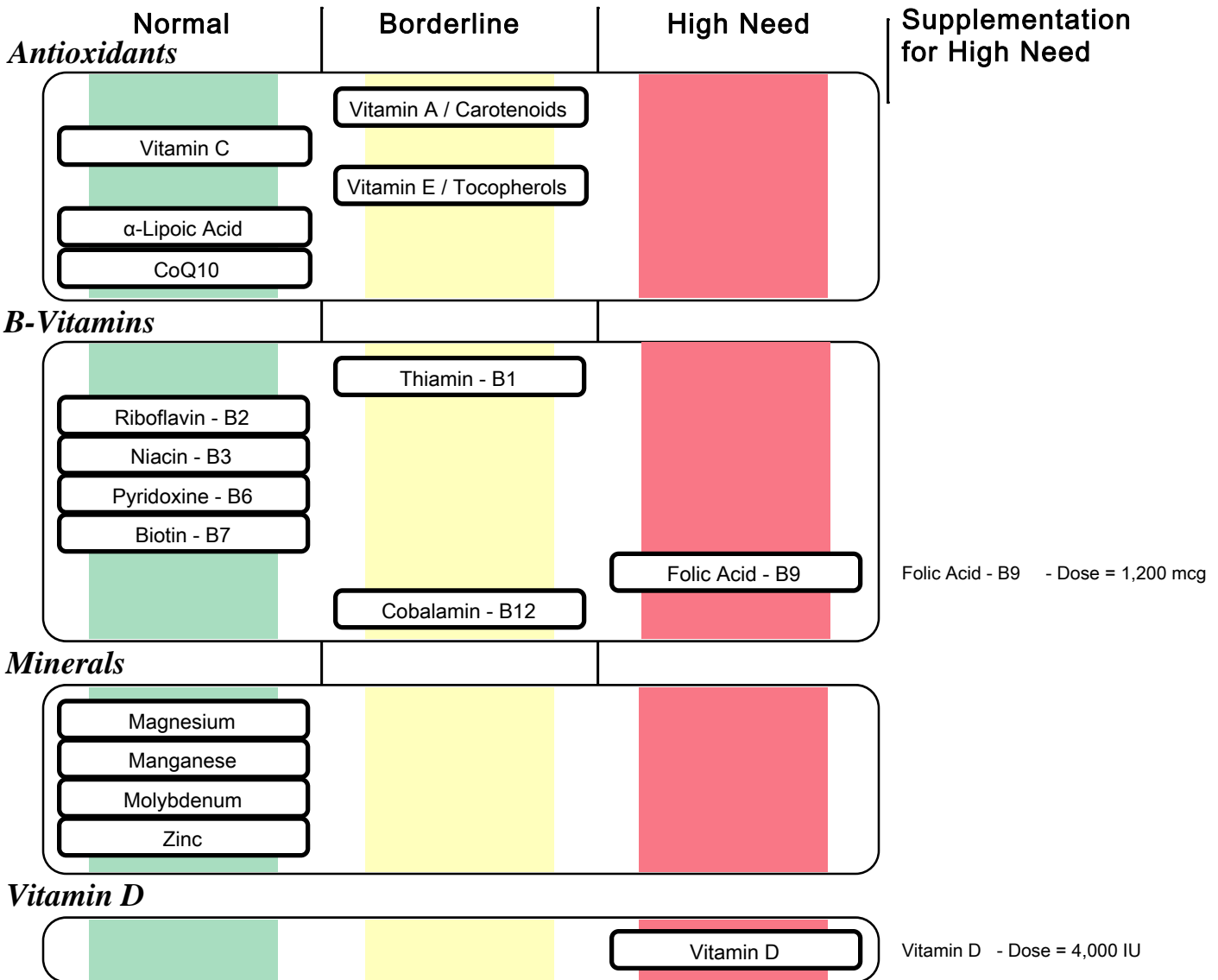




Patient: **FEMALE**
TEST

NutrEval Results Overview



SUGGESTED SUPPLEMENT SCHEDULE

Supplements	Daily Recommended Intake (DRI)	Patient's Daily Recommendations	Provider Daily Recommendations
Antioxidants			
Vitamin A / Carotenoids	2,333 IU	5,000 IU	
Vitamin C	75 mg	250 mg	
Vitamin E / Tocopherols	22 IU	200 IU	
α-Lipoic Acid		50 mg	
CoQ10		30 mg	
B-Vitamins			
Thiamin - B1	1.1 mg	25 mg	
Riboflavin - B2	1.1 mg	10 mg	
Niacin - B3	14 mg	20 mg	
Pyridoxine - B6	1.5 mg	10 mg	
Biotin - B7	30 mcg	100 mcg	
Folic Acid - B9	400 mcg	1,200 mcg	
Cobalamin - B12	2.4 mcg	500 mcg	
Minerals			
Magnesium	320 mg	400 mg	
Manganese	1.8 mg	3.0 mg	
Molybdenum	45 mcg	75 mcg	
Zinc	8 mg	10 mg	
Essential Fatty Acids			
Omega-3 Oils	500 mg	1,000 mg	
Digestive Support			
Probiotics		50 billion CFU	
Pancreatic Enzymes		10,000 IU	
Other Vitamins			
Vitamin D	800 IU	4,000 IU	
Amino Acid		Amino Acid	
	mg/day		mg/day
Arginine	0	Methionine	0
Asparagine	0	Phenylalanine	0
Cysteine	0	Serine	0
Glutamine	334	Taurine	0
Glycine	327	Threonine	0
Histidine	0	Tryptophan	0
Isoleucine	0	Tyrosine	0
Leucine	0	Valine	0
Lysine	0		

Recommendations for age and gender-specific supplementation are set by comparing levels of nutrient functional need to optimal levels as described in the peer-reviewed literature. They are provided as guidance for short-term support of nutritional deficiencies only.

The Suggested Supplemental Schedule is provided at the request of the ordering practitioner. Any application of it as a therapeutic intervention is to be determined by the ordering practitioner.

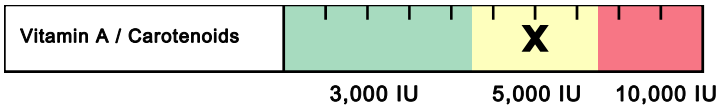
Key

Normal	Borderline	High Need

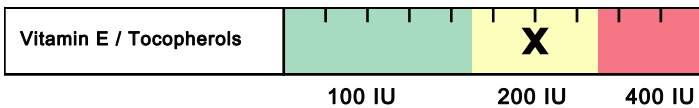
NutrEval *Interpretation At-A-Glance*
FMV amino acids

Nutritional Needs

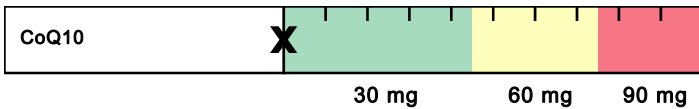
Antioxidants



- ▶ Beta-carotene & other carotenoids are converted to vitamin A (retinol), involved in vision, antioxidant & immune function, gene expression & cell growth.
- ▶ Vitamin A deficiency may occur with chronic alcoholism, zinc deficiency, hypothyroidism, or oral contraceptives containing estrogen & progesterin.
- ▶ Deficiency may result in night blindness, impaired immunity, healing & tissue regeneration, increased risk of infection, leukoplakia or keratosis.
- ▶ Food sources include cod liver oil, fortified cereals & milk, eggs, sweet potato, pumpkin, carrot, cantaloupe, mango, spinach, broccoli, kale & butternut squash.



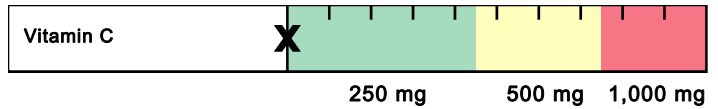
- ▶ Alpha-tocopherol (body's main form of vitamin E) functions as an antioxidant, regulates cell signaling, influences immune function and inhibits coagulation.
- ▶ Deficiency may occur with malabsorption, cholestyramine, colestipol, isoniazid, orlistat, olestra and certain anti-convulsants (e.g., phenobarbital, phenytoin).
- ▶ Deficiency may result in peripheral neuropathy, ataxia, muscle weakness, retinopathy, and increased risk of CVD, prostate cancer and cataracts.
- ▶ Food sources include oils (olive, soy, corn, canola, safflower, sunflower), eggs, nuts, seeds, spinach, carrots, avocado, dark leafy greens and wheat germ.



