NutriPATH PATHOLOGY · **PATIENT REPORT**

16 Harker St, Burwood VIC, 3125 • info@nutripath.com.au • 1300 688 522

26135-0078

Dr Test Doctor (Test Doctor) Test Clinic. 123 Test Street, Test Suburb Victoria 3125

Lab ID
Patient ID P000060
Ext ID 26135-0078

Test Patient

Sex: Female • 56yrs • 01-Jan-70

123 Home Street, Test Suburb VIC 3125

RECEIVED
15-May-26

Clinical Notes Difficulty Sleeping, Fatigue, Brain Fog, Headaches

ORGANIC ACIDS PROFILING (LC-MS/MS/MS)

Specimen type - Urine, Spot

Collected
12-Apr-26

MICROBIAL OVERGROWTH

2-Hydroxyphenylacetic Acid (2-HPAA)
3,4-Dihydroxyphenylpropionic Acid (DHPPA)
4-Cresol
4-Hydroxyphenyllactic Acid (4-HPLA)
Furancarbonylglycine

YEAST/FUNGAL METABOLITES

5-Hydroxymethyl-2-furoic Acid
Tricarballylic Acid

ENVIRONMENTAL EXPOSURES

Quinolinic Acid

MITOCHONDRIA/ENERGY

2-Hydroxybutyric Acid
Succinic Acid

FATTY ACID / KETONE METABOLISM

Adipic Acid

CARBOHYDRATE / GLYCAEMIC METABOLISM

2-Hydroxybutyric Acid

NEUROTRANSMITTER METABOLISM

Kynurenic Acid
Quinolinic Acid
Quinolinic Acid/5-HIAA Ratio
Vanillylmandelic Acid (VMA)

AMINO ACIDS METABOLISM

4-Hydroxyphenyllactic Acid (4-HPLA)
Guanidinoacetic Acid
Phenylpyruvic Acid

VITAMINS / NUTRITIONAL MARKERS

3-Hydroxy-3-methylglutaric Acid (CoQ10)
Kynurenic Acid
Pantothenic Acid (Vit B5)
Pyroglutamic Acid
Quinolinic Acid

DETOXIFICATION / GLUTATHIONE FUNCTION

5-Hydroxymethyl-2-furoic Acid
Pyroglutamic Acid

OXIDATIVE DAMAGE / INFLAMMATION

Pyroglutamic Acid
Quinolinic Acid

OXALATE METABOLISM

N/A

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ORGANIC ACIDS INTRODUCTION

Organic acids are small carbon-based molecules produced as by-products of everyday metabolic processes in the body. They are measurable in urine and serve as functional windows into the biochemical pathways that govern energy production, detoxification, immune regulation, neurotransmitter synthesis, and nutritional status. Organic acid testing offers a uniquely deep and clinically meaningful picture of a patient's metabolic health.

The Organic Acids Test (OAT) is a comprehensive urine-based analysis that examines over 120 individual metabolic markers across the following key domains:

Microbial Overgrowth - Identifies markers associated with bacterial dysbiosis and overgrowth of potentially pathogenic organisms in the gastrointestinal tract, including phenolic and aromatic compounds produced by microbial fermentation of dietary substrates.

Yeast & Fungal Metabolites - Screens for metabolites associated with yeast and fungal overgrowth, including Candida-related markers such as arabinitol, tartaric acid, and tricarballylic acid, which may interfere with mitochondrial function and nutrient absorption.

Mitochondrial & Energy Metabolism - Evaluates the functional integrity of the citric acid (TCA) cycle through measurement of key intermediates including citric acid, cis-aconitic acid, isocitric acid, and alpha-ketoglutaric acid.

Fatty Acid Oxidation & Ketone Metabolism - Assesses the efficiency of both mitochondrial beta-oxidation and the alternative omega-oxidation pathway through dicarboxylic acid markers, providing insight into fatty acid handling, carnitine sufficiency, and metabolic flexibility.

Amino Acid & Branched-Chain Metabolism - Examines the catabolism of essential and branched-chain amino acids, identifying impairments in leucine, isoleucine, valine, methionine, threonine, and tyrosine pathways that may contribute to fatigue, mood dysregulation, and neurological symptoms.

Neurotransmitter Metabolism -Profiles the major catecholamine and indole pathways, including dopamine, norepinephrine, and serotonin metabolites, as well as the tryptophan-kynurenine pathway, offering functional insight into mood, cognition, stress response, and sleep regulation.

Vitamins & Nutritional Cofactor Markers - Provides functional assessment of B-vitamin status (B1, B2, B3, B5, B6, B12, folate, biotin), glutathione and antioxidant capacity, and key cofactors required for enzymatic activity across multiple metabolic pathways.

Detoxification & Glutathione Function - Evaluates markers of phase I and phase II hepatic detoxification, glutathione cycling via pyroglutamic acid, and NAC status, reflecting the body's capacity to manage oxidative burden and chemical exposure.

Oxidative Stress & Inflammation - Measures markers of DNA oxidative damage and inflammatory mediators, including 8-hydroxy-deoxyguanosine and leukotriene E4, providing a functional picture of systemic oxidative load.

Oxalate Metabolism - Screens for markers of excess oxalate production or absorption, which may be associated with gut dysbiosis, dietary factors, or impaired detoxification.

Environmental & Xenobiotic Exposures - Identifies urinary metabolites of common environmental chemicals including xylene, benzene, toluene, phthalates, parabens, and endocrine-disrupting compounds such as Bisphenol A (BPA) and Bisphenol S (BPS), providing objective evidence of toxic load.

Please Note: We acknowledge that certain compounds appear in multiple sections of the report. This is intentional, as these analytes have relevance across different clinical pathways. Their inclusion in multiple categories supports more accurate pattern recognition and enhances the interpretive value of Organic Acids testing.

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MICROBIAL OVERGROWTH

	TEST	RESULT	H/L	REFERENCE	UNITS
1	Benzoic Acid	4.80		(<9.30)	mmol/molCR
2	Hippuric Acid	188.0		(<603.0)	mmol/molCR
3	4-Hydroxybenzoic Acid (4-HBA)	0.23		(<0.57)	mmol/molCR
4	3,4-Dihydroxybenzoic Acid (3,4-DHBA)	31.00		(<45.00)	mmol/molCR
5	3-Hydroxyphenylacetic Acid (3-HPAA)	2.80		(<10.00)	mmol/molCR
6	3,4-Dihydroxyphenylpropionic Acid (DHPPA)	6.60	H	(<5.30)	mmol/molCR
7	Furancarbonylglycine	7.30	H	(<2.00)	mmol/molCR
	Amino Acid Metabolism				
8	Phenylacetic Acid (PAA)	1.22		(<3.90)	mmol/molCR
9	Phenylpropionic Acid	0.04		(<0.10)	mmol/molCR
10	3-Phenyllactic Acid (3-PLA)	0.98		(<2.00)	mmol/molCR
11	2-Hydroxyphenylacetic Acid (2-HPAA)	11.10	H	(0.05-0.70)	mmol/molCR
12	4-Hydroxyphenyllactic Acid (4-HPLA)	12.40	H	(<3.90)	mmol/molCR
	SCFA Metabolism				
13	3-Hydroxypropionic Acid (3-HPA)	4.10		(<17.00)	mmol/molCR
	Clostridial Markers				
14	4-Cresol	14.30	H	(<3.00)	ug/mgCR
15	4-Hydroxyphenylacetic Acid (4-HPAA)	3.90		(<14.60)	mmol/molCR
16	Indoleacetic Acid (IAA)	3.90		(<11.00)	mmol/molCR
17	3-(3-Hydroxyphenyl)-3-hydroxypropionic Acid (HPHPA)	41.60		(<120.00)	mmol/molCR

