The Great Plains Laboratory, Inc.

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Requisition #:	386879	***Physician:***	JAMES NEUBRANDER
Patient Name:	Finn Schureman	***Date of Collection:***	5/14/2015
Patient Age:	2	***Time of Collection:***	00:00 AM
Patient Sex:	M	***Print Date:***	05/22/2015

Organic Acids Test - Nutritional and Metabolic Profile

Metabolic Markers in Urine	Reference Range *(mmol/mol creatinine)*		Patient Value	Reference Population - Males Under Age 13
Intestinal Microbial Overgrowth				
Yeast and Fungal Markers				
1 Citramalic	≤ 5.0	H	6.0	6.0
2 5-Hydroxymethyl-2-furoic	≤ 28		4.9	4.9
3 3-Oxoglutaric	≤ 0.46		0	0.00
4 Furan-2,5-dicarboxylic	≤ 18		4.2	4.2
5 Furancarbonylglycine	≤ 3.1		0.43	0.43
6 Tartaric	≤ 6.5		1.1	1.1
7 Arabinose	≤ 50	H	373	373
8 Carboxycitric	≤ 25		0	0.00
9 Tricarballylic	≤ 1.3		0.23	0.23
Bacterial Markers				
10 Hippuric	≤ 680		166	166
11 2-Hydroxyphenylacetic	≤ 0.86	H	0.94	0.94
12 4-Hydroxybenzoic	≤ 3.0	H	7.9	7.9
13 4-Hydroxyhippuric	≤ 30	H	105	105
14 DHPPA (Beneficial Bacteria)	≤ 0.59		0.53	0.53
Clostridia Bacterial Markers				
15 4-Hydroxyphenylacetic 2.0 - 32 *(C. difficile, C. stricklandii, C. lituseburense & others)*			28	28
16 HPHPA *(C. sporogenes, C. caloritolerans, C. botulinum & others)*	≤ 220	H	645	645
17 4-Cresol *(C. difficile)*	≤ 84		19	19
18 3-Indoleacetic 0.60 - 14 *(C. stricklandii, C. lituseburense, C. subterminale & others)*		H	20	20

Testing performed by The Great Plains Laboratory, Inc., Lenexa, Kansas. The Great Plains Laboratory has developed and determined the performance characteristics of this test. This test has not been evaluated by the U.S. FDA; the FDA does not currently regulate such testing.

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Metabolic Markers in Urine		Reference Range *(mmol/mol creatinine)*			Patient Value	Reference Population - Males Under Age 13
Oxalate Metabolites						
19	Glyceric	0.74	- 13		12	12
20	Glycolic	27	- 221		42	42
21	Oxalic	35	- 185	H	476	476
Glycolytic Cycle Metabolites						
22	Lactic	2.6	- 48		39	39
23	Pyruvic	0.32	- 8.8		6.8	6.8
Mitochondrial Markers - Krebs Cycle Metabolites						
24	Succinic		≤ 23	H	36	36
25	Fumaric		≤ 1.8		1.1	1.1
26	Malic		≤ 2.3		0.43	0.43
27	2-Oxoglutaric		≤ 96		78	78
28	Aconitic	9.8	- 39		13	13
29	Citric		≤ 597	H	1 261	1261
Mitochondrial Markers - Amino Acid Metabolites						
30	3-Methylglutaric	0.01	- 0.97	H	1.0	1.0
31	3-Hydroxyglutaric		≤ 16		2.4	2.4
32	3-Methylglutaconic		≤ 6.9		2.8	2.8
Neurotransmitter Metabolites						
Phenylalanine and Tyrosine Metabolites						
33	Homovanillic (HVA) *(dopamine)*	0.49	- 13		10.0	10
34	Vanillylmandelic (VMA) *(norepinephrine, epinephrine)*	0.72	- 6.4		3.6	3.6
35	HVA / VMA Ratio	0.23	- 2.8		2.8	2.8
Tryptophan Metabolites						
36	5-Hydroxyindoleacetic (5-HIAA) *(serotonin)*		≤ 11		3.8	3.8
37	Quinolinic	0.48	- 8.8		3.0	3.0
38	Kynurenic		≤ 4.2		0.52	0.52
39	Quinolinic / 5-HIAA Ratio		≤ 2.5		0.79	0.79

