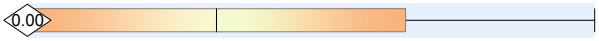
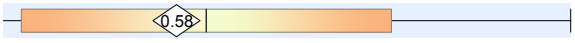
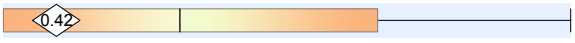
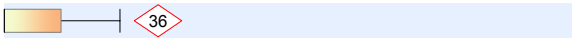
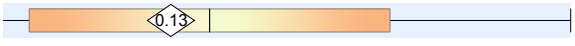
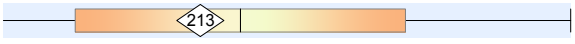
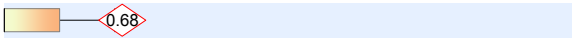
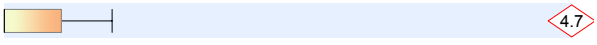
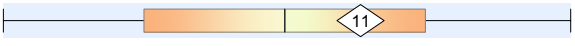
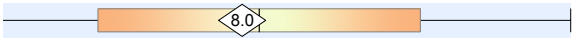
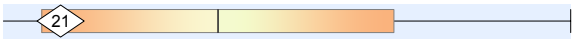
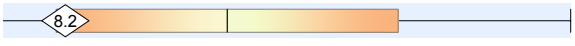
Nordic Laboratories

PATIENT:	**Sample Report**			TEST REF:	**TST-##-#####**
TEST NUMBER:	##########	COLLECTED:	dd/mm/yyyy	PRACTITIONER:	**Nordic Laboratories**
PATIENT NUMBER:	##########	RECEIVED:	dd/mm/yyyy	ADDRESS:	
GENDER:	Female	TESTED:	dd/mm/yyyy		
AGE:	58				
DATE OF BIRTH:	dd-mm-yyyy				

TEST NAME: Organic Acids Test (OAT)

Organic Acids Test - Nutritional and Metabolic Profile

Metabolic Markers in Urine	Reference Range (mmol/mol creatinine)			Patient Value	Reference Population - Females Age 13 and Over
Intestinal Microbial Overgrowth					
Yeast and Fungal Markers					
1 Citramalic	≤ 3.6			0.64	
2 5-Hydroxymethyl-2-furoic	≤ 14			4.2	
3 3-Oxoglutaric	≤ 0.33			0	
4 Furan-2,5-dicarboxylic	≤ 16			7.4	
5 Furancarbonylglycine	≤ 1.9			0.58	
6 Tartaric	≤ 4.5			0.42	
7 Arabinose	≤ 29		H	50	
8 Carboxycitric	≤ 29		H	36	
9 Tricarballylic	≤ 0.44			0.13	
Bacterial Markers					
10 Hippuric	≤ 613			213	
11 2-Hydroxyphenylacetic	0.06	- 0.66	H	0.68	
12 4-Hydroxybenzoic	≤ 1.3		H	4.7	
13 4-Hydroxyhippuric	0.79	- 17		11	
14 DHPPA (Beneficial Bacteria)	≤ 0.38			0.12	
Clostridia Bacterial Markers					
15 4-Hydroxyphenylacetic *(C. difficile, C. stricklandii, C. lituseburense & others)*	≤ 19			8.0	
16 HPHPA *(C. sporogenes, C. caloritolerans, C. botulinum & others)*	≤ 208			21	
17 4-Cresol *(C. difficile)*	≤ 75			8.2	
18 3-Indoleacetic *(C. stricklandii, C. lituseburense, C. subterminale & others)*	≤ 11			1.1	

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PATIENT NUMBER:	##########	RECEIVED:	dd/mm/yyyy	ADDRESS:	
GENDER:	Female	TESTED:	dd/mm/yyyy		
AGE:	58				
DATE OF BIRTH:	dd-mm-yyyy				

TEST NAME: Organic Acids Test (OAT)

