



# The Great Plains Laboratory, Inc.

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Requisition #: 226924

Physician Name: Ron Hunninghake

Patient Name: Jonathan Barnett

Date of Collection: 12/15/2010

Patient Age: 54

Time of Collection: 06:00 AM

Sex: M

Print Date: 12/21/2010



## Organic Acids Test - Nutritional and Metabolic Profile

### Metabolic Markers in Urine

Reference Range  
(mmol/mol creatinine)

Patient  
Value

Reference Population - Males Age 13 and Over

### Intestinal Microbial Overgrowth

#### Yeast and Fungal Markers

Marker	Reference Range	Patient Value	Reference Population - Males Age 13 and Over
1 Citramalic	0.11 - 2.0	1.1	1.1
2 5-Hydroxymethyl-2-furoic	≤ 18	13	13
3 3-Oxoglutaric	≤ 0.11	H 1.8	1.8
4 Furan-2,5-dicarboxylic	≤ 13	H 35	35
5 Furancarboxylglycine	≤ 2.3	0.17	0.17
6 Tartaric	≤ 5.3	1.3	1.3
7 Arabinose	≤ 20	H 51	51
8 Carboxycitric	≤ 20	3.9	3.9

#### Malabsorption and Bacterial Markers

Marker	Reference Range	Patient Value	Reference Population - Males Age 13 and Over
9 2-Hydroxyphenylacetic	0.03 - 0.47	0.44	0.44
10 4-Hydroxyphenylacetic	≤ 18	10	10
11 4-Hydroxybenzoic	0.01 - 0.73	H 1.7	1.7
12 4-Hydroxyhippuric	≤ 14	11	11
13 Hippuric	≤ 241	H 300	300
14 3-Indoleacetic	≤ 6.8	2.6	2.6
15 Succinic	≤ 5.3	H 19	19
16 HPPA (Clostridia marker)	≤ 102	87	87
17 DHPPA (beneficial bacteria)	≤ 0.23	0.06	0.06

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## Oxalate Metabolites

18	Glyceric	0.21 - 4.9	H	6.0	
19	Glycolic	18 - 81		31	
20	Oxalic	8.9 - 67	H	85	

## Glycolytic Cycle Metabolites

21	Lactic	0.74 - 19		9.3	
22	Pyruvic	0.28 - 6.7		3.8	
23	2-Hydroxybutyric	≤ 1.2		0.56	

## Krebs Cycle Metabolites

24	Succinic	≤ 5.3	H	19	
25	Fumaric	≤ 0.49		0.44	
26	Malic	≤ 1.1	H	1.3	
27	2-Oxoglutaric	≤ 18		1.6	
28	Aconitic	4.1 - 23		14	
29	Citric	2.2 - 260		224	

## Neurotransmitter Metabolites

30	Homovanillic (HVA)	0.39 - 2.2		1.9	
31	Vanillylmandelic (VMA)	0.53 - 2.2		1.1	
32	5-Hydroxyindoleacetic (5-HIAA)	≤ 2.9		0.47	
33	Quinolinic	0.52 - 2.4	H	9.2	
34	Kynurenic	0.12 - 1.8	H	1.9	
35	Quinolinic / 5-HIAA Ratio	≤ 2.5	H	20	
36	Quinolinic / Kynurenic Ratio	0.22 - 3.0	H	4.9	

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## Pyrimidine Metabolites

37	Uracil	≤ 6.9	4.3	
38	Thymine	≤ 0.36	0.17	

## Ketone and Fatty Acid Oxidation

39	3-Hydroxybutyric	≤ 1.9	1.7	
40	Acetoacetic	≤ 10	0	
41	4-Hydroxybutyric	≤ 4.3	0.22	
42	Ethylmalonic	0.13 - 2.7	1.6	
43	Methylsuccinic	≤ 2.3	1.8	
44	Adipic	≤ 2.9	H 8.3	
45	Suberic	≤ 1.9	H 3.1	
46	Sebacic	≤ 0.14	H 0.36	

