

TEST NAME: Organic Acids Test (OAT)

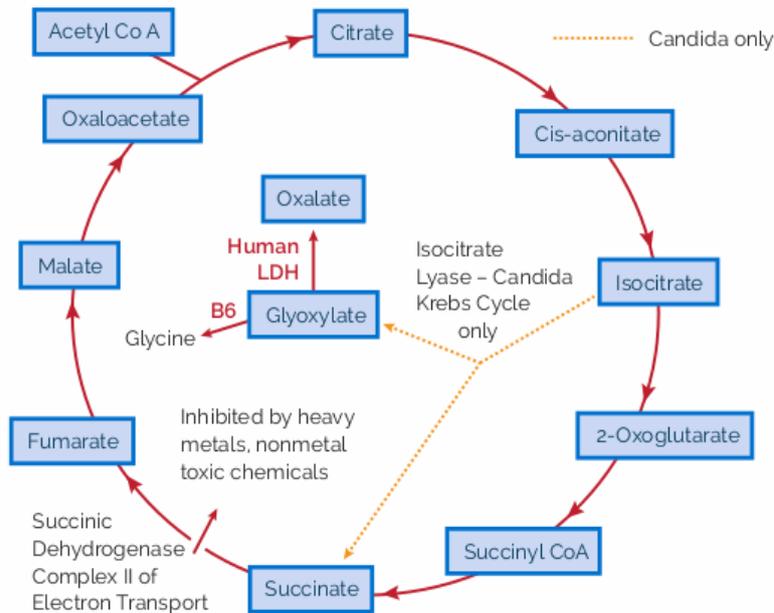


Organic Acids Test - Nutritional and Metabolic Profile

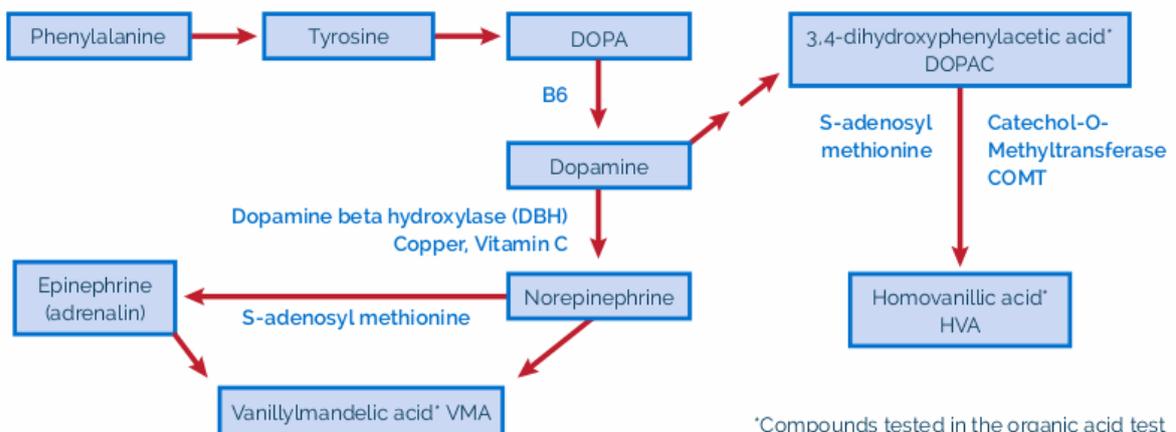
Metabolic Markers in Urine	Reference Range (mmol/mol creatinine)	Patient Value	Reference Population - Males Age 13 and Over
<b>Intestinal Microbial Overgrowth</b>			
<b>Yeast and Fungal Markers</b>			
1 Citramalic	0.11 - 2.0	0.70	
2 5-Hydroxymethyl-2-furoic <i>(Aspergillus)</i>	≤ 18	14	
3 3-Oxoglutaric	≤ 0.11	0.04	
4 Furan-2,5-dicarboxylic <i>(Aspergillus)</i>	≤ 13	10	
5 Furancarboxylglycine <i>(Aspergillus)</i>	≤ 2.3	0	
6 Tartaric <i>(Aspergillus)</i>	≤ 5.3	2.0	
7 Arabinose	≤ 20	H 48	
8 Carboxycitric	≤ 20	0.01	
9 Tricarballic <i>(Fusarium)</i>	≤ 0.58	0.19	
<b>Bacterial Markers</b>			
10 Hippuric	≤ 241	4.9	
11 2-Hydroxyphenylacetic	0.03 - 0.47	0.46	
12 4-Hydroxybenzoic	≤ 0.73	H 2.1	
13 4-Hydroxyhippuric	≤ 14	9.1	
14 DHPPA (Beneficial Bacteria)	≤ 0.23	H 0.24	
<b>Clostridia Bacterial Markers</b>			
15 4-Hydroxyphenylacetic <i>(C. difficile, C. stricklandii, C. lituseburense &amp; others)</i>	≤ 18	6.2	
16 HPPHA <i>(C. sporogenes, C. caloritolerans, C. botulinum &amp; others)</i>	≤ 102	41	
17 4-Cresol <i>(C. difficile)</i>	≤ 39	4.4	
18 3-Indoleacetic <i>(C. stricklandii, C. lituseburense, C. subterminale &amp; others)</i>	≤ 6.8	0.47	

