Genova Diagnostics®
Innovative Testing for Optimal Health
63 Zillicoa Street
Asheville, NC 28801

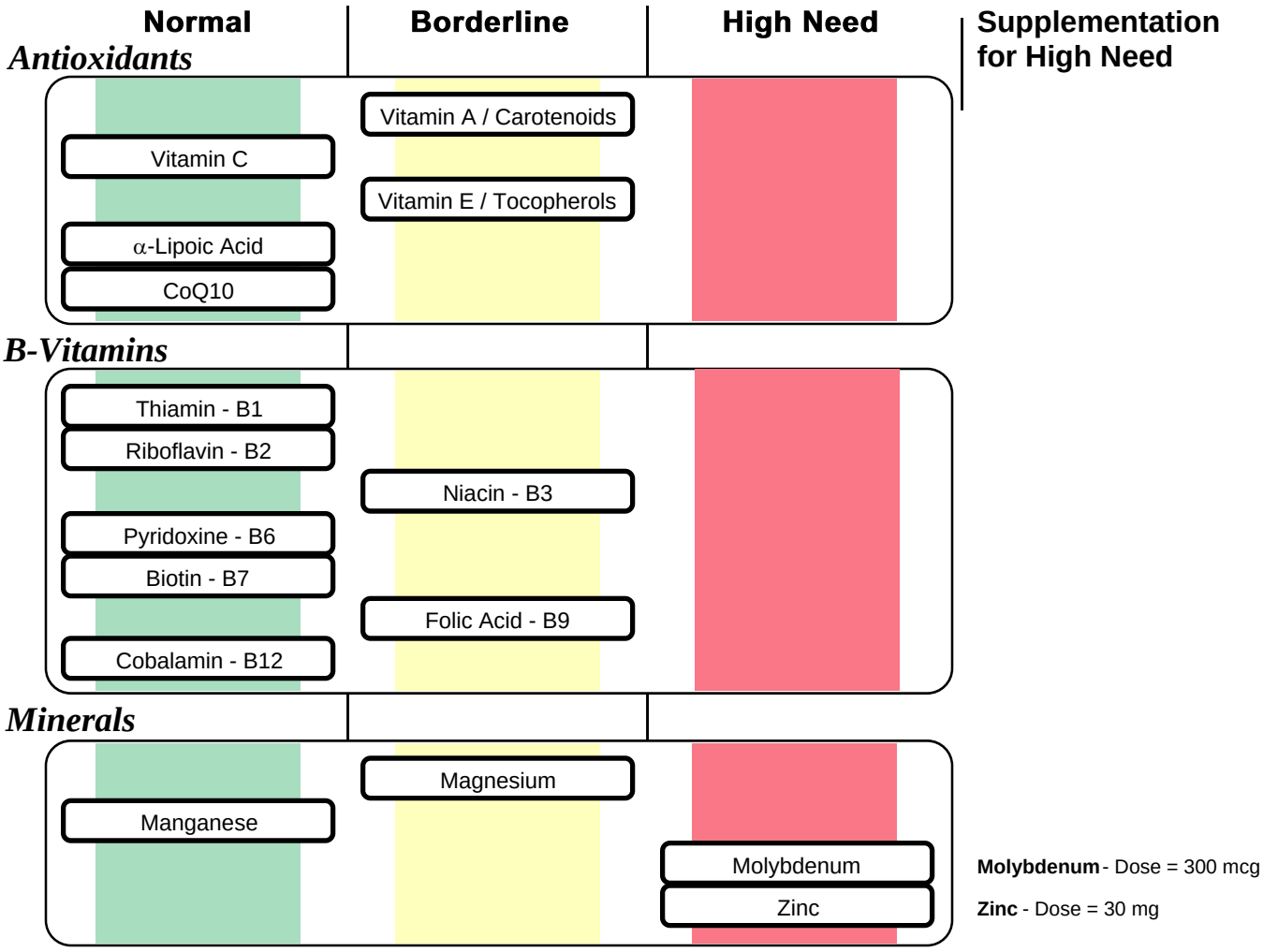
NutrEval® FMV amino acids

Patient: **SHAWN BEAN**
DOB: February 10, 1973
Sex: M
MRN: 0000713446

Order Number: D7030055
Completed: May 18, 2011
Received: May 03, 2011
Collected: May 02, 2011

Michael Overbeck MD
1100 Fayette St
Conshohocken, PA 19428

Results Overview



Molybdenum - Dose = 300 mcg

Zinc - Dose = 30 mg

SUGGESTED SUPPLEMENT SCHEDULE

Supplements	Daily Recommended Intake (DRI)	Patient's Daily Recommendations	Provider Daily Recommendations
Antioxidants			
Vitamin A / Carotenoids	3,000 IU	5,000 IU	
Vitamin C	90 mg	250 mg	
Vitamin E / Tocopherols	22 IU	200 IU	
α-Lipoic Acid		50 mg	
CoQ10		30 mg	
B-Vitamins			
Thiamin - B1	1.2 mg	10 mg	
Riboflavin - B2	1.3 mg	10 mg	
Niacin - B3	16 mg	30 mg	
Pyridoxine - B6	1.3 mg	10 mg	
Biotin - B7	30 mcg	100 mcg	
Folic Acid - B9	400 mcg	800 mcg	
Cobalamin - B12	2.4 mcg	100 mcg	
Minerals			
Magnesium	420 mg	600 mg	
Manganese	2.3 mg	3.0 mg	
Molybdenum	45 mcg	300 mcg	
Zinc	11 mg	30 mg	
Essential Fatty Acids			
Omega-3 Oils	500 mg	500 mg	
Digestive Support			
Probiotics		10 billion CFU	
Pancreatic Enzymes		5,000 IU	
Other Vitamins			
Vitamin D	600 IU		

Amino Acid	mg/day	Amino Acid	mg/day
Arginine	655	Methionine	0
Asparagine	0	Phenylalanine	192
Cysteine	0	Serine	0
Glutamine	257	Taurine	0
Glycine	2,283	Threonine	0
Histidine	0	Tryptophan	0
Isoleucine	147	Tyrosine	253
Leucine	0	Valine	421
Lysine	828		

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

Interpretation At-A-Glance

Nutritional Needs

Antioxidants

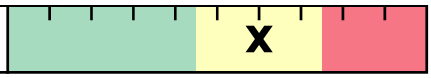
Vitamin A / Carotenoids

- 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 & progestin.
- 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.

Vitamin C

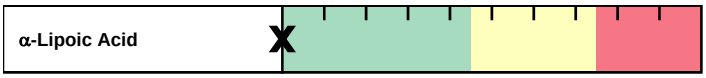
- Vitamin C is an antioxidant (also used in the regeneration of other antioxidants). It is involved in cholesterol metabolism, the production & function of WBCs and antibodies, and the synthesis of collagen, norepinephrine and carnitine.
- Deficiency may occur with oral contraceptives, aspirin, diuretics 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.

Vitamin E / Tocopherols



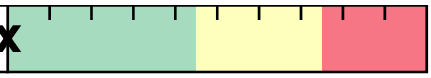
- Alpha-tocopherol (body's main form of vitamin E) functions as an antioxidant, regulates cell signaling, influences immune function and inhibits coagulation.
- Deficiency may occur with malabsorption, cholestyramine, colestipol, isoniazid, orlistat, olestra and certain anti-convulsants (e.g., phenobarbital, 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.

α-Lipoic Acid



- 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.

CoQ10

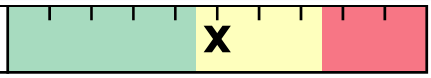


- CoQ10 is a powerful antioxidant that is synthesized in the body and contained in cell membranes. CoQ10 is also essential for energy production & pH regulation.
- CoQ10 deficiency may occur with HMG-CoA reductase inhibitors (statins), several anti-diabetic medication classes (biguanides, sulfonylureas) 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.

Glutathione

- 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.

Plant-based Antioxidants



- 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.).

Key

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

NutrEval FMV amino acids

Interpretation At-A-Glance

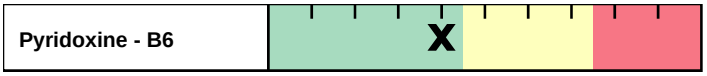
Nutritional Needs

B-Vitamins

Thiamin - B1

- 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.

Pyridoxine - B6



- 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.

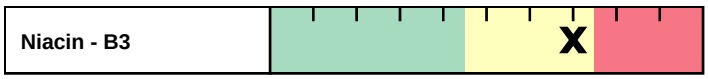
Riboflavin - B2

- 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 - B7

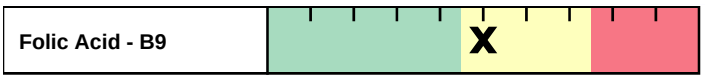
- Biotin is a cofactor for enzymes involved in functions such as fatty acid synthesis, mitochondrial FA oxidation, gluconeogenesis and DNA replication & transcription.
- Deficiency may result from certain inborn errors, chronic intake of raw egg whites, long-term TPN, anticonvulsants, high-dose B5, sulfa 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.

Niacin - B3



- 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 - B9



- 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.

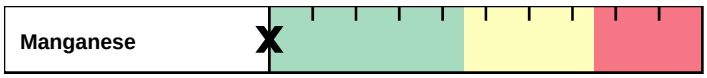
Cobalamin - B12

- 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.

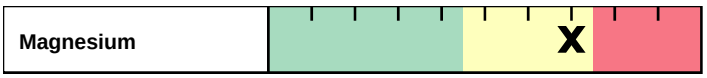
NutrEval FMV amino acids — Interpretation At-A-Glance

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.