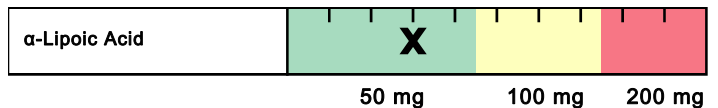
- ▶ CoQ10 is a powerful antioxidant that is synthesized in the body and contained in cell membranes. CoQ10 is also essential for energy production & pH regulation.
- ▶ CoQ10 deficiency may occur with HMG-CoA reductase inhibitors (statins), several anti-diabetic medication classes (biguanides, sulfonylureas) or beta-blockers.
- ▶ Low levels may aggravate oxidative stress, diabetes, cancer, congestive heart failure, cardiac arrhythmias, gingivitis and neurologic diseases.
- ▶ Main food sources include meat, poultry, fish, soybean, canola oil, nuts and whole grains. Moderate sources include fruits, vegetables, eggs and dairy.



- ▶ Oxidative stress is the imbalance between the production of free radicals and the body's ability to readily detoxify these reactive species and/or repair the resulting damage with anti-oxidants.
- ▶ Oxidative stress can be endogenous (energy production and inflammation) or exogenous (exercise, exposure to environmental toxins).
- ▶ Oxidative stress has been implicated clinically in the development of neurodegenerative diseases, cardiovascular diseases and chronic fatigue syndrome.
- ▶ Antioxidants may be found in whole food sources (e.g., brightly colored fruits & vegetables, green tea, turmeric) as well as nutraceuticals (e.g., resveratrol, EGCG, lutein, lycopene, ginkgo, milk thistle, etc.).



- ▶ Vitamin C is an antioxidant (also used in the regeneration of other antioxidants). It is involved in cholesterol metabolism, the production & function of WBCs and antibodies, and the synthesis of collagen, norepinephrine and carnitine.
- ▶ Deficiency may occur with oral contraceptives, aspirin, diuretics or NSAIDs.
- ▶ Deficiency can result in scurvy, swollen gingiva, periodontal destruction, loose teeth, sore mouth, soft tissue ulcerations, or increased risk of infection.
- ▶ Food sources include oranges, grapefruit, strawberries, tomato, sweet red pepper, broccoli and potato.



- ▶ α-Lipoic acid plays an important role in energy production, antioxidant activity (including the regeneration of vitamin C and glutathione), insulin signaling, cell signaling and the catabolism of α-keto acids and amino acids.
- ▶ High biotin intake can compete with lipoic acid for cell membrane entry.
- ▶ Optimal levels of α-lipoic acid may improve glucose utilization and protect against diabetic neuropathy, vascular disease and age-related cognitive decline.
- ▶ Main food sources include organ meats, spinach and broccoli. Lesser sources include tomato, peas, Brussels sprouts and brewer's yeast.



- ▶ Glutathione (GSH) is composed of cysteine, glutamine & glycine. GSH is a source of sulfate and plays a key role in antioxidant activity and detoxification of toxins.
- ▶ GSH requirement is increased with high-fat diets, cigarette smoke, cystinuria, chronic alcoholism, chronic acetaminophen use, infection, inflammation and toxic exposure.
- ▶ Deficiency may result in oxidative stress & damage, impaired detoxification, altered immunity, macular degeneration and increased risk of chronic illness.
- ▶ Food sources of GSH precursors include meats, poultry, fish, soy, corn, nuts, seeds, wheat germ, milk and cheese.

Key

- ▶ Function
- ▶ Causes of Deficiency
- ▶ Complications of Deficiency
- ▶ Food Sources

Nutritional Needs

B-Vitamins



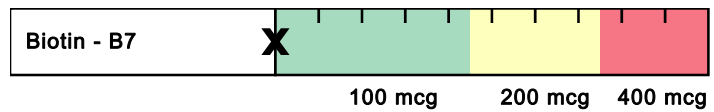
- ▶ B1 is a required cofactor for enzymes involved in energy production from food, and for the synthesis of ATP, GTP, DNA, RNA and NADPH.
- ▶ Low B1 can result from chronic alcoholism, diuretics, digoxin, oral contraceptives and HRT, or large amounts of tea & coffee (contain anti-B1 factors).
- ▶ B1 deficiency may lead to dry beriberi (e.g., neuropathy, muscle weakness), wet beriberi (e.g., cardiac problems, edema), encephalopathy or dementia.
- ▶ Food sources include lentils, whole grains, wheat germ, Brazil nuts, peas, organ meats, brewer's yeast, blackstrap molasses, spinach, milk & eggs.



- ▶ B6 (as P5P) is a cofactor for enzymes involved in glycogenolysis & gluconeogenesis, and synthesis of neurotransmitters, heme, B3, RBCs and nucleic acids.
- ▶ Low B6 may result from chronic alcoholism, long-term diuretics, estrogens (oral contraceptives and HRT), anti-TB meds, penicillamine, L-DOPA or digoxin.
- ▶ B6 deficiency may result in neurologic symptoms (e.g., irritability, depression, seizures), oral inflammation, impaired immunity or increased homocysteine.
- ▶ Food sources include poultry, beef, beef liver, fish, whole grains, wheat germ, soybean, lentils, nuts & seeds, potato, spinach and carrots.



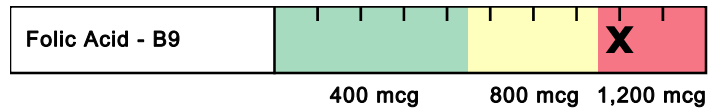
- ▶ B2 is a key component of enzymes involved in antioxidant function, energy production, detoxification, methionine metabolism and vitamin activation.
- ▶ Low B2 may result from chronic alcoholism, some anti-psychotic medications, oral contraceptives, tricyclic antidepressants, quinacrine or adriamycin.
- ▶ B2 deficiency may result in oxidative stress, mitochondrial dysfunction, low uric acid, low B3 or B6, high homocysteine, anemia or oral & throat inflammation.
- ▶ Food sources include milk, cheese, eggs, whole grains, beef, chicken, wheat germ, fish, broccoli, asparagus, spinach, mushrooms and almonds.



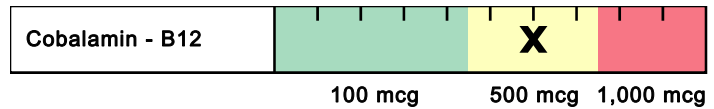
- ▶ Biotin is a cofactor for enzymes involved in functions such as fatty acid synthesis, mitochondrial FA oxidation, gluconeogenesis and DNA replication & transcription.
- ▶ Deficiency may result from certain inborn errors, chronic intake of raw egg whites, long-term TPN, anticonvulsants, high-dose B5, sulfa drugs & other antibiotics.
- ▶ Low levels may result in neurologic symptoms (e.g., paresthesias, depression), hair loss, scaly rash on face or genitals or impaired immunity.
- ▶ Food sources include yeast, whole grains, wheat germ, eggs, cheese, liver, meats, fish, wheat, nuts & seeds, avocado, raspberries, sweet potato and cauliflower.



- ▶ B3 is used to form NAD and NADP, involved in energy production from food, fatty acid & cholesterol synthesis, cell signaling, DNA repair & cell differentiation.
- ▶ Low B3 may result from deficiencies of tryptophan (B3 precursor), B6, B2 or Fe (cofactors in B3 production), or from long-term isoniazid or oral contraceptive use.
- ▶ B3 deficiency may result in pellagra (dermatitis, diarrhea, dementia), neurologic symptoms (e.g., depression, memory loss), bright red tongue or fatigue.
- ▶ Food sources include poultry, beef, organ meats, fish, whole grains, peanuts, seeds, lentils, brewer's yeast and lima beans.



