GENOVA DIAGNOSTICS EUROPE

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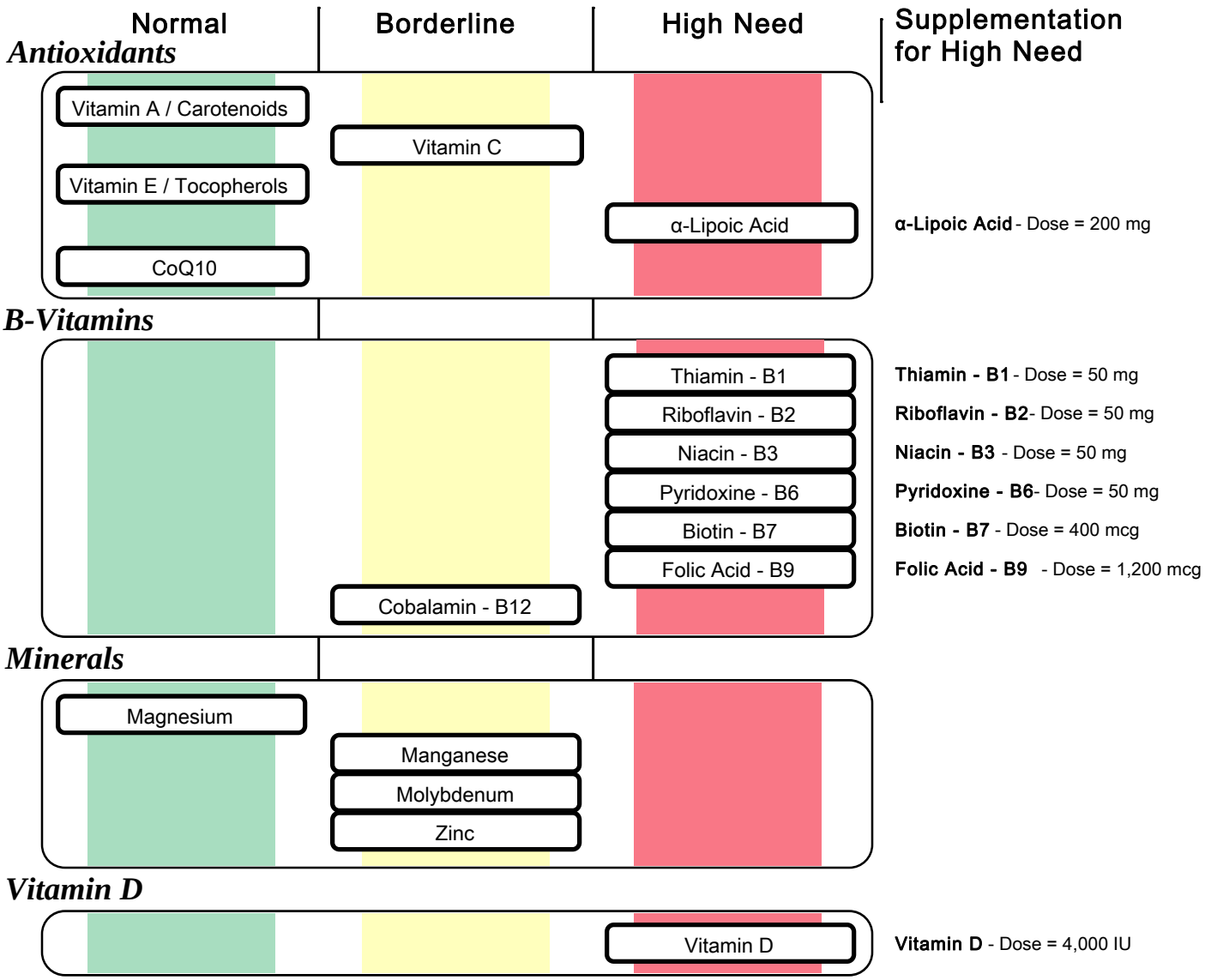
NutrEval Results Overview



SUGGESTED SUPPLEMENT SCHEDULE

Supplements	Daily Recommended Intake (DRI)	Patient's Daily Recommendations	Provider Daily Recommendations
Antioxidants			
Vitamin A / Carotenoids	2,333 IU	3,000 IU	
Vitamin C	75 mg	500 mg	
Vitamin E / Tocopherols	22 IU	100 IU	
α-Lipoic Acid		200 mg	
CoQ10		30 mg	
B-Vitamins			
Thiamin - B1	1.1 mg	50 mg	
Riboflavin - B2	1.1 mg	50 mg	
Niacin - B3	14 mg	50 mg	
Pyridoxine - B6	1.3 mg	50 mg	
Biotin - B7	30 mcg	400 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	5.0 mg	
Molybdenum	45 mcg	150 mcg	
Zinc	8 mg	20 mg	
Essential Fatty Acids			
Omega-3 Oils	500 mg	500 mg	
Digestive Support			
Probiotics		25 billion CFU	
Pancreatic Enzymes		5,000 IU	
Other Vitamins			
Vitamin D	600 IU	4,000 IU	

Amino Acid	mg/day	Amino Acid	mg/day
Arginine	186	Methionine	0
Asparagine	0	Phenylalanine	0
Cysteine	0	Serine	0
Glutamine	22	Taurine	0
Glycine	0	Threonine	36
Histidine	0	Tryptophan	0
Isoleucine	0	Tyrosine	0
Leucine	0	Valine	0
Lysine	362		

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.

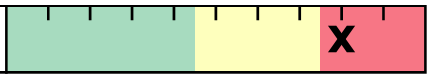
Vitamin C

- 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.

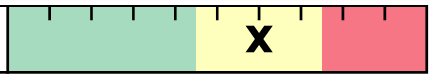
CoQ10

- 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

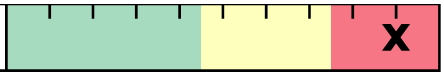
NutrEval FMV amino acids

Interpretation At-A-Glance

Nutritional Needs

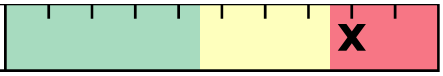
B-Vitamins

Thiamin - B1



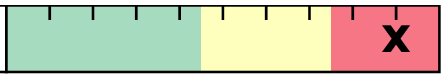
- B1 is a required cofactor for enzymes involved in energy production from food, and for the synthesis of ATP, GTP, DNA, RNA and NADPH.
- Low B1 can result from chronic alcoholism, diuretics, digoxin, oral contraceptives and HRT, or large amounts of tea & coffee (contain anti-B1 factors).
- B1 deficiency 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



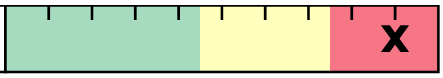
- B6 (as P5P) is a cofactor for enzymes involved in glycogenolysis & gluconeogenesis, and synthesis of neurotransmitters, heme, B3, RBCs and nucleic acids.
- Low B6 may result from chronic alcoholism, long-term diuretics, estrogens (oral contraceptives and HRT), anti-TB meds, penicillamine, L-DOPA or 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



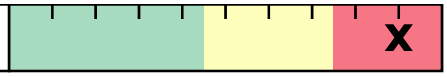
- B2 is a key component of enzymes involved in antioxidant function, energy production, detoxification, methionine metabolism and vitamin activation.
- Low B2 may result from chronic alcoholism, some anti-psychotic medications, oral contraceptives, tricyclic antidepressants, quinacrine or adriamycin.
- 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



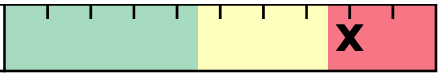
- Biotin is a cofactor for enzymes involved in functions such as fatty acid synthesis, mitochondrial FA oxidation, gluconeogenesis and DNA replication & transcription.
- Deficiency may result from certain inborn errors, chronic intake of raw egg whites, long-term TPN, anticonvulsants, high-dose B5, sulfa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



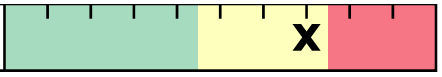
- B3 is used to form NAD and NADP, involved in energy production from food, fatty acid & cholesterol synthesis, cell signaling, DNA repair & cell differentiation.
- Low B3 may result from deficiencies of tryptophan (B3 precursor), B6, B2 or Fe (cofactors in B3 production), or from long-term isoniazid 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



