



**Genova**  
**Diagnostics**  
Europe

Improving Healthcare for Chronic Disease

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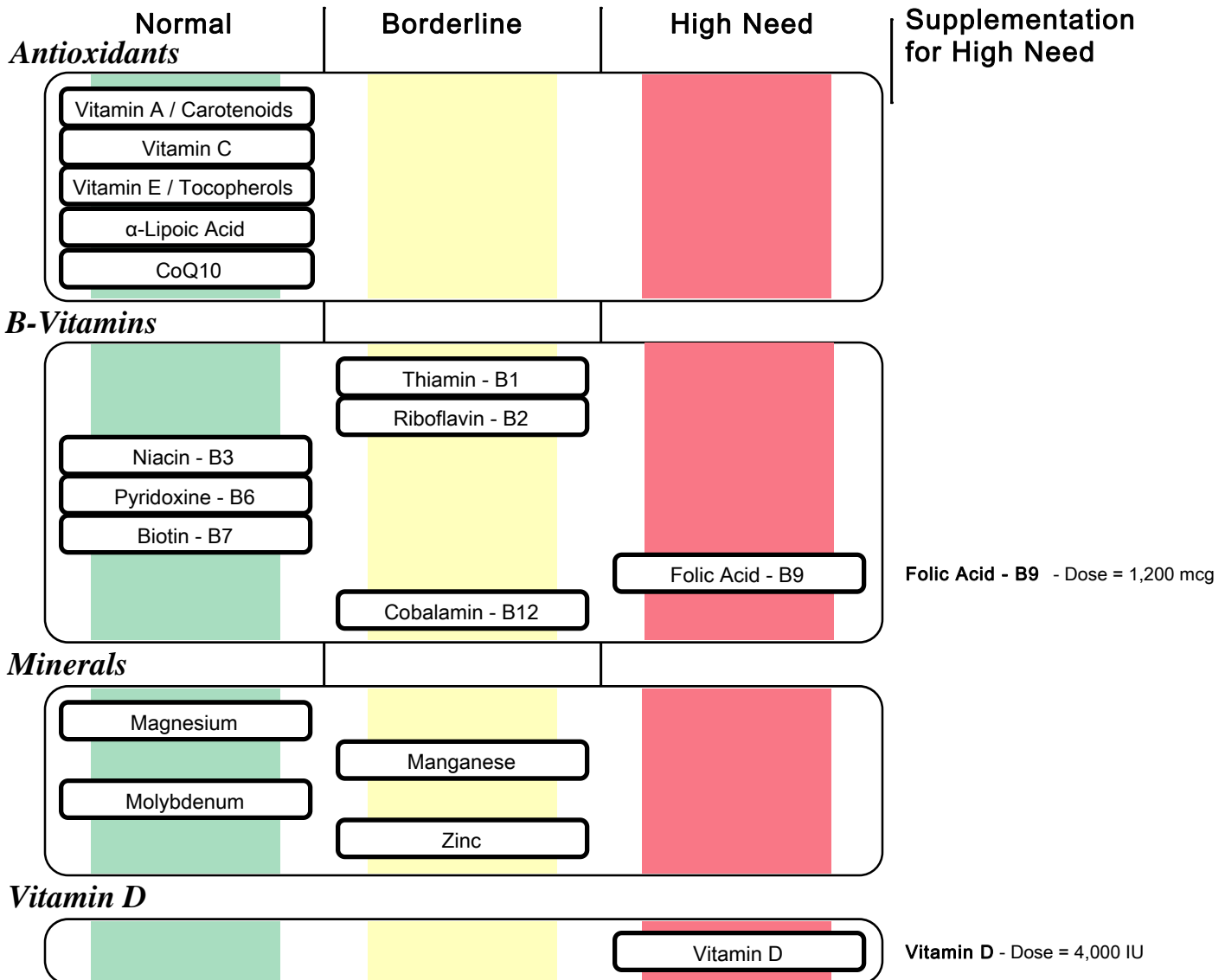
Patient: **PAUL**  
**BARDIN**  
DOB: January 27, 1975  
Sex: M  
MRN: 0001757026

**Order Number: F4260192**

Completed: November 06, 2012  
Received: October 26, 2012  
Collected: October 25, 2012  
Route Number: A155652

Rita Arora  
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London, SW7 3HZ  
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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	3,000 IU	3,000 IU	
Vitamin C	90 mg	250 mg	
Vitamin E / Tocopherols	22 IU	100 IU	
α-Lipoic Acid		50 mg	
CoQ10		30 mg	
B-Vitamins			
Thiamin - B1	1.2 mg	25 mg	
Riboflavin - B2	1.3 mg	25 mg	
Niacin - B3	16 mg	20 mg	
Pyridoxine - B6	1.3 mg	10 mg	
Biotin - B7	30 mcg	100 mcg	
Folic Acid - B9	400 mcg	1,200 mcg	
Cobalamin - B12	2.4 mcg	500 mcg	
Minerals			
Magnesium	420 mg	400 mg	
Manganese	2.3 mg	5.0 mg	
Molybdenum	45 mcg	75 mcg	
Zinc	11 mg	20 mg	
Essential Fatty Acids			
Omega-3 Oils	500 mg	500 mg	
Digestive Support			
Probiotics		10 billion CFU	
Pancreatic Enzymes		5,000 IU	
Other Vitamins			
Vitamin D	600 IU	4,000 IU	
Amino Acid		mg/day	
Arginine	655	Methionine	38
Asparagine	0	Phenylalanine	0
Cysteine	0	Serine	322
Glutamine	70	Taurine	0
Glycine	1,675	Threonine	0
Histidine	90	Tryptophan	0
Isoleucine	0	Tyrosine	253
Leucine	0	Valine	356
Lysine	1,164		

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

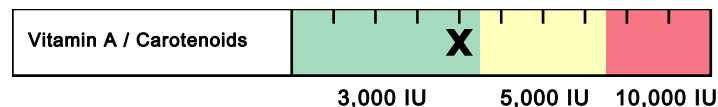
High Need



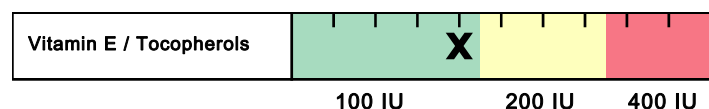
## Interpretation At-A-Glance

### Nutritional Needs

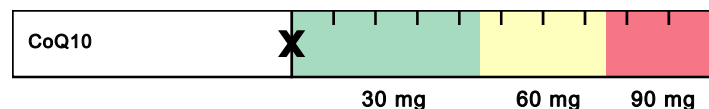
#### Antioxidants



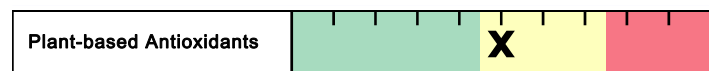
- ▶ Beta-carotene & other carotenoids are converted to vitamin A (retinol), involved in vision, antioxidant & immune function, gene expression & cell growth.
- ▶ Vitamin A deficiency may occur with chronic alcoholism, zinc deficiency, hypothyroidism, or oral contraceptives containing estrogen & progestin.
- ▶ 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.



