MOSAIC DIAGNOSTICS

OAT ORGANIC ACIDS TEST

Requisition #:	9900001			*Practitioner:*	NO PHYSICIAN
Patient Name:	Sample Report			*Date of Collection:*	12/01/2022
Date of Birth:	12/01/2021	*Patient Age:*	1	*Time of Collection:*	Not Given
Patient Sex:	F			*Report Date:*	08/04/2023

Organic Acids Test - Nutritional and Metabolic Profile

Metabolic Markers in Urine	Reference Range (mmol/mol creatinine)		Patient Value	Reference Population - Females Under Age 13
Intestinal Microbial Overgrowth				
Yeast and Fungal Markers				
1 Citramalic	≤ 5.3		1.0	1.0
2 5-Hydroxymethyl-2-furoic *(Aspergillus)*	≤ 30		4.0	4.0
3 3-Oxoglutaric	≤ 0.52	H	2.0	2.0
4 Furan-2,5-dicarboxylic *(Aspergillus)*	≤ 22		0	0.00
5 Furancarbonylglycine *(Aspergillus)*	≤ 3.6		0	0.00
6 Tartaric *(Aspergillus)*	≤ 3.9	H	111	111
7 Arabinose	≤ 56		0	0.00
8 Carboxycitric	≤ 34		0	0.00
9 Tricarballylic *(Fusarium)*	≤ 0.86	H	5.0	5.0
Bacterial Markers				
10 Hippuric	≤ 717		1.0	1.0
11 2-Hydroxyphenylacetic	≤ 1.1	H	4.0	4.0
12 4-Hydroxybenzoic	0.09 - 2.0	L	0	0.00
13 4-Hydroxyhippuric	≤ 27		0	0.00
14 DHPPA (Beneficial Bacteria)	≤ 0.73	H	7.0	7.0
Clostridia Bacterial Markers				
15 4-Hydroxyphenylacetic *(C. difficile, C. stricklandii, C. lituseburense & others)*	≤ 30		2.0	2.0
16 HPHPA *(C. sporogenes, C. caloritolerans, C. botulinum & others)*	≤ 227		5.0	5.0
17 4-Cresol *(C. difficile)*	≤ 76		5.0	5.0
18 3-Indoleacetic *(C. stricklandii, C. lituseburense, C. subterminale & others)*	≤ 11		5.0	5.0

This test was developed, and its performance characteristics determined by Mosaic Diagnostics, LLC. It has not been cleared or approved by the US Food and Drug Administration.

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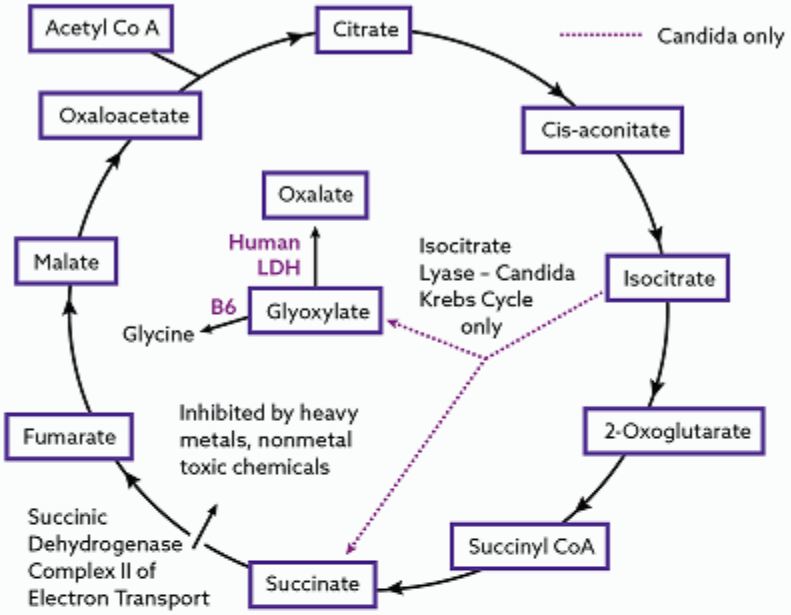
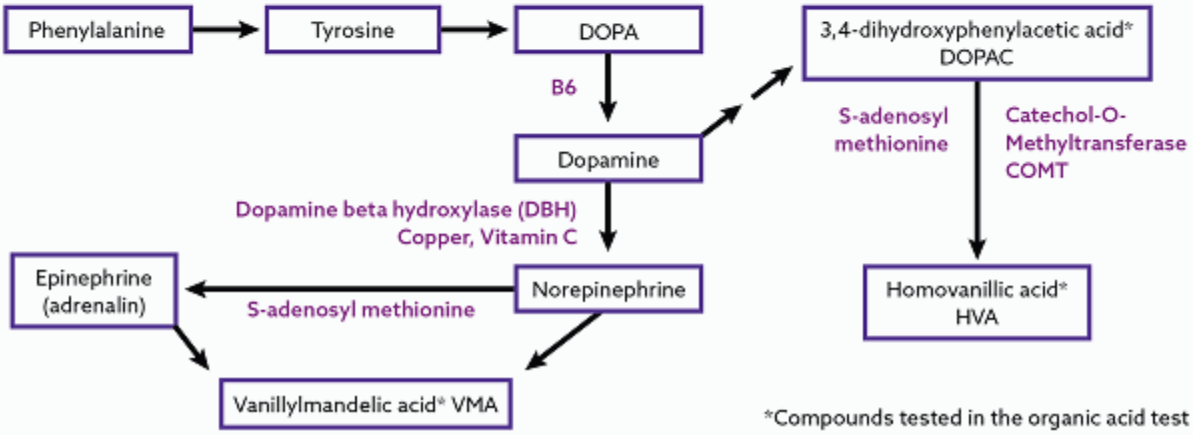
Human Krebs Cycle showing Candida Krebs Cycle variant that causes excess Oxalate via Glyoxylate