YEASTS & FUNGAL METABOLITES

	TEST	RESULT	H/L	REFERENCE	UNITS
18	Arabinitol	18.6		(<36.0)	mmol/molCR
19	Arabinose	12.20		(<32.00)	mmol/molCR
20	Citramalic Acid	0.95		(<3.60)	mmol/molCR
21	3-Oxoglutaric Acid	0.36		(<0.50)	mmol/molCR
	Aspergillus Markers				
22	5-Hydroxymethyl-2-furoic Acid	22.10	H	(<10.00)	mmol/molCR
23	Furan-2,5-dicarboxylic Acid	5.20		(<15.00)	mmol/molCR
24	Tartaric Acid	6.90		(<15.00)	mmol/molCR
	Fusarium				
25	Tricarballylic Acid	21.40	H	(<0.44)	mmol/molCR

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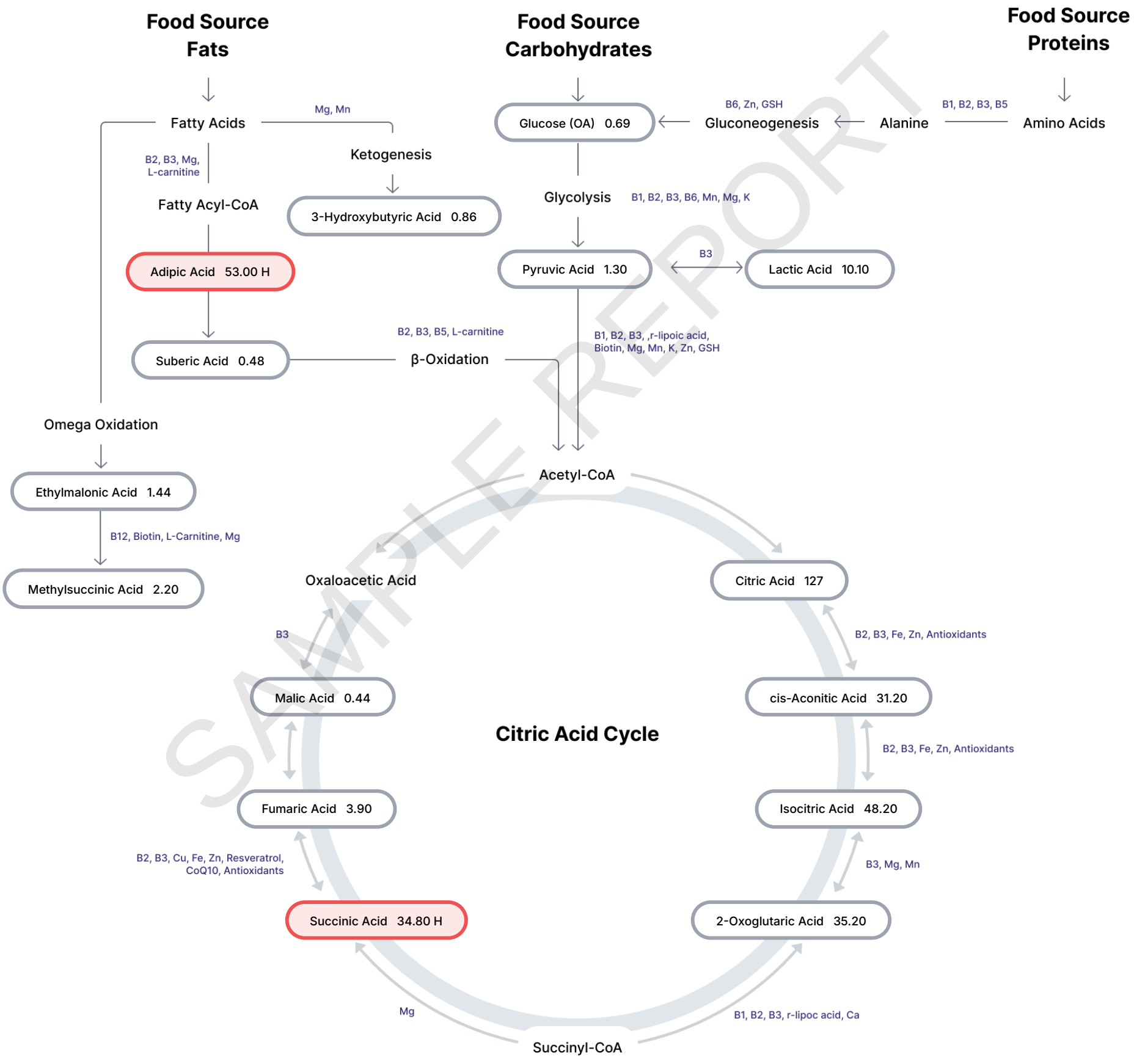
Organic Acids Pathway



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MITOCHONDRIAL & ENERGY METABOLISM

(B Comp, Cr, CoQ10, Lipoic Acid, Amino Acids, Mg)

	TEST	RESULT	H/L	REFERENCE	UNITS
	Carbohydrate & Glycaemic Metabolism				
26	Glucose (OA)	0.69		(<1.10)	ug/mgCR
27	Pyruvic Acid	1.30		(0.50-8.70)	mmol/molCR
28	Lactic Acid	10.10		(<48.00)	mmol/molCR
29	2-Hydroxybutyric Acid	38.10	H	(<6.90)	mmol/molCR
	Citric Acid Cycle				
30	Citric Acid	127		(40-507)	mmol/molCR
31	cis-Aconitic Acid	31.20		(3.50-36.00)	mmol/molCR
32	Isocitric Acid	48.20		(5.00-65.00)	mmol/molCR
33	2-Oxoglutaric Acid	35.20		(2.00-45.00)	mmol/molCR
34	Succinic Acid	34.80	H	(1.00-15.00)	mmol/molCR
35	Fumaric Acid	3.90		(<8.60)	mmol/molCR
36	Malic Acid	0.44		(<1.80)	mmol/molCR
	Ketone/Energy Intermediates				
37	Acetoacetic Acid	1.90		(<10.00)	mmol/molCR
38	3-Hydroxybutyric Acid	0.86		(<3.10)	mmol/molCR
39	2-Hydroxybutyric Acid	38.10	H	(<6.90)	mmol/molCR
	Mitochondrial Markers				
40	Methylsuccinic Acid	2.20		(<10.80)	mmol/molCR
41	2-Methylglutaric Acid	0.22		(<0.76)	mmol/molCR
42	3-Methylglutaric Acid	2.63		(<8.50)	mmol/molCR
43	2-Hydroxyglutaric Acid	7.70		(<15.00)	mmol/molCR
44	3-Hydroxyglutaric Acid	1.45		(<5.50)	mmol/molCR
45	Malonic Acid	3.50		(<9.70)	mmol/molCR
46	Mevalonolactone	0.75		(<2.00)	mmol/molCR
47	2,4-Dihydroxybutanoic Acid	3.10		(<10.00)	mmol/molCR
48	N-Acetylaspartic Acid	1.66		(<15.00)	mmol/molCR
49	3-Methylglutaconic Acid	0.59		(<5.50)	mmol/molCR