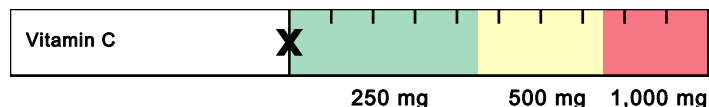
- ▶ Alpha-tocopherol (body's main form of vitamin E) functions as an antioxidant, regulates cell signaling, influences immune function and inhibits coagulation.
- ▶ Deficiency may occur with malabsorption, cholestyramine, colestipol, isoniazid, orlistat, olestra and certain anti-convulsants (e.g., phenobarbital,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



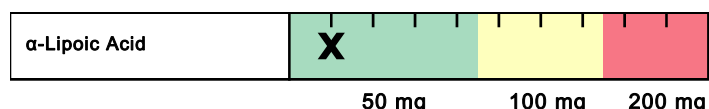
- ▶ CoQ10 is a powerful antioxidant that is synthesized in the body and contained in cell membranes. CoQ10 is also essential for energy production & pH regulation.
- ▶ CoQ10 deficiency may occur with HMG-CoA reductase inhibitors (statins), several anti-diabetic medication classes (biguanides, sulfonylureas)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



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



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



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



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

#### Key

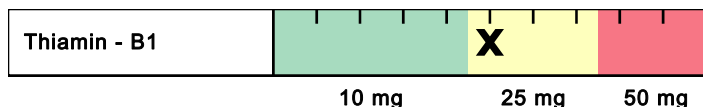
- ▶ Function
- ▶ Causes of Deficiency
- ▶ Complications of Deficiency
- ▶ Food Sources



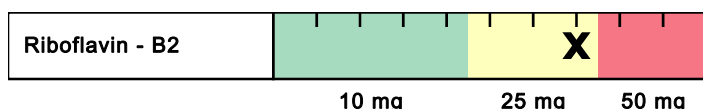
## Interpretation At-A-Glance

### Nutritional Needs

#### B-Vitamins



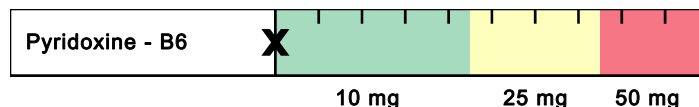
- ▶ B1 is a required cofactor for enzymes involved in energy production from food, and for the synthesis of ATP, GTP, DNA, RNA and NADPH.
- ▶ Low B1 can result from chronic alcoholism, diuretics, digoxin, oral contraceptives and HRT, or large amounts of tea & coffee (contain anti-B1 factors).
- ▶ B1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



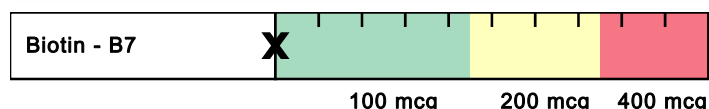
- ▶ B2 is a key component of enzymes involved in antioxidant function, energy production, detoxification, methionine metabolism and vitamin activation.
- ▶ Low B2 may result from chronic alcoholism, some anti-psychotic medications, oral contraceptives, tricyclic antidepressants, quinacrine or adriamycin.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



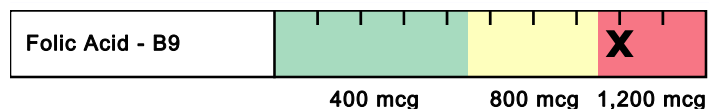
- ▶ B3 is used to form NAD and NADP, involved in energy production from food, fatty acid & cholesterol synthesis, cell signaling, DNA repair & cell differentiation.
- ▶ Low B3 may result from deficiencies of tryptophan (B3 precursor), B6, B2 or Fe (cofactors in B3 production), or from long-term isoniazid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



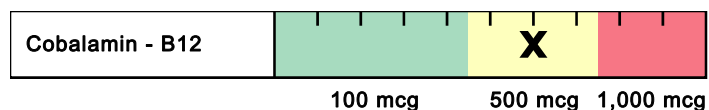
- ▶ B6 (as P5P) is a cofactor for enzymes involved in glycogenolysis & gluconeogenesis, and synthesis of neurotransmitters, heme, B3, RBCs and nucleic acids.
- ▶ Low B6 may result from chronic alcoholism, long-term diuretics, estrogens (oral contraceptives and HRT), anti-TB meds, penicillamine, L-DOPA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



- ▶ 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.



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



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



## Interpretation At-A-Glance

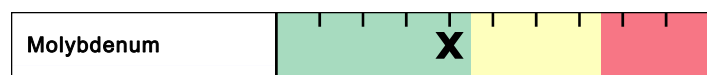
### Nutritional Needs

#### Minerals



3.0 mg 5.0 mg 7.0 mg

- ▶ 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.



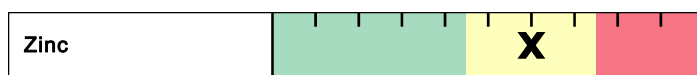
75 mcg 150 mcg 300 mcg

- ▶ 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).



400 mg 600 mg 800 mg

- ▶ 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.



10 mg 20 mg 30 mg

- ▶ 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



500 mg 1,000 mg 2,000 mg

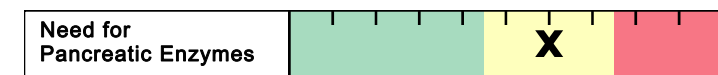
- ▶ Omega-3 (O3) and Omega-6 (O6) fatty acids are polyunsaturated fatty acids that cannot be synthesized by the human body. They are classified as essential nutrients and must be obtained from dietary sources.
- ▶ The standard American diet is much higher in O6 than O3 fatty acids. Deficiency of EFAs 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  $\alpha$ -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



10 B CFU 25 B CFU 50 B CFU

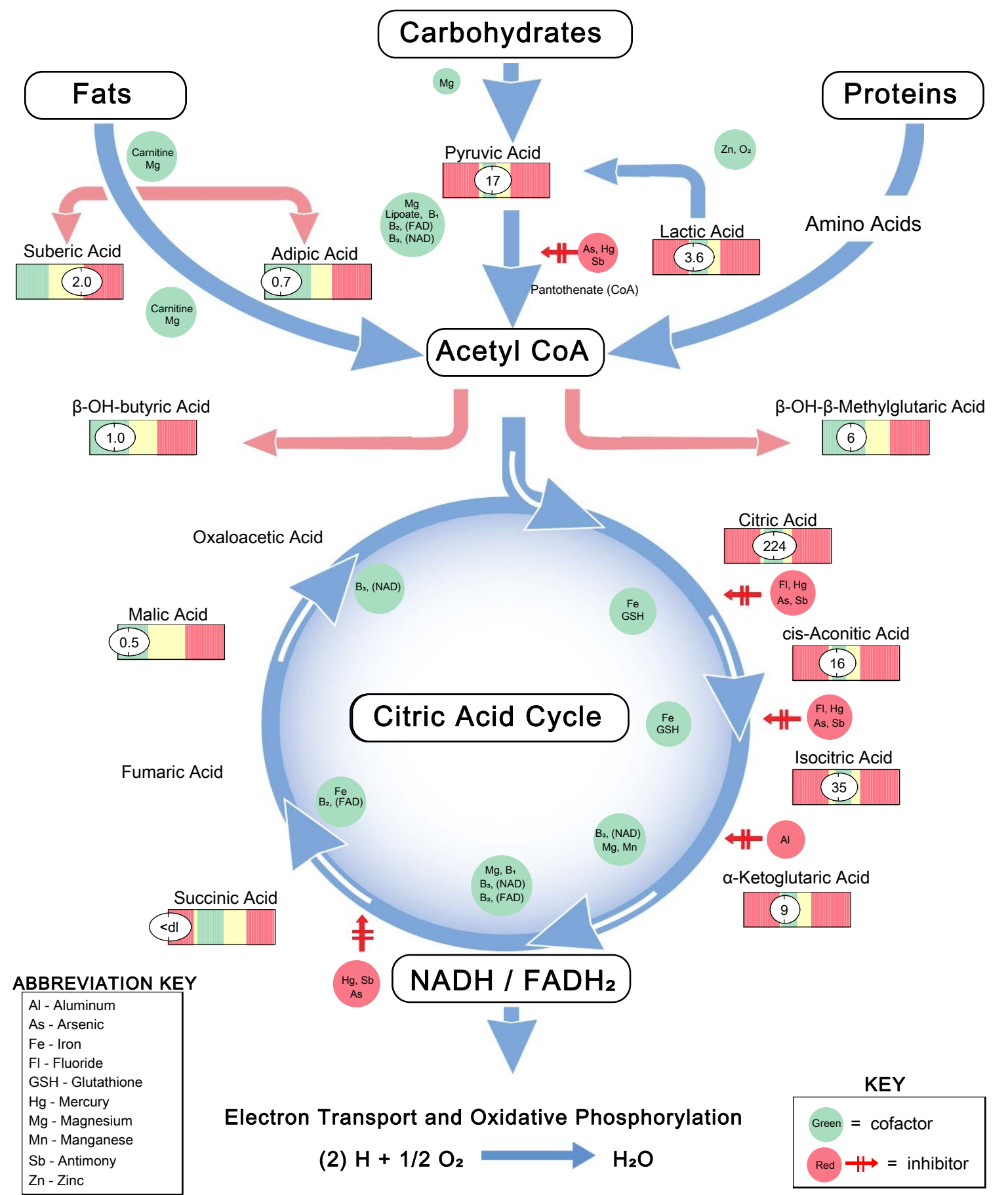
- ▶ Probiotics have many functions. These include: production of some B vitamins and vitamin K; enhance digestion & absorption; decrease severity of diarrheal illness; modulate of immune function & intestinal permeability.
- ▶ Alterations of gastrointestinal microflora may result from C-section delivery, 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.