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 Under Age 13
Oxalate Metabolites			
19 Glyceric	0.71 - 9.5	1.0	1.0
20 Glycolic	20 - 202	L 1.0	1.0
21 Oxalic	15 - 174	L 5.0	5.0
Glycolytic Cycle Metabolites			
22 Lactic	0.18 - 44	10	10
23 Pyruvic	0.88 - 9.1	L 0	0.00
Mitochondrial Markers - Krebs Cycle Metabolites			
24 Succinic	≤ 15	0.50	0.50
25 Fumaric	0.04 - 1.3	0.50	0.50
26 Malic	≤ 2.2	1.0	1.0
27 2-Oxoglutaric	≤ 81	1.0	1.0
28 Aconitic	11 - 35	L 1.0	1.0
29 Citric	59 - 440	L 1.0	1.0
Mitochondrial Markers - Amino Acid Metabolites			
30 3-Methylglutaric	0.07 - 0.95	H 1.0	1.0
31 3-Hydroxyglutaric	≤ 11	5.0	5.0
32 3-Methylglutaconic	≤ 6.4	0	0.00
Neurotransmitter Metabolites			
Phenylalanine and Tyrosine Metabolites			
33 Homovanillic (HVA) *(dopamine)*	≤ 14	0	0.00
34 Vanillylmandelic (VMA) *(norepinephrine, epinephrine)*	0.87 - 5.9	1.0	1.0
35 HVA / VMA Ratio	0.12 - 3.0	L 0	0.00
36 Dihydroxyphenylacetic (DOPAC) *(dopamine)*	0.07 - 4.0	H 5.0	5.0
37 HVA/ DOPAC Ratio	1.5 - 2.6	L 0	0.00
Tryptophan Metabolites			
38 5-Hydroxyindoleacetic (5-HIAA) *(serotonin)*	≤ 7.7	1.0	1.0
39 Quinolinic	0.63 - 6.7	L 0	0.00
40 Kynurenic	≤ 4.1	1.0	1.0

