



Requisition #: 1048007  
 Patient Name: Naveed Aslam  
 Patient Age: 53  
 Patient Sex: M  
 Specimen Id.: 1048007-2

Practitioner: REGENERUS LAB  
 Date of Collection: 03/23/2022  
 Time of Collection: 07:00 AM  
 Print Date: 04/04/2022



## 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	1.1	
2 5-Hydroxymethyl-2-furoic (Aspergillus)	≤ 18	0.18	
3 3-Oxoglutaric	≤ 0.11	0.03	
4 Furan-2,5-dicarboxylic (Aspergillus)	≤ 13	0.25	
5 Furancarboxylglycine (Aspergillus)	≤ 2.3	0.21	
6 Tartaric (Aspergillus)	≤ 5.3	0.47	
7 Arabinose	≤ 20	8.7	
8 Carboxycitric	≤ 20	H 43	
9 Tricarballic (Fusarium)	≤ 0.58	0.28	

#### Bacterial Markers

10 Hippuric	≤ 241	178	
11 2-Hydroxyphenylacetic	0.03 - 0.47	0.26	
12 4-Hydroxybenzoic	≤ 0.73	H 3.4	
13 4-Hydroxyhippuric	≤ 14	H 97	
14 DHPPA (Beneficial Bacteria)	≤ 0.23	0.08	

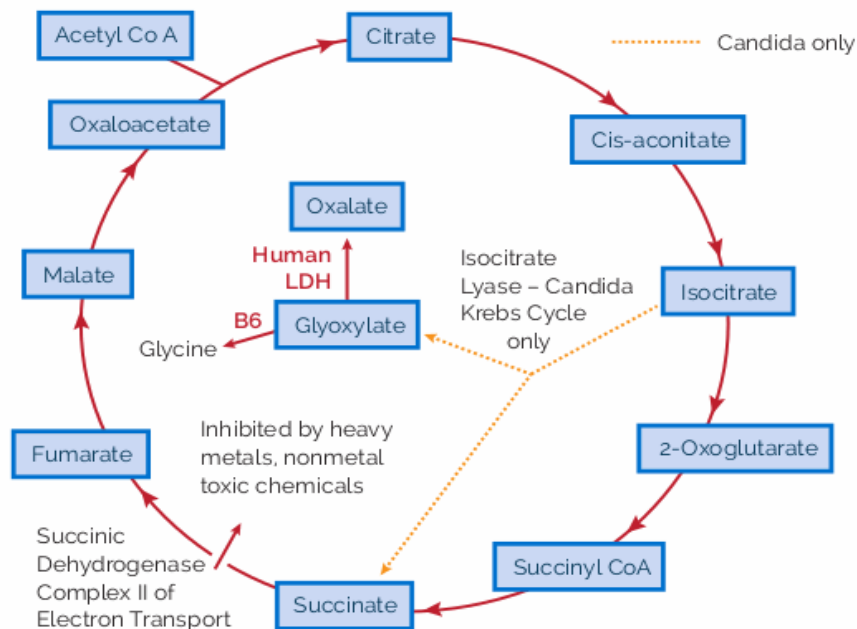
#### Clostridia Bacterial Markers

15 4-Hydroxyphenylacetic (C. difficile, C. stricklandii, C. lituseburens & others)	≤ 18	5.3	
16 HPPHA (C. sporogenes, C. caloritolerans, C. botulinum & others)	≤ 102	6.4	
17 4-Cresol (C. difficile)	≤ 39	4.8	
18 3-Indoleacetic (C. stricklandii, C. lituseburens, C. subterminale & others)	≤ 6.8	5.3	

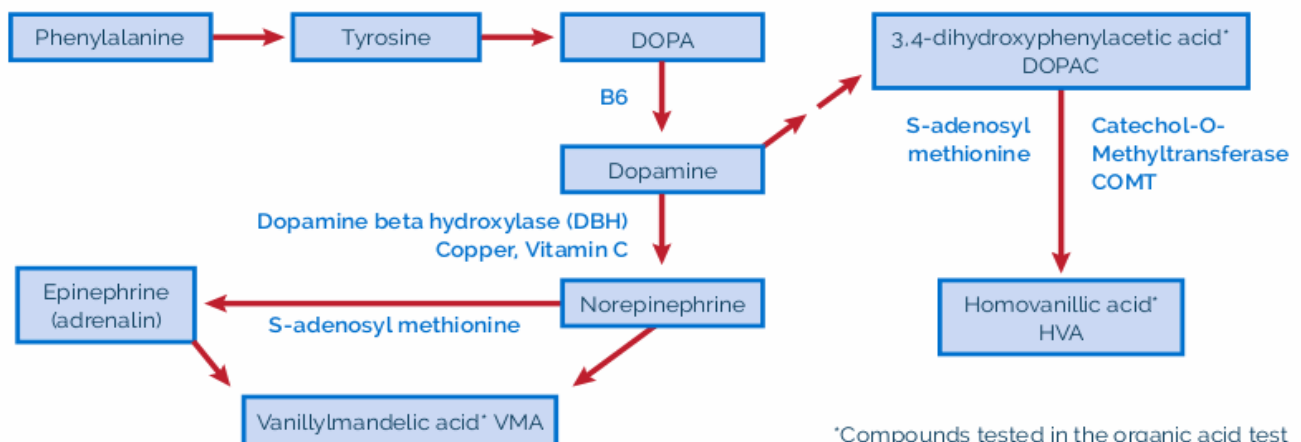
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## Human Krebs Cycle showing Candida Krebs Cycle variant that causes excess Oxalate via Glyoxylate