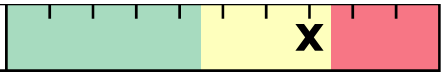
- B12 plays important roles in energy production from fats & proteins, methylation, synthesis of hemoglobin & RBCs, and maintenance of nerve cells, DNA & RNA.
- Low B12 may result from alcoholism, malabsorption, hypochlorhydria (e.g., from atrophic gastritis, H. pylori infection, pernicious anemia, H2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

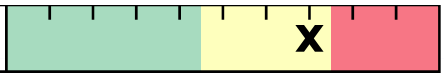


- Manganese plays an important role in antioxidant function, gluconeogenesis, the urea cycle, cartilage & bone formation, energy production and digestion.
- Impaired absorption of Mn may occur with excess intake of Fe, Ca, Cu, folic acid, or phosphorous compounds, or use of long-term TPN, Mg-containing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

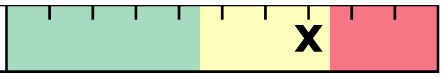
- Magnesium is involved in >300 metabolic reactions. Key areas include energy production, bone & ATP formation, muscle & nerve conduction and cell signaling.
- Deficiency may occur with malabsorption, alcoholism, hyperparathyroidism, renal disorders (wasting), diabetes, diuretics, digoxin or high doses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

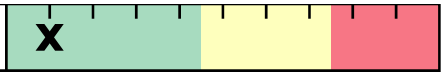
Zinc



- 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

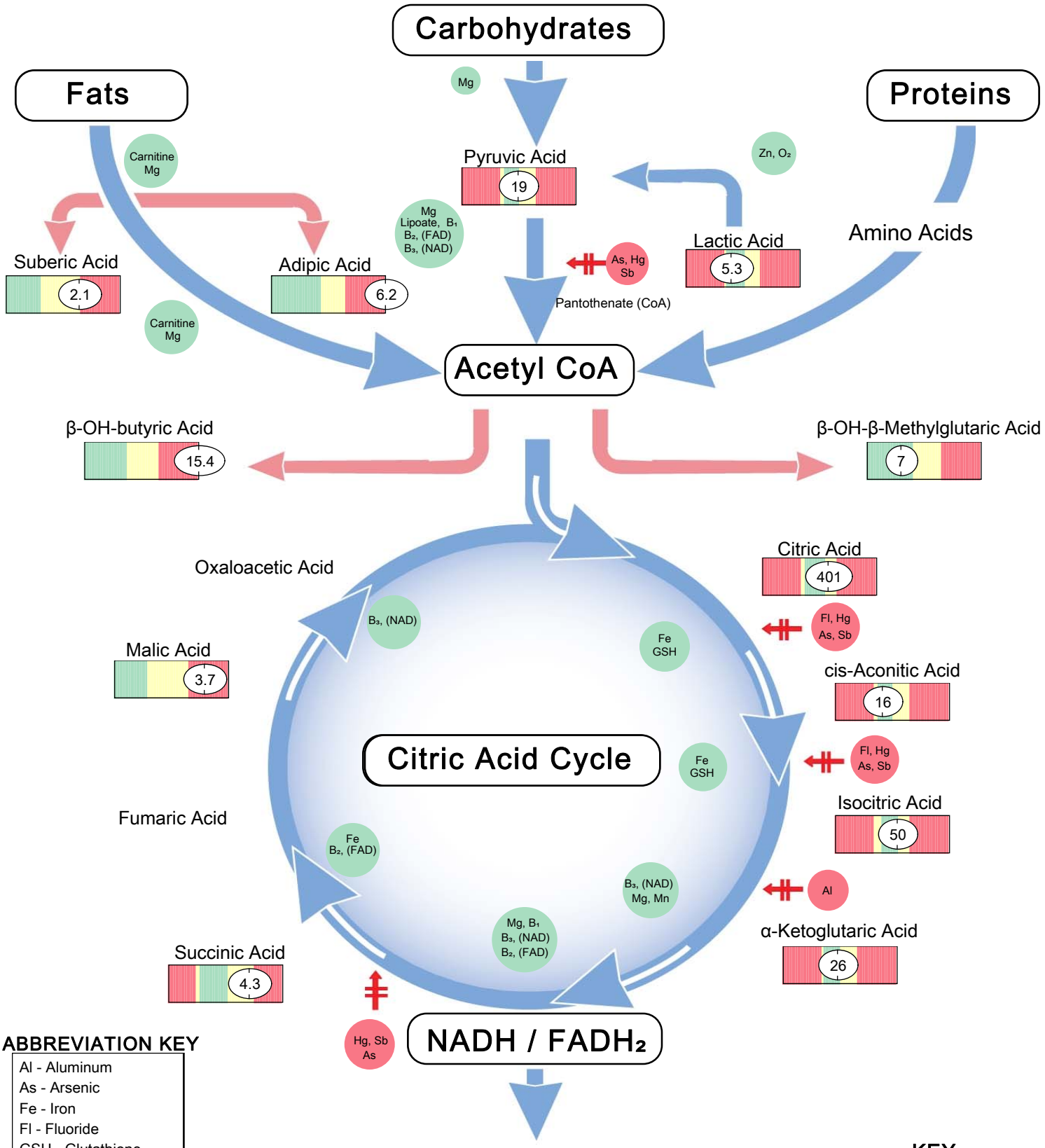
Need for Probiotics

- 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.

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

KEY

Green = cofactor

Red = inhibitor

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)	1.5	<= 5.3
3-Hydroxyphenylacetic Acid	6.5	<= 8.1
4-Hydroxyphenylacetic Acid	15	<= 29
Benzoic Acid	0.14	<= 0.05
Hippuric Acid	<dl	<= 603

Yeast / Fungal Dysbiosis Markers

		Reference Range
Arabinose	80	<= 96
Citramalic Acid	2.5	<= 5.8
Tartaric Acid	<dl	<= 15

Cellular Energy & Mitochondrial Metabolites

Carbohydrate Metabolism

		Reference Range
Lactic Acid	5.3	1.9-19.8
Pyruvic Acid	19	7-32
β-OH-Butyric Acid (BHBA)	15.4	<= 2.8

Energy Metabolism

		Reference Range
Citric Acid	401	40-520
Cis-Aconitic Acid	16	10-36
Isocitric Acid	50	22-65
α-Ketoglutaric Acid (AKG)	26	4-52
Succinic Acid	4.3	0.4-4.6
Malic Acid	3.7	<= 3.0
β-OH-β-Methylglutaric Acid (HMG)	7	<= 15

Fatty Acid Metabolism

		Reference Range
Adipic Acid	6.2	<= 2.8
Suberic Acid	2.1	<= 2.1

Creatinine Concentration

		Reference Range
Creatinine ◆	10.9	3.1-19.5 mmol/L

Neurotransmitter Metabolites

		Reference Range
Vanilmandelic Acid	1.4	0.4-3.6
Homovanillic Acid	1.7	1.2-5.3
5-OH-indoleacetic Acid	12.4	3.8-12.1
3-Methyl-4-OH-phenylglycol	0.08	0.02-0.22
Kynurenic Acid	10.4	<= 7.1
Quinolinic Acid	4.3	<= 9.1
Kynurenic / Quinolinic Ratio	2.42	>= 0.44

Vitamin Markers

		Reference Range
α-Ketoadipic Acid	1.1	<= 1.7
α-Ketoisovaleric Acid	0.85	<= 0.97
α-Ketoisocaproic Acid	0.95	<= 0.89
α-Keto-β-Methylvaleric Acid	2.5	<= 2.1
Formiminoglutamic Acid (FIGlu)	2.2	<= 1.5
Glutaric Acid	0.50	<= 0.51
Isovalerylglycine	3.8	<= 3.7
Methylmalonic Acid	1.0	<= 1.9
Xanthurenic Acid	0.96	<= 0.96
3-Hydroxypropionic Acid	13	5-22
3-Hydroxyisovaleric Acid	33	<= 29

Toxin & Detoxification Markers

		Reference Range
α-Ketophenylacetic Acid (from Styrene)	0.29	<= 0.46
α-Hydroxyisobutyric Acid (from MTBE)	5.4	<= 6.7
Orotic Acid	1.23	0.33-1.01
Pyroglutamic Acid	29	16-34

Tyrosine Metabolism

		Reference Range
Homogentisic Acid	5	<= 19
2-Hydroxyphenylacetic Acid	0.68	<= 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 ◆, the assay has not been cleared by the U.S. Food and Drug Administration.

