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Ext ID 26096-0001

Test Patient

Sex: Female • 56yrs • 01-Jan-70

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

Specimen type - Urine, Spot

Collected
02-Apr-26

MICROBIAL OVERGROWTH

3,4-Dihydroxybenzoic Acid (3,4-DHBA)
3-Hydroxyphenylacetic Acid (3-HPAA)
4-Hydroxybenzoic Acid (4-HBA) (Urine, Spot)
Benzoic Acid (Urine, Spot)
Furancarbonylglycine

YEAST/FUNGAL METABOLITES

5-Hydroxymethyl-2-furoic Acid (Urine, Spot)
Arabinose

ENVIRONMENTAL EXPOSURES

3-Methylhippuric Acid
4-Hydroxybenzoic Acid (4-HBA) (Urine, Spot)
5-Methylcytosine
Benzoic Acid (Urine, Spot)
Quinolinic Acid (Urine, Spot)
t,t-Muconic Acid

MITOCHONDRIA/ENERGY

2-Oxoglutaric Acid
Isocitric Acid
Mevalonolactone

FATTY ACID / KETONE METABOLISM

Adipic Acid

CARBOHYDRATE / GLYCAEMIC METABOLISM

N/A

NEUROTRANSMITTER METABOLISM

Kynurenic Acid (Urine, Spot)
Quinolinic Acid (Urine, Spot)
Quinolinic Acid/5-HIAA Ratio
Xanthurenic Acid (Urine, Spot)

AMINO ACIDS METABOLISM

2-Oxoisocaproic Acid
Guanidinoacetic Acid
N-Acetylphenylalanine
Phenylpyruvic Acid

VITAMINS / NUTRITIONAL MARKERS

Kynurenic Acid (Urine, Spot)
Quinolinic Acid (Urine, Spot)
Xanthurenic Acid (Urine, Spot)

DETOXIFICATION / GLUTATHIONE FUNCTION

5-Hydroxymethyl-2-furoic Acid (Urine, Spot)

OXIDATIVE DAMAGE / INFLAMMATION

8-hydroxy-deoxyguanosine
Leukotriene E4
Quinolinic Acid (Urine, Spot)

OXALATE METABOLISM

N/A

Lab Director: Jillian Harrington, PhD, HCLD (ABB). CLIA#99D2282774

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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 (Urine, Spot)	25.00	H	(<9.30)	mmol/molCR
2	Hippuric Acid (Urine, Spot)	330.0		(<603.0)	mmol/molCR
3	4-Hydroxybenzoic Acid (4-HBA) (Urine, Spot)	0.64	H	(<0.57)	mmol/molCR
4	3,4-Dihydroxybenzoic Acid (3,4-DHBA)	62.00	H	(<45.00)	mmol/molCR
5	3-Hydroxyphenylacetic Acid (3-HPAA)	12.00	H	(<10.00)	mmol/molCR
6	3,4-Dihydroxyphenylpropionic Acid (DHPPA)	2.90		(<5.30)	mmol/molCR
7	Furancarbonylglycine	2.90	H	(<2.00)	mmol/molCR
Amino Acid Metabolism					
8	Phenylacetic Acid (PAA)	2.50		(<3.90)	mmol/molCR
9	Phenylpropionic Acid	0.08		(<0.10)	mmol/molCR
10	3-Phenyllactic Acid (3-PLA)	1.50		(<2.00)	mmol/molCR
11	2-Hydroxyphenylacetic Acid (2-HPAA)	0.60		(0.05-0.70)	mmol/molCR
12	4-Hydroxyphenyllactic Acid (4-HPLA) (Urine, Spot)	3.50		(<3.90)	mmol/molCR
SCFA Metabolism					
13	3-Hydroxypropionic Acid (3-HPA)	6.60		(<17.00)	mmol/molCR
Clostridial Markers					
14	4-Cresol	0.90		(<3.00)	ug/mgCR
15	4-Hydroxyphenylacetic Acid (4-HPAA)	10.20		(<14.60)	mmol/molCR
16	Indoleacetic Acid (IAA)	8.70		(<11.00)	mmol/molCR
17	3-(3-Hydroxyphenyl)-3-hydroxypropionic Acid (HPHPA)	59.00		(<120.00)	mmol/molCR

YEASTS & FUNGAL METABOLITES

TEST		RESULT	H/L	REFERENCE	UNITS
18	Arabinitol	28.0		(<36.0)	mmol/molCR
19	Arabinose	42.00	H	(<32.00)	mmol/molCR
20	Citramalic Acid	2.20		(<3.60)	mmol/molCR
21	3-Oxoglutaric Acid	0.38		(<0.50)	mmol/molCR
Aspergillus Markers					
22	5-Hydroxymethyl-2-furoic Acid (Urine, Spot)	18.50	H	(<10.00)	mmol/molCR
23	Furan-2,5-dicarboxylic Acid (Urine, Spot)	12.90		(<15.00)	mmol/molCR
24	Tartaric Acid	11.60		(<15.00)	mmol/molCR
Fusarium					

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TEST		RESULT	H/L	REFERENCE	UNITS
25	Tricarballylic Acid	**0.21**		(<0.44)	mmol/molCR