## Major pathways in the synthesis and breakdown of catecholamine neurotransmitters in the absence of microbial inhibitors



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## Oxalate Metabolites

19 Glyceric	0.21 - 4.9	4.1	
20 Glycolic	18 - 81	H 87	
21 Oxalic	8.9 - 67	H 79	

## Glycolytic Cycle Metabolites

22 Lactic	0.74 - 19	16	
23 Pyruvic	0.28 - 6.7	0.39	

## Mitochondrial Markers - Krebs Cycle Metabolites

24 Succinic	≤ 5.3	2.8	
25 Fumaric	≤ 0.49	0.21	
26 Malic	≤ 1.1	0.69	
27 2-Oxoglutaric	≤ 18	17	
28 Aconitic	4.1 - 23	5.0	
29 Citric	2.2 - 260	161	

## Mitochondrial Markers - Amino Acid Metabolites

30 3-Methylglutaric	0.02 - 0.38	H 0.41	
31 3-Hydroxyglutaric	≤ 4.6	2.9	
32 3-Methylglutaconic	0.38 - 2.0	1.1	

## Neurotransmitter Metabolites

### Phenylalanine and Tyrosine Metabolites

33 Homovanillic (HVA) (dopamine)	0.39 - 2.2	2.2	
34 Vanillylmandelic (VMA) (norepinephrine, epinephrine)	0.53 - 2.2	1.7	
35 HVA / VMA Ratio	0.32 - 1.4	1.3	
36 Dihydroxyphenylacetic (DOPAC) (dopamine)	0.27 - 1.9	H 3.1	
37 HVA / DOPAC Ratio	0.17 - 1.6	0.71	

### Tryptophan Metabolites

38 5-Hydroxyindoleacetic (5-HIAA) (serotonin)	≤ 2.9	1.7	
39 Quinolinic	0.52 - 2.4	1.7	
40 Kynurenic	≤ 1.8	0.68	



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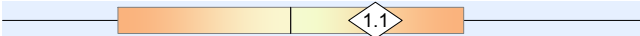
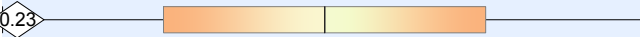
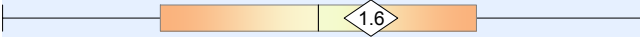
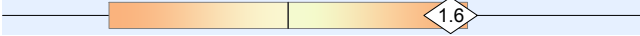
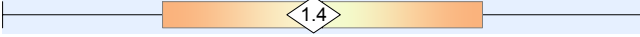
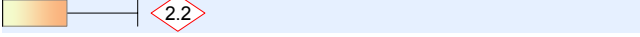
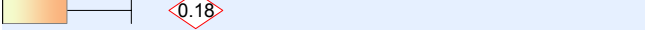
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Metabolic Markers in Urine      Reference Range (mmol/mol creatinine)      Patient Value      Reference Population - Males Age 13 and Over

## Pyrimidine Metabolites - Folate Metabolism

41 Uracil	≤ 6.9	3.1	
42 Thymine	≤ 0.36	0.08	

## Ketone and Fatty Acid Oxidation

43 3-Hydroxybutyric	≤ 1.9	1.1	
44 Acetoacetic	≤ 10	0.23	
45 Ethylmalonic	0.13 - 2.7	1.6	
46 Methylsuccinic	≤ 2.3	1.6	
47 Adipic	≤ 2.9	1.4	
48 Suberic	≤ 1.9	<b>H</b> 2.2	
49 Sebacic	≤ 0.14	<b>H</b> 0.18	

## Nutritional Markers

### Vitamin B12

50 Methylmalonic *	≤ 2.3	1.3	
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### Vitamin B6

51 Pyridoxic (B6)	≤ 26	1.2	
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### Vitamin B5

52 Pantothenic (B5)	≤ 5.4	1.3	
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### Vitamin B2 (Riboflavin)

53 Glutaric *	≤ 0.43	0.16	
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### Vitamin C

54 Ascorbic	10 - 200	<b>L</b> 1.3	
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### Vitamin Q10 (CoQ10)

55 3-Hydroxy-3-methylglutaric *	≤ 26	8.0	
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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	0.56	
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\* A high value for this marker may indicate a deficiency of this vitamin.