Amino Acids (Urine FMV)

Nutritionally Essential Amino Acids

Amino Acid	Reference Range
Arginine	10-64
Histidine	296-1,136
Isoleucine	24-58
Leucine	30-87
Lysine	45-286
Methionine	30-82
Phenylalanine	26-71
Taurine	68-538
Threonine	65-252
Tryptophan	28-111
Valine	23-61

Nonessential Protein Amino Acids

Amino Acid	Reference Range
Alanine	146-486
Asparagine	49-182
Aspartic Acid	35-86
Cysteine	21-78
Cystine	26-78
γ-Aminobutyric Acid	<= 31
Glutamic Acid	5-21
Glutamine	172-570
Proline	2-18
Tyrosine	33-124

Creatinine Concentration

	Reference Range
Creatinine ◆	3.1-19.5 mmol/L

Amino Acid Reference Ranges are Age Specific

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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	22-192
Cystathionine	6-33
3-Methylhistidine	131-318

Urea Cycle Markers

	Reference Range
Ammonia	14.0-49.0 mmol/g creatinine
Citrulline	12-45
Ornithine	4-21
Urea ◆	168-465 mmol/g creatinine

Glycine/Serine Metabolites

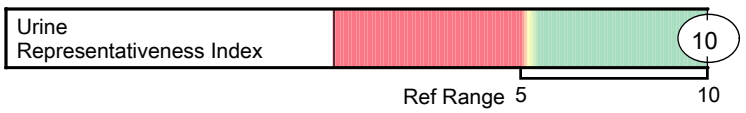
	Reference Range
Glycine	639-3,306
Serine	187-568
Ethanolamine	208-514
Phosphoethanolamine	18-70
Phosphoserine	28-63
Sarcosine	<= 48

Dietary Peptide Related Markers

	Reference Range
Anserine (dipeptide)	7-126
Carnosine (dipeptide)	10-104
1-Methylhistidine	92-1,046
β-Alanine	<= 21

Markers for Urine Representativeness

	Reference Range
Glutamine/Glutamate	>= 10
Ammonia	14.0-49.0 mmol/g creatinine
Arginine/Ornithine	>= 1.1



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	4.52	>= 0.16 wt %
Docosapentaenoic (DPA) 22:5 n3	2.45	>= 1.14 wt %
Docosahexaenoic (DHA) 22:6 n3	8.0	>= 2.1 wt %
% Omega 3s	15.1	>= 3.8

Omega 9 Fatty Acids

Analyte	(olive oil)	Reference Range
Oleic 18:1 n9	12	10-13 wt %
Nervonic 24:1 n9	3.7	2.1-3.5 wt %
% Omega 9s	15.6	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	16	14-17 wt %
Arachidic C20:0	0.23	0.22-0.35 wt %
Behenic C22:0	0.95	0.92-1.68 wt %
Tricosanoic C23:0	0.21	0.12-0.18 wt %
Lignoceric C24:0	2.2	2.1-3.8 wt %
Pentadecanoic C15:0	0.17	0.07-0.15 wt %
Margaric C17:0	0.36	0.22-0.37 wt %
% Saturated Fats	40.3	39.8-43.6

Omega 6 Fatty Acids

Analyte	(vegetable oil, grains, most meats, dairy)	Reference Range
Linoleic (LA) 18:2 n6	8.9	10.5-16.9 wt %
γ-Linolenic (GLA) 18:3 n6	0.04	0.03-0.13 wt %
Dihomo-γ-linolenic (DGLA) 20:3 n6	0.70	>= 1.19 wt %
Arachidonic (AA) 20:4 n6	17	15-21 wt %
Docosatetraenoic (DTA) 22:4 n6	1.11	1.50-4.20 wt %
Eicosadienoic 20:2 n6	0.18	<= 0.26 wt %
% Omega 6s	27.7	30.5-39.7

Monounsaturated Fats

Omega 7 Fats		Reference Range
Palmitoleic 16:1 n7	0.27	<= 0.64 wt %
Vaccenic 18:1 n7	0.73	<= 1.13 wt %
Trans Fat		
Elaidic 18:1 n9t	0.34	<= 0.59 wt %

Delta - 6 Desaturase Activity

	Upregulated Functional Impaired	
Linoleic / DGLA 18:2 n6 / 20:3 n6	12.8	6.0-12.3

Cardiovascular Risk

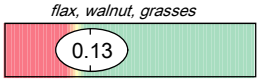
Analyte		Reference Range
Omega 6s / Omega 3s	1.8	3.4-10.7
AA / EPA 20:4 n6 / 20:5 n3	4	12-125
Omega 3 Index	12.5	>= 4.0

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

Essential Fatty Acid Metabolism

Omega 3 Family

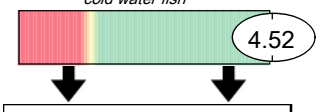
α-Linolenic Acid
flax, walnut, grasses



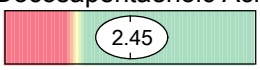
Stearidonic acid

Eicosatetraenoic acid, ETA

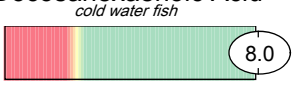
Eicosapentaenoic Acid
cold water fish



Docosapentaenoic Acid



Docosahexaenoic Acid
cold water fish



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

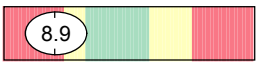
Delta-5 Desaturase
Vitamin and Mineral Cofactors:
FAD (B2), Niacin (B3)
Pyridoxal-5-phosphate (B6)
Vitamin C, Insulin, Zn, Mg

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

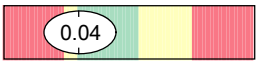
Elongase
Delta-6 Desaturase
Vitamin and Mineral Cofactors:
FAD (B2), Niacin (B3)
Pyridoxal-5-phosphate (B6), Biotin
Vitamin C, Zn, Mg, Carnitine
Pantothenic Acid (B5)

Omega 6 Family

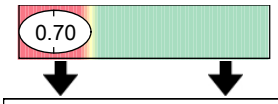
Linoleic Acid
grains, vegetable oils



γ-Linolenic Acid
evening primrose, borage, black currant



Dihomo-γ-Linolenic Acid



Arachidonic Acid
animal fats

Docosatetraenoic Acid

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
Glutathione (whole blood)	705	>=669 micromol/L
Lipid Peroxides (urine)	6.9	<=10.0 micromol/g Creat.
8-OHdG (urine)	9	<=16 mcg/g Creat.
Coenzyme Q10, Ubiquinone (plasma)	0.73	0.43-1.49 mcg/mL

The Oxidative Stress reference ranges are based on an adult population.

Vitamin D (Serum)

	Inside Range	Outside Range	Reference Range
25 - OH Vitamin D ◆		24	50-100 ng/mL

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 (RBCs)

Nutrient Elements

Element	Reference Range	Reference Range
Copper	0.495	0.466-0.721 mcg/g
Magnesium	53.3	30.1-56.5 mcg/g
Manganese	0.011	0.007-0.038 mcg/g
Potassium	3,019	2,220-3,626 mcg/g
Selenium	0.49	0.25-0.76 mcg/g
Zinc	8.6	7.8-13.1 mcg/g

Toxic Elements

Element	Reference Range	Reference Range
Lead	0.034	<= 0.048 mcg/g
Mercury	0.0113	<= 0.0039 mcg/g
Antimony	0.001	<= 0.002 mcg/g
Arsenic	0.029	<= 0.071 mcg/g
Cadmium	<dl	<= 0.001 mcg/g
Tin	<dl	<= 0.0009 mcg/g

The Elemental reference ranges are based on an adult population.

Lab Comments

Lab Comments

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.

Homocysteine (Plasma)

GENOVA DIAGNOSTICS EUROPE

Parkgate House
356 West Barnes Lane
New Malden, Surrey KT3 6NB

63 Zillicoa Street
Asheville, NC 28801 USA

Homocysteine

	Inside Range	Outside Range	Reference Range
Homocysteine		3.00	3.70-10.40 umol/L

Commentary

Lab Comments

The reference range for homocysteine is based on the sex-specific 5th to 95th percentile values for men and women (20 to 39 years of age) in the NHANES nutritionally replete cohort. Annals of Internal Medicince 1999; 131 (331-338).

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.

Homocysteine is BELOW the REFERENCE level for this person. Low homocysteine translates into diminished cardiovascular risk and is an important BENEFICIAL finding for this person. Positive genetic, nutritional and lifestyle factors are likely to be responsible for this favorable finding.

