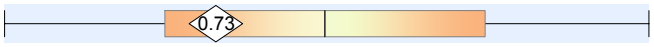
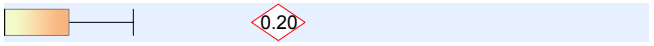
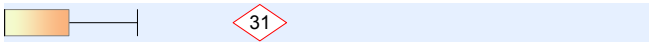
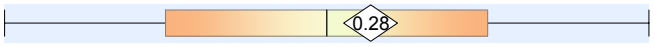
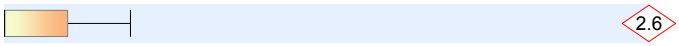
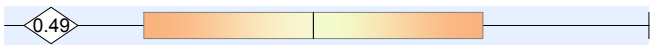
MOSAIC DIAGNOSTICS

OAT ORGANIC ACIDS TEST

Requisition #:		*Practitioner:*	JAN VOJACEK
Patient Name:		*Date of Collection:*	05/24/2024
Date of Birth:	*Patient Age:*	*Time of Collection:*	05:45 AM
Patient Sex:	M	*Report Date:*	06/12/2024

Organic Acids Test - Nutritional and Metabolic Profile

Metabolic Markers in Urine	Reference Range (mmol/mol creatinine)		Patient Value	Reference Population - Males Age 13 and Over
Intestinal Microbial Overgrowth				
Yeast and Fungal Markers				
1 Citramalic	0.11 - 2.0		0.73	
2 5-Hydroxymethyl-2-furoic *(Aspergillus)*	≤ 18		7.5	
3 3-Oxoglutaric	≤ 0.11	H	0.20	
4 Furan-2,5-dicarboxylic *(Aspergillus)*	≤ 13		5.3	
5 Furancarbonylglycine *(Aspergillus)*	≤ 2.3		0.40	
6 Tartaric *(Aspergillus)*	≤ 5.3		0.69	
7 Arabinose	≤ 20	H	31	
8 Carboxycitric	≤ 20		0.04	
9 Tricarballylic *(Fusarium)*	≤ 0.58		0.08	
Bacterial Markers				
10 Hippuric	≤ 241	H	426	
11 2-Hydroxyphenylacetic	0.03 - 0.47		0.28	
12 4-Hydroxybenzoic	≤ 0.73	H	2.6	
13 4-Hydroxyhippuric	≤ 14		9.3	
14 DHPPA (Beneficial Bacteria)	≤ 0.23		0.05	
Clostridia Bacterial Markers				
15 4-Hydroxyphenylacetic *(C. difficile, C. stricklandii, C. lituseburense & others)*	≤ 18		5.2	
16 HPHPA *(C. sporogenes, C. caloritolerans, C. botulinum & others)*	≤ 102		16	
17 4-Cresol *(C. difficile)*	≤ 39		4.5	
18 3-Indoleacetic *(C. stricklandii, C. lituseburense, C. subterminale & others)*	≤ 6.8		0.49	