- ▶ Folic acid plays a key role in coenzymes involved in DNA and SAMe synthesis, methylation, nucleic acids & amino acid metabolism and RBC production.
- ▶ Low folate may result from alcoholism, high-dose NSAIDs, diabetic meds, H2 blockers, some diuretics and anti-convulsants, SSRIs, methotrexate, trimethoprim, pyrimethamine, triamterene, sulfasalazine or cholestyramine.
- ▶ Folate deficiency can result in anemia, fatigue, low methionine, increased homocysteine, impaired immunity, heart disease, birth defects and CA risk.
- ▶ Food sources include fortified grains, green vegetables, beans & legumes.



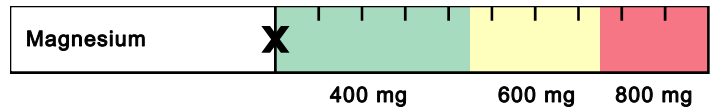
- ▶ B12 plays important roles in energy production from fats & proteins, methylation, synthesis of hemoglobin & RBCs, and maintenance of nerve cells, DNA & RNA.
- ▶ Low B12 may result from alcoholism, malabsorption, hypochlorhydria (e.g., from atrophic gastritis, H. pylori infection, pernicious anemia, H2 blockers, PPIs), vegan diets, diabetic meds, cholestyramine, chloramphenicol, neomycin or colchicine.
- ▶ B12 deficiency can lead to anemia, fatigue, neurologic symptoms (e.g., paresthesias, memory loss, depression, dementia), methylation defects or chromosome breaks.
- ▶ Food sources include shellfish, red meat poultry, fish, eggs, milk and cheese.

Nutritional Needs

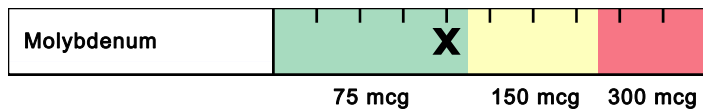
Minerals



- ▶ Manganese plays an important role in antioxidant function, gluconeogenesis, the urea cycle, cartilage & bone formation, energy production and digestion.
- ▶ Impaired absorption of Mn may occur with excess intake of Fe, Ca, Cu, folic acid, or phosphorous compounds, or use of long-term TPN, Mg-containing antacids or laxatives.
- ▶ Deficiency may result in impaired bone/connective tissue growth, glucose & lipid dysregulation, infertility, oxidative stress, inflammation or hyperammonemia.
- ▶ Food sources include whole grains, legumes, dried fruits, nuts, dark green leafy vegetables, liver, kidney and tea.



- ▶ Magnesium is involved in >300 metabolic reactions. Key areas include energy production, bone & ATP formation, muscle & nerve conduction and cell signaling.
- ▶ Deficiency may occur with malabsorption, alcoholism, hyperparathyroidism, renal disorders (wasting), diabetes, diuretics, digoxin or high doses of zinc.
- ▶ Low Mg may result in muscle weakness/spasm, constipation, depression, hypertension, arrhythmias, hypocalcemia, hypokalemia or personality changes.
- ▶ Food sources include dark leafy greens, oatmeal, buckwheat, unpolished grains, chocolate, milk, nuts & seeds, lima beans and molasses.

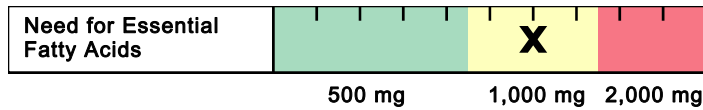


- ▶ Molybdenum is a cofactor for enzymes that convert sulfites to sulfate, and nucleotides to uric acid, and that help metabolize aldehydes & other toxins.
- ▶ Low Mo levels may result from long-term TPN that does not include Mo.
- ▶ Mo deficiency may result in increased sulfite, decreased plasma uric acid (and antioxidant function), deficient sulfate, impaired sulfation (detoxification), neurologic disorders or brain damage (if severe deficiency).
- ▶ Food sources include buckwheat, beans, grains, nuts, beans, lentils, meats and vegetables (although Mo content of plants depends on soil content).



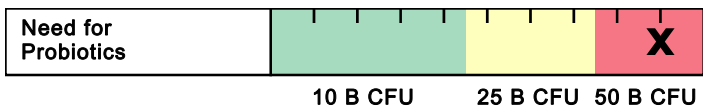
- ▶ Zinc plays a vital role in immunity, protein metabolism, heme synthesis, growth & development, reproduction, digestion and antioxidant function.
- ▶ Low levels may occur with malabsorption, alcoholism, chronic diarrhea, diabetes, excess Cu or Fe, diuretics, ACE inhibitors, H2 blockers or digoxin.
- ▶ Deficiency can result in hair loss and skin rashes, also impairments in growth & healing, immunity, sexual function, taste & smell and digestion.
- ▶ Food sources include oysters, organ meats, soybean, wheat germ, seeds, nuts, red meat, chicken, herring, milk, yeast, leafy and root vegetables.

Essential Fatty Acids

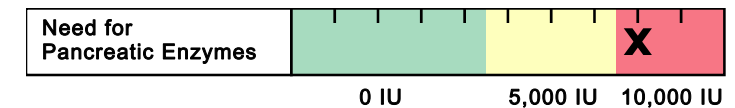


- ▶ Omega-3 (O3) and Omega-6 (O6) fatty acids are polyunsaturated fatty acids that cannot be synthesized by the human body. They are classified as essential nutrients and must be obtained from dietary sources.
- ▶ The standard American diet is much higher in O6 than O3 fatty acids. Deficiency of EFAs may result from poor dietary intake and/or poor conversion from food sources.
- ▶ EFA deficiency is associated with decreased growth & development of infants and children, dry skin/rash, poor wound healing, and increased risk of infection, cardiovascular and inflammatory diseases.
- ▶ Dietary sources of the O6 Linoleic Acid (LA) include vegetable oils, nuts, seeds and some vegetables. Dietary sources of the O3 a-Linolenic Acid (ALA) include flaxseeds, walnuts, and their oils. Fish (mackerel, salmon, sardines) are the major dietary sources of the O3 fatty acids EPA and DHA.

Digestive Support



- ▶ Probiotics have many functions. These include: production of some B vitamins and vitamin K; enhance digestion & absorption; decrease severity of diarrheal illness; modulate of immune function & intestinal permeability.
- ▶ Alterations of gastrointestinal microflora may result from C-section delivery, antibiotic use, improved sanitation, decreased consumption of fermented foods and use of certain drugs.
- ▶ Some of the diseases associated with microflora imbalances include: IBS, IBD, fibromyalgia, chronic fatigue syndrome, obesity, atopic illness, colic and cancer.
- ▶ Food sources rich in probiotics are yogurt, kefir and fermented foods.



- ▶ Pancreatic enzymes are secreted by the exocrine glands of the pancreas and include protease/peptidase, lipase and amylase.
- ▶ Pancreatic exocrine insufficiency may be primary or secondary in nature. Any indication of insufficiency warrants further evaluation for underlying cause (i.e., celiac disease, small intestine villous atrophy, small bowel bacterial overgrowth).
- ▶ A high functional need for digestive enzymes suggests that there is an impairment related to digestive capacity.
- ▶ Determining the strength of the pancreatic enzyme support depends on the degree of functional impairment. Supplement potency is based on the lipase units present in both prescriptive and non-prescriptive agents.



Interpretation At-A-Glance

Functional Imbalances



- Mitochondria are a primary site of generation of reactive oxygen species. Oxidative damage is considered an important factor in decline of physiologic function that occurs with aging and stress.
- Mitochondrial defects have been identified in cardiovascular disease, fatigue syndromes, neurologic disorders such as Parkinson's and Alzheimer's disease, as well as a variety of genetic conditions. Common nutritional deficiencies can impair mitochondrial efficiency.

