Adrenal Stress Profile (Saliva)

GENOVA DIAGNOSTICS EUROPE

46-50 Coombe Road
New Malden
Surrey KT3 4QF

63 Zillicoa Street
Asheville, NC 28801 USA

Patient:

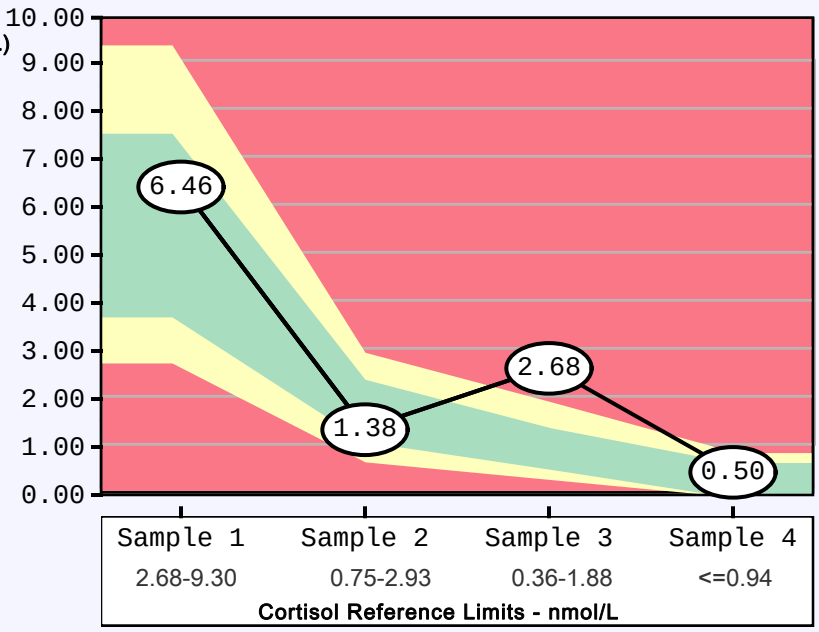
DOB:
Sex: F
MRN:

Order Number: Completed:
Received:
Collected:

Salivary Cortisol and DHEA

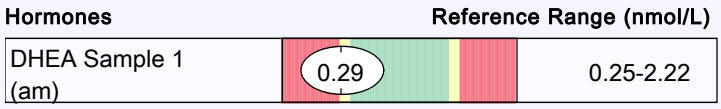
Cortisol Levels

			Reference Range (nmol/L)
Sample 1 Post Awakening	6.46		2.68-9.30
Sample 2 (+ 4 - 5 Hours)	1.38		0.75-2.93
Sample 3 (+ 4 - 5 Hours)	2.68	H	0.36-1.88
Sample 4 (Prior to Sleep)	0.50		<=0.94
Sum of Cortisol	11.020		



DHEA Level

DHEA : Cortisol Ratio	0.04	L	0.05-0.32



Testing performed by Genova Diagnostics, Inc. 63 Zillicoa St., Asheville, NC 28801-0174

Commentary

Please note the cortisol reference ranges have been updated due to a change in the assay manufacturer.

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.

Cortisol reference ranges are based on samples collected over one day during the following time periods (+/- 2hrs):
#1: 7AM - 9AM
#2: 11AM - 1PM
#3: 3PM - 5PM
#4: 10PM - 12PM

Results for samples collected outside the recommended time period should be interpreted with caution as the stated

Commentary

reference range may not apply.

For the patient:
This profile measures the levels of cortisol and DHEA and provides an evaluation of how cortisol levels differ throughout the day. Cortisol levels typically peak shortly after rising and are at their lowest after the onset of sleep. Cortisol is involved in many important functions in your body, including the metabolism and utilization of proteins, carbohydrates and fats, your body's response to physiological or psychological stress, and the control of inflammation and proper blood sugar levels. Cortisol also helps maintain proper blood pressure, normal nerve and brain activity and normal heart and immune function. DHEA also plays a role in the metabolism of protein, carbohydrates and fats, and works with cortisol to help maintain proper blood sugar levels. DHEA helps regulate body weight, blood pressure and immune function, and is used by the body to make the hormones, testosterone and estradiol. Too much or too little of cortisol or DHEA can lead to illness, and it is important that these two hormones be in balance with each other.

For the Physician:

In this profile, Sample 1 (Post awakening) cortisol level is within the reference range. Because cortisol levels are typically at their peak shortly after awakening, morning cortisol may be a good indicator of peak adrenal gland function. Morning cortisol levels within reference range suggest a component of normal adrenal function with regard to peak circadian activity.

Sample 2 cortisol level is within the reference range. Mid-day cortisol levels may be a good indication of adaptive adrenal gland function since they represent the adrenal glands' response to the demands of the first few hours of the day. Mid-day cortisol levels within reference range suggest a component of normal adrenal function in regard to adaptive response.

Sample 3 cortisol level is above the reference range. Afternoon cortisol levels may be a good indication of glycaemic control exerted by the adrenal gland since they represent a postprandial sample. High afternoon levels suggest a degree of adrenal hyperfunction with increased adrenal assistance in glycaemic control. Other possible causes of high salivary cortisol include stress, heavy exercise, pregnancy, smoking, obesity, depression, alcoholism, or if significantly elevated, adrenal hyperplasia and Cushing's syndrome.

Sample 4 cortisol level is within the reference range. Late-night cortisol levels may be a good indication of baseline adrenal gland function since they typically represent the lowest level during the day. Normal late-night cortisol levels suggest normal adrenal function with regard to baseline circadian activity.

DHEA is within the reference range. Proper levels contribute to the ideal metabolism of proteins, carbohydrates and fats, including efficient glycaemic control.