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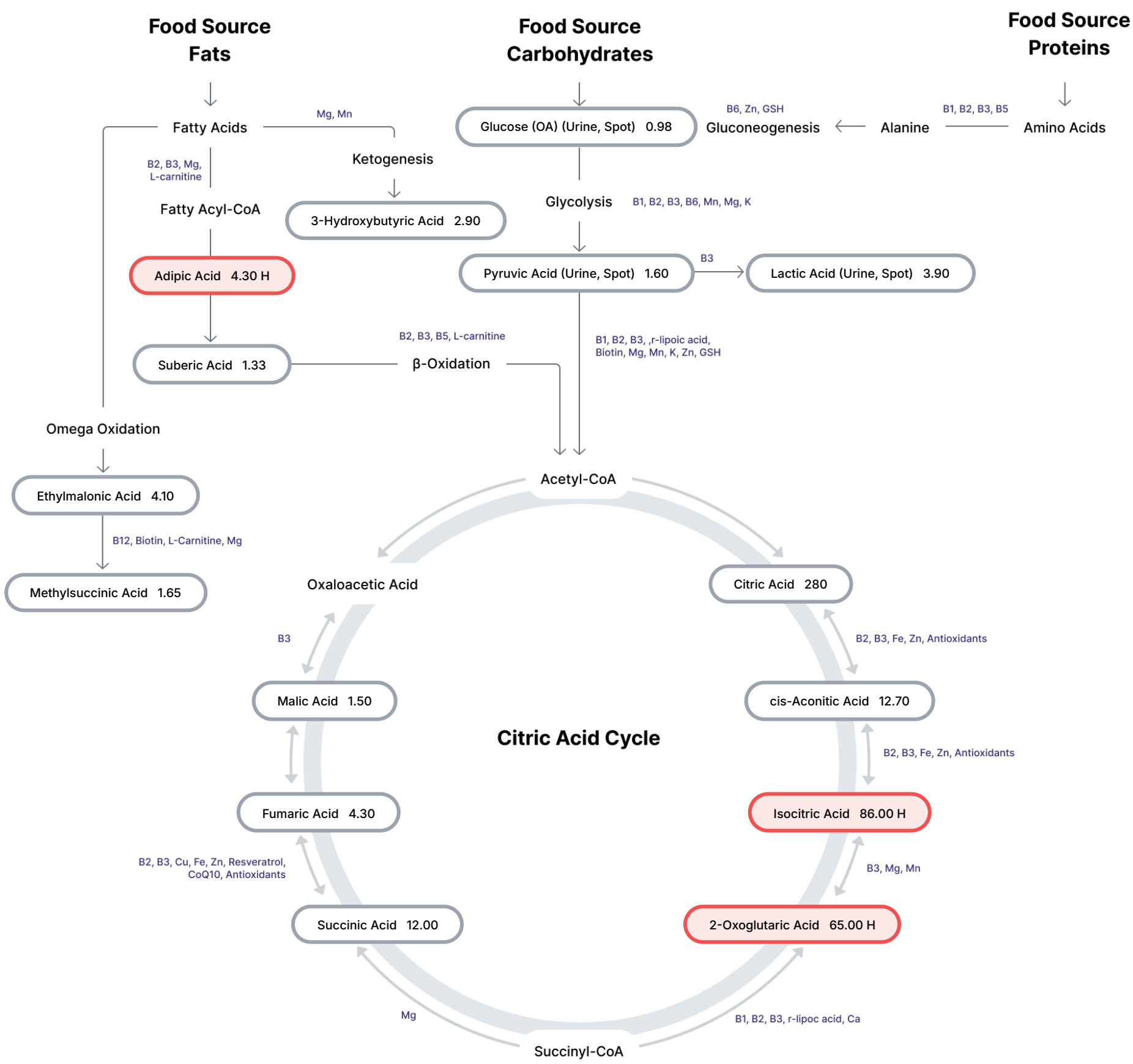
Organic Acids Pathway



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MITOCHONDRIAL & ENERGY METABOLISM

(B Comp, CoQ10, Amino Acids, Mg)

TEST		RESULT	H/L	REFERENCE	UNITS
26	Glucose (OA) (Urine, Spot)	0.98		(<1.10)	ug/mgCR
27	Pyruvic Acid (Urine, Spot)	1.60		(0.50-8.70)	mmol/molCR
28	Lactic Acid (Urine, Spot)	3.90		(<48.00)	mmol/molCR
Citric Acid Cycle					
29	Citric Acid	280		(40-507)	mmol/molCR
30	cis-Aconitic Acid	12.70		(3.50-36.00)	mmol/molCR
31	Isocitric Acid	86.00	H	(5.00-65.00)	mmol/molCR
32	2-Oxoglutaric Acid	65.00	H	(2.00-45.00)	mmol/molCR
33	Succinic Acid	12.00		(1.00-15.00)	mmol/molCR
34	Fumaric Acid	4.30		(<8.60)	mmol/molCR
35	Malic Acid	1.50		(<1.80)	mmol/molCR
Ketone/Energy Intermediates					
36	Acetoacetic Acid	8.80		(<10.00)	mmol/molCR
37	3-Hydroxybutyric Acid	2.90		(<3.10)	mmol/molCR
38	2-Hydroxybutyric Acid (Urine, Spot)	4.10		(<6.90)	mmol/molCR
Mitochondrial Markers					
39	Methylsuccinic Acid	1.65		(<10.80)	mmol/molCR
40	2-Methylglutaric Acid	0.45		(<0.76)	mmol/molCR
41	3-Methylglutaric Acid (Urine, Spot)	2.90		(<8.50)	mmol/molCR
42	2-Hydroxyglutaric Acid	12.90		(<15.00)	mmol/molCR
43	3-Hydroxyglutaric Acid	4.20		(<5.50)	mmol/molCR
44	Malonic Acid (Urine, Spot)	5.90		(<9.70)	mmol/molCR
45	Mevalonolactone	2.80	H	(<2.00)	mmol/molCR
46	2,4-Dihydroxybutanoic Acid	5.80		(<10.00)	mmol/molCR
47	N-Acetylaspartic Acid	10.40		(<15.00)	mmol/molCR
48	3-Methylglutaconic Acid	6.32	H	(<5.50)	mmol/molCR