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FATTY ACID OXIDATION & KETONE METABOLISM

	TEST	RESULT	H/L	REFERENCE	UNITS
50	Adipic Acid	53.00	H	(<3.80)	mmol/molCR
51	Pimelic Acid	1.86		(<4.00)	mmol/molCR
52	Suberic Acid	0.48		(<2.20)	mmol/molCR
53	Azelaic Acid	3.10		(<10.00)	mmol/molCR
54	Sebacic Acid	0.15		(<0.24)	mmol/molCR
55	Ethylmalonic Acid	1.44		(<5.80)	mmol/molCR
56	Propionylglycine	0.86		(<2.00)	mmol/molCR
57	N-Butyrylglycine	0.63		(<3.00)	mmol/molCR
58	Isovalerylglycine	0.09		(<4.50)	mmol/molCR
59	N-(2-Methylbutyryl)glycine	0.12		(<2.00)	mmol/molCR
60	3-Methylcrotonylglycine	2.20		(<10.00)	mmol/molCR
61	Tiglylglycine	0.55		(<10.00)	mmol/molCR
62	Hexanoylglycine	1.47		(<10.00)	mmol/molCR
63	Suberylglycine	0.85		(<3.00)	mmol/molCR

VITAMIN & NUTRITIONAL COFACTOR MARKERS

	TEST	RESULT	H/L	REFERENCE	UNITS
	B-Vitamin Functional Markers				
64	Methylmalonic Acid (MMA)	1.24		(<1.90)	mmol/molCR
65	Formiminoglutamic Acid (FIGLU)	1.08		(<1.50)	mmol/molCR
66	Xanthurenic Acid	0.71		(<0.96)	mmol/molCR
67	Kynurenic Acid	72.00	H	(<2.20)	mmol/molCR
68	Quinolinic Acid	71.00	H	(<9.10)	mmol/molCR
69	Picolinic Acid	2.30		(<10.28)	mmol/molCR
70	3-Hydroxyisovaleric Acid	8.20		(<29.00)	mmol/molCR
	Vitamin-specific/Antioxidant Markers				
71	Pyroglutamic Acid	74.00	H	(4.50-33.00)	mmol/molCR
72	N-Acetylcysteine (NAC)	0.17		(0.02-0.28)	mmol/molCR
73	Glutaric Acid (Vit B2)	0.11		(0.02-0.36)	mmol/molCR
74	Pantothenic Acid (Vit B5)	0.09	L	(0.10-10.00)	mmol/molCR
75	Pyridoxic Acid (Vit B6)	4.90		(0.68-34.00)	mmol/molCR
76	Ascorbic Acid (Vit C)	74.00		(0.50-200.00)	mmol/molCR
77	Methylcitric Acid (Biotin/Vitamin H)	5.10		(0.10-15.00)	mmol/molCR
78	3-Hydroxy-3-methylglutaric Acid (CoQ10)	81.00	H	(0.10-5.00)	mmol/molCR

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PYRIMIDINE METABOLITES - Folate Metabolism

TEST		RESULT	H/L	REFERENCE	UNITS
79	Thymine	0.13		(<0.60)	mmol/molCR
80	Uracil	2.86		(<9.00)	mmol/molCR

AMINO ACID & BRANCHED-CHAIN METABOLISM

(B1, B2, B3, B5, B6, B12, Folate, Biotin)

TEST		RESULT	H/L	REFERENCE	UNITS
Branched-Chain Ketoacids					
81	2-Oxoisovaleric Acid	1.26		(<4.10)	mmol/molCR
82	2-Oxoisocaproic Acid	0.21		(<0.65)	mmol/molCR
83	3-Methyl-2-oxovaleric Acid	0.55		(<2.00)	mmol/molCR
84	3-Methylglutaric Acid	2.63		(<8.50)	mmol/molCR
85	Succinylacetone	0.03		(<0.50)	mmol/molCR
Downstream Metabolites					
86	2-Hydroxyisovaleric Acid	1.12		(<4.10)	mmol/molCR
87	2-Hydroxyisocaproic Acid	0.63		(<1.50)	mmol/molCR
88	2-Oxobutyric Acid	2.20		(<7.00)	mmol/molCR
89	2-Oxo-4-methiolbutyric Acid (KMBA)	92.00	H	(<1.50)	mmol/molCR
Amino Acid Metabolism					
90	Phenylpyruvic Acid	93.00	H	(<2.00)	mmol/molCR
91	Homogentisic Acid	0.47		(<1.00)	mmol/molCR
92	N-Acetylphenylalanine	1.15		(<5.00)	mmol/molCR
93	Mandelic Acid	145.0		(<340.0)	ug/gCR
94	Malonic Acid	3.50		(<9.70)	mmol/molCR
95	4-Hydroxyphenyllactic Acid (4-HPLA)	12.40	H	(<3.90)	mmol/molCR
96	2-Oxoadipic Acid	0.81		(<2.00)	mmol/molCR
97	Guanidinoacetic Acid	1100.00	H	(0.50-3.00)	mmol/molCR
98	Guanidinobutyric Acid	0.22		(0.10-1.00)	mmol/molCR
99	3-Methylglutaric Acid	2.63		(<8.50)	mmol/molCR
100	N-Acetylglycine	0.79		(<5.00)	mmol/molCR

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NEUROTRANSMITTER METABOLITES

(Phenylalanine, Tyrosine)

