



Requisition #: 1113390
 Patient Name: Abby Albert
 Date of Birth: 12/14/1982
 Patient Sex: F
 Specimen Id.: 1113390-2

Patient Age: 39

Practitioner: RN LABS
 Date of Collection: 09/20/2022
 Time of Collection: 06:15 AM
 Print Date: 10/07/2022

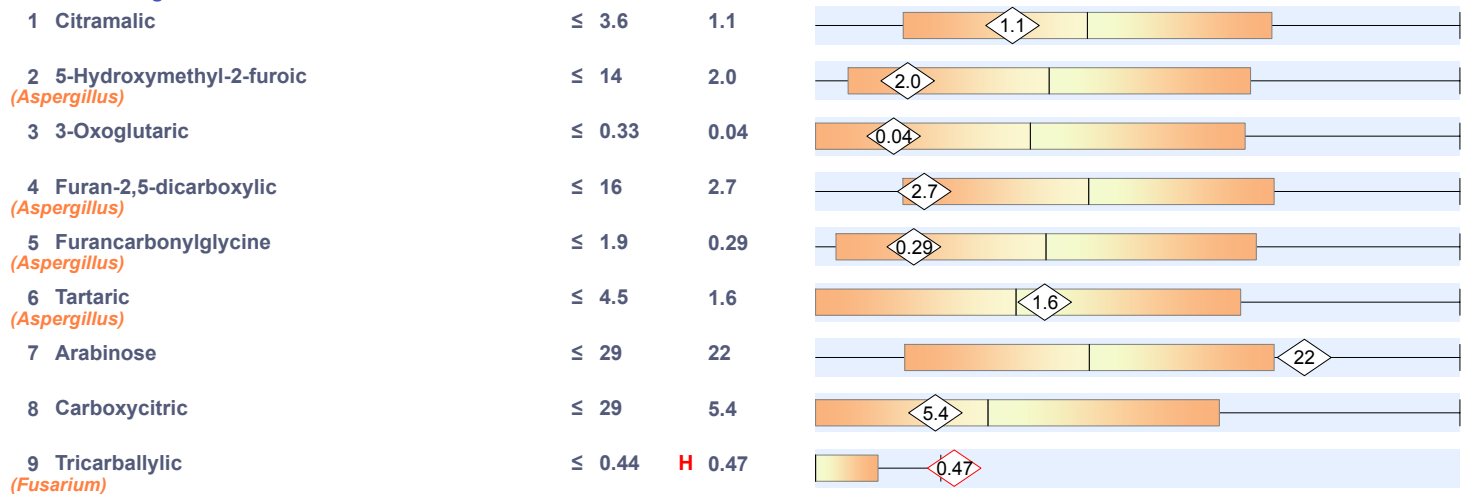


Organic Acids Test - Nutritional and Metabolic Profile

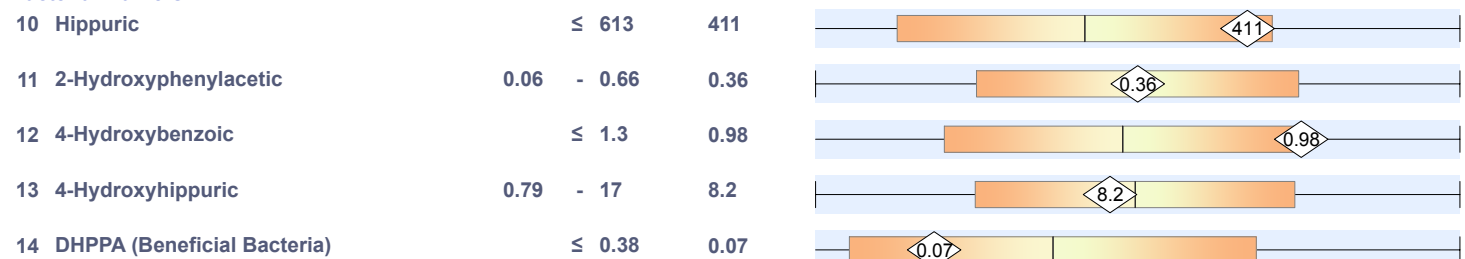
Metabolic Markers in Urine	Reference Range (mmol/mol creatinine)	Patient Value	Reference Population - Females Age 13 and Over
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Intestinal Microbial Overgrowth