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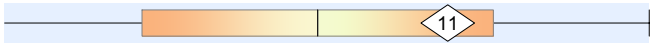
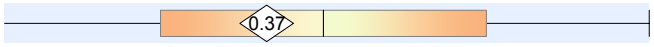
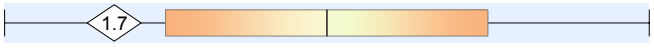
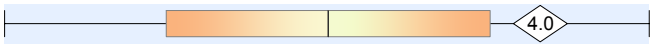
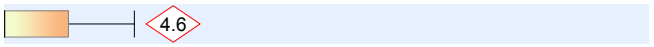
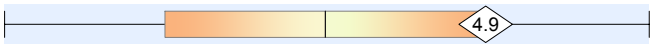
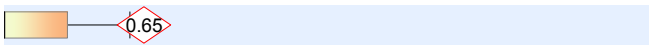
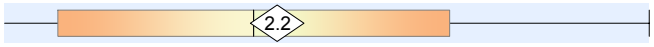
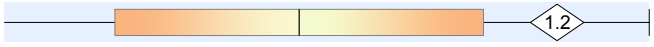
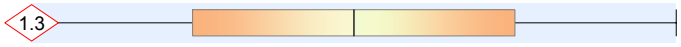
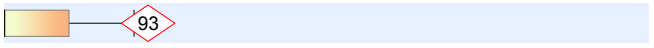
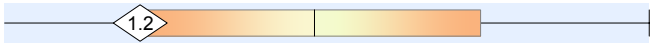
Metabolic Markers in Urine		Reference Range *(mmol/mol creatinine)*		Patient Value	Reference Population - Males Under Age 13
Pyrimidine Metabolites - Folate Metabolism					
40	Uracil	≤ 16		11	
41	Thymine	≤ 0.91		0.37	
Ketone and Fatty Acid Oxidation					
42	3-Hydroxybutyric	≤ 4.8		2.1	
43	Acetoacetic	≤ 10		1.7	
44	4-Hydroxybutyric	≤ 4.7		1.3	
45	Ethylmalonic	0.06 - 4.8		4.0	
46	Methylsuccinic	≤ 4.0	H	4.6	
47	Adipic	0.19 - 6.5		4.9	
48	Suberic	≤ 7.0	H	14	
49	Sebacic	≤ 0.61	H	0.65	
Nutritional Markers					
Vitamin B12					
50	Methylmalonic ✱	≤ 5.2		2.2	
Vitamin B6					
51	Pyridoxic (B6)	≤ 53		4.5	
Vitamin B5					
52	Pantothenic (B5)	≤ 14		11	
Vitamin B2 (Riboflavin)					
53	Glutaric ✱	≤ 1.4		1.2	
Vitamin C					
54	Ascorbic	10 - 200	L	1.3	
Vitamin Q10 (CoQ10)					
55	3-Hydroxy-3-methylglutaric ✱	≤ 88	H	93	
Glutathione Precursor and Chelating Agent					
56	N-Acetylcysteine (NAC)	≤ 0.34		0.12	
Biotin (Vitamin H)					
57	Methylcitric ✱	≤ 5.7		1.2	

