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 #:	530624	***Physician:***	REGENERUS LAB
Patient Name:	Paul Burgess	***Date of Collection:***	9/2/2017
Patient Age:	51	***Time of Collection:***	07:00 AM
Patient Sex:	M	***Print Date:***	10/16/2017

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				
1 Citramalic	0.11 - 2.0		0.38	0.38
2 5-Hydroxymethyl-2-furoic	≤ 18		5.1	5.1
3 3-Oxoglutaric	≤ 0.11		0.06	0.06
4 Furan-2,5-dicarboxylic	≤ 13		3.5	3.5
5 Furancarbonylglycine	≤ 2.3		2.1	2.1
6 Tartaric	≤ 5.3		0.97	0.97
7 Arabinose	≤ 20	H	23	23
8 Carboxycitric	≤ 20		1.2	1.2
9 Tricarballylic	≤ 0.58		0.16	0.16
Bacterial Markers				
10 Hippuric	≤ 241	H	426	426
11 2-Hydroxyphenylacetic	0.03 - 0.47		0.31	0.31
12 4-Hydroxybenzoic	0.01 - 0.73		0.63	0.63
13 4-Hydroxyhippuric	≤ 14		6.6	6.6
14 DHPPA (Beneficial Bacteria)	≤ 0.23	H	0.34	0.34
Clostridia Bacterial Markers				
15 4-Hydroxyphenylacetic *(C. difficile, C. stricklandii, C. lituseburense & others)*	≤ 18		11	11
16 HPHPA *(C. sporogenes, C. caloritolerans, C. botulinum & others)*	≤ 102	H	121	121
17 4-Cresol *(C. difficile)*	≤ 39		25	25
18 3-Indoleacetic *(C. stricklandii, C. lituseburense, C. subterminale & others)*	≤ 6.8		0.24	0.24

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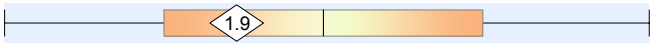
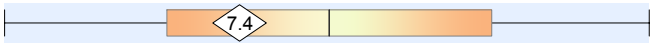
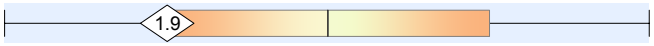
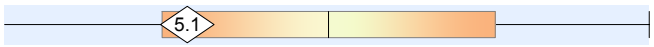
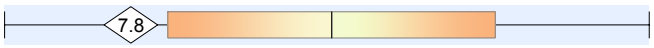
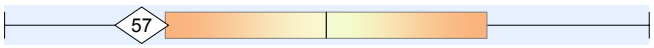
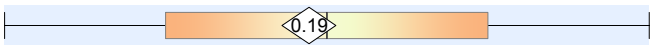
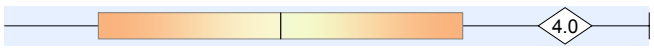
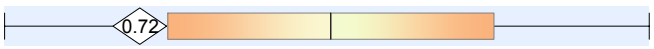
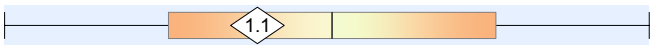
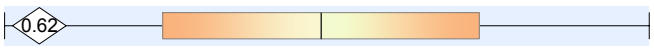
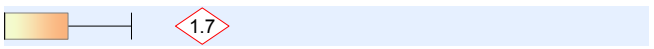
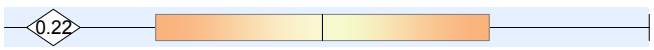
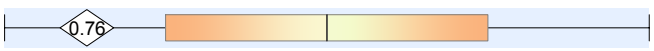
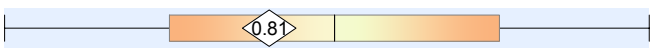
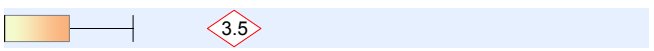
Metabolic Markers in Urine		Reference Range *(mmol/mol creatinine)*			Patient Value	Reference Population - Males Age 13 and Over
Oxalate Metabolites						
19	Glyceric	0.21	- 4.9		1.9	
20	Glycolic	18	- 81		53	
21	Oxalic	8.9	- 67	H	109	
Glycolytic Cycle Metabolites						
22	Lactic	0.74	- 19		7.4	
23	Pyruvic	0.28	- 6.7		1.9	
Mitochondrial Markers - Krebs Cycle Metabolites						
24	Succinic		≤ 5.3		1.8	
25	Fumaric		≤ 0.49		0.05	
26	Malic		≤ 1.1		1.0	
27	2-Oxoglutaric		≤ 18		5.1	
28	Aconitic	4.1	- 23		7.8	
29	Citric	2.2	- 260		57	
Mitochondrial Markers - Amino Acid Metabolites						
30	3-Methylglutaric	0.02	- 0.38		0.19	
31	3-Hydroxyglutaric		≤ 4.6		4.0	
32	3-Methylglutaconic	0.38	- 2.0		0.72	
Neurotransmitter Metabolites						
Phenylalanine and Tyrosine Metabolites						
33	Homovanillic (HVA) *(dopamine)*	0.39	- 2.2		1.1	
34	Vanillylmandelic (VMA) *(norepinephrine, epinephrine)*	0.53	- 2.2		0.62	
35	HVA / VMA Ratio	0.32	- 1.4	H	1.7	
Tryptophan Metabolites						
36	5-Hydroxyindoleacetic (5-HIAA) *(serotonin)*		≤ 2.9		0.22	
37	Quinolinic	0.52	- 2.4		0.76	
38	Kynurenic	0.12	- 1.8		0.81	
39	Quinolinic / 5-HIAA Ratio		≤ 2.5	H	3.5	

