MOSAIC DIAGNOSTICS

OAT ORGANIC ACIDS TEST

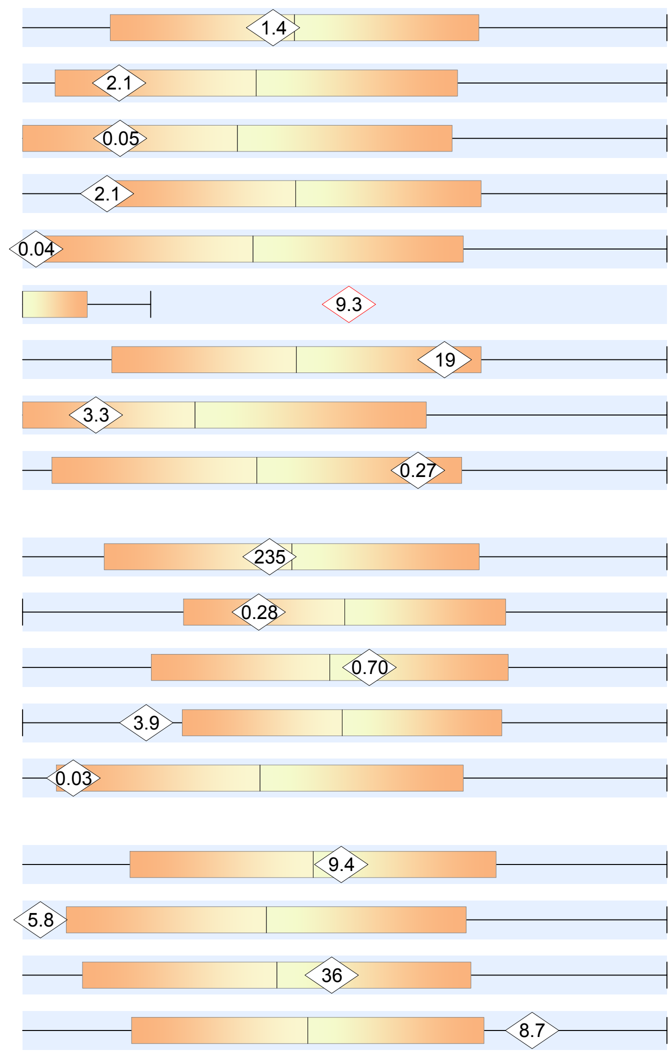
Requisition #:	[redacted]			*Practitioner:*	Marsha Meyers
Patient Name:				*Date of Collection:*	06/04/2024
Date of Birth:		*Patient Age:*	73	*Time of Collection:*	04:08 AM
Patient Sex:	F			*Report Date:*	06/14/2024

Organic Acids Test - Nutritional and Metabolic Profile

Intestinal Microbial Overgrowth

Metabolic Markers in Urine	Reference Range (mmol/mol creatinine)		Patient Value
Yeast and Fungal Markers			
1 Citramalic		≤ 3.6	1.4
2 5-Hydroxymethyl-2-furoic *(Aspergillus)*		≤ 14	2.1
3 3-Oxoglutaric		≤ 0.33	0.05
4 Furan-2,5-dicarboxylic *(Aspergillus)*		≤ 16	2.1
5 Furancarbonylglycine *(Aspergillus)*		≤ 1.9	0.04
6 Tartaric *(Aspergillus)*		≤ 4.5	H 9.3
7 Arabinose		≤ 29	19
8 Carboxycitric		≤ 29	3.3
9 Tricarballylic *(Fusarium)*		≤ 0.44	0.27
Bacterial Markers			
10 Hippuric		≤ 613	235
11 2-Hydroxyphenylacetic	0.06	- 0.66	0.28
12 4-Hydroxybenzoic		≤ 1.3	0.70
13 4-Hydroxyhippuric	0.79	- 17	3.9
14 DHPPA (Beneficial Bacteria)		≤ 0.38	0.03
Clostridia Bacterial Markers			
15 4-Hydroxyphenylacetic *(C. difficile, C. stricklandii, C. lituseburense & others)*		≤ 19	9.4
16 HPHPA *(C. sporogenes, C. caloritolerans, C. botulinum & others)*		≤ 208	5.8
17 4-Cresol *(C. difficile)*		≤ 75	36
18 3-Indoleacetic *(C. stricklandii, C. lituseburense, C. subterminale & others)*		≤ 11	8.7