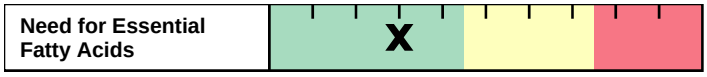


- Magnesium is involved in >300 metabolic reactions. Key areas include energy production, bone & ATP formation, muscle & nerve conduction and cell signaling.
- Deficiency may occur with malabsorption, alcoholism, hyperparathyroidism, renal disorders (wasting), diabetes, diuretics, digoxin or high doses 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.

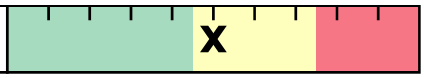
Interpretation At-A-Glance

Functional Imbalances

Mitochondrial Dysfunction

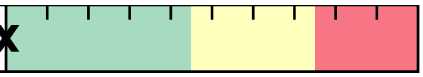
- Mitochondria are a primary site of generation of reactive oxygen species. Oxidative damage is considered an important factor in decline of physiologic function that occurs with aging and stress.
- Mitochondrial defects have been identified in cardiovascular disease, fatigue syndromes, neurologic disorders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

Toxic Exposure



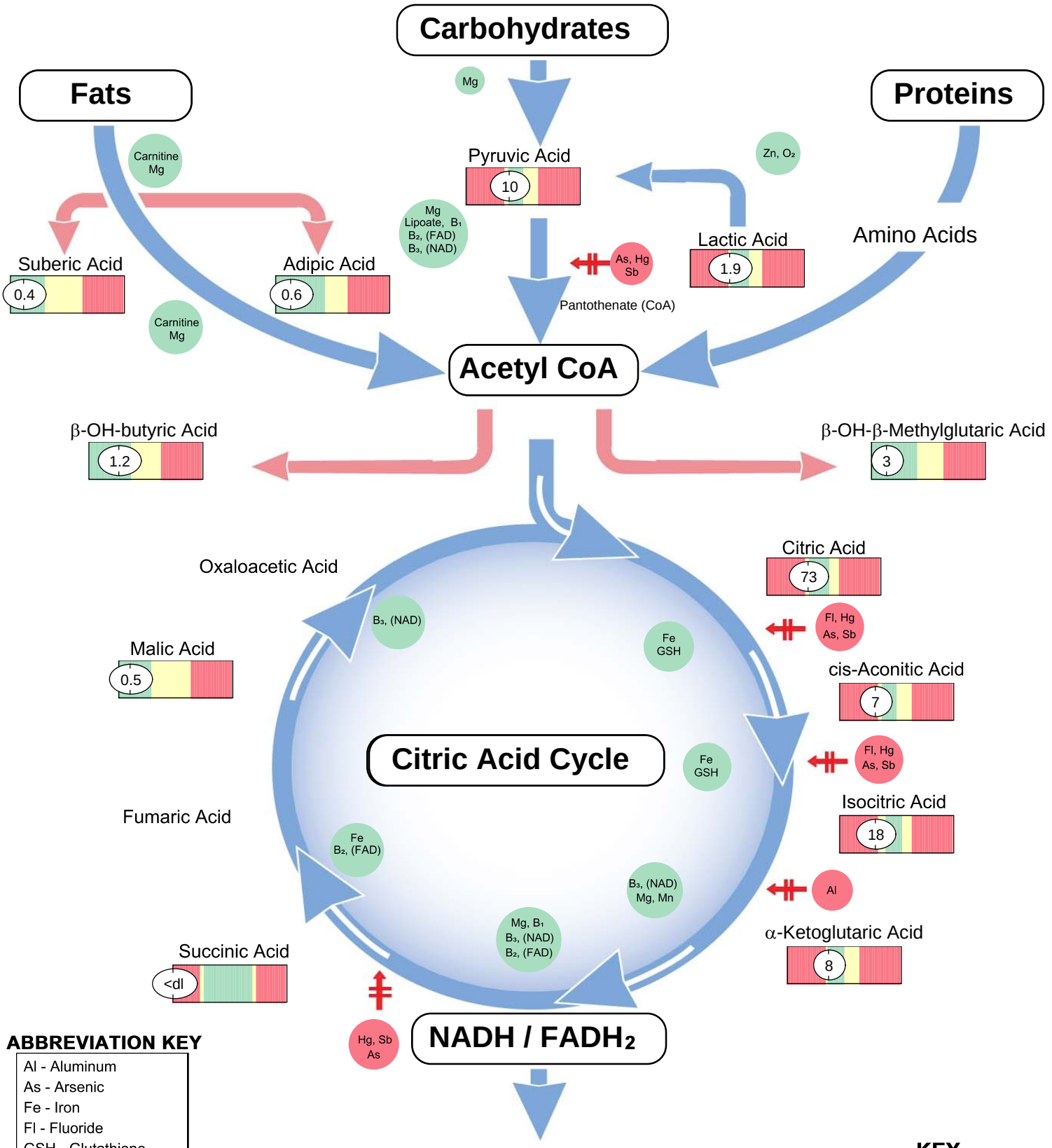
- Methyl tert-Butyl Ether (MTBE) is a common gasoline additive used to increase octane ratings, and has been found to contaminate ground water supplies where gasoline is stored. Inhalation of MTBE may cause nose and throat irritation, as well as headaches, nausea, dizziness and mental confusion. Animal 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.

Need for Methylation



- 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.

Krebs Cycle At-A-Glance



ABBREVIATION KEY

Al - Aluminum
As - Arsenic
Fe - Iron
Fl - Fluoride
GSH - Glutathione
Hg - Mercury
Mg - Magnesium
Mn - Manganese
Sb - Antimony
Zn - Zinc

Electron Transport and Oxidative Phosphorylation

$(2)\ H + 1/2\ O_2 \rightarrow H_2O$

KEY

Green = cofactor

Red = inhibitor

Metabolic Analysis Markers

All biomarkers reported in mmol/mol creatinine unless otherwise noted.

Malabsorption and Dysbiosis Markers

Malabsorption Markers

		Reference Range
Indoleacetic Acid (IAA)	1.2	<= 4.2
Phenylacetic Acid (PAA)	0.03	<= 0.12

Bacterial Dysbiosis Markers

		Reference Range
Dihydroxyphenylpropionic Acid (DHPPA)	0.4	<= 12.8
3-Hydroxyphenylacetic Acid	3.5	<= 8.1
4-Hydroxyphenylacetic Acid	5	<= 29
Benzoic Acid	0.02	<= 0.05
Hippuric Acid	215	<= 603

Yeast / Fungal Dysbiosis Markers

		Reference Range
Arabinose	14	<= 96
Citramalic Acid	1.0	<= 5.8
Tartaric Acid	<dl	<= 15

Cellular Energy & Mitochondrial Metabolites

Carbohydrate Metabolism

		Reference Range
Lactic Acid	1.9	1.9-19.8
Pyruvic Acid	10	7-32
β-OH-Butyric Acid (BHBA)	1.2	<= 2.8

Energy Metabolism

		Reference Range
Citric Acid	73	40-520
Cis-Aconitic Acid	7	10-36
Isocitric Acid	18	22-65
α-Ketoglutaric Acid (AKG)	8	4-52
Succinic Acid	<dl	0.4-4.6
Malic Acid	0.5	<= 3.0
β-OH-β-Methylglutaric Acid (HMG)	3	<= 15

Fatty Acid Metabolism

		Reference Range
Adipic Acid	0.6	<= 2.8
Suberic Acid	0.4	<= 2.1

Creatinine Concentration

		Reference Range
Creatinine ◆	9.6	3.1-19.5 mmol/L

Neurotransmitter Metabolites

		Reference Range
Vanilmandelic Acid	0.4	0.4-3.6
Homovanillic Acid	1.2	1.2-5.3
5-OH-indoleacetic Acid	2.3	3.8-12.1
3-Methyl-4-OH-phenylglycol	0.03	0.02-0.22
Kynurenic Acid	1.9	<= 7.1
Quinolinic Acid	2.4	<= 9.1
Kynurenic / Quinolinic Ratio	0.79	>= 0.44

Vitamin Markers

		Reference Range
α-Ketoadipic Acid	0.3	<= 1.7
α-Ketoisovaleric Acid	0.33	<= 0.97
α-Ketoisocaproic Acid	0.35	<= 0.89
α-Keto-β-Methylvaleric Acid	0.6	<= 2.1
Formiminoglutamic Acid (FIGlu)	0.9	<= 1.5
Glutaric Acid	0.10	<= 0.51
Isovalerylglycine	1.4	<= 3.7
Methylmalonic Acid	0.7	<= 1.9
Xanthurenic Acid	0.24	<= 0.96
3-Hydroxypropionic Acid	5	5-22
3-Hydroxyisovaleric Acid	7	<= 29

Toxin & Detoxification Markers

		Reference Range
α-Ketophenylacetic Acid (from Styrene)	0.16	<= 0.46
α-Hydroxyisobutyric Acid (from MTBE)	4.6	<= 6.7
Orotic Acid	0.29	0.33-1.01
Pyroglutamic Acid	14	16-34

Tyrosine Metabolism

		Reference Range
Homogentisic Acid	3	<= 19
2-Hydroxyphenylacetic Acid	0.37	<= 0.76

Metabolic Analysis Reference Ranges are Age Specific

The performance characteristics of all assays have been verified by Genova Diagnostics, Inc. Unless otherwise noted with ◆ as cleared by the U.S. Food and Drug Administration, assays are For Research Use Only.