✱ ***A high value for this marker may indicate a deficiency of this vitamin.***

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Metabolic Markers in Urine		Reference Range (mmol/mol creatinine)			Patient Value	Reference Population - Males Under Age 13
Indicators of Detoxification						
Glutathione						
58	Pyroglutamic ✱	13	- 62		55	55
59	2-Hydroxybutyric ✱	0.19	- 2.0		1.0	1.0
Ammonia Excess						
60	Orotic	0.04	- 0.80		0.39	0.39
Aspartame, salicylates, or GI bacteria						
61	2-Hydroxyhippuric		≤ 1.2	H	2.2	2.2
Amino Acid Metabolites						
62	2-Hydroxyisovaleric		≤ 0.55		0	0.00
63	2-Oxoisovaleric		≤ 2.5		0	0.00
64	3-Methyl-2-oxovaleric		≤ 1.1		0	0.00
65	2-Hydroxyisocaproic		≤ 0.68		0.08	0.08
66	2-Oxoisocaproic		≤ 0.46		0	0.00
67	2-Oxo-4-methiolbutyric		≤ 0.33		0.17	0.17
68	Mandelic		≤ 0.30		0	0.00
69	Phenyllactic		≤ 0.19		0.17	0.17
70	Phenylpyruvic		≤ 4.0		1.1	1.1
71	Homogentisic		≤ 0.61		0.25	0.25
72	4-Hydroxyphenyllactic	0.05	- 1.1		0.44	0.44
73	N-Acetylaspartic		≤ 5.9		0	0.00
74	Malonic		≤ 18		2.8	2.8
Mineral Metabolism						
75	Phosphoric	1 000	- 7 300		3 314	3314

✱ ***A high value for this marker may indicate a Glutathione deficiency.***

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Indicator of Fluid Intake

76	*Creatinine	45	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 ± 2SD of the mean. Reference ranges are age and gender specific, consisting of Male Adult (≥13 years), Female Adult (≥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.

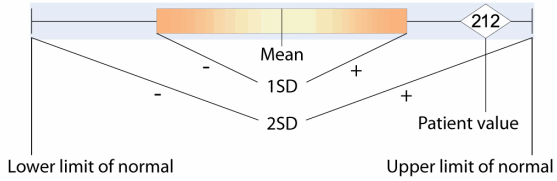
The first graph will occur when the value of the patient is within the reference (normal) range, defined as the mean plus or minus two standard deviations.

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.

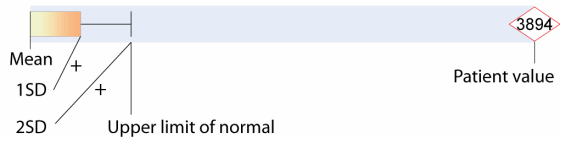
Example of Value Within Reference Range

Metabolic Markers in Urine	Reference Range (mmol/mol creatinine)		Patient Result	Reference Range - Males Age 13 and Under
Intestinal Microbial Overgrowth				
HPHPA (Clostridia marker)	<	219 .9	212	