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## Indicators of Detoxification

### Glutathione

58 Pyroglutamic \*      5.7 - 25      23      

### Methylation, Toxic exposure

59 2-Hydroxybutyric \*\*      ≤ 1.2      H 1.8      

### Ammonia Excess

60 Orotic      ≤ 0.46      0.16      

### Aspartame, salicylates, or GI bacteria

61 2-Hydroxyhippuric      ≤ 0.86      0.66      

\* A high value for this marker may indicate a Glutathione deficiency.  
 \*\* High values may indicate methylation defects and/or toxic exposures.

## Amino Acid Metabolites

62 2-Hydroxyisovaleric      ≤ 2.0      0.25      

63 2-Oxoisovaleric      ≤ 2.0      0.08      

64 3-Methyl-2-oxovaleric      ≤ 2.0      0.10      

65 2-Hydroxyisocaproic      ≤ 2.0      0      

66 2-Oxoisocaproic      ≤ 2.0      0.02      

67 2-Oxo-4-methylbutyric      ≤ 2.0      0.07      

68 Mandelic      ≤ 2.0      0.11      

69 Phenyllactic      ≤ 2.0      0      

70 Phenylpyruvic      ≤ 2.0      0.02      

71 Homogentisic      ≤ 2.0      0.01      

72 4-Hydroxyphenyllactic      ≤ 2.0      0.14      

73 N-Acetylaspartic      ≤ 38      1.1      

74 Malonic      ≤ 9.9      6.5      

75 4-Hydroxybutyric      ≤ 4.3      0      

## Mineral Metabolism

76 Phosphoric      1,000 - 4,900      L 963      

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## Indicator of Fluid Intake

77 \*Creatinine 44 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).

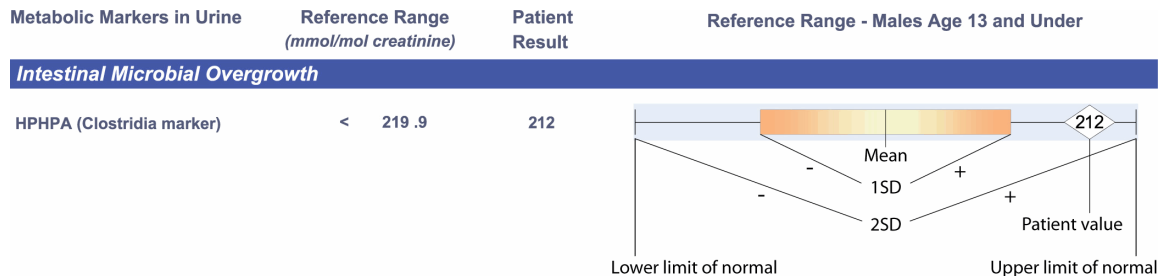
There are two types of graphical representations of patient values found in the new report format of both the standard Organic Acids Test and the Microbial Organic Acids Test.

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.

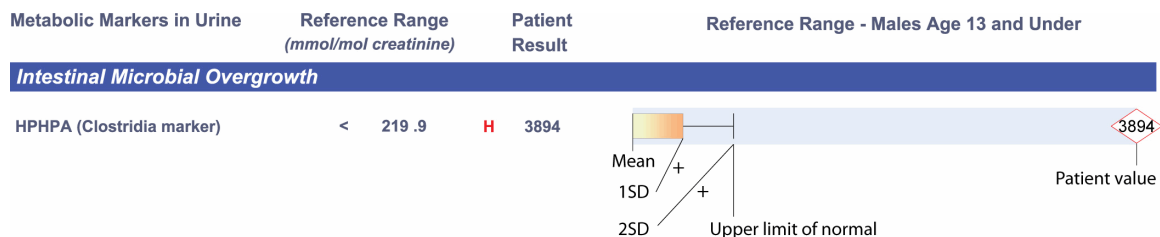
The second graph will occur when the value of the patient exceeds the upper limit of normal. In such cases, the graphical reference range is "shrunk" so that the degree of abnormality can be appreciated at a glance. In this case, the lower limits of normal are not shown, only the upper limit of normal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