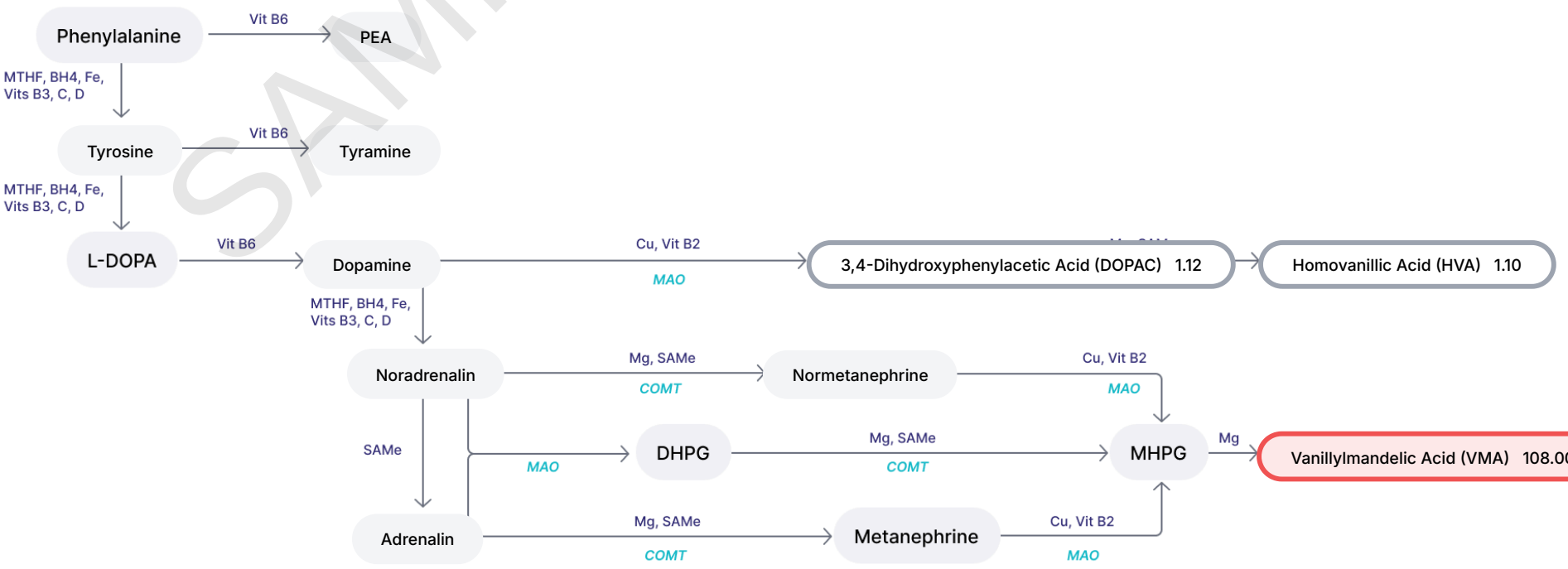
TEST	RESULT	H/L	REFERENCE	UNITS
Inhibitory (Serotonin)				
101 5-Hydroxyindoleacetic Acid (5-HIAA)	2.89		(<4.30)	mmol/molCR
Excitatory (Dopamine)				
102 3,4-Dihydroxyphenylacetic Acid (DOPAC)	1.12		(0.08-3.50)	mmol/molCR
103 3-Methyl-4-hydroxyphenylglycol (MHPG)	0.16		(0.05-0.50)	mmol/molCR
104 Homovanillic Acid (HVA)	1.10		(0.10-5.30)	mmol/molCR
105 Vanillylmandelic Acid (VMA)	108.00	H	(0.40-3.60)	mmol/molCR
106 HVA/DOPAC Ratio	1.0		(<10.0)	ratio
107 HVA/VMA Ratio	0.0		(<2.0)	ratio

ADRENAL STRESS (Overnight)

SERVICE	RESULT	H/L	REFERENCE	UNITS
108 Cortisol (OA)	12.8		(1.0-63.8)	ug/gCR

Legend Not Tested | Within Range | Out of Range | L = Low, LL = Critically Low | H = High, HH = Critically High | Regulator | *Enzyme*

Excitatory Pathway



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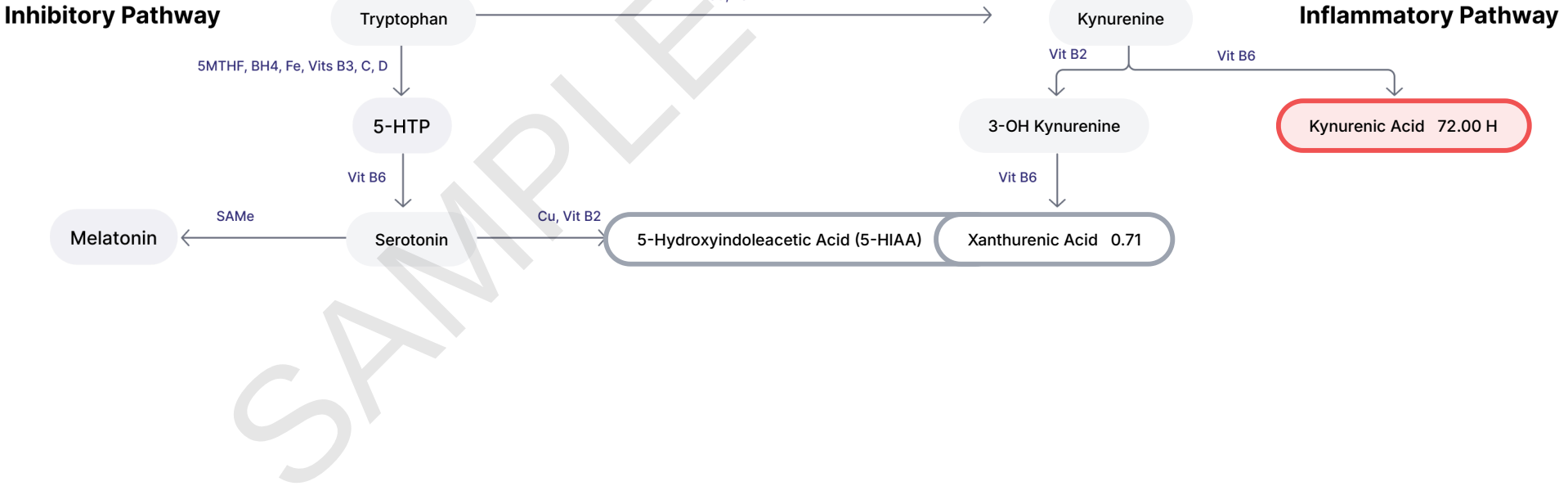
TRYPTOPHAN/KYNURENINE PATHWAY

(Tryptophan, B6, Antioxidants)

TEST	RESULT	H/L	REFERENCE	UNITS
Inhibitory				
109 5-Hydroxyindoleacetic Acid (5-HIAA)	2.89		(<4.30)	mmol/molCR
Inflammatory				
110 Kynurenic Acid	72.00	H	(<2.20)	mmol/molCR
111 Quinolinic Acid	71.00	H	(<9.10)	mmol/molCR
112 Picolinic Acid	2.30		(<10.28)	mmol/molCR
113 Xanthurenic Acid	0.71		(<0.96)	mmol/molCR
114 Kynurenic/Quinolinic Ratio	1.0		(<2.0)	ratio
115 Quinolinic Acid/5-HIAA Ratio	24.6	H	(<5.0)	ratio