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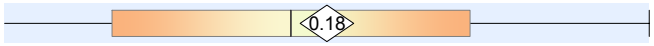
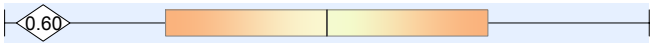
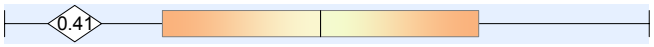
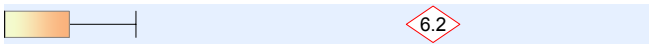
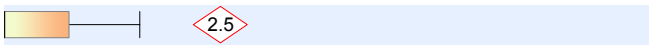
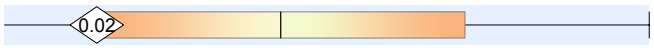
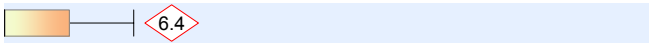
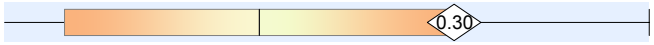
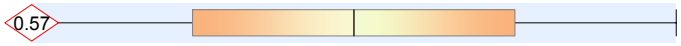
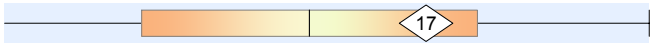
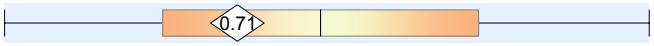
Patient Name:	Paul Burgess	***Date of Collection:***	9/2/2017

Metabolic Markers in Urine		Reference Range *(mmol/mol creatinine)*		Patient Value	Reference Population - Males Age 13 and Over
Pyrimidine Metabolites - Folate Metabolism					
40	Uracil	≤ 6.9		2.4	
41	Thymine	≤ 0.36		0.18	
Ketone and Fatty Acid Oxidation					
42	3-Hydroxybutyric	≤ 1.9		0.11	
43	Acetoacetic	≤ 10		0.60	
44	4-Hydroxybutyric	≤ 4.3		0.65	
45	Ethylmalonic	0.13 - 2.7		0.41	
46	Methylsuccinic	≤ 2.3		0.89	
47	Adipic	≤ 2.9	H	6.2	
48	Suberic	≤ 1.9	H	2.5	
49	Sebacic	≤ 0.14		0.02	
Nutritional Markers					
Vitamin B12					
50	Methylmalonic ✱	≤ 2.3		0.36	
Vitamin B6					
51	Pyridoxic (B6)	≤ 26		1.6	
Vitamin B5					
52	Pantothenic (B5)	≤ 5.4	H	6.4	
Vitamin B2 (Riboflavin)					
53	Glutaric ✱	≤ 0.43		0.30	
Vitamin C					
54	Ascorbic	10 - 200	L	0.57	
Vitamin Q10 (CoQ10)					
55	3-Hydroxy-3-methylglutaric ✱	≤ 26		17	
Glutathione Precursor and Chelating Agent					
56	N-Acetylcysteine (NAC)	≤ 0.13		0	
Biotin (Vitamin H)					
57	Methylcitric ✱	0.15 - 1.7		0.71	

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

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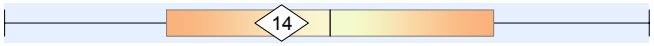
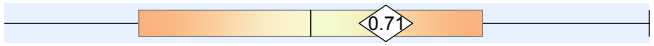
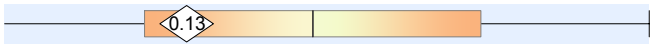
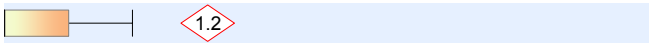
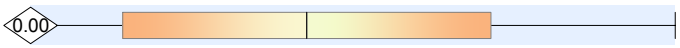
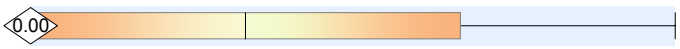
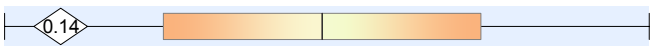
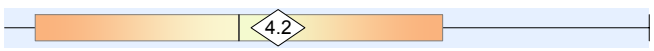
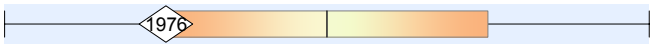
Requisition #:	530624	***Physician:***	REGENERUS LAB
Patient Name:	Paul Burgess	***Date of Collection:***	9/2/2017