CARBOHYDRATE & GLYCAEMIC METABOLISM

(B1, B3, Cr, Lipoic Acid, CoQ10)

TEST		RESULT	H/L	REFERENCE	UNITS
49	Glucose (OA) (Urine, Spot)	0.98		(<1.10)	ug/mgCR
50	Pyruvic Acid (Urine, Spot)	1.60		(0.50-8.70)	mmol/molCR
51	Lactic Acid (Urine, Spot)	3.90		(<48.00)	mmol/molCR
52	2-Hydroxybutyric Acid (Urine, Spot)	4.10		(<6.90)	mmol/molCR

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FATTY ACID OXIDATION & KETONE METABOLISM

	TEST	RESULT	H/L	REFERENCE	UNITS
53	Adipic Acid	4.30	H	(<3.80)	mmol/molCR
54	Pimelic Acid	1.80		(<4.00)	mmol/molCR
55	Suberic Acid	1.33		(<2.20)	mmol/molCR
56	Azelaic Acid	5.90		(<10.00)	mmol/molCR
57	Sebacic Acid	0.19		(<0.24)	mmol/molCR
58	Ethylmalonic Acid	4.10		(<5.80)	mmol/molCR
59	Propionylglycine	1.50		(<2.00)	mmol/molCR
60	N-Butyrylglycine	1.89		(<3.00)	mmol/molCR
61	Isovalerylglycine	3.80		(<4.50)	mmol/molCR
62	N-(2-Methylbutyryl)glycine	1.66		(<2.00)	mmol/molCR
63	3-Methylcrotonylglycine	1.80		(<10.00)	mmol/molCR
64	Tiglylglycine	3.88		(<10.00)	mmol/molCR
65	Hexanoylglycine	4.10		(<10.00)	mmol/molCR
66	Suberylglycine	1.58		(<3.00)	mmol/molCR

VITAMIN & NUTRITIONAL COFACTOR MARKERS

	TEST	RESULT	H/L	REFERENCE	UNITS
	B-Vitamin Functional Markers				
67	Methylmalonic Acid (MMA)	1.65		(<1.90)	mmol/molCR
68	Formiminoglutamic Acid (FIGLU)	0.98		(<1.50)	mmol/molCR
69	Xanthurenic Acid (Urine, Spot)	1.92	H	(<0.96)	mmol/molCR
70	Kynurenic Acid (Urine, Spot)	3.10	H	(<2.20)	mmol/molCR
71	Quinolinic Acid (Urine, Spot)	11.80	H	(<9.10)	mmol/molCR
72	Picolinic Acid (Urine, Spot)	4.20		(<10.28)	mmol/molCR
73	3-Hydroxyisovaleric Acid	20.40		(<29.00)	mmol/molCR
	Glutathione/Antioxidant Support				
74	Pyroglutamic Acid (Urine, Spot)	28.80		(4.50-33.00)	mmol/molCR
75	N-Acetylcysteine (NAC) (Urine, Spot)	0.05		(0.02-0.28)	mmol/molCR
	Vitamin-specific Markers				
76	Glutaric Acid (Vit B2)	0.28		(0.02-0.36)	mmol/molCR
77	Pantothenic Acid (Vit B5)	3.30		(0.10-10.00)	mmol/molCR
78	Pyridoxic Acid (Vit B6)	25.90		(0.68-34.00)	mmol/molCR
79	Ascorbic Acid (Vit C) (Urine, Spot)	59.00		(0.50-200.00)	mmol/molCR
80	Methylcitric Acid (Biotin/Vitamin H)	4.40		(0.10-15.00)	mmol/molCR

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TEST		RESULT	H/L	REFERENCE	UNITS
81	3-Hydroxy-3-methylglutaric Acid (CoQ10)	3.50		(0.10-5.00)	mmol/molCR

PYRIMIDINE METABOLITES - Folate Metabolism

TEST		RESULT	H/L	REFERENCE	UNITS
82	Thymine (Urine, Spot)	0.42		(<0.60)	mmol/molCR
83	Uracil (Urine, Spot)	8.10		(<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					
84	2-Oxoisovaleric Acid	1.95		(<4.10)	mmol/molCR
85	2-Oxoisocaproic Acid	0.88	H	(<0.65)	mmol/molCR
86	3-Methyl-2-oxovaleric Acid	1.74		(<2.00)	mmol/molCR
87	3-Methylglutaric Acid (Urine, Spot)	2.90		(<8.50)	mmol/molCR
88	Succinylacetone	0.41		(<0.50)	mmol/molCR
Downstream Metabolites					
89	2-Hydroxyisovaleric Acid	3.00		(<4.10)	mmol/molCR
90	2-Hydroxyisocaproic Acid	1.21		(<1.50)	mmol/molCR
91	2-Oxobutyric Acid	2.95		(<7.00)	mmol/molCR
92	2-Oxo-4-methiolbutyric Acid (KMBA)	3.20	H	(<1.50)	mmol/molCR
Amino Acid Metabolism					
93	Phenylpyruvic Acid	4.50	H	(<2.00)	mmol/molCR
94	Homogentisic Acid	1.30	H	(<1.00)	mmol/molCR
95	N-Acetylphenylalanine	6.60	H	(<5.00)	mmol/molCR
96	Mandelic Acid (Urine, Spot)	188.0		(<340.0)	ug/gCR
97	Malonic Acid (Urine, Spot)	5.90		(<9.70)	mmol/molCR
98	4-Hydroxyphenyllactic Acid (4-HPLA) (Urine, Spot)	3.50		(<3.90)	mmol/molCR
99	2-Oxoadipic Acid	1.60		(<2.00)	mmol/molCR
100	Guanidinoacetic Acid	4.80	H	(0.50-3.00)	mmol/molCR
101	Guanidinobutyric Acid	0.88		(0.10-1.00)	mmol/molCR
102	3-Methylglutaric Acid (Urine, Spot)	2.90		(<8.50)	mmol/molCR
103	N-Acetylglycine	1.69		(<5.00)	mmol/molCR