Reference Population - Females Age 13 and Over



This test was developed, and its performance characteristics determined by Mosaic Diagnostics Laboratory. It has not been cleared or approved by the US Food and Drug Administration, however, does comply with CLIA regulations for clinical use.

The results should be interpreted in conjunction with the complete clinical picture, given patient history and presentation, and at the discretion of the medical provider.

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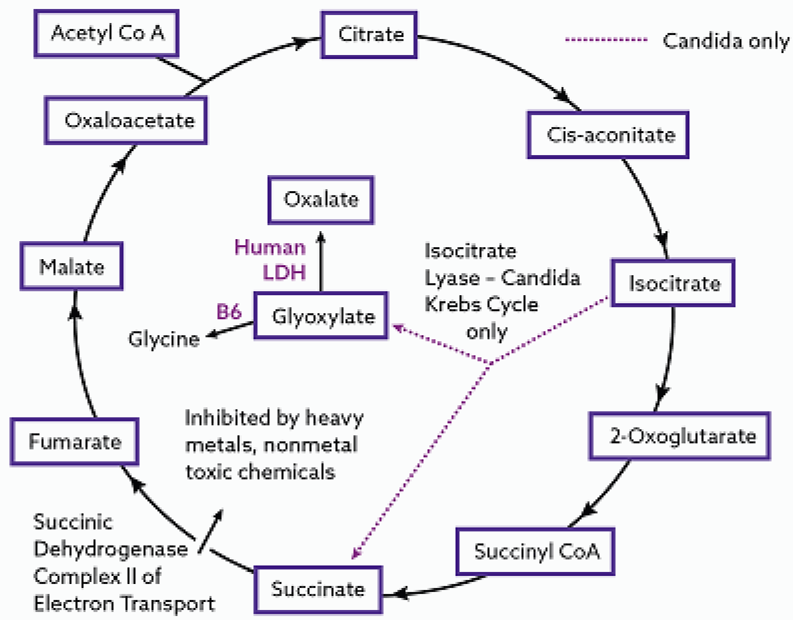
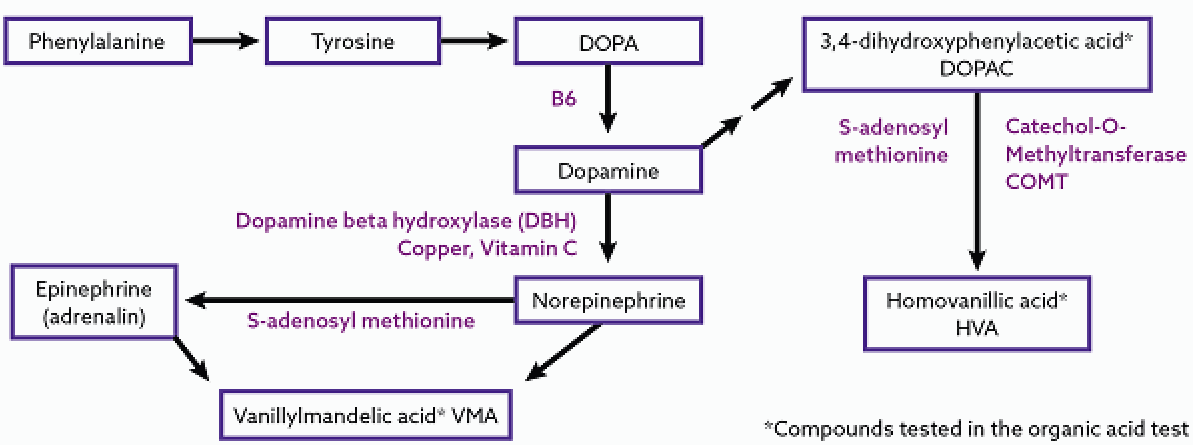
Human Krebs Cycle showing Candida Krebs Cycle variant that causes excess Oxalate via Glyoxylate



