The Great Plains Laboratory, Inc.

William Shaw, Ph.D., Director | 11813 West 77th Street, Lenexa, KS 66214 | (913) 341-8949 | Fax (913) 341-6207

Requisition #:	430628	***Physician:***	BIOLAB
Patient Name:		***Date of Collection:***	3/10/2016
Patient Age:		***Time of Collection:***	07:30 AM
Patient Sex:		***Print Date:***	03/23/2016

Organic Acids Test - Nutritional and Metabolic Profile

Metabolic Markers in Urine	Reference Range *(mmol/mol creatinine)*		Patient Value	Reference Population - Females Age 13 and Over
Intestinal Microbial Overgrowth				
Yeast and Fungal Markers				
1 Citramalic	≤ 3.6		1.3	1.3
2 5-Hydroxymethyl-2-furoic	≤ 14		3.6	3.6
3 3-Oxoglutaric	≤ 0.33		0	0.00
4 Furan-2,5-dicarboxylic	≤ 16		4.8	4.8
5 Furancarbonylglycine	≤ 1.9		0.67	0.67
6 Tartaric	≤ 4.5		0.86	0.86
7 Arabinose	≤ 29	H	40	40
8 Carboxycitric	≤ 29		3.3	3.3
9 Tricarballylic	≤ 0.44		0.37	0.37
Bacterial Markers				
10 Hippuric	≤ 613	H	1 071	1071
11 2-Hydroxyphenylacetic	0.06 - 0.66	H	1.3	1.3
12 4-Hydroxybenzoic	≤ 1.3		0.72	0.72
13 4-Hydroxyhippuric	0.79 - 17		13	13
14 DHPPA (Beneficial Bacteria)	≤ 0.38		0.04	0.04
Clostridia Bacterial Markers				
15 4-Hydroxyphenylacetic *(C. difficile, C. stricklandii, C. lituseburense & others)*	≤ 19		17	17
16 HPHPA *(C. sporogenes, C. caloritolerans, C. botulinum & others)*	≤ 208	H	248	248
17 4-Cresol *(C. difficile)*	≤ 75		50	50
18 3-Indoleacetic *(C. stricklandii, C. lituseburense, C. subterminale & others)*	≤ 11		2.8	2.8

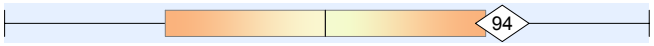
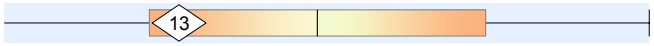
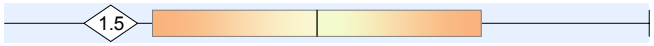
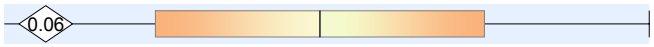
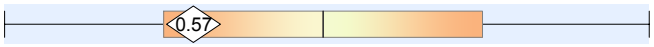
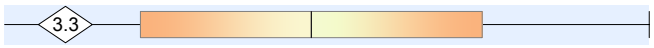
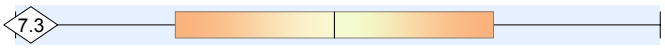
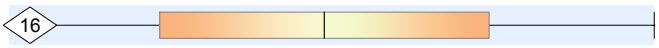
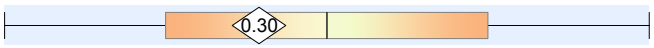
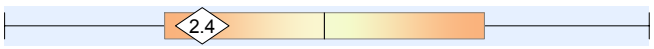
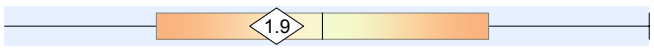
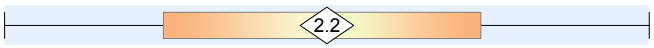
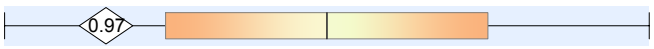
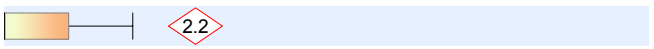
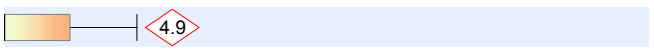
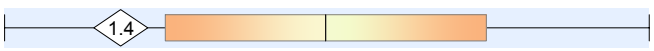
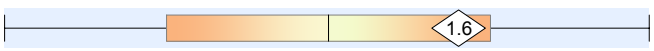
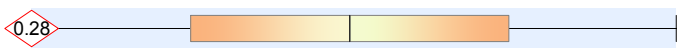
The Great Plains Laboratory, Inc.