	Metabolic Markers in Urine	Reference Range (mmol/mol creatinine)		Patient Value	Reference Population - Females Age 13 and Over
Oxalate Metabolites					
19	Glyceric	0.77 - 7.0		4.5	4.5
20	Glycolic	16 - 117	H	208	208
21	Oxalic	6.8 - 101	H	121	121
Glycolytic Cycle Metabolites					
22	Lactic	≤ 48	H	71	71
23	Pyruvic	≤ 9.1		0.90	0.90
Mitochondrial Markers - Krebs Cycle Metabolites					
24	Succinic	≤ 9.3		5.7	5.7
25	Fumaric	≤ 0.94		0.58	0.58
26	Malic	0.06 - 1.8		1.2	1.2
27	2-Oxoglutaric	≤ 35		12	12
28	Aconitic	6.8 - 28		10	10
29	Citric	≤ 507		259	259
Mitochondrial Markers - Amino Acid Metabolites					
30	3-Methylglutaric	≤ 0.76		0.28	0.28
31	3-Hydroxyglutaric	≤ 6.2		4.4	4.4
32	3-Methylglutaconic	≤ 4.5		1.3	1.3
Neurotransmitter Metabolites					
Phenylalanine and Tyrosine Metabolites					
33	Homovanillic (HVA) *(dopamine)*	0.80 - 3.6		2.2	2.2
34	Vanillylmandelic (VMA) *(norepinephrine, epinephrine)*	0.46 - 3.7		1.2	1.2
35	HVA / VMA Ratio	0.16 - 1.8		1.8	1.8
Tryptophan Metabolites					
36	5-Hydroxyindoleacetic (5-HIAA) *(serotonin)*	≤ 4.3		0.79	0.79
37	Quinolinic	0.85 - 3.9		2.7	2.7
38	Kynurenic	0.17 - 2.2		1.6	1.6
39	Quinolinic / 5-HIAA Ratio	0.42 - 2.0	H	3.4	3.4

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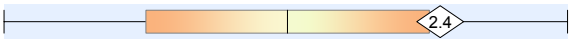
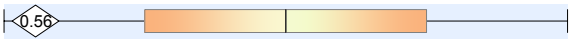
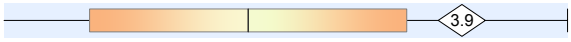
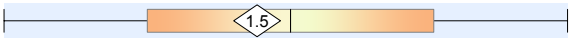
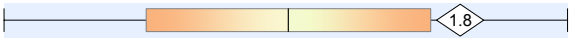
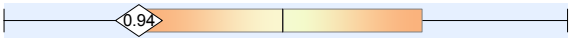
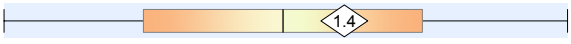
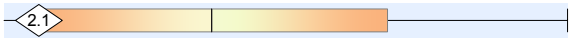
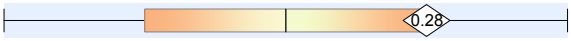
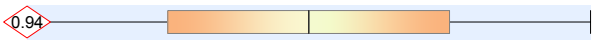
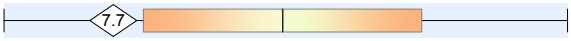
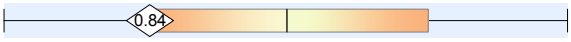
GENDER:	Female	TESTED:	dd/mm/yyyy	ADDRESS:	
AGE:	58				
DATE OF BIRTH:	dd-mm-yyyy				

TEST NAME: Organic Acids Test (OAT)

	Metabolic Markers in Urine	Reference Range (mmol/mol creatinine)	Patient Value	Reference Population - Females Age 13 and Over
Pyrimidine Metabolites - Folate Metabolism				
40	Uracil	≤ 9.7	4.0	
41	Thymine	≤ 0.56	0.21	
Ketone and Fatty Acid Oxidation				
42	3-Hydroxybutyric	≤ 3.1	2.4	
43	Acetoacetic	≤ 10	0.56	
44	4-Hydroxybutyric	≤ 4.8	3.9	
45	Ethylmalonic	0.44 - 2.8	1.5	
46	Methylsuccinic	0.10 - 2.2	1.8	
47	Adipic	0.04 - 3.8	0.94	
48	Suberic	0.18 - 2.2	1.4	
49	Sebacic	≤ 0.24	0.11	
Nutritional Markers				
Vitamin B12				
50	Methylmalonic ✱	≤ 2.3	1.6	
Vitamin B6				
51	Pyridoxic (B6)	≤ 34	2.1	
Vitamin B5				
52	Pantothenic (B5)	≤ 10	4.7	
Vitamin B2 (Riboflavin)				
53	Glutaric ✱	0.04 - 0.36	0.28	
Vitamin C				
54	Ascorbic	10 - 200	L 0.94	
Vitamin Q10 (CoQ10)				
55	3-Hydroxy-3-methylglutaric ✱	0.17 - 39	7.7	
Glutathione Precursor and Chelating Agent				
56	N-Acetylcysteine (NAC)	≤ 0.28	0	
Biotin (Vitamin H)				
57	Methylcitric ✱	0.19 - 2.7	0.84	