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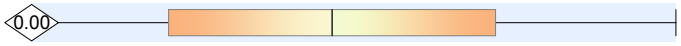
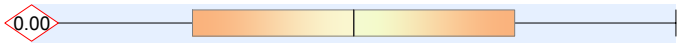
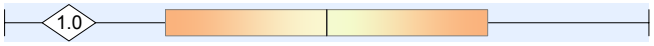
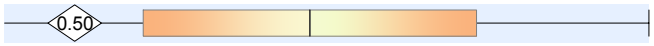
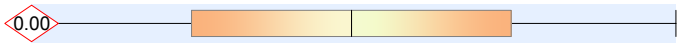
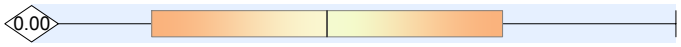
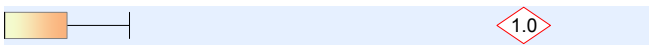
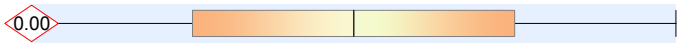
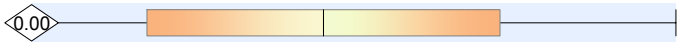
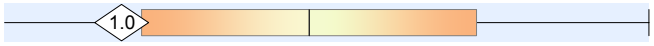
Metabolic Markers in Urine	Reference Range *(mmol/mol creatinine)*	Patient Value	Reference Population - Females Under Age 13
Pyrimidine Metabolites - Folate Metabolism			
41 Uracil	≤ 19	0	
42 Thymine	0.01 - 0.89	L 0	
Ketone and Fatty Acid Oxidation			
43 3-Hydroxybutyric	≤ 4.1	0	
44 Acetoacetic	≤ 10	1.0	
45 Ethylmalonic	≤ 4.6	0.50	
46 Methylsuccinic	≤ 4.3	0	
47 Adipic	≤ 9.7	0	
48 Suberic	≤ 9.5	0	
49 Sebacic	≤ 0.37	H 1.0	
Nutritional Markers			
Vitamin B12			
50 Methylmalonic ✱	≤ 6.2	5.0	
Vitamin B6			
51 Pyridoxic (B6)	≤ 59	1.0	
Vitamin B5			
52 Pantothenic (B5)	≤ 26	1.0	
Vitamin B2 (Riboflavin)			
53 Glutaric ✱	≤ 1.1	1.0	
Vitamin C			
54 Ascorbic	10 - 200	L 0	
Vitamin Q10 (CoQ10)			
55 3-Hydroxy-3-methylglutaric ✱	≤ 101	0	
Glutathione Precursor and Chelating Agent			
56 N-Acetylcysteine (NAC)	≤ 0.41	0	
Biotin (Vitamin H)			
57 Methylcitric ✱	≤ 5.5	1.0	

✱ ***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 - Females Under Age 13

Indicators of Detoxification

Glutathione

58	Pyroglutamic ✱	7.0 - 63	L	0	0.00

Methylation, Toxic exposure

59	2-Hydroxybutyric ✱✱	≤ 2.2	H	5.0	5.0

Ammonia Excess

60	Orotic	≤ 0.88		0	0.00

Aspartame, salicylates, or GI bacteria

61	2-Hydroxyhippuric	≤ 1.2		1.0	1.0

✱ ***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		1.0	1.0
63	2-Oxoisovaleric	≤ 2.4		0	0.00
64	3-Methyl-2-oxovaleric	≤ 2.0	H	3.0	3.0
65	2-Hydroxyisocaproic	≤ 2.0		0	0.00
66	2-Oxoisocaproic	≤ 2.0		0	0.00
67	2-Oxo-4-methiolbutyric	≤ 2.0		0	0.00
68	Mandelic	≤ 2.0		1.0	1.0
69	Phenyllactic	≤ 2.3		0	0.00
70	Phenylpyruvic	≤ 2.3		0	0.00
71	Homogentisic	≤ 2.0		0	0.00
72	4-Hydroxyphenyllactic	≤ 2.0		1.0	1.0
73	N-Acetylaspartic	≤ 38		0	0.00
74	Malonic	≤ 12		1.0	1.0
75	4-Hydroxybutyric	≤ 3.4		2.3	2.3

Mineral Metabolism

76	Phosphoric	1,000 - 7,300	L	380	380

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Indicator of Fluid Intake

77	***Creatinine**	**100**	**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.

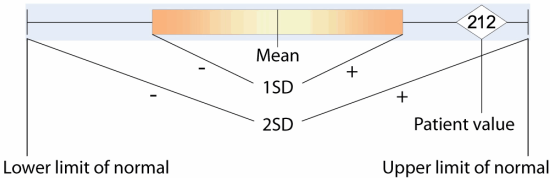
The first graph will occur when the value of the patient is within the reference (normal) range, defined as the mean plus or minus two standard deviations.

