**** Current supplements noted at end of report

The Great Plains Laboratory, Inc.

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Requisition #:	537053	***Physician:***	RESEARCH NUTRITION
Patient Name:		***Date of Collection:***	10/31/2017
Patient Age:		***Time of Collection:***	03:00 AM
Patient Sex:	M	***Print Date:***	11/10/2017

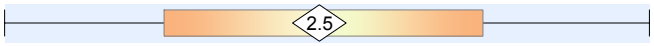
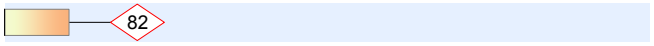
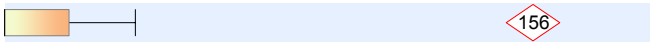
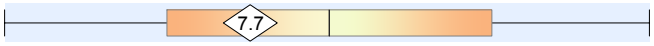
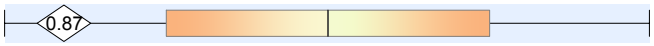
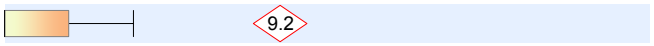
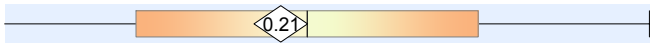
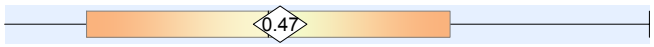
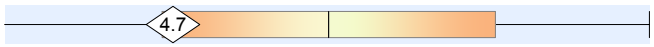
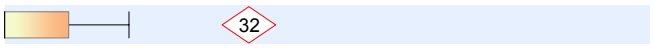
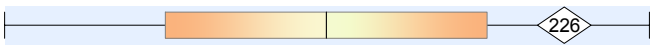
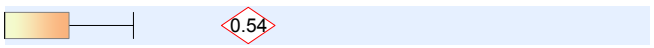
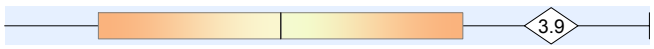
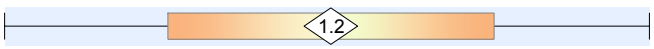
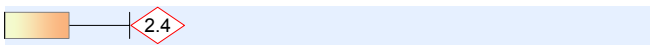
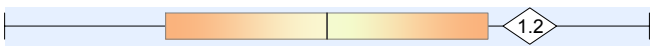
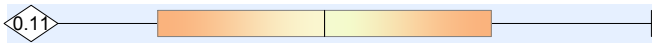
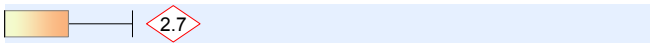
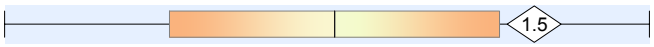
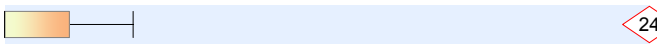
Organic Acids Test - Nutritional and Metabolic Profile

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		0.71	0.71
2 5-Hydroxymethyl-2-furoic	≤ 18		7.6	7.6
3 3-Oxoglutaric	≤ 0.11		0.02	0.02
4 Furan-2,5-dicarboxylic	≤ 13		7.1	7.1
5 Furancarbonylglycine	≤ 2.3		0	0.00
6 Tartaric	≤ 5.3		0.77	0.77
7 Arabinose	≤ 20	H	103	103
8 Carboxycitric	≤ 20		1.1	1.1
9 Tricarballylic	≤ 0.58		0.15	0.15
Bacterial Markers				
10 Hippuric	≤ 241		127	127
11 2-Hydroxyphenylacetic	0.03 - 0.47		0.37	0.37
12 4-Hydroxybenzoic	0.01 - 0.73	H	0.93	0.93
13 4-Hydroxyhippuric	≤ 14		10	10
14 DHPPA (Beneficial Bacteria)	≤ 0.23	H	0.26	0.26
Clostridia Bacterial Markers				
15 4-Hydroxyphenylacetic *(C. difficile, C. stricklandii, C. lituseburense & others)*	≤ 18		13	13
16 HPHPA *(C. sporogenes, C. caloritolerans, C. botulinum & others)*	≤ 102		38	38
17 4-Cresol *(C. difficile)*	≤ 39		23	23
18 3-Indoleacetic *(C. stricklandii, C. lituseburense, C. subterminale & others)*	≤ 6.8		0.61	0.61

The Great Plains Laboratory, Inc.