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.

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.

5-Hydroxyindoleacetic Acid (5-HIAA) is elevated. 5-HIAA is a normal urine metabolite of the neurotransmitter serotonin, which is formed from the essential amino acid, tryptophan. 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.

Slightly or moderately elevated 5-HIAA may result from increased formation of serotonin, a vasoconstrictor and smooth-muscle contractor, in the small intestine. Secondary inflammatory responses may be present. Slightly or moderately increased 5-HIAA may also be a dietary artifact from consumption of relatively large amounts of bananas, plantain, pineapple, kiwi fruit, plums, avocado, walnuts or pecans. Similarly, the medications acetaminophen and guaifenesin can elevate urinary 5-HIAA. Elevated 5-HIAA may also occur if methylation by S-adenosylmethionine (SAM) is impaired, as methylation of serotonin is needed to produce other products of serotonin: melatonin and 5-methoxy-3-indoleacetic acid (a waste metabolite like 5-HIAA). Notably high levels of 5-HIAA (and serotonin) are found in carcinoid disease, where malignant cells in the intestine, particularly the ileum, produce excess serotonin.

Malic Acid (malate) is measured to be elevated. An important intermediate of the citric acid cycle in cell mitochondria, malic acid or malate is formed from fumaric acid (fumarate), and it becomes oxaloacetic acid. Malic acid also participates in the malate-aspartate shuttle, a cellular process in which malate and a proton (H+) can enter the mitochondrion from the cytosol. This brings a chemical reducing equivalent, H+, inside the mitochondrial membrane. This is the mechanism whereby the NADH produced in glycolysis can enter the mitochondria to participate in oxidative phosphorylation.

Malic acid can be elevated if its dehydrogenation to oxaloacetic acid is reduced; this dehydrogenase enzyme requires vitamin B3 as NAD. Malate can also be high if oxaloacetic acid is high. Use of D-malic acid (or D,L-malate) as a nutritional supplement, instead of L-malic acid will also cause elevated urine levels, since this compound will interfere with its metabolism. Only L-malic acid can be utilized properly.

Commentary

Impairments in pyruvate metabolism with elevated pyruvate and lactate usually result in elevated malate, as well. (Refer to commentary for these analytes.)

Adipic Acid and/or **Suberic Acid** is elevated in the urine. Adipic acid and suberic acid are both products of omega-oxidation of fatty acids, a process that occurs when normal beta-oxidation (inside cell mitochondria) is impaired. Since carnitine is necessary for the transport of long-chain fatty acids into the mitochondrial matrix, carnitine insufficiency can lead to elevations in these compounds. Another possible cause of sub-optimal beta-oxidation is weakness of the dehydrogenase process in beta-oxidation of fatty acids (which inserts a double bond in the fatty acid being oxidized). This dehydrogenation uses vitamin B2 as FAD. Decreased dehydrogenase activity will limit the rate at which fatty acids can be broken down into acetyl CoA for subsequent entry into the citric acid cycle and/or for ketone formation.

This abnormal omega-oxidation of fatty acids involve the addition of a carboxyl group to the methyl end (or the "omega end") of the fatty acid, resulting in a dicarboxylic acid (carboxyl groups at both ends). Some degree of normal beta-oxidation may follow this. Individuals with excessive amounts of adipic or suberic acids often present with lethargy, fatigue, and hypoglycemia.

On the other hand, adipic acid and/or suberic acid excess can also result from a shift towards ketosis (increased beta-oxidation activity). In ketosis, C-10 to C-14 monocarboxylic fatty acids are mobilized from adipose tissue to the liver, and some of this becomes adipic acid or suberic acid. Furthermore, individuals in a state of fasting or starvation or with diabetic ketosis may also show elevated urinary levels of adipic or suberic acids since omega-oxidation is upregulated and followed by beta-oxidation in order to produce succinyl CoA. This allows for improved utilization of acetyl CoA and increased ATP production. Alternatively, succinyl CoA becomes malate in the citric acid cycle, and malate is free to leave the mitochondria and enter the cytoplasm where it can be oxidized to oxaloacetate and eventually lead to gluconeogenesis. In this manner, even in severe fasting, additional small amounts of glucose, critical for neurological function, can be formed.

Beta-hydroxybutyric Acid (BHBA) is elevated. BHBA is a "ketone body". Excess BHBA is consistent with ketosis or ketoacidosis (a type of metabolic acidosis). Under normal conditions, carbohydrates or fatty acids are metabolized to acetyl CoA; the acetyl CoA then enters the mitochondria and combines with oxaloacetic acid to form citric acid, in the first "step" of the citric acid cycle. In the case of a high-fat diet or inadequate carbohydrates (leading to low oxaloacetic acid and increased breakdown of fat for energy), more acetyl CoA is formed, which then forms acetoacetyl CoA and eventually leads to the formation of BHBA and other ketones.

The ketone BHBA cannot be metabolized in liver cells, and it is transported via the blood stream to muscle, brain, heart and kidney tissues for oxidation, to meet their energy needs. Ketoacidosis may occur with: fasting, anorexia or starvation, diabetes, or during and following an extended period of severe exercise. Ketogenic diets may also result in elevated urine BHBA. Less common conditions causing ketoacidosis are those originating from metabolic or (severe) nutritional deficiencies such as methylmalonic aciduria, due to B12 deficiency.

The severity of ketosis is not accurately reflected by the degree of ketonuria. Only a small amount of the body burden of ketones is excreted in urine; most must be oxidized in extrahepatic tissue using (and depleting) available oxygen.

Alpha-ketoisocaproic Acid (AKIC) is measured to be elevated. This organic acid is formed from the essential amino acid leucine by transamination. Moderate elevations of AKIC usually mean that AKIC's further metabolism to the compound, isovaleryl-CoA, is impaired either by coenzyme/cofactor insufficiency or by (genetic) weakness in the enzyme complex. The dehydrogenase enzyme complex that accomplishes this requires lipoic acid, vitamin B1 as thiamin pyrophosphate and vitamin B2 as FAD. Vitamin B3 as NAD is a necessary cofactor; it removes hydrogen to

Commentary

become NADH. Coenzyme A (requiring pantothenic acid, cysteine, magnesium, and ATP) is a second, necessary cofactor. Elevated levels of AKIV may indicate insufficiencies of any of these nutrients.

The toxic elements arsenic, antimony, mercury and cadmium may also weaken lipoic acid and dehydrogenase activity. Very high AKIC and its sister keto acids (alpha-ketoisovaleric, alpha-keto-beta-methylvaleric) constitute a rare disorder called "maple syrup urine disease". When AKIC is elevated, leucine may also be elevated per urine or plasma urine amino acid analysis.

Very rare (less than 1 in 100,000) causes of elevated AKIC include metabolic weakness in enzymes that metabolize the organic acids formed from isovaleryl-CoA in the oxidative catabolism of leucine. Several disorders may result from these defects; however, their differential diagnosis is beyond the scope of this test, and should be evaluated by a metabolic specialist.

Alpha-keto-beta-methylvaleric Acid (AKBM) is measured to be elevated. AKBM comes from the essential amino acid isoleucine via transamination. Moderate elevations of AKBM usually mean that AKBM's further metabolism to the compound, alpha-methylbutyryl-CoA, is impaired either by coenzyme/cofactor insufficiency or by (genetic) weakness in the enzyme complex. The dehydrogenase enzyme complex that accomplishes this requires lipoic acid, vitamin B1 as thiamin pyrophosphate and vitamin B2 as FAD. Vitamin B3 as NAD is a necessary cofactor, which removes hydrogen to become NADH. The other necessary cofactor is coenzyme A, requiring pantothenic acid, cysteine, and magnesium. Elevated levels of AKBM may indicate low levels of any of these nutrients.

The toxic elements arsenic, antimony, mercury and cadmium may also weaken lipoic acid and dehydrogenase activity. Very high elevation of AKBM and its sister keto acids (alpha-ketoisovaleric, alpha-ketoisocaproic) constitute a rare disorder called "maple syrup urine disease". When AKBM is elevated, isoleucine may also be elevated per urine or plasma amino acid analysis.