Requisition #:

Patient Name:

Physician: BIOLAB

Date of Collection: 3/10/2016

Metabolic Markers in Urine		Reference Range (mmol/mol creatinine)			Patient Value	Reference Population - Females Age 13 and Over
Oxalate Metabolites						
19	Glyceric	0.77 - 7.0			3.7	
20	Glycolic	16 - 117			94	
21	Oxalic	6.8 - 101		H	179	
Glycolytic Cycle Metabolites						
22	Lactic	≤ 48			13	
23	Pyruvic	≤ 9.1			1.5	
Mitochondrial Markers - Krebs Cycle Metabolites						
24	Succinic	≤ 9.3			3.0	
25	Fumaric	≤ 0.94			0.06	
26	Malic	0.06 - 1.8			0.57	
27	2-Oxoglutaric	≤ 35			3.3	
28	Aconitic	6.8 - 28			7.3	
29	Citric	≤ 507			16	
Mitochondrial Markers - Amino Acid Metabolites						
30	3-Methylglutaric	≤ 0.76			0.30	
31	3-Hydroxyglutaric	≤ 6.2			2.4	
32	3-Methylglutaconic	≤ 4.5			1.9	
Neurotransmitter Metabolites						
Phenylalanine and Tyrosine Metabolites						
33	Homovanillic (HVA) *(dopamine)*	0.80 - 3.6			2.2	
34	Vanillylmandelic (VMA) *(norepinephrine, epinephrine)*	0.46 - 3.7			0.97	
35	HVA / VMA Ratio	0.16 - 1.8		H	2.2	
Tryptophan Metabolites						
36	5-Hydroxyindoleacetic (5-HIAA) *(serotonin)*	≤ 4.3		H	4.9	
37	Quinolinic	0.85 - 3.9			1.4	
38	Kynurenic	0.17 - 2.2			1.6	
39	Quinolinic / 5-HIAA Ratio	0.42 - 2.0		L	0.28	

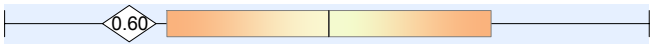
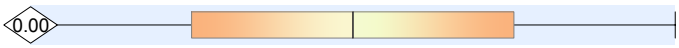
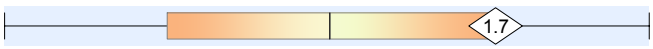
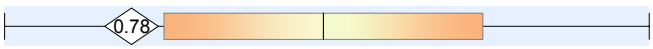
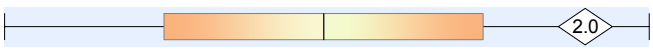
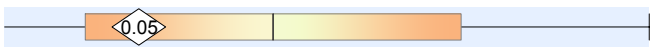
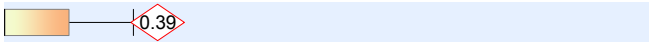
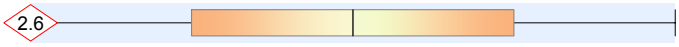
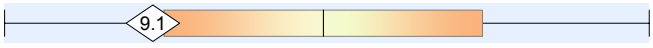
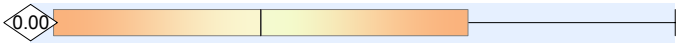
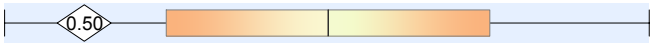
The Great Plains Laboratory, Inc.