Legend Not Tested | Within Range | Out of Range | L = Low, LL = Critically Low | H = High, HH = Critically High | Regulator | *Enzyme*



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DETOXIFICATION & GLUTATHIONE FUNCTION

(Arg, NAC, Meth, Mg, Antioxidants)

TEST	RESULT	H/L	REFERENCE	UNITS
Ammonia Metabolism				
116 Orotic Acid	0.88		(<3.20)	mmol/molCR
Glutathione Metabolism				
117 Pyroglutamic Acid	74.00	H	(4.50-33.00)	mmol/molCR
118 N-Acetylcysteine (NAC)	0.17		(0.02-0.28)	mmol/molCR
119 Glucaric Acid	4.85		(<11.00)	mmol/molCR
Phase I / Xenobiotic Markers				
120 2-Hydroxyhippuric Acid	0.79		(<1.50)	mmol/molCR
121 5-Hydroxymethyl-2-furoic Acid	22.10	H	(<10.00)	mmol/molCR
122 Furan-2,5-dicarboxylic Acid	5.20		(<15.00)	mmol/molCR

OXIDATIVE STRESS & INFLAMMATION

(Vitamin C, Other Antioxidants)

TEST	RESULT	H/L	REFERENCE	UNITS
123 8-hydroxy-deoxyguanosine	0.93		(<2.70)	umol/molCR
124 Leukotriene E4	48.60		(<100.00)	pg/mgCR
125 Quinolinic Acid	71.00	H	(<9.10)	mmol/molCR
126 Pyroglutamic Acid	74.00	H	(4.50-33.00)	mmol/molCR
127 Ascorbic Acid (Vit C)	74.00		(0.50-200.00)	mmol/molCR
128 Quercetin	0.19		(0.01-2.00)	mmol/molCR

OXALATE METABOLISM

TEST	RESULT	H/L	REFERENCE	UNITS
129 Glycolic Acid	19.10		(<67.00)	mmol/molCR
130 Glyceric Acid	2.20		(<6.00)	mmol/molCR
131 Oxalic Acid	13.50		(<78.00)	mmol/molCR

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ENVIRONMENTAL / XENOBIOTIC EXPOSURE

	TEST	RESULT	H/L	REFERENCE	UNITS
	Toluene Exposure				
132	Hippuric Acid	188.0		(<603.0)	mmol/molCR
133	Benzoic Acid	4.80		(<9.30)	mmol/molCR
	Paraben Exposure				
134	4-Hydroxybenzoic Acid (4-HBA)	0.23		(<0.57)	mmol/molCR
	Styrene Exposure				
135	Mandelic Acid	145.0		(<340.0)	ug/gCR
136	Phenylglyoxylic Acid	131.0		(<300.0)	ug/gCR
137	Mandelic Acid + Phenylglyoxylic Acid	276.0		(<610.0)	ug/gCR
	Benzene Exposure				
138	t,t-Muconic Acid	<DL		(<0.12)	mmol/molCR
	Phthalate Exposure				
139	Phthalic Acid	26.00		(<170.00)	ug/gCR
140	Monoethyl Phthalate	13.00		(<100.00)	ug/gCR
141	Quinolinic Acid	71.00	H	(<9.10)	mmol/molCR
	METB Exposure				
142	2-Hydroxylsobutyric Acid	1.05		(<6.90)	mmol/molCR
	Xylene Exposure				
143	2-Methylhippuric Acid	0.01		(<0.04)	mmol/molCR
144	3-Methylhippuric Acid	<DL		(<0.11)	mmol/molCR
145	4-Methylhippuric Acid	0.00		(<1.80)	mmol/molCR
	Trimethylbenzene Exposure				
146	3,4-Dimethylhippuric Acid	0.00		(<0.01)	mmol/molCR
147	4-Hydroxyhippuric Acid	0.00		(<16.50)	mmol/molCR
	Nucleotide Turnover/Methylation				
148	5-Methylcytosine	13.00		(10.00-50.00)	mmol/molCR
149	Uracil	2.86		(<9.00)	mmol/molCR
150	Thymine	0.13		(<0.60)	mmol/molCR

TEST		RESULT	H/L	REFERENCE	UNITS
151	Creatinine, Urine	3.10		(2.47-19.20)	mmol/L

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NUTRITION GUIDE

SERVICE	Nutritional Need	DOSE	UNITS
Vitamins			
Vitamin-B1	Moderate	90.0	mg
Vitamin-B2	HIGH	80.0	mg
Vitamin-B3	Adequate	0.0	mg
Vitamin-B5	Mild	60.0	mg
Vitamin-B6	HIGH	20.0	mg
Biotin	Adequate	0.0	ug
Vitamin B12	Adequate	0.0	ug
Vitamin-C	Moderate	660.0	mg
Vitamin-E	Adequate	0.0	U
Minerals			
Chromium	Adequate	0.0	ug
Iron	HIGH	18.0	mg
Magnesium	Adequate	0.0	mg
Manganese	Moderate	6.0	mg
Vanadium	Mild	20.0	ug
Antioxidants/Cofactors			
alpha-Lipoic Acid	Adequate	0.0	mg
Calcium-D-glucarate	Adequate	0.0	mg
Coenzyme Q10	Adequate	0.0	mg
Glutathione		10.0	mg
N-Acetylcysteine	Adequate	0.0	mg

NUTRITION GUIDE

SERVICE	Nutritional Need	DOSE	UNITS
Amino Acids			
5-HTP	HIGH	80.0	mg
Acetyl-L-Carnitine	Adequate	0.0	mg
L-Arginine	Adequate	0.0	mg
Aspartic Acid	Adequate	0.0	mg
Glutamine	Adequate	0.0	mg
Glycine	Adequate	0.0	mg
Lysine	Adequate	0.0	mg
Methionine	Adequate	0.0	mg
Ornithine	Adequate	0.0	mg
Phenylalanine	Adequate	0.0	mg
Serine	Adequate	0.0	mg
Taurine	Adequate	0.0	mg
Tryptophan	Adequate	0.0	mg
Tyrosine	Adequate	0.0	mg
Probiotics			
D-Lactate-free probiotics	Mild	20.0	billion CFU
Lactobacillus	HIGH	40.0	billion CFU
Probiotics (Multistrain)	Mild	20.0	billion CFU
Other			
Malic Acid	Adequate	0.0	mg
EPA/DHA	Adequate	0.0	mg
Folinic Acid	Adequate	0.0	ug

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About the Nutrition Guide & Supplement Schedule

The Nutrition Guide presented in this report represents a personalised supplementation schedule generated directly from the organic acid findings of this individual patient. It is not a generic recommendation it is dynamically calculated for each patient based on the specific metabolic pattern identified across all measured domains.