A low DHEA: cortisol ratio is generally associated with chronic stress and hypothalamic-pituitary-adrenal imbalances. While often observed in individuals as they age, it may also be associated with cognitive and mood disorders, anxiety, and depressive symptoms. DHEA levels in women tend to decrease more rapidly with aging (especially between 50-60 years of age) than DHEA levels in men.

Metabolic Analysis Profile (Urine)

Physician Copy

GENOVA DIAGNOSTICS EUROPE

46-50 Coombe Road
New Malden
Surrey KT3 4QF

63 Zillicoa Street
Asheville, NC 28801 USA

Patient:

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Sex: F
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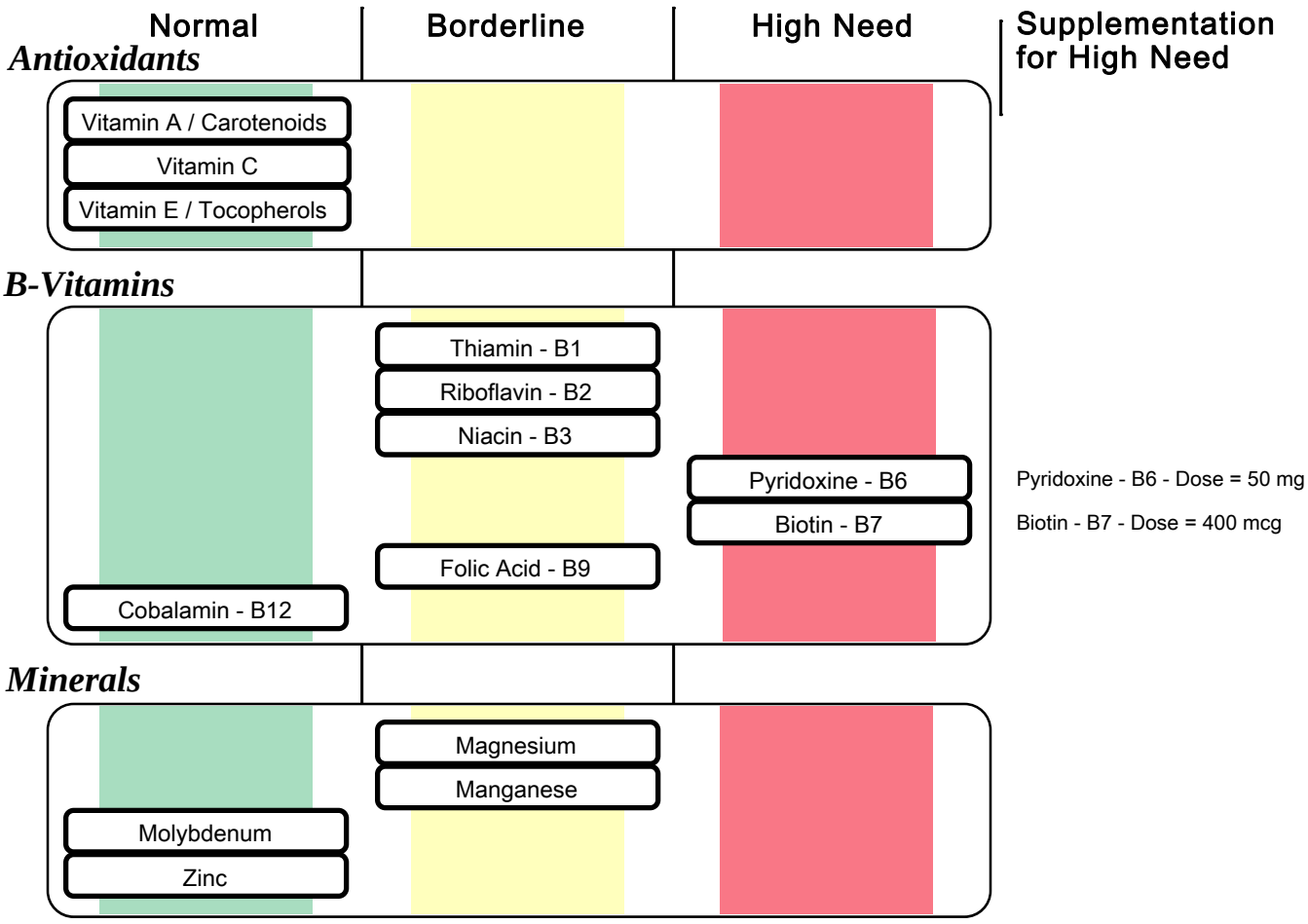
Results Overview



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	250 mg	
Vitamin E / Tocopherols	22 IU	100 IU	
B-Vitamins			
Thiamin - B1	1.1 mg	25 mg	
Riboflavin - B2	1.1 mg	25 mg	
Niacin - B3	14 mg	30 mg	
Pyridoxine - B6	1.3 mg	50 mg	
Biotin - B7	30 mcg	400 mcg	
Folic Acid - B9	400 mcg	800 mcg	
Cobalamin - B12	2.4 mcg	100 mcg	
Minerals			
Magnesium	320 mg	600 mg	
Manganese	1.8 mg	5.0 mg	
Molybdenum	45 mcg	75 mcg	
Zinc	8 mg	10 mg	
Digestive Support			
Probiotics		50 billion CFU	
Pancreatic Enzymes		5,000 IU	

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.