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



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Metabolic Markers in Urine	Reference Range (mmol/mol creatinine)		Patient Value	Reference Population - Females Age 13 and Over
Oxalate Metabolites				
19 Glyceric	0.77 - 7.0		4.4	4.4
20 Glycolic	16 - 117		49	49
21 Oxalic	6.8 - 101	H	186	186
Glycolytic Cycle Metabolites				
22 Lactic	≤ 48		29	29
23 Pyruvic	≤ 9.1		1.9	1.9
Mitochondrial Markers - Krebs Cycle Metabolites				
24 Succinic	≤ 9.3	H	45	45
25 Fumaric	≤ 0.94	H	6.3	6.3
26 Malic	0.06 - 1.8	H	11	11
27 2-Oxoglutaric	≤ 35		14	14
28 Aconitic	6.8 - 28		20	20
29 Citric	≤ 507		287	287
Mitochondrial Markers - Amino Acid Metabolites				
30 3-Methylglutaric	≤ 0.76		0.21	0.21
31 3-Hydroxyglutaric	≤ 6.2		3.4	3.4
32 3-Methylglutaconic	≤ 4.5		1.1	1.1
Neurotransmitter Metabolites				
Phenylalanine and Tyrosine Metabolites				
33 Homovanillic (HVA) *(dopamine)*	0.80 - 3.6		2.1	2.1
34 Vanillylmandelic (VMA) *(norepinephrine, epinephrine)*	0.46 - 3.7		2.1	2.1
35 HVA / VMA Ratio	0.16 - 1.8		1.0	1.0
36 Dihydroxyphenylacetic (DOPAC) *(dopamine)*	0.08 - 3.5		2.2	2.2
37 HVA/ DOPAC Ratio	0.10 - 1.8		0.95	0.95
Tryptophan Metabolites				
38 5-Hydroxyindoleacetic (5-HIAA) *(serotonin)*	≤ 4.3		0.72	0.72
39 Quinolinic	0.85 - 3.9		1.7	1.7
40 Kynurenic	≤ 2.2		0.75	0.75

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Patient Name:	[redacted]	*Date of Collection:*	06/04/2024

Metabolic Markers in Urine	Reference Range *(mmol/mol creatinine)*		Patient Value
Pyrimidine Metabolites - Folate Metabolism			
41 Uracil		≤ 9.7	6.9
42 Thymine		≤ 0.56	0.25
Ketone and Fatty Acid Oxidation			
43 3-Hydroxybutyric		≤ 3.1	1.3
44 Acetoacetic		≤ 10	0.93
45 Ethylmalonic	0.44	- 2.8	H 3.0
46 Methylsuccinic	0.10	- 2.2	1.7
47 Adipic	0.04	- 3.8	1.1
48 Suberic	0.18	- 2.2	1.8
49 Sebacic		≤ 0.24	0.24
Nutritional Markers			
Vitamin B12			
50 Methylmalonic ✱		≤ 2.3	0.95
Vitamin B6			
51 Pyridoxic (B6)		≤ 34	3.3
Vitamin B5			
52 Pantothenic (B5)		≤ 10	2.8
Vitamin B2 (Riboflavin)			
53 Glutaric ✱	0.04	- 0.36	0.20
Vitamin C			
54 Ascorbic	10	- 200	L 0.69
Vitamin Q10 (CoQ10)			
55 3-Hydroxy-3-methylglutaric ✱	0.17	- 39	22
Glutathione Precursor and Chelating Agent			
56 N-Acetylcysteine (NAC)		≤ 0.28	0
Biotin (Vitamin H)			
57 Methylcitric ✱	0.19	- 2.7	0.66