Amino Acids (FMV)

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

Nutritionally Essential Amino Acids

Amino Acid	Reference Range
Arginine	10-64
Histidine	271-993
Isoleucine	17-52
Leucine	25-77
Lysine	34-226
Methionine	26-69
Phenylalanine	22-61
Taurine	80-545
Threonine	52-192
Tryptophan	23-88
Valine	19-53

Nonessential Protein Amino Acids

Amino Acid	Reference Range
Alanine	103-392
Asparagine	37-134
Aspartic Acid	27-74
Cysteine	19-70
Cystine	23-68
γ-Aminobutyric Acid	<= 23
Glutamic Acid	3-15
Glutamine	153-483
Proline	2-14
Tyrosine	28-113

Creatinine Concentration

	Reference Range
Creatinine ◆	3.1-19.5 mmol/L

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Intermediary Metabolites

B Vitamin Markers	Reference Range
α-Aminoadipic Acid	11-73
α-Amino-N-butyric Acid	9-49
β-Aminoisobutyric Acid	19-163
Cystathionine	6-29
3-Methylhistidine	134-302

Urea Cycle Markers	Reference Range
Ammonia	12.0-41.0 mmol/g creatinine
Citrulline	9-40
Ornithine	3-16
Urea ◆	150-380 mmol/g creatinine

Glycine/Serine Metabolites	Reference Range
Glycine	434-1,688
Serine	135-426
Ethanolamine	156-422
Phosphoethanolamine	14-50
Phosphoserine	26-64
Sarcosine	<= 41

Dietary Peptide Related Markers

	Reference Range
Anserine (dipeptide)	8-118
Carnosine (dipeptide)	12-120
1-Methylhistidine	83-1,008
β-Alanine	<= 17

Markers for Urine Representativeness

	Reference Range
Glutamine/Glutamate	>= 12
Ammonia	12.0-41.0 mmol/g creatinine
Arginine/Ornithine	>= 1.0

Urine Representativeness Index

Ref Range 5 – 10

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	0.20	>= 0.09 wt %
Eicosapentaenoic (EPA) 20:5 n3	0.52	>= 0.16 wt %
Docosapentaenoic (DPA) 22:5 n3	2.34	>= 1.14 wt %
Docosahexaenoic (DHA) 22:6 n3	5.8	>= 2.1 wt %
% Omega 3s	8.8	>= 3.8

Omega 9 Fatty Acids

Analyte	(olive oil)	Reference Range
Oleic 18:1 n9	9	10-13 wt %
Nervonic 24:1 n9	2.2	2.1-3.5 wt %
% Omega 9s	11.6	13.3-16.6

Saturated Fatty Acids

Analyte	(meat, dairy, coconuts, palm oils)	Reference Range
Palmitic C16:0	20	18-23 wt %
Stearic C18:0	18	14-17 wt %
Arachidic C20:0	0.18	0.22-0.35 wt %
Behenic C22:0	0.69	0.92-1.68 wt %
Tricosanoic C23:0	0.15	0.12-0.18 wt %
Lignoceric C24:0	2.0	2.1-3.8 wt %
Pentadecanoic C15:0	0.07	0.07-0.15 wt %
Margaric C17:0	0.25	0.22-0.37 wt %
% Saturated Fats	41.2	39.8-43.6

Omega 6 Fatty Acids

Analyte	(vegetable oil, grains, most meats, dairy)	Reference Range
Linoleic (LA) 18:2 n6	12.1	10.5-16.9 wt %
γ-Linolenic (GLA) 18:3 n6	0.06	0.03-0.13 wt %
Dihomo-γ-linolenic (DGLA) 20:3 n6	1.26	>= 1.19 wt %
Arachidonic (AA) 20:4 n6	20	15-21 wt %
Docosatetraenoic (DTA) 22:4 n6	2.83	1.50-4.20 wt %
Eicosadienoic 20:2 n6	0.35	<= 0.26 wt %
% Omega 6s	37.1	30.5-39.7

Monounsaturated Fats

Omega 7 Fats		Reference Range
Palmitoleic 16:1 n7	0.19	<= 0.64 wt %
Vaccenic 18:1 n7	0.79	<= 1.13 wt %
Trans Fat		
Elaidic 18:1 n9t	0.31	<= 0.59 wt %

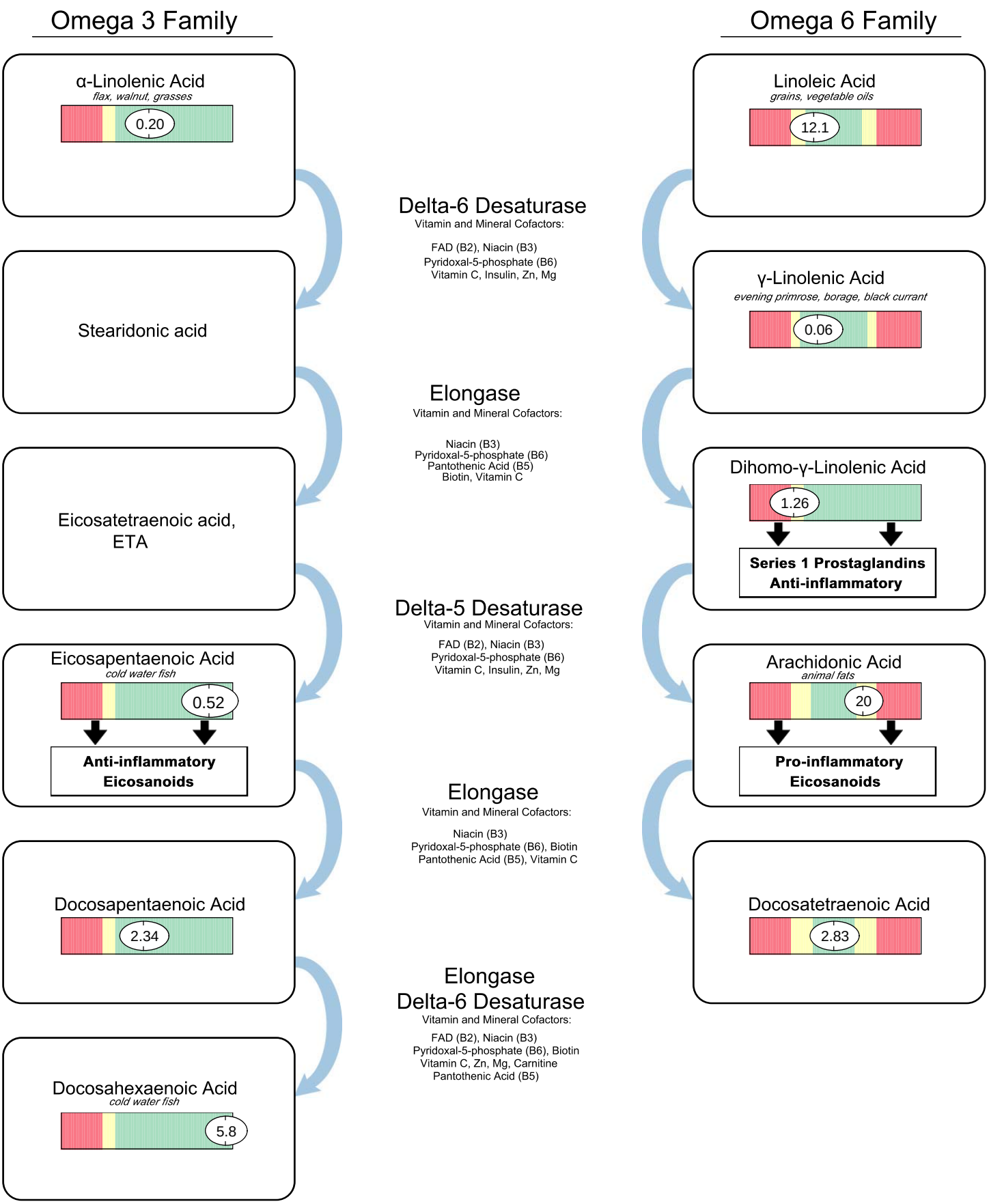
Delta - 6 Desaturase Activity

	Upregulated Functional Impaired	
Linoleic / DGLA 18:2 n6 / 20:3 n6	9.6	6.0-12.3

Cardiovascular Risk

Analyte		Reference Range
Omega 6s / Omega 3s	4.2	3.4-10.7
AA / EPA 20:4 n6 / 20:5 n3	39	12-125
Omega 3 Index	6.3	>= 4.0

Essential Fatty Acid Metabolism



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

Oxidative Stress Markers

Oxidative Stress Markers

		Reference Range
Glutathione (whole blood)	1,110	>= 669 micromol/L
Lipid Peroxides (urine)	3.7	<=10.0 micromol/g Creat.
8-OHdG (urine)	4	<=16 mcg/g Creat.
Coenzyme Q10, Ubiquinone (plasma)	0.75	0.46-1.72 mcg/mL

Elemental Markers (RBCs)

Nutrient Elements

Element	Reference Range	Reference Range
Copper	0.475	0.466-0.721 mcg/g
Magnesium	34.0	30.1-56.5 mcg/g
Manganese	0.013	0.007-0.038 mcg/g
Potassium	2,454	2,220-3,626 mcg/g
Selenium	0.23	0.25-0.76 mcg/g
Zinc	6.5	7.8-13.1 mcg/g

Toxic Elements

Element	Reference Range	Reference Range
Lead	0.017	<= 0.048 mcg/g
Mercury	0.0040	<= 0.0039 mcg/g
Antimony	0.001	<= 0.002 mcg/g
Arsenic	0.010	<= 0.071 mcg/g
Cadmium	0.000	<= 0.001 mcg/g
Tin	<dl	<= 0.0009 mcg/g

Lab Comments

The performance characteristics of all assays have been verified by Genova Diagnostics, Inc. Unless otherwise noted with ◆ as cleared by the U.S. Food and Drug Administration, assays are For Research Use Only.

Metabolic Analysis Markers

Commentary

All these markers are within their reference ranges; there are no abnormals.