Kynurenic Acid is high; it is a possible metabolite of tryptophan, and it comes directly from kynurenine, an intermediate in tryptophan metabolism. Metabolism of kynurenine is dependent upon reduced, phosphorylated vitamin B3 as NADPH and upon kynureninase which is very sensitive to vitamin B6 function as pyridoxal 5-phosphate. Usually, elevated kynurenic acid means that vitamin B6 is functionally deficient. This impacts the body's ability to form nicotinic acid (vitamin B3) and picolinic acid, both of which are eventual metabolites of kynurenine and tryptophan. Severe vitamin B3 deficiency results in pellagra. Vitamin B6 deficiency symptoms are consistent with elevated kynurenic acid: fatigue, irritability, GI distress, neuritis and neuropathy.

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

Elevated FIGlu can be coincident with homocystinuria and predisposition to cardiovascular disease. In children,

Commentary

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.

Isovalerylglycine (IVG) is a product of leucine catabolism, and has been observed to be elevated in the urine with increased leucine intake, anorexia nervosa or an enzyme defect. There are numerous variants of the enzyme errors, some of which respond to combined treatment with carnitine and glycine by increasing the excretion (detoxification) of the IVG. There are additional cases where riboflavin appears to be an important way to correct the metabolic difficulties. High levels could be due to high leucine intake, dramatic dietary issues such as anorexia or potential enzyme defect, which are often accompanied by significant muscular symptoms such as weakness or hypotonia. High levels of IVG benefit therapeutically from carnitine, glycine, and riboflavin.

Orotic Acid is elevated. Orotic acid is an amine, ring-structured organic acid formed from aspartic acid and carbamoyl phosphate. Carbamoyl phosphate is also the metabolite that brings waste nitrogen into the urea cycle for detoxication and disposal. Orotic acid combines with ribosyl phosphate to produce the pyrimidine nucleotides uridine, thymidine and cytidine. Elevated orotic acid and orotic aciduria can have several possible causes.

Alcoholism can cause a moderate increase of urine orotic acid if liver damage has occurred. Also barbiturates may cause pronounced elevations in orotic acid (barbituric acid inhibits the enzyme orotate phosphoribosyl transferase). The nutritional deficits that might limit pyrimidine synthesis from orotic acid could be a cause of mild/moderate orotic aciduria. These include magnesium, glutamine and the nutrients needed for methylene tetrahydrofolate: either folic acid, vitamin B6, and serine; or folic acid, vitamins B3 and B6, and glycine.

Urea cycle dysfunction with weak activity of arginase or other enzymes (uncommon) can increase orotic acid as compensation; orotic aciduria then contributes to nitrogen disposal. Lysinuric protein intolerance also features orotic aciduria. In this condition, lysine is markedly elevated and interferes with arginase.) These urea-cycle related orotic acidurias are uncommon. Weakness of the pyrimidine-nucleotide-synthesis enzymes can be another, uncommon reason for elevated urine orotic acid, as can purine enzyme disorders (nucleoside phosphorylase). Allopurinol, a drug used for gout conditions, and "6azur" (6-azuridine) and "5FU" (5-fluorouracil), used for chemotherapy in cancer, result in orotic aciduria.

3-OH isovaleric acid is elevated. A high level of 3-hydroxyisovaleric acid (3-HIVA) is a dependable indicator of biotin deficiency. 3-OH-isovaleric acid may also be elevated in smokers and those on long-term therapy with phenytoin or carbemazapine. Chronic intake of raw egg whites is an unusual cause of biotin deficiency as the uncooked egg whites contain a compound, avidin, which interferes with biotin utilization. Common symptoms of insufficient biotin levels include: alopecia, dermatitis and hypotonia, and in severe manifestation of this deficiency, even seizures and ataxia. Most individuals with elevated 3-OH-isovaleric acid related to biotin metabolism respond well to biotin supplementation.

Commentary

<dl = Unable to calculate results due to less than detectable levels of analyte.
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.**

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.

Beta-aminoisobutyric acid (B-AIB) is a product of catabolism of pyrimidine nucleotides and it is an intermediate of valine-to-succinic acid metabolism. In valine-to-succinic acid metabolism, B-AIB is directly formed from methylmalonic acid semialdehyde. B-AIB is elevated for this individual which implies one of four possible conditions.

1. Vitamin B12 coenzyme function (as adenosylcobalamin) is weak. Elevated methylmalonic acid in urine (methylmalonic aciduria) would confirm this. Vitamin B12 deficiency or adenosylcobalamin coenzyme defect would be causative.
2. Vitamin B6 coenzyme function (as pyridoxal phosphate) is weak. B-AIB also transaminates to its keto analog.
3. The specific B-AIB-to-pyruvic acid transaminase is weak or absent. This is considered a benign variant of metabolism and is present in about 25% of Chinese and Japanese individuals and in about 8% of Scandinavian and Northwestern Europeans.

Commentary

4. Accelerated catabolism of DNA and RNA is occurring. Catabolism of damaged or diseased tissue, tumors and malignancy feature increased formation and excretion of B-AIB.

In addition to the above conditions, Downs syndrome individuals usually are B-AIB excretors. It is not known whether one of the above four mechanisms is responsible.

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 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.

3-Methylhistidine is elevated. This methylated form of histidine comes from muscle tissue, both dietary and endogenous, where it is part of the muscle proteins actin and myosin. A moderate level of urine 3-methylhistidine is normal. Elevated levels suggest either an inordinately high intake of dietary actin/myosin or accelerated catabolism

Commentary

of muscle tissue in this individual. 3-Methylhistidine excretion is increased after strenuous physical exercise and also occurs in muscle wasting conditions - dystrophies, lack of exercise, extended bed rest, terminal stages of severe illness. Convulsions and seizures also feature increased urinary 3-methylhistidine. There is some correlation between elevated 3-methylhistidine and increased need for vitamin B12, folic acid, methionine and perhaps histidine if it is marginal or low.

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.

The essential amino acid **leucine** is elevated. A mild or moderate isolated elevation of leucine usually reflects insufficient vitamin B6 or pyridoxal 5-phosphate. Metabolic disorders of leucine are expected to involve the similarly structured essential amino acids, isoleucine and valine, and a urine leucine level above 200 micromoles per 24 hours would be expected. The other branched-chain essential amino acids are not elevated.

Glutamic acid is measured to be elevated in urine. Dicarboxylic hyperaminoaciduria is not present. The known conditions consistent with isolated glutamic aciduria are as follows.

(1) Ingestion of excessive levels of monosodium glutamate "MSG"
(2) Ingestion of nutritional supplements containing large amounts of glutamic acid
(3) Gout or pregout, check blood/urine uric acid levels
(4) Some imbalance or impairment in purine metabolism
(5) Metabolic or renal acidosis

Conditions (1) and (2) are expected to normalize soon after the dietary source is discontinued. Conditions (3) and (4) are often best corrected by a low purine diet. Purine metabolism disorders are uncommon and differential diagnosis is difficult. In metabolic or renal acidosis, glutaminase in the kidneys forms glutamic acid and ammonia which becomes basic ammonium hydroxide. This is a normal pH balancing mechanism for compensating acidosis. Acidoses can feature increased serum anion gap (ketoacidoses of diabetes or alcoholism or lactic acidoses of respiratory insufficiency, chemical toxicity, circulatory problems, etc.) or may be hyperchloremic with normal anion gap (renal tubular acidosis, hypoaldosteronism, alkali-loss diarrhea).

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

Linoleic acid (LA) is below the reference range. LA is found in large quantities in virtually all vegetable oils (corn, peanut, soy, sunflower, safflower, canola, etc.). Given the large quantities of vegetable oil in the typical western diet, LA is usually seen only in people on a fat-free or severely fat-restricted diet. LA is the precursor essential fat for GLA, DGLA and arachidonic acid. Other dietary sources of LA include avocados, nuts, and seeds.

Linoleic acid stimulates normal cellular division and cellular repair. Inadequate LA may result in eczema-like skin eruptions, behavioral disturbances, increased thirst, growth retardation, and impaired wound healing.

Dihomo Gamma Linolenic Acid (DGLA) is below the reference 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

Commentary

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 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.

Pentadecanoic acid and/or Tricosanoic acid are above the reference 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.

The linoleic/DGLA ratio is high which may indicate impaired delta-6 desaturase enzyme activity. Impaired delta-6 activity would be confirmed if the eicosadienoic/DGLA ratio were also high since eicosadienoic acid represents the elongation of linoleic acid before it has undergone desaturation. However, a high linoleic/DGLA ratio, alone, is sufficient to suggest impaired delta-6 activity. Typical remedies include supplementing with vitamins B2, B3, B6, C, and the minerals zinc and magnesium. The enzyme may be bypassed by supplementing oils containing pre-formed GLA oils such as evening primrose, borage, or black currant seed oil.