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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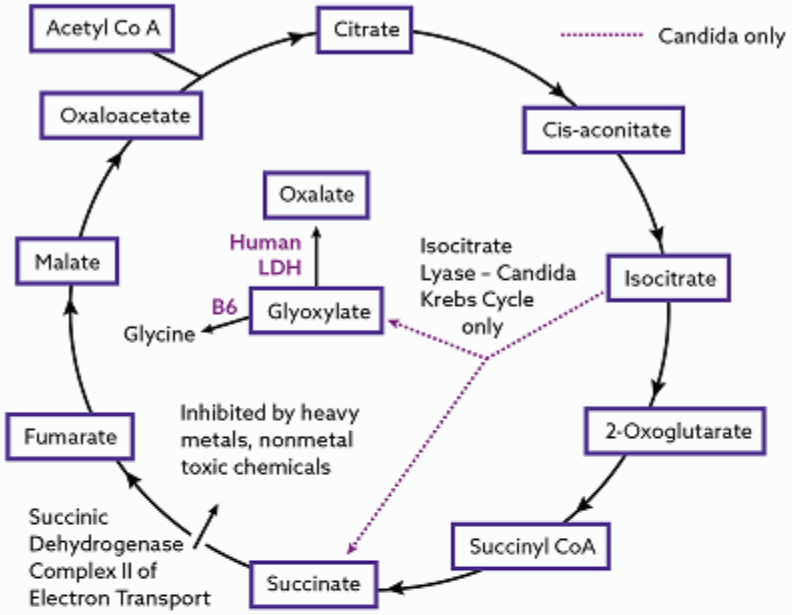
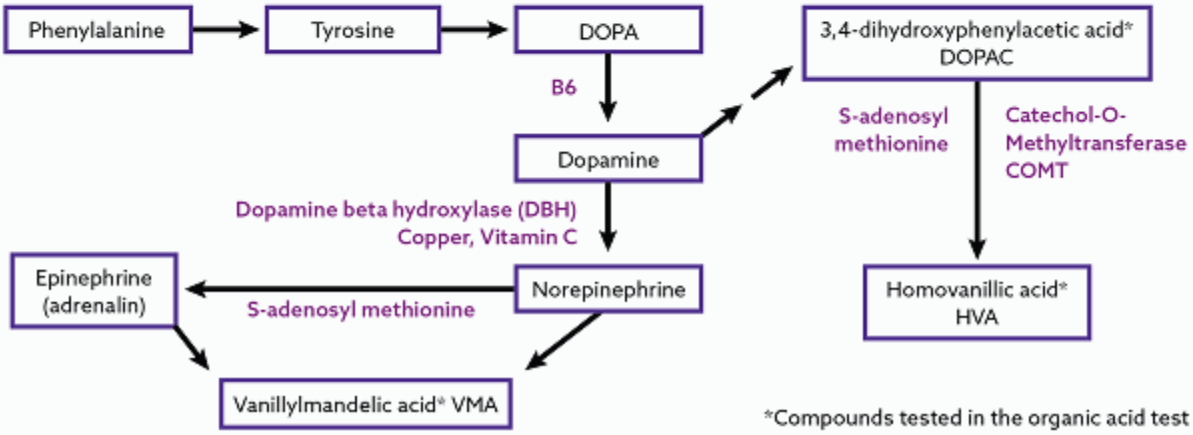
Requisition #:		***Practitioner:***	JAN VOJACEK
Patient Name:		***Date of Collection:***	05/24/2024

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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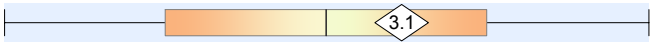
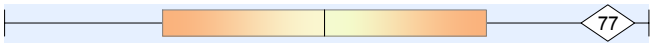
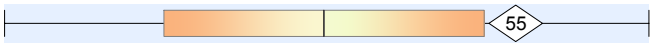
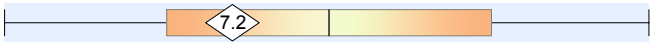
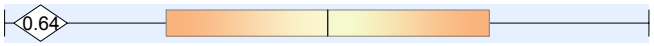
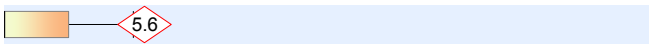
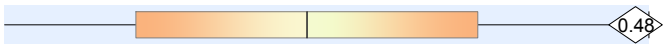
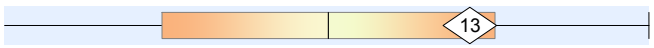
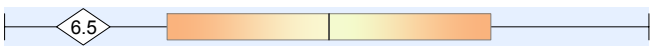
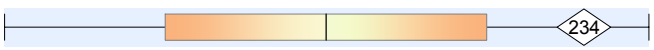
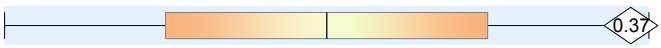
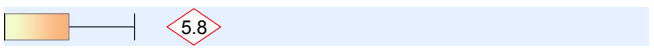
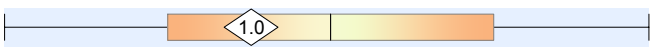
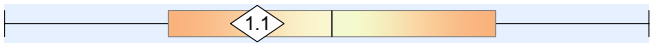
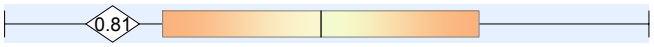
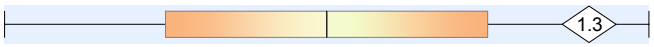
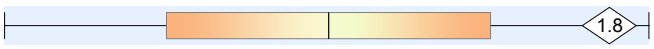
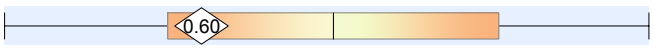
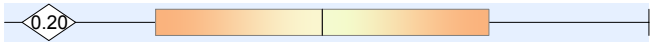
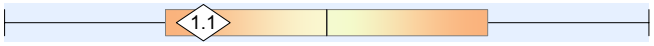
Requisition #:

Patient Name:

Practitioner: JAN VOJACEK

Date of Collection: 05/24/2024

Metabolic Markers in Urine	Reference Range (mmol/mol creatinine)	Patient Value	Reference Population - Males Age 13 and Over
Oxalate Metabolites			
19 Glyceric	0.21 - 4.9	3.1	
20 Glycolic	18 - 81	77	
21 Oxalic	8.9 - 67	55	
Glycolytic Cycle Metabolites			
22 Lactic	0.74 - 19	7.2	
23 Pyruvic	0.28 - 6.7	0.64	
Mitochondrial Markers - Krebs Cycle Metabolites			
24 Succinic	≤ 5.3	H 5.6	
25 Fumaric	≤ 0.49	0.48	
26 Malic	≤ 1.1	0.96	
27 2-Oxoglutaric	≤ 18	13	
28 Aconitic	4.1 - 23	6.5	
29 Citric	2.2 - 260	234	
Mitochondrial Markers - Amino Acid Metabolites			
30 3-Methylglutaric	0.02 - 0.38	0.37	
31 3-Hydroxyglutaric	≤ 4.6	H 5.8	
32 3-Methylglutaconic	0.38 - 2.0	1.0	
Neurotransmitter Metabolites			
Phenylalanine and Tyrosine Metabolites			
33 Homovanillic (HVA) *(dopamine)*	0.39 - 2.2	1.1	
34 Vanillylmandelic (VMA) *(norepinephrine, epinephrine)*	0.53 - 2.2	0.81	
35 HVA / VMA Ratio	0.32 - 1.4	1.3	
36 Dihydroxyphenylacetic (DOPAC) *(dopamine)*	0.27 - 1.9	1.8	
37 HVA/ DOPAC Ratio	0.17 - 1.6	0.60	
Tryptophan Metabolites			
38 5-Hydroxyindoleacetic (5-HIAA) *(serotonin)*	≤ 2.9	0.20	
39 Quinolinic	0.52 - 2.4	1.1	
40 Kynurenic	≤ 1.8	0.15	

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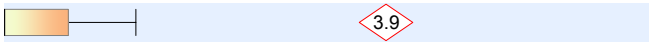
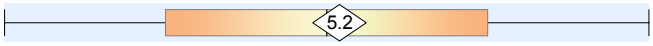
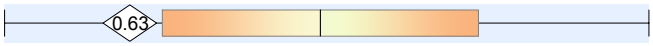
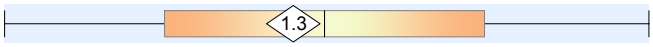
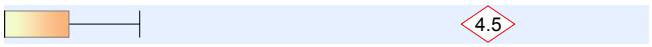
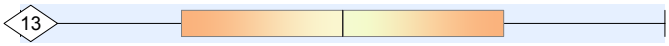
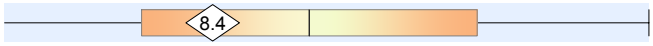
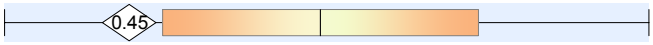
Date of Collection: 05/24/2024