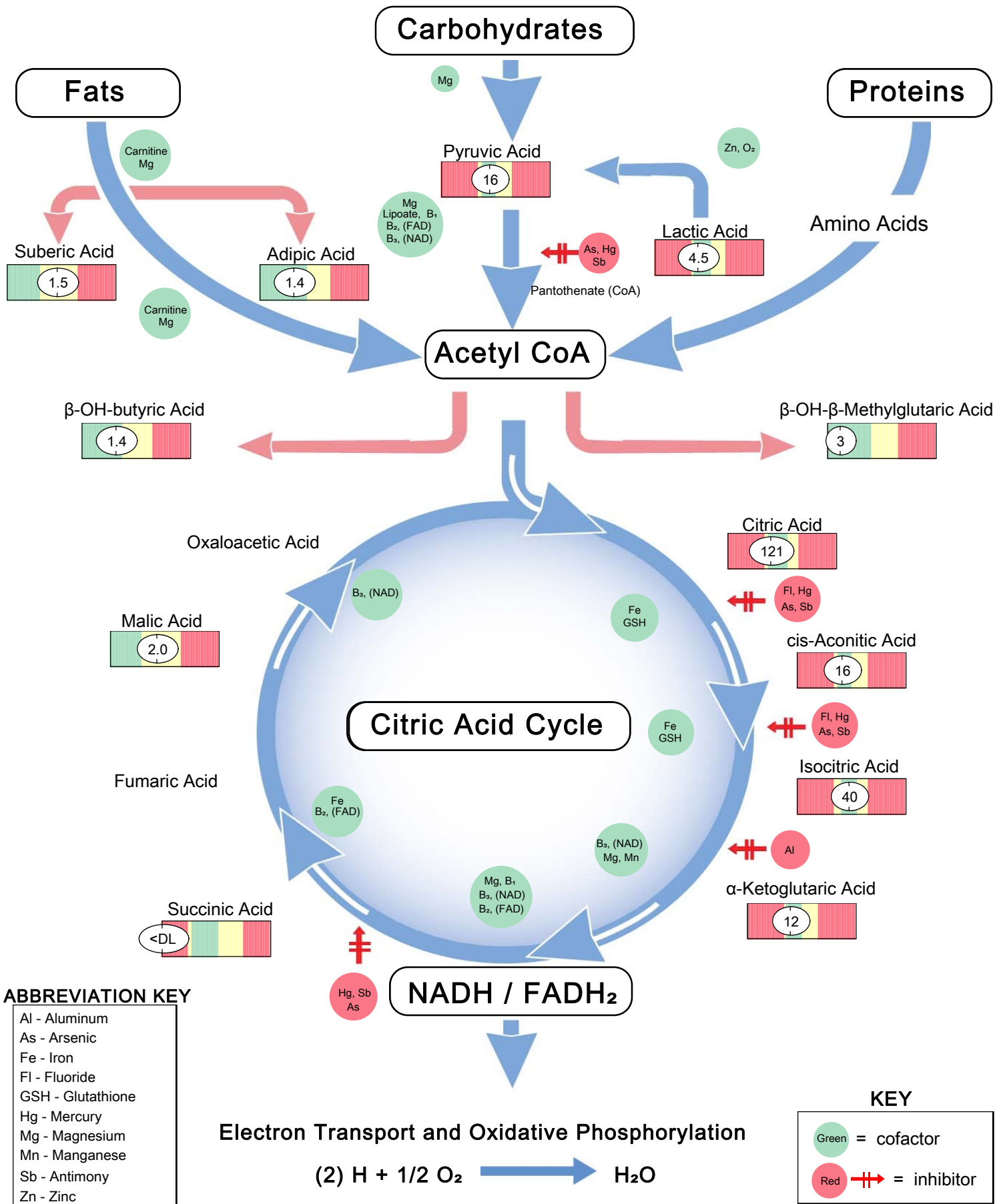


- Methylation is an enzymatic process that is critical for both synthesis and inactivation. DNA, estrogen and neurotransmitter metabolism are all dependent on appropriate methylation activity.
- B vitamins and other nutrients (methionine, magnesium, selenium) functionally support catechol-O-methyltransferase (COMT), the enzyme responsible for methylation.



- Methyl tert-Butyl Ether (MTBE) is a common gasoline additive used to increase octane ratings, and has been found to contaminate ground water supplies where gasoline is stored. Inhalation of MTBE may cause nose and throat irritation, as well as headaches, nausea, dizziness and mental confusion. Animal studies suggest that drinking MTBE may cause gastrointestinal irritation, liver and kidney damage and nervous system effects.
- Styrene is classified by the US EPA as a "potential human carcinogen," and is found widely distributed in commercial products such as rubber, plastic, insulation, fiberglass, pipes, food containers and carpet backing.
- Levels of these toxic substances should be examined within the context of the body's functional capacity for methylation and need for glutathione.

Krebs Cycle At-A-Glance



All biomarkers reported in mmol/mol creatinine unless otherwise noted. **Metabolic Analysis Markers (Urine)**

Malabsorption and Dysbiosis Markers

Malabsorption Markers	Reference Range
Indoleacetic Acid (IAA)	1.5 <= 4.2
Phenylacetic Acid (PAA)	0.13 <= 0.12

Bacterial Dysbiosis Markers	Reference Range
Dihydroxyphenylpropionic Acid (DHPPA)	8.5 <= 5.3
3-Hydroxyphenylacetic Acid	3.9 <= 8.1
4-Hydroxyphenylacetic Acid	19 <= 29
Benzoic Acid	0.06 <= 0.05
Hippuric Acid	166 <= 603

Yeast / Fungal Dysbiosis Markers	Reference Range
Arabinose	27 <= 96
Citramalic Acid	2.4 <= 5.8
Tartaric Acid	<DL <= 15

Cellular Energy & Mitochondrial Metabolites

Carbohydrate Metabolism	Reference Range
Lactic Acid	4.5 1.9-19.8
Pyruvic Acid	16 7-32
β-OH-Butyric Acid (BHBA)	1.4 <= 2.8

Energy Metabolism	Reference Range
Citric Acid	121 40-520
Cis-Aconitic Acid	16 10-36
Isocitric Acid	40 22-65
α-Ketoglutaric Acid (AKG)	12 4-52
Succinic Acid	<DL 0.4-4.6
Malic Acid	2.0 <= 3.0
β-OH-β-Methylglutaric Acid (HMG)	3 <= 15

Fatty Acid Metabolism	Reference Range
Adipic Acid	1.4 <= 2.8
Suberic Acid	1.5 <= 2.1

Creatinine Concentration

Reference Range
Creatinine ♦ 12.0 3.1-19.5 mmol/L

Methodology: GCMS, LC/MS/MS, Alkaline Picrate

The performance characteristics of all assays have been verified by Genova Diagnostics, Inc. Unless otherwise noted with ♦, the assay has not been cleared by the U.S. Food and Drug Administration.

Neurotransmitter Metabolites

Reference Range
Vanilmandelic Acid 1.2 0.4-3.6
Homovanillic Acid 2.2 1.2-5.3
5-OH-indoleacetic Acid 9.6 3.8-12.1
3-Methyl-4-OH-phenylglycol 0.09 0.02-0.22
Kynurenic Acid 4.5 <= 7.1
Quinolinic Acid 3.3 <= 9.1
Kynurenic / Quinolinic Ratio 1.36 >= 0.44

Vitamin Markers

Reference Range
α-Ketoadipic Acid 0.8 <= 1.7
α-Ketoisovaleric Acid 0.50 <= 0.97
α-Ketoisocaproic Acid 0.46 <= 0.89
α-Keto-β-Methylvaleric Acid 1.5 <= 2.1
Formiminoglutamic Acid (FIGlu) 2.5 <= 1.5
Glutaric Acid 0.30 <= 0.51
Isovalerylglycine 2.0 <= 3.7
Methylmalonic Acid 0.9 <= 1.9
Xanthurenic Acid 0.37 <= 0.96
3-Hydroxypropionic Acid 10 5-22
3-Hydroxyisovaleric Acid 15 <= 29

Toxin & Detoxification Markers

Reference Range
α-Ketophenylacetic Acid (from Styrene) 0.24 <= 0.46
α-Hydroxyisobutyric Acid (from MTBE) 3.9 <= 6.7
Orotic Acid 0.82 0.33-1.01
Pyroglutamic Acid 18 16-34

Tyrosine Metabolism

Reference Range
Homogentisic Acid 12 <= 19
2-Hydroxyphenylacetic Acid 0.40 <= 0.76

Metabolic Analysis Reference Ranges are Age Specific

Amino Acids (Urine FMV)

All biomarkers reported in micromol/g creatinine unless otherwise noted.