Requisition #: 537053 **Physician:** RESEARCH NUTRITION

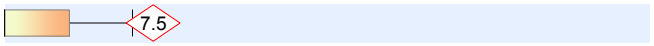
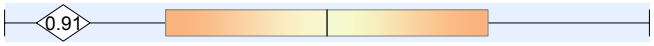
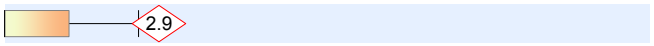
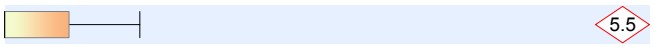
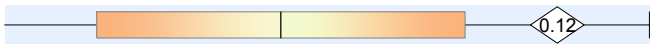
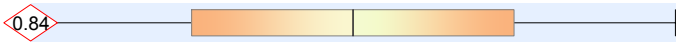
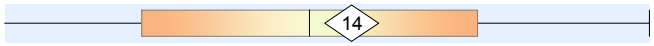
Patient Name: **Date of Collection:** 10/31/2017

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		2.5	
20	Glycolic	18	- 81	H	82	
21	Oxalic	8.9	- 67	H	156	
Glycolytic Cycle Metabolites						
22	Lactic	0.74	- 19		7.7	
23	Pyruvic	0.28	- 6.7		0.87	
Mitochondrial Markers - Krebs Cycle Metabolites						
24	Succinic		≤ 5.3	H	9.2	
25	Fumaric		≤ 0.49		0.21	
26	Malic		≤ 1.1		0.47	
27	2-Oxoglutaric		≤ 18		4.7	
28	Aconitic	4.1	- 23	H	32	
29	Citric	2.2	- 260		226	
Mitochondrial Markers - Amino Acid Metabolites						
30	3-Methylglutaric	0.02	- 0.38	H	0.54	
31	3-Hydroxyglutaric		≤ 4.6		3.9	
32	3-Methylglutaconic	0.38	- 2.0		1.2	
Neurotransmitter Metabolites						
Phenylalanine and Tyrosine Metabolites						
33	Homovanillic (HVA) *(dopamine)*	0.39	- 2.2	H	2.4	
34	Vanillylmandelic (VMA) *(norepinephrine, epinephrine)*	0.53	- 2.2		2.0	
35	HVA / VMA Ratio	0.32	- 1.4		1.2	
Tryptophan Metabolites						
36	5-Hydroxyindoleacetic (5-HIAA) *(serotonin)*		≤ 2.9		0.11	
37	Quinolinic	0.52	- 2.4	H	2.7	
38	Kynurenic	0.12	- 1.8		1.5	
39	Quinolinic / 5-HIAA Ratio		≤ 2.5	H	24	

The Great Plains Laboratory, Inc.