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

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TEST NUMBER:	##########	COLLECTED:	dd/mm/yyyy		
PATIENT NUMBER:	##########	RECEIVED:	dd/mm/yyyy	PRACTITIONER:	**Nordic Laboratories**
GENDER:	Female	TESTED:	dd/mm/yyyy	ADDRESS:	
AGE:	58				
DATE OF BIRTH:	dd-mm-yyyy				

TEST NAME: Organic Acids Test (OAT)

	Metabolic Markers in Urine	Reference Range (mmol/mol creatinine)				Patient Value	Reference Population - Females Age 13 and Over
Indicators of Detoxification							
Glutathione							
58	Pyroglutamic ✱	10	-	33		27	27
59	2-Hydroxybutyric ✱	0.03	-	1.8	H	2.8	2.8
Ammonia Excess							
60	Orotic	0.06	-	0.54	H	0.66	0.66
Aspartame, salicylates, or GI bacteria							
61	2-Hydroxyhippuric		≤	1.3		0.45	0.45
	A high value for this marker may indicate a Glutathione deficiency.						
Amino Acid Metabolites							
62	2-Hydroxyisovaleric		≤	0.42		0	0.00
63	2-Oxoisovaleric		≤	2.1		0	0.00
64	3-Methyl-2-oxovaleric		≤	0.87		0.49	0.49
65	2-Hydroxyisocaproic		≤	0.48	H	0.97	0.97
66	2-Oxoisocaproic		≤	0.37		0.17	0.17
67	2-Oxo-4-methiolbutyric		≤	0.16		0.09	0.09
68	Mandelic		≤	0.21		0.13	0.13
69	Phenyllactic		≤	0.20		0	0.00
70	Phenylpyruvic	0.20	-	1.9		1.1	1.1
71	Homogentisic		≤	0.36		0.07	0.07
72	4-Hydroxyphenyllactic		≤	0.80		0.37	0.37
73	N-Acetylaspartic		≤	3.0		0.54	0.54
74	Malonic		≤	9.7		2.8	2.8
Mineral Metabolism							
75	Phosphoric	1,000	-	5,000		3,725	3725

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GENDER:	Female	TESTED:	dd/mm/yyyy		
AGE:	58				
DATE OF BIRTH:	dd-mm-yyyy				

TEST NAME: Organic Acids Test (OAT)

Indicator of Fluid Intake

76	*Creatinine	51	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.

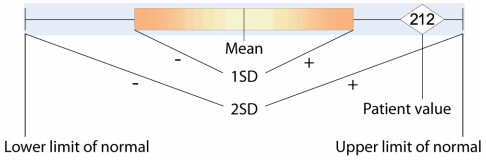
The first graph will occur when the value of the patient is within the reference (normal) range, defined as the mean plus or minus two standard deviations.

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.