0 IU 5,000 IU 10,000 IU

- ▶ 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





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

**Malabsorption and Dysbiosis Markers****Malabsorption Markers**

		Reference Range
Indoleacetic Acid (IAA)	0.9	<= 4.2
Phenylacetic Acid (PAA)	0.06	<= 0.12

**Bacterial Dysbiosis Markers**

Dihydroxyphenylpropionic Acid (DHPPA)	2.6	<= 5.3
3-Hydroxyphenylacetic Acid	1.5	<= 8.1
4-Hydroxyphenylacetic Acid	9	<= 29
Benzoic Acid	0.02	<= 0.05
Hippuric Acid	100	<= 603

**Yeast / Fungal Dysbiosis Markers**

Arabinose	36	<= 96
Citramalic Acid	1.4	<= 5.8
Tartaric Acid	8	<= 15

**Cellular Energy & Mitochondrial Metabolites****Carbohydrate Metabolism**

Lactic Acid	3.6	1.9-19.8
Pyruvic Acid	17	7-32
$\beta$ -OH-Butyric Acid (BHBA)	1.0	<= 2.8

**Energy Metabolism**

Citric Acid	224	40-520
Cis-Aconitic Acid	16	10-36
Isocitric Acid	35	22-65
$\alpha$ -Ketoglutaric Acid (AKG)	9	4-52
Succinic Acid	<dl	0.4-4.6
Malic Acid	0.5	<= 3.0
$\beta$ -OH- $\beta$ -Methylglutaric Acid (HMG)	6	<= 15

**Fatty Acid Metabolism**

Adipic Acid	0.7	<= 2.8
Suberic Acid	2.0	<= 2.1

**Creatinine Concentration**

		Reference Range
Creatinine ♦	17.2	3.1-19.5 mmol/L

**Neurotransmitter Metabolites****Reference Range**

Vanilmandelic Acid	0.9	0.4-3.6
Homovanillic Acid	1.8	1.2-5.3
5-OH-indoleacetic Acid	6.3	3.8-12.1
3-Methyl-4-OH-phenylglycol	0.05	0.02-0.22
Kynurenic Acid	1.4	<= 7.1
Quinolinic Acid	5.7	<= 9.1
Kynurenic / Quinolinic Ratio	0.25	>= 0.44

**Vitamin Markers****Reference Range**

$\alpha$ -Ketoadipic Acid	0.3	<= 1.7
$\alpha$ -Ketoisovaleric Acid	0.39	<= 0.97
$\alpha$ -Ketoisocaproic Acid	0.41	<= 0.89
$\alpha$ -Keto- $\beta$ -Methylvaleric Acid	1.3	<= 2.1
Formiminoglutamic Acid (FIGlu)	1.6	<= 1.5
Glutaric Acid	0.38	<= 0.51
Isovalerylglycine	0.9	<= 3.7
Methylmalonic Acid	0.9	<= 1.9
Xanthurenic Acid	0.42	<= 0.96
3-Hydroxypropionic Acid	8	5-22
3-Hydroxyisovaleric Acid	14	<= 29

**Toxin & Detoxification Markers****Reference Range**

$\alpha$ -Ketophenylacetic Acid (from Styrene)	0.13	<= 0.46
$\alpha$ -Hydroxyisobutyric Acid (from MTBE)	3.6	<= 6.7
Orotic Acid	0.38	0.33-1.01
Pyroglutamic Acid	25	16-34

**Tyrosine Metabolism****Reference Range**

Homogentisic Acid	8	<= 19
2-Hydroxyphenylacetic Acid	0.39	<= 0.76

Metabolic Analysis Reference Ranges are Age Specific

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

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

*Amino Acids (FMV)***Nutritionally Essential Amino Acids**

Amino Acid	Reference Range
Arginine	11 10-64
Histidine	372 271-993
Isoleucine	27 17-52
Leucine	45 25-77
Lysine	37 34-226
Methionine	32 26-69
Phenylalanine	35 22-61
Taurine	578 80-545
Threonine	87 52-192
Tryptophan	34 23-88
Valine	19 19-53

**Nonessential Protein Amino Acids**

Amino Acid	Reference Range
Alanine	121 103-392
Asparagine	61 37-134
Aspartic Acid	55 27-74
Cysteine	38 19-70
Cystine	18 23-68
γ-Aminobutyric Acid	8 ≤ 23
Glutamic Acid	12 3-15
Glutamine	202 153-483
Proline	3 2-14
Tyrosine	35 28-113

**Creatinine Concentration**

	Reference Range
Creatinine ♦	16.3 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 ♦ as cleared by the U.S. Food and Drug Administration, assays are For Research Use Only.

**Intermediary Metabolites**

B Vitamin Markers	Reference Range
α-Aminoadipic Acid	58 11-73
α-Amino-N-butyric Acid	25 9-49
β-Aminoisobutyric Acid	86 19-163
Cystathionine	8 6-29
3-Methylhistidine	232 134-302

**Urea Cycle Markers**

Ammonia	16.4 12.0-41.0 mmol/g creatinine
Citrulline	27 9-40
Ornithine	2 3-16
Urea ♦	251 150-380 mmol/g creatinine

**Glycine/Serine Metabolites**

Glycine	450 434-1,688
Serine	160 135-426
Ethanolamine	109 156-422
Phosphoethanolamine	21 14-50
Phosphoserine	23 26-64
Sarcosine	29 ≤ 41

**Dietary Peptide Related Markers**

	Reference Range
Anserine (dipeptide)	187 8-118
Carnosine (dipeptide)	18 12-120
1-Methylhistidine	1,624 83-1,008
β-Alanine	6 ≤ 17

**Markers for Urine Representativeness**

	Reference Range
Glutamine/Glutamate	17 ≥ 12
Ammonia	16.4 12.0-41.0 mmol/g creatinine
Arginine/Ornithine	5.5 ≥ 1.0

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
$\alpha$ -Linolenic (ALA) 18:3 n3	0.27	$\geq 0.09$ wt %
Eicosapentaenoic (EPA) 20:5 n3	1.11	$\geq 0.16$ wt %
Docosapentaenoic (DPA) 22:5 n3	2.11	$\geq 1.14$ wt %
Docosahexaenoic (DHA) 22:6 n3	6.6	$\geq 2.1$ wt %
<b>% Omega 3s</b>	10.1	$\geq 3.8$

### Omega 9 Fatty Acids

Analyte	(olive oil)	Reference Range
Oleic 18:1 n9	12	10-13 wt %
Nervonic 24:1 n9	4.3	2.1-3.5 wt %
<b>% Omega 9s</b>	16.8	13.3-16.6