Requisition #: 537053 *Physician:* RESEARCH NUTRITION

Patient Name: *Date of Collection:* 10/31/2017

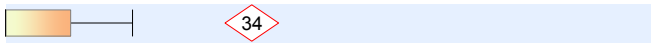
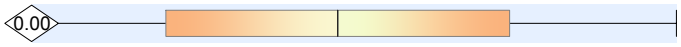
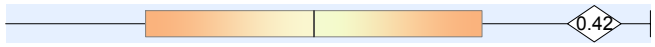
	Metabolic Markers in Urine	Reference Range (mmol/mol creatinine)		Patient Value	Reference Population - Males Age 13 and Over
Pyrimidine Metabolites - Folate Metabolism					
40	Uracil	≤ 6.9	H	7.5	
41	Thymine	≤ 0.36		0.22	
Ketone and Fatty Acid Oxidation					
42	3-Hydroxybutyric	≤ 1.9		1.9	
43	Acetoacetic	≤ 10		0.91	
44	4-Hydroxybutyric	≤ 4.3		0	
45	Ethylmalonic	0.13 - 2.7	H	2.9	
46	Methylsuccinic	≤ 2.3		1.5	
47	Adipic	≤ 2.9		1.6	
48	Suberic	≤ 1.9	H	5.5	
49	Sebacic	≤ 0.14		0.12	
Nutritional Markers					
	Vitamin B12				
50	Methylmalonic ✱	≤ 2.3		1.4	
	Vitamin B6				
51	Pyridoxic (B6)	≤ 26		2.5	
	Vitamin B5				
52	Pantothenic (B5)	≤ 5.4		2.7	
	Vitamin B2 (Riboflavin)				
53	Glutaric ✱	≤ 0.43		0.30	
	Vitamin C				
54	Ascorbic	10 - 200	L	0.84	
	Vitamin Q10 (CoQ10)				
55	3-Hydroxy-3-methylglutaric ✱	≤ 26		14	
	Glutathione Precursor and Chelating Agent				
56	N-Acetylcysteine (NAC)	≤ 0.13		0.08	
	Biotin (Vitamin H)				
57	Methylcitric ✱	0.15 - 1.7		1.1	

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

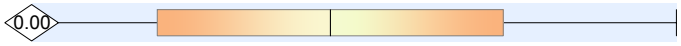
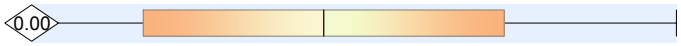
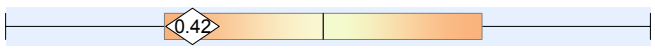
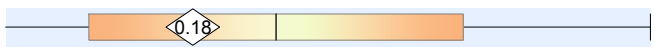
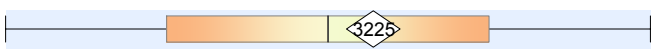
The Great Plains Laboratory, Inc.

Requisition #: 537053 *Physician:* RESEARCH NUTRITION

Patient Name: *Date of Collection:* 10/31/2017

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	H	34	
59	2-Hydroxybutyric ✱		≤	1.2		0	
Ammonia Excess							
60	Orotic		≤	0.46		0.42	
Aspartame, salicylates, or GI bacteria							
61	2-Hydroxyhippuric		≤	0.86		0.40	

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

Amino Acid Metabolites							
62	2-Hydroxyisovaleric		≤	0.41		0	
63	2-Oxoisovaleric		≤	1.5		0	
64	3-Methyl-2-oxovaleric		≤	0.56		0.40	
65	2-Hydroxyisocaproic		≤	0.39		0	
66	2-Oxoisocaproic		≤	0.34		0.06	
67	2-Oxo-4-methiolbutyric		≤	0.14		0.09	
68	Mandelic		≤	0.09		0.07	
69	Phenyllactic		≤	0.10		0	
70	Phenylpyruvic	0.02	-	1.4		0.42	
71	Homogentisic		≤	0.23		0.01	
72	4-Hydroxyphenyllactic		≤	0.62		0.18	
73	N-Acetylaspartic		≤	2.5		0.41	
74	Malonic		≤	9.9		4.1	
Mineral Metabolism							
75	Phosphoric	1 000	-	4 900		3 225	

Indicator of Fluid Intake

76	*Creatinine	231	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.

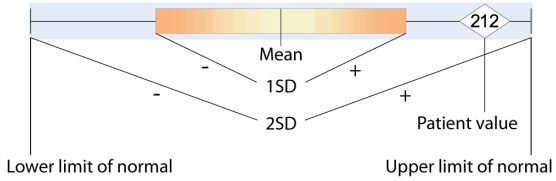
The first graph will occur when the value of the patient is within the reference (normal) range, defined as the mean plus or minus two standard deviations.

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.