Metabolic Markers in Urine		Reference Range *(mmol/mol creatinine)*		Patient Value		Reference Population - Males Age 13 and Over
Indicators of Detoxification						
Glutathione						
58	Pyroglutamic ✱	5.7 - 25		14		
59	2-Hydroxybutyric ✱	≤ 1.2		0.71		
Ammonia Excess						
60	Orotic	≤ 0.46		0.13		
Aspartame, salicylates, or GI bacteria						
61	2-Hydroxyhippuric	≤ 0.86	H	1.2		

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

Amino Acid Metabolites					
62	2-Hydroxyisovaleric	≤ 0.41		0.39	
63	2-Oxoisovaleric	≤ 1.5		0.04	
64	3-Methyl-2-oxovaleric	≤ 0.56		0.23	
65	2-Hydroxyisocaproic	≤ 0.39		0.10	
66	2-Oxoisocaproic	≤ 0.34		0.08	
67	2-Oxo-4-methiolbutyric	≤ 0.14		0	
68	Mandelic	≤ 0.09		0	
69	Phenyllactic	≤ 0.10		0.10	
70	Phenylpyruvic	0.02 - 1.4		0.14	
71	Homogentisic	≤ 0.23		0.02	
72	4-Hydroxyphenyllactic	≤ 0.62		0.35	
73	N-Acetylaspartic	≤ 2.5		0.57	
74	Malonic	≤ 9.9		4.2	
Mineral Metabolism					
75	Phosphoric	1 000 - 4 900		1 976	

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Requisition #:	530624	*Physician:*	REGENERUS LAB
Patient Name:	Paul Burgess	*Date of Collection:*	9/2/2017

Indicator of Fluid Intake

76	*Creatinine	221	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.

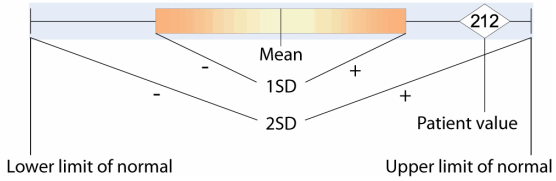
The first graph will occur when the value of the patient is within the reference (normal) range, defined as the mean plus or minus two standard deviations.

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.