### Saturated Fatty Acids

Analyte	(meat, dairy, coconuts, palm oils)	Reference Range
Palmitic C16:0	18	18-23 wt %
Stearic C18:0	19	14-17 wt %
Arachidic C20:0	0.26	0.22-0.35 wt %
Behenic C22:0	1.02	0.92-1.68 wt %
Tricosanoic C23:0	0.19	0.12-0.18 wt %
Lignoceric C24:0	2.8	2.1-3.8 wt %
Pentadecanoic C15:0	0.07	0.07-0.15 wt %
Margaric C17:0	0.20	0.22-0.37 wt %
<b>% Saturated Fats</b>	40.9	39.8-43.6

### Omega 6 Fatty Acids

Analyte	(vegetable oil, grains, most meats, dairy)	Reference Range
Linoleic (LA) 18:2 n6	10.2	10.5-16.9 wt %
$\gamma$ -Linolenic (GLA) 18:3 n6	0.06	0.03-0.13 wt %
Dihomo- $\gamma$ -linolenic (DGLA) 20:3 n6	1.23	$\geq 1.19$ wt %
Arachidonic (AA) 20:4 n6	18	15-21 wt %
Docosatetraenoic (DTA) 22:4 n6	1.79	1.50-4.20 wt %
Eicosadienoic 20:2 n6	0.23	$\leq 0.26$ wt %
<b>% Omega 6s</b>	31.1	30.5-39.7

### Monounsaturated Fats

Omega 7 Fats	Reference Range
Palmitoleic 16:1 n7	0.16 $\leq 0.64$ wt %
Vaccenic 18:1 n7	0.77 $\leq 1.13$ wt %

### Trans Fat

Elaidic 18:1 n9t	0.15 $\leq 0.59$ wt %
---------------------	-----------------------

### Delta - 6 Desaturase Activity

	Upregulated	Functional	Impaired	
Linoleic / DGLA 18:2 n6 / 20:3 n6		8.3		6.0-12.3

### Cardiovascular Risk

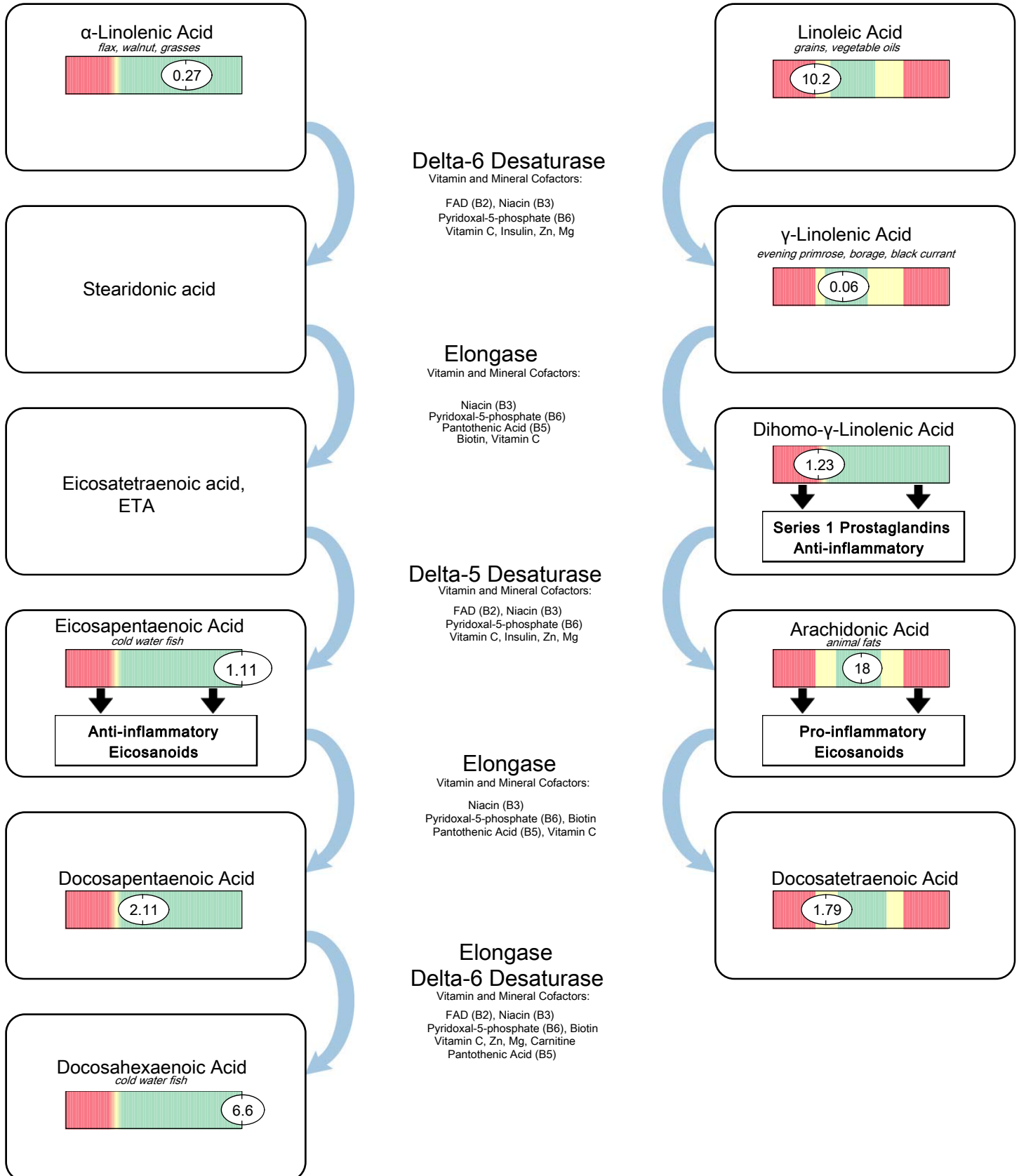
Analyte	Reference Range
Omega 6s / Omega 3s	3.1 3.4-10.7
AA / EPA 20:4 n6 / 20:5 n3	16 12-125
Omega 3 Index	7.7 $\geq 4.0$

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

## Essential Fatty Acid Metabolism

# Omega 3 Family

## Omega 6 Family



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

## Oxidative Stress Markers

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

Inside Range    Outside Range    **Reference Range**

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)

## Nutrient Elements

The Elemental reference ranges are based on an adult population.

Element	Reference Range	Reference Range
---------	-----------------	-----------------

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

## Metabolic Analysis Markers

### Commentary

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

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

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

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

**Kynurenic Acid/Quinolinic Acid ratio** is low. The downstream tryptophan metabolite quinolinic acid is generally recognized as a damaging, pro-oxidation factor due to its stimulation of NMDA receptors. Another tryptophan metabolite, Kynurenic acid, appears to protect the nervous system from this oxidative stress and neuronal damage. Thus a low KA/QA ratio is associated with inflammation, the underweight phase of anorexia and overall neurotoxicity.

## Amino Acid Markers (FMV)

### Commentary

Commentary is provided to the practitioner for educational purposes, and should not be interpreted as diagnostic or treatment recommendations. Diagnosis and treatment decisions are the responsibility of the practitioner.

#### REPRESENTATIVENESS INDEX

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

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

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

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

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

**Anserine**, a dietary dipeptide, is higher than the reference range. This peptide comes from fish, fowl and some animal protein, principally from tuna, salmon, chicken, turkey, duck and rabbit. Anserine does not occur in human organs but dietary uptake is common for non-vegetarians. Elevated anserine may result from a dietary overload of protein, may be temporary or episodic, and may have no clinical consequence. However, zinc deficiency can be a cause of peptidase weakness; also, pancreatic dysfunction or digestive disorder can result in increased uptake and excretion of anserine. Elevated anserine together with subnormal levels of essential or semiessential amino acids is consistent with incomplete digestive proteolysis and malabsorption. Further diagnostic testing to assess maldigestion should be considered.