5-Hydroxyindoleacetic Acid (5-HIAA) is measured to be low. 5-HIAA is a normal urine metabolite of the neurotransmitter serotonin, which is formed from the essential amino acid tryptophan. Low levels of 5-HIAA have been noted in mood disorders, anxiety, insomnia, migraine, and eating disorders.

Virtually all blood serotonin and most urine 5-HIAA comes from serotonin formation outside of the CNS. This occurs primarily in tissues in the abdominal cavity, especially the gastrointestinal tract, pancreas and spleen. Subnormal 5-HIAA can be the result of: a tryptophan-deficient diet, maldigestion or malabsorption that limits tryptophan uptake, tissue oxygen deficit or biopterin deficiency (limits 5-hydroxytryptophan formation), vitamin B6 deficiency (limits serotonin formation), monoamine oxidase (MAO) inhibition or deficiency of the coenzyme, FAD (limits serotonin conversion to 5-HIAA). Malabsorption of tryptophan can lead to urine elevations of indoleacetic acid (IAA), another analyte reported on this profile.

Cis-aconitic Acid (CAA) is measured to be subnormal, while its precursor, citric acid is not deficient. This may occur with low carbohydrate diets, fasting or anorexic conditions; citric acid may then be only marginal. On the other hand, normal or elevated citric acid with low cis-aconitic acid may be the result of urinary citrate wasting (leading to deficiency) or of weakness in the enzyme that transforms citrate into cis-aconitate. This enzyme (aconitase) requires cysteine or glutathione in the reduced state and ferrous iron (Fe+2). Oxidant stress can inhibit aconitase as can toxic elements that bind to sulfhydryl groups (mercury, arsenic, antimony). Fluorine or fluoride that forms fluorocitrate is also a very potent inhibitor of aconitase.

Isocitric Acid is measured to be subnormal. This may be due to deficient or low-normal levels of its precursors, cis-aconitate or citrate. It may also be due to depleted alpha-ketoglutarate, which it forms in the citric acid cycle; check for low alpha-ketoglutarate in this report. The same enzyme, aconitase, forms cis-aconitic acid and isocitric acid, and requires reduced cysteine or glutathione and ferrous iron (Fe+2). Oxidant stress can inhibit it, as can arsenic, mercury or antimony. Fluoride that becomes fluorocitrate can strongly inhibit aconitase and impair formation of isocitrate.

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.

Pyroglutamic Acid (5-oxoproline) is measured to be subnormal. Pyroglutamic acid primarily arises during the "gamma-glutamyl cycle" which splits glutathione (GSH) into cysteinylglycine and a gamma-glutamyl moiety. The gamma-glutamyl part attaches to another amino acid or short-chain peptide, or joins with an element (essential mineral) for transport across a membrane or into a cell. Uptake of such nutrients from the small intestine depends primarily upon this process. The enzyme gamma-glutamylcyclotransferase finishes the transport function by splitting off what was carried and changing the gamma-glutamyl part into pyroglutamic acid. Insufficiency of pyroglutamic acid may result from: insufficiency of reduced glutathione ("GSH"), toxicity (causing depletion of GSH), oxidant stress limiting the reduced form of glutathione, magnesium deficiency which can limit the rate of endogenous GSH formation, and deficiency of cysteine (the rate-limiting amino acid for GSH formation). Symptoms of nutrient malabsorption, oxidant stress and/or toxicity are consistent with subnormal pyroglutamate.

Commentary

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.

REPRESENTATIVENESS INDEX

Urine amino acid levels usually are representative of blood levels and reflect dietary uptake and metabolism as well as excretion. However, abnormal renal clearance, loss of urine during the collection period, decay or spoilage, and presence of blood in the urine could cause the urine specimen to be unrepresentative. The possibility of such problems can be judged from analytical measurements which are portrayed in the first section of the report: Markers for Urine Representativeness.

The **glutamine/glutamate ratio** can indicate specimen decay. When aged or improperly preserved, urine glutamine decays to glutamic acid and ammonia. However, in metabolic acidosis some glutamine is transformed into glutamic acid and ammonium ion as a pH-balancing mechanism. Also, high glutamic acid occurs in gout. Hence, low glutamine/glutamate ratio may reflect decay or it may be of metabolic origin. High glutamine/glutamate ratio is metabolic and does not reflect on specimen representativeness.

The **ammonia concentration** , if elevated, usually indicates overall decay of amino acids. An exception would be elevated ammonia concentration with hyperammonemia of metabolic or bacterial origin. Very low ammonia concentration suggests low urine nitrogen levels and may occur in protein-deficient diets. Blood amino acid levels may then be normal or low-normal.

The **arginine/ornithine ratio** generally reflects whether the sample is purely urine or whether hematuria is present. A low ratio is consistent with blood in the urine. This is not foolproof, because high ornithine relative to arginine also may occur with a specific urea cycle weakness (OCT enzyme dysfunction, rare), and with pyridoxal phosphate or transamination weakness affecting ornithine. Urine should not be collected for acid analysis by women during menses. Blood in urine can notably distort the results.

The computer scores the above four Markers for Representativeness and computes a Representativeness Index. An index of 10 means all markers are within expected limits. **An index below 5 suggests a repeat amino acid analysis with a new urine specimen.**

Valine , a branched-chain structured amino acid, is measured to be low. This nutritionally essential amino acid is required for formation of body proteins and enzymes and it is normally in physiological balance with the other two similarly structured amino acids, leucine and isoleucine. The branched-chain structure of valine makes it very important to the formation of flexible collagen tissues, such as elastin in ligaments. Valine is relatively abundant in all protein foods. Low valine may result from a poor quality diet or from gastrointestinal dysfunction, particularly from digestive peptidase dysfunction. Zinc deficiency, pancreatic insufficiency, acidic small intestine, food reactivities and malabsorption may be involved.

Alanine , a nonessential protein amino acid, is low. Alanine may come directly from dietary protein, or it can be formed in body cells from serine or from pyruvic acid. Tryptophan, an essential amino acid, and cysteine are minor sources of endogenously-produced alanine. Low urine alanine can be a consequence of poor renal clearance, in which case 24-hour urine creatinine is expected to be low and blood alanine levels may be elevated. Deficient alanine is consistent with a low-protein diet or protein malnutrition. Gastrointestinal dysfunction with poor digestive proteolysis or malabsorption may lead to alanine insufficiency. Occasionally, cases of adrenocortical insufficiency feature impaired conversion of pyruvic acid to alanine and low urinary alanine. No symptomatology is attributed specifically to subnormal alanine.

Aspartic acid , a protein amino acid, is measured to be low. This amino acid comes from dietary protein, and it may be formed endogenously. Endogenous formation occurs by amino group transfer from glutamic acid to oxaloacetic

Commentary

acid using the familiar "ALT". This enzyme is dependent upon vitamin B6 as pyridoxal 5-phosphate as a coenzyme. Low urine aspartic acid can be a consequence of poor renal clearance, in which case 24-hour urine creatinine is expected to be low and blood aspartic acid level may be elevated. Deficient aspartic acid can result from dietary protein insufficiency, gastrointestinal dysfunction and occasionally is secondary to vitamin B6 deficiency or dysfunction.

Cystine is measured to be low. Cystine is the oxidized or dimer form of cysteine; it is two cysteines linked together with a sulfur-sulfur bond. In the form of cysteine it is a protein amino acid and a key component of glutathione, coenzyme A, many enzymes, and of insulin. Cystine has dietary sources and is the extracellular form of cysteine. Low cystine is consistent with protein malnutrition, gastrointestinal dysfunctions, or impaired metabolism of methionine. In oxidant stress cystine (and cysteine) may be low or the cysteine/cystine ratio may be below about 0.75 (please check the cysteine result).

Glycine, a major nonessential, protein-forming amino acid, is low. Glycine comes from digestion of dietary protein, and it has multiple routes for endogenous formation and removal. The amino acids threonine and serine are important sources of glycine as is glycolysis. Subnormal glycine usually accompanies multiple hypoaminoaciduria conditions and may occur in chronic or late stage protein malnutrition. After two days of fasting or in early stages of starvation, elevated glycine may occur from catabolism of body protein. Later, as body muscle and protein is depleted, decreased urinary glycine occurs. Negative nitrogen balance and deficiencies of threonine and serine are expected with low urine glycine.

Alpha-amino-N-butyric acid (A-ANB), an intermediary product of threonine and methionine metabolism, is measured to be low. This is not a protein-forming amino acid, and a low level usually reflects low levels of either threonine or methionine. An immediate precursor of A-ANB is alpha-keto-N-butyric acid which is formed together with cysteine from cystathionine. Low cysteine or cystine and low A-ANB with normal or high cystathionine suggests pyridoxal 5-phosphate coenzyme dysfunction or increased need for vitamin B6, regardless of methionine or threonine level.

Phosphoethanolamine is measured to be low. Like ethanolamine, phosphoethanolamine is an intermediate in the serine-to-choline metabolism sequence. It is a precursor of phosphatidylcholine, choline and the neurotransmitter, acetylcholine. Formation of phosphoethanolamine from ethanolamine requires phosphorylation, a magnesium-dependent process. The most common reasons for insufficient phosphoethanolamine are magnesium deficiency or dysfunction, ethanolamine deficiency and serine deficiency. Low phosphoethanolamine is significant if cholinergic functions are limited.

Phosphoserine is measured to be low. Phosphoserine is a product of glycolysis and is formed by amino group transfer from glutamic acid to phosphohydroxypyruvic acid. Low levels of phosphoserine may be secondary to: vitamin B6 deficiency (as coenzyme pyridoxal phosphate for amino group transfer), low pyruvic acid, impaired glycolysis, or serine insufficiency where phosphoserine is consumed as a source for serine.