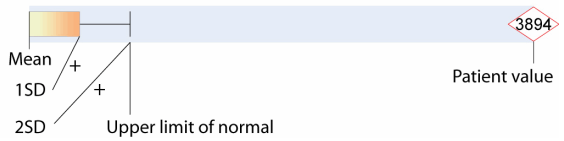
Example of Value Within Reference Range

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	

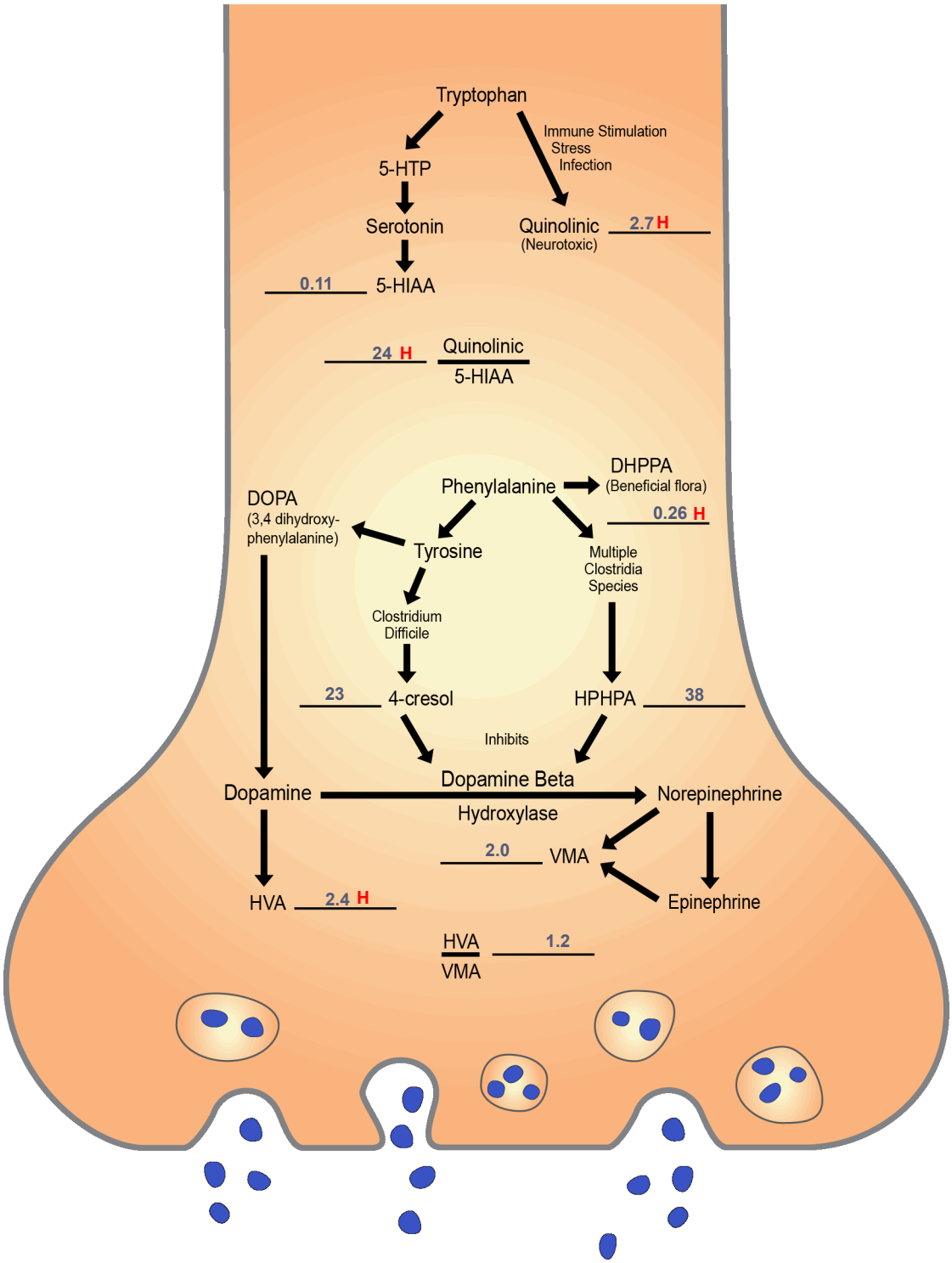


Example of Elevated Value

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 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 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 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 aconitic acid (Marker 28) may indicate an additional requirement for reduced glutathione. Aconitase is dependent upon glutathione to metabolize citric and aconitic acids.

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.