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.

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.

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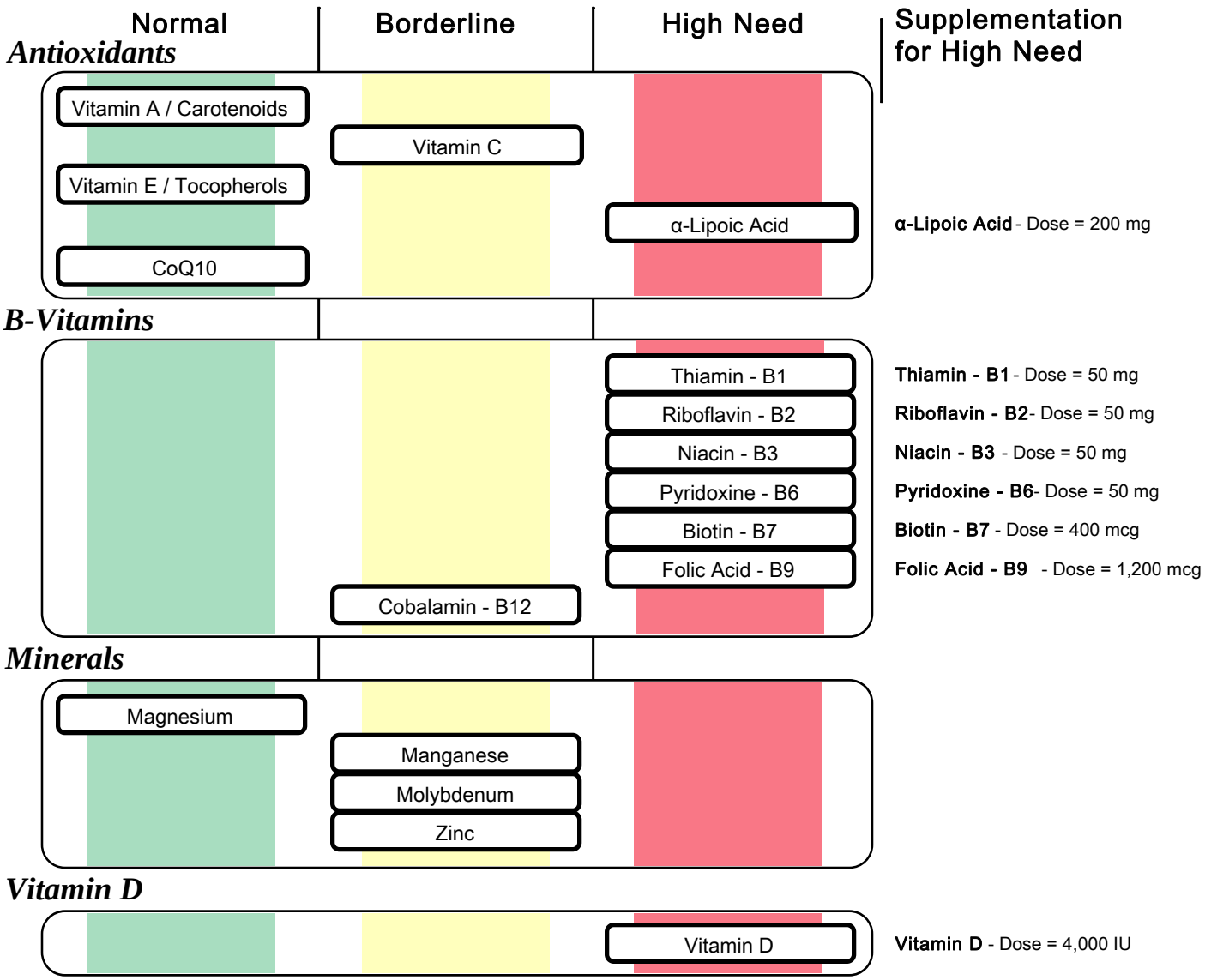
NutrEval Results Overview



SUGGESTED SUPPLEMENT SCHEDULE

Supplements	Daily Recommended Intake (DRI)	Patient's Daily Recommendations	Provider Daily Recommendations
Antioxidants			
Vitamin A / Carotenoids	2,333 IU	3,000 IU	
Vitamin C	75 mg	500 mg	
Vitamin E / Tocopherols	22 IU	100 IU	
α-Lipoic Acid		200 mg	
CoQ10		30 mg	
B-Vitamins			
Thiamin - B1	1.1 mg	50 mg	
Riboflavin - B2	1.1 mg	50 mg	
Niacin - B3	14 mg	50 mg	
Pyridoxine - B6	1.3 mg	50 mg	
Biotin - B7	30 mcg	400 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	5.0 mg	
Molybdenum	45 mcg	150 mcg	
Zinc	8 mg	20 mg	
Essential Fatty Acids			
Omega-3 Oils	500 mg	500 mg	
Digestive Support			
Probiotics		25 billion CFU	
Pancreatic Enzymes		5,000 IU	
Other Vitamins			
Vitamin D	600 IU	4,000 IU	

Amino Acid	mg/day	Amino Acid	mg/day
Arginine	186	Methionine	0
Asparagine	0	Phenylalanine	0
Cysteine	0	Serine	0
Glutamine	22	Taurine	0
Glycine	0	Threonine	36
Histidine	0	Tryptophan	0
Isoleucine	0	Tyrosine	0
Leucine	0	Valine	0
Lysine	362		

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 FMV amino acids 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.

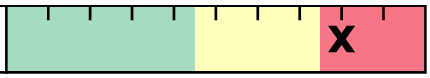
Vitamin C

- 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.

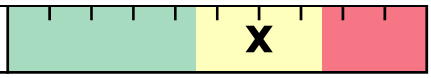
CoQ10

- 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

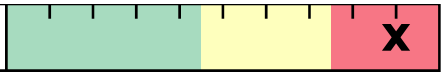
NutrEval FMV amino acids

Interpretation At-A-Glance

Nutritional Needs

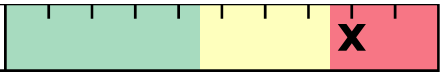
B-Vitamins

Thiamin - B1



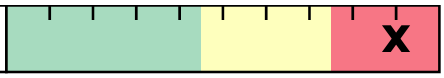
- B1 is a required cofactor for enzymes involved in energy production from food, and for the synthesis of ATP, GTP, DNA, RNA and NADPH.
- Low B1 can result from chronic alcoholism, diuretics, digoxin, oral contraceptives and HRT, or large amounts of tea & coffee (contain anti-B1 factors).
- B1 deficiency 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



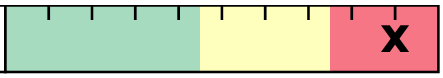
- B6 (as P5P) is a cofactor for enzymes involved in glycogenolysis & gluconeogenesis, and synthesis of neurotransmitters, heme, B3, RBCs and nucleic acids.
- Low B6 may result from chronic alcoholism, long-term diuretics, estrogens (oral contraceptives and HRT), anti-TB meds, penicillamine, L-DOPA or 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

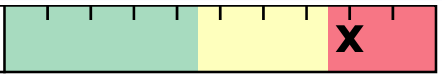


- Biotin is a cofactor for enzymes involved in functions such as fatty acid synthesis, mitochondrial FA oxidation, gluconeogenesis and DNA replication & transcription.
- Deficiency may result from certain inborn errors, chronic intake of raw egg whites, long-term TPN, anticonvulsants, high-dose B5, sulfa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

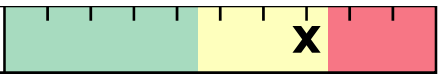
- B3 is used to form NAD and NADP, involved in energy production from food, fatty acid & cholesterol synthesis, cell signaling, DNA repair & cell differentiation.
- Low B3 may result from deficiencies of tryptophan (B3 precursor), B6, B2 or Fe (cofactors in B3 production), or from long-term isoniazid 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



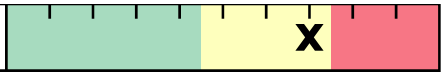
- B12 plays important roles in energy production from fats & proteins, methylation, synthesis of hemoglobin & RBCs, and maintenance of nerve cells, DNA & RNA.
- Low B12 may result from alcoholism, malabsorption, hypochlorhydria (e.g., from atrophic gastritis, H. pylori infection, pernicious anemia, H2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