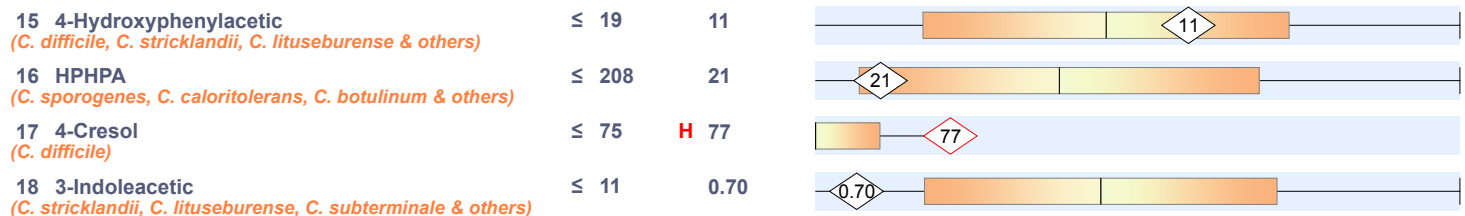
Yeast and Fungal Markers



Bacterial Markers



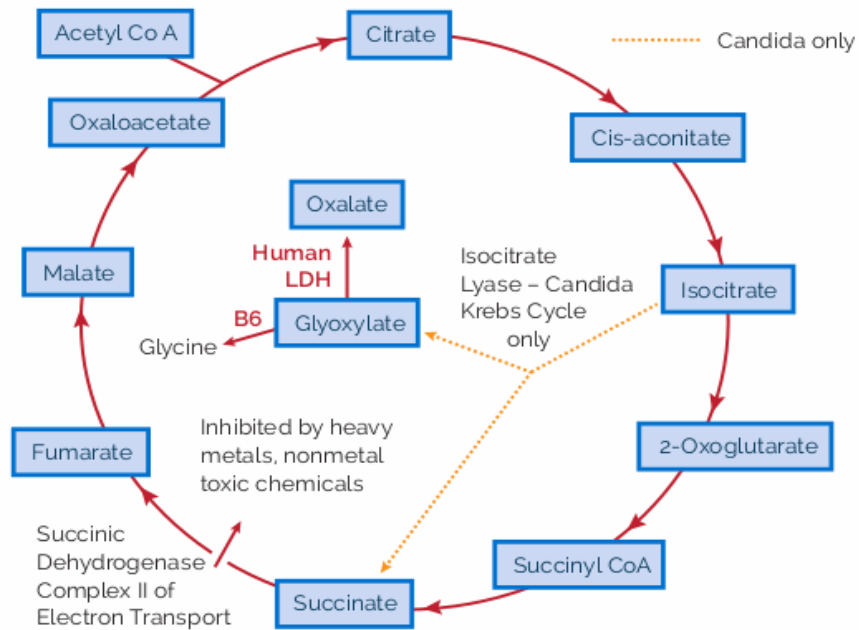
Clostridia Bacterial Markers



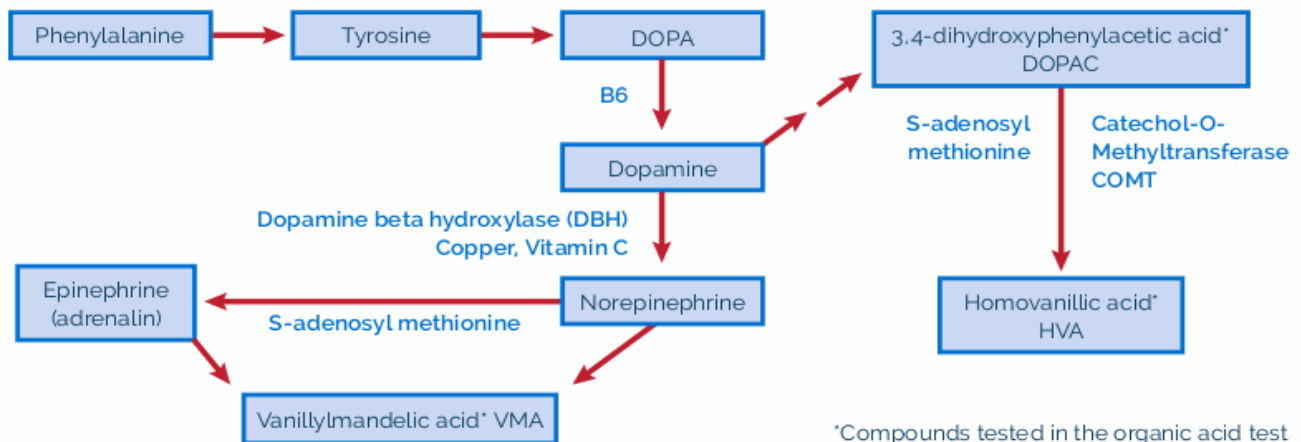
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Human Krebs Cycle showing Candida Krebs Cycle variant that causes excess Oxalate via Glyoxylate