## Nutritional Markers

<b>Vitamin B12</b>				
47	Methylmalonic	≤ 2.3	1.3	
<b>Vitamin B6</b>				
48	Pyridoxic	≤ 26	11	
<b>Vitamin B5</b>				
49	Pantothenic	≤ 5.4	H 35	
<b>Vitamin B2 (Riboflavin)</b>				
50	Glutaric	≤ 0.43	H 1.5	
<b>Vitamin C</b>				
51	Ascorbic	10 - 200	H 2676	
<b>Vitamin Q10 (CoQ10)</b>				
52	3-Hydroxy-3-methylglutaric	≤ 26	10	
<b>Glutathione Precursor and Chelating Agent</b>				
53	N-Acetylcysteine	≤ 0.13	0.10	

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## Nutritional Markers

### Biotin (Vitamin H)

54	Methylcitric	0.15 - 1.7	0.73	
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## Indicators of Detoxification

55	Pyroglutamic	5.7 - 25	23	
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56	Orotic	≤ 0.46	0.33	
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57	2-Hydroxyhippuric	≤ 0.86	0.84	
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## Amino Acid Metabolites

58	2-Hydroxyisovaleric	≤ 0.41	0	
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59	2-Oxoisovaleric	≤ 1.5	0	
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60	3-Methyl-2-oxovaleric	≤ 0.56	0.22	
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61	2-Hydroxyisocaproic	≤ 0.39	0.04	
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62	2-Oxoisocaproic	≤ 0.34	0.04	
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63	2-Oxo-4-methylbutyric	≤ 0.14	0.05	
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64	Mandelic	≤ 0.09	0	
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65	Phenyllactic	≤ 0.10	0.03	
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66	Phenylpyruvic	0.02 - 1.4	0.25	
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67	Homogentisic	≤ 0.23	0.05	
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68	4-Hydroxyphenyllactic	≤ 0.62	0.38	
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69	N-Acetylaspartic	≤ 2.5	2.0	
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70	Malonic	≤ 9.9	0	
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71	3-Methylglutaric	0.02 - 0.38	H 0.43	
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## Bone Metabolites

72	Phosphoric	332 - 5 040	H 8 519	
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## Indicator of Fluid Intake

73 \*Creatinine

107 mg/dL

\*The creatinine test is performed to adjust metabolic marker results for differences in fluid intake. Urinary creatinine has limited diagnostic value due to variability as a result of recent fluid intake. Samples are rejected if creatinine is below 20 mg/dL unless the client requests results knowing of our rejection criteria.

### Explanation of Report Format

The reference ranges for organic acids were established using samples collected from typical individuals of all ages with no known physiological or psychological disorders. The ranges were determined by calculating the mean and standard deviation (SD) and are defined as  $\pm 2SD$  of the mean. Reference ranges are age and gender specific, consisting of Male Adult ( $\geq 13$  years), Female Adult ( $\geq 13$  years), Male Child ( $< 13$  years), and Female Child ( $< 13$  years).

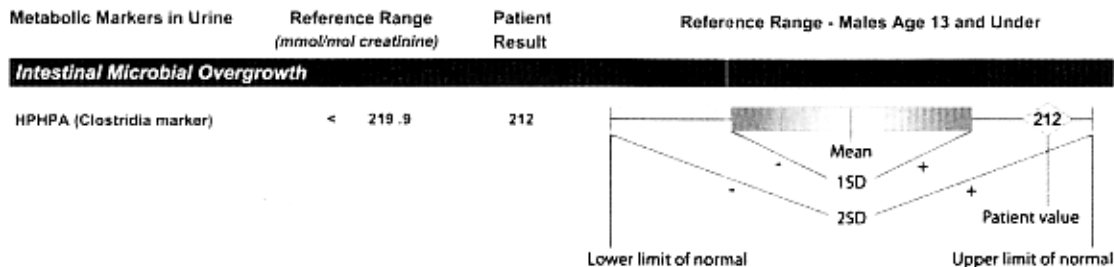
There are two types of graphical representations of patient values found in the new report format of both the standard Organic Acids Test and the Microbial Organic Acids Test.

The first graph will occur when the value of the patient is within the reference (normal) range, defined as the mean plus or minus two standard deviations.