NutrEval FMV amino acids Interpretation At-A-Glance

Nutritional Needs

Minerals

Manganese

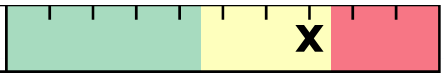


- Manganese plays an important role in antioxidant function, gluconeogenesis, the urea cycle, cartilage & bone formation, energy production and digestion.
- Impaired absorption of Mn may occur with excess intake of Fe, Ca, Cu, folic acid, or phosphorous compounds, or use of long-term TPN, Mg-containing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

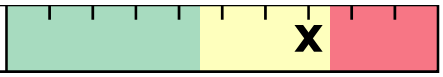
- Magnesium is involved in >300 metabolic reactions. Key areas include energy production, bone & ATP formation, muscle & nerve conduction and cell signaling.
- Deficiency may occur with malabsorption, alcoholism, hyperparathyroidism, renal disorders (wasting), diabetes, diuretics, digoxin or high doses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

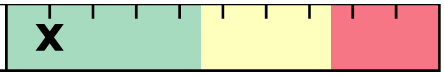
Zinc



- 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

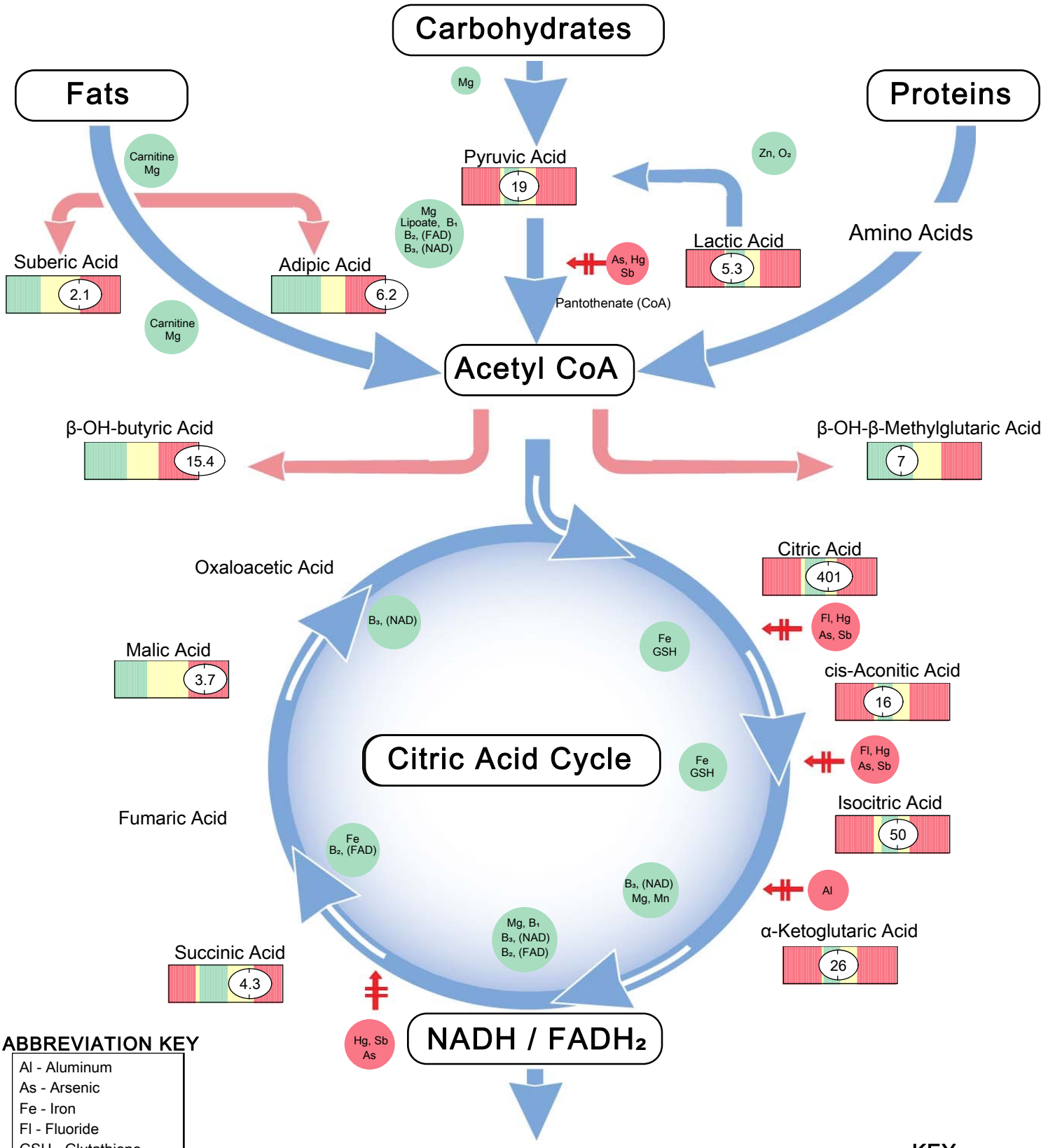
Need for Probiotics

- 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.

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 (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)	1.5	<= 5.3
3-Hydroxyphenylacetic Acid	6.5	<= 8.1
4-Hydroxyphenylacetic Acid	15	<= 29
Benzoic Acid	0.14	<= 0.05
Hippuric Acid	<dl	<= 603

Yeast / Fungal Dysbiosis Markers

		Reference Range
Arabinose	80	<= 96
Citramalic Acid	2.5	<= 5.8
Tartaric Acid	<dl	<= 15

Cellular Energy & Mitochondrial Metabolites

Carbohydrate Metabolism

		Reference Range
Lactic Acid	5.3	1.9-19.8
Pyruvic Acid	19	7-32
β-OH-Butyric Acid (BHBA)	15.4	<= 2.8

Energy Metabolism

		Reference Range
Citric Acid	401	40-520
Cis-Aconitic Acid	16	10-36
Isocitric Acid	50	22-65
α-Ketoglutaric Acid (AKG)	26	4-52
Succinic Acid	4.3	0.4-4.6
Malic Acid	3.7	<= 3.0
β-OH-β-Methylglutaric Acid (HMG)	7	<= 15

Fatty Acid Metabolism

		Reference Range
Adipic Acid	6.2	<= 2.8
Suberic Acid	2.1	<= 2.1

Creatinine Concentration

		Reference Range
Creatinine ◆	10.9	3.1-19.5 mmol/L

Neurotransmitter Metabolites

		Reference Range
Vanilmandelic Acid	1.4	0.4-3.6
Homovanilic Acid	1.7	1.2-5.3
5-OH-indoleacetic Acid	12.4	3.8-12.1
3-Methyl-4-OH-phenylglycol	0.08	0.02-0.22
Kynurenic Acid	10.4	<= 7.1
Quinolinic Acid	4.3	<= 9.1
Kynurenic / Quinolinic Ratio	2.42	>= 0.44

Vitamin Markers

		Reference Range
α-Ketoadipic Acid	1.1	<= 1.7
α-Ketoisovaleric Acid	0.85	<= 0.97
α-Ketoisocaproic Acid	0.95	<= 0.89
α-Keto-β-Methylvaleric Acid	2.5	<= 2.1
Formiminoglutamic Acid (FIGlu)	2.2	<= 1.5
Glutaric Acid	0.50	<= 0.51
Isovalerylglycine	3.8	<= 3.7
Methylmalonic Acid	1.0	<= 1.9
Xanthurenic Acid	0.96	<= 0.96
3-Hydroxypropionic Acid	13	5-22
3-Hydroxyisovaleric Acid	33	<= 29

Toxin & Detoxification Markers

		Reference Range
α-Ketophenylacetic Acid (from Styrene)	0.29	<= 0.46
α-Hydroxyisobutyric Acid (from MTBE)	5.4	<= 6.7
Orotic Acid	1.23	0.33-1.01
Pyroglutamic Acid	29	16-34

Tyrosine Metabolism

		Reference Range
Homogentisic Acid	5	<= 19
2-Hydroxyphenylacetic Acid	0.68	<= 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 ◆, the assays have not been cleared by the U.S. Food and Drug Administration.