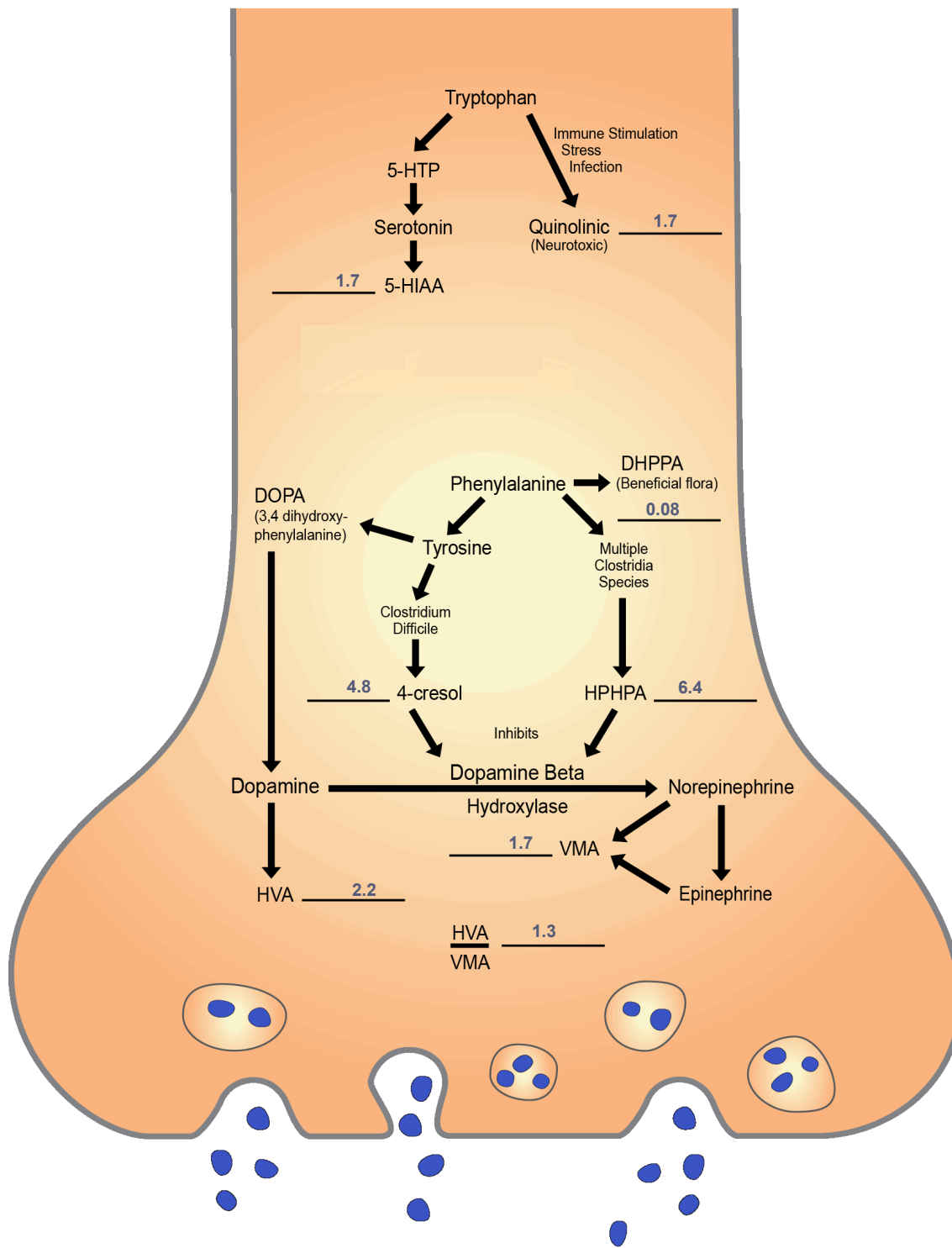
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## Neurotransmitter Metabolism Markers



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 (1-8)** Elevations of one or more metabolites indicate a yeast/fungal overgrowth of the gastrointestinal (GI) tract. Prescription or natural (botanical) anti-fungals, along with supplementation of high potency multi-strain probiotics, may reduce yeast/fungal levels.