TEST NAME: Organic Acids Test (OAT)

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



\*Compounds tested in the organic acid test

TEST NAME: Organic Acids Test (OAT)

Metabolic Markers in Urine	Reference Range (mmol/mol creatinine)	Patient Value	Reference Population - Males Age 13 and Over
<b>Oxalate Metabolites</b>			
19 Glyceric	0.21 - 4.9	1.6	
20 Glycolic	18 - 81	31	
21 Oxalic	8.9 - 67	<b>H</b> 117	
<b>Glycolytic Cycle Metabolites</b>			
22 Lactic	0.74 - 19	<b>H</b> 68	
23 Pyruvic	0.28 - 6.7	1.7	
<b>Mitochondrial Markers - Krebs Cycle Metabolites</b>			
24 Succinic	≤ 5.3	3.2	
25 Fumaric	≤ 0.49	0.07	
26 Malic	≤ 1.1	0.45	
27 2-Oxoglutaric	≤ 18	2.0	
28 Aconitic	4.1 - 23	7.9	
29 Citric	2.2 - 260	<b>H</b> 269	
<b>Mitochondrial Markers - Amino Acid Metabolites</b>			
30 3-Methylglutaric	0.02 - 0.38	0.09	
31 3-Hydroxyglutaric	≤ 4.6	2.6	
32 3-Methylglutaconic	0.38 - 2.0	0.57	
<b>Neurotransmitter Metabolites</b>			
<b>Phenylalanine and Tyrosine Metabolites</b>			
33 Homovanillic (HVA) (dopamine)	0.39 - 2.2	2.0	
34 Vanillylmandelic (VMA) (norepinephrine, epinephrine)	0.53 - 2.2	1.2	
35 HVA / VMA Ratio	0.32 - 1.4	<b>H</b> 1.7	
36 Dihydroxyphenylacetic (DOPAC) (dopamine)	0.27 - 1.9	1.1	
37 HVA/ DOPAC Ratio	0.17 - 1.6	<b>H</b> 1.8	
<b>Tryptophan Metabolites</b>			
38 5-Hydroxyindoleacetic (5-HIAA) (serotonin)	≤ 2.9	0.76	
39 Quinolinic	0.52 - 2.4	1.4	
40 Kynurenic	≤ 1.8	0.46	



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PRACTITIONER:

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RECEIVED: XX/XX/XXXX

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AGE: XX

TESTED: XX/XX/XXXX

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TEST NAME: Organic Acids Test (OAT)

