



# The Great Plains Laboratory, Inc.

William Shaw, Ph.D., Director

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Requisition #: 411947

Physician: ANDREW ROSTENBERG DC

Patient Name: Kevin Olheiser

Date of Collection: 11/7/2015

Patient Age: 36

Time of Collection: 07:00 AM

Patient Sex: M

Print Date: 11/16/2015



## 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	0.61	
2	5-Hydroxymethyl-2-furoic	≤ 18	1.5	
3	3-Oxoglutaric	≤ 0.11	0	
4	Furan-2,5-dicarboxylic	≤ 13	2.1	
5	Furancarbonylglycine	≤ 2.3	0.68	
6	Tartaric	≤ 5.3	0.47	
7	Arabinose	≤ 20	H 33	
8	Carboxycitric	≤ 20	13	
9	Tricarballic	≤ 0.58	0.11	

#### Bacterial Markers

10	Hippuric	≤ 241	H 429	
11	2-Hydroxyphenylacetic	0.03 - 0.47	H 0.64	
12	4-Hydroxybenzoic	0.01 - 0.73	0.72	
13	4-Hydroxyhippuric	≤ 14	5.1	
14	DHPPA (Beneficial Bacteria)	≤ 0.23	0.07	

#### Clostridia Bacterial Markers

15	4-Hydroxyphenylacetic ( <i>C. difficile</i> , <i>C. stricklandii</i> , <i>C. lituseburens</i> & others)	≤ 18	15	
16	HPHPA ( <i>C. sporogenes</i> , <i>C. caloritolerans</i> , <i>C. botulinum</i> & others)	≤ 102	64	
17	4-Cresol ( <i>C. difficile</i> )	≤ 39	29	
18	3-Indoleacetic ( <i>C. stricklandii</i> , <i>C. lituseburens</i> , <i>C. subterminale</i> & others)	≤ 6.8	1.8	

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

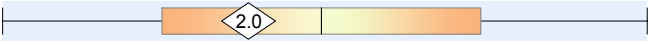
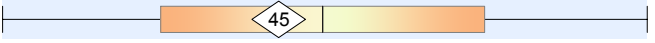

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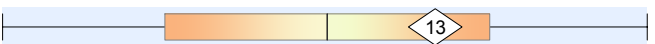
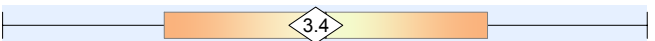
Physician: ANDREW ROSTENBERG DC  
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Metabolic Markers in Urine      Reference Range (mmol/mol creatinine)      Patient Value      Reference Population - Males Age 13 and Over

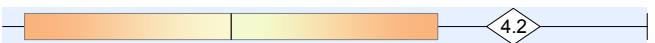


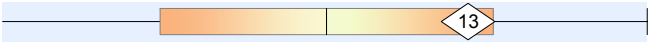
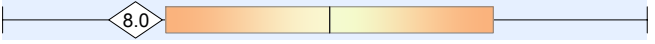
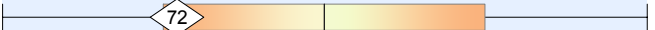
## Oxalate Metabolites

19	Glyceric	0.21 - 4.9	2.0	
20	Glycolic	18 - 81	45	
21	Oxalic	8.9 - 67	H 110	

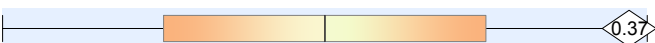
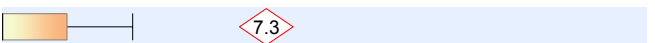
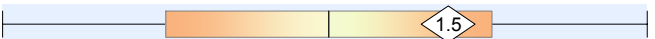
## Glycolytic Cycle Metabolites

22	Lactic	0.74 - 19	13	
23	Pyruvic	0.28 - 6.7	3.4	

## Mitochondrial Markers - Krebs Cycle Metabolites

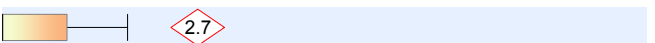
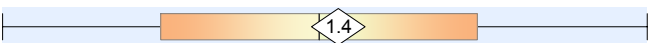
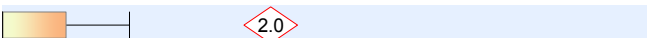
24	Succinic	≤ 5.3	4.2	
25	Fumaric	≤ 0.49	0.20	
26	Malic	≤ 1.1	0.43	
27	2-Oxoglutaric	≤ 18	13	
28	Aconitic	4.1 - 23	8.0	
29	Citric	2.2 - 260	72	