**High 4-hydroxybenzoic acid and/or 4-hydroxyhippuric acid (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 Ca<sup>2+</sup>-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 glycolic (20):** in the absence of oxalic is most likely a result of GI yeast overgrowth (*Aspergillus*, *Penicillium*, *Candida*) or due to dietary sources containing glycerol/glycerine. Glycolic acid had also been found to be a metabolite in *Acetobacter*, *Acidithiobacillus*, *Alcanigenes*, *Corynebacterium*, *Cryptococcus*, *Escherichia*, *Gluconobacter*, *Kluyveromyces*, *Leptospirillum*, *Pichia*, *Rhodococcus*, *Rhodotorula* and *Saccharomyces* (PMID: 11758919; PMID: 26360870; PMID: 14390024).

**High oxalic (21) with or without elevated glyceric (19) or glycolic acids (20)** 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.



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<b>Specimen Id.:</b>	1048007-2		

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. In addition, oxalate deposits in the breast have been associated with breast cancer.

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.

People with abnormally high markers characteristic of the genetic diseases should do the following:

1. Avoid spinach, soy, nuts, and berries for one month.
2. If *Candida* is present, treat *Candida* for at least one month.
3. Repeat the organic acid test having abstained 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.

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**High 3-methylglutaric and/or high 3-methylglutaconic acids (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 quadriplegia). Recommendations include supplementation with coenzyme Q-10, L-carnitine and acetyl-L-carnitine, riboflavin, nicotinamide, and vitamin E.

**High 3,4-dihydroxyphenylacetic acid (DOPAC) (36)** 3,4-dihydroxyphenylacetic acid (DOPAC) is an intermediate in the metabolism of dopamine. Values may be elevated due to increased intake of amino acid precursors of DOPAC such as phenylalanine, tyrosine, or DOPA. Values may be elevated due to factors that inhibit dopamine beta hydroxylase (DBH) like Clostridia metabolites, the mold metabolite fusaric acid, pharmaceuticals such as disulfiram, or food additives like aspartame, or to deficiencies of the DBH enzyme due to copper deficiency, vitamin C deficiency, or malic acid deficiency. Single nucleotide polymorphisms (SNPs) of DBH or catechol-O-methyltransferase (COMT) that result in reduced enzyme activities also result in increased amounts of DOPAC. SNPs of COMT are available on **The Great Plains Laboratory DNA methylation pathway test** which can be performed on a cheek swab. Deficiencies of S-adenosylmethionine (S-ame) also are associated with high amounts of DOPAC. DOPAC may also be increased when bananas are ingested the day before urine collection.

**High ethylmalonic, methylsuccinic, adipic, suberic, or sebacic acids (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 may be beneficial.

**Pyridoxic acid (B6) levels below the mean (51)** may be associated with less than optimum health conditions (low intake, malabsorption, or dysbiosis). Supplementation with B6 or a multivitamin may be beneficial.

**Pantothenic acid (B5) levels below the mean (52)** may be associated with less than optimum health conditions. Supplementation with B5 or a multivitamin may be beneficial.

**Ascorbic acid (vitamin C) levels below the mean (54)** may indicate a less than optimum level of the antioxidant vitamin C. Individuals who consume large amounts of vitamin C can still have low values if the sample is taken 12 or more hours after intake. Supplementation with buffered vitamin C taken 2 or 3 times a day is suggested.

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**High 2-hydroxybutyric acid (59)** This organic acid is elevated when there is increased production of sulfur amino acids derived from homocysteine. The reasons for an increase can be due to the following reasons (which are not mutually exclusive):

1. There is increased need for glutathione to detoxify a host of toxic chemicals, resulting in increased shunting of homocysteine into the production of cysteine for glutathione. This is the most common reason.
2. There are genetic variants of the DNA such that methylation of homocysteine by betaine homocysteine methyl transferase or methionine synthase is impaired. . SNPs of genes in the methylation cycle are available on **The Great Plains DNA methylation pathway test** which can be performed on a cheek swab.
3. There are nutritional deficiencies of betaine, methylcobalamin, or methyltetrahydrofolate that reduce the enzyme activities of the enzymes in #2 above.
4. There is a genetic variant in cystathionine beta synthase (CBS) enzyme such that there is excessive shunting of homocysteine into cysteine production that results in excessive 2-hydroxybutyric acid formation.
5. Onset of diabetes mellitus or excessive alcohol use.
6. Presence of certain genetic diseases such as lactic acidosis, glutaric aciduria type II, dihydrolipoyl dehydrogenase (E3) deficiency, and propionic aciduria.

**Low phosphoric acid or its base conjugate phosphate (76)** is associated with hypoparathyroidism, pseudohypoparathyroidism, low nutritional phosphate intake (unusual on a Western diet), parathyroidectomy, and vitamin D deficiency. Phosphate excretion is directly proportional to dietary intake and is highly variable. Phosphate excretion is diurnal with lowest values occurring in the early morning. Testing for vitamin D status should be considered.

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.

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