Metabolic Markers in Urine	Reference Range (mmol/mol creatinine)	Patient Value	Reference Population - Males Age 13 and Over
<b>Pyrimidine Metabolites - Folate Metabolism</b>			
41 Uracil	≤ 6.9	3.4	
42 Thymine	≤ 0.36	0.17	
<b>Ketone and Fatty Acid Oxidation</b>			
43 3-Hydroxybutyric	≤ 1.9	0.93	
44 Acetoacetic	≤ 10	0.64	
45 Ethylmalonic	0.13 - 2.7	0.62	
46 Methylsuccinic	≤ 2.3	1.0	
47 Adipic	≤ 2.9	0.88	
48 Suberic	≤ 1.9	<b>H</b> 2.0	
49 Sebacic	≤ 0.14	0.14	
<b>Nutritional Markers</b>			
<b>Vitamin B12</b>			
50 Methylmalonic *	≤ 2.3	0.97	
<b>Vitamin B6</b>			
51 Pyridoxic (B6)	≤ 26	<b>H</b> 33	
<b>Vitamin B5</b>			
52 Pantothenic (B5)	≤ 5.4	<b>H</b> 6.0	
<b>Vitamin B2 (Riboflavin)</b>			
53 Glutaric *	≤ 0.43	0.16	
<b>Vitamin C</b>			
54 Ascorbic	10 - 200	<b>L</b> 1.4	
<b>Vitamin Q10 (CoQ10)</b>			
55 3-Hydroxy-3-methylglutaric *	≤ 26	9.2	
<b>Glutathione Precursor and Chelating Agent</b>			
56 N-Acetylcysteine (NAC)	≤ 0.13	0	
<b>Biotin (Vitamin H)</b>			
57 Methylcitric *	0.15 - 1.7	0.62	

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



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XXXXXXXXXXXXXXXXXX

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XXXXXXXXXXXXXXXXXXXXXXXXXX

TEST NAME: Organic Acids Test (OAT)

Metabolic Markers in Urine	Reference Range (mmol/mol creatinine)	Patient Value	Reference Population - Males Age 13 and Over
<b>Indicators of Detoxification</b>			
<b>Glutathione</b>			
58 Pyroglutamic *	5.7 - 25	19	
<b>Methylation, Toxic exposure</b>			
59 2-Hydroxybutyric ***	≤ 1.2	0.50	
<b>Ammonia Excess</b>			
60 Orotic	≤ 0.46	0.29	
<b>Aspartame, salicylates, or GI bacteria</b>			
61 2-Hydroxyhippuric	≤ 0.86	0.70	

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

<b>Amino Acid Metabolites</b>			
62 2-Hydroxyisovaleric	≤ 2.0	0.18	
63 2-Oxoisovaleric	≤ 2.0	0.02	
64 3-Methyl-2-oxovaleric	≤ 2.0	0.07	
65 2-Hydroxyisocaproic	≤ 2.0	0.27	
66 2-Oxoisocaproic	≤ 2.0	0.04	
67 2-Oxo-4-methiolbutyric	≤ 2.0	0.10	
68 Mandelic	≤ 2.0	0.13	
69 Phenyllactic	≤ 2.0	0	
70 Phenylpyruvic	≤ 2.0	0	
71 Homogentisic	≤ 2.0	0.01	
72 4-Hydroxyphenyllactic	≤ 2.0	0.18	
73 N-Acetylaspartic	≤ 38	1.3	
74 Malonic	≤ 9.9	2.8	
75 4-Hydroxybutyric	≤ 4.3	1.6	

<b>Mineral Metabolism</b>			
76 Phosphoric	1,000 - 4,900	L 685	

**TEST NAME: Organic Acids Test (OAT)**

**Indicator of Fluid Intake**

77 \*Creatinine 117 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).

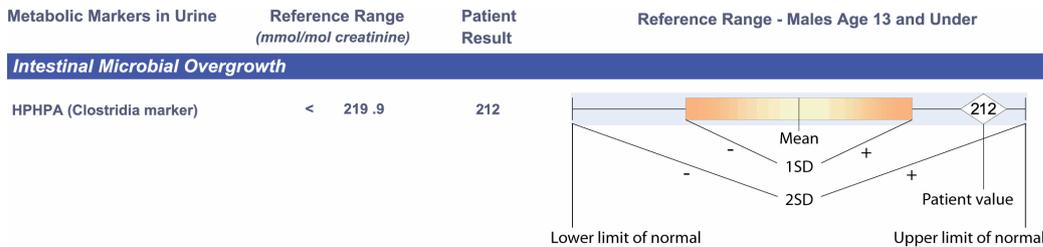
There are two types of graphical representations of patient values found in the new report format of both the standard Organic Acids Test and the Microbial Organic Acids Test.

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.