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



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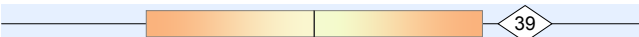
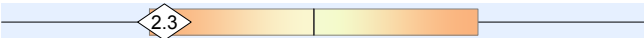
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Metabolic Markers in Urine Reference Range (mmol/mol creatinine) Patient Value Reference Population - Females Age 13 and Over

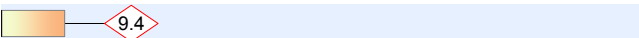
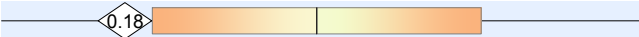
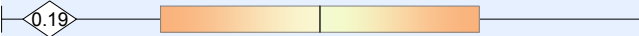
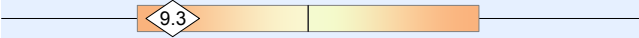
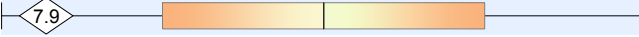
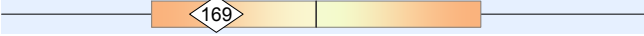
Oxalate Metabolites

19	Glyceric	0.77 - 7.0	1.8	
20	Glycolic	16 - 117	72	
21	Oxalic	6.8 - 101	H 131	

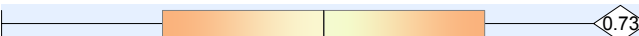
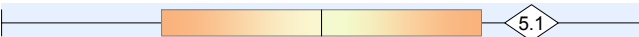
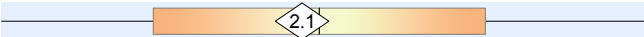
Glycolytic Cycle Metabolites

22	Lactic	≤ 48	39	
23	Pyruvic	≤ 9.1	2.3	

Mitochondrial Markers - Krebs Cycle Metabolites

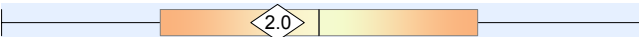
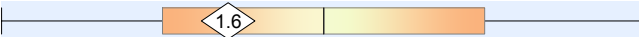
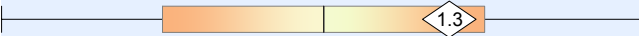
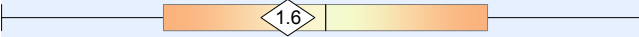
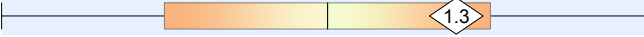
24	Succinic	≤ 9.3	H 9.4	
25	Fumaric	≤ 0.94	0.18	
26	Malic	0.06 - 1.8	0.19	
27	2-Oxoglutaric	≤ 35	9.3	
28	Aconitic	6.8 - 28	7.9	
29	Citric	≤ 507	169	

Mitochondrial Markers - Amino Acid Metabolites

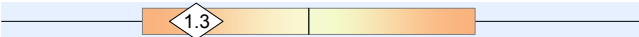
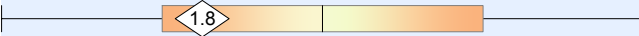
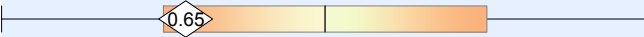
30	3-Methylglutaric	≤ 0.76	0.73	
31	3-Hydroxyglutaric	≤ 6.2	5.1	
32	3-Methylglutaconic	≤ 4.5	2.1	

Neurotransmitter Metabolites

Phenylalanine and Tyrosine Metabolites

33	Homovanillic (HVA) (dopamine)	0.80 - 3.6	2.0	
34	Vanillylmandelic (VMA) (norepinephrine, epinephrine)	0.46 - 3.7	1.6	
35	HVA / VMA Ratio	0.16 - 1.8	1.3	
36	Dihydroxyphenylacetic (DOPAC) (dopamine)	0.08 - 3.5	1.6	
37	HVA / DOPAC Ratio	0.10 - 1.8	1.3	