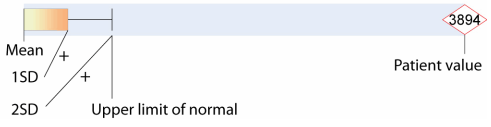
Example of Value Within Reference Range

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	



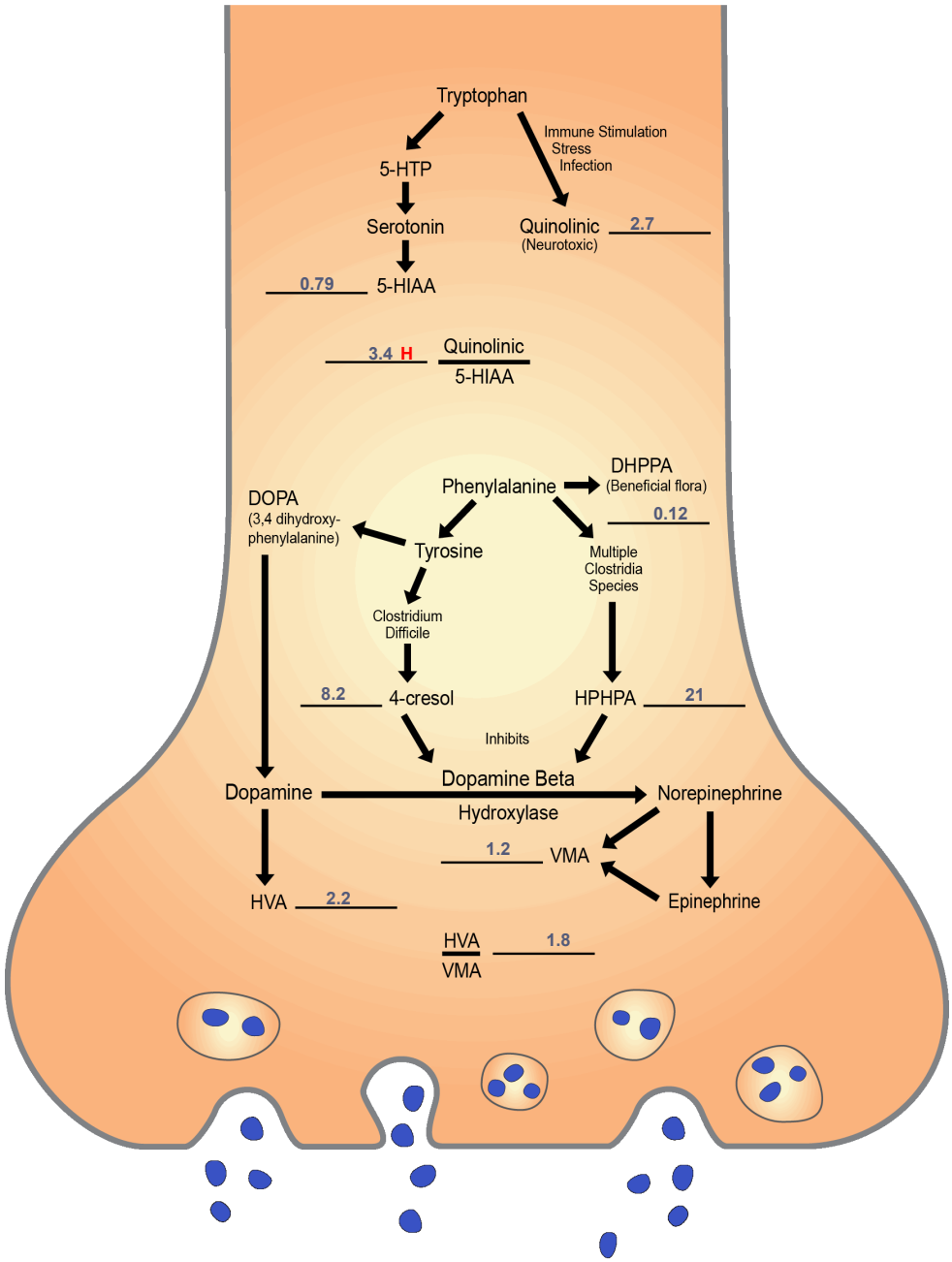
Example of Elevated Value

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	



TEST NAME: Organic Acids Test (OAT)

Neurotransmitter Metabolism Markers



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

TEST NAME: Organic Acids Test (OAT)

Interpretation

High yeast/fungal metabolites (Markers 1,2,3,4,5,6,7,8) indicate a yeast/fungal overgrowth of the gastrointestinal tract. Prescription or natural (botanical) anti-fungals, along with supplementation of high potency multi-strain probiotics (20-50 billion cfu's), may reduce yeast/fungal levels.

High 2-hydroxyphenylacetic acid (Marker 11) is associated with intestinal bacteria overgrowth and with the genetic disease phenylketonuria (PKU).

High 4-hydroxybenzoic acid and/or 4-hydroxyhippuric acid (Markers 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]).

High oxalic with or without elevated glyceric or glycolic acids (Markers 19,20,21) may be associated with the genetic hyperoxalurias, autism, women with vulvar pain, fibromyalgia, and may also be due to high vitamin C intake. However, kidney stone formation from oxalic acid was not correlated with vitamin C intake in a very large study. Besides being present in varying concentrations in most vegetables and fruits, oxalates, the mineral conjugate base forms of oxalic acid, are also byproducts of molds such as *Aspergillus* and *Penicillium* and probably *Candida*. If yeast or fungal markers are elevated, antifungal therapy may reduce excess oxalates. High oxalates may cause anemia that is difficult to treat, skin ulcers, muscles pains, and heart abnormalities. Elevated oxalic acid is also the result of anti-freeze (ethylene glycol) poisoning. Oxalic acid is a toxic metabolite of trichloroacetic acid and other environmental pollutants. In addition, decomposing vitamin C may form oxalates during transport or storage.

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

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

TEST NAME: Organic Acids Test (OAT)

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.