Reference Population - Females Age 13 and Over

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

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Patient Name: [redacted] **Date of Collection:** 06/04/2024

Metabolic Markers in Urine	Reference Range (mmol/mol creatinine)		Patient Value	Reference Population - Females Age 13 and Over
Indicators of Detoxification				
Glutathione				
58 Pyroglutamic ✱	10 - 33		16	16
Methylation, Toxic exposure				
59 2-Hydroxybutyric ✱✱	0.03 - 1.8		0.77	0.77
Ammonia Excess				
60 Orotic	0.06 - 0.54		0.36	0.36
Aspartame, salicylates, or GI bacteria				
61 2-Hydroxyhippuric	≤ 1.3	H	2.3	2.3

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

✱✱ ***High values may indicate methylation defects and/or toxic exposures.***

Amino Acid Metabolites

Low values are not associated with inadequate protein intake and have not been demonstrated to indicate specific amino acid deficiencies.

Metabolic Markers in Urine	Reference Range (mmol/mol creatinine)	Patient Value	Reference Population - Females Age 13 and Over
62 2-Hydroxyisovaleric	≤ 2.0	0.09	0.09
63 2-Oxoisovaleric	≤ 2.1	0.07	0.07
64 3-Methyl-2-oxovaleric	≤ 2.0	0.09	0.09
65 2-Hydroxyisocaproic	≤ 2.0	0	0.00
66 2-Oxoisocaproic	≤ 2.0	0.06	0.06
67 2-Oxo-4-methiolbutyric	≤ 2.0	0.19	0.19
68 Mandelic	≤ 2.0	0.13	0.13
69 Phenyllactic	≤ 2.0	0.10	0.10
70 Phenylpyruvic	≤ 2.0	0	0.00
71 Homogentisic	≤ 2.0	0.07	0.07
72 4-Hydroxyphenyllactic	≤ 2.0	0.26	0.26
73 N-Acetylaspartic	≤ 38	1.7	1.7
74 Malonic	≤ 9.7	2.0	2.0
75 4-Hydroxybutyric	≤ 4.8	2.2	2.2

Mineral Metabolism

Metabolic Markers in Urine	Reference Range (mmol/mol creatinine)	Patient Value	Reference Population - Females Age 13 and Over
76 Phosphoric	1,000 - 5,000	1,911	1911

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Indicator of Fluid Intake

77	*Creatinine	68	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).

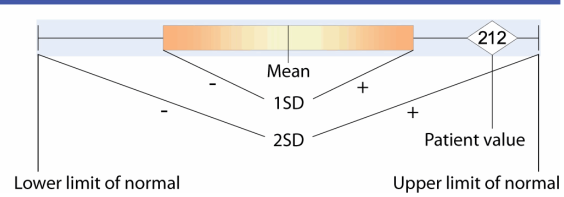
There are two types of graphical representations of patient values found in the new report format of both the standard Organic Acids Test and the Microbial Organic Acids Test.

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.

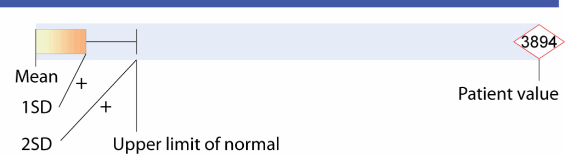
The second graph will occur when the value of the patient exceeds the upper limit of normal. In such cases, the graphical reference range is "shrunk" so that the degree of abnormality can be appreciated at a glance. In this case, the lower limits of normal are not shown, only the upper limit of normal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