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

**Ethanolamine**, an intermediate of the serine-to-choline metabolism sequence, is measured to be low. Ethanolamine is formed metabolically from serine and phosphatidylethanolamine; this endogenous formation is pyridoxal phosphate dependent and requires adequate serine. Consequences of ethanolamine insufficiency may be limited or insufficient levels of phosphoethanolamine, phosphatidylcholine and choline. Acetylcholine, the neurotransmitter, is formed from choline. Dietary lecithin provides an independent source of the neurotransmitter precursors. Ethanolamine insufficiency is significant if cholinergic functions are limited.

## Commentary

**Ornithine** is measured to be low. Ornithine is a non-protein-forming intermediate of the urea cycle for detoxication of nitrogen. It is a metabolic precursor of glutamic acid; and with glycine it leads to polyamines (putrescine, spermidine). Low ornithine may be of no clinical consequence and may be secondary to a low protein diet. However, low ornithine in conjunction with high arginine and with low urea could indicate urea cycle difficulties.

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

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

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

- 
- Conjugation of cholesterol (as cholyl-coenzyme A) to form taurocholic acid, an important component of bile and a major utilization of cholesterol.
- Mediation of the flux of electrolyte elements at the plasma membrane of cells. Deficient taurine may result in 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<sup>-</sup>, in leukocytes and facilitates effective phagocytosis by enhancing survival of leukocytes. Deficient taurine may lead to increased inflammatory response to: toxins, foreign proteins, and xenobiotic chemicals including aldehydes, alcohols, amines, petroleum solvents, and chlorine or chlorite (bleach).
- Neurotransmitter functions. Taurine strongly influences neuronal concentrations and activities of GABA and glutamic acid. Taurine can have anti-convulsant and anti-epileptic effects.

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



Commentary

symptomatology. 1-Methylhistidine itself is not known to be detrimental.

# *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 within the reference range, but below the functional physiologic range. DGLA is the main precursor fat for the production of highly anti-inflammatory eicosanoids, especially the series 1 prostaglandins. Low DGLA is often associated with inflammatory conditions such as heart disease, arthritis, inflammatory bowel disorders, eczema, and psoriasis. Since DGLA-derived eicosanoids also promote smooth muscle

### Commentary

relaxation, low DGLA levels may contribute to increased smooth muscle contraction, and subsequently to conditions like hypertension, asthma, painful menstruation, and irritable bowel syndrome.

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

## *Oxidative Stress Markers*

### **Commentary**

Commentary is provided to the practitioner for educational purposes, and should not be interpreted as diagnostic or treatment recommendations. Diagnosis and treatment decisions are the responsibility of the practitioner.

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

#### **Deficient or Insufficient levels:**

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

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

*Elemental Markers (RBCs)***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.

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

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

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

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

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



**Genova**  
**Diagnostics**  
Europe  
*Improving Healthcare for Chronic Disease*

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Patient: **PAUL**  
**BARDIN**  
DOB: January 27, 1975  
Sex: M  
MRN: 0001757026

**Order Number: F4260192**  
Completed: November 06, 2012  
Received: October 26, 2012  
Collected: October 25, 2012  
Route Number: A155652

Rita Arora  
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### NutrEval Results Overview

Normal	Borderline	High Need	Supplementation for High Need
<b>Antioxidants</b>			
Vitamin A / Carotenoids			
Vitamin C			
Vitamin E / Tocopherols			
α-Lipoic Acid			
CoQ10			
<b>B-Vitamins</b>			
	Thiamin - B1		
	Riboflavin - B2		
Niacin - B3			
Pyridoxine - B6			
Biotin - B7			
		Folic Acid - B9	Folic Acid - B9 - Dose = 1,200 mcg
	Cobalamin - B12		
<b>Minerals</b>			
Magnesium			
	Manganese		
Molybdenum			
	Zinc		
<b>Vitamin D</b>			
		Vitamin D	Vitamin D - Dose = 4,000 IU



SUGGESTED SUPPLEMENT SCHEDULE

Supplements	Daily Recommended Intake (DRI)	Patient's Daily Recommendations	Provider Daily Recommendations
Antioxidants			
Vitamin A / Carotenoids	3,000 IU	3,000 IU	
Vitamin C	90 mg	250 mg	
Vitamin E / Tocopherols	22 IU	100 IU	
α-Lipoic Acid		50 mg	
CoQ10		30 mg	
B-Vitamins			
Thiamin - B1	1.2 mg	25 mg	
Riboflavin - B2	1.3 mg	25 mg	
Niacin - B3	16 mg	20 mg	
Pyridoxine - B6	1.3 mg	10 mg	
Biotin - B7	30 mcg	100 mcg	
Folic Acid - B9	400 mcg	1,200 mcg	
Cobalamin - B12	2.4 mcg	500 mcg	
Minerals			
Magnesium	420 mg	400 mg	
Manganese	2.3 mg	5.0 mg	
Molybdenum	45 mcg	75 mcg	
Zinc	11 mg	20 mg	
Essential Fatty Acids			
Omega-3 Oils	500 mg	500 mg	
Digestive Support			
Probiotics		10 billion CFU	
Pancreatic Enzymes		5,000 IU	
Other Vitamins			
Vitamin D	600 IU	4,000 IU	
Amino Acid		mg/day	
Arginine	655	Methionine	38
Asparagine	0	Phenylalanine	0
Cysteine	0	Serine	322
Glutamine	70	Taurine	0
Glycine	1,675	Threonine	0
Histidine	90	Tryptophan	0
Isoleucine	0	Tyrosine	253
Leucine	0	Valine	356
Lysine	1,164		

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

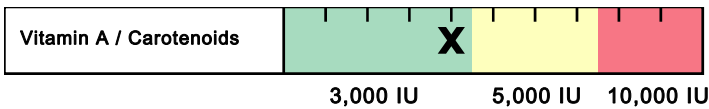
High Need



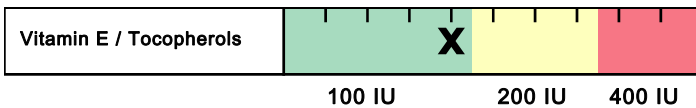
## Interpretation At-A-Glance

### Nutritional Needs

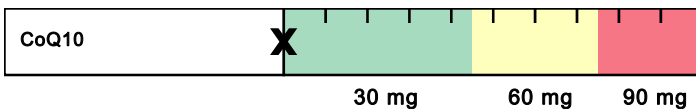
#### Antioxidants



- ▶ 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.