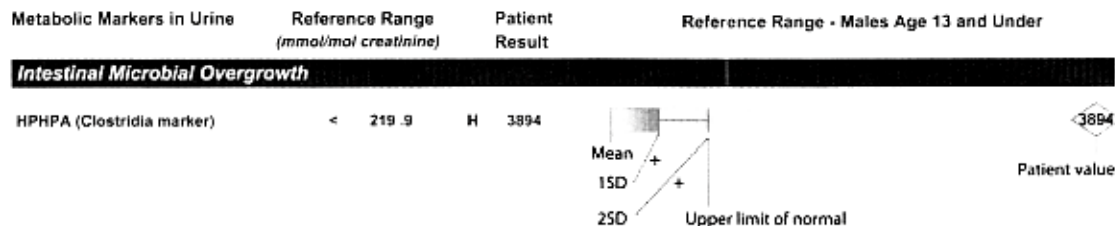
The second graph will occur when the value of the patient exceeds the upper limit of normal. In such cases, the graphical reference range is "shrunk" so that the degree of abnormality can be appreciated at a glance. In this case, the lower limits of normal are not shown, only the upper limit of normal is shown.

In both cases, the value of the patient is given to the left of the graph and is repeated on the graph inside a diamond. If the value is within the normal range, the diamond will be outlined in black. If the value is high or low, the diamond will be outlined in red.

### Example of Value Within Reference Range



### Example of Elevated Value



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## Interpretation

**High yeast/fungal metabolites** indicate a yeast/fungal overgrowth of the gastrointestinal tract. Prescription or natural (botanical) anti-fungals, along with supplementation of high potency multi-strain probiotics (20-50 billion cfu's), may reduce yeast/fungal levels.

**High 4-hydroxybenzoic acid and/or 4-hydroxyhippuric acid** may be due to bacterial overgrowth of the GI tract, intake of fruits containing polyphenols rich in anthocyanins, flavonols, and hydroxycinnamates such as blueberries, or from paraben exposure. 4-Hydroxybenzoic acid may be excreted as its glycine conjugate 4-hydroxyhippuric acid. High levels of these paraben metabolites in urine (>10 mmol /mol creatinine) may result from excessive exposure to parabens. Parabens are 4-hydroxybenzoic acid alkyl esters with antimicrobial properties. Parabens are common preservatives allowed in foods, drugs, cosmetics and toiletries, but also they have a long history of use in a variety of pharmaceutical products intended for either injection, inhalation, oral, topical, rectal or vaginal administration. Parabens were considered to be safe because of their low toxicity profile and their long history of safe use; however, recent studies have begun to challenge this view. In 1998, Routledge *et al.* (Toxicol. Appl. Pharmacol. 153,12-19), reported parabens having estrogenic activity *in vitro*. A number of *in vivo* studies have further elucidated potential endocrine disruption by parabens that may affect reproduction or promote tumor growth. Parabens have been found at high levels in breast cancer biopsies, although a definitive relationship with breast cancer has not been demonstrated. Parabens may contribute to mitochondrial failure by uncoupling oxidative phosphorylation and depleting cellular ATP. 4-Hydroxyhippuric acid has been found to be an inhibitor of Ca<sup>2+</sup>-ATPase in end-stage renal failure. Some individuals experience skin reactions as most parabens are readily and completely absorbed through the skin and from the gastrointestinal tract. Eliminate all sources of parabens and use sauna therapy, the Hubbard detoxification protocol employing niacin supplementation, or glutathione supplementation (oral, intravenous, transdermal, or precursors such as N-acetyl cysteine [NAC]) to accelerate paraben excretion.

**High hippuric acid** may derive from food, from the action of gastrointestinal bacteria, or from exposure to the chemical solvent toluene. Hippuric acid is a conjugate of glycine and benzoic acid formed in the liver. Most hippuric acid in urine is derived from microbial breakdown of chlorogenic acid to benzoic acid, a common substance found in beverages and in many fruits and vegetables, including apples, pears, tea, coffee, sunflower seeds, carrots, blueberries, cherries, potatoes, tomatoes, eggplant, sweet potatoes, and peaches. Benzoic acid is also a food preservative and present in high amounts in cranberry juice. The workplace is the most common source of toluene exposure, but toluene may be absorbed from outgassing of new carpets and other building materials or absorbed during recreational abuse of solvents such as glue-sniffing. Because most of the hippuric acid in urine is from gastrointestinal sources, this marker is a poor indicator of toluene exposure and is being replaced by other markers in occupational safety testing. Bacterial overgrowth can be treated with natural anti-bacterial agents and/or 30-50 billion cfu's of probiotics that include *Lactobacillus rhamnosus*.