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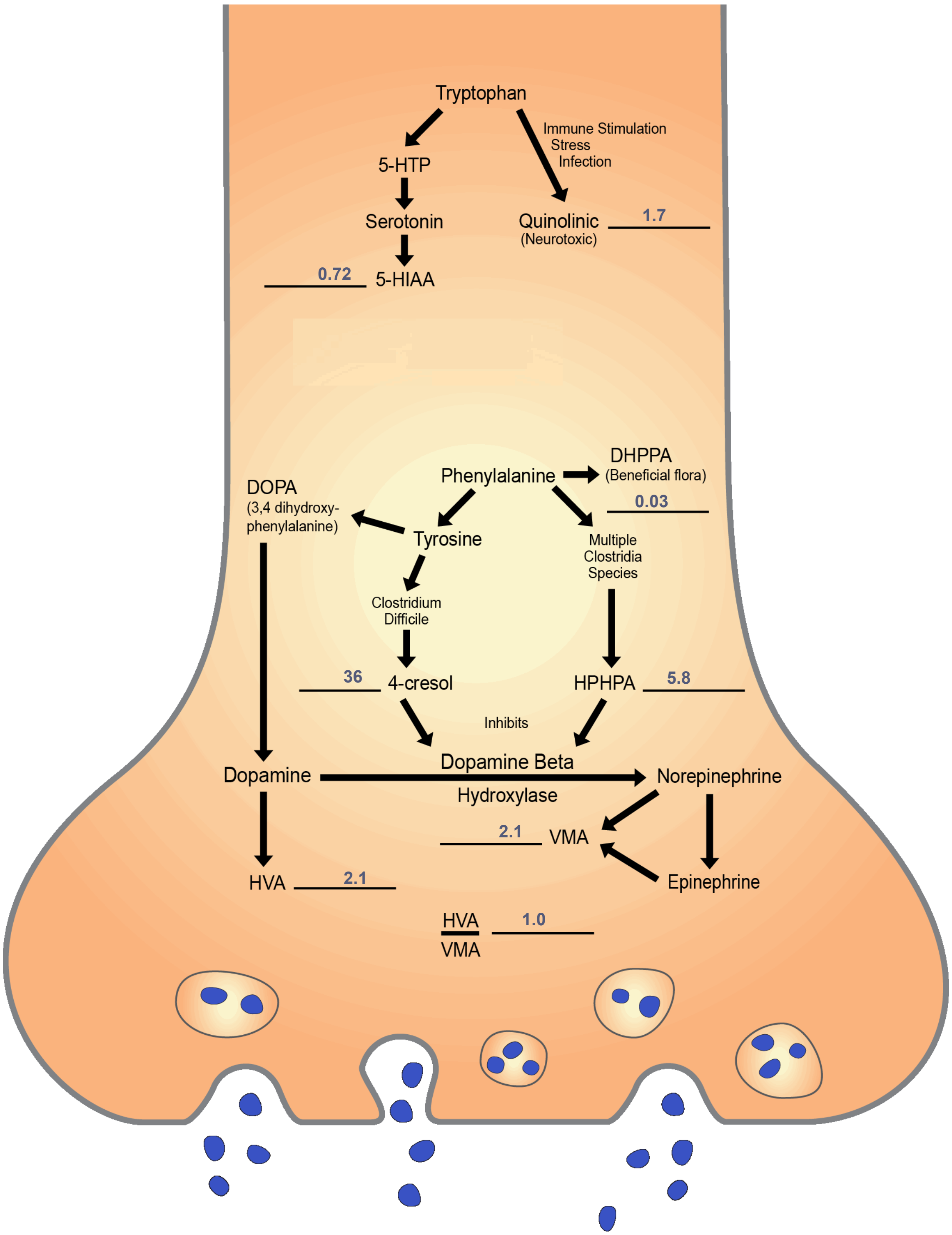
Neurotransmitter Metabolism Markers



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 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. 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.