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.

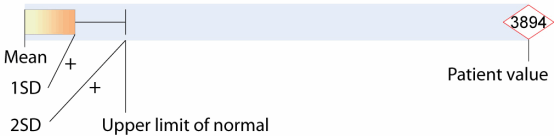
Example of Value Within Reference Range

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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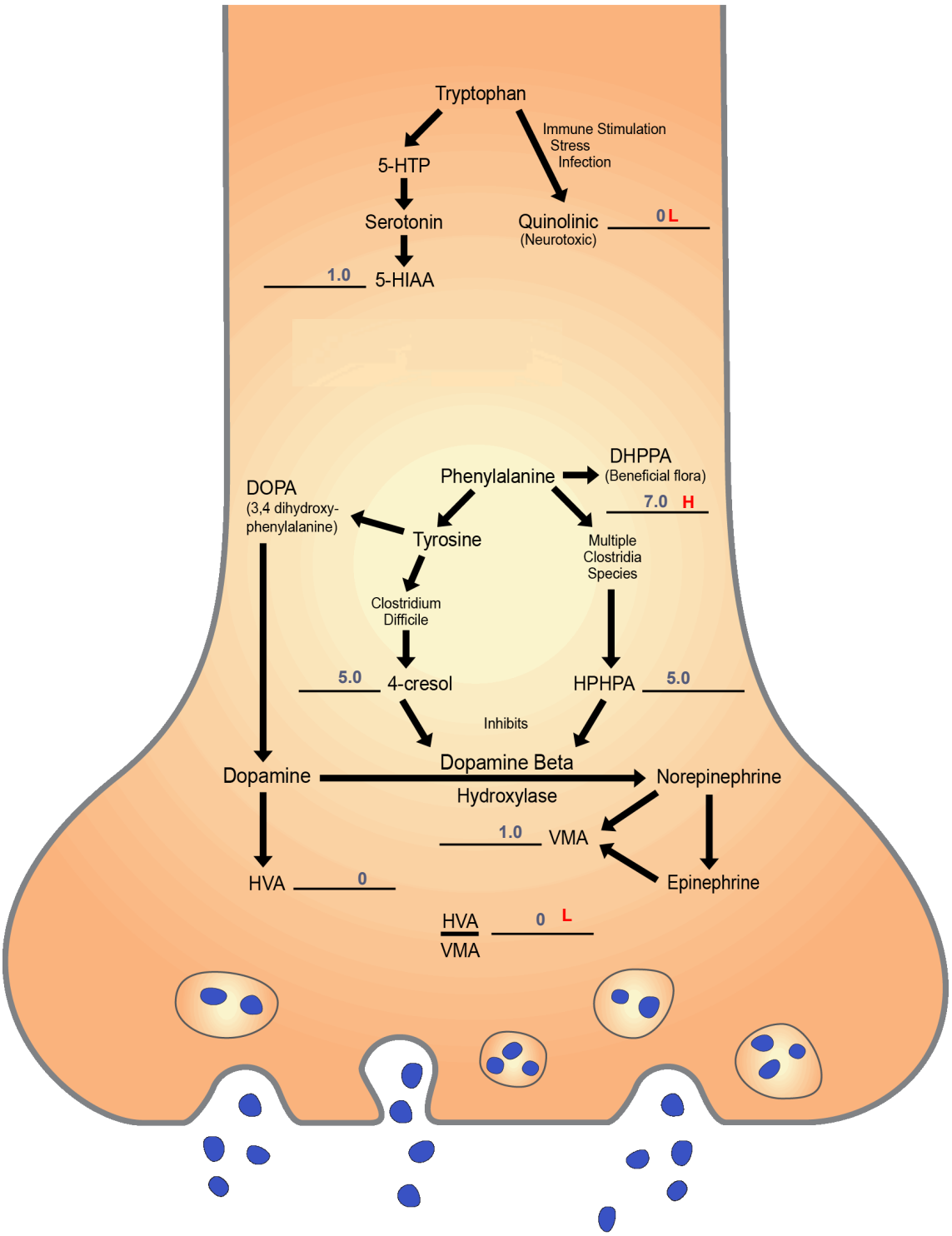
Neurotransmitter Metabolism Markers