**High succinic acid** may indicate a relative deficiency of riboflavin and/or coenzyme Q10 that are needed to supply cofactors for succinic dehydrogenase in the Krebs cycle. Suggest supplementation with a minimum of 20mg riboflavin (which could be provided through a high quality multivitamin) and/or 50 mg per day of coenzyme Q10.

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**High oxalic, glyceric or glycolic acid** may be found in the genetic hyperoxalurias, in autism, in women with vulvar pain, in fibromyalgia, and may also be due to vitamin C overuse. However, kidney stone formation from oxalic acid was not correlated with vitamin C intake in a very large study. Besides being present in varying concentrations in most vegetables and fruits, oxalates, the mineral conjugate base forms of oxalic acid, are also byproducts of molds such as *Aspergillus* and *Penicillium* and probably *Candida*. If yeast or fungal markers are elevated, antifungal therapy may reduce excess oxalates. High oxalates may cause anemia that is difficult to treat, skin ulcers, muscles pains, and heart abnormalities. Elevated oxalic acid is also found in anti-freeze (ethylene glycol) poisoning and is a toxic metabolite of trichloroacetic acid or other compounds from environmental sources.

Elevated oxalate values with concomitant increases in glycolic acid may indicate genetic hyperoxaluria (type I) whereas increased glyceric acid may indicate a genetic hyperoxaluria (type II). Increased oxalic acid with normal values of these metabolites rules out a genetic cause for elevated oxalate. Elevated glycolic or glyceric acids with normal oxalate levels appears not to have an important clinical significance.

Regardless of its source, high oxalic acid may contribute to kidney stones and may also reduce ionized calcium. Oxalic acid absorption from the gastrointestinal tract may be reduced by supplementation with calcium citrate before meals. Vitamin B-6, arginine, vitamin E, chondroitin sulfate, taurine, selenium, omega-3 fatty acids and/or N-acetyl glucosamine supplements may also be useful to reduce oxalates and/or their toxicity. Excessive fats in the diet may cause elevated oxalate if fatty acids are poorly absorbed because of bile salt deficiency. Nonabsorbed free fatty acids bind calcium to form insoluble soaps, reducing calcium's ability to bind oxalate and increase its absorption. If taurine is low in a plasma amino acid profile, supplementation with taurine may help stimulate bile salt production (taurocholic acid), leading to better fatty acid absorption and diminished oxalate absorption.

Bone tends to be the major repository of excess oxalate in patients with primary hyperoxaluria. Bone oxalate levels are negligible in healthy subjects. Oxalate deposition in the skeleton tends to increase bone resorption and decrease osteoblast activity.

Oxalates may also be deposited in the kidneys, joints, eyes, muscles, blood vessels, brain, and heart and may contribute to muscle pain in fibromyalgia. Oxalate crystal formation in the eyes may be a source of severe eye pain in individuals with autism who may exhibit eye-poking behaviors. High oxalates in the gastrointestinal tract also may significantly reduce absorption of essential metals such as calcium, magnesium, zinc, and others.

A low oxalate diet may also be useful in the reduction of body oxalates even when dysbiosis of GI flora is the major source of oxalates. Foods especially high in oxalates include spinach, beets, chocolate, soy, peanuts, wheat bran, tea, cashews, pecans, almonds, berries, and many others. A complete list of high oxalate foods is available online

**High malic acid** indicates a greater requirement for nutrients such as niacin and CoQ10. If malic acid is simultaneously elevated with citric, fumaric and alpha-ketoglutaric acids, Cytochrome C Oxidase deficiency is strongly suggested, indicating mitochondrial energy pathway dysfunction.

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**High quinolinic acid** may be a sign of neural excitotoxicity. Quinolinic acid is derived from the amino acid tryptophan and is neurotoxic at high levels. Excitotoxic substances like quinolinic acid may stimulate nerve cells so much that the nerve cells die. Brain toxicity due to quinolinic acid has been implicated in Alzheimer's disease, autism, Huntington's disease, stroke, dementia of old age, depression, HIV-associated dementia, and schizophrenia. However, quinolinic acid is an important intermediate that the body uses to make the essential nutritional cofactor nicotinamide adenine dinucleotide (NAD), which can also be derived from niacin (B3). Quinolinic acid is an excitotoxic stimulant of certain brain cells that have NMDA-type receptors. It is also a metal chelator, and an inhibitor of enzymes that allow the body to produce the sugar glucose when it is low. High levels of quinolinic acid may inhibit heart contractions, cause lipid peroxidation in the brain, and cause increased apoptosis (programmed cell death) of human astrocytes in the brain. In addition, phthalates inhibit the conversion of quinolinic acid to NAD. Excessive immune stimulation, resulting in overproduction of cytokines like interferon, also stimulates the overproduction of quinolinic acid.