The schedule is generated using a proprietary weighted algorithm developed by our clinical and scientific team. The algorithm analyses the full pattern of organic acid results not individual markers in isolation and assigns a weighted score to each finding based on its functional significance, its relationship to nutrient-dependent enzymatic pathways, and its interaction with other metabolic markers present in the same report. The outcome of this calculation determines both the nutritional priority level (Adequate, Mild, Moderate, or High) and the suggested starting dose for each nutrient, amino acid, probiotic, or cofactor listed.

Important Disclaimer

The Nutrition Guide and supplementation suggestions in this report are generated for clinical decision-support purposes only and are not intended as standalone medical advice or a prescription.

The proprietary algorithm used to generate this schedule is based on established relationships between organic acid biomarkers and nutritional cofactor requirements. However, it operates solely on biochemical data and cannot account for a patient's complete medical history, current medications, existing diagnoses, organ function, pregnancy or breastfeeding status, or any other health condition that may influence the safety or appropriateness of the suggested nutrients and doses.

The final therapeutic decision including whether to implement, modify, or withhold any recommendation in this report rests entirely with the treating practitioner. It is the practitioner's responsibility to integrate these findings with the full clinical picture before making any therapeutic recommendation.

This report does not diagnose, treat, cure, or prevent any disease or medical condition.

Nutritional Guide Practitioner Notes

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Bacterial Dysbiosis Comment

DIHYDROXYPHENYLPROPIONIC ACID ELEVATED (URINE):

Elevated urinary dihydroxyphenylpropionic acid suggests increased microbial metabolism of dietary polyphenols or altered handling of aromatic compounds within the gut. This metabolite is commonly produced by intestinal bacteria from plant-derived phenolic substrates.

Clinically, elevated dihydroxyphenylpropionic acid may be associated with gastrointestinal symptoms such as bloating or altered bowel habits, as well as non-specific symptoms including fatigue or chemical sensitivity, although findings are often asymptomatic.

From an organic acid pattern perspective, elevated dihydroxyphenylpropionic acid may be observed alongside other aromatic and polyphenol-derived metabolites, such as hippuric acid or phenylacetic acid, reflecting increased microbial fermentation activity within the gut.

From a functional medicine perspective, this finding should be interpreted in the context of dietary polyphenol intake, gut microbiome balance, and intestinal transit rather than as a marker of pathology in isolation.

FURANCARBONYLGLYCINE ELEVATED:

Furancarbonylglycine (FCG) is a glycine-conjugated metabolite formed during the hepatic detoxification of furan-derived compounds, which are commonly generated during the thermal processing of foods and from certain environmental exposures.

Elevated urinary furancarbonylglycine may reflect increased exposure to furan-containing compounds, with subsequent metabolism via glycine conjugation pathways and renal excretion. This is most commonly associated with dietary intake of heat-processed foods, although environmental sources may also contribute.

As a phase II detoxification product, this marker primarily reflects exposure and metabolic processing rather than intrinsic metabolic dysfunction, and is therefore non-specific when interpreted in isolation.

Suggested Treatment Considerations:

Consider review of dietary intake, particularly consumption of highly processed or heat-treated foods. Assessment of environmental exposures may be appropriate where clinically indicated. No specific medical intervention is typically required beyond addressing identifiable sources. Interpretation should be guided by the overall clinical context and associated findings.

2-HYDROXYPHENYLACETIC ACID ELEVATED:

2-Hydroxyphenylacetic acid is a phenolic metabolite derived from the metabolism of the amino acid tyrosine, arising through combined host and gastrointestinal microbial pathways. It represents a minor urinary metabolite reflecting aromatic amino acid metabolism and microbial activity.

Elevated urinary 2-hydroxyphenylacetic acid may be associated with increased microbial metabolism of tyrosine within the gastrointestinal tract, and may be observed in the context of altered gut microbial activity or dysbiosis. In addition, elevations may reflect increased substrate availability or altered host metabolism of aromatic amino acids.

This marker is non-specific and should be interpreted in conjunction with other phenolic metabolites and markers of gastrointestinal function to assess potential patterns of microbial metabolism.

Suggested Treatment Considerations:

Consider correlation with other markers of microbial metabolism and gastrointestinal function where clinically indicated. Review dietary intake of protein and aromatic amino acids. Further evaluation of gastrointestinal health and microbial balance may be appropriate. Management should be guided by the overall clinical context and associated laboratory findings rather than the isolated marker elevation.

4-CRESOL ELEVATED (URINE):

p-Cresol is primarily produced by gut bacteria during protein fermentation (notably from tyrosine). Elevation often indicates dysbiosis and increased putrefactive metabolism, and can place additional demand on sulphation pathways.

Clinically, elevations may be associated with bloating, constipation, fatigue, brain fog, and chemical sensitivity. Interpretation should consider recent exposure timing, hydration status, and overall detoxification capacity.

From a functional medicine perspective, management focuses on improving bowel transit, reducing excessive protein putrefaction, increasing dietary fibre and polyphenols, and targeted gut microbiome support; ensure adequate sulphation support (e.g., B6, magnesium, molybdenum where indicated).

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TRICARBALLYLIC ACID ELEVATED (URINE):

Elevated urinary tricarballylic acid suggests exposure to fungal-derived metabolites, commonly associated with certain mycotoxins, or altered gut microbial metabolism.

Clinically, elevated tricarballylic acid may be associated with fatigue, gastrointestinal symptoms, or reduced mineral availability, although symptoms are often non-specific.

From an organic acid pattern perspective, tricarballylic acid may be observed alongside other markers of microbial or mycotoxin-related metabolic interference, and it may competitively inhibit aconitase activity within the TCA cycle.

From a functional medicine perspective, this finding should be interpreted in the context of gut microbial balance, environmental exposure history, and mitochondrial energy patterns.

Yeast Dysbiosis Comment

5-HYDROXYMETHYL-2-FUROIC ACID ELEVATED:

5-Hydroxymethyl-2-furoic acid (5-HMFA) is a urinary metabolite derived from the metabolism of 5-hydroxymethylfurfural (HMF), a compound formed during the thermal processing of carbohydrate-containing foods, particularly under high heat conditions (e.g. baking, roasting, or caramelisation).

Elevated urinary 5-HMFA typically reflects increased dietary exposure to heat-processed foods, with subsequent hepatic metabolism and excretion. As such, this marker is primarily considered an indicator of dietary intake and exposure, rather than endogenous metabolic dysfunction.

In some contexts, elevated levels may also reflect increased formation of furan-derived compounds associated with carbohydrate degradation during cooking. This finding is non-specific and should be interpreted in conjunction with dietary history and other relevant exposure markers.

Suggested Treatment Considerations:

Review dietary intake, particularly consumption of highly processed or heat-treated foods. Where appropriate, consider reducing intake of foods subjected to high-temperature processing. No specific medical intervention is typically required beyond addressing identifiable dietary sources. Interpretation should be guided by the overall clinical context and associated findings.