## Mitochondrial Markers - Amino Acid Metabolites

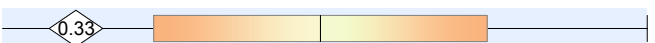
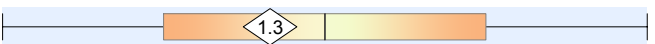
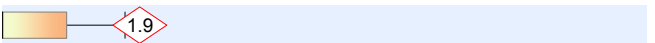
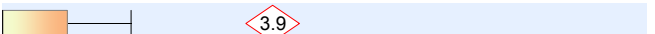
30	3-Methylglutaric	0.02 - 0.38	0.37	
31	3-Hydroxyglutaric	≤ 4.6	H 7.3	
32	3-Methylglutaconic	0.38 - 2.0	1.5	

## Neurotransmitter Metabolites

### Phenylalanine and Tyrosine Metabolites

33	Homovanillic (HVA) (dopamine)	0.39 - 2.2	H 2.7	
34	Vanillylmandelic (VMA) (norepinephrine, epinephrine)	0.53 - 2.2	1.4	
35	HVA / VMA Ratio	0.32 - 1.4	H 2.0	

### Tryptophan Metabolites

36	5-Hydroxyindoleacetic (5-HIAA) (serotonin)	≤ 2.9	0.33	
37	Quinolinic	0.52 - 2.4	1.3	
38	Kynurenic	0.12 - 1.8	H 1.9	
39	Quinolinic / 5-HIAA Ratio	≤ 2.5	H 3.9	

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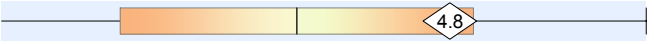
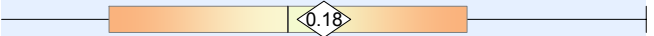
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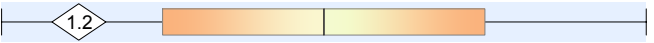

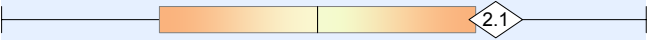

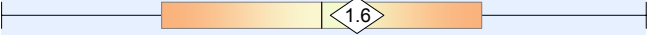

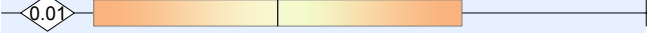
11/7/2015

Metabolic Markers in Urine	Reference Range (mmol/mol creatinine)	Patient Value	Reference Population - Males Age 13 and Over
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## Pyrimidine Metabolites - Folate Metabolism

40	Uracil	≤ 6.9	4.8	
41	Thymine	≤ 0.36	0.18	

## Ketone and Fatty Acid Oxidation

42	3-Hydroxybutyric	≤ 1.9	1.1	
43	Acetoacetic	≤ 10	1.2	
44	4-Hydroxybutyric	≤ 4.3	1.8	
45	Ethylmalonic	0.13 - 2.7	2.1	
46	Methylsuccinic	≤ 2.3	1.1	
47	Adipic	≤ 2.9	1.6	
48	Suberic	≤ 1.9	1.2	
49	Sebacic	≤ 0.14	0.01	

## Nutritional Markers

### Vitamin B12

50	Methylmalonic *	≤ 2.3	0.88	
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### Vitamin B6

51	Pyridoxic (B6)	≤ 26	3.0	
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### Vitamin B5

52	Pantothenic (B5)	≤ 5.4	H 6.1	
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### Vitamin B2 (Riboflavin)

53	Glutaric *	≤ 0.43	0.42	
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### Vitamin C

54	Ascorbic	10 - 200	L 0.43	
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### Vitamin Q10 (CoQ10)

55	3-Hydroxy-3-methylglutaric *	≤ 26	13	
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### Glutathione Precursor and Chelating Agent

56	N-Acetylcysteine (NAC)	≤ 0.13	0	
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### Biotin (Vitamin H)

57	Methylcitric *	0.15 - 1.7	0.31	
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\* A high value for this marker may indicate a deficiency of this vitamin.

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## Metabolic Markers in Urine

Reference Range  
(mmol/mol creatinine)

Patient  
Value

Reference Population - Males Age 13 and Over

### Indicators of Detoxification

#### Glutathione

58	Pyroglutamic *	5.7 - 25	H	27	
59	2-Hydroxybutyric *	≤ 1.2		1.2	

#### Ammonia Excess

60	Orotic	≤ 0.46		0.34	
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#### Aspartame, salicylates, or GI bacteria

61	2-Hydroxyhippuric	≤ 0.86		0.86	
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\* 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		0.44	
65	2-Hydroxyisocaproic	≤ 0.39		0	
66	2-Oxoisocaproic	≤ 0.34		0.10	
67	2-Oxo-4-methylbutyric	≤ 0.14		0.03	
68	Mandelic	≤ 0.09		0.08	
69	Phenyllactic	≤ 0.10		0.05	
70	Phenylpyruvic	0.02 - 1.4		0.77	
71	Homogentisic	≤ 0.23		0	
72	4-Hydroxyphenyllactic	≤ 0.62		0.13	
73	N-Acetylaspartic	≤ 2.5		0	
74	Malonic	≤ 9.9		4.9	