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NEUROTRANSMITTER METABOLITES

(Phenylalanine, Tyrosine)

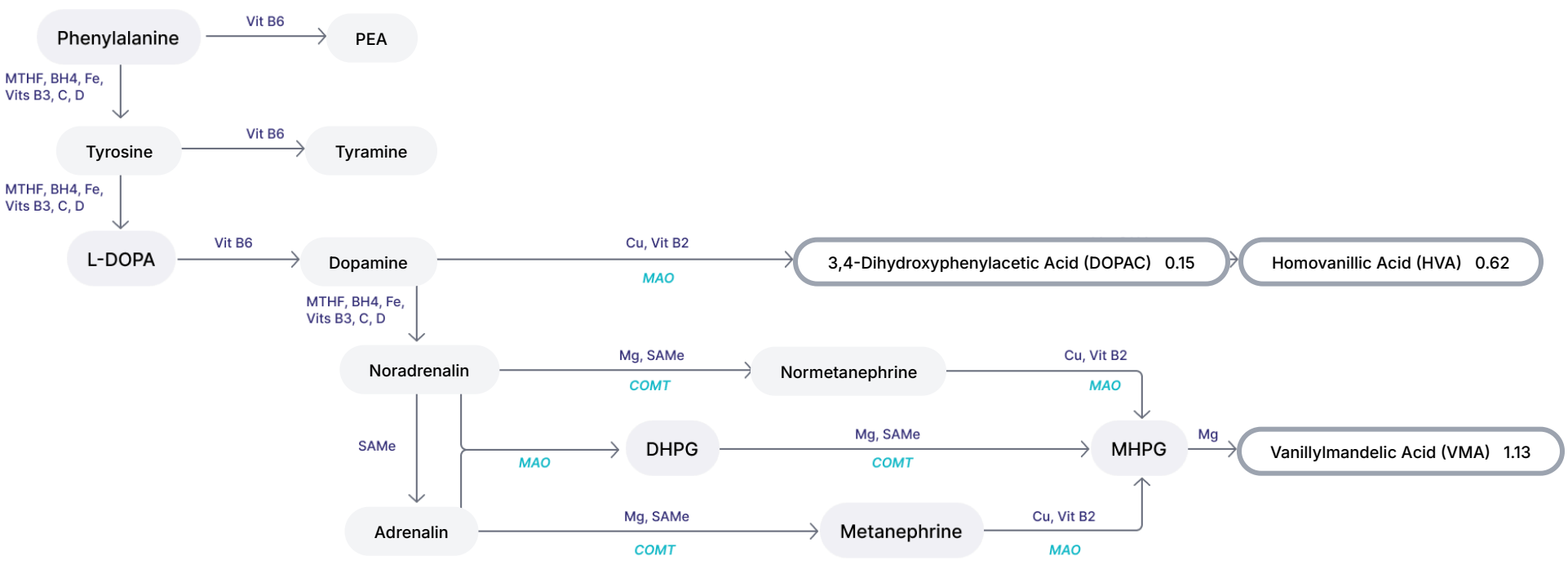
TEST	RESULT	H/L	REFERENCE	UNITS
Inhibitory (Serotonin)				
104 5-Hydroxyindoleacetic Acid (5-HIAA)	1.14		(<4.30)	mmol/molCR
Excitatory (Dopamine)				
105 3,4-Dihydroxyphenylacetic Acid (DOPAC)	0.15		(0.08-3.50)	mmol/molCR
106 3-Methyl-4-hydroxyphenylglycol (MHPG)	0.18		(0.05-0.50)	mmol/molCR
107 Homovanillic Acid (HVA)	0.62		(0.10-5.30)	mmol/molCR
108 Vanillylmandelic Acid (VMA)	1.13		(0.40-3.60)	mmol/molCR
109 HVA/DOPAC Ratio	4.1		(<10.0)	ratio
110 HVA/VMA Ratio	0.5		(<2.0)	ratio

ADRENAL STRESS (Overnight)

SERVICE	RESULT	H/L	REFERENCE	UNITS
111 Cortisol (OA)	25.3		(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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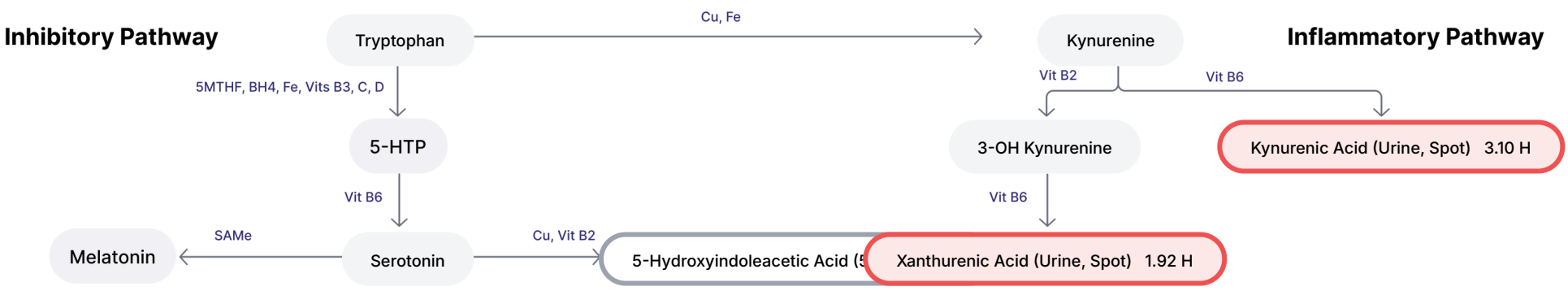
TRYPTOPHAN/KYNURENINE PATHWAY