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 cholyl-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

Commentary

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 chamicals 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 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).

1-Methylhistidine is found to be elevated; it is a component of the dietary peptide anserine. Anserine is beta-alanyl-1-methyl-L-histidine, and it is known to come from chicken, turkey, duck, rabbit, tuna and salmon. Other food sources (especially trout and fowl) also are likely but are not documented. The peptidase enzyme that hydrolyzes anserine is present in the small intestine and also present in liver, spleen, and kidney tissues and in blood serum. Some direct uptake of dietary anserine is normal, and moderate levels of urinary 1-methylhistidine are normal. However, high levels suggest increased uptake of short-chain peptides, possibly increased gut permeability, and increased hydrolysis of short-chain dietary peptides by peptidases in blood, liver and spleen. Elevated 1-methylhistidine suggests one or more of: dietary overload of anserine-source foods, increased gut permeability, and decreased activity of digestive peptidases in the small intestine. There may or may not be associated symptomatology. 1-Methylhistidine itself is not known to be detrimental.

Cystathionine is an intermediary metabolite of the essential amino acid methionine, and cystathionine is subnormal per the urine analysis. Cystathionine is preceded by homocysteine, and it leads to cysteine and alpha-ketobutyric acid. Cystathionine formation from homocysteine requires the amino acid serine and vitamin B6 as coenzyme pyridoxal 5-phosphate (P 5-P). Low cystathionine with normal (or high) methionine and normal homocystine may indicate limited serine but usually indicates increased need for vitamin B6 or pyridoxal phosphate.

Depending upon need for and levels of cysteine, cystine and taurine, this problem may or may not have associated symptoms and may only be a transient physiological imbalance. However, if low cystathionine reflects a significant weakness in the activity of its formation enzyme (cystathionine beta-synthase), then clinical abnormalities could be associated with this finding. Pathologies associated with impaired cystathionine beta-synthase include: ectopia lentis, myopia, osteoporosis, scoliosis, CNS disorders, and arterial and venous thromboemboli.

Essential & Metabolic Fatty Acids Markers (RBCs)

Commentary

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

Dihomo Gamma Linolenic Acid (DGLA) is within the reference range, but below the functional physiologic range. DGLA is the main precursor fat for the production of highly anti-inflammatory eicosanoids, especially the series 1 prostaglandins. Low DGLA is often associated with inflammatory conditions such as heart disease, arthritis, inflammatory bowel disorders, eczema, and psoriasis. Since DGLA-derived eicosanoids also promote smooth muscle relaxation, low DGLA levels may contribute to increased smooth muscle contraction, and subsequently to conditions like hypertension, asthma, painful menstruation, and irritable bowel syndrome.

Low DGLA can result from impaired conversion of linoleic acid into gamma-linolenic acid (and subsequently into DGLA) or from an increased conversion of DGLA into arachidonic acid or both. Delta-6 desaturase is the enzyme responsible for converting LA into GLA and may be impaired with age, alcohol use, genetic defect, or nutrient

Commentary

deficiency. An elevated linoleic/DGLA ratio or an elevated eicosadienoic/DGLA ratio (see p.3 of this report) would strongly suggest impaired delta-6 desaturase activity. Supplementation with GLA-containing oils like evening primrose, borage or black currant seed oils bypasses delta-6 desaturase.

A low DGLA/arachidonic acid ratio (see p.3 of this report) would indicate a likely increased activity of delta-5 desaturase. Insulin activates delta-5 desaturase. A high carbohydrate (sugars and starch) diet increases insulin secretion and action in the body. Consumption of a higher protein and higher fiber and complex carbohydrate diet reduces insulin action in the body. Eicosapentaenoic acid (EPA) supplementation, found in fish and fish oils, has also been shown to reduce delta-5 desaturase activity, reducing the conversion of DGLA into AA.

Oleic acid is below the reference range. Oleic acid is important in maintaining cell membrane fluidity. Low oleic acid may indicate decreased delta-9 desaturase activity. Vitamin and mineral cofactor supplementation should be considered. These include B vitamins (especially B2, B3, and B6), vitamin C, zinc, and magnesium.

Olive oil is ~80% oleic acid; using olive oil as the primary dietary oil can increase oleic acid in cell membrane phospholipids. High-oleic safflower oil and high-oleic sunflower oil are available in health food stores and also constitute excellent sources of oleic acid.

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.

Commentary

Mercury is above the reference range. Possible sources of mercury (Hg) include: contaminated shellfish or seafood, contaminated water supplies, dental amalgams and/or recent dental work, laboratory equipment, barometers, thermometers, certain specially-formulated fungicides, old paint containing Hg fungicide and mining and smelting operations.

At least 90% of blood organic mercury rapidly distributes to erythrocytes, and at least 60% of elemental mercury may reside transiently in erythrocytes. Most inorganic mercury does not enter the erythrocyte. Mercury has strong affinity for sulfhydryl (-SH) sites on proteins and enzymes throughout the body and deposits in many tissues and organs. The kidneys eventually carry much of the body burden regardless of route of exposure or chemical form of the Hg. Elemental and inorganic Hg eventually distribute predominately to liver and kidney. Excretion is slow - kidney Hg via urine and liver Hg via feces. Elemental Hg vapor may be dissolved in blood, may enter erythrocytes, and can deposit in brain tissue. Organic Hg (methyl, ethyl) binds to enzymes, proteins and glutathione in blood and various tissues, circulates rather freely, and has a long retention half-time in the body (approximately two months). Hg interferes with catalase, monoamine oxidase, mixed-function oxidases and cytochrome P-450 in liver tissue, and stimulates thionein formation and is distributed there partly as mercury-metallothionein. In cell mitochondria, organic Hg, especially methyl mercury, disrupts respiration, decreases synthesis of RNA and can be mutagenic by altering chromosome structure.

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

Selenium (Se) is below the reference range. This element activates glutathione peroxidase which facilitates glutathione's antioxidant function, and it activates prohormone iodothyronine deiodinase which helps to balance levels of thyroid hormone. Of whole blood selenium, approximately one-third is carried in serum (bound to alpha2 and beta1 globulins) and two thirds resides inside the erythrocytes bound to glutathione peroxidase and to other proteins. Approximately 10% of erythrocyte selenium is bound to glutathione peroxidase.

Low selenium can produce two physiological imbalances: (1) oxidant stress due to lowered antioxidant activity of glutathione, and (2) normal or high T4 and subnormal T3. Reasons for low selenium include: poor quality diet or diet of foods grown in low-selenium soils, intestinal malabsorption, and urinary wasting of selenium which may occur with cystinuria. Cystinuria, renal wasting of cystine, is assessed by urine amino acid analysis.

Symptoms and pathological consequences of insufficient selenium include: muscle aches, hypothyroid function, sclerosing of tissue, anemia, increased dental caries, and increased inflammatory responses. Also reported are higher incidence of malignancy, increased thrombosis, and increased cardiovascular disease.

Zinc is below the reference range. Erythrocytes contain 75 to 85% of total whole blood zinc, bound to enzymes and to cell membranes. Important erythrocyte enzymes containing zinc are carbonic anhydrase and Cu,Zn-SOD. Zinc deficiency features lowered levels of zinc in erythrocytes with reduced activity of carbonic anhydrase occurring probably before reduced SOD activity occurs. However, erythrocyte zinc binding is relatively strong, and packed RBCs may not be the most sensitive or early indicator of zinc insufficiency. More sensitive are digestive peptidases (leucine aminopeptidase, carboxypeptidase, carnosinase). Urine amino acid analysis with measurement of anserine and carnosine peptides can be a very sensitive indicator of zinc function as an enzyme activator.

Zinc can be deficient in intestinal malabsorption, alcoholism, chronic ingestion of highly-processed or "junk" foods, chronic diarrhea, overuse of diuretics, and nephrotic syndrome. Excess copper interferes with zinc binding in blood plasma and reduces zinc retention. Excess iron intake may impair zinc absorption in the small intestine. Diabetes mellitus and hyperaminoaciduria may feature urinary wasting of zinc. Signs, symptoms, and conditions consistent with zinc insufficiency include: incomplete digestive proteolysis, food reactivities, reduced taste, reduced night vision, muscle aches, slowed wound healing, hair loss, dermatitis and sexual impotency. Mild lactic acidosis can be secondary

Commentary

to deficient zinc; lactic acid dehydrogenase requires zinc. Disordered insulin secretion from the pancreas can occur because insulin is stored in beta cells as a crystalline hexameric zinc complex. Immune dysfunction with impaired T-lymphocyte activity can be secondary to zinc insufficiency. In children, delayed growth or stunted growth may occur.