Tryptophan Metabolites

38	5-Hydroxyindoleacetic (5-HIAA) (serotonin)	≤ 4.3	1.3	
39	Quinolinic	0.85 - 3.9	1.8	
40	Kynurenic	≤ 2.2	0.65	

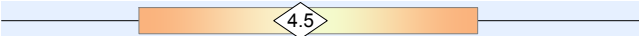
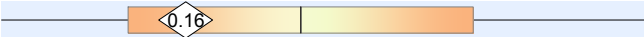
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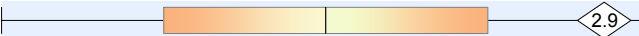
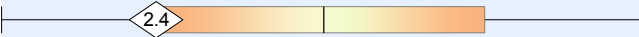
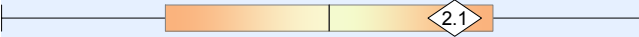
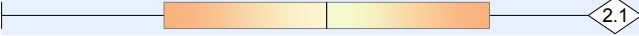
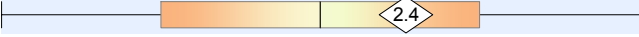
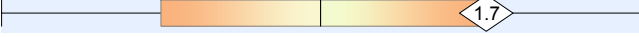
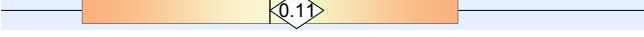
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Metabolic Markers in Urine Reference Range (mmol/mol creatinine) Patient Value Reference Population - Females Age 13 and Over

Pyrimidine Metabolites - Folate Metabolism

41 Uracil	≤ 9.7	4.5	
42 Thymine	≤ 0.56	0.16	

Ketone and Fatty Acid Oxidation

43 3-Hydroxybutyric	≤ 3.1	2.9	
44 Acetoacetic	≤ 10	2.4	
45 Ethylmalonic	0.44 - 2.8	2.1	
46 Methylsuccinic	0.10 - 2.2	2.1	
47 Adipic	0.04 - 3.8	2.4	
48 Suberic	0.18 - 2.2	1.7	
49 Sebacic	≤ 0.24	0.11	

Nutritional Markers

Vitamin B12

50 Methylmalonic *	≤ 2.3	1.4	
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Vitamin B6

51 Pyridoxic (B6)	≤ 34	2.0	
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Vitamin B5

52 Pantothenic (B5)	≤ 10	2.5	
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Vitamin B2 (Riboflavin)

53 Glutaric *	0.04 - 0.36	0.36	
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Vitamin C

54 Ascorbic	10 - 200	L 0.67	
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Vitamin Q10 (CoQ10)

55 3-Hydroxy-3-methylglutaric *	0.17 - 39	17	
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Glutathione Precursor and Chelating Agent

56 N-Acetylcysteine (NAC)	≤ 0.28	0.10	
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Biotin (Vitamin H)

57 Methylcitric *	0.19 - 2.7	0.60	
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* A high value for this marker may indicate a deficiency of this vitamin.

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Indicators of Detoxification

Glutathione

58 Pyroglutamic * 10 - 33 21 

Methylation, Toxic exposure

59 2-Hydroxybutyric ** 0.03 - 1.8 0.99 

Ammonia Excess

60 Orotic 0.06 - 0.54 0.14 

Aspartame, salicylates, or GI bacteria

61 2-Hydroxyhippuric ≤ 1.3 0.94 

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

Amino Acid Metabolites

62 2-Hydroxyisovaleric ≤ 2.0 0.29 

63 2-Oxoisovaleric ≤ 2.1 0.05 

64 3-Methyl-2-oxovaleric ≤ 2.0 0.06 

65 2-Hydroxyisocaproic ≤ 2.0 0.78 

66 2-Oxoisocaproic ≤ 2.0 0.01 

67 2-Oxo-4-methiolbutyric ≤ 2.0 0.02 

68 Mandelic ≤ 2.0 0.28 

69 Phenyllactic ≤ 2.0 0.10 

70 Phenylpyruvic ≤ 2.0 0 

71 Homogentisic ≤ 2.0 0.05 

72 4-Hydroxyphenyllactic ≤ 2.0 0.21 

73 N-Acetylaspartic ≤ 38 1.3 

74 Malonic ≤ 9.7 3.0 

75 4-Hydroxybutyric ≤ 4.8 0 

Mineral Metabolism

76 Phosphoric 1,000 - 5,000 1,983 

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Indicator of Fluid Intake

77 *Creatinine 121 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 (≥ 13 years), Female Adult (≥ 13 years), Male Child (< 13 years), and Female Child (< 13 years).