### Mineral Metabolism

75	Phosphoric	1 000 - 4 900		2 714	
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## Indicator of Fluid Intake

76 \*Creatinine 143 mg/dL

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

### Explanation of Report Format

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

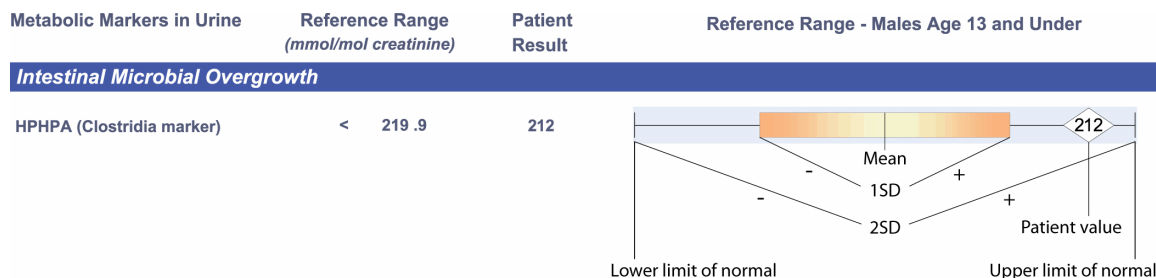
There are two types of graphical representations of patient values found in the new report format of both the standard Organic Acids Test and the Microbial Organic Acids Test.

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

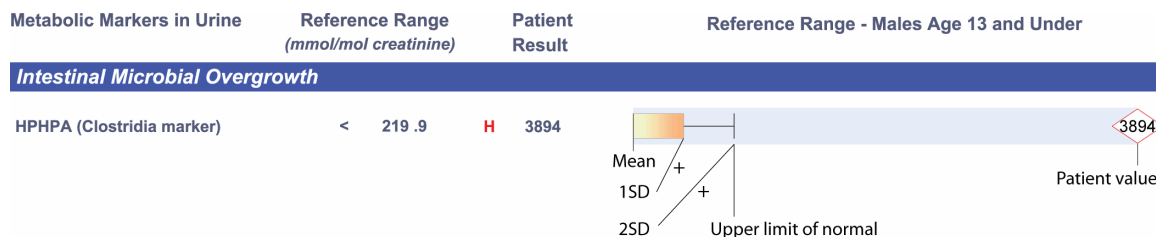
The second graph will occur when the value of the patient exceeds the upper limit of normal. In such cases, the graphical reference range is "shrunk" so that the degree of abnormality can be appreciated at a glance. In this case, the lower limits of normal are not shown, only the upper limit of normal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