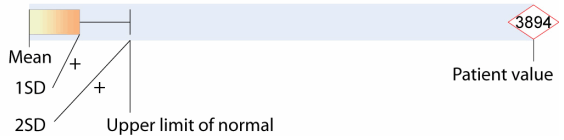
Example of Value Within Reference Range

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	

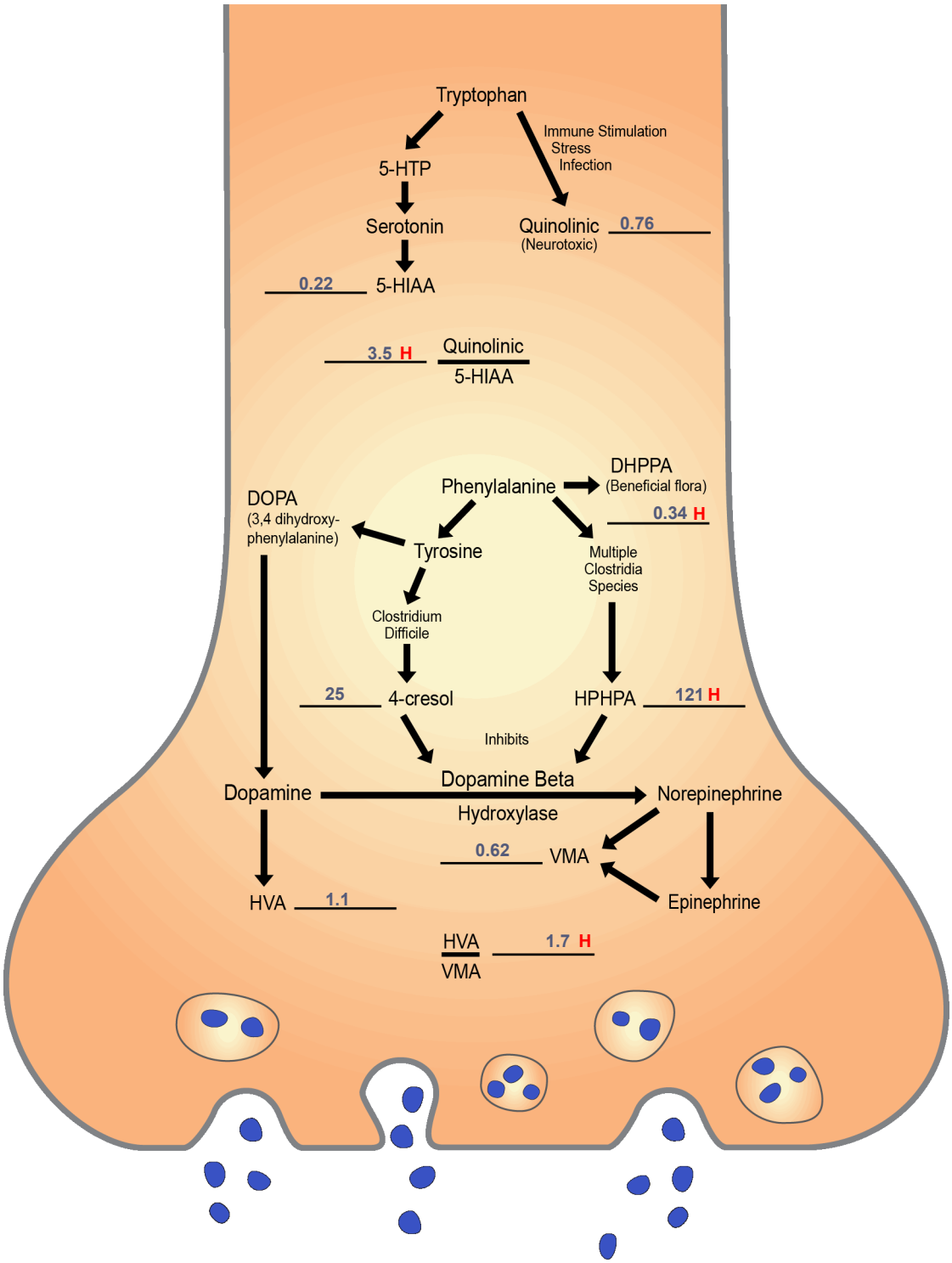


Example of Elevated Value

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 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 DHPPA (3,4 dihydroxyphenylpropionic acid) (Marker 14) indicates excessive intake of chlorogenic 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. Harmless or beneficial bacteria such as Lactobacilli, Bifidobacteria, and E. coli mediate the breakdown of chlorogenic acid to 3,4-dihydroxyphenylpropionic acid (DHPPA), and high values may indicate increased amounts of these species in the GI tract. In addition, one *Clostridia* species, *C. orbiscindens*, can convert the flavanoids luteolin and eriodictyol, occurring only in a relatively small food group that includes parsley, thyme, celery, and sweet red pepper to 3,4-dihydroxyphenylpropionic acid. The quantity of *Clostridia orbiscindens* in the GI tract is negligible (approximately 0.1% of the total bacteria) compared to the predominant flora of *Lactobacilli, Bifidobacteria,* and *E. coli*. Consequently, this marker is essentially useless as a general *Clostridia* marker but may be a good indicator of the presence of beneficial flora.

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>.

HVA levels below the mean (Marker 33) may indicate lower production of the neurotransmitter dopamine, perhaps due to low dietary intake of the amino acid precursors phenylalanine or tyrosine. Homovanillic acid is a metabolite of the neurotransmitter dopamine. Supplementation with phenylalanine or tyrosine may be beneficial. Enzyme cofactors magnesium, B6 (pyridoxine) or biopterin may also be deficient; neurotransmitter levels may increase with supplementation with these cofactors if these are deficient.

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.

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 quinolinic acid / 5-HIAA ratio (Marker 39) indicates an imbalance of these organic acids and may be a sign of neural excitotoxicity. Quinolinic acid is an excitotoxic stimulant of certain brain cells that have NMDA-type receptors. Overstimulated nerve cells may 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 derived from the amino acid tryptophan and 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).

An elevated ratio is not specific for a particular medical condition and is commonly associated with excessive inflammation due to recurrent infections. If quinolinic acid is not elevated, low 5-HIAA from serotonin may be the source of the imbalance. Supplementation with 5-HTP may increase serotonin levels, but 5-HTP is not metabolized to quinolinic acid. Immune overstimulation, excess adrenal production of cortisol due to stress, or high exposure to phthalates may also increase the quinolinic acid/5-HIAA acid ratio.

The drug deprenyl or the dietary supplements carnitine, melatonin, capsaicin, turmeric (curcumin) and garlic may 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. Inositol hexaniacinate as an adult dose of 500-1000 mg does not cause niacin flush.

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.

High pantothenic acid (B5) (Marker 52) 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.

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.

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.