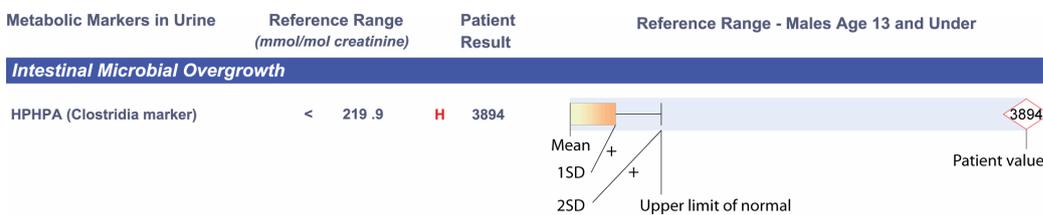
The second graph will occur when the value of the patient exceeds the upper limit of normal. In such cases, the graphical reference range is "shrunk" so that the degree of abnormality can be appreciated at a glance. In this case, the lower limits of normal are not shown, only the upper limit of normal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

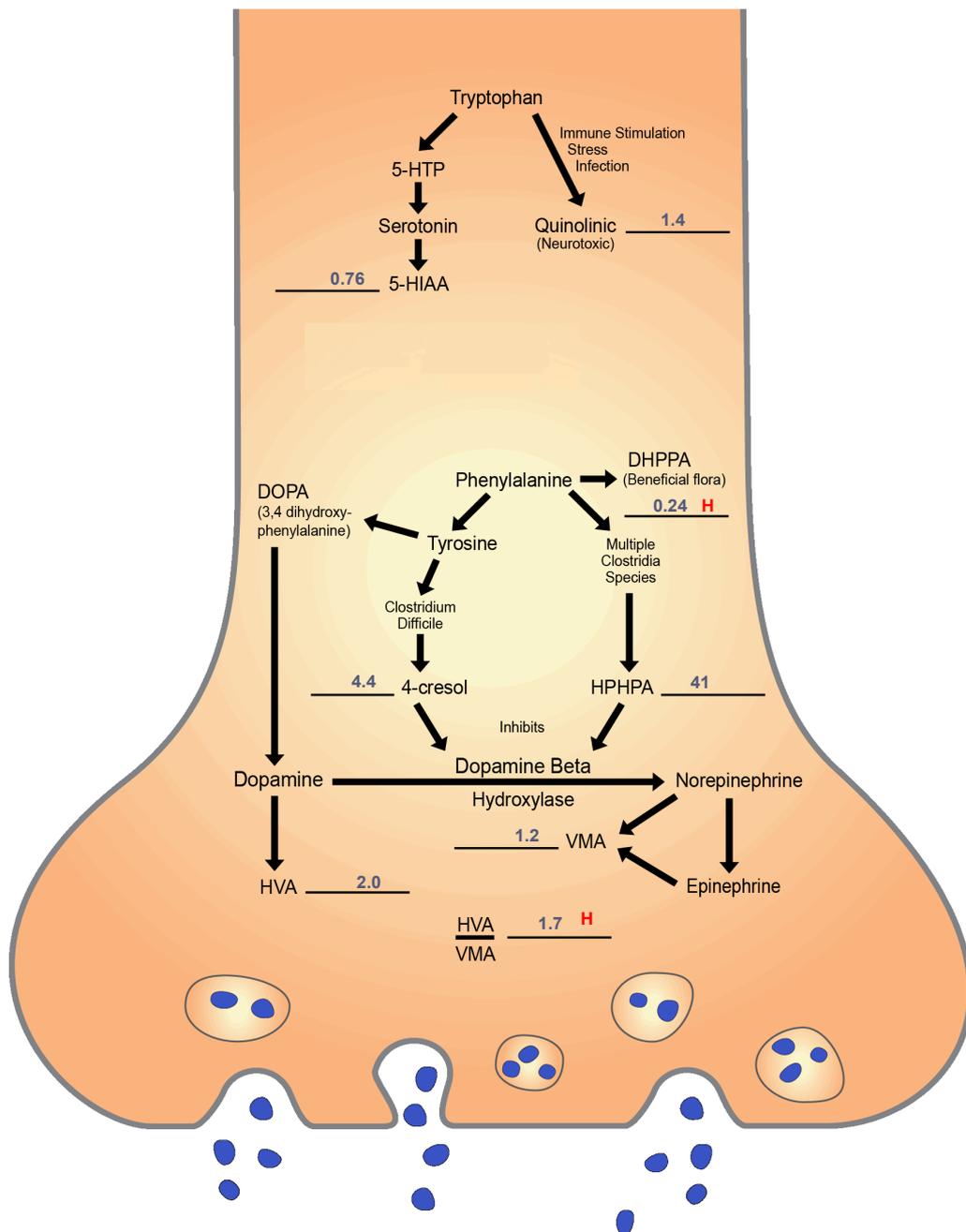


**Example of Elevated Value**



TEST NAME: Organic Acids Test (OAT)