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	4.7	
42 Thymine	≤ 0.36	0.15	
Ketone and Fatty Acid Oxidation			
43 3-Hydroxybutyric	≤ 1.9	H 3.9	
44 Acetoacetic	≤ 10	5.2	
45 Ethylmalonic	0.13 - 2.7	0.63	
46 Methylsuccinic	≤ 2.3	0.99	
47 Adipic	≤ 2.9	1.3	
48 Suberic	≤ 1.9	H 4.5	
49 Sebacic	≤ 0.14	0.14	
Nutritional Markers			
Vitamin B12			
50 Methylmalonic ✱	≤ 2.3	0.95	
Vitamin B6			
51 Pyridoxic (B6)	≤ 26	1.1	
Vitamin B5			
52 Pantothenic (B5)	≤ 5.4	1.5	
Vitamin B2 (Riboflavin)			
53 Glutaric ✱	≤ 0.43	0.32	
Vitamin C			
54 Ascorbic	10 - 200	13	
Vitamin Q10 (CoQ10)			
55 3-Hydroxy-3-methylglutaric ✱	≤ 26	8.4	
Glutathione Precursor and Chelating Agent			
56 N-Acetylcysteine (NAC)	≤ 0.13	0.10	
Biotin (Vitamin H)			
57 Methylcitric ✱	0.15 - 1.7	0.45	

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

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Patient Name:
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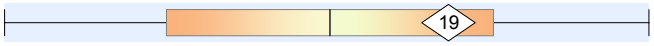
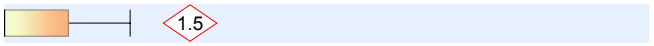
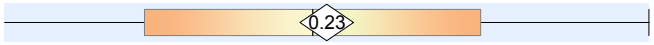
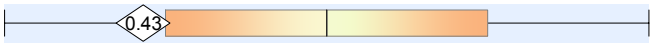
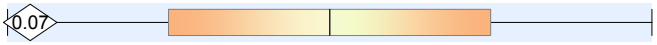
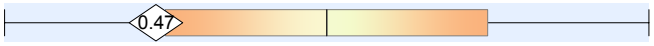
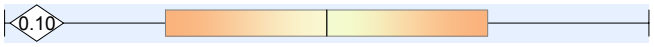
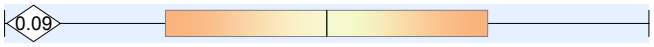
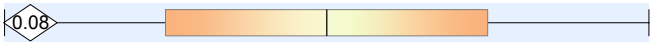
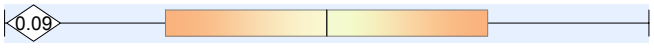
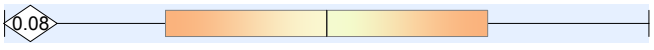
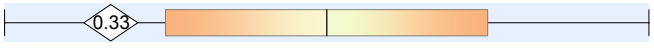
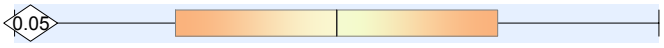
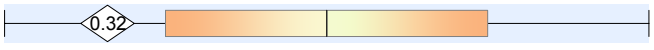
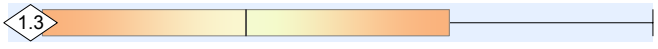
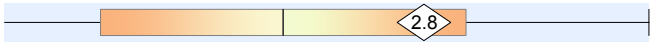
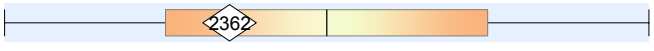
Date of Collection: 05/24/2024

Metabolic Markers in Urine	Reference Range (mmol/mol creatinine)	Patient Value	Reference Population - Males Age 13 and Over
Indicators of Detoxification			
Glutathione			
58 Pyroglutamic *	5.7 - 25	19	
Methylation, Toxic exposure			
59 2-Hydroxybutyric **	≤ 1.2	H 1.5	
Ammonia Excess			
60 Orotic	≤ 0.46	0.23	
Aspartame, salicylates, or GI bacteria			
61 2-Hydroxyhippuric	≤ 0.86	0.05	

* *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.