Mitochondrial/Energy Metabolism Comment

SUCCINIC ACID ELEVATED (URINE):

Elevated urinary succinic acid suggests impaired downstream processing within the tricarboxylic acid (TCA) cycle or reduced efficiency of the electron transport chain. Accumulation of succinic acid may reflect mitochondrial bottlenecks or altered redox balance.

Clinically, elevated succinic acid may be associated with fatigue, reduced exercise tolerance, and symptoms related to impaired aerobic energy production.

From an organic acid pattern perspective, elevated succinic acid often occurs alongside elevations in alpha-ketoglutaric acid and other downstream TCA intermediates, indicating congestion within mitochondrial energy pathways and increased reliance on compensatory metabolic mechanisms.

From a functional medicine perspective, elevated succinic acid should be interpreted in the context of mitochondrial efficiency, oxidative stress, and overall metabolic demand rather than concentration alone.

Elevated succinate may indicate a deficiency of Riboflavin and CoQ10.

Fatty Acid Oxidation & Ketone Metabolism

ADIPIC ACID ELEVATED (URINE):

Adipic acid is a dicarboxylic fatty acid that increases when mitochondrial beta-oxidation is inefficient, often reflecting impaired fatty acid utilisation or carnitine insufficiency.

Clinically, elevations may be associated with fatigue, reduced exercise tolerance, muscle weakness, brain fog, or difficulty regulating weight. Interpretation should consider dietary intake, medications, and the broader metabolic context.

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From a functional medicine perspective, management focuses on supporting mitochondrial beta-oxidation with carnitine, riboflavin (B2), niacin (B3), magnesium, CoQ10, optimising glycaemic control, and reducing excess dietary fat load, addressing underlying contributors rather than isolated suppression of the marker.

2-HYDROXYBUTYRIC ACID ELEVATED (URINE):

Alpha-hydroxybutyric acid is linked to glutathione demand and early insulin resistance. Elevation reflects oxidative stress and altered glucose metabolism.

Clinically, elevations may be associated with fatigue, blood sugar instability, and reduced stress tolerance. Results should be interpreted alongside diet, environmental exposures, medications, and overall clinical context.

From a functional medicine perspective, management focuses on improving insulin sensitivity, supporting glutathione synthesis, and reducing oxidative load, with emphasis on reducing exposure and supporting metabolic clearance pathways.

Treatment: Treatment may involve lifestyle changes, such as diet and exercise, and ensuring adequate antioxidant intake.

B-Vitamins/Amino Acids Comment

2-OXO-4-METHYLTHIOBUTYRIC ACID ELEVATED:

Description:

2-Oxo-4-methylthiobutyric acid (KMBA; also known as α-keto-γ-methiolbutyric acid) is the α-keto acid analogue of methionine, formed at the entry point of methionine catabolism via transamination. The reaction is catalysed by branched-chain aminotransferases, which transfer the amino group from methionine to an α-keto acid acceptor (typically α-ketoglutarate), producing KMBA. Under normal conditions, KMBA is further processed through the methionine salvage pathway to propionyl-CoA and methanethiol, ultimately entering the TCA cycle via succinyl-CoA. Elevated urinary KMBA reflects impaired downstream catabolism due to insufficient B6, B12, or folate cofactors; excess methionine substrate availability; reduced methylation capacity; or non-enzymatic generation from methionine via reactive oxygen species under conditions of elevated oxidative stress. KMBA therefore signals potential disruption at the critical junction between methionine metabolism, SAMe synthesis, transsulfuration, and glutathione production.

Clinical Significance:

Elevated KMBA indicates impaired methionine catabolism and/or transsulfuration pathway dysfunction with downstream consequences for methylation and glutathione synthesis. Reduced SAMe production impairs methylation reactions essential for neurotransmitter synthesis (dopamine, serotonin, norepinephrine), DNA and histone methylation, phospholipid metabolism, and immune regulation. Impaired transsulfuration reduces cysteine availability and consequently limits glutathione production compromising phase II detoxification, antioxidant defence, and the body's capacity to manage chemical and oxidative burden. If remethylation is concurrently impaired, homocysteine may accumulate, representing an independent cardiovascular and neurological risk factor. Clinically, this pattern is associated with fatigue, cognitive impairment and brain fog, poor detoxification, chemical sensitivity, mood dysregulation, and susceptibility to inflammatory and autoimmune conditions. The combination of elevated KMBA, elevated xanthurenic acid, and low pyroglutamic acid is a strong functional indicator of B6 insufficiency as the primary driver. Plasma homocysteine and serum B12, folate, and B6 levels should be assessed to directly characterise methylation pathway status.

PHENYLPYRUVIC ACID ELEVATED:

Phenylpyruvic acid is a keto-acid formed during the catabolism of phenylalanine, typically generated when phenylalanine is transaminated rather than converted via its primary metabolic pathway to tyrosine.

Elevated urinary phenylpyruvic acid may reflect impaired phenylalanine metabolism, most notably due to reduced activity of phenylalanine hydroxylase, as seen in phenylketonuria (PKU). In such cases, accumulation of phenylalanine leads to increased production of alternative metabolites, including phenylpyruvate.

Milder elevations may be observed in the context of increased phenylalanine load, altered amino acid metabolism, or metabolic stress, although these findings are non-specific and should be interpreted cautiously.

Given its clinical relevance, particularly in inherited metabolic disorders, this marker should be interpreted in conjunction with plasma amino acid profiles and other phenylalanine-related metabolites.

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Consider correlation with plasma phenylalanine levels and clinical history. In cases of significant or persistent elevation, further evaluation for disorders of phenylalanine metabolism, including phenylketonuria, may be warranted. Referral for specialist metabolic assessment may be appropriate. Management should be guided by the underlying clinical diagnosis and overall context.

GUANIDINOACETIC ACID ELEVATED:

Guanidinoacetic acid (GAA) is an intermediate in creatine biosynthesis, formed from arginine and glycine via arginine:glycine amidinotransferase (AGAT) and subsequently converted to creatine by guanidinoacetate methyltransferase (GAMT) using S-adenosylmethionine (SAM) as a methyl donor.

Elevated urinary GAA may indicate impaired conversion of guanidinoacetate to creatine, which can occur in the context of guanidinoacetate methyltransferase (GAMT) deficiency, a rare inherited metabolic disorder. More commonly, elevated levels may reflect relative inefficiency in methylation-dependent conversion, increased endogenous synthesis, or altered creatine metabolism.

From a biochemical perspective, accumulation of GAA may be associated with increased demand on methylation pathways, as the conversion of GAA to creatine represents a significant consumer of methyl groups. As such, elevations may also be observed in the context of impaired one-carbon metabolism or reduced methyl donor availability.

This marker should be interpreted cautiously and in conjunction with other indicators of creatine metabolism and methylation status.