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

### Example of Value Within Reference Range



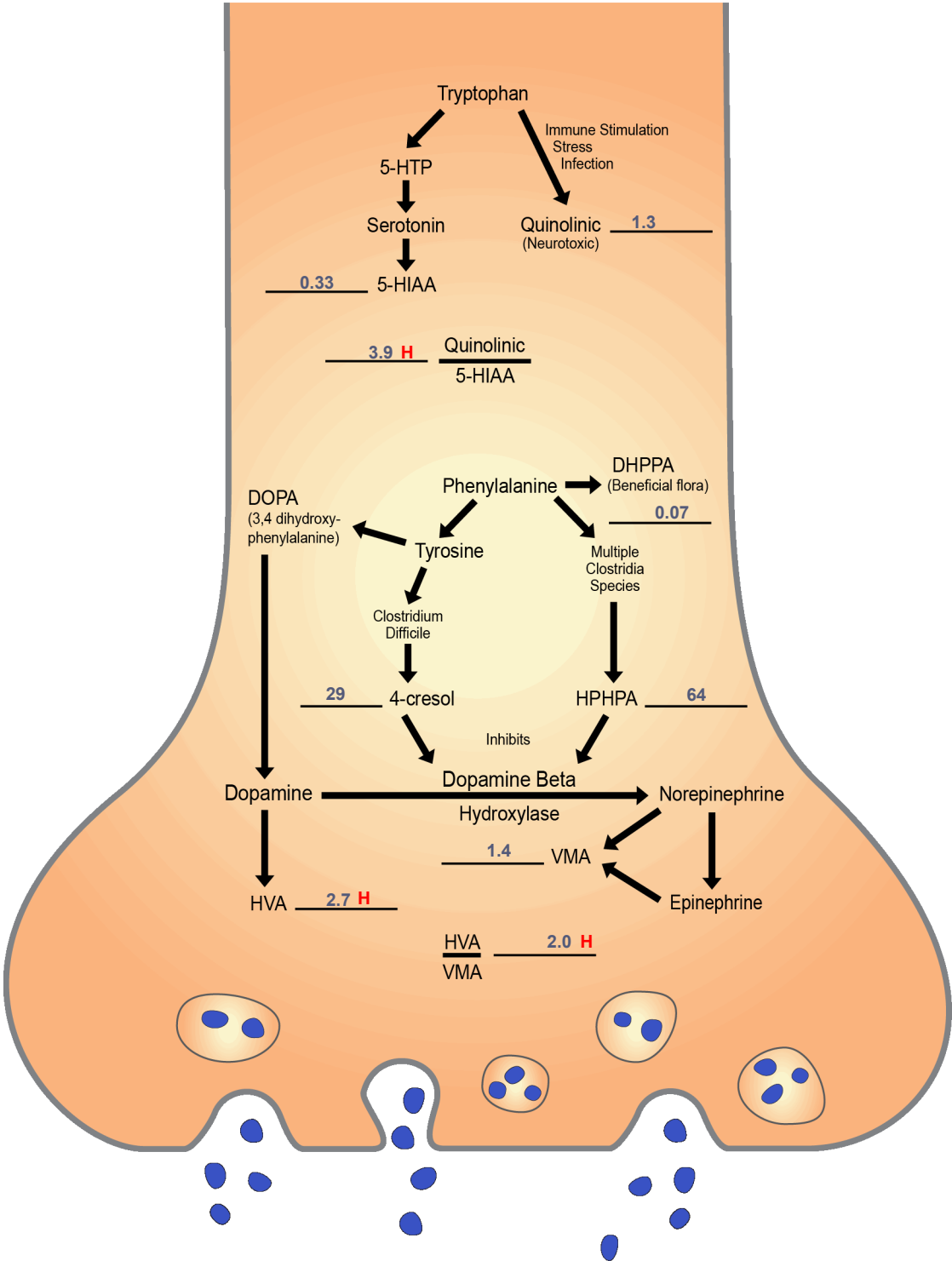
### Example of Elevated Value



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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.

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## 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 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.

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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 3-hydroxyglutaric (Marker 31)** is a metabolite associated with the genetic disease glutaric aciduria type I, which is due to a deficiency of glutaryl CoA dehydrogenase, an enzyme involved in the breakdown of lysine, hydroxylysine, and tryptophan. Other elevated organic acids may include glutaric and glutaconic acids. 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 have developed bleeding in the brain or eyes that may be mistaken for the effects of child abuse. This abnormality should be confirmed by additional testing of enzyme deficiencies and/or DNA at a pediatric medical genetics center (Morton et al., Am J. Med. Genetics **41**: 89-95, 1991). Elevated values may also be found in hepatic carnitine palmitoyltransferase I deficiency, short-chain acyl dehydrogenase deficiency (SCAD), or ketosis. Mitochondrial dysfunction induced by glutaric acid metabolites causes astrocytes to adopt a proliferative phenotype, which may underlie neuronal loss, white matter abnormalities and macrocephalia. Values in glutaric aciduria type I range from 60-3000 mmol/mol creatinine. Values higher than normal but less than 60 mmol/mol creatinine may be due to mild glutaric acidemia type I or to the other causes indicated above. Treatment of this disorder includes special diets low in lysine and supplementation with carnitine or acetyl-L-carnitine (1000-2000 mg/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.



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**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 HPPHA, 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 kynurenic acid (Marker 38)** may result from vitamin B-6 (pyridoxine) deficiency, immune stimulation or ingestion of tryptophan supplements. The kynurenine pathway is the main path of tryptophan metabolism. Although kynurenic acid may be elevated in vitamin B-6 (pyridoxine) deficiency, excretion of pyridoxic acid itself, as the major metabolite of B-6, is a much better marker for deficiency. Kynurenine (KYN) is the central compound of the pathway which splits into two separate branches: to kynurenic acid and to quinolinic acid, the precursor of the coenzyme NAD. Endogenous kynurenic acid is an antagonist to the excitatory amino acid alpha 7-nicotinic acetylcholine and to N-methyl-D-aspartate (NMDA) receptors. In several studies, kynurenic acid has been protective against the neurotoxic effects of quinolinic acid, which is a specific agonist of NMDA receptors and a potent producer of free radicals. The pathogenesis of several neurodegenerative disorders has been demonstrated to involve imbalances in the kynurenine pathway, including Alzheimer's disease, Parkinson disease, multiple sclerosis, and amyotrophic laterosclerosis (ALS).

**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.

**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.

# The Great Plains Laboratory, Inc.

Requisition #: 411947

Physician:

ANDREW ROSTENBERG DC

Patient Name: Kevin Olheiser

Date of Collection:

11/7/2015

## **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) <<http://www.NBNUS.com>> , or call 877-575-2467.

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

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