62 2-Hydroxyisovaleric	≤ 2.0	0.43	
63 2-Oxoisovaleric	≤ 2.0	0.07	
64 3-Methyl-2-oxovaleric	≤ 2.0	0.47	
65 2-Hydroxyisocaproic	≤ 2.0	0.10	
66 2-Oxoisocaproic	≤ 2.0	0.09	
67 2-Oxo-4-methiolbutyric	≤ 2.0	0.08	
68 Mandelic	≤ 2.0	0.09	
69 Phenyllactic	≤ 2.0	0.08	
70 Phenylpyruvic	≤ 2.0	0.33	
71 Homogentisic	≤ 2.0	0.05	
72 4-Hydroxyphenyllactic	≤ 2.0	0.32	
73 N-Acetylaspartic	≤ 38	1.3	
74 Malonic	≤ 9.9	4.4	
75 4-Hydroxybutyric	≤ 4.3	2.8	
Mineral Metabolism			
76 Phosphoric	1,000 - 4,900	2,362	

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Requisition #:
Patient Name:
Practitioner: JAN VOJACEK
Date of Collection: 05/24/2024

Indicator of Fluid Intake

77	*Creatinine	82	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.

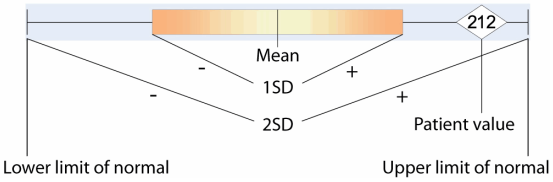
The first graph will occur when the value of the patient is within the reference (normal) range, defined as the mean plus or minus two standard deviations.

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.