Suggested Treatment Considerations:

Consider evaluation of creatine metabolism and methylation capacity, including assessment of relevant nutrients such as folate and vitamin B12 where clinically indicated. Review dietary intake and consider factors influencing methylation demand. In cases of significant elevation or clinical suspicion, further investigation for inborn errors of metabolism may be warranted. Management should be guided by the overall clinical context and associated laboratory findings.

Neurotransmitter Metabolism Comment

VANILMANDELLIC ACID (VMA) ELEVATED (URINE):

Elevated urinary vanilmandellic acid suggests increased catecholamine turnover and heightened sympathetic nervous system activation.

Clinically, elevated vanilmandellic acid may be associated with anxiety, palpitations, headaches, sleep disturbance, irritability, and stress intolerance.

From an organic acid pattern perspective, elevated vanilmandellic acid commonly occurs with elevated homovanillic acid and increased glycolytic markers such as pyruvic or lactic acid, reflecting sustained stress-driven metabolic demand.

From a functional medicine perspective, elevated vanilmandellic acid should be interpreted in relation to cortisol rhythm, inflammatory burden, and stimulant exposure rather than being viewed in isolation.

KYNURENIC ACID ELEVATED (URINE):

Kynurenic acid reflects diversion of tryptophan metabolism down the kynurenine pathway, often driven by inflammation or immune activation.

Clinically, elevations may be associated with fatigue, mood changes, cognitive dysfunction, or pain syndromes. Interpretation should consider dietary intake, medications, and the broader metabolic context.

From a functional medicine perspective, management focuses on addressing inflammatory drivers, optimising vitamin B6 status, and supporting immune balance, addressing underlying contributors rather than isolated suppression of the marker.

Consider: Supplementation with Vitamin B6.

QUINOLINIC ACID ELEVATED (URINE):

Quinolinic acid is a neuroactive metabolite within the kynurenine pathway. Elevation suggests inflammatory activation and excitotoxic stress.

Clinically, elevations may be associated with anxiety, depression, cognitive changes, and pain sensitivity. Interpretation should consider dietary intake, medications, and the broader metabolic context.

From a functional medicine perspective, management focuses on reducing neuroinflammation, supporting antioxidant defences, and optimising B-vitamin status, addressing underlying contributors rather than isolated suppression of the marker.

Consider: Elimination of high tryptophan foods; supplementation with melatonin, B6, turmeric, garlic.

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QUINOLINIC ACID/5-HIAA RATIO ELEVATED:

Description:

An elevated quinolinic acid/5-HIAA ratio reflects disproportionate tryptophan shunting through the excitotoxic kynurenine-quinolinic acid pathway at the expense of serotonin synthesis, indicating neuroinflammatory-driven tryptophan catabolism.

Clinical Significance:

Elevated ratio is a significant indicator of neuroinflammation, IDO (indoleamine 2,3-dioxygenase) enzyme upregulation, and serotonin depletion with concomitant excitotoxic stress. Associated with treatment-resistant depression, suicidal ideation, chronic fatigue, cognitive impairment, and neurodegenerative conditions. IDO is activated by pro-inflammatory cytokines (IFN-γ, TNF-α, IL-6).

Suggested Treatment:

Identify and address inflammatory triggers (gut dysbiosis, chronic infections, autoimmunity, dietary). Anti-inflammatory interventions: omega-3 fatty acids, curcumin, resveratrol. Support serotonin synthesis: L-tryptophan, 5-HTP, B6, zinc, SAMe. Consider IDO-modulating strategies. Neurological and psychiatric review if symptoms are severe.

Detoxification / Oxidative Damage Comment:

PYROGLUTAMIC ACID ELEVATED (URINE):

Elevated urinary pyroglutamic acid suggests increased demand on glutathione synthesis and recycling, often reflecting oxidative stress or limited availability of glutathione precursors. Accumulation may occur when the gamma-glutamyl cycle is under increased metabolic pressure.

Clinically, elevated pyroglutamic acid may be associated with fatigue, increased sensitivity to environmental exposures, inflammatory symptoms, or impaired detoxification tolerance, although symptoms are often non-specific.

From an organic acid pattern perspective, elevated pyroglutamic acid may be observed alongside elevated oxidative stress markers such as 8-hydroxy-2-deoxyguanosine and reduced availability of sulfur-containing compounds, and may coexist with low N-acetylcysteine or altered glycine metabolism.

From a functional medicine perspective, elevated pyroglutamic acid should be interpreted in the context of oxidative burden, glutathione precursor availability, and overall redox balance rather than as an isolated abnormality.

Consider: Supplementation with glutathione or N-acetyl-cysteine.

4-HYDROXYPHENYLLACTIC ACID ELEVATED (URINE):

Elevated urinary 4-hydroxyphenyllactic acid suggests altered tyrosine metabolism, commonly driven by gut microbial activity or impaired downstream conversion pathways.

Clinically, elevated levels may be associated with fatigue, gastrointestinal disturbance, or neurocognitive symptoms, though presentations vary.

From an organic acid pattern perspective, elevation may occur alongside p-hydroxyphenylacetic acid and other aromatic metabolites, indicating increased microbial fermentation of aromatic amino acids.

From a functional medicine perspective, this finding should be interpreted in the context of gut microbial balance and overall aromatic amino acid metabolism rather than as a primary metabolic disorder.

Environmental/Xenobiotic Exposure Comment:

ENVIRONMENTAL POLLUTANTS PROFILE:

The reported markers in the Environmental Pollutants Profile commonly originate from industrial/manufacturing products or their associated byproducts. Exposures are often occupationally-related and typically through either inhalation or topical exposure.

Metabolism of these products occurs via the liver detoxification pathways leading to excretion into the urine. Chronic exposures may also lead to build up of these products in fatty tissue deposits.

QUINOLINIC ACID ELEVATED (URINE):

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Elevated urinary quinolinic acid suggests increased flux through the kynurenine pathway of tryptophan metabolism, often associated with immune activation or inflammatory signalling. Quinolinic acid is a neuroactive metabolite with excitatory properties.

Clinically, elevated quinolinic acid may be associated with neurocognitive symptoms such as brain fog, mood disturbance, irritability, sleep disruption, or heightened pain sensitivity, although symptom expression varies.

From an organic acid pattern perspective, elevated quinolinic acid is often observed alongside alterations in other tryptophan metabolites, indicating inflammatory diversion of tryptophan metabolism away from serotonin and melatonin pathways.

From a functional medicine perspective, this finding should be interpreted in the context of immune activation, inflammatory burden, and overall balance of tryptophan metabolism rather than as a primary neurological disorder.

Treatment: Treatments for high quinolinic acid focus on reducing its production and counteracting its neurotoxic effects, including dietary changes like increasing vitamin B6 and consuming antioxidants (e.g., sulforaphane, green tea polyphenols, curcumin), and supplements (melatonin, selenium, theanine).

Methodology

Liquid Chromatography-Mass Spectrometry (LC-MS/MS/MS), Gas Chromatography-MS (GC/MS), Automated Chemistry/Immunochemistry