Genova Diagnostics®
Innovative Testing for Optimal Health
63 Zillicoa Street
Asheville, NC 28801

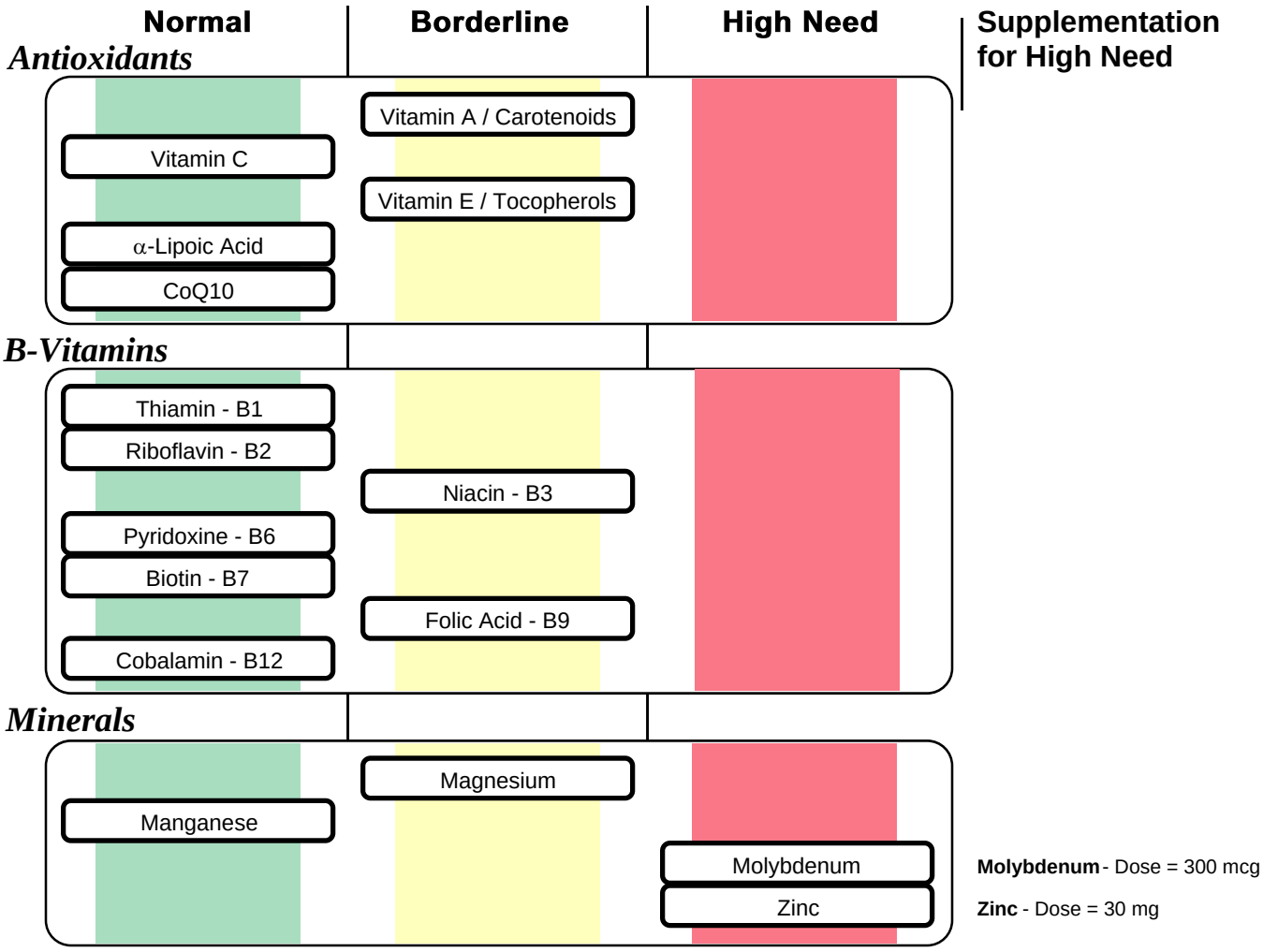
NutrEval® FMV amino acids

Patient: **SHAWN BEAN**
DOB: February 10, 1973
Sex: M
MRN: 0000713446

Order Number: D7030055
Completed: May 18, 2011
Received: May 03, 2011
Collected: May 02, 2011

Michael Overbeck MD
1100 Fayette St
Conshohocken, PA 19428

Results Overview



SUGGESTED SUPPLEMENT SCHEDULE

Supplements	Daily Recommended Intake (DRI)	Patient's Daily Recommendations	Provider Daily Recommendations
Antioxidants			
Vitamin A / Carotenoids	3,000 IU	5,000 IU	
Vitamin C	90 mg	250 mg	
Vitamin E / Tocopherols	22 IU	200 IU	
α-Lipoic Acid		50 mg	
CoQ10		30 mg	
B-Vitamins			
Thiamin - B1	1.2 mg	10 mg	
Riboflavin - B2	1.3 mg	10 mg	
Niacin - B3	16 mg	30 mg	
Pyridoxine - B6	1.3 mg	10 mg	
Biotin - B7	30 mcg	100 mcg	
Folic Acid - B9	400 mcg	800 mcg	
Cobalamin - B12	2.4 mcg	100 mcg	
Minerals			
Magnesium	420 mg	600 mg	
Manganese	2.3 mg	3.0 mg	
Molybdenum	45 mcg	300 mcg	
Zinc	11 mg	30 mg	
Essential Fatty Acids			
Omega-3 Oils	500 mg	500 mg	
Digestive Support			
Probiotics		10 billion CFU	
Pancreatic Enzymes		5,000 IU	
Other Vitamins			
Vitamin D	600 IU		

Amino Acid	mg/day	Amino Acid	mg/day
Arginine	655	Methionine	0
Asparagine	0	Phenylalanine	192
Cysteine	0	Serine	0
Glutamine	257	Taurine	0
Glycine	2,283	Threonine	0
Histidine	0	Tryptophan	0
Isoleucine	147	Tyrosine	253
Leucine	0	Valine	421
Lysine	828		

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

Interpretation At-A-Glance

Nutritional Needs

Antioxidants

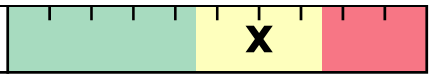
Vitamin A / Carotenoids

- 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 & progestin.
- 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.

Vitamin C

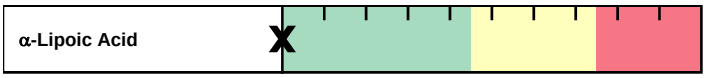
- Vitamin C is an antioxidant (also used in the regeneration of other antioxidants). It is involved in cholesterol metabolism, the production & function of WBCs and antibodies, and the synthesis of collagen, norepinephrine and carnitine.
- Deficiency may occur with oral contraceptives, aspirin, diuretics 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.

Vitamin E / Tocopherols



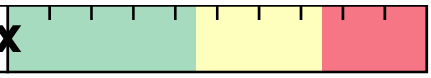
- Alpha-tocopherol (body's main form of vitamin E) functions as an antioxidant, regulates cell signaling, influences immune function and inhibits coagulation.
- Deficiency may occur with malabsorption, cholestyramine, colestipol, isoniazid, orlistat, olestra and certain anti-convulsants (e.g., phenobarbital, 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.

α-Lipoic Acid



- 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.

CoQ10

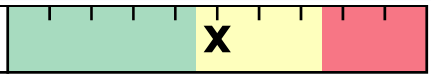


- CoQ10 is a powerful antioxidant that is synthesized in the body and contained in cell membranes. CoQ10 is also essential for energy production & pH regulation.
- CoQ10 deficiency may occur with HMG-CoA reductase inhibitors (statins), several anti-diabetic medication classes (biguanides, sulfonylureas) 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.

Glutathione

- 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.

Plant-based Antioxidants



- 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 nutriceuticals (e.g., resveratrol, EGCG, lutein, lycopene, ginkgo, milk thistle, etc.).

Key

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

NutrEval FMV amino acids

Interpretation At-A-Glance

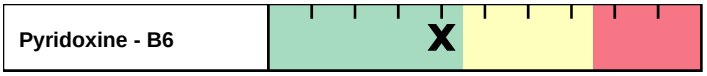
Nutritional Needs

B-Vitamins

Thiamin - B1

- 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.

Pyridoxine - B6



- 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.

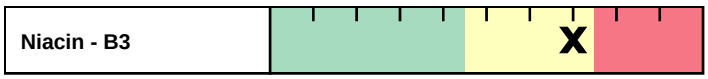
Riboflavin - B2

- 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 - B7

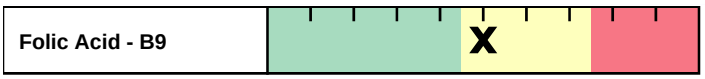
- Biotin is a cofactor for enzymes involved in functions such as fatty acid synthesis, mitochondrial FA oxidation, gluconeogenesis and DNA replication & transcription.
- Deficiency may result from certain inborn errors, chronic intake of raw egg whites, long-term TPN, anticonvulsants, high-dose B5, sulfa 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.

Niacin - B3



- 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 - B9



- 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.

Cobalamin - B12

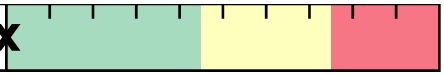
- B12 plays important roles in energy production from fats & proteins, methylation, synthesis of hemoglobin & RBCs, and maintenance of nerve cells, DNA & RNA.
- Low B12 may result from alcoholism, malabsorption, hypochlorhydria (e.g., from atrophic gastritis, H. pylori infection, pernicious anemia, H2 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.

Interpretation At-A-Glance

Nutritional Needs

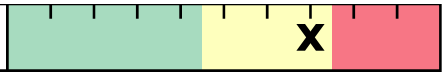
Minerals

Manganese



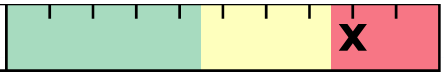
- Manganese plays an important role in antioxidant function, gluconeogenesis, the urea cycle, cartilage & bone formation, energy production and digestion.
- Impaired absorption of Mn may occur with excess intake of Fe, Ca, Cu, folic acid, or phosphorous compounds, or use of long-term TPN, Mg-containing 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



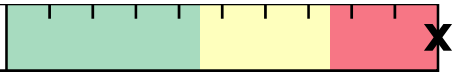
- Magnesium is involved in >300 metabolic reactions. Key areas include energy production, bone & ATP formation, muscle & nerve conduction and cell signaling.
- Deficiency may occur with malabsorption, alcoholism, hyperparathyroidism, renal disorders (wasting), diabetes, diuretics, digoxin or high doses 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



- 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).