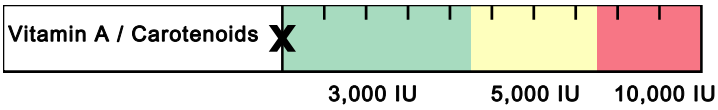
Key

Normal **Borderline** **High Need**

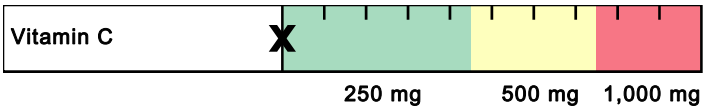
Metabolic Analysis Profile Interpretation At-A-Glance

Nutritional Needs

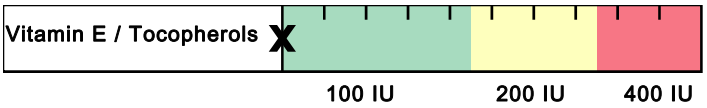
Antioxidants



- 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.



- 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.



- 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.

Key

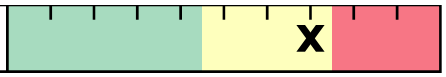
- Function
- Causes of Deficiency
- Complications of Deficiency
- Food Sources

Metabolic Analysis Profile Interpretation At-A-Glance

Nutritional Needs

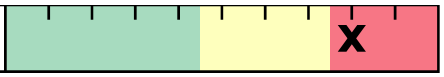
B-Vitamins

Thiamin - B1



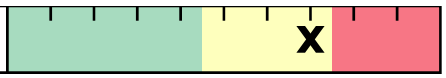
- B1 is a required cofactor for enzymes involved in energy production from food, and for the synthesis of ATP, GTP, DNA, RNA and NADPH.
- Low B1 can result from chronic alcoholism, diuretics, digoxin, oral contraceptives and HRT, or large amounts of tea & coffee (contain anti-B1 factors).
- B1 deficiency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



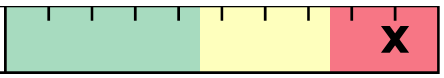
- B6 (as P5P) is a cofactor for enzymes involved in glycogenolysis & gluconeogenesis, and synthesis of neurotransmitters, heme, B3, RBCs and nucleic acids.
- Low B6 may result from chronic alcoholism, long-term diuretics, estrogens (oral contraceptives and HRT), anti-TB meds, penicillamine, L-DOPA or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



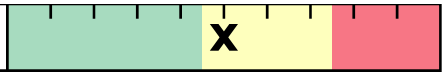
- B2 is a key component of enzymes involved in antioxidant function, energy production, detoxification, methionine metabolism and vitamin activation.
- Low B2 may result from chronic alcoholism, some anti-psychotic medications, oral contraceptives, tricyclic antidepressants, quinacrine or adriamycin.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



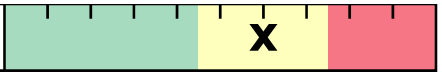
- Biotin is a cofactor for enzymes involved in functions such as fatty acid synthesis, mitochondrial FA oxidation, gluconeogenesis and DNA replication & transcription.
- Deficiency may result from certain inborn errors, chronic intake of raw egg whites, long-term TPN, anticonvulsants, high-dose B5, sulfa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

Metabolic Analysis Profile Interpretation At-A-Glance

Nutritional Needs

Minerals

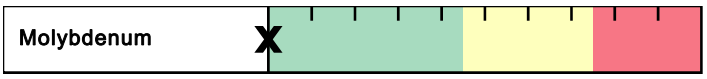
Manganese

- 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

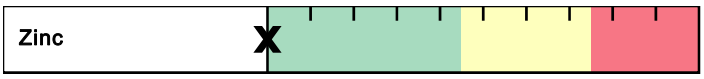
- Magnesium is involved in >300 metabolic reactions. Key areas include energy production, bone & ATP formation, muscle & nerve conduction and cell signaling.
- Deficiency may occur with malabsorption, alcoholism, hyperparathyroidism, renal disorders (wasting), diabetes, diuretics, digoxin or high doses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

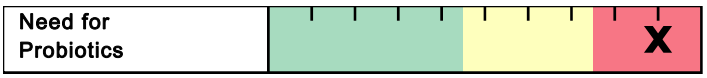
Zinc



- 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.

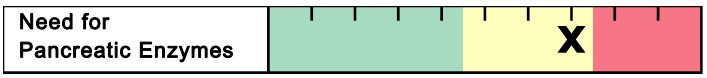
Digestive Support

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.

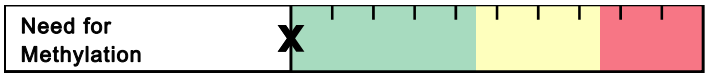
Metabolic Analysis Profile Interpretation At-A-Glance

Functional Imbalances

Mitochondrial Dysfunction

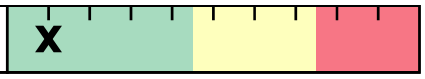
- Mitochondria are a primary site of generation of reactive oxygen species. Oxidative damage is considered an important factor in decline of physiologic function that occurs with aging and stress.
- Mitochondrial defects have been identified in cardiovascular disease, fatigue syndromes, neurologic disorders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