Nutritionally Essential Amino Acids

Amino Acid	Reference Range
Arginine	10 (3-43)
Histidine	255 (124-894)
Isoleucine	16 (3-28)
Leucine	37 (4-46)
Lysine	81 (11-175)
Methionine	9 (2-18)
Phenylalanine	70 (8-71)
Taurine	457 (21-424)
Threonine	64 (17-135)
Tryptophan	50 (5-53)
Valine	28 (7-49)

Nonessential Protein Amino Acids

Amino Acid	Reference Range
Alanine	169 (63-356)
Asparagine	56 (25-166)
Aspartic Acid	<DL (<= 14)
Cysteine (FMV urine)	25 (8-74)
Cystine (FMV Urine)	56 (10-104)
γ-Aminobutyric Acid	1 (<= 5)
Glutamic Acid	8 (4-27)
Glutamine	173 (110-632)
Proline	7 (1-13)
Tyrosine	163 (11-135)

Creatinine Concentration

Reference Range
Creatinine ♦ 11.9 (3.1-19.5 mmol/L)

Amino Acid reference ranges are age specific.

The performance characteristics of all assays have been verified by Genova Diagnostics, Inc. Unless otherwise noted with ♦, the assays have not been cleared by the U.S. Food and Drug Administration.

Methodology: LC/MS/MS, Alkaline Picrate

Intermediary Metabolites

B Vitamin Markers	Reference Range
α-Aminoadipic	43 (2-47)
α-Amino-N-butyric Acid	11 (2-25)
β-Aminoisobutyric Acid	84 (11-160)
Cystathionine	23 (2-68)
3-Methylhistidine	160 (44-281)

Urea Cycle Markers

Citrulline	1.4 (0.6-3.9)
Ornithine	9 (2-21)
Urea ♦	264 (168-465 mmol/g creatinine)

Glycine/Serine Metabolites

Glycine	176 (95-683)
Serine	129 (40-163)
Ethanolamine	91 (50-235)
Phosphoethanolamine	5 (1-13)
Phosphoserine	7 (3-13)
Sarcosine	1.1 (<= 1.1)

Dietary Peptide Related Markers

Reference Range
Anserine (dipeptide) 60.5 (0.4-105.1)
Carnosine (dipeptide) 29 (1-28)
1-Methylhistidine 923 (38-988)
β-Alanine 25 (<= 22)

Essential and Metabolic Fatty Acids Markers (RBCs)

Omega 3 Fatty Acids		
Analyte	(cold water fish, flax, walnut)	Reference Range
α-Linolenic (ALA) 18:3 n3	0.13	>= 0.09 wt %
Eicosapentaenoic (EPA) 20:5 n3	0.59	>= 0.16 wt %
Docosapentaenoic (DPA) 22:5 n3	1.79	>= 1.14 wt %
Docosahexaenoic (DHA) 22:6 n3	3.2	>= 2.1 wt %
% Omega 3s	5.8	>= 3.8

Omega 9 Fatty Acids		
Analyte	(olive oil)	Reference Range
Oleic 18:1 n9	11	10-13 wt %
Nervonic 24:1 n9	2.2	2.1-3.5 wt %
% Omega 9s	13.7	13.3-16.6

Saturated Fatty Acids		
Analyte	(meat, dairy, coconuts, palm oils)	Reference Range
Palmitic C16:0	21	18-23 wt %
Stearic C18:0	20	14-17 wt %
Arachidic C20:0	0.28	0.22-0.35 wt %
Behenic C22:0	0.78	0.92-1.68 wt %
Tricosanoic C23:0	0.17	0.12-0.18 wt %
Lignoceric C24:0	2.5	2.1-3.8 wt %
Pentadecanoic C15:0	0.08	0.07-0.15 wt %
Margaric C17:0	0.28	0.22-0.37 wt %
% Saturated Fats	44.6	39.8-43.6

Methodology: GCMS

Omega 6 Fatty Acids		
Analyte	(vegetable oil, grains, most meats, dairy)	Reference Range
Linoleic (LA) 18:2 n6	14.1	10.5-16.9 wt %
γ-Linolenic (GLA) 18:3 n6	0.07	0.03-0.13 wt %
Dihomo-γ-linolenic (DGLA) 20:3 n6	1.70	>= 1.19 wt %
Arachidonic (AA) 20:4 n6	16	15-21 wt %
Docosatetraenoic (DTA) 22:4 n6	2.60	1.50-4.20 wt %
Eicosadienoic 20:2 n6	0.32	<= 0.26 wt %
% Omega 6s	34.4	30.5-39.7

Monounsaturated Fats		
Analyte		Reference Range
Palmitoleic 16:1 n7	0.30	<= 0.64 wt %
Vaccenic 18:1 n7	0.85	<= 1.13 wt %
Trans Fat		
Elaidic 18:1 n9t	0.38	<= 0.59 wt %

Delta - 6 Desaturase Activity		
Analyte	Upregulated Functional Impaired	Reference Range
Linoleic / DGLA 18:2 n6 / 20:3 n6	8.3	6.0-12.3

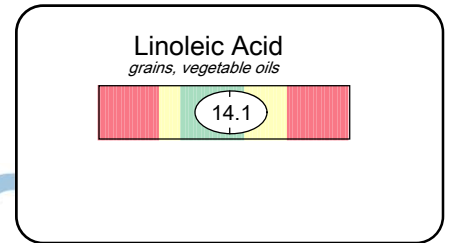
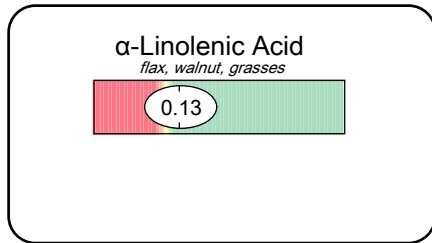
Cardiovascular Risk		
Analyte		Reference Range
Omega 6s / Omega 3s	6.0	3.4-10.7
AA / EPA 20:4 n6 / 20:5 n3	26	12-125
Omega 3 Index	3.8	>= 4.0

The Essential Fatty Acid reference ranges are based on an adult population.

Essential Fatty Acid Metabolism

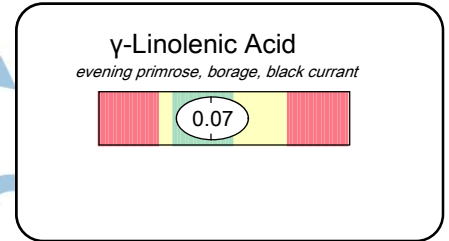
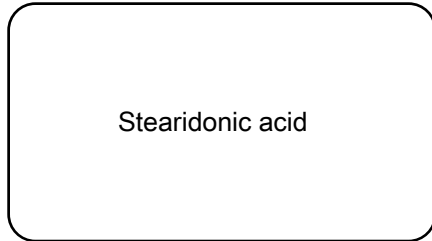
Omega 3 Family

Omega 6 Family



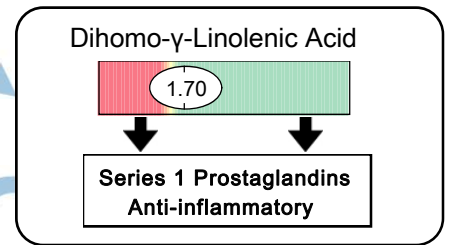
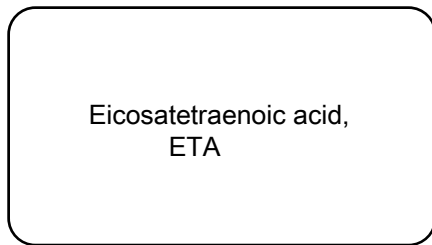
Delta-6 Desaturase

Vitamin and Mineral Cofactors:
FAD (B2), Niacin (B3)
Pyridoxal-5-phosphate (B6)
Vitamin C, Insulin, Zn, Mg