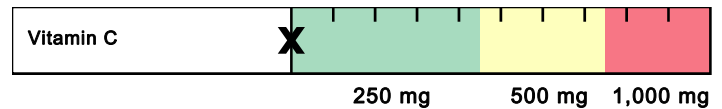
- ▶ Alpha-tocopherol (body's main form of vitamin E) functions as an antioxidant, regulates cell signaling, influences immune function and inhibits coagulation.
- ▶ Deficiency may occur with malabsorption, cholestyramine, colestipol, isoniazid, orlistat, olestra and certain anti-convulsants (e.g., phenobarbital,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



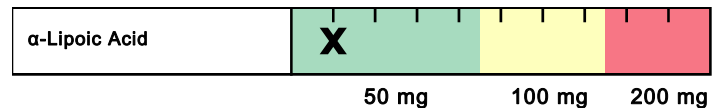
- ▶ CoQ10 is a powerful antioxidant that is synthesized in the body and contained in cell membranes. CoQ10 is also essential for energy production & pH regulation.
- ▶ CoQ10 deficiency may occur with HMG-CoA reductase inhibitors (statins), several anti-diabetic medication classes (biguanides, sulfonylureas)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



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



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



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



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

#### Key

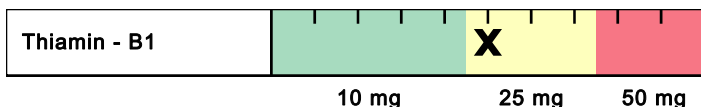
- ▶ Function
- ▶ Causes of Deficiency
- ▶ Complications of Deficiency
- ▶ Food Sources



## Interpretation At-A-Glance

### Nutritional Needs

#### B-Vitamins



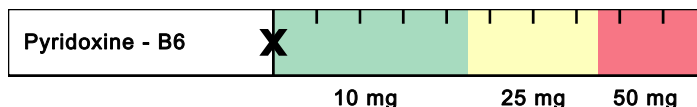
- ▶ B1 is a required cofactor for enzymes involved in energy production from food, and for the synthesis of ATP, GTP, DNA, RNA and NADPH.
- ▶ Low B1 can result from chronic alcoholism, diuretics, digoxin, oral contraceptives and HRT, or large amounts of tea & coffee (contain anti-B1 factors).
- ▶ B1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



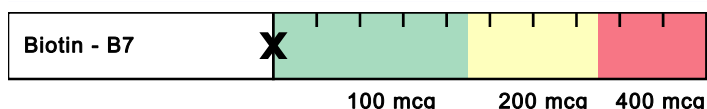
- ▶ B2 is a key component of enzymes involved in antioxidant function, energy production, detoxification, methionine metabolism and vitamin activation.
- ▶ Low B2 may result from chronic alcoholism, some anti-psychotic medications, oral contraceptives, tricyclic antidepressants, quinacrine or adriamycin.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



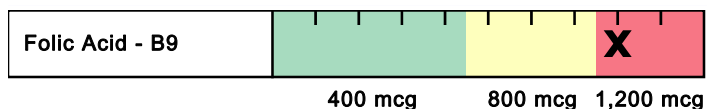
- ▶ B3 is used to form NAD and NADP, involved in energy production from food, fatty acid & cholesterol synthesis, cell signaling, DNA repair & cell differentiation.
- ▶ Low B3 may result from deficiencies of tryptophan (B3 precursor), B6, B2 or Fe (cofactors in B3 production), or from long-term isoniazid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



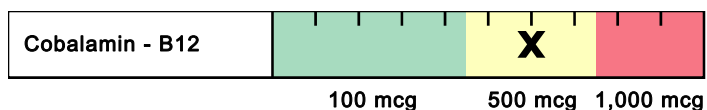
- ▶ B6 (as P5P) is a cofactor for enzymes involved in glycogenolysis & gluconeogenesis, and synthesis of neurotransmitters, heme, B3, RBCs and nucleic acids.
- ▶ Low B6 may result from chronic alcoholism, long-term diuretics, estrogens (oral contraceptives and HRT), anti-TB meds, penicillamine, L-DOPA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



- ▶ 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.



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



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



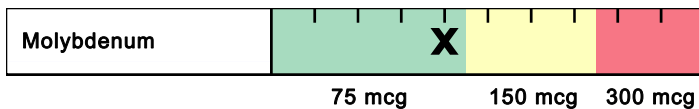
## Interpretation At-A-Glance

### Nutritional Needs

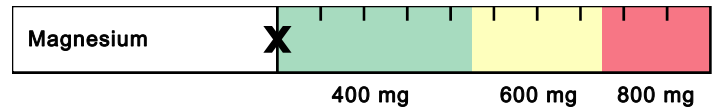
#### Minerals



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



- 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).

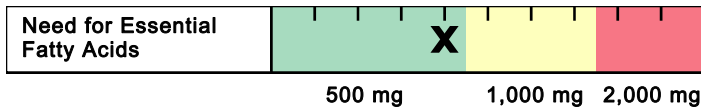


- Magnesium is involved in >300 metabolic reactions. Key areas include energy production, bone & ATP formation, muscle & nerve conduction and cell signaling.
- Deficiency may occur with malabsorption, alcoholism, hyperparathyroidism, renal disorders (wasting), diabetes, diuretics, digoxin or high doses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



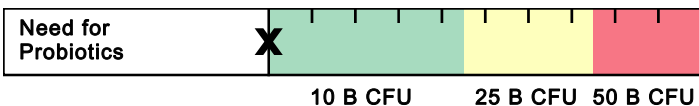
- Zinc plays a vital role in immunity, protein metabolism, heme synthesis, growth & development, reproduction, digestion and antioxidant function.
- Low levels may occur with malabsorption, alcoholism, chronic diarrhea, diabetes, excess Cu or Fe, diuretics, ACE inhibitors, H2 blockers or digoxin.
- Deficiency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

#### Essential Fatty Acids

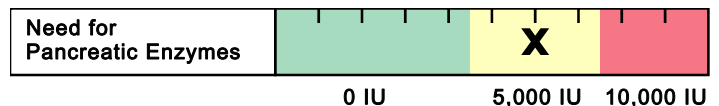


- Omega-3 (O3) and Omega-6 (O6) fatty acids are polyunsaturated fatty acids that cannot be synthesized by the human body. They are classified as essential nutrients and must be obtained from dietary sources.
- The standard American diet is much higher in O6 than O3 fatty acids.
- Deficiency of EFAs may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