Need for Methylation



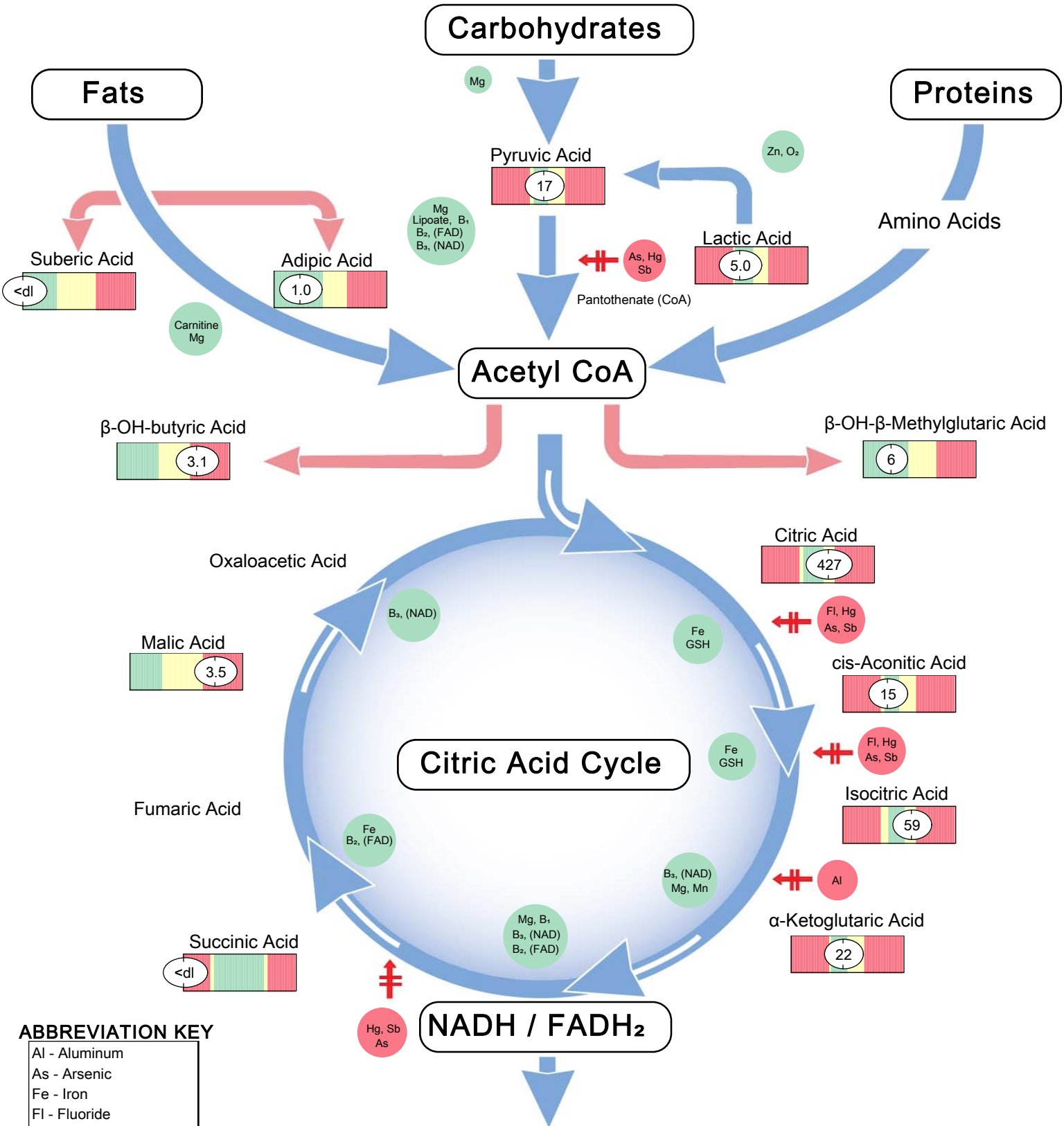
- Methylation is an enzymatic process that is critical for both synthesis and inactivation. DNA, estrogen and neurotransmitter metabolism are all dependent on appropriate methylation activity.
- B vitamins and other nutrients (methionine, magnesium, selenium) functionally support catechol-O-methyltransferase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

Toxic Exposure



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

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)

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

Malabsorption and Dysbiosis Markers

Malabsorption Markers		Reference Range
Indoleacetic Acid (IAA)	1.8	<= 4.2
Phenylacetic Acid (PAA)	0.19	<= 0.12
Bacterial Dysbiosis Markers		
Dihydroxyphenylpropionic Acid (DHPPA)	5.8	<= 5.3
3-Hydroxyphenylacetic Acid	3.7	<= 8.1
4-Hydroxyphenylacetic Acid	10	<= 29
Benzoic Acid	>1.14	<= 0.05
Hippuric Acid	264	<= 603
Yeast / Fungal Dysbiosis Markers		
Arabinose	72	<= 96
Citramalic Acid	6.4	<= 5.8
Tartaric Acid	<dl	<= 15

Cellular Energy & Mitochondrial Metabolites

Carbohydrate Metabolism		Reference Range
Lactic Acid	5.0	1.9-19.8
Pyruvic Acid	17	7-32
β-OH-Butyric Acid (BHBA)	3.1	<= 2.8
Energy Metabolism		
Citric Acid	427	40-520
Cis-Aconitic Acid	15	10-36
Isocitric Acid	59	22-65
α-Ketoglutaric Acid (AKG)	22	4-52
Succinic Acid	<dl	0.4-4.6
Malic Acid	3.5	<= 3.0
β-OH-β-Methylglutaric Acid (HMG)	6	<= 15
Fatty Acid Metabolism		
Adipic Acid	1.0	<= 2.8
Suberic Acid	<dl	<= 2.1

Creatinine Concentration

		Reference Range
Creatinine ◆	4.3	3.1-19.5 mmol/L

Neurotransmitter Metabolites

		Reference Range
Vanilmandelic Acid	1.6	0.4-3.6
Homovanillic Acid	2.3	1.2-5.3
5-OH-indoleacetic Acid	12.3	3.8-12.1
3-Methyl-4-OH-phenylglycol	0.08	0.02-0.22
Kynurenic Acid	4.9	<= 7.1
Quinolinic Acid	3.8	<= 9.1
Kynurenic / Quinolinic Ratio	1.29	>= 0.44