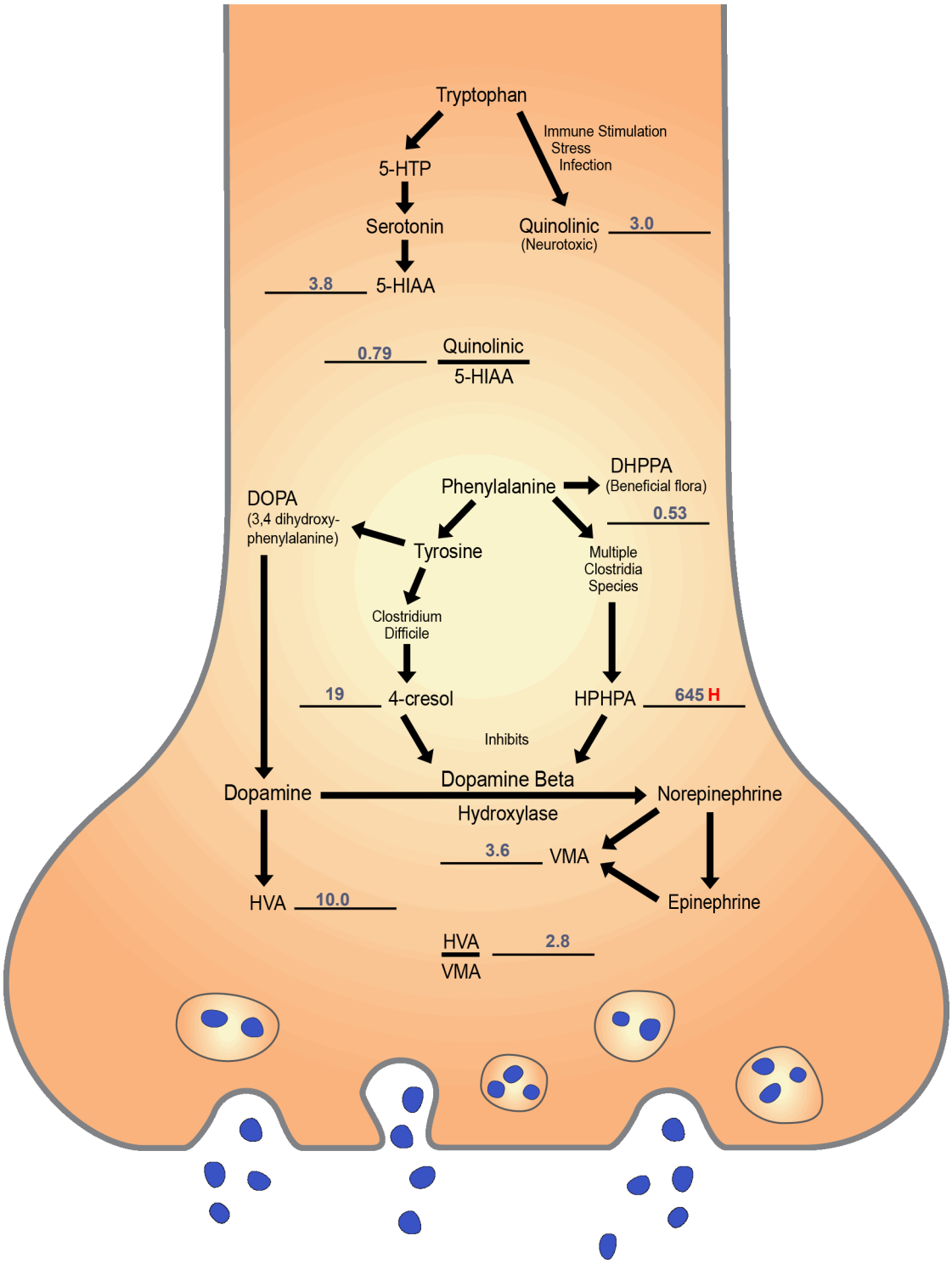


Example of Elevated Value

Metabolic Markers in Urine	Reference Range (mmol/mol creatinine)		Patient Result	Reference Range - Males Age 13 and Under
Intestinal Microbial Overgrowth				
HPHPA (Clostridia marker)	<	219 .9	H 3894	



Neurotransmitter Metabolism Markers



The diagram contains the patient's test results for neurotransmitter metabolites and shows their relationship with key biochemical pathways within the axon terminal of nerve cells. The effect of microbial byproducts on the blockage of the conversion of dopamine to norepinephrine is also indicated.

Interpretation

High yeast/fungal metabolites (Markers 1,2,3,4,5,6,7,8) 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 2-hydroxyphenylacetic acid (Marker 11) is associated with intestinal bacteria overgrowth and with the genetic disease phenylketonuria (PKU).

High 4-hydroxybenzoic acid and/or 4-hydroxyhippuric acid (Markers 12,13) may be due to bacterial overgrowth of the GI tract, intake of fruits such as blueberries rich in polyphenols (anthocyanins, flavonols, and hydroxycinnamates), or may be from paraben additive exposure. Parabens are 4-hydroxybenzoic acid alkyl esters with antimicrobial properties. 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 common preservatives allowed in foods, drugs, cosmetics and toiletries, but they also have a long history of use in a variety of pharmaceutical products for injection, inhalation, oral, topical, rectal or vaginal administration. Some individuals experience skin reactions as most parabens are readily and completely absorbed through the skin and the GI tract. Parabens have been considered safe because of their low toxicity profile and their long history of safe use; however, recent studies 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 affecting 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 Ca2+- ATPase in end-stage renal failure. Eliminate all sources of parabens. To accelerate paraben excretion, use sauna therapy, the Hubbard detoxification protocol employing niacin supplementation, or glutathione supplementation (oral, intravenous, transdermal, or precursors such as N-acetyl cysteine [NAC]).