### Digestive Support

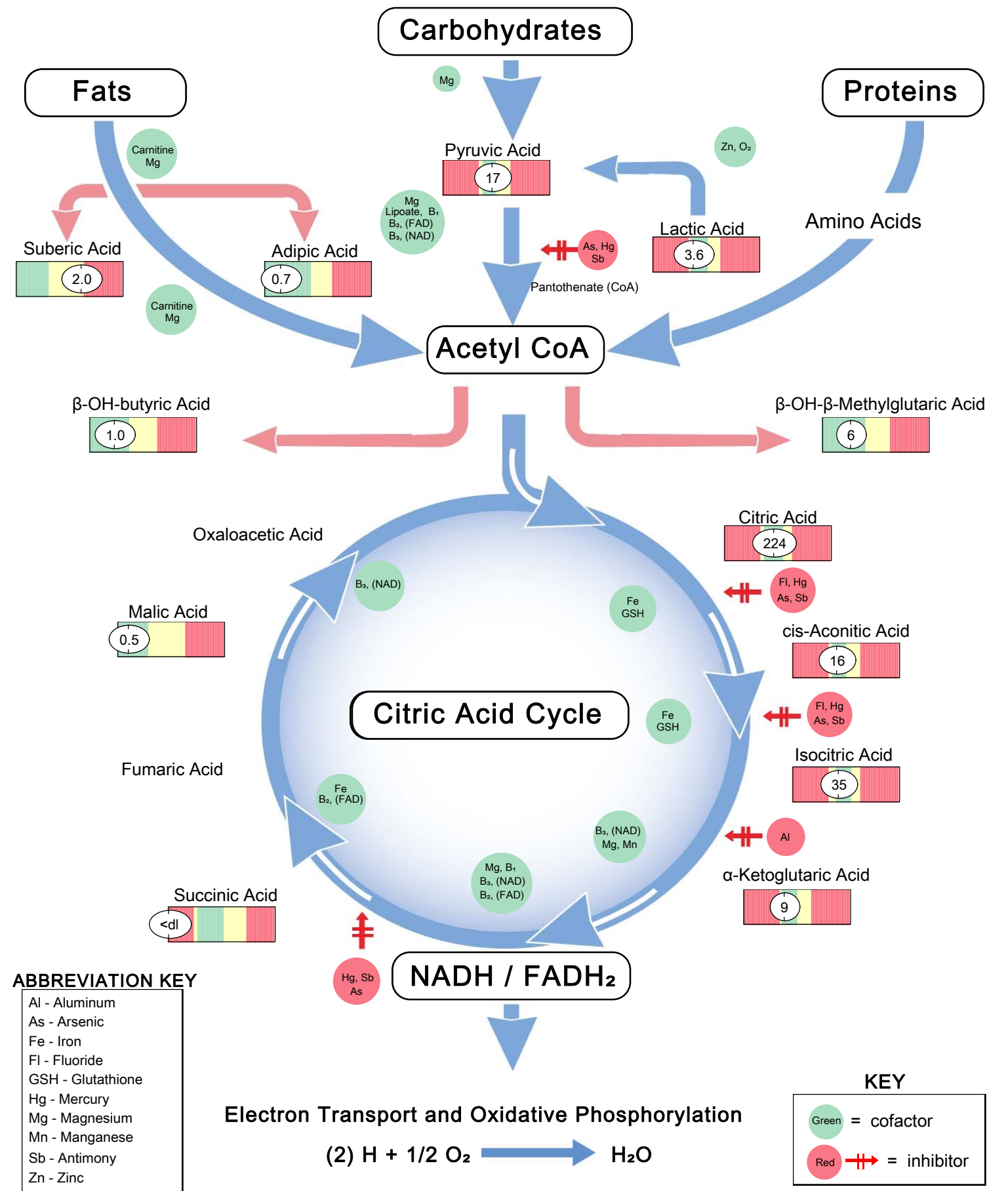


- Probiotics have many functions. These include: production of some B vitamins and vitamin K; enhance digestion & absorption; decrease severity of diarrheal illness; modulate of immune function & intestinal permeability.
- Alterations of gastrointestinal microflora may result from C-section delivery,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



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

Krebs Cycle At-A-Glance



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

**Malabsorption and Dysbiosis Markers****Malabsorption Markers**

		Reference Range
Indoleacetic Acid (IAA)	0.9	<= 4.2
Phenylacetic Acid (PAA)	0.06	<= 0.12

**Bacterial Dysbiosis Markers**

Dihydroxyphenylpropionic Acid (DHPPA)	2.6	<= 5.3
3-Hydroxyphenylacetic Acid	1.5	<= 8.1
4-Hydroxyphenylacetic Acid	9	<= 29
Benzoic Acid	0.02	<= 0.05
Hippuric Acid	100	<= 603

**Yeast / Fungal Dysbiosis Markers**

Arabinose	36	<= 96
Citramalic Acid	1.4	<= 5.8
Tartaric Acid	8	<= 15

**Cellular Energy & Mitochondrial Metabolites****Carbohydrate Metabolism**

Lactic Acid	3.6	1.9-19.8
Pyruvic Acid	17	7-32
$\beta$ -OH-Butyric Acid (BHBA)	1.0	<= 2.8

**Energy Metabolism**

Citric Acid	224	40-520
Cis-Aconitic Acid	16	10-36
Isocitric Acid	35	22-65
$\alpha$ -Ketoglutaric Acid (AKG)	9	4-52
Succinic Acid	<dl	0.4-4.6
Malic Acid	0.5	<= 3.0
$\beta$ -OH- $\beta$ -Methylglutaric Acid (HMG)	6	<= 15

**Fatty Acid Metabolism**

Adipic Acid	0.7	<= 2.8
Suberic Acid	2.0	<= 2.1

**Creatinine Concentration**

		Reference Range
Creatinine ♦	17.2	3.1-19.5 mmol/L

**Neurotransmitter Metabolites****Reference Range**

Vanilmandelic Acid	0.9	0.4-3.6
Homovanillic Acid	1.8	1.2-5.3
5-OH-indoleacetic Acid	6.3	3.8-12.1
3-Methyl-4-OH-phenylglycol	0.05	0.02-0.22
Kynurenic Acid	1.4	<= 7.1
Quinolinic Acid	5.7	<= 9.1
Kynurenic / Quinolinic Ratio	0.25	>= 0.44

**Vitamin Markers****Reference Range**

$\alpha$ -Ketoadipic Acid	0.3	<= 1.7
$\alpha$ -Ketoisovaleric Acid	0.39	<= 0.97
$\alpha$ -Ketoisocaproic Acid	0.41	<= 0.89
$\alpha$ -Keto- $\beta$ -Methylvaleric Acid	1.3	<= 2.1
Formiminoglutamic Acid (FIGlu)	1.6	<= 1.5
Glutaric Acid	0.38	<= 0.51
Isovalerylglycine	0.9	<= 3.7
Methylmalonic Acid	0.9	<= 1.9
Xanthurenic Acid	0.42	<= 0.96
3-Hydroxypropionic Acid	8	5-22
3-Hydroxyisovaleric Acid	14	<= 29

**Toxin & Detoxification Markers****Reference Range**

$\alpha$ -Ketophenylacetic Acid (from Styrene)	0.13	<= 0.46
$\alpha$ -Hydroxyisobutyric Acid (from MTBE)	3.6	<= 6.7
Orotic Acid	0.38	0.33-1.01
Pyroglutamic Acid	25	16-34

**Tyrosine Metabolism****Reference Range**

Homogentisic Acid	8	<= 19
2-Hydroxyphenylacetic Acid	0.39	<= 0.76

Metabolic Analysis Reference Ranges are Age Specific

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



*Amino Acids (FMV)*

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

**Nutritionally Essential Amino Acids**

Amino Acid	Reference Range
Arginine	11 10-64
Histidine	372 271-993
Isoleucine	27 17-52
Leucine	45 25-77
Lysine	37 34-226
Methionine	32 26-69
Phenylalanine	35 22-61
Taurine	578 80-545
Threonine	87 52-192
Tryptophan	34 23-88
Valine	19 19-53

**Nonessential Protein Amino Acids**

Amino Acid	Reference Range
Alanine	121 103-392
Asparagine	61 37-134
Aspartic Acid	55 27-74
Cysteine	38 19-70
Cystine	18 23-68
γ-Aminobutyric Acid	8 ≤ 23
Glutamic Acid	12 3-15
Glutamine	202 153-483
Proline	3 2-14
Tyrosine	35 28-113

**Creatinine Concentration**

	Reference Range
Creatinine ♦	16.3 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 ♦ as cleared by the U.S. Food and Drug Administration, assays are For Research Use Only.

**Intermediary Metabolites**

B Vitamin Markers	Reference Range
α-Aminoadipic Acid	58 11-73
α-Amino-N-butyric Acid	25 9-49
β-Aminoisobutyric Acid	86 19-163
Cystathionine	8 6-29
3-Methylhistidine	232 134-302

**Urea Cycle Markers**

Ammonia	16.4 12.0-41.0 mmol/g creatinine
Citrulline	27 9-40
Ornithine	2 3-16
Urea ♦	251 150-380 mmol/g creatinine

**Glycine/Serine Metabolites**

Glycine	450 434-1,688
Serine	160 135-426
Ethanolamine	109 156-422
Phosphoethanolamine	21 14-50
Phosphoserine	23 26-64
Sarcosine	29 ≤ 41

**Dietary Peptide Related Markers**

	Reference Range
Anserine (dipeptide)	187 8-118
Carnosine (dipeptide)	18 12-120
1-Methylhistidine	1,624 83-1,008
β-Alanine	6 ≤ 17

**Markers for Urine Representativeness**

	Reference Range
Glutamine/Glutamate	17 ≥ 12
Ammonia	16.4 12.0-41.0 mmol/g creatinine
Arginine/Ornithine	5.5 ≥ 1.0

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
$\alpha$ -Linolenic (ALA) 18:3 n3	0.27	$\geq 0.09$ wt %
Eicosapentaenoic (EPA) 20:5 n3	1.11	$\geq 0.16$ wt %
Docosapentaenoic (DPA) 22:5 n3	2.11	$\geq 1.14$ wt %
Docosahexaenoic (DHA) 22:6 n3	6.6	$\geq 2.1$ wt %
<b>% Omega 3s</b>	10.1	$\geq 3.8$