Requisition #:

Patient Name:

Physician: BIOLAB

Date of Collection: 3/10/2016

	Metabolic Markers in Urine	Reference Range (mmol/mol creatinine)		Patient Value	Reference Population - Females Age 13 and Over
Pyrimidine Metabolites - Folate Metabolism					
40	Uracil	≤ 9.7	H	16	
41	Thymine	≤ 0.56		0.36	
Ketone and Fatty Acid Oxidation					
42	3-Hydroxybutyric	≤ 3.1		0.60	
43	Acetoacetic	≤ 10		0	
44	4-Hydroxybutyric	≤ 4.8		0.53	
45	Ethylmalonic	0.44 - 2.8		1.3	
46	Methylsuccinic	0.10 - 2.2		1.7	
47	Adipic	0.04 - 3.8		0.78	
48	Suberic	0.18 - 2.2		2.0	
49	Sebacic	≤ 0.24		0.05	
Nutritional Markers					
Vitamin B12					
50	Methylmalonic ✱	≤ 2.3		1.6	
Vitamin B6					
51	Pyridoxic (B6)	≤ 34		15	
Vitamin B5					
52	Pantothenic (B5)	≤ 10		4.1	
Vitamin B2 (Riboflavin)					
53	Glutaric ✱	0.04 - 0.36	H	0.39	
Vitamin C					
54	Ascorbic	10 - 200	L	2.6	
Vitamin Q10 (CoQ10)					
55	3-Hydroxy-3-methylglutaric ✱	0.17 - 39		9.1	
Glutathione Precursor and Chelating Agent					
56	N-Acetylcysteine (NAC)	≤ 0.28		0	
Biotin (Vitamin H)					
57	Methylcitric ✱	0.19 - 2.7		0.50	

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

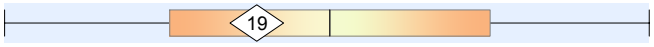
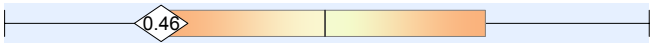
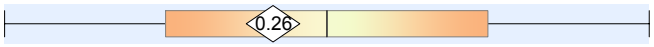
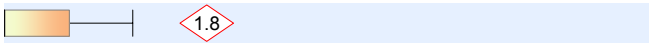
The Great Plains Laboratory, Inc.

Requisition #:

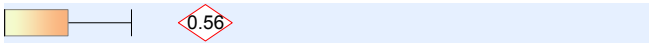
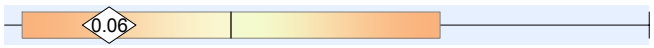
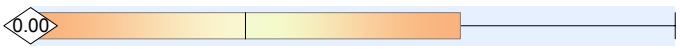
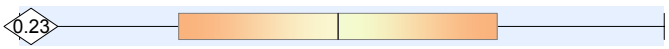
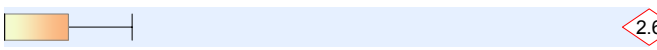
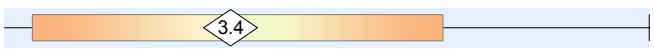
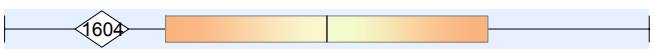
Patient Name:

Physician: BIOLAB

Date of Collection: 3/10/2016

Metabolic Markers in Urine		Reference Range (mmol/mol creatinine)			Patient Value	Reference Population - Females Age 13 and Over
Indicators of Detoxification						
Glutathione						
58	Pyroglutamic ✱	10 - 33			19	
59	2-Hydroxybutyric ✱	0.03 - 1.8			0.46	
Ammonia Excess						
60	Orotic	0.06 - 0.54			0.26	
Aspartame, salicylates, or GI bacteria						
61	2-Hydroxyhippuric	≤ 1.3		H	1.8	

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

Amino Acid Metabolites						
62	2-Hydroxyisovaleric	≤ 0.42		H	0.56	
63	2-Oxoisovaleric	≤ 2.1			0	
64	3-Methyl-2-oxovaleric	≤ 0.87			0.35	
65	2-Hydroxyisocaproic	≤ 0.48			0	
66	2-Oxoisocaproic	≤ 0.37			0.06	
67	2-Oxo-4-methiolbutyric	≤ 0.16			0.02	
68	Mandelic	≤ 0.21			0	
69	Phenyllactic	≤ 0.20			0.15	
70	Phenylpyruvic	0.20 - 1.9			0.23	
71	Homogentisic	≤ 0.36			0.10	
72	4-Hydroxyphenyllactic	≤ 0.80		H	2.6	
73	N-Acetylaspartic	≤ 3.0			0.40	
74	Malonic	≤ 9.7			3.4	
Mineral Metabolism						
75	Phosphoric	1 000 - 5 000			1 604	

Indicator of Fluid Intake

76	*Creatinine	50	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.