High HPHPA (3-(3-hydroxyphenyl)-3-hydroxypropionic acid) (Marker 16) is an abnormal phenylalanine metabolite produced when byproducts of *Clostridium* bacteria combine with human metabolites. High concentrations of this compound cause abnormal behavior by inhibiting metabolism of dopamine to epinephrine, resulting in high levels of the dopamine metabolite homovanillic acid (HVA) in the urine and insufficient epinephrine/norepinephrine in the body. It is associated with behavioral, gastrointestinal, and neuropsychiatric symptoms including tic disorders, depression, autism, schizophrenia, aggression, seizures, anorexia, obsessive compulsive disorder, and hyperactivity. Neuropsychiatric effects are more common when values exceed 500 mmol/mol creatinine.

The *Clostridia* species that cause the greatest quantities of urinary HPHPA are *C. sporogenes, C. caloritolerans,* and *C. botulinum*. Additionally, *C. mangenoti, C. ghoni, C. bifermentans, C. caproicum, and C. sordellii* are also capable of causing elevated urinary levels of HPHPA.

HPHPA precursors are **not** produced by *C.perfringens* -types A-F*, C.tetani, C.subterminale C.capitovale, C.septicum, C.difficile, C.histolyticum,* or *C.tertium.*

C. botulinum would appear to be an unlikely source unless clinical symptoms of botulism are present. The botulinum toxin can cause a severe flaccid paralytic <http://en.wikipedia.org/wiki/Flaccid_paralysis> disease in humans and animals and is the most potent toxin known to humankind, with a lethal dose of less than 1 µg in humans. Symptoms of botulism include weakness, impaired vision, fatigue, and impaired speech. This may then be followed by weakness of the arms, chest muscles and legs. Surprisingly, symptoms may sometimes be mild and the severity of symptoms appears to be modulated by the amount of beneficial flora in the intestinal tract. In food borne botulism, symptoms generally begin 18 to 36 hours after eating contaminated food, but they can occur as early as 6 hours or as late as 10 days. *C. caloritolerans* is so named because it can survive at the boiling point for 8 hours. Its extreme resistance to heat may allow common food borne transmission. *C. sporogenes* is the name given to strains of *Clostridium botulinum* that do not produce botulinum <http://en.wikipedia.org/wiki/Botulinum> neurotoxins. *C. sporogenes* differs from C. botulinum by a single gene. C. sporogenes is ubiquitous in nature and is commonly found in the flora of humans. *C. sordellii* can be pathogenic and has been implicated in fatal toxic shock syndrome among women of child bearing age.

Treatment with Metronidazole or Vancomycin is almost 100% effective in killing parent *Clostridia* organisms but not their spores. At least three months of probiotic therapy is recommended after antimicrobial treatment due to spore formation by *Clostridia* species. *Clostridia* overgrowth can sometimes be controlled by supplementation with *Lactobacillus rhamnosus GG* (Culturelle) or *Saccharomyces boulardii.* Phenalalanine or tyrosine supplements should be avoided because of the possibility of conversion to HPHPA or other toxic byproducts.

High 3-Indoleacetic acid (Marker 18) is a byproduct of the Clostridia species *C. stricklandii, C. lituseburense, C. subterminale, and C. putrefaciens* in the GI tract. No information on the pathogenicity of these species is available. This compound can be elevated in Hartnup's disease, a genetic disease involving a defect in the renal and intestinal transport of neutral amino acids, which are excreted in amounts 5-20x normal in the urine. The elevated urine amino acids in this disease include: alanine, serine, threonine, valine, leucine, isoleucine, phenylalanine, tyrosine, tryptophan, histidine, asparagine, and glutamine. Urinary acidic and basic amino acids, along with proline and hydroxyproline, are usually normal in this disorder, and plasma amino acids are close to normal. Clinical symptoms may include skin rash and neuropsychiatric symptoms such as ataxia, psychotic behavior, seizures and depression. If Hartnup's disease has been determined, treatment with high doses of the vitamin niacin (50-300 mg per day) is recommended. Reference: *The Metabolic Basis of Inherited Disease, Sixth Edition*, Vol. 2, pp. 2515-2527, 1989.