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 tricarballylic acid (propane-1,2,3-tricarboxylic acid) (9) could be caused by the intake of corn or corn-based food contaminated with fumonisins, a group of mycotoxins produced primarily by *F. verticillioides*, and other related species. Tricarballylic acid is released from fumonisins during passage through the gastrointestinal tract . Tricarballylic acid is an inhibitor of the enzyme aconitase and therefore interferes with the Krebs cycle. The main symptoms of aconitase deficiency are myopathy and exercise intolerance. It may also act as a magnesium chelator. Tricarballylic acid is also metabolite of a component of a substance in modified corn starch, octenylsuccinic acid, found in a number of infant formulas such as Nutramigen, Vivonex, and Pregestimil. In addition, tricarballylic acid is a byproduct of beet sugar and maple sugar refining and might appear after ingestion of these sugars. Tricarballylic acid is also released from fumonisins upon certain food processing conditions. Clinical syndromes due to the intact mycotoxin are rare and characterized by abdominal pain and diarrhea. A specific role for fumonisins in the development of neural tube defects was suggested after the appearance of a cluster of such defects in Texas associated with consumption of corn from the heavily fumonisin-contaminated 1989 corn crop. More recent studies have shown that fumonisin B1 inhibits folate metabolism in cultured cells. Confirmation of Fusarium species can be done by the urine Mycotoxin test of The Great Plains Laboratory .

High 2-hydroxyphenylacetic acid (11) is associated with intestinal bacteria overgrowth and higher levels with the genetic disease phenylketonuria (PKU). Additional metabolites that can become elevated in PKU include mandelic acid, phenylpyruvic, and phenyllactic. The diagnosis of PKU is more likely if the individual has an elevation in more than one of these metabolites.

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.

Low or low normal citric acid (29) may be due to impaired function of the Krebs cycle, low dietary intake of citrate-containing foods such as citrus fruits and juices, potassium deficiency, acidosis (especially renal tubular acidosis), chronic kidney failure, diabetes, hypoparathyroidism, or excessive muscle activity. Low values may indicate increased risk of oxalate kidney stone formation, especially if oxalic acid is elevated also. Supplement with calcium or magnesium citrate if oxalic acid is elevated.

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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 quadriparesis). Recommendations include supplementation with coenzyme Q-10, L-carnitine and acetyl-L-carnitine, riboflavin, nicotinamide, and vitamin E.

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.

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. Such SNPs are available on **The Great Plains DNA methylation pathway test** which can be performed on a cheek swab. 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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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.

Low HVA/DOPAC ratio (37) HVA and DOPAC are the major metabolites of dopamine. A decrease in the conversion of DOPAC to HVA is most commonly caused by a genetic deficiency of catechol-O-methyltransferase (COMT) and/or a nutritional deficiency of S-adenosyl methionine (S-ame). Individuals with this abnormality might benefit from supplementation with S-ame and/or other methylation cofactors such as methyltetrahydrofolate or methylcobalamin that increase endogenous Sam-e. SNPs of COMT 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.

Low pyroglutamic acid (58) the diagnostic significance of low pyroglutamic acid is unclear.

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Requisition #:	9900001	***Practitioner:***	NO PHYSICIAN
Patient Name:	Sample Report	***Date of Collection:***	12/01/2022

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.

High 3-methyl-2-oxovaleric acid (64) is a metabolite of the amino acid isoleucine, and elevated values are seen in the genetic disease MSUD (maple syrup urine disease). Individuals with slight to moderate elevations may benefit from supplementing with thiamine.* Individuals high in all MSUD metabolites and have values that exceed 20 times the upper limit may benefit from very high doses (5-20 mg/kg/day) of thiamine.

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.

Low citramalic, 2-hydroxyphenylacetic, 4-hydroxyphenylacetic, 4-hydroxybenzoic, 4-hydroxyhippuric, 3-indoleacetic, glyceric, glycolic, oxalic, lactic, pyruvic, 3-Methylglutaric, 3-methylglutaconic, 2-hydroxybutyric, fumaric, malic, aconitic, quinolinic, kynurenic, thymine, ethylmalonic, methylsuccinic, adipic, suberic, glutaric, 3-hydroxy-3-methylglutaric, methylcitric, or orotic values have no known clinical significance.

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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