Vitamin Markers

		Reference Range
α-Ketoadipic Acid	1.0	<= 1.7
α-Ketoisovaleric Acid	0.51	<= 0.97
α-Ketoisocaproic Acid	0.62	<= 0.89
α-Keto-β-Methylvaleric Acid	1.6	<= 2.1
Formiminoglutamic Acid (FIGlu)	1.4	<= 1.5
Glutaric Acid	0.37	<= 0.51
Isovalerylglycine	2.7	<= 3.7
Methylmalonic Acid	1.2	<= 1.9
Xanthurenic Acid	1.44	<= 0.96
3-Hydroxypropionic Acid	9	5-22
3-Hydroxyisovaleric Acid	30	<= 29

Toxin & Detoxification Markers

		Reference Range
α-Ketophenylacetic Acid (from Styrene)	0.29	<= 0.46
α-Hydroxyisobutyric Acid (from MTBE)	5.3	<= 6.7
Orotic Acid	1.10	0.33-1.01
Pyroglutamic Acid	31	16-34

Tyrosine Metabolism

		Reference Range
Homogentisic Acid	13	<= 19
2-Hydroxyphenylacetic Acid	0.50	<= 0.76

Methodology: GCMS, LCMSMS, Enzymatic and Kinetic (Jaffe)

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.

Metabolic Analysis Markers (Urine)

Commentary

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

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

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

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

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

Citramalic Acid is elevated. With a chemical structure very similar to that of malic acid, citramalate may cause metabolic interference with malate. This is of concern because malic acid has extra-mitochondrial functions, as with the "malate shuttle" for carrying reducing equivalents (protons) into the mitochondria. While the metabolic interference aspect is uncertain, the presence of citramalate in the urine indicates intestinal dysbiosis. Not formed in human tissues, citramalate may be formed by anaerobic bacteria such as clostridia, as well as by yeast/fungi. A stool analysis with bacteriology or microbiology is suggested.

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

Commentary

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.

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.

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.

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

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.

Xanthurenic acid (XA) is a metabolite of tryptophan. When tryptophan intake is increased, the XA excretion increases. Xanthurenic acid secretion is higher among women in general, in women taking oral contraceptives, and in women with PMS, thus there appears a relationship to high XA excretion and high estrogen levels, likely to also manifest in pregnant women. An increased excretion of XA is also present in vitamin B6 deficiency, and is normalized by administration of vitamin B6.

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,

Commentary

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.

INTERPRETATION AT A GLANCE

5-HIAA is measured to be high.

Possible Conditions	Possible Remedies	Confirming Tests
Excessive dietary intake of bananas, pineapple, kiwi, walnuts, plums, avocados or pecans	None, or dietary intervention	Diet analysis
Recent use of guaifenesin or acetaminophen	None	Medication history
Methylation deficit (consistent with sub-normal melatonin, sleep disorder, fatigue)	L-Methionine 500-1000 mg/d, Magnesium 100-200 mg/d, Same as beneficial	Plasma amino acid analysis RBC element (Mg) analysis
Abdominal/intestinal inflammation with increased vasoconstrictor dysbiosis/leaky gut	Dietary intervention Correction of intestinal flora, digestive aids	Stool analysis for dysbiosis, maldigestion Intestinal permeability Food reactivity assessment
Serotonin utilization disorder or possibly secondary to medications	As prescribed by neuroendocrinologist or psychiatrist	Medication, history, (depression, migraine, bulimia, panic attacks)
Carcinoid syndrome (usually features markedly high 5-HIAA)	As prescribed by oncologist	Plasma serotonin endoscopy, CT-scan, biopsy

Commentary

Malic Acid is elevated. Malic Acid is elevated. Malic Acid is elevated.

Possible Conditions	Possible Remedies	Confirming Tests
Use of DL-malic acid supplements	Change to L-forms	Check labels
Malic dehydrogenase inhibition		
Pyruvate excess	See Elevated Pyruvate	See Elevated Pyruvate
Inhibited "malic enzyme"		
Hyperglycemia	Treat for dysglycemia	Dysglycemia assessment
Insulin insufficiency	Treat for diabetes	Diabetes evaluation
Glucagon excess		
Hypothyroidism	Treat for hypothyroidism	Thyroid function assay
Mitochondrial myopathy	Referral to a specialist	Muscle cell histology

Beta-Hydroxybutyric Acid is elevated, consistent with ketoacidosis.

Possible conditions	Possible Remedies	Confirming Tests
Alcoholism	Detoxification and counseling dietary intervention	Patient history Diet assessment
Starvation, anorexia	Dietary intervention	Diet assessment
Diabetic state	Treat for diabetes	Test for diabetes
Impaired Krebs Cycle enzyme kinetics	See other abnormal results	See other abnormal results
Toxicity	Detoxification	Toxicity assessments
Electrolyte imbalance	Replenish electrolytes	Blood electrolytes, especially potassium
Low tissue oxygenation	See Elevated Lactic Acid	See Elevated Lactic Acid
Metabolic organic acidemia/aciduria, e.g., methylmalonic aciduria (MMA should be elevated)	Vitamin B12 100-2000 ug/d, if MMA is also elevated; Referral to metabolism specialist" in "Possible Remedies	Biochemical genetics testing

Metabolic Analysis Profile (Urine)