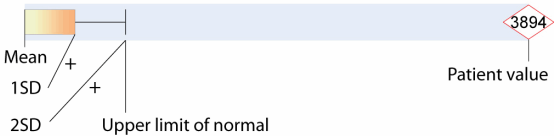
Example of Value Within Reference Range

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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Requisition #:
Patient Name:
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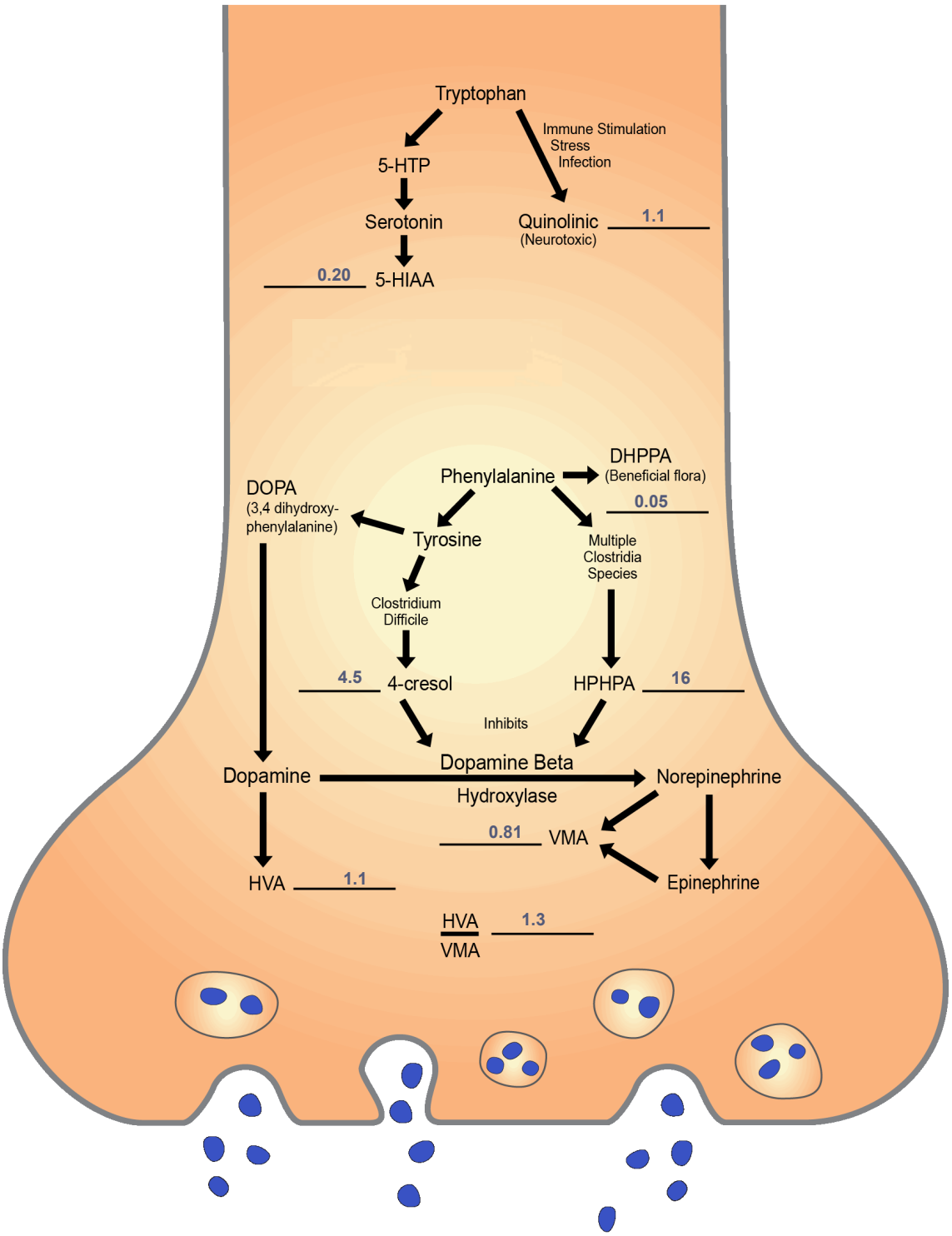
Neurotransmitter Metabolism Markers



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 hippuric acid (10) may derive from food, GI bacterial activity, or exposure to the solvent toluene. Hippuric acid is a conjugate of glycine and benzoic acid formed in the liver. Most hippuric acid in urine is derived from microbial breakdown of chlorogenic acid to benzoic acid. Chlorogenic acid is a common substance in beverages and in many fruits and vegetables, including apples, pears, tea, coffee, sunflower seeds, carrots, blueberries, cherries, potatoes, tomatoes, eggplant, sweet potatoes, and peaches. Benzoic acid is present in high amounts in cranberry juice and is a food preservative. The workplace is the most common source of toluene exposure, but toluene may be absorbed from outgassing of new carpets and other building materials, or absorbed during recreational abuse of solvents such as glue-sniffing. Because most hippuric acid in urine is from GI sources, this marker is a poor indicator of toluene exposure and is being replaced by other markers in occupational safety testing. Bacterial overgrowth can be treated with natural anti-bacterial agents and/or probiotics (30-50 billion cfu's) that include *Lactobacillus rhamnosus*.

High 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 Ca2+-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]).

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Requisition #:		***Practitioner:***	JAN VOJACEK
Patient Name:		***Date of Collection:***	05/24/2024

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

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.

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Requisition #:		***Practitioner:***	JAN VOJACEK
Patient Name:		***Date of Collection:***	05/24/2024

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.

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 5 HIAA 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 3-hydroxybutyric acids (43) and/or acetoacetic acids (44) indicate increased metabolic utilization of fatty acids. These ketones are associated with diabetes mellitus, fasting, dieting (ketogenic or SCD diet), or illness such as nausea or flu, among many other causes.

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.

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.

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