High HVA (Marker 33) may result from toxic metal exposure (including lead, aluminum, manganese, and mercury), presumably due to increased release of dopamine from neurons. Heavy metal testing (blood or hair) might be useful to determine if such exposure is significant. Homovanillic acid (HVA), a dopamine metabolite, is often elevated due to stress-induced catecholamine output from the adrenal gland which depletes vitamin C. Supplementation with vitamin C (ascorbate) may be helpful in such cases.

Elevated HVA may also result from the intake of L-DOPA, dopamine, phenylalanine, or tyrosine. If values are more than double the upper limit of normal, the possibility of catecholamine-secreting tumors can be ruled out by 24- hour VMA and/or HVA testing in urine. Even in this subgroup, the incidence of tumors is extremely rare. High HVA may be associated with *Clostridia* or toxoplasmosis infection. If HVA is elevated and VMA is normal, avoid supplementation with phenylalanine or tyrosine until *Clostridia* or toxoplasmosis is treated.

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 (Marker 37) may be a sign of inflammation and/or neural excitotoxicity. Quinolinic acid is derived from the amino acid tryptophan and is neurotoxic at high levels. As an excitotoxic stimulant of certain brain cells that have NMDA-type receptors, high quinolinic acid may cause nerve cell death with continuous stimulation. 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. High levels of quinolinic acid may inhibit heart contractions, cause lipid peroxidation in the brain, and increase apoptosis (programmed cell death) of astrocytes in human brain. The level of quinolinic acid is also highly correlated with the degree of arthritis impairment.

Quinolinic acid is also a metal chelator, and inhibits enzymes that allow the body to produce glucose when needed. Excessive immune stimulation and chronic inflammation, resulting in overproduction of cytokines like interferon, stimulates overproduction of quinolinic acid. However, quinolinic acid is an important intermediate in making the essential nutritional cofactor nicotinamide adenine dinucleotide (NAD), which is also derived from niacin (B3). Phthalates inhibit the conversion of quinolinic acid to NAD.

Treatment of excessive levels of quinolinic acid can be achieved by multiple approaches: reducing tryptophan supplements, preventing repeated infections and subsequent immune overstimulation by: supplementation with colostrum, transfer factor and probiotics; reducing the use of immune modulators like interferon that increase quinolinic acid production; or reducing the numbers of vaccines given at one time or increasing the interval between vaccinations. In addition, 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. A high quinolinic acid/ 5-hydroxyindoleacetic acid ratio would be indicative of immune overstimulation and/or phthalate toxicity.

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 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 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 pyroglutamic acid (Marker 58)

Elevated pyroglutamic acid (oxoproline) is most commonly due to intracellular glutathione deficiency due to toxic exposures such as acetaminophen toxicity. Pyroglutamic acid (5-oxoproline) is formed from intracellular gamma-glutamylcysteine. This conversion is regulated by intracellular glutathione. When intracellular glutathione is low or there is a genetic deficiency of glutathione synthetase, high amounts of gamma-glutamylcysteine and its metabolite pyroglutamic acid are formed. Intracellular glutathione deficiency and high pyroglutamic acid are commonly caused by moderate doses of acetaminophen (paracetamol), vigabatrin (Sabril®). and certain antibiotics (flucloxacillin, netimicin) or exposure to toxic environmental chemicals that deplete glutathione such as halogenated hydrocarbons (e.g. DDT, PCBs, and many others). High pyroglutamic acid may also be caused by genetic deficiency of the enzyme oxoprolinase that breaks down pyroglutamic acid and may also be associated with urea cycle disorders, propionic acidemia, hawkinsinuria, Stevens-Johnson syndrome with severe burns, homocystinuria, prematurity, glycine deficiency, and infants on synthetic formulas. Treatment most often includes supplementation with either N-acetyl cysteine or glutathione.

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.

Supplements I was on when this test was taken:

- B2 and R5P
- P5P
- Biotin
- Niacitol and possibly some Niacinamide (only occasionally)
- Hydroxy/Adenosyl B12 drops
- Benfotamine (had stopped a few weeks prior to test)
- Lithium
- Metagenics Mineral complex only few times per week
- Tri-Fortify glutathione
- Argentyn23 and lauricidin