Patient Copy

GENOVA DIAGNOSTICS EUROPE

46-50 Coombe Road
New Malden
Surrey KT3 4QF

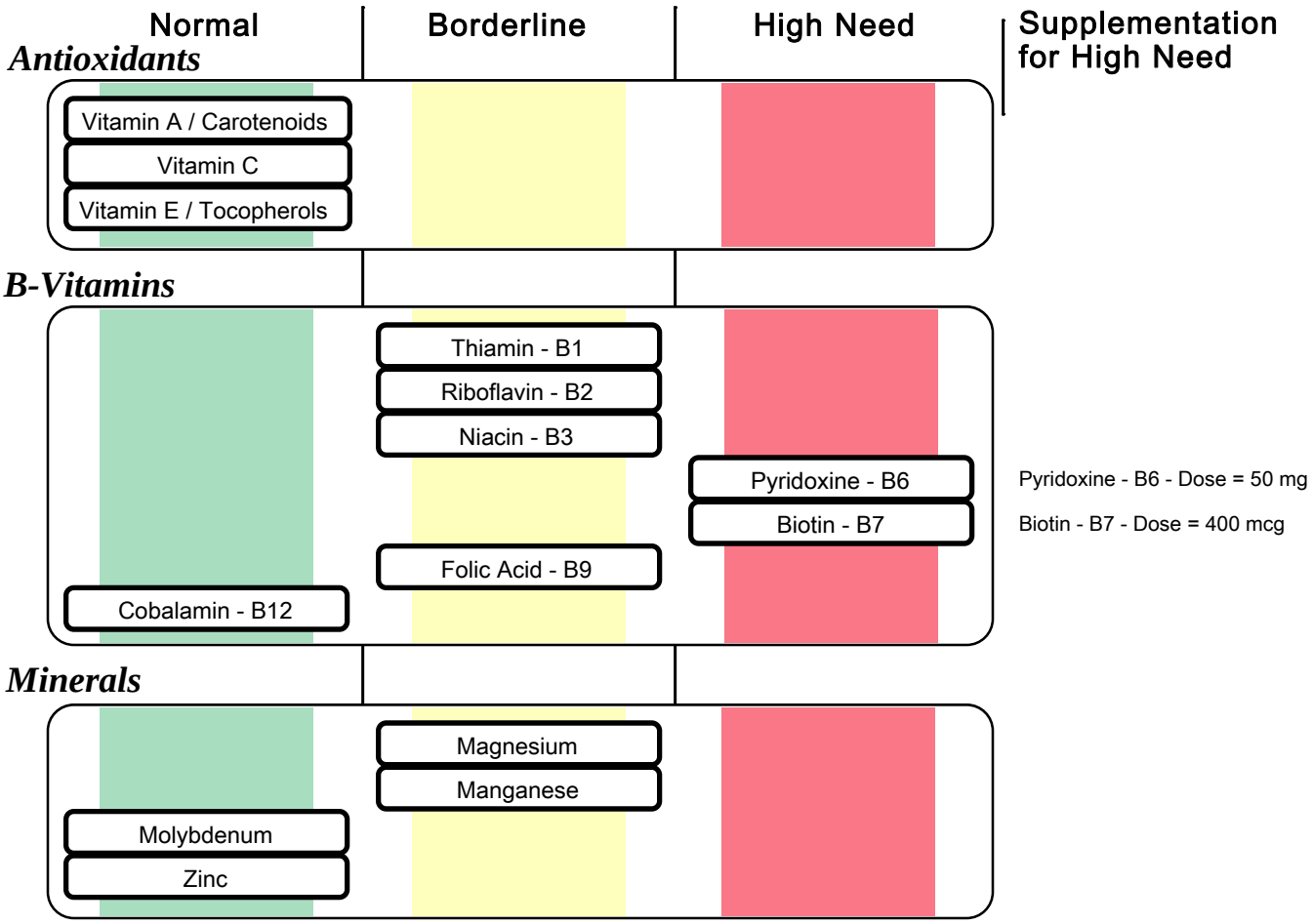
63 Zillicoa Street
Asheville, NC 28801 USA

Patient:

DOB:
Sex: F
MRN:

Order Number: K9160051
Completed:
Received:
Collected:

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	250 mg	
Vitamin E / Tocopherols	22 IU	100 IU	
B-Vitamins			
Thiamin - B1	1.1 mg	25 mg	
Riboflavin - B2	1.1 mg	25 mg	
Niacin - B3	14 mg	30 mg	
Pyridoxine - B6	1.3 mg	50 mg	
Biotin - B7	30 mcg	400 mcg	
Folic Acid - B9	400 mcg	800 mcg	
Cobalamin - B12	2.4 mcg	100 mcg	
Minerals			
Magnesium	320 mg	600 mg	
Manganese	1.8 mg	5.0 mg	
Molybdenum	45 mcg	75 mcg	
Zinc	8 mg	10 mg	
Digestive Support			
Probiotics		50 billion CFU	
Pancreatic Enzymes		5,000 IU	

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.

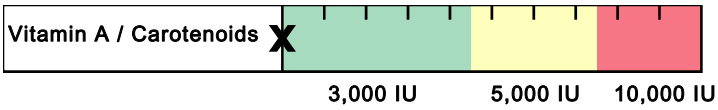
Key

Normal **Borderline** **High Need**

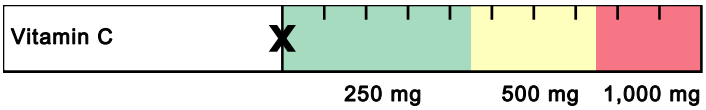
Metabolic Analysis Profile Interpretation At-A-Glance

Nutritional Needs

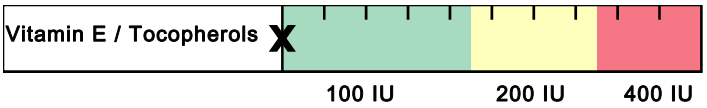
Antioxidants



- 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.



- 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.



- 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.

