Interpretation At-A-Glance Details

Antioxidants

Vitamin C

Contributing Biomarkers:
Glutathione

α-Lipoic Acid

Contributing Biomarkers:
α-Keto-β-Methylvaleric Acid
α-Ketoisocaproic Acid
Glutathione
Isoleucine
Pyruvic Acid

Glutathione

Contributing Biomarkers:
Glutathione

Plant-based Antioxidants

Contributing Biomarkers:
Glutathione

B-Vitamins

Thiamin - B1

Contributing Biomarkers:
5-OH-Indoleacetic Acid
α-Keto-β-Methylvaleric Acid
α-Ketoisocaproic Acid
Citrulline
Isoleucine
Proline
Pyruvic Acid

Riboflavin - B2

Contributing Biomarkers:
Adipic Acid
Glutaric Acid
Pyruvic Acid
Sarcosine

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Folic Acid - B9

Contributing Biomarkers:
Formiminoglutamic Acid
Sarcosine

Cobalamin - B12

Contributing Biomarkers:
Cysteine
Formiminoglutamic Acid
Isoleucine
Sarcosine

Minerals

Manganese

Contributing Biomarkers:
5-OH-Indoleacetic Acid

Molybdenum

Contributing Biomarkers:
Cysteine

Magnesium

Contributing Biomarkers:
Ethanolamine
Phosphoethanolamine
Isocitric Acid

Essential Fatty Acids

Need for Essential Fatty Acids

Contributing Biomarkers:
Omega 3 Index
Dihomo-γ-linolenic Acid

Digestive Support

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Need for Probiotics	**Contributing Biomarkers:** Benzoic Acid Dihydroxyphenylpropionic Acid
Need for Pancreatic Enzymes	**Contributing Biomarkers:** Dihydroxyphenylpropionic Acid

Functional Imbalances

Mitochondrial Dysfunction	**Contributing Biomarkers:** Adipic Acid Glutathione Glutaric Acid
Need for Methylation	**Contributing Biomarkers:** Glutathione Sarcosine
Toxic Exposure	**Contributing Biomarkers:** Glutaric Acid

Commentary

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.

Tartaric Acid is measured to be above the normal range. Widely distributed in fruits, tartaric acid is particularly high in grapes, raisins, and in wine. It may also be found in some soft drinks and baked goods (as "cream of tartar"). Therefore, the elevation of tartaric acid may be due to recent ingestion of higher than usual amounts of some of these foods. In chemical structure, tartaric acid is very similar to malic acid; thus, elevated blood or tissue levels may interfere with malic acid's role in the "malate shuttle", which carries reducing equivalents (protons) into the mitochondria. Aside from dietary sources, elevated urine tartrate can be the result of (intestinal) yeast overgrowth. A stool analysis with microbiology is suggested if dietary sources are ruled out.

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

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

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

Glutaric Acid is measured to be high. This organic acid is formed from the essential amino acids lysine (primarily) and tryptophan, via alpha ketoadipic acid (AKAA) and glutaryl-CoA. Glutaric acid is elevated when glutaryl CoA