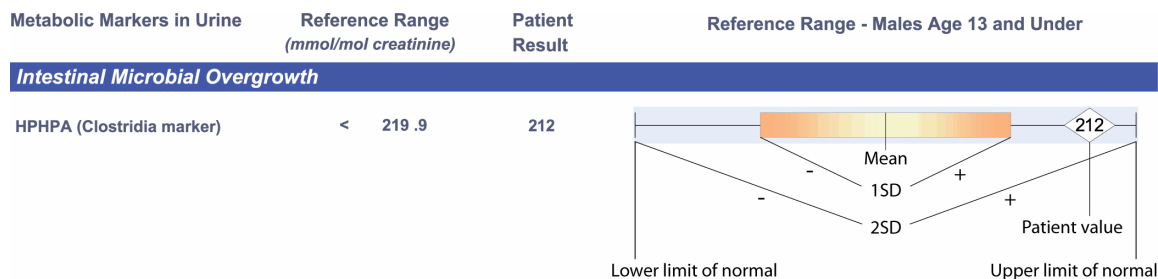
There are two types of graphical representations of patient values found in the new report format of both the standard Organic Acids Test and the Microbial Organic Acids Test.

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.

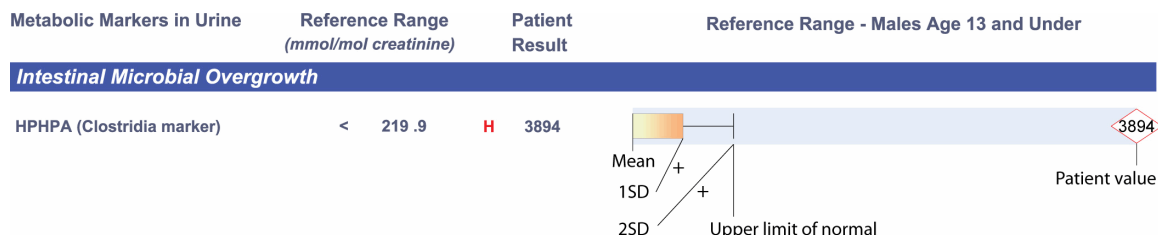
The second graph will occur when the value of the patient exceeds the upper limit of normal. In such cases, the graphical reference range is "shrunk" so that the degree of abnormality can be appreciated at a glance. In this case, the lower limits of normal are not shown, only the upper limit of normal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