Treatment of excessive levels of quinolinic acid can be achieved by multiple approaches: lowering tryptophan supplements, preventing repeated infections and subsequent immune overstimulation by the use of colostrum, transfer factor and probiotics, reducing the use of immune modulators like interferon that increase quinolinic acid production, reducing the numbers of vaccines given at one time or increasing the time interval between vaccines. In addition, the drug deprenyl or the dietary supplements carnitine, melatonin, capsaicin, turmeric (curcumin) and garlic can reduce brain damage caused by quinolinic acid. Niacin (nicotinic acid) and niacinamide may also reduce quinolinic acid production by decreasing tryptophan shunting to the quinolinic acid pathway. A high quinolinic acid/ 5-hydroxyindoleacetic acid ratio or high quinolinic/kynurenic acid ratio would be indicative of immune over stimulation and/or phthalate toxicity.

**High kynurenic acid** may be a result of vitamin B-6 (pyridoxine) deficiency, immune stimulation and/or ingestion of tryptophan supplements. Tryptophan is metabolized via several pathways, the main one being the kynurenine pathway. Kynurenic acid may also be elevated in vitamin B-6 (pyridoxine) deficiency, but pyridoxic acid, the major metabolite of B-6, is a much better marker of deficiency. A central compound of the pathway is kynurenine (KYN), which can be metabolized in two separate ways: one branch resulting in kynurenic acid, and the other quinolinic acid, the precursor of the coenzyme NAD. Kynurenic acid is one of the few known endogenous excitatory amino acid receptor antagonists to alpha 7-nicotinic acetylcholine receptors and N-methyl-D-aspartate (NMDA) receptors. Kynurenic acid has proven to be neuroprotective in several experimental settings against the neurotoxic effects of quinolinic acid, a specific agonist of NMDA receptors and a potent producer of toxic free radicals. The pathogenesis of a number of neurodegenerative disorders has been demonstrated to involve multiple imbalances of kynurenine pathway metabolism, including Alzheimer's disease, Parkinson disease, multiple sclerosis, and amyotrophic laterosclerosis (ALS). Extremely high amounts of kynurenic acid in urine may be due to the genetic disorder kynureninase deficiency, which can be confirmed by DNA testing. Common clinical features of kynureninase deficiency include photosensitive rash, ataxia, and mental abnormality. If the genetic disease is present, the conversion of tryptophan to niacin may be impaired. Patients with this genetic disorder or who simply produce excessive kynurenic acid for other reasons may respond favorably to niacin supplements.

**High quinolinic acid/ 5-HIAA ratio** indicates an imbalance of these organic acids. An elevated ratio is not specific for a particular medical condition and may be commonly associated with excessive inflammation due to recurrent infections, or excessive tryptophan intake. Immune overstimulation, excess adrenal production of cortisol due to stress, or high exposure to phthalates may also increase the quinolinic acid/5-HIAA ratio.

**High quinolinic acid/kynurenic acid ratio** indicates an imbalance of these organic acids. An elevated ratio is not specific for a particular medical condition and may be commonly associated with excessive inflammation due to recurrent infections, or excessive tryptophan intake. Immune overstimulation, excess adrenal production of cortisol due to stress, or high exposure to phthalates may also increase the quinolinic acid/kynurenic acid ratio.

**High ethylmalonic, methylsuccinic, adipic, suberic, or sebacic acids** may be due to fatty acid oxidation disorders, carnitine deficiency, fasting, or to increased intake of the medium-chain triglycerides found in coconut oil, MCT oil, and some infant formulas. The fatty acid oxidation defects are associated with hypoglycemia, apnea episodes, lethargy, and coma. [An acyl carnitine profile (Duke University Biochemical Genetics Laboratory, <http://medgenetics.pediatrics.duke.edu>) can rule out fatty acid oxidation defects.] Regardless of cause, supplementation with L-carnitine or acetyl-L-carnitine (500-1000 mg per day) may be beneficial.