(Tryptophan, B6, Antioxidants)

TEST		RESULT	H/L	REFERENCE	UNITS
Inhibitory					
112	5-Hydroxyindoleacetic Acid (5-HIAA)	1.14		(<4.30)	mmol/molCR
Inflammatory					
113	Kynurenic Acid (Urine, Spot)	3.10	H	(<2.20)	mmol/molCR
114	Quinolinic Acid (Urine, Spot)	11.80	H	(<9.10)	mmol/molCR
115	Picolinic Acid (Urine, Spot)	4.20		(<10.28)	mmol/molCR
116	Xanthurenic Acid (Urine, Spot)	1.92	H	(<0.96)	mmol/molCR
117	Kynurenic/Quinolinic Ratio	0.3		(<2.0)	ratio
118	Quinolinic Acid/5-HIAA Ratio	10.4	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				
119 Orotic Acid	1.95		(<3.20)	mmol/molCR
Glutathione Metabolism				
120 Pyroglutamic Acid (Urine, Spot)	28.80		(4.50-33.00)	mmol/molCR
121 N-Acetylcysteine (NAC) (Urine, Spot)	0.05		(0.02-0.28)	mmol/molCR
122 Glucaric Acid	8.40		(<11.00)	mmol/molCR
Phase I / Xenobiotic Markers				
123 2-Hydroxyhippuric Acid	0.80		(<1.50)	mmol/molCR
124 5-Hydroxymethyl-2-furoic Acid (Urine, Spot)	18.50	H	(<10.00)	mmol/molCR
125 Furan-2,5-dicarboxylic Acid (Urine, Spot)	12.90		(<15.00)	mmol/molCR

OXIDATIVE STRESS & INFLAMMATION

(Vitamin C, Other Antioxidants)

TEST	RESULT	H/L	REFERENCE	UNITS
126 8-hydroxy-deoxyguanosine	3.80	H	(<2.70)	umol/molCR
127 Leukotriene E4	195.00	H	(<100.00)	pg/mgCR
128 Quinolinic Acid (Urine, Spot)	11.80	H	(<9.10)	mmol/molCR
129 Pyroglutamic Acid (Urine, Spot)	28.80		(4.50-33.00)	mmol/molCR
130 Ascorbic Acid (Vit C) (Urine, Spot)	59.00		(0.50-200.00)	mmol/molCR
131 Quercetin	0.68		(0.01-2.00)	mmol/molCR

OXALATE METABOLISM

TEST	RESULT	H/L	REFERENCE	UNITS
132 Glycolic Acid	20.80		(<67.00)	mmol/molCR
133 Glyceric Acid	3.80		(<6.00)	mmol/molCR
134 Oxalic Acid	42.00		(<78.00)	mmol/molCR

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ENVIRONMENTAL / XENOBIOTIC EXPOSURE

TEST	RESULT	H/L	REFERENCE	UNITS
Toluene Exposure				
135 Hippuric Acid (Urine, Spot)	330.0		(<603.0)	mmol/molCR
136 Benzoic Acid (Urine, Spot)	25.00	H	(<9.30)	mmol/molCR
Paraben Exposure				
137 4-Hydroxybenzoic Acid (4-HBA) (Urine, Spot)	0.64	H	(<0.57)	mmol/molCR
Styrene Exposure				
138 Mandelic Acid (Urine, Spot)	188.0		(<340.0)	ug/gCR
139 Phenylglyoxylic Acid	106.0		(<300.0)	ug/gCR
140 Mandelic Acid + Phenylglyoxylic Acid	294.0		(<610.0)	ug/gCR
Benzene Exposure				
141 t,t-Muconic Acid	0.20	H	(<0.12)	mmol/molCR
Phthalate Exposure				
142 Phthalic Acid	149.00		(<170.00)	ug/gCR
143 Monoethyl Phthalate	60.30		(<100.00)	ug/gCR
144 Quinolinic Acid (Urine, Spot)	11.80	H	(<9.10)	mmol/molCR
METB Exposure				
145 2-Hydroxylsobutyric Acid	3.90		(<6.90)	mmol/molCR
Xylene Exposure				
146 2-Methylhippuric Acid	0.01		(<0.04)	mmol/molCR
147 3-Methylhippuric Acid	0.34	H	(<0.11)	mmol/molCR
148 4-Methylhippuric Acid	1.69		(<1.80)	mmol/molCR
Trimethylbenzene Exposure				
149 3,4-Dimethylhippuric Acid	0.00		(<0.01)	mmol/molCR
150 4-Hydroxyhippuric Acid	6.23		(<16.50)	mmol/molCR
Nucleotide Turnover/Methylation				
151 5-Methylcytosine	68.00	H	(10.00-50.00)	mmol/molCR
152 Uracil (Urine, Spot)	8.10		(<9.00)	mmol/molCR
153 Thymine (Urine, Spot)	0.42		(<0.60)	mmol/molCR

TEST	RESULT	H/L	REFERENCE	UNITS
154 Creatinine, Urine	8.50		(2.47-19.20)	mmol/L

Lab Director: Jillian Harrington, PhD, HCLD (ABB). CLIA#99D2282774

*** US BioTek** US BioTek. 16020 Linden Av N, Shoreline WA 98133

Lab ID
Patient ID P000065
Ext ID 26096-0001

Test Patient

Sex: Female • 56yrs • 01-Jan-70

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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 (Urine, Spot)	Adequate	0.0	mg
Glutathione	Adequate	0.0	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

BENZOIC ACID ELEVATED (URINE):

Elevated urinary benzoic acid suggests increased exposure to benzoate-containing foods, preservatives, or altered gut microbial metabolism. Benzoic acid is detoxified primarily through glycine conjugation to form hippuric acid.