Key

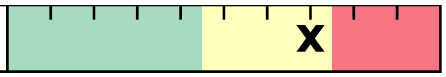
- Function
- Causes of Deficiency
- Complications of Deficiency
- Food Sources

Metabolic Analysis Profile Interpretation At-A-Glance

Nutritional Needs

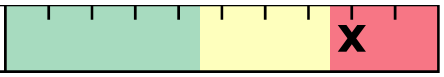
B-Vitamins

Thiamin - B1



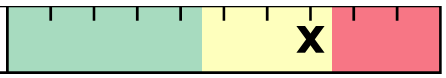
- B1 is a required cofactor for enzymes involved in energy production from food, and for the synthesis of ATP, GTP, DNA, RNA and NADPH.
- Low B1 can result from chronic alcoholism, diuretics, digoxin, oral contraceptives and HRT, or large amounts of tea & coffee (contain anti-B1 factors).
- B1 deficiency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



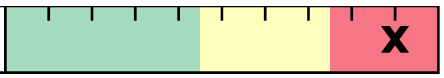
- B6 (as P5P) is a cofactor for enzymes involved in glycogenolysis & gluconeogenesis, and synthesis of neurotransmitters, heme, B3, RBCs and nucleic acids.
- Low B6 may result from chronic alcoholism, long-term diuretics, estrogens (oral contraceptives and HRT), anti-TB meds, penicillamine, L-DOPA or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



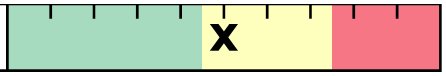
- B2 is a key component of enzymes involved in antioxidant function, energy production, detoxification, methionine metabolism and vitamin activation.
- Low B2 may result from chronic alcoholism, some anti-psychotic medications, oral contraceptives, tricyclic antidepressants, quinacrine or adriamycin.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



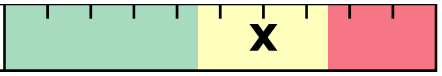
- Biotin is a cofactor for enzymes involved in functions such as fatty acid synthesis, mitochondrial FA oxidation, gluconeogenesis and DNA replication & transcription.
- Deficiency may result from certain inborn errors, chronic intake of raw egg whites, long-term TPN, anticonvulsants, high-dose B5, sulfa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

Metabolic Analysis Profile Interpretation At-A-Glance

Nutritional Needs

Minerals

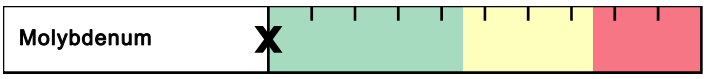
Manganese

- 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

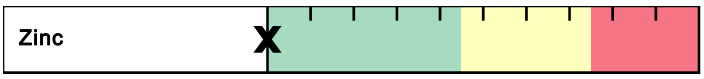
- Magnesium is involved in >300 metabolic reactions. Key areas include energy production, bone & ATP formation, muscle & nerve conduction and cell signaling.
- Deficiency may occur with malabsorption, alcoholism, hyperparathyroidism, renal disorders (wasting), diabetes, diuretics, digoxin or high doses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

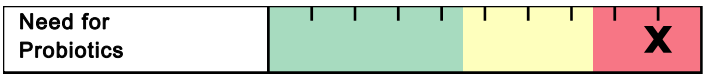
Zinc



- 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.

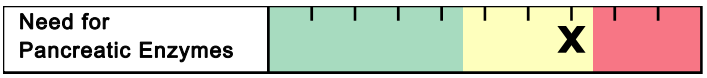
Digestive Support

Need for Probiotics



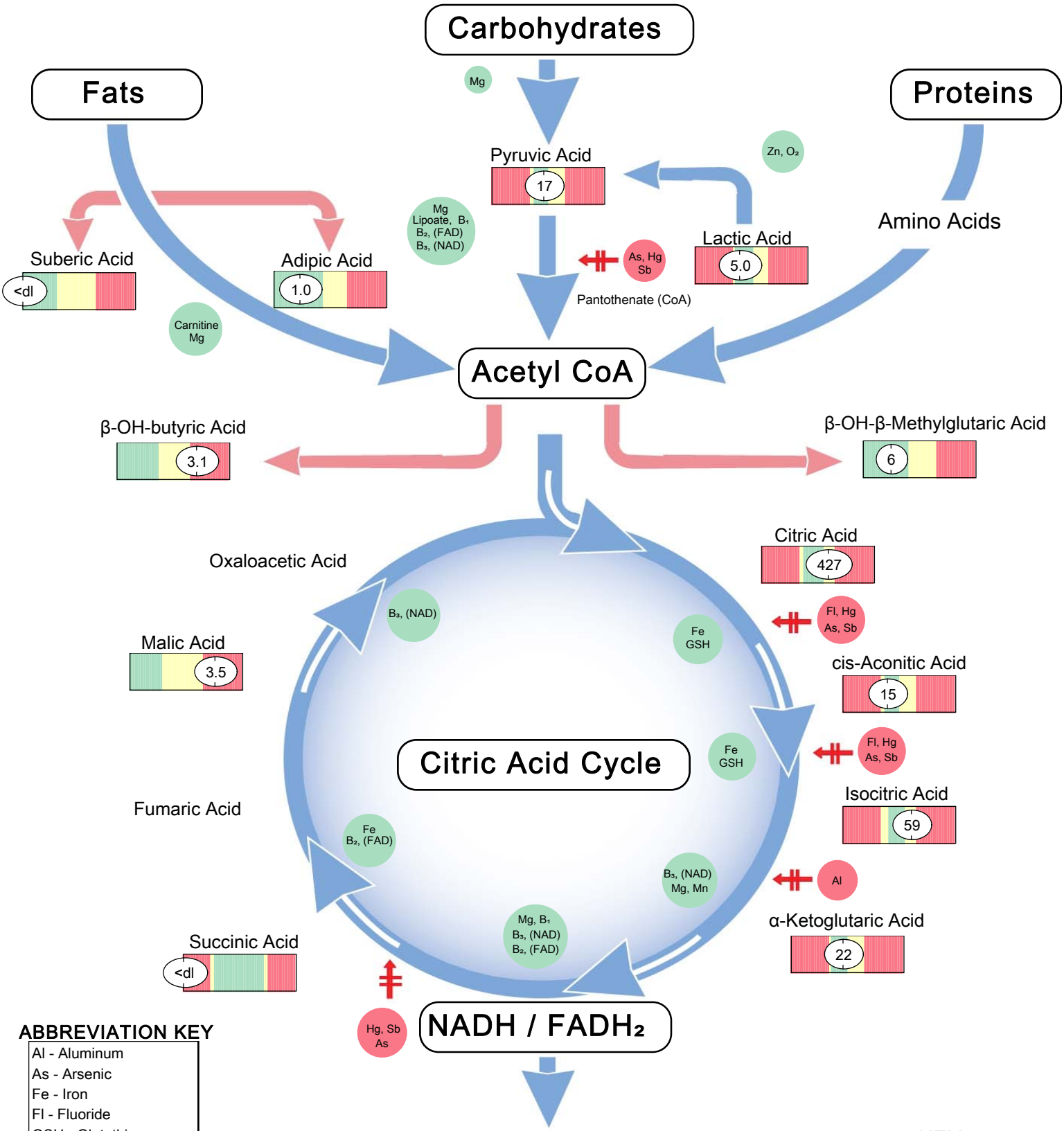
- Probiotics have many functions. These include: production of some B vitamins and vitamin K; enhance digestion & absorption; decrease severity of diarrheal illness; modulate of immune function & intestinal permeability.
- Alterations of gastrointestinal microflora may result from C-section delivery,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