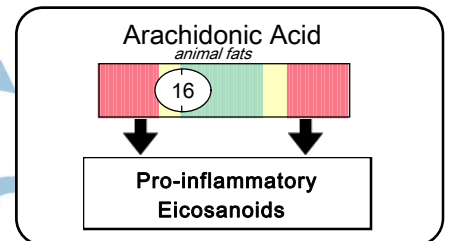
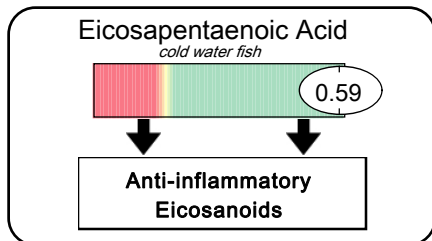
Elongase

Vitamin and Mineral Cofactors:
Niacin (B3)
Pyridoxal-5-phosphate (B6)
Pantothenic Acid (B5)
Biotin, Vitamin C



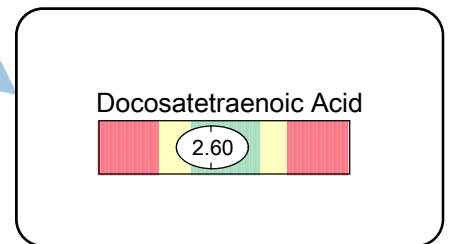
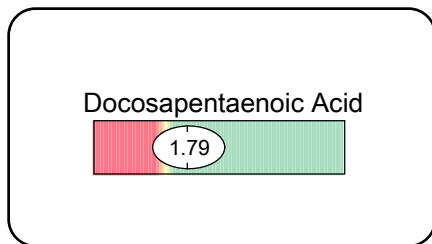
Delta-5 Desaturase

Vitamin and Mineral Cofactors:
FAD (B2), Niacin (B3)
Pyridoxal-5-phosphate (B6)
Vitamin C, Insulin, Zn, Mg



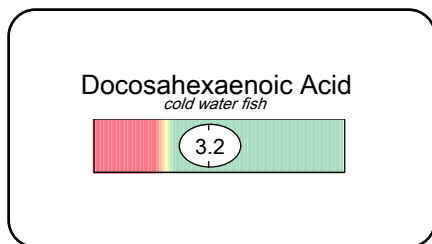
Elongase

Vitamin and Mineral Cofactors:
Niacin (B3)
Pyridoxal-5-phosphate (B6), Biotin
Pantothenic Acid (B5), Vitamin C



Elongase Delta-6 Desaturase

Vitamin and Mineral Cofactors:
FAD (B2), Niacin (B3)
Pyridoxal-5-phosphate (B6), Biotin
Vitamin C, Zn, Mg, Carnitine
Pantothenic Acid (B5)



This test was developed and its performance characteristics determined by Genova Diagnostics, Inc. It has not been cleared by the U.S. Food and Drug Administration.

Oxidative Stress Markers

Oxidative Stress Markers

Reference Range

Methodology: Colorimetric, thiobarbituric acid reactive substances (TBARS), Alkaline Picrate, Hexokinase/G-6-PDH, LC/MS/MS, HPLC

Glutathione (whole blood)		>=669 micromol/L
Lipid Peroxides (urine)		<=10.0 micromol/g Creat.
8-OHdG (urine)		<=15 mcg/g Creat.
Coenzyme Q10, Ubiquinone (serum)		0.43-1.49 mcg/mL

The Oxidative Stress reference ranges are based on an adult population. The performance characteristics of the Oxidative Stress Markers have been verified by Genova Diagnostics, Inc. They have not been cleared by the U.S. Food and Drug Administration.

Vitamin D (Serum)

Inside Range Outside Range Reference Range

Methodology: Chemiluminescent

25 - OH Vitamin D ♦		50-100 ng/mL
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Deficiency = < 20 ng/mL (< 50 nmol/L)
 Insufficiency = 20-49 ng/mL (50-124 nmol/L)
 Optimal = 50-100 ng/mL (125-250 nmol/L)
 Excessive = > 100 ng/mL (> 250 nmol/L)

Elemental Markers

Nutrient Elements

Element Reference Range Reference Range

Copper (plasma)		75.3-192.0 mcg/dL
Magnesium (RBC)		30.1-56.5 mcg/g
Manganese (whole blood)		3.0-16.5 mcg/L
Potassium (RBC)		2,220-3,626 mcg/g
Selenium (whole blood)		109-330 mcg/L
Zinc (plasma)		64.3-159.4 mcg/dL

The Elemental reference ranges are based on an adult population.

The performance characteristics of the Elemental Markers have been verified by Genova Diagnostics, Inc. They have not been cleared by the U.S. Food and Drug Administration.

Elemental testing performed by Genova Diagnostics, Inc. 3425 Corporate Way, Duluth, GA 30096 - Robert M. David, PhD, Lab Director - CLIA Lic. #11D0255349 - Medicare Lic. #34-8475

Toxic Elements*

Element Reference Range Reference Range

Lead		<= 2.81 mcg/dL
Mercury		<= 4.35 mcg/L
Arsenic		<= 13.7 mcg/L
Cadmium		<= 1.22 mcg/L
Tin		<= 0.39 mcg/L

* All toxic Elements are measured in whole blood.

Methodology: ICP-MS

Commentary

Please note the reference range for 8-OHdG (urine) has been updated.

Interpretation At-A-Glance Details

Antioxidants

Vitamin A / Carotenoids

Contributing Biomarkers:

β-Alanine
Cystine
Cysteine
Lipid Peroxides
Taurine

Vitamin E / Tocopherols

Contributing Biomarkers:

β-Alanine
Cystine
Cysteine
Lipid Peroxides
Taurine

α-Lipoic Acid

Contributing Biomarkers:

Lipid Peroxides

Glutathione

Contributing Biomarkers:

Lipid Peroxides
Pyroglutamic Acid

Plant-based Antioxidants

Contributing Biomarkers:

Cystine
Cysteine
Lipid Peroxides
Taurine

B-Vitamins

Thiamin - B1

Contributing Biomarkers:

5-OH-Indoleacetic Acid
α-Keto-β-Methylvaleric Acid
Taurine
Tyrosine

Interpretation At-A-Glance Details

Pyridoxine - B6

Contributing Biomarkers:
 β-Alanine
 Cysteine
 Tyrosine

Folic Acid - B9

Contributing Biomarkers:
 Formiminoglutamic Acid

Cobalamin - B12

Contributing Biomarkers:
 Cysteine
 Formiminoglutamic Acid
 Succinic Acid

Minerals

Manganese

Contributing Biomarkers:
 5-OH-Indoleacetic Acid

Molybdenum

Contributing Biomarkers:
 Taurine

Ethanolamine

Phosphoetr

Essential Fatty Acids

**Need for
 Essential Fatty Acids**

Contributing Biomarkers:
 Omega 3 Index

Digestive Support

Interpretation At-A-Glance Details**Need for Probiotics****Contributing Biomarkers:**

Benzoic Acid

 β -Alanine

Dihydroxyphenylpropionic Acid

Phenylacetic Acid

**Need for
Pancreatic Enzymes****Contributing Biomarkers:**

1-Methylhistidine

Dihydroxyphenylpropionic Acid

Phenylacetic Acid

Functional Imbalances**Mitochondrial Dysfunction****Contributing Biomarkers:**

Formiminoglutamic Acid

Need for Methylation**Contributing Biomarkers:**

Formiminoglutamic Acid

Metabolic Analysis Markers

Commentary

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.

Phenylacetic Acid (PAA) is elevated. If the essential amino acid phenylalanine is not sufficiently digested and absorbed in the small intestine, it is carried to the large bowel where anaerobic bacteria convert it to phenylethylamine. This is then absorbed, and in body tissues such as the liver, it is converted by deamination to PAA, which is excreted in the urine. Some species of Clostridia can produce PAA directly from aromatic amino acids. Its presence at elevated levels indicates one or more of the following: gastric hypochlorhydria or pepsin inactivity, impaired digestive peptidase function in the small intestine, rate-limited or insufficient absorption or mucosal transport in the small intestine, abnormal intestinal motility (partly regulated by cholecystokinin and secretin), or presence of colonic or other bacteria in the small intestine (dysbiosis).