Amino Acids (Urine FMV)

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

Nutritionally Essential Amino Acids

Amino Acid		Reference Range
Arginine	16	10-64
Histidine	682	296-1,136
Isoleucine	45	24-58
Leucine	90	30-87
Lysine	70	45-286
Methionine	51	30-82
Phenylalanine	52	26-71
Taurine	1,604	68-538
Threonine	92	65-252
Tryptophan	77	28-111
Valine	49	23-61

Nonessential Protein Amino Acids

Amino Acid		Reference Range
Alanine	118	146-486
Asparagine	86	49-182
Aspartic Acid	66	35-86
Cysteine	86	21-78
Cystine	29	26-78
γ-Aminobutyric Acid	2	<= 31
Glutamic Acid	23	5-21
Glutamine	234	172-570
Proline	11	2-18
Tyrosine	71	33-124

Creatinine Concentration

		Reference Range
Creatinine ◆	10.7	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.

Intermediary Metabolites

B Vitamin Markers		Reference Range
α-Aminoadipic Acid	108	11-73
α-Amino-N-butyric Acid	11	9-49
β-Aminoisobutyric Acid	304	22-192
Cystathionine	<dl	6-33
3-Methylhistidine	375	131-318

Urea Cycle Markers		
Ammonia	40.1	14.0-49.0 mmol/g creatinine
Citrulline	44	12-45
Ornithine	9	4-21
Urea ◆	440	168-465 mmol/g creatinine

Glycine/Serine Metabolites		
Glycine	1,109	639-3,306
Serine	279	187-568
Ethanolamine	321	208-514
Phosphoethanolamine	27	18-70
Phosphoserine	32	28-63
Sarcosine	47	<= 48

Dietary Peptide Related Markers

		Reference Range
Anserine (dipeptide)	44	7-126
Carnosine (dipeptide)	61	10-104
1-Methylhistidine	1,599	92-1,046
β-Alanine	7	<= 21

Markers for Urine Representativeness

		Reference Range
Glutamine/Glutamate	10	>= 10
Ammonia	40.1	14.0-49.0 mmol/g creatinine
Arginine/Ornithine	1.8	>= 1.1

Urine Representativeness Index	10	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	0.13	>= 0.09 wt %
Eicosapentaenoic (EPA) 20:5 n3	4.52	>= 0.16 wt %
Docosapentaenoic (DPA) 22:5 n3	2.45	>= 1.14 wt %
Docosahexaenoic (DHA) 22:6 n3	8.0	>= 2.1 wt %
% Omega 3s	15.1	>= 3.8

Omega 9 Fatty Acids

Analyte	(olive oil)	Reference Range
Oleic 18:1 n9	12	10-13 wt %
Nervonic 24:1 n9	3.7	2.1-3.5 wt %
% Omega 9s	15.6	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	16	14-17 wt %
Arachidic C20:0	0.23	0.22-0.35 wt %
Behenic C22:0	0.95	0.92-1.68 wt %
Tricosanoic C23:0	0.21	0.12-0.18 wt %
Lignoceric C24:0	2.2	2.1-3.8 wt %
Pentadecanoic C15:0	0.17	0.07-0.15 wt %
Margaric C17:0	0.36	0.22-0.37 wt %
% Saturated Fats	40.3	39.8-43.6

Omega 6 Fatty Acids

Analyte	(vegetable oil, grains, most meats, dairy)	Reference Range
Linoleic (LA) 18:2 n6	8.9	10.5-16.9 wt %
γ-Linolenic (GLA) 18:3 n6	0.04	0.03-0.13 wt %
Dihomo-γ-linolenic (DGLA) 20:3 n6	0.70	>= 1.19 wt %
Arachidonic (AA) 20:4 n6	17	15-21 wt %
Docosatetraenoic (DTA) 22:4 n6	1.11	1.50-4.20 wt %
Eicosadienoic 20:2 n6	0.18	<= 0.26 wt %
% Omega 6s	27.7	30.5-39.7

Monounsaturated Fats

Omega 7 Fats		Reference Range
Palmitoleic 16:1 n7	0.27	<= 0.64 wt %
Vaccenic 18:1 n7	0.73	<= 1.13 wt %
Trans Fat		
Elaidic 18:1 n9t	0.34	<= 0.59 wt %

Delta - 6 Desaturase Activity

	Upregulated Functional Impaired	
Linoleic / DGLA 18:2 n6 / 20:3 n6	12.8	6.0-12.3

Cardiovascular Risk

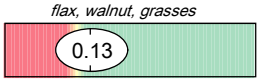
Analyte		Reference Range
Omega 6s / Omega 3s	1.8	3.4-10.7
AA / EPA 20:4 n6 / 20:5 n3	4	12-125
Omega 3 Index	12.5	>= 4.0

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

Essential Fatty Acid Metabolism

Omega 3 Family

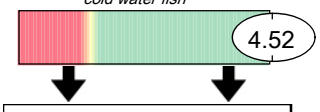
α-Linolenic Acid
flax, walnut, grasses



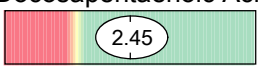
Stearidonic acid

Eicosatetraenoic acid, ETA

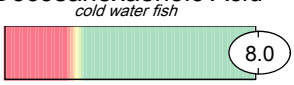
Eicosapentaenoic Acid
cold water fish



Docosapentaenoic Acid



Docosahexaenoic Acid
cold water fish



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

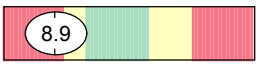
Delta-5 Desaturase
Vitamin and Mineral Cofactors:
FAD (B2), Niacin (B3)
Pyridoxal-5-phosphate (B6)
Vitamin C, Insulin, Zn, Mg

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

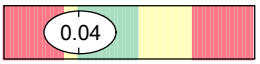
Elongase
Delta-6 Desaturase
Vitamin and Mineral Cofactors:
FAD (B2), Niacin (B3)
Pyridoxal-5-phosphate (B6), Biotin
Vitamin C, Zn, Mg, Carnitine
Pantothenic Acid (B5)

Omega 6 Family

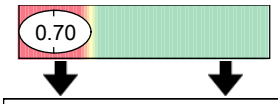
Linoleic Acid
grains, vegetable oils



γ-Linolenic Acid
evening primrose, borage, black currant



Dihomo-γ-Linolenic Acid



Arachidonic Acid
animal fats

Docosatetraenoic Acid

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
Glutathione (whole blood)	705	>=669 micromol/L
Lipid Peroxides (urine)	6.9	<=10.0 micromol/g Creat.
8-OHdG (urine)	9	<=16 mcg/g Creat.
Coenzyme Q10, Ubiquinone (plasma)	0.73	0.43-1.49 mcg/mL

The Oxidative Stress reference ranges are based on an adult population.

Vitamin D (Serum)

	Inside Range	Outside Range	Reference Range
25 - OH Vitamin D ◆		24	50-100 ng/mL

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 (RBCs)

Nutrient Elements

Element	Reference Range	Reference Range
Copper	0.495	0.466-0.721 mcg/g
Magnesium	53.3	30.1-56.5 mcg/g
Manganese	0.011	0.007-0.038 mcg/g
Potassium	3,019	2,220-3,626 mcg/g
Selenium	0.49	0.25-0.76 mcg/g
Zinc	8.6	7.8-13.1 mcg/g

The Elemental reference ranges are based on an adult population.

Toxic Elements

Element	Reference Range	Reference Range
Lead	0.034	<= 0.048 mcg/g
Mercury	0.0113	<= 0.0039 mcg/g
Antimony	0.001	<= 0.002 mcg/g
Arsenic	0.029	<= 0.071 mcg/g
Cadmium	<dl	<= 0.001 mcg/g
Tin	<dl	<= 0.0009 mcg/g

Lab Comments

Lab Comments

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.

Homocysteine (Plasma)

GENOVA DIAGNOSTICS EUROPE

Parkgate House
356 West Barnes Lane
New Malden, Surrey KT3 6NB

63 Zillicoa Street
Asheville, NC 28801 USA

Homocysteine

	Inside Range	Outside Range	Reference Range
Homocysteine		3.00	3.70-10.40 umol/L

Commentary

Lab Comments

The reference range for homocysteine is based on the sex-specific 5th to 95th percentile values for men and women (20 to 39 years of age) in the NHANES nutritionally replete cohort. Annals of Internal Medicince 1999; 131 (331-338).

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.

Homocysteine is BELOW the REFERENCE level for this person. Low homocysteine translates into diminished cardiovascular risk and is an important BENEFICIAL finding for this person. Positive genetic, nutritional and lifestyle factors are likely to be responsible for this favorable finding.