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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AGE: XX	TESTED: XX/XX/XXXX
	PRACTITIONER: XXXXXXXXXXXXXXXXXX XXXXXXXXXXXXXXXXXXXXXX

## TEST NAME: Organic Acids Test (OAT)

### 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 DHPPA (3,4 dihydroxyphenylpropionic acid) (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.



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**TEST NAME: Organic Acids Test (OAT)**

**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.

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. Plasma oxalate values greater than 50 micromol/L are consistent with genetic oxalate diseases and may serve as an alternate confirmation test.



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## TEST NAME: Organic Acids Test (OAT)

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.

**High lactic acid and/or high pyruvic acid (22,23)** may be caused by many nonspecific factors, such as vigorous exercise, bacterial overgrowth of the GI tract, shock, poor perfusion, anemia, mitochondrial dysfunction or damage, and many other causes. Conversion of pyruvic acid to acetyl-CoA requires the cofactors coenzyme A (derived from pantothenic acid), lipoic acid, FAD derived from riboflavin, and thiamine. However, the possibility of an inborn error of metabolism increases as the value exceeds 300 mmol/mol creatinine. Values greater than 1000 mmol/mol creatinine indicate a much higher likelihood of an inborn error of metabolism. There are many inborn errors of metabolism that present elevated lactic acid, including disorders of sugar metabolism and pyruvate dehydrogenase deficiency.

**High citric acid (29)** may be due to increased intake of foods containing citric acid or as a result of intestinal yeast that either produce citric acid or perhaps inhibit the human citric acid cycle.

**Vanillylmandelic acid (VMA) levels (34) below the mean** indicate low production and/or decreased metabolism of the neurotransmitters norepinephrine and epinephrine. Vanillylmandelic acid is a metabolite of the neurotransmitters norepinephrine and epinephrine. Low production of VMA can be due to decreased intake or absorption of norepinephrine's and epinephrine's precursor amino acids such as phenylalanine and/or tyrosine, decreased quantities of cofactors needed for biosynthesis of norepinephrine and epinephrine such as tetrahydrobiopterin and vitamin B6 coenzyme or decreased amounts of cofactors such as S-adenosylmethionine (Sam-e) needed to convert norepinephrine and epinephrine to VMA. In addition, a number of genetic variations such as single nucleotide polymorphisms (SNPs) or mutations in MAO or COMT genes can cause reduced production of VMA. VMA values below the mean but which are much lower than HVA values are usually due to impairment of dopamine beta hydroxylase due to Clostridia metabolites, the mold metabolite fusaric acid, pharmaceuticals such as disulfiram, or food additives like aspartame or deficiencies of cofactors such as vitamin C or copper. Values may be decreased in patients on monoamine oxidase (MAO) inhibitors. Another cause for a low VMA value is a genetic variation (single nucleotide polymorphism or SNP) of the DBH enzyme. Patients with low VMA due to Clostridia metabolites or genetic DBH deficiency should not be supplemented with phenylalanine, tyrosine, or L-DOPA.



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TEST NAME: Organic Acids Test (OAT)

**High HVA/VMA ratio (35)** the HVA/VMA ratio reflects the balance between dopamine and norepinephrine/epinephrine production by catecholamine producing neurons in the central nervous system, sympathetic nervous system, and adrenal gland. The most common reason for an elevation of the HVA/VMA ratio is a decreased conversion of dopamine to norepinephrine. The enzyme responsible for this conversion, dopamine beta-hydroxylase (DBH), is copper and vitamin C dependent so an elevated ratio could be due to deficiencies of these cofactors. **The most common reason** for this elevated ratio is inhibition of this enzyme by Clostridia byproducts including HPHPA, 4-cresol, or 4-hydroxyphenylacetic acid. Other causes of an increased ratio include inhibition of DBH by the mold metabolite fusaric acid, pharmaceuticals such as disulfiram, or food additives like aspartame. Another cause for an elevated ratio is a genetic variation (single nucleotide polymorphism or SNP) of the DBH enzyme. Alternatively, the activity of the DBH enzyme can be measured on blood serum. Individuals with low DBH activity can be treated with the drug Droxidopa™, which provides adequate norepinephrine by an alternate biochemical pathway. High ratios are common in a large number of neuropsychiatric diseases regardless of the reason for DBH deficiency.