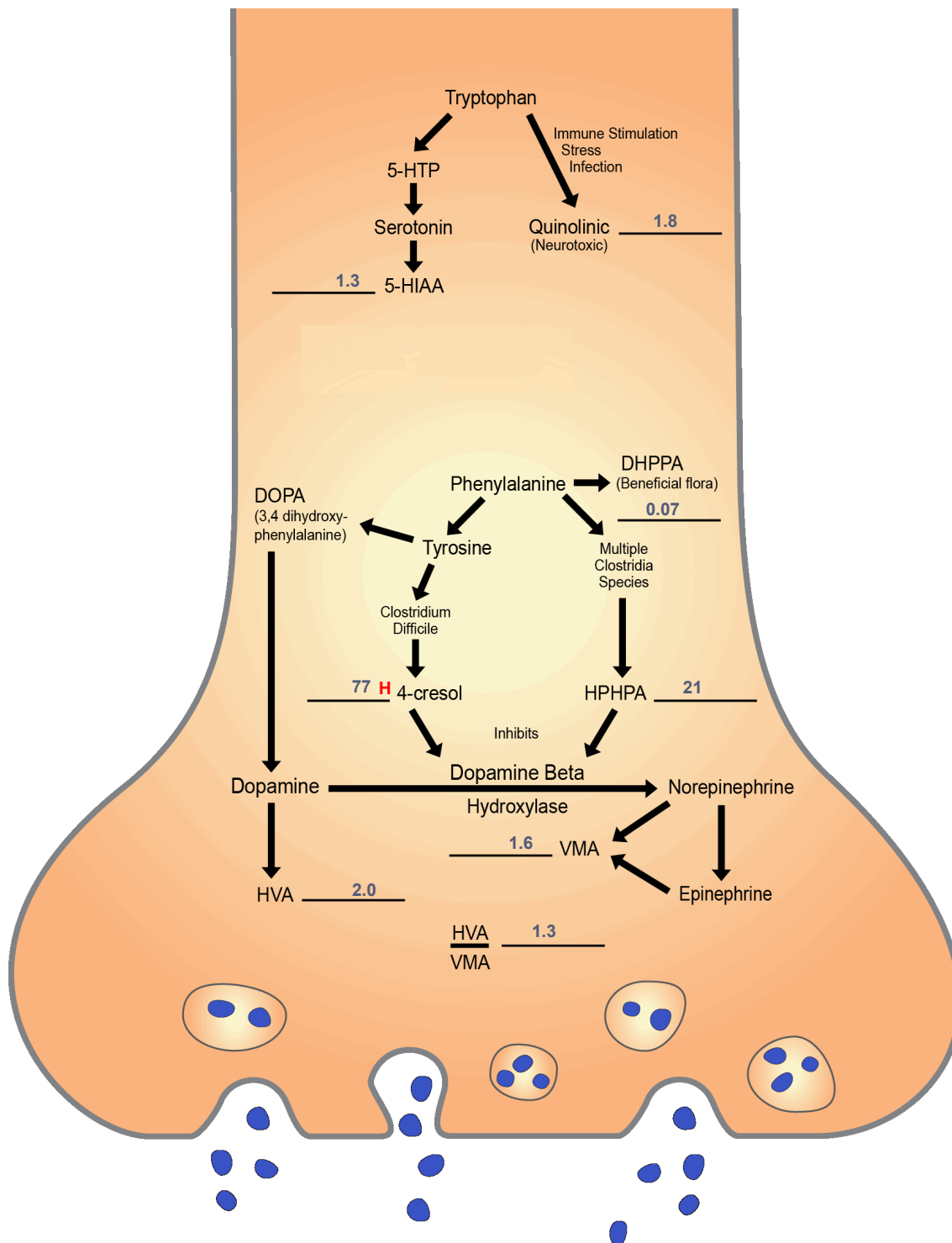
Example of Elevated Value



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Neurotransmitter Metabolism Markers



The diagram contains the patient's test results for neurotransmitter metabolites and shows their relationship with key biochemical pathways within the axon terminal of nerve cells. The effect of microbial byproducts on the blockage of the conversion of dopamine to norepinephrine is also indicated.

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Interpretation

High tricarballic 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. Tricarballic acid is released from fumonisins during passage through the gastrointestinal tract. Tricarballic 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. Tricarballic acid is also a 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, tricarballic acid is a byproduct of beet sugar and maple sugar refining and might appear after ingestion of these sugars. Tricarballic 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 4-cresol (p-cresol) (17) in the urine is most commonly due to *C. difficile* and *C. scatologenes* in the gastrointestinal tract. Most other *Clostridia* species and all other bacteria species do not produce this metabolite. Since *C. scatologenes* is not commonly isolated from stool samples, *C. difficile* would be the most likely source of this compound.

The major clinical significance of 4-cresol is that it is a potent inhibitor of brain dopamine-beta-hydroxylase, the enzyme that converts dopamine to norepinephrine. High concentrations of this compound cause abnormal behavior by inhibiting metabolism of dopamine to norepinephrine, resulting in high levels of the dopamine metabolite homovanillic acid (HVA) in the urine and insufficient norepinephrine in the central nervous system. High urine values of 4-cresol are associated with the most severe clinical symptoms in autism, multiple sclerosis, neurotoxicity, hallucinations, and other neurological and psychiatric disorders. 4-Cresol is also a metabolite of toluene, wood tar creosote, and menthofuran (derived from the mint flavoring agent pennyroyal).

Treatment with Metronidazole or Vancomycin is almost 100% effective in killing parent organisms, including *C. difficile*, but not their spores. At least three months of probiotic therapy is recommended after antimicrobial treatment due to spore formation by *Clostridia* species. *Clostridia* overgrowth can sometimes be controlled by supplementation with *Lactobacillus rhamnosus* GG (Culturelle) or *Saccharomyces boulardi*.