Additionally, some elevation of PAA may occur in the uncommon instances of phenylketonuria and with Type I tyrosinemia (tyrosinosis). With phenylketonuria, 2-hydroxyphenylacetate (2-HPAA) would be significantly elevated. An amino acid analysis also is helpful in diagnosing such conditions.

2,3 Dihydroxyphenylpropionic acid (DHPPA) is elevated. This organic acid is a byproduct of the bacterial metabolism of phenylalanine, tyrosine, and/or tryptophan. Research has identified various species of Clostridia in the *in-vitro* production of this compound. Other research on quinoline demonstrates production of DHPPA by Pseudomonas species. Presence of elevated levels of DHPPA in the urine may thus suggest overgrowth of Clostridia and/or Pseudomonas, as well as a degree of malabsorption of aromatic amino acids. A comprehensive stool analysis is suggested.

Benzoic acid is a common food component, especially in fruits and in particular berries/cranberries. It is also a common food additive/preservative. Benzoic acid is also formed by gut microflora metabolism of phenylalanine and dietary polyphenols. Elevated levels may thus reflect dietary intake (for example strawberries), imbalanced gut flora or a high intake of polyphenols or phenylalanine. Older studies note a relationship between decreased cognitive function and increased BA in the urine.

Succinic acid participates in the citric acid cycle, acting to donate electrons to the mitochondrial electron transport and leading to formation of fumaric acid. Common in foods such as cantaloupe, it is also a food additive, providing flow-altering effects and a tart flavor. It appears that lacto-ovo vegetarians may show decreased levels in the urine and chronic fatigue patients may also show low levels, although studies on this topic are mixed. Low levels may also be an indicator of B12 or folate deficiency.

Formiminoglutamic Acid "FIGlu" is elevated in the urine. FIGlu stands for formiminoglutamic acid, a substance produced in body tissue from the dietary amino acid histidine. FIGlu needs tetrahydrofolate (THF), a reduced form of folic acid, to be changed into forms that are metabolically useful.

Elevated urine FIGlu can occur with several circumstances. Dietary deficiency of folic acid or severe oxidant stress that limits biologic reduction of folic acid to the THF form can cause this elevation. Histidine as a supplemented nutrient can contribute to urine FIGlu levels, especially if taken in amounts that exceed 50 mg/Kg body weight. Metabolism of folic acid can be impaired if vitamin B12 is insufficient or if its metabolism is disordered. So, elevated FIGlu also can mean that some form of B12 or cobalamin is needed. The enzyme that promotes processing of FIGlu and THF requires pyridoxal 5-phosphate as a coenzyme, and vitamin B6 deficiency also may contribute to elevated FIGlu. Finally, there are rare disorders in purine synthesis that impair normal utilization of folate forms that come from FIGlu and THF. Abnormal levels of uric acid, succinylpurines, inosine or adenosine may be investigated if FIGlu

Commentary

levels remain elevated despite folate, cobalamin, pyridoxine and antioxidant therapy.

Elevated FIGlu can be coincident with homocystinuria and predisposition to cardiovascular disease. In children, elevated FIGlu and folate and/or vitamin B12 dysfunctions may be associated with mental retardation, autism, growth failure and seizures. Folate and/or vitamin B12 insufficiencies can be secondary to gastrointestinal disorders or poor quality diet, and deficiencies of both have been noted in elderly populations.

Amino Acid Markers (FMV)

Commentary

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.

Carnosine, a dietary dipeptide, is higher than the reference range. This peptide comes from fish and some animal protein, principally from tuna, salmon, beef and pork. Carnosine also is present at very low levels in human muscle (approximately 0.1% by weight). Elevated carnosine may result from a dietary overload of protein, may be temporary or episodic, and may have no clinical consequence. However, zinc deficiency can be a cause of peptidase weakness; also, pancreatic dysfunction or digestive disorder can result in increased uptake and excretion of carnosine. Elevated carnosine together with subnormal levels of essential or semiessential amino acids is consistent with incomplete digestive proteolysis and malabsorption. Further diagnostic testing to assess maldigestion should be considered. Rarely, muscle catabolism or dystrophic conditions may feature elevated urine carnosine. Metabolic carnosinemia is described (C.R. Scriver et al., *The Metabolic Basis of Inherited Disease*, 5th ed, McGraw Hill, pp 570-585, 1983).

Beta-alanine is measured to be high in the urine. Often this amino acid is elevated when the dietary peptides anserine and carnosine are elevated because they contain beta-alanine. Beta-alanine also is a breakdown product of the pyrimidine bases cytosine and uracil. Catabolism of damaged or diseased body tissue, tumors and malignancy feature increased production and urinary disposal of beta-alanine. Besides elevated anserine or carnosine and accelerated catabolism of unwanted body tissue, the next most likely source of beta-alanine is imbalanced gut flora. Some beta-alanine is produced by normal gut flora which also make pantothenic acid from it. Elevated levels of staphylococcus or streptococcus, use of antibiotics, and breakdown of yeast or fungi in the body can result in increased levels of urinary beta-alanine. Continuously elevated beta-alanine can be detrimental by impairing renal conservation of taurine.

Taurine is measured to be elevated in the urine, which is consistent with excess dietary intake, or with urinary wasting due to poor renal conservation. Excessive dietary intake of taurine-rich sources like seafood (especially shellfish), and from liver and organ meats may elevate plasma blood levels, as may consumption of taurine-supplemented sports and stimulant drinks. Urinary wasting can be secondary to generally increased renal clearance or nephrotic syndromes. Wasting can also occur when the similarly-structured amino acid beta-alanine is elevated or is present in kidney tubules. In molybdenum deficiency or sulfite oxidase impairment, elevated urine taurine results as a mode of sulfur excretion.

Renal wasting of taurine can be medically significant if it affects one or more of taurine's many important functions

-
- Conjugation of cholesterol (as choly-coenzyme A) to form taurocholic acid, an important component of bile and a major utilization of cholesterol.
- Mediation of the flux of electrolyte elements at the plasma membrane of cells. Deficient taurine may result in increased cellular calcium and sodium and reduced magnesium.
- Increased resistance to aggregation of blood platelets and decreased thromboxane release if aggregation does occur.
- Sparing of magnesium - globally. Urinary magnesium wasting can result from taurine insufficiency. Magnesium deficiency may cause fatigue, depression, muscle tremor and hypertension.
- Antioxidant functions. Taurine scavenges excess hypochlorite ion, OCl⁻, in leukocytes and facilitates effective phagocytosis by enhancing survival of leukocytes. Deficient taurine may lead to increased inflammatory response to: toxins, foreign proteins, and xenobiotic chemicals including aldehydes, alcohols, amines, petroleum solvents, and chlorine or chlorite (bleach).
- Neurotransmitter functions. Taurine strongly influences neuronal concentrations and activities of GABA and glutamic acid. Taurine can have anti-convulsant and anti-epileptic effects.

Pathologies attributed to taurine insufficiency include: biliary insufficiency, fat malabsorption (steatorrhea), cardiac arrhythmia, congestive heart failure, poor vision, retinal degeneration, granulomatous disorder of neutrophils, immune

Commentary

dysfunction, enhanced inflammatory response to xenobiotics, convulsions and seizures.

The uncommon condition of overall taurine excess (hypertaurinuria with hypertaurinemia) usually is insufficiency of sulfite oxidase activity, possibly due to molybdenum deficiency. In this condition there is increased urinary sulfites and decreased sulfates. If molybdenum is deficient, uric acid levels are reduced, xanthine is increased and aldehyde detoxication is impaired (aldehyde intolerance).