**High HVA/DOPAC ratio (37)** HVA and DOPAC are the major metabolites of dopamine. An increase in the conversion of DOPAC to HVA might be due to excessive supplementation of S-adenosyl methionine (S-ame) and/or supplements such as methyltetrahydrofolate or methylcobalamin that increase endogenous Sam-e.

**5-hydroxyindoleacetic acid (5HIAA) (38) levels below the mean** may indicate lower production and/or decreased metabolism of the neurotransmitter serotonin. 5-hydroxy-indoleacetic acid is a metabolite of serotonin. Low values have been correlated with symptoms of depression. Low production of 5HIAA can be due to decreased intake or absorption of serotonin's precursor amino acid tryptophan, decreased quantities of cofactors needed for biosynthesis of serotonin such as tetrahydrobiopterin and vitamin B6 coenzyme. In addition, a number of genetic variations such as single nucleotide polymorphisms (SNPs) or mutations can cause reduced production of 5HIAA. Values may be decreased in patients on monoamine oxidase (MAO) inhibitors that are drugs or foods that contain tyramine such as Chianti wine and vermouth, fermented foods such as cheeses, fish, bean curd, sausage, bologna, pepperoni, sauerkraut, and salami.

**Slight elevation in suberic acid (48)** is consistent with overnight fasting or increased fat in the diet. Regardless of cause, supplementation with L-carnitine or acetyl-L-carnitine may be beneficial.

**High pyridoxic acid (51)** indicates high recent intake of vitamin B6. Pyridoxic acid is a major metabolite of vitamin B6. Because some individuals may require very high doses of vitamin B6, high values do not necessarily indicate the need to reduce vitamin B6 intake.



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AGE: XX	TESTED: XX/XX/XXXX	XXXXXXXXXXXXXXXXXXXXX

**TEST NAME: Organic Acids Test (OAT)**

**High pantothenic acid (B5) (52)** most commonly indicates recent intake of pantothenic acid as a supplement. Pantothenic acid is an essential B vitamin that is converted to coenzyme A (unrelated to vitamin A). Coenzyme A is needed for the synthesis of fatty acids, cholesterol, and acetyl choline and is also needed for the Krebs cycle and fatty acid catabolism. Because some individuals may require high doses of pantothenic acid, high values do not necessarily indicate the need to reduce pantothenic acid intake. However, if a patient who **does not take B-vitamin** supplements has high values of pantothenic acid, especially if the values are 20 or more times the upper limit of normal, the individual may have a genetic deficiency in the conversion of pantothenic acid to pantothenic acid-phosphate, which is the first step in the production of coenzyme A. It may be useful to retest after one week off all B-vitamin supplementation; individuals with PKAN would be expected to still have very elevated pantothenic acid levels even with no supplementation. This disease is called pantothenate kinase-associated neurodegeneration (PKAN), an inborn error of metabolism characterized by iron accumulation in the basal ganglia and by the presence of dystonia, dysarthria, Parkinson symptoms, and retinal degeneration. In mild variants of this disease, psychiatric illnesses such as schizoaffective disorder, hallucinations, obsessive compulsive disorder, speech defects, and depression are common. Mutations in pantothenate kinase 2 (PANK2), the rate-limiting enzyme in mitochondrial coenzyme A biosynthesis, represent the most common genetic cause of this disorder. Other biochemical abnormalities commonly found on the organic acid test in this disorder include elevated lactate, pyruvate, and Krebs cycle intermediates. Confirmation of mutant DNA requires special genetic testing.

Treatment for the illness is currently focused on giving high doses of pantothenic acid to stimulate any residual enzyme. Doses as high as 10 g per day have been ingested with few side effects. Other suggested therapies are increased supplementation with cholesterol, fat soluble vitamins, and bile salts. Since Lactobacillus species produce pantothenic acid phosphate, supplementation with high doses of probiotics might also 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.

**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.