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, muscle pains, and heart abnormalities. Elevated oxalic acid is also the result of anti-freeze (ethylene glycol) poisoning. Oxalic acid is a toxic metabolite of trichloroacetic acid and other environmental pollutants. In addition, decomposing vitamin C may form oxalates during transport or storage.

Elevated oxalate values with a concomitant increase in glycolic acid may indicate genetic hyperoxaluria (type I), whereas increased glyceric acid may indicate a genetic hyperoxaluria (type II). Elevated oxalic acid with normal levels of glyceric or glycolic metabolites rules out a genetic cause for high oxalate. However, elevated oxalates may be due to a new genetic disorder, hyperoxaluria type III.

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Regardless of its source, high oxalic acid may contribute to kidney stones and may also reduce ionized calcium. Oxalic acid absorption from the GI tract may be reduced by calcium citrate supplementation before meals. Vitamin B6, arginine, vitamin E, chondroitin sulfate, taurine, selenium, omega-3 fatty acids and/or N-acetyl glucosamine supplements may also reduce oxalates and/or their toxicity. Excessive fats in the diet may cause elevated oxalate if fatty acids are poorly absorbed because of bile salt deficiency. Unabsorbed free fatty acids bind calcium to form insoluble soaps, reducing calcium's ability to bind oxalate and increase its absorption. If taurine is low in a plasma amino acid profile, supplementation with taurine (1000 mg/day) may help stimulate bile salt production (taurocholic acid), leading to better fatty acid absorption and diminished oxalate absorption.

High levels of oxalates are common in autism. Malabsorption of fat and intestinal *Candida* overgrowth are probably the major causes for elevated oxalates in this disorder. Even individuals with elevated glyceric or glycolic acids may not have a genetic disease. To rule out genetic diseases in those people with abnormally high markers characteristic of the genetic diseases, do the following steps: (1) Follow the nutritional steps indicated in this interpretation for one month; (2) If *Candida* is present, treat *Candida* for at least one month; (3) Repeat the organic acid test after abstaining from vitamin C supplements for 48 hours; (4) If the biochemical markers characteristic of genetic oxalate disorders are still elevated in the repeat test, consider DNA tests for the most common mutations of oxalate metabolism. DNA testing for type I hyperoxaluria is available from the Mayo Clinic, Rochester, MN as test #89915 "AGXT Gene, Full Gene Analysis" and, for the p.Gly170Arg mutation only, as # 83643 "Alanine: Glyoxylate Aminotransferase [AGXT] Mutation Analysis [G170R], Blood". Another option to confirm the genetic disease is a plasma oxalate test, also available from the Mayo Clinic (Phone 507.266.5700). Plasma oxalate values greater than 50 micromol/L are consistent with genetic oxalate diseases and may serve as an alternate confirmation test.

Bone tends to be the major repository of excess oxalate in patients with primary hyperoxaluria. Bone oxalate levels are negligible in healthy subjects. Oxalate deposition in the skeleton tends to increase bone resorption and decrease osteoblast activity.

Oxalates may also be deposited in the kidneys, joints, eyes, muscles, blood vessels, brain, and heart and may contribute to muscle pain in fibromyalgia. Oxalate crystal formation in the eyes may be a source of severe eye pain in individuals with autism who may exhibit eye-poking behaviors. High oxalates in the GI tract also may significantly reduce absorption of essential minerals such as calcium, magnesium, zinc, and others. In addition, oxalate deposits in the breast have been associated with breast cancer.

A low oxalate diet may also be particularly useful in the reduction of body oxalates even if dysbiosis of GI flora is the major source of oxalates. Foods especially high in oxalates include spinach, beets, chocolate, soy, peanuts, wheat bran, tea, cashews, pecans, almonds, berries, and many others.

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

1. Avoid spinach, soy, nuts, and berries for one month.
2. If *Candida* is present, treat *Candida* for at least one month.
3. Repeat the organic acid test having abstained from vitamin C supplements for 48 hours.
4. If the biochemical markers characteristic of genetic oxalate disorders are still elevated in the repeat test, consider DNA tests for the most common mutations of oxalate metabolism.

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High 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.

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.

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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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 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 as Chianti wine and vermouth, fermented foods such as cheeses, fish, bean curd, sausage, bologna, pepperoni, sauerkraut, and salami.

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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