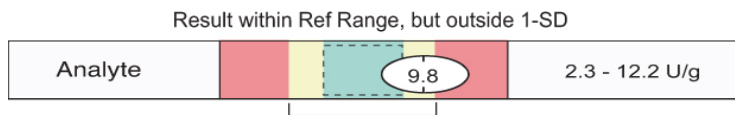
Tyrosine , a nonessential, protein-forming amino acid, is elevated. Tyrosinemia may coexist with tyrosinuria and the most common cause is either vitamin B6 deficiency or pyridoxal 5-phosphate dysfunction, or weakness of the tyrosine transaminase enzyme and increased need for vitamin B6 or pyridoxal 5-phosphate. Occasionally, down-regulation of tyrosine transaminase and elevated urine tyrosine is a consequence of adrenocortical insufficiency provided that this condition does not also reduce uptake of dietary tyrosine. Corresponding urine elevations of tyrosine are slight or mild, not severe. In severe cases or with acute tyrosinuria and tyrosinemia the condition is sometimes referred to as Tyrosinemia Type II or Richner-Hanhart syndrome. Most instances are of moderate degree, and are corrected by vitamin B6 or pyridoxal 5-phosphate supplementation. Untreated or severe conditions may feature neurological disorders, behavioral problems and hyperactivity (in children), photophobia, eye lesions or hazy corneas, and skin lesions in the form of hyperkeratotic plaques on elbows, knees, palms, and soles. Failure of vitamin B6 to clear the problem suggests need for a low phenylalanine and low tyrosine diet.

Essential & Metabolic Fatty Acids Markers (RBCs)

Commentary

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.

The **Reference Range** is a statistical interval representing 95% or 2 Standard Deviations (2 S.D.) of the reference population. One Standard Deviation (1 S.D.) is a statistical interval representing 68% of the reference population. Values between 1 and 2 S.D. are not necessarily abnormal. Clinical correlation is suggested. (See example below)



Fatty Acids and Your Health

Doctors and nutritionists used to think that all fat was merely a way for the body to store calories for later use as energy, since, as we all know too well, if we eat excess food, our body converts those calories to fat. Only in the last century have we discovered that some fats are absolutely essential to health. Our bodies cannot make these fats, and so we must get them from our food, or our health will suffer. These Essential Fatty Acids (EFAs) have many functions in the body: they are the precursors for local "hormones"; they regulate all inflammation as well as all smooth muscle contraction and relaxation. These local hormones are given names like prostaglandins, leukotrienes and thromboxanes. EFAs are also essential components for all cell membranes. Their importance for health cannot be overemphasized since the brain, nerves, eyes, connective tissue, skin, blood vessels, and every cell in the body depend on a proper balance of essential fatty acids for optimal function. It is the fats found in red blood cell membranes, known as phospholipids, that this test measures.

Essential fatty acids are classified into fat "families": omega 3 fats and omega 6 fats. Non-essential fat "families" include omega-9 fats, saturated fats, omega-7 fats, and trans-fats. Optimal health depends on the proper balance of all fats - both essential and non-essential fats - in the diet. Proper balance means adequate amounts of each individual fat, without having too much, and maintaining proper balance between the various "families" of fats. Fat health also means avoiding potentially harmful fats such as trans fats found in shortening, margarine, fried foods and dairy. A proper balance of fatty acids will lead to mental health and proper nerve function, a healthy heart and circulatory system, reduced inflammation in general, proper gastrointestinal and lung function, a more balanced immune system, and even healthy skin, hair and nails. Fatty acid balance is also critical for the health of all pregnant women and their babies since the developing brain and nervous system of the baby requires large amounts of EFAs that must come from the mother. Fatty acid imbalances have been seen in many disease processes including heart disease, hypertension, insulin resistance and diabetes, asthma, painful menstruation, pre-menstrual syndrome (PMS), depression, attention deficit hyperactivity disorder (ADHD), senility, obsessive-compulsive disorder, and post-partum depression.

This Essential and Metabolic Fatty Acid Analysis allows your health care practitioner to examine the fats found in your red blood cell membranes. These fats represent the types of fats your body has available to make cell membranes and the local "hormones" that control inflammation and smooth muscle contraction throughout the body. Following your health care practitioner's advice on diet and fatty acid supplementation is likely to restore your fatty acids to a state of healthy balance.

Results of Your Individual Essential and Metabolic Fatty Acid Analysis

The % of total saturated fats is above the reference range. Saturated fats are found in meats and dairy products.

Commentary

Saturated fats fit much more tightly together in membranes than do unsaturated fats, contributing to the structural rigidity of membranes. Too many saturated fats, however, may make cell membranes too rigid, slowing the passage of nutrients into, and of waste products out of, cells. Cell signaling and hormone-receptor binding may also be adversely affected.

Increased consumption of polyunsaturated fats, especially omega-3 and omega-6 fats and decreased consumption of meat and dairy should lower the % saturated fat.

Pentadecanoic acid and/or Tricosanoic acid are within the reference range but above the functional physiologic range. Odd chain fatty acids are produced when endogenous fatty acid synthesis begins with propionic acid (3-carbon fatty acid) as substrate rather than acetic acid (2-carbon). Propionate is found in high quantities in butter and other dairy products. Propionate is also one of the short chain fatty acids produced by our gut bacteria in the fermentation (digestion) of water-soluble fiber. With adequate B12 and biotin, propionate can be converted into succinate for use in the citric acid cycle and energy production. High levels of odd chain fatty acids in cell membranes may indicate an increased need for B12 and biotin, or may result from an exceptionally high water-soluble fiber diet.

Oxidative Stress Markers

Commentary

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.

The performance characteristics of this assay have been verified by Genova Diagnostics, Inc. This assay for Vitamin D has been cleared by the U.S. Food and Drug Administration.

Deficient or Insufficient levels:

Vitamin D is a hormone produced in the skin during exposure to sunlight or consumed in the diet, and converted to its active form, calcitriol, in the liver and kidneys. Vitamin D helps regulate serum calcium and phosphorus levels by increasing intestinal absorption of calcium and stimulating tubular reabsorption of calcium. Vitamin D also affects numerous other functions in the body.

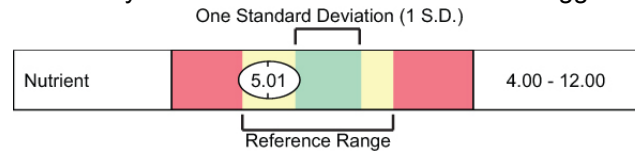
Calcitriol deficiency can result in rickets or osteomalacia due to under-mineralization of the growing skeleton or demineralization of the adult skeleton, respectively. Hypovitaminosis D also increases the risk of infection, cancer, autoimmune disease, hypertension, arteriosclerosis, diabetes and/or insulin resistance, musculoskeletal pain, epilepsy, and migraine.

*Elemental Markers***Commentary**

Testing Methodology: ICP-MS

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.

The **Reference Range** is a statistical interval representing 95% or 2 Standard Deviations (2 S.D.) of the reference population. One Standard Deviation (1 S.D.) is a statistical interval representing 68% of the reference population. Values between 1 and 2 S.D. are not necessarily abnormal. Clinical correlation is suggested. (See example below)



The reference range for Lead is set at NHANES 95th percentile.

https://www.cdc.gov/biomonitoring/pdf/FourthReport_UpdatedTables_Volume1_Jan2017.pdf

The reference range for Cadmium is set at NHANES 95th percentile.

https://www.cdc.gov/biomonitoring/pdf/FourthReport_UpdatedTables_Volume1_Jan2017.pdf

The reference range for Mercury is set at NHANES 95th percentile.

https://www.cdc.gov/biomonitoring/pdf/FourthReport_UpdatedTables_Volume1_Jan2017.pdf

Magnesium is above the reference range. Published literature acknowledges only the following causative conditions: poor renal clearance or renal insufficiency, parenteral overdose, and excessive oral use of magnesium salts together with impaired renal clearance.

Interchange between serum and cell magnesium can be rapid, and serum magnesium is closely controlled by homeostasis mechanisms. Normally, renal transport of magnesium is more rapid than intestinal absorption, and blood elevations are transient occurrences. Signs and symptoms consistent with magnesium excess are: hypotension, hypothermia, vasodilation, nausea and diarrhea with oral magnesium excess, and CNS depression with sleepiness. Reduced voluntary muscle control may occur.