High oxalic with or without elevated glyceric or glycolic acids (Markers 19,20,21) may be associated with the genetic hyperoxalurias, autism, women with vulvar pain, fibromyalgia, and may also be due to high vitamin C intake. 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 the result of anti-freeze (ethylene glycol) poisoning. Oxalic acid is a toxic metabolite of trichloroacetic acid and other environmental pollutants. In addition, decomposing vitamin C may form oxalates during transport or storage.

Elevated oxalate values with a concomitant increase in glycolic acid may indicate genetic hyperoxaluria (type I), whereas increased glyceric acid may indicate a genetic hyperoxaluria (type II). Elevated oxalic acid with normal levels of glyceric or glycolic metabolites rules out a genetic cause for high oxalate. However, elevated oxalates may be due to a new genetic disorder, hyperoxaluria type III.

Regardless of its source, high oxalic acid may contribute to kidney stones and may also reduce ionized calcium. Oxalic acid absorption from the GI tract may be reduced by calcium citrate supplementation before meals. Vitamin B6, arginine, vitamin E, chondroitin sulfate, taurine, selenium, omega-3 fatty acids and/or N-acetyl glucosamine supplements may also 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. Unabsorbed 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 (1000 mg/day) may help stimulate bile salt production (taurocholic acid), leading to better fatty acid absorption and diminished oxalate absorption.

High levels of oxalates are common in autism. Malabsorption of fat and intestinal *Candida* overgrowth are probably the major causes for elevated oxalates in this disorder. Even individuals with elevated glyceric or glycolic acids may not have a genetic disease. To rule out genetic diseases in those people with abnormally high markers characteristic of the genetic diseases, do the following steps: (1) Follow the nutritional steps indicated in this interpretation for one month; (2) If *Candida* is present, treat *Candida* for at least one month; (3) Repeat the organic acid test after abstaining from vitamin C supplements for 48 hours; (4) If the biochemical markers characteristic of genetic oxalate disorders are still elevated in the repeat test, consider DNA tests for the most common mutations of oxalate metabolism. DNA testing for type I hyperoxaluria is available from the Mayo Clinic, Rochester, MN as test #89915 "*AGXT* Gene, Full Gene Analysis" and, for the p.Gly170Arg mutation only, as # 83643 "Alanine:Glyoxylate Aminotransferase [*AGXT*] Mutation Analysis [G170R], Blood"). Another option to confirm the genetic disease is a plasma oxalate test, also available from the Mayo Clinic (Phone 507.266.5700). Plasma oxalate values greater than 50 micromol/L are consistent with genetic oxalate diseases and may serve as an alternate confirmation test.

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 GI tract also may significantly reduce absorption of essential minerals such as calcium, magnesium, zinc, and others.

A low oxalate diet may also be particularly useful in the reduction of body oxalates even if 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 at <http://www.greatplainslaboratory.com/home/eng/oxalates.asp>.

High succinic acid (Marker 24) may indicate a relative deficiency of riboflavin and/or coenzyme Q10 (cofactors for succinic dehydrogenase in the Krebs cycle). Supplementation with a minimum of 20 mg riboflavin (which could be provided through a high quality multivitamin) and/or 50 mg/day of coenzyme Q10 is recommended. Clinical observation suggests that succinic acid levels also decrease after treatment for GI dysbiosis.

High citric acid (Marker 29) may be due to increased intake of citric acid-containing foods or as a result of intestinal yeast that either produce citric acid or perhaps inhibit the human citric acid cycle. Increased citric acid may also indicate depletion of glutathione, which is required for the enzyme aconitase to metabolize both aconitic and citric acids. If pyroglutamic acid is also high, consider supplements of reduced glutathione, n-acetyl cysteine (NAC), or lipoic acid.