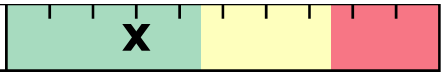
Zinc



- 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

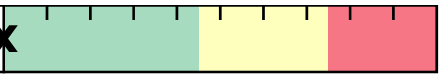
Need for Essential Fatty Acids



- 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.

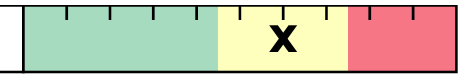
Digestive Support

Need for Probiotics



- 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.

Need for Pancreatic Enzymes



- 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.

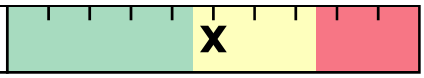
Interpretation At-A-Glance

Functional Imbalances

Mitochondrial Dysfunction

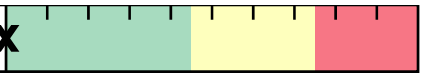
- Mitochondria are a primary site of generation of reactive oxygen species. Oxidative damage is considered an important factor in decline of physiologic function that occurs with aging and stress.
- Mitochondrial defects have been identified in cardiovascular disease, fatigue syndromes, neurologic disorders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

Toxic Exposure



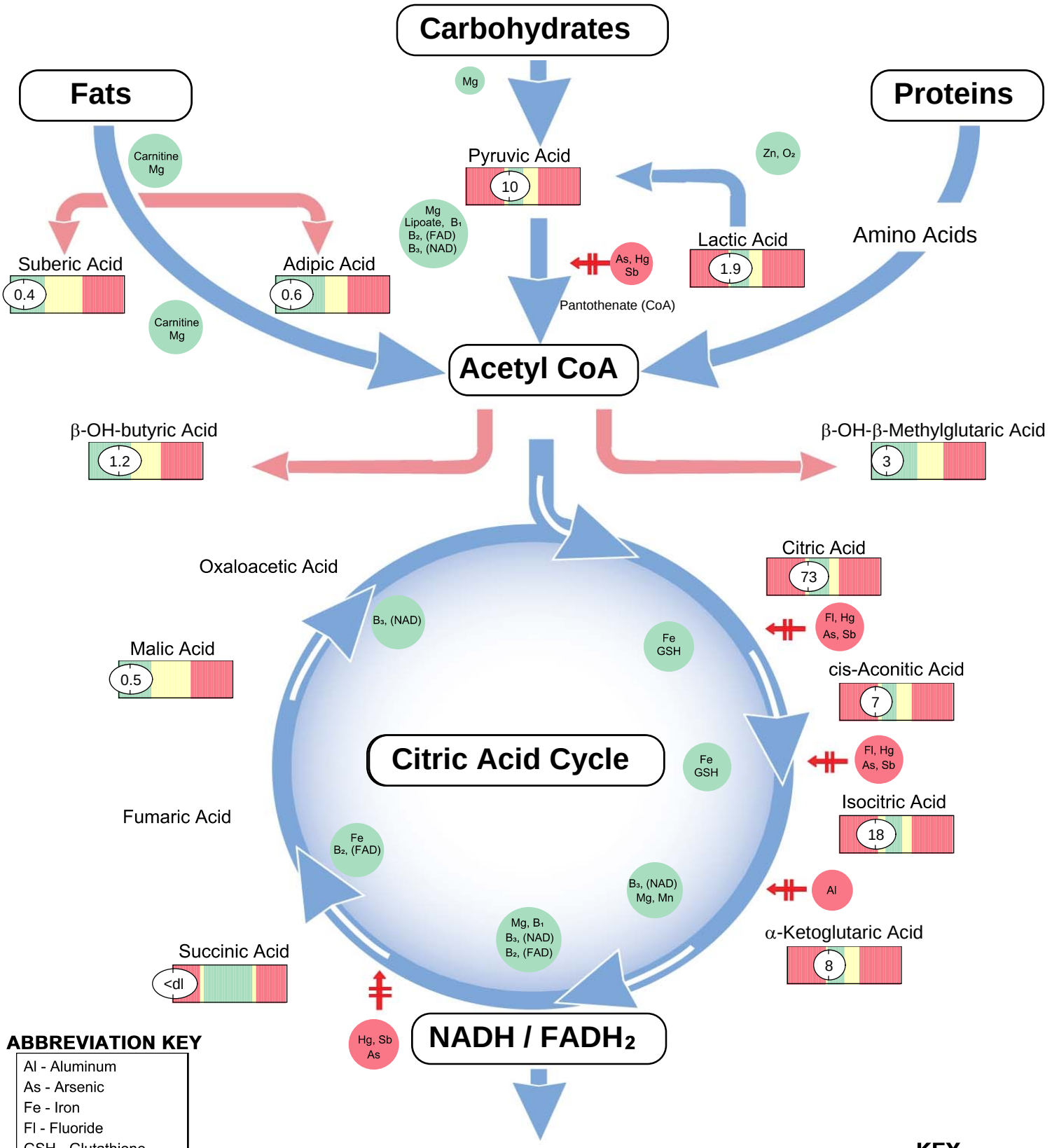
- Methyl tert-Butyl Ether (MTBE) is a common gasoline additive used to increase octane ratings, and has been found to contaminate ground water supplies where gasoline is stored. Inhalation of MTBE may cause nose and throat irritation, as well as headaches, nausea, dizziness and mental confusion. Animal 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.

Need for Methylation



- 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.

Krebs Cycle At-A-Glance



ABBREVIATION KEY

Al - Aluminum
As - Arsenic
Fe - Iron
Fl - Fluoride
GSH - Glutathione
Hg - Mercury
Mg - Magnesium
Mn - Manganese
Sb - Antimony
Zn - Zinc

Electron Transport and Oxidative Phosphorylation

$(2)\ H + 1/2\ O_2 \rightarrow H_2O$

KEY

Green = cofactor

Red = inhibitor

Metabolic Analysis Markers

All biomarkers reported in mmol/mol creatinine unless otherwise noted.

Malabsorption and Dysbiosis Markers

Malabsorption Markers

		Reference Range
Indoleacetic Acid (IAA)	1.2	<= 4.2
Phenylacetic Acid (PAA)	0.03	<= 0.12

Bacterial Dysbiosis Markers

		Reference Range
Dihydroxyphenylpropionic Acid (DHPPA)	0.4	<= 12.8
3-Hydroxyphenylacetic Acid	3.5	<= 8.1
4-Hydroxyphenylacetic Acid	5	<= 29
Benzoic Acid	0.02	<= 0.05
Hippuric Acid	215	<= 603

Yeast / Fungal Dysbiosis Markers

		Reference Range
Arabinose	14	<= 96
Citramalic Acid	1.0	<= 5.8
Tartaric Acid	<dl	<= 15

Cellular Energy & Mitochondrial Metabolites

Carbohydrate Metabolism

		Reference Range
Lactic Acid	1.9	1.9-19.8
Pyruvic Acid	10	7-32
β-OH-Butyric Acid (BHBA)	1.2	<= 2.8

Energy Metabolism

		Reference Range
Citric Acid	73	40-520
Cis-Aconitic Acid	7	10-36
Isocitric Acid	18	22-65
α-Ketoglutaric Acid (AKG)	8	4-52
Succinic Acid	<dl	0.4-4.6
Malic Acid	0.5	<= 3.0
β-OH-β-Methylglutaric Acid (HMG)	3	<= 15

Fatty Acid Metabolism

		Reference Range
Adipic Acid	0.6	<= 2.8
Suberic Acid	0.4	<= 2.1

Creatinine Concentration

		Reference Range
Creatinine ◆	9.6	3.1-19.5 mmol/L

Neurotransmitter Metabolites

		Reference Range
Vanilmandelic Acid	0.4	0.4-3.6
Homovanillic Acid	1.2	1.2-5.3
5-OH-indoleacetic Acid	2.3	3.8-12.1
3-Methyl-4-OH-phenylglycol	0.03	0.02-0.22
Kynurenic Acid	1.9	<= 7.1
Quinolinic Acid	2.4	<= 9.1
Kynurenic / Quinolinic Ratio	0.79	>= 0.44

Vitamin Markers

		Reference Range
α-Ketoadipic Acid	0.3	<= 1.7
α-Ketoisovaleric Acid	0.33	<= 0.97
α-Ketoisocaproic Acid	0.35	<= 0.89
α-Keto-β-Methylvaleric Acid	0.6	<= 2.1
Formiminoglutamic Acid (FIGlu)	0.9	<= 1.5
Glutaric Acid	0.10	<= 0.51
Isovalerylglycine	1.4	<= 3.7
Methylmalonic Acid	0.7	<= 1.9
Xanthurenic Acid	0.24	<= 0.96
3-Hydroxypropionic Acid	5	5-22
3-Hydroxyisovaleric Acid	7	<= 29

Toxin & Detoxification Markers

		Reference Range
α-Ketophenylacetic Acid (from Styrene)	0.16	<= 0.46
α-Hydroxyisobutyric Acid (from MTBE)	4.6	<= 6.7
Orotic Acid	0.29	0.33-1.01
Pyroglutamic Acid	14	16-34

Tyrosine Metabolism

		Reference Range
Homogentisic Acid	3	<= 19
2-Hydroxyphenylacetic Acid	0.37	<= 0.76

Metabolic Analysis Reference Ranges are Age Specific

The performance characteristics of all assays have been verified by Genova Diagnostics, Inc. Unless otherwise noted with ◆ as cleared by the U.S. Food and Drug Administration, assays are For Research Use Only.