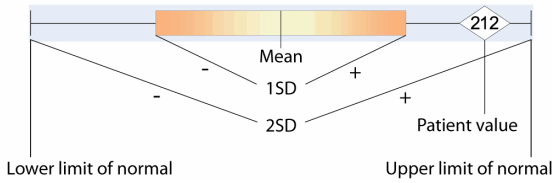
The first graph will occur when the value of the patient is within the reference (normal) range, defined as the mean plus or minus two standard deviations.

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.

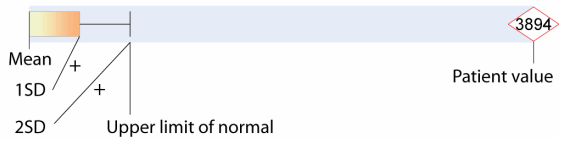
Example of Value Within Reference Range

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	



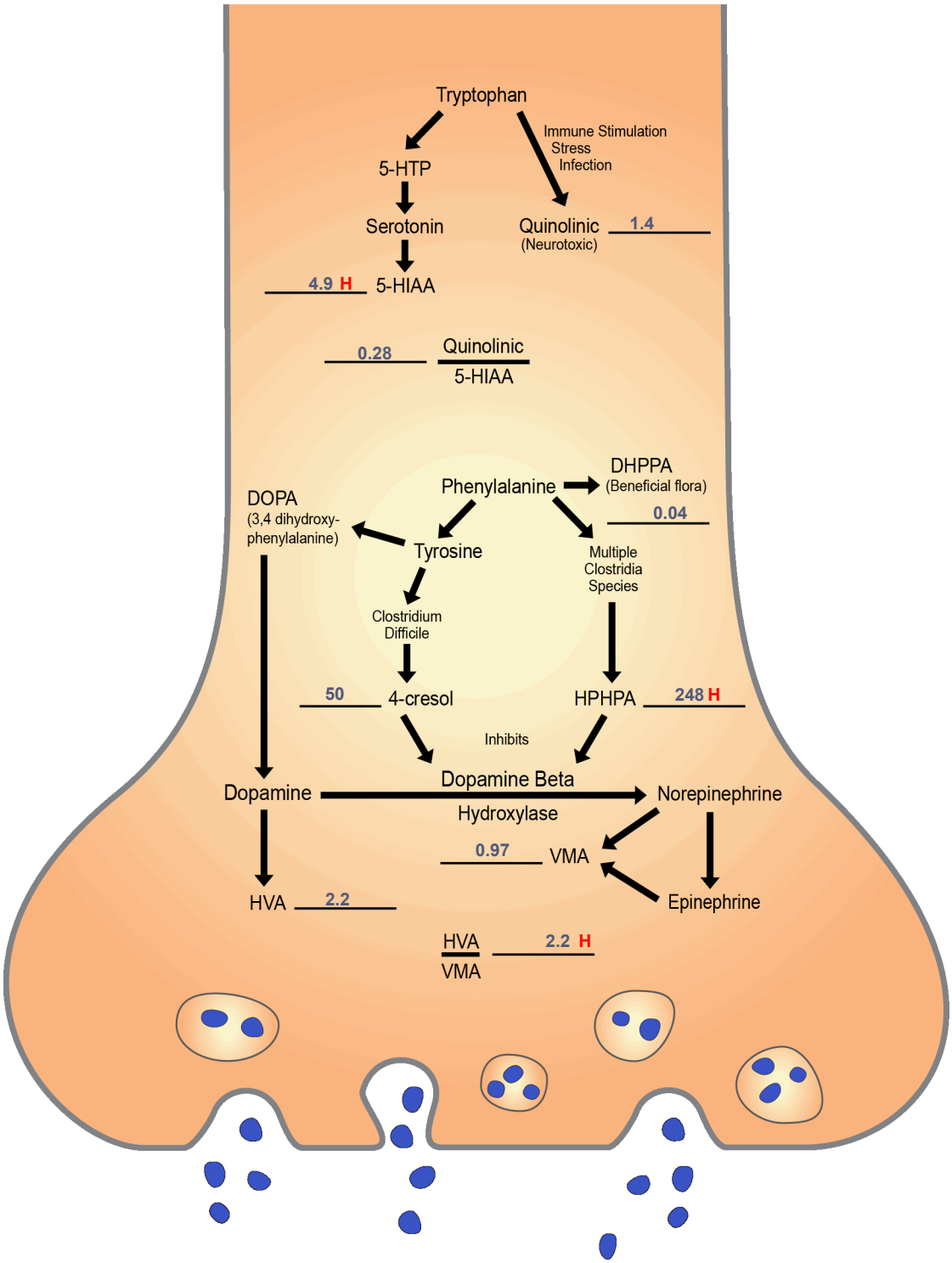
Example of Elevated Value

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	



Requisition #:		***Physician:***	BIOLAB
Patient Name:		***Date of Collection:***	3/10/2016

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 hippuric acid (Marker 10) may derive from food, GI bacterial activity, or exposure to the 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. Chlorogenic acid is a common substance 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 present in high amounts in cranberry juice and is a food preservative. 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 hippuric acid in urine is from GI 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 probiotics (30-50 billion cfu's) that include *Lactobacillus rhamnosus*.

High 2-hydroxyphenylacetic acid (Marker 11) is associated with intestinal bacteria overgrowth and with the genetic disease phenylketonuria (PKU).

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 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>.

VMA levels below the mean (Marker 34) may indicate lower production of the neurotransmitter norepinephrine or the hormone adrenaline, perhaps due to low dietary intake of the amino acid precursors phenylalanine or tyrosine. Vanylmandelic acid (VMA) is a metabolite of norepinephrine or adrenaline. Low VMA may also result from blocked conversion of dopamine to norepinephrine by *Clostridia* metabolites. Supplementation with phenylalanine or tyrosine may be beneficial. Enzyme cofactors magnesium, B6 (pyridoxine) or biopterin may also be deficient and respond to supplementation.

High HVA/VMA ratio (Marker 35) The most common reason for an elevation of the HVA/VMA ratio is the decreased conversion of dopamine to norepinephrine and epinephrine. The enzyme responsible for this conversion, dopamine betahydroxylase, is copper and vitamin C dependent, so an elevated ratio could be due to deficiencies of these cofactors. Another common factor is inhibition of this enzyme by *Clostridia* byproducts. A high HPHPA, 4-Cresol, or other elevations of metabolites would be consistent with the latter explanation.