Clinically, elevated benzoic acid may be associated with headaches, fatigue, gastrointestinal discomfort, or chemical sensitivity, although symptoms are often non-specific.

From an organic acid pattern perspective, elevated benzoic acid may be observed alongside elevated hippuric acid, indicating increased benzoate load or increased demand on glycine-dependent conjugation pathways.

From a functional medicine perspective, elevated benzoic acid should be interpreted in the context of dietary intake, glycine availability, gut microbial activity, and overall detoxification capacity rather than as an isolated finding.

Consider: treatment for dysbiosis and diet changes, mucosal support, pre and probiotics

4-HYDROXYBENZOIC ACID ELEVATED (URINE):

Elevated urinary 4-hydroxybenzoic acid suggests increased exposure to parabens or altered metabolism of aromatic compounds. It may also arise from gut microbial metabolism of dietary polyphenols.

Clinically, elevated levels may be associated with non-specific symptoms such as headaches, fatigue, or chemical sensitivity.

From an organic acid pattern perspective, elevated 4-hydroxybenzoic acid may be observed alongside other aromatic metabolites, reflecting increased aromatic compound load or altered gut microbial activity.

From a functional medicine perspective, this finding should be interpreted in the context of environmental exposure, dietary polyphenol intake, and gut microbiome balance.

Consider: Treatment for dysbiosis and diet changes, mucosal support, pre and probiotics.

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.

3,4-DIHYDROXYBENZOIC ACID ELEVATED:

Description:

Elevated 3,4-dihydroxybenzoic acid reflects increased catecholamine metabolism, enhanced polyphenol biotransformation by gut microbiota, or oxidative degradation of dopamine.

Clinical Significance:

Persistent elevation may indicate excessive catecholamine turnover, increased oxidative stress, or heightened gut microbial phenolic metabolism. Associated with inflammatory states and dysbiosis patterns.

Suggested Treatment:

Evaluate catecholamine pathway function and oxidative stress markers. Support antioxidant status with N-acetylcysteine, glutathione precursors, and vitamin C. Address gut dysbiosis if implicated.

3-HYDROXYPHENYLACETIC ACID ELEVATED:

Description:

Elevated 3-hydroxyphenylacetic acid reflects overgrowth of Clostridia or other aromatic amino acid-fermenting bacteria, producing excessive phenolic metabolites in the gut lumen.

Clinical Significance:

Marked elevation is a recognised biomarker of intestinal bacterial overgrowth (particularly Clostridial dysbiosis) and increased gut permeability. Associated with neurological symptoms, fatigue, and cognitive impairment due to systemic absorption of phenolic compounds.

Suggested Treatment:

Consider targeted antimicrobial or herbal antimicrobial therapy (e.g., oregano oil, berberine). Implement gut restoration protocol including dietary modification, probiotics, and intestinal barrier support (L-glutamine, zinc carnosine). Retest after intervention.

Yeast Dysbiosis Comment

ARABINOSE ELEVATED:

Arabinose is a pentose sugar that may be detected in urine as a product of dietary intake and gastrointestinal microbial metabolism. It is not a primary endogenous human metabolite and is typically present at low concentrations.

Elevated urinary arabinose may be associated with increased gastrointestinal microbial production of arabinose, which has been described in the context of altered gut microbial activity, including possible overrepresentation of certain yeast or bacterial species. However, this finding is non-specific and may also be influenced by dietary sources and intestinal metabolism.

As such, arabinose should not be used in isolation as a diagnostic marker of a specific organism, and is best interpreted alongside other markers of microbial metabolism and gastrointestinal function.

Suggested Treatment Considerations:

Consider correlation with other markers of gastrointestinal microbial activity and clinical features suggestive of altered gut function. Review dietary intake and factors that may influence microbial balance. Further evaluation of gastrointestinal health may be appropriate where clinically indicated. Management should be guided by the overall clinical context and associated laboratory findings rather than the isolated elevation of this marker.

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

ISOCITRIC ACID ELEVATED (URINE):

Elevated urinary isocitric acid suggests altered downstream TCA cycle efficiency or compensatory metabolic flux.

Clinically, symptoms are non-specific.

From an organic acid pattern perspective, interpretation alongside alpha-ketoglutaric acid assists in determining whether downstream enzymatic congestion is present.

From a functional medicine perspective, this finding should be interpreted as part of an overall mitochondrial pattern.

A high level is suggestive of inhibition to the enzyme by Aluminum.

Supplementation Recommendations: Cofactors needed to increase the breakdown of isocitrate to alpha-ketoglutarate are: Vit B3, (NAD), Mg, Mn.

2-OXOGLUTARIC ACID ELEVATED (URINE)

Elevated urinary alpha-ketoglutaric acid suggests a bottleneck within the TCA cycle and altered nitrogen handling.

Clinically, elevated alpha-ketoglutaric acid may be associated with fatigue, cognitive symptoms, or reduced exercise tolerance.