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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 succinic acid (24) The most common cause of elevated succinic acid is exposure to toxic chemicals which impairs mitochondria function. The most useful tests for confirming toxic chemical exposure are **The Great Plains Laboratory GPL-TOX test** on urine for 172 chemicals and the hair metals test. Succinic acid is metabolized by the mitochondrial enzyme succinic dehydrogenase, which is significant in that it is both a Krebs cycle enzyme and a component- complex 2-of the mitochondrial electron transport chain, making this metabolite a marker of mitochondrial complex 2 as well as Krebs cycle dysfunction. A sampling of toxic chemicals that have been associated with mitochondrial dysfunction include glyphosate, 2, 4-dichlorophenoxyacetic acid (2, 4-D), organophosphate pesticides, mercury, and lead. Approximately 95% of elevated succinic acid results are associated with toxic chemical exposure. Succinic acid in the organic acid test and tiglylglycine in the **GPLTOX test** are two of the most useful markers for mitochondrial dysfunction. Tiglylglycine is a marker for mitochondrial respiratory chain complex I dysfunction while elevated succinic acid indicates respiratory complex 2 dysfunction. Occasionally both succinic acid and tiglylglycine may be elevated in mitochondrial dysfunction. Other Krebs cycle markers may also be elevated when severe chemical toxicity is present. In general, the severity of the chemical toxicity is correlated with higher values of succinic acid.

Less common causes of elevated succinic acid are mitochondrial mutations which may be due to mutations in the nuclear or the mitochondrial DNA for mitochondrial proteins such as Kearns-Sayres disorder. Succinic acid is a metabolite of gamma aminobutyric acid (GABA) so supplementation with GABA may also increase succinic acid.

High fumaric acid (25) may be due to impaired Krebs cycle function, defect of the enzyme fumarase or a defect in mitochondrial function. Recommendations for supporting mitochondrial function include supplementation with coenzyme Q10, L-carnitine or acetyl-L-carnitine, riboflavin, nicotinamide, and vitamin E.* All of these supplements are known to improve mitochondrial dysfunction.

High malic acid (26) indicates a greater requirement for the nutrients niacin and coenzyme Q10.* Malic acid simultaneously elevated with citric, fumaric and alpha-ketoglutaric acids may indicate a possible Cytochrome C Oxidase deficiency. Mitochondrial energy pathway dysfunction would be expected.

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Homovanillic acid (HVA) levels (33) below the mean indicate low production and/or decreased metabolism of the neurotransmitter dopamine. Homovanillic acid is a metabolite of the neurotransmitter dopamine. Low production of HVA can be due to decreased intake or absorption of dopamine's precursor amino acids such as phenylalanine and/or tyrosine, decreased quantities of cofactors needed for biosynthesis of dopamine such as tetrahydrobiopterin and vitamin B6 coenzyme or decreased amounts of cofactors such as S-adenosylmethionine (Sam-e) needed to convert dopamine to HVA. In addition, a number of genetic variations such as single nucleotide polymorphisms (SNPs) or mutations can cause reduced production of HVA due to enzymes with decreased function. HVA values below the mean but which are much higher than VMA values are usually due to impairment of dopamine beta hydroxylase due to excessive 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 also be decreased in patients on monoamine oxidase (MAO) inhibitors. 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 HVA. Such SNPs are available on **The Great Plains DNA methylation pathway test** which can be performed on a cheek swab.

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. Such SNPs are available on **The Great Plains DNA methylation pathway test** which can be performed on a cheek swab. Values may be decreased in patients on monoamine oxidase (MAO) inhibitors that are drugs or foods that contain tyramine such such as Chianti wine and vermouth, fermented foods such as cheeses, fish, bean curd, sausage, bologna, pepperoni, sauerkraut, and salami.

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.

High 2-hydroxyhippuric acid (61) may result from ingestion of aspartame (Nutrasweet®), salicylates (aspirin), dietary salicylates, or from GI bacteria converting tyrosine or phenylalanine to salicylic acid. For more information about salicylates in foods go to <http://www.feingold.org/salicylate.php>. 2-Hydroxyhippuric acid is a conjugate of hydroxybenzoic acid (salicylic acid) and glycine. Very high 2-hydroxyhippuric also inhibits dopamine beta-hydroxylase resulting in elevated HVA, decreased VMA, and elevated HVA/VMA ratio.

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.

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