High 5-hydroxyindoleacetic acid (5-HIAA) (Marker 36) may occur in celiac or tropical sprue, carcinoid tumors, or from ingestion of foods high in serotonin, such as avocado, banana, tomato, plum, walnut, pineapple or eggplant. Elevated values may also result from supplementing with tryptophan itself or 5-hydroxy-tryptophan (5-HTP); if this is the case, a high value does not necessarily indicate the need to reduce or eliminate supplementation. It is possible that excessive tryptophan intake can lead to overproduction of the neurotoxic and inflammatory metabolite quinolinic acid. (See quinolinic acid value and interpretation.)

High uracil with normal/elevated thymine (Markers 40, 41) is an abnormality that is found in about 10% of children with autism. Because folic acid is involved as a methyl donor in the conversion of uracil to thymine, this elevation may indicate a deficiency of folic acid or a defect in folic acid metabolism. Regardless of cause, supplementation with folic acid, folinic acid or methyl folate may be beneficial.

High glutaric acid (Marker 53) 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 (20-100 mg/day) and coenzyme Q-10 (50-100 mg/day) may be beneficial.

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 academia 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 sebacic acids.

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 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.

High 2-hydroxyisovaleric acid and/or 2-hydroxyisocaproic acid (Markers 62,65) may be due to the genetic disease MSUD (maple syrup urine disease) or dihydrolipoyl dehydrogenase deficiency. Individuals with slight to moderate elevations may benefit from supplementing with high doses (5-20 mg/kg/day) of thiamine.

High 4-hydroxyphenyllactic acid (Marker 72) is associated with tyrosinemia, which can be due to immature development of enzymes in infants or to genetic deficiencies. Even a mild case would have levels at least of 100 mmol/mol creatinine. Values between the upper limit of normal and 100 mmol creatinine may be due to the heterozygous genetic carrier state, or mild disease or unknown physiological conditions.

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.