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.

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

VMA levels below the mean (Marker 34) may indicate lower production of the neurotransmitter norepinephrine or the hormone adrenaline, perhaps due to low dietary intake of the amino acid precursors phenylalanine or tyrosine. Vanylmandelic acid (VMA) is a metabolite of norepinephrine or adrenaline. Low VMA may also result from blocked conversion of dopamine to norepinephrine by *Clostridia* metabolites. Supplementation with phenylalanine or tyrosine may be beneficial. Enzyme cofactors magnesium, B6 (pyridoxine) or biopterin may also be deficient and respond to supplementation.

5-hydroxyindoleacetic acid (5-HIAA) levels below the mean (Marker 36) may indicate lower production of the neurotransmitter serotonin. 5-hydroxy-indoleacetic acid is a metabolite of serotonin. Low values have been correlated with symptoms of depression. Supplementation with the precursor 5-HTP (5-hydroxytryptophan) at 50-300 mg/day may be beneficial. Supplementation with tryptophan itself may form the neurotoxic metabolite quinolinic acid, however, 5-HTP is not metabolized to quinolinic acid. Excessive tryptophan supplementation has been associated with eosinophilia myalgia syndrome.

TEST NAME: Organic Acids Test (OAT)

High quinolinic acid / 5-HIAA ratio (Marker 39) indicates an imbalance of these organic acids and may be a sign of neural excitotoxicity. Quinolinic acid is an excitotoxic stimulant of certain brain cells that have NMDA-type receptors. Overstimulated nerve cells may die. Brain toxicity due to quinolinic acid has been implicated in Alzheimer's disease , autism, Huntington's disease, stroke, dementia of old age, depression, HIV-associated dementia, and schizophrenia. However, quinolinic acid is derived from the amino acid tryptophan and is an important intermediate that the body uses to make the essential nutritional cofactor nicotinamide adenine dinucleotide (NAD), which can also be derived from niacin (B3).

An elevated ratio is not specific for a particular medical condition and is commonly associated with excessive inflammation due to recurrent infections. If quinolinic acid is not elevated, low 5- HIAA from serotonin may be the source of the imbalance. Supplementation with 5-HTP may increase serotonin levels, but 5-HTP is not metabolized to quinolinic acid. Immune overstimulation, excess adrenal production of cortisol due to stress, or high exposure to phthalates may also increase the quinolinic acid/5- HIAA acid ratio.

The drug deprenyl or the dietary supplements carnitine, melatonin, capsaicin, turmeric (curcumin) and garlic may reduce brain damage caused by quinolinic acid. Niacin (nicotinic acid) and niacinamide may also reduce quinolinic acid production by decreasing tryptophan shunting to the quinolinic acid pathway. Inositol hexaniacinate as an adult dose of 500-1000 mg does not cause niacin flush.

Pyridoxic acid (B6) levels below the mean (Marker 51) may be associated with less than optimum health conditions (low intake, malabsorption, or dysbiosis). Supplementation with B6 (20 - 50 mg/day) or a multivitamin may be beneficial.

Ascorbic acid (vitamin C) levels below the mean (Marker 54) may indicate a less than optimum level of the antioxidant vitamin C. Suggested supplementation is 1000 mg/ day of buffered vitamin C, divided into 2-3 doses.

High 2-hydroxybutyric acid (Marker 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.
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.

Slightly elevated orotic acid (Marker 60) levels (less than 5 mmol/mol creatinine) are commonly associated with dysbiosis. In this case, the use of probiotics may be beneficial. Elevated orotic acid may also indicate a disorder of ammonia metabolism. It is also possible, but unlikely, that this individual may have an undiagnosed inborn error of metabolism of the urea cycle.

High 2-hydroxyisovaleric acid and/or 2-hydroxyisocaproic acid (Markers 62,65) may be due to the genetic disease MSUD (maple syrup urine disease) or dihydrolipoyl dehydrogenase deficiency. Individuals with slight to moderate elevations may benefit from supplementing with high doses (5-20 mg/kg/day) of thiamine.

Low values for amino acid metabolites (Markers 62-74) indicate the absence of genetic disorders of amino acid metabolism. These markers are deamination (ammonia removed) byproducts that are very elevated only when a key enzyme has low activity; slight elevations may indicate a genetic variation or heterozygous condition which may be mitigated with diet or supplementation. Low values are not associated with inadequate protein intake and have not been proven to indicate specific amino acid deficiencies.