### Omega 9 Fatty Acids

Analyte	(olive oil)	Reference Range
Oleic 18:1 n9	12	10-13 wt %
Nervonic 24:1 n9	4.3	2.1-3.5 wt %
<b>% Omega 9s</b>	16.8	13.3-16.6

### Saturated Fatty Acids

Analyte	(meat, dairy, coconuts, palm oils)	Reference Range
Palmitic C16:0	18	18-23 wt %
Stearic C18:0	19	14-17 wt %
Arachidic C20:0	0.26	0.22-0.35 wt %
Behenic C22:0	1.02	0.92-1.68 wt %
Tricosanoic C23:0	0.19	0.12-0.18 wt %
Lignoceric C24:0	2.8	2.1-3.8 wt %
Pentadecanoic C15:0	0.07	0.07-0.15 wt %
Margaric C17:0	0.20	0.22-0.37 wt %
<b>% Saturated Fats</b>	40.9	39.8-43.6

### Omega 6 Fatty Acids

Analyte	(vegetable oil, grains, most meats, dairy)	Reference Range
Linoleic (LA) 18:2 n6	10.2	10.5-16.9 wt %
$\gamma$ -Linolenic (GLA) 18:3 n6	0.06	0.03-0.13 wt %
Dihomo- $\gamma$ -linolenic (DGLA) 20:3 n6	1.23	$\geq 1.19$ wt %
Arachidonic (AA) 20:4 n6	18	15-21 wt %
Docosatetraenoic (DTA) 22:4 n6	1.79	1.50-4.20 wt %
Eicosadienoic 20:2 n6	0.23	$\leq 0.26$ wt %
<b>% Omega 6s</b>	31.1	30.5-39.7

### Monounsaturated Fats

Omega 7 Fats	Reference Range
Palmitoleic 16:1 n7	0.16 $\leq 0.64$ wt %
Vaccenic 18:1 n7	0.77 $\leq 1.13$ wt %

### Trans Fat

Elaidic 18:1 n9t	0.15 $\leq 0.59$ wt %
---------------------	-----------------------

### Delta - 6 Desaturase Activity

	Upregulated	Functional	Impaired	
Linoleic / DGLA 18:2 n6 / 20:3 n6		8.3		6.0-12.3

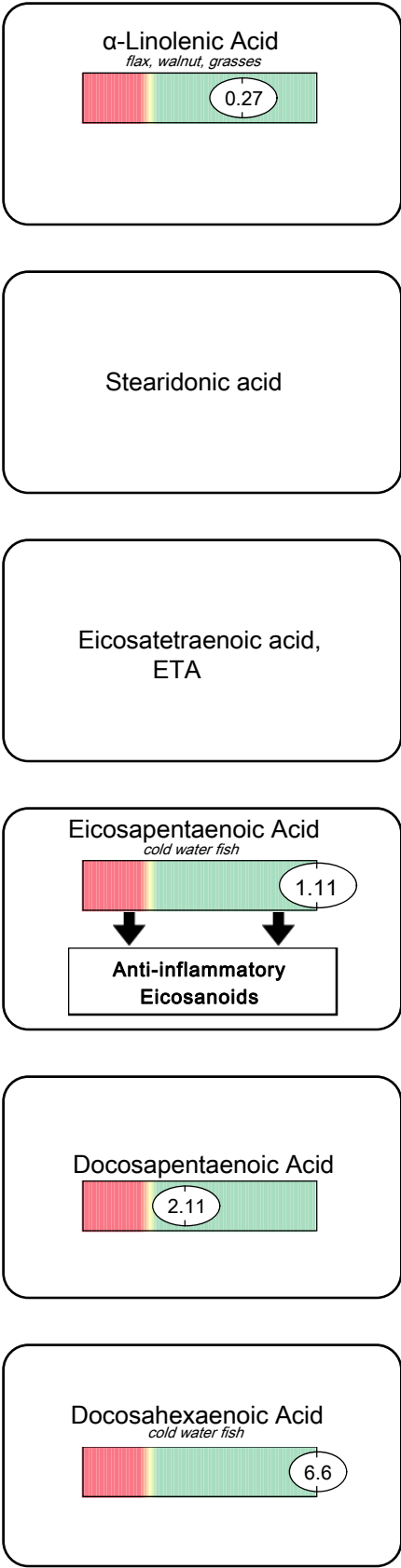
### Cardiovascular Risk

Analyte	Reference Range
Omega 6s / Omega 3s	3.1 3.4-10.7
AA / EPA 20:4 n6 / 20:5 n3	16 12-125
Omega 3 Index	7.7 $\geq 4.0$

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

Essential Fatty Acid Metabolism

Omega 3 Family



Delta-6 Desaturase

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

Elongase

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

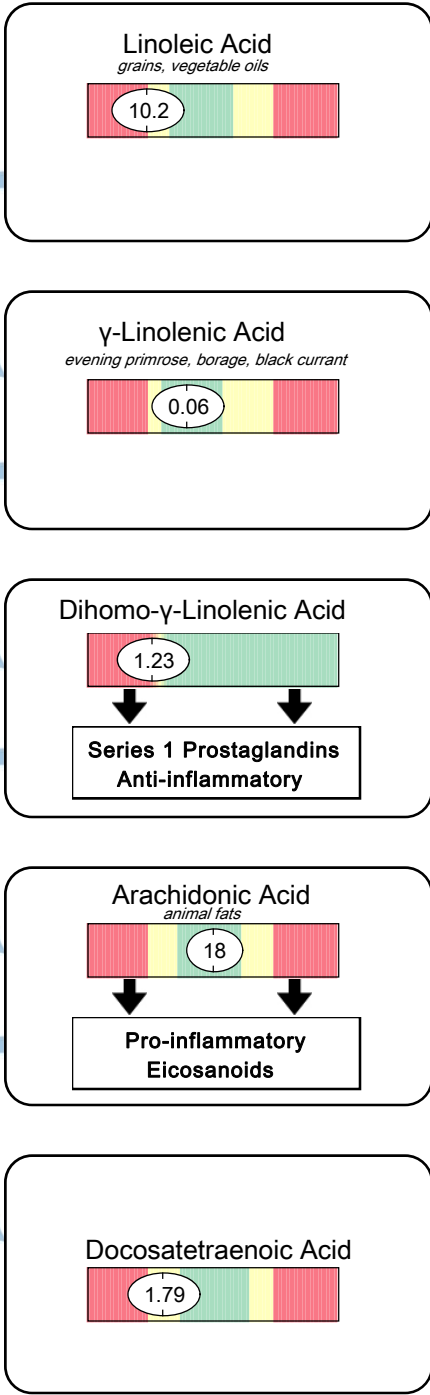
Elongase

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



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

## Oxidative Stress Markers

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

Inside Range    Outside Range    **Reference Range**

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)

## Nutrient Elements

The Elemental reference ranges are based on an adult population.

## Toxic Elements

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Patient: **PAUL**  
**BARDIN**

DOB: January 27, 1975

Sex: M

MRN: 0001757026

**Order Number: F4260192**

Completed: November 06, 2012

Received: October 26, 2012

Collected: October 25, 2012

Route Number: A155652

Rita Arora

Rita Arora

35-37 Old Brompton Road

London, SW7 3HZ

Great Britain and Northern Ireland

## Homocysteine

	Inside Range	Outside Range	Reference Range
Homocysteine	7.53		5.20-11.40 umol/L

## Commentary

This report contains an updated reference range for the biomarker Homocysteine due to a laboratory equipment update. The updated reference range 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 Medicine 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 WITHIN the REFERENCE range. As elevated homocysteine is a factor which increases cardiovascular risk, normal levels are highly desirable and beneficial. Continued attention to nutritional influences such as vitamin B6, B12 and folic acid will help maintain this level.