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**High pantothenic acid** indicates high recent intake of pantothenic acid. Pantothenic acid is an essential B vitamin. Since some individuals may require very high doses of pantothenic acid, high values do not necessarily indicate the need to reduce pantothenic acid intake.

**High glutaric acid** can result from glutaric acidemias, fatty acid oxidation defects, riboflavin deficiency, ingestion of medium-chain triglycerides, metabolic effects of valproic acid (Depakene), and celiac disease. The genetic disorders are usually diagnosed in children but have occasionally been detected in adults. The probability of a genetic disease is higher when values exceed 10 mmol/mol creatinine but such diseases may also be present with lower urine values. DNA tests have been developed for the confirmation of both types of genetic disorders but may not be commercially available. This compound may be elevated in about 10% of children with autism. Regardless of the cause, supplementation with riboflavin (20-100mg) and coenzyme Q10 (50-100 mg per day) may be helpful.

Glutaric acidemia type I is associated with elevations of 3-hydroxyglutaric and glutaconic acid. Normal values of 3-hydroxyglutaric acid greatly reduce but do not completely eliminate the possibility of glutaric acidemia type I. This disease has been associated with clinical symptoms ranging from near normal to encephalopathy, cerebral palsy, and other neurological abnormalities. Some individuals with glutaric acidemia type I have developed bleeding in the brain or eyes that may be mistaken for the effects of child abuse. Treatment of this disorder includes special diets low in lysine and carnitine supplementation.

Glutaric acidemia type II, also called acyl-CoA dehydrogenase deficiency, caused by a genetic defect in one of the mitochondrial electron transport proteins, is associated with dysmorphic features, seizures, hypoglycemia, and developmental delay. Glutaric acidemia II is commonly associated with elevations of 2-hydroxyglutaric acid as well as isovalerylglycine, hexanoylglycine, isobutyrylglycine, ethylmalonic acid, methylsuccinic acid, and adipic, suberic, and sebatic acids.

**High ascorbic acid** (vitamin C) may be elevated as a result of supplementation. An elevated value of ascorbic acid does not mean that this amount of vitamin C is not nutritionally desirable.

**High 3-methylglutaric and/or high 3-methylglutaconic acids** may be due to reduced capacity to metabolize the amino acid leucine. This abnormality is found in the genetic disease methylglutaconic aciduria and in mitochondrial disorders in which there are severe deficiencies of the respiratory complexes (Complex I, NADH ubiquinone oxidoreductase and complex IV, cytochrome c oxidase). Typical results found in genetic defects are above 10 mmol/mol creatinine. A few non-generic conditions including pregnancy and kidney failure may also produce elevation of these organic acids in urine. Confirmation of the genetic disease requires enzymes and/or DNA testing. Multiple genetic defects can cause the biochemical abnormality. Confirmation of mitochondrial disorder usually requires tissue biopsy for mitochondria testing. Symptoms differ with the different types of genetic disorders but in severe cases may include speech delay, delayed development of both mental and motor skills (psychomotor delay), metabolic acidosis, abnormal muscle tone (dystonia), and spasms and weakness affecting the arms and legs (spastic quadriplegia). Recommendations include supplementation with coenzyme Q-10 (300-600 mg), NAD 25-50mg, L-carnitine and acetyl-L-carnitine (1000-2000 mg), riboflavin (40-80 mg), nicotinamide (40-80 mg), biotin (4-8 mg), and vitamin E (200-400 IU's) per day.

**High phosphoric acid or its base conjugate phosphate** is associated with hyperparathyroidism, vitamin D-resistant rickets, immobilization following paraplegia or fracture due to bone resorption, vitamin D intoxication, blood lead levels above 1.5 ppm, renal tubular damage, familial hypophosphatemia, high nutritional intake of phosphate, and metabolic acidosis. Phosphate excretion is directly proportional to dietary intake. Foods high in phosphate include sodas, candy, ice cream, chocolate, mayonnaise, frozen pizza and processed cakes, cookies and meats. Phosphate excretion is diurnal with lowest values occurring in the early morning.

*Certain uses of the compounds arabinose, citramalic, tartaric, 3-oxoglutaric, carboxycitric, 3,4-dihydroxyphenylpropionic acid, and 3-(3-hydroxyphenyl)-3-hydroxypropionic acid in their application to autism in the Organic Acid Test and Microbial Organic Acid Test are protected by USA patent 5,686,311 granted to The Great Plains Laboratory, Inc., November 11, 1997.*