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)

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

Malabsorption and Dysbiosis Markers

Malabsorption Markers		Reference Range
Indoleacetic Acid (IAA)	1.8	<= 4.2
Phenylacetic Acid (PAA)	0.19	<= 0.12
Bacterial Dysbiosis Markers		
Dihydroxyphenylpropionic Acid (DHPPA)	5.8	<= 5.3
3-Hydroxyphenylacetic Acid	3.7	<= 8.1
4-Hydroxyphenylacetic Acid	10	<= 29
Benzoic Acid	>1.14	<= 0.05
Hippuric Acid	264	<= 603
Yeast / Fungal Dysbiosis Markers		
Arabinose	72	<= 96
Citramalic Acid	6.4	<= 5.8
Tartaric Acid	<dl	<= 15

Cellular Energy & Mitochondrial Metabolites

Carbohydrate Metabolism		Reference Range
Lactic Acid	5.0	1.9-19.8
Pyruvic Acid	17	7-32
β-OH-Butyric Acid (BHBA)	3.1	<= 2.8
Energy Metabolism		
Citric Acid	427	40-520
Cis-Aconitic Acid	15	10-36
Isocitric Acid	59	22-65
α-Ketoglutaric Acid (AKG)	22	4-52
Succinic Acid	<dl	0.4-4.6
Malic Acid	3.5	<= 3.0
β-OH-β-Methylglutaric Acid (HMG)	6	<= 15
Fatty Acid Metabolism		
Adipic Acid	1.0	<= 2.8
Suberic Acid	<dl	<= 2.1

Creatinine Concentration

		Reference Range
Creatinine ◆	4.3	3.1-19.5 mmol/L

Neurotransmitter Metabolites

		Reference Range
Vanilmandelic Acid	1.6	0.4-3.6
Homovanillic Acid	2.3	1.2-5.3
5-OH-indoleacetic Acid	12.3	3.8-12.1
3-Methyl-4-OH-phenylglycol	0.08	0.02-0.22
Kynurenic Acid	4.9	<= 7.1
Quinolinic Acid	3.8	<= 9.1
Kynurenic / Quinolinic Ratio	1.29	>= 0.44

Vitamin Markers

		Reference Range
α-Ketoadipic Acid	1.0	<= 1.7
α-Ketoisovaleric Acid	0.51	<= 0.97
α-Ketoisocaproic Acid	0.62	<= 0.89
α-Keto-β-Methylvaleric Acid	1.6	<= 2.1
Formiminoglutamic Acid (FIGlu)	1.4	<= 1.5
Glutaric Acid	0.37	<= 0.51
Isovalerylglycine	2.7	<= 3.7
Methylmalonic Acid	1.2	<= 1.9
Xanthurenic Acid	1.44	<= 0.96
3-Hydroxypropionic Acid	9	5-22
3-Hydroxyisovaleric Acid	30	<= 29

Toxin & Detoxification Markers

		Reference Range
α-Ketophenylacetic Acid (from Styrene)	0.29	<= 0.46
α-Hydroxyisobutyric Acid (from MTBE)	5.3	<= 6.7
Orotic Acid	1.10	0.33-1.01
Pyroglutamic Acid	31	16-34

Tyrosine Metabolism

		Reference Range
Homogentisic Acid	13	<= 19
2-Hydroxyphenylacetic Acid	0.50	<= 0.76

Methodology: GCMS, LCMSMS, Enzymatic and Kinetic (Jaffe)

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.

Lipid Peroxides (Urine)

GENOVA DIAGNOSTICS EUROPE

46-50 Coombe Road
New Malden
Surrey KT3 4QF

63 Zillicoa Street
Asheville, NC 28801 USA

Patient:

Order Number:

Completed:

DOB:

Received:

Sex: F

Collected:

MRN:

Lipid Peroxides (Urine)

		Reference Range
Urine Lipid Peroxides	5.9	<= 10.0 umol/g Creat.

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.

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.

Urine lipid peroxides is a marker of free radical damage in the body. An elevated level may reflect excess free radical production and/or insufficient antioxidants. Free radical damage is thought to underlie many processes such as atherosclerosis, chronic fatigue syndrome, cancer, cardiovascular disease, Parkinson's disease, Alzheimer's, and aging.