From an organic acid pattern perspective, elevation often occurs alongside elevated succinic acid, reflecting downstream congestion and potential oxidative stress.

From a functional medicine perspective, this finding should be interpreted in the context of mitochondrial efficiency, redox balance, and amino-acid metabolism.

Elevations can be seen with nutrient cofactor deficiencies needed for the enzymatic conversion of α ketoglutarate such as vitamin B3, zinc, magnesium, manganese.

MEVALONOLACTONE ELEVATED:

Mevalonolactone is the lactone form of mevalonic acid, an intermediate in the mevalonate pathway, which is essential for the synthesis of cholesterol, isoprenoids, and other biologically important molecules. This pathway is regulated by HMG-CoA reductase, the rate-limiting enzyme in cholesterol biosynthesis.

Elevated urinary mevalonolactone may reflect increased activity of the mevalonate pathway, potentially indicating upregulated cholesterol and isoprenoid synthesis. This pattern may be observed in the context of metabolic dysregulation, increased anabolic demand, or altered lipid metabolism. In some cases, elevations may also be associated with inflammatory states, as the mevalonate pathway is involved in the production of intermediates important for immune cell function.

While increased levels may provide insight into pathway activity, this finding is non-specific and should be interpreted in conjunction with lipid markers, inflammatory indicators, and overall metabolic context.

Suggested Treatment Considerations:

Consider correlation with lipid profile and markers of metabolic and inflammatory status. Review dietary and metabolic factors influencing cholesterol synthesis where clinically indicated. Further evaluation may be warranted in cases of persistent or significant elevation. Management should be guided by the overall clinical context and associated laboratory findings.

3-METHYLGLUTACONIC ACID ELEVATED:

3-Methylglutaconic acid (3-MGC) is a dicarboxylic acid intermediate in the leucine catabolism pathway. Under normal conditions, 3-methylglutaconyl-CoA is hydrated by the enzyme 3-methylglutaconyl-CoA hydratase to form HMG-CoA, which enters ketogenesis. When this step is impaired — due to genetic enzyme deficiency or acquired mitochondrial dysfunction — 3-MGC accumulates and is excreted in urine. Beyond leucine metabolism, 3-MGC has been established as a broader non-specific marker of mitochondrial inner membrane dysfunction, elevated across a range of mitochondrial disorders including Barth syndrome (TAZ gene mutation), DNAJC19-related cardiomyopathy, and SERAC1-related MEGDEL syndrome. In the functional medicine context, mild to moderate elevation most commonly reflects acquired mitochondrial stress and nutritional cofactor insufficiency (CoQ10, B2, carnitine, magnesium) rather than primary genetic deficiency.

Clinical Significance:

Elevated urinary 3-MGC is a meaningful indicator of mitochondrial dysfunction and warrants assessment of the broader mitochondrial marker pattern. Isolated mild elevation in a clinically well adult most likely reflects functional mitochondrial stress, cofactor depletion, or elevated oxidative burden rather than a primary genetic disorder. Elevation is most significant when found alongside globally reduced TCA cycle intermediates (low citric acid, isocitric acid, 2-oxoglutaric acid), indicating reduced mitochondrial throughput rather than an isolated

enzyme defect. Clinically, elevation is associated with chronic fatigue and post-exertional malaise, exercise intolerance, reduced stamina, cognitive impairment, and in more severe presentations — cardiomyopathy and skeletal myopathy. Barth syndrome should be considered in males with elevated 3-MGC and cardiomyopathy. Marked elevation (greater than 10× the upper reference limit) accompanied by neurological symptoms, cardiomyopathy, myopathy, or significant neurodevelopmental concerns warrants referral for metabolic genetics evaluation to exclude primary mitochondrial disease.

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.

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.

B-Vitamins/Amino Acids Comment

2-OXOISOCAPROIC ACID ELEVATED (URINE):

Elevated urinary alpha-ketoisocaproic acid suggests impaired leucine catabolism and mitochondrial inefficiency.

Clinically, this may be associated with fatigue, muscle weakness, and reduced exercise tolerance.

From an organic acid pattern perspective, elevation commonly occurs with 3-methylglutaric acid and other BCAA ketoacids, reflecting congestion within BCAA metabolic pathways.

From a functional medicine perspective, this finding should be interpreted in the context of overall mitochondrial function and nutrient sufficiency rather than as an isolated abnormality.

Consider supplementation with B1, B2, B3, B5, and lipoate to support enzyme function.

XANTHURENIC ACID ELEVATED (URINE):

Elevated urinary xanthurenic acid suggests altered tryptophan metabolism, most commonly reflecting reduced vitamin B6-dependent enzymatic activity.

Clinically, elevated xanthurenic acid may be associated with fatigue, mood disturbance, and impaired stress tolerance.

From an organic acid pattern perspective, elevations often occur alongside kynurenic and quinolinic acid, indicating inflammatory diversion of tryptophan metabolism.

From a functional medicine perspective, this finding should be interpreted in the context of vitamin B6 status and inflammatory burden.

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.

N-ACETYLPHENYLALANINE ELEVATED:

N-acetylphenylalanine is an acetylated derivative of the amino acid phenylalanine, formed through N-acetylation pathways as part of amino acid metabolism and phase II detoxification processes. It represents a minor urinary metabolite reflecting aromatic amino acid metabolism and conjugation activity.

Elevated urinary N-acetylphenylalanine may reflect increased flux through phenylalanine metabolism and/or enhanced N-acetylation activity, which may occur in the context of increased substrate availability, altered amino acid metabolism, or metabolic demand. In addition, elevations may be influenced by gastrointestinal microbial metabolism of aromatic compounds, contributing to altered phenylalanine handling.