Amino Acids (FMV)

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

Nutritionally Essential Amino Acids

Amino Acid	Reference Range
Arginine	10-64
Histidine	271-993
Isoleucine	17-52
Leucine	25-77
Lysine	34-226
Methionine	26-69
Phenylalanine	22-61
Taurine	80-545
Threonine	52-192
Tryptophan	23-88
Valine	19-53

Nonessential Protein Amino Acids

Amino Acid	Reference Range
Alanine	103-392
Asparagine	37-134
Aspartic Acid	27-74
Cysteine	19-70
Cystine	23-68
γ-Aminobutyric Acid	<= 23
Glutamic Acid	3-15
Glutamine	153-483
Proline	2-14
Tyrosine	28-113

Creatinine Concentration

	Reference Range
Creatinine ◆	3.1-19.5 mmol/L

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Intermediary Metabolites

B Vitamin Markers	Reference Range
α-Aminoadipic Acid	11-73
α-Amino-N-butyric Acid	9-49
β-Aminoisobutyric Acid	19-163
Cystathionine	6-29
3-Methylhistidine	134-302

Urea Cycle Markers	Reference Range
Ammonia	12.0-41.0 mmol/g creatinine
Citrulline	9-40
Ornithine	3-16
Urea ◆	150-380 mmol/g creatinine

Glycine/Serine Metabolites	Reference Range
Glycine	434-1,688
Serine	135-426
Ethanolamine	156-422
Phosphoethanolamine	14-50
Phosphoserine	26-64
Sarcosine	<= 41

Dietary Peptide Related Markers

	Reference Range
Anserine (dipeptide)	8-118
Carnosine (dipeptide)	12-120
1-Methylhistidine	83-1,008
β-Alanine	<= 17

Markers for Urine Representativeness

	Reference Range
Glutamine/Glutamate	>= 12
Ammonia	12.0-41.0 mmol/g creatinine
Arginine/Ornithine	>= 1.0

Urine Representativeness Index

Ref Range 5 – 10

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	0.20	>= 0.09 wt %
Eicosapentaenoic (EPA) 20:5 n3	0.52	>= 0.16 wt %
Docosapentaenoic (DPA) 22:5 n3	2.34	>= 1.14 wt %
Docosahexaenoic (DHA) 22:6 n3	5.8	>= 2.1 wt %
% Omega 3s	8.8	>= 3.8

Omega 9 Fatty Acids

Analyte	(olive oil)	Reference Range
Oleic 18:1 n9	9	10-13 wt %
Nervonic 24:1 n9	2.2	2.1-3.5 wt %
% Omega 9s	11.6	13.3-16.6

Saturated Fatty Acids

Analyte	(meat, dairy, coconuts, palm oils)	Reference Range
Palmitic C16:0	20	18-23 wt %
Stearic C18:0	18	14-17 wt %
Arachidic C20:0	0.18	0.22-0.35 wt %
Behenic C22:0	0.69	0.92-1.68 wt %
Tricosanoic C23:0	0.15	0.12-0.18 wt %
Lignoceric C24:0	2.0	2.1-3.8 wt %
Pentadecanoic C15:0	0.07	0.07-0.15 wt %
Margaric C17:0	0.25	0.22-0.37 wt %
% Saturated Fats	41.2	39.8-43.6

Omega 6 Fatty Acids

Analyte	(vegetable oil, grains, most meats, dairy)	Reference Range
Linoleic (LA) 18:2 n6	12.1	10.5-16.9 wt %
γ-Linolenic (GLA) 18:3 n6	0.06	0.03-0.13 wt %
Dihomo-γ-linolenic (DGLA) 20:3 n6	1.26	>= 1.19 wt %
Arachidonic (AA) 20:4 n6	20	15-21 wt %
Docosatetraenoic (DTA) 22:4 n6	2.83	1.50-4.20 wt %
Eicosadienoic 20:2 n6	0.35	<= 0.26 wt %
% Omega 6s	37.1	30.5-39.7

Monounsaturated Fats

Omega 7 Fats		Reference Range
Palmitoleic 16:1 n7	0.19	<= 0.64 wt %
Vaccenic 18:1 n7	0.79	<= 1.13 wt %
Trans Fat		
Elaidic 18:1 n9t	0.31	<= 0.59 wt %

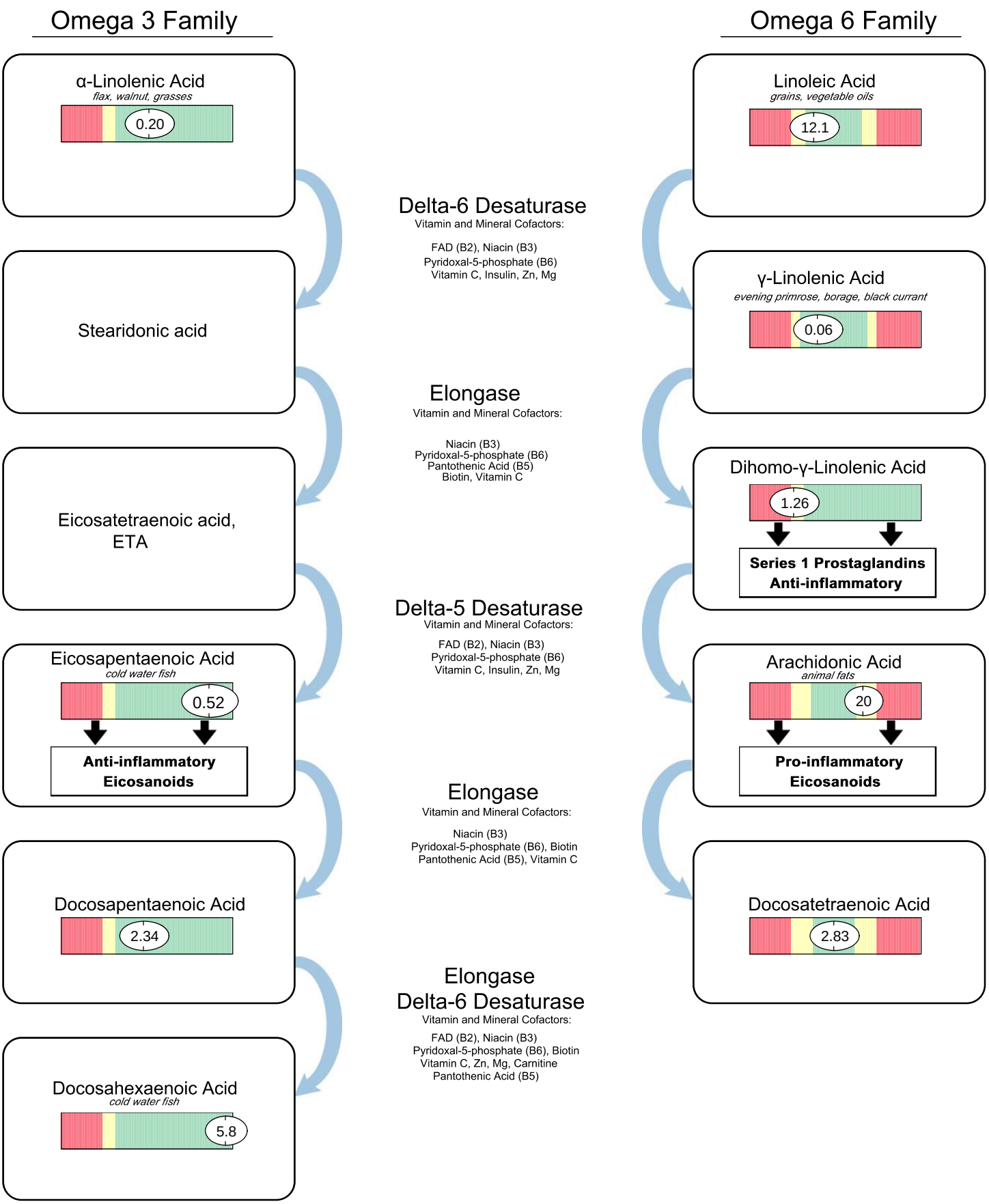
Delta - 6 Desaturase Activity

	Upregulated Functional Impaired	
Linoleic / DGLA 18:2 n6 / 20:3 n6	9.6	6.0-12.3

Cardiovascular Risk

Analyte		Reference Range
Omega 6s / Omega 3s	4.2	3.4-10.7
AA / EPA 20:4 n6 / 20:5 n3	39	12-125
Omega 3 Index	6.3	>= 4.0

Essential Fatty Acid Metabolism



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

Oxidative Stress Markers

Oxidative Stress Markers

		Reference Range
Glutathione (whole blood)	1,110	>= 669 micromol/L
Lipid Peroxides (urine)	3.7	<=10.0 micromol/g Creat.
8-OHdG (urine)	4	<=16 mcg/g Creat.
Coenzyme Q10, Ubiquinone (plasma)	0.75	0.46-1.72 mcg/mL

Elemental Markers (RBCs)

Nutrient Elements

Element	Reference Range	Reference Range
Copper	0.475	0.466-0.721 mcg/g
Magnesium	34.0	30.1-56.5 mcg/g
Manganese	0.013	0.007-0.038 mcg/g
Potassium	2,454	2,220-3,626 mcg/g
Selenium	0.23	0.25-0.76 mcg/g
Zinc	6.5	7.8-13.1 mcg/g

Toxic Elements

Element	Reference Range	Reference Range
Lead	0.017	<= 0.048 mcg/g
Mercury	0.0040	<= 0.0039 mcg/g
Antimony	0.001	<= 0.002 mcg/g
Arsenic	0.010	<= 0.071 mcg/g
Cadmium	0.000	<= 0.001 mcg/g
Tin	<dl	<= 0.0009 mcg/g

Lab Comments

The performance characteristics of all assays have been verified by Genova Diagnostics, Inc. Unless otherwise noted with ◆ as cleared by the U.S. Food and Drug Administration, assays are For Research Use Only.