High 3-methylglutaric and/or high 3-methylglutaconic acids (Markers 30,32) 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.). Small elevations may be due to impairment of mitochondrial function and may respond to the recommended supplements below. Typical results found in genetic defects are above 10 mmol/mol creatinine. A few non-genetic 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 within 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 quadriparesis). 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.

5-hydroxyindoleacetic acid (5-HIAA) levels below the mean (Marker 36) may indicate lower production of the neurotransmitter serotonin. 5-hydroxy-indoleacetic acid is a metabolite of serotonin. Low values have been correlated with symptoms of depression. Supplementation with the precursor 5-HTP (5-hydroxytryptophan) at 50-300 mg/day may be beneficial. Supplementation with tryptophan itself may form the neurotoxic metabolite quinolinic acid, however, 5-HTP is not metabolized to quinolinic acid. Excessive tryptophan supplementation has been associated with eosinophilia myalgia syndrome.

High ethylmalonic, methylsuccinic, adipic, suberic, or sebacic acids (Markers 45,46,47,48,49) 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.

Pyridoxic acid (B6) levels below the mean (Marker 51) may be associated with less than optimum health conditions (low intake, malabsorption, or dysbiosis). Supplementation with B6 (20 - 50 mg/day) or a multivitamin may be beneficial.

Ascorbic acid (vitamin C) levels below the mean (Marker 54) may indicate a less than optimum level of the antioxidant vitamin C. Suggested supplementation is 1000 mg/day of buffered vitamin C, divided into 2-3 doses.

High 3-hydroxy-3-methylglutaric acid (Marker 55) is seen in the genetic disease 3-hydroxy 3-methylglutaric aciduria. Typical values observed in the genetic disease are 200-11,000mmol/mol creatinine. The cause of less significant increases in this urinary metabolite is unknown. 3-Hydroxy-3-methylglutaric aciduria may cause vomiting, lethargy, hypotonia, and apnea, sometimes evolving to coma. Laboratory tests reveal metabolic acidosis with severe hypoketotic hypoglycemia on fasting or during acute illness, hyperammonemia, and abnormal liver function. Preliminary diagnosis is based on a pattern of organic acids in urine which includes 3-hydroxy-3-methylglutaric, 3-hydroxyisovaleric, 3-methylglutaconic, 3-methylglutaric, and 3-methylcrotonic acids. Because yeast also produces this compound and yeast metabolites are frequently elevated along with this compound; slight increases may be yeast-related. Reduced activity of 3-hydroxy 3-methylglutaryl Co A reductase, a critical enzyme at the beginning of the cholesterol synthesis pathway, may also elevate this compound. Check cholesterol values when this compound is elevated up to 300 mmol/mol creatinine. Slight elevations may result from coenzyme Q10 deficiency. Supplementation with coenzyme Q10 at 50 - 120 mg/day may be beneficial.

High 2-hydroxyhippuric acid (Marker 61) may result after ingestion of aspartame (Nutrasweet®) or salicylates (aspirin), or from GI bacteria converting tyrosine or phenylalanine to salicylic acid. 2-Hydroxyhippuric acid is a conjugate of hydroxybenzoic acid (salicylic acid) and glycine.

Low values for amino acid metabolites (Markers 62-74) indicate the absence of genetic disorders of amino acid metabolism. These markers are deamination (ammonia removed) byproducts that are very elevated only when a key enzyme has low activity; slight elevations may indicate a genetic variation or heterozygous condition which may be mitigated with diet or supplementation. Low values are not associated with inadequate protein intake and have not been proven to indicate specific amino acid deficiencies.

High quality nutritional supplements can be purchased through your practitioner or at New Beginnings Nutritionals, www.NBNUS.com <http://www.NBNUS.com> , or call 877-575-2467.

The nutritional recommendations in this test are not approved by the US FDA. Supplement recommendations are not intended to treat, cure, or prevent any disease and do not take the place of medical advice or treatment from a healthcare professional.

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.