This marker is non-specific and is best interpreted alongside other metabolites within the phenylalanine/tyrosine pathway and markers of microbial activity to assess potential patterns of metabolic or gastrointestinal involvement.

Suggested Treatment Considerations:

Consider correlation with other markers of aromatic amino acid metabolism and gastrointestinal function. Review dietary protein intake and assess for factors influencing microbial metabolism where clinically indicated. Further evaluation should be guided by the overall clinical context and associated laboratory findings rather than the isolated marker elevation.

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

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.

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:

8-HYDROXY-2-DEOXYGUANOSINE ELEVATED (URINE):

8-OHdG is a marker of oxidative DNA damage. Elevation reflects increased oxidative stress and impaired antioxidant defence.

Clinically, elevations may be associated with fatigue, accelerated ageing features, inflammatory symptoms. Interpretation should consider dietary intake, medications, and the broader metabolic context.

From a functional medicine perspective, management focuses on reducing oxidative load, enhancing antioxidant capacity, and addressing environmental or metabolic stressors, addressing underlying contributors rather than isolated suppression of the marker.

Consider: Supplementation with antioxidants such as vitamin C, E, N-acetyl cysteine, lipoate.

LEUKOTRIENE E4 ELEVATED:

Leukotriene E4 (LTE4) is a stable urinary metabolite of the cysteinyl leukotrienes (LTC4, LTD4, and LTE4), which are lipid mediators derived from arachidonic acid via the 5-lipoxygenase pathway. These mediators play a key role in inflammatory and immune responses, particularly in MCAS, bronchoconstriction, vascular permeability, and recruitment of inflammatory cells.

Elevated urinary LTE4 reflects increased systemic leukotriene production and is considered a non-invasive marker of inflammatory pathway activation, particularly within allergic and respiratory conditions. Increased levels may be observed in association with asthma, allergic disease, mast cell activation, and other inflammatory states, reflecting upregulation of leukotriene-mediated signalling.

As LTE4 represents a downstream metabolite, it provides an indication of overall leukotriene pathway activity rather than a specific site of inflammation, and should be interpreted in conjunction with clinical findings and other markers of inflammatory or immune activity.

Suggested Treatment Considerations:

Consider correlation with clinical features of inflammatory or allergic disease, including respiratory symptoms or hypersensitivity patterns. Evaluation of factors contributing to inflammatory activation, including environmental exposures and immune triggers, may be appropriate. Management should be directed toward the underlying inflammatory process, with further investigation guided by the overall clinical context and associated laboratory findings.

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.

3-METHYLHIPPURIC ACID ELEVATED (URINE):

Elevated urinary 3-methylhippuric acid suggests increased exposure to xylene, as this metabolite represents glycine conjugation of xylene-derived methylbenzoic acids. Urinary methylhippuric acids are well-established biomarkers of xylene exposure.

Clinically, elevated 3-methylhippuric acid may be associated with headaches, dizziness, fatigue, mucosal irritation, or central nervous system symptoms, depending on exposure magnitude and duration.

From an organic acid pattern perspective, elevated 3-methylhippuric acid may be observed alongside increased hippuric acid or other aromatic conjugates, reflecting increased aromatic solvent burden and glycine-dependent detoxification demand.

From a functional medicine perspective, this finding should be interpreted in the context of occupational, environmental, or household solvent exposure (e.g. paints, fuels, adhesives), alongside assessment of hepatic conjugation capacity and glycine availability, with emphasis on exposure identification and reduction.

Treatment: Treatment options include limiting exposure to xylenes and supportive supplements such as glycine and N-acetyl cysteine can support natural detoxification.

BENZOIC ACID ELEVATED (URINE):

Elevated urinary benzoic acid suggests increased exposure to benzoate-containing foods, preservatives, or altered gut microbial metabolism. Benzoic acid is detoxified primarily through glycine conjugation to form hippuric acid.

Clinically, elevated benzoic acid may be associated with headaches, fatigue, gastrointestinal discomfort, or chemical sensitivity, although symptoms are often non-specific.

From an organic acid pattern perspective, elevated benzoic acid may be observed alongside elevated hippuric acid, indicating increased benzoate load or increased demand on glycine-dependent conjugation pathways.

From a functional medicine perspective, elevated benzoic acid should be interpreted in the context of dietary intake, glycine availability, gut microbial activity, and overall detoxification capacity rather than as an isolated finding.

Treatment: Limiting exposure to toluene. Supportive supplements such as glycine and N-acetyl cysteine can support natural detoxification.

t,t-MUCONIC ACID ELEVATED (URINE):

Elevated urinary t,t-muconic acid suggests increased exposure to benzene, as this metabolite represents a recognised biomarker of benzene metabolism and detoxification.

Clinically, elevated t,t-muconic acid may be associated with fatigue, headaches, dizziness, or non-specific neurological symptoms, although clinical effects are highly dependent on exposure magnitude and duration.

From an organic acid pattern perspective, elevated t,t-muconic acid may be observed alongside other aromatic or solvent-related metabolites, reflecting increased aromatic hydrocarbon burden and hepatic detoxification demand.

From a functional medicine perspective, this finding should be interpreted in the context of environmental and occupational exposure sources (e.g. fuel vapours, tobacco smoke, solvents), indoor air quality, and overall detoxification capacity, with primary emphasis on exposure identification and reduction.

Treatment: Treatment usually involves removing/limiting exposure; and therefore avoiding chronic exposure which can lead to severe consequences.

QUINOLINIC ACID ELEVATED (URINE):

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).

4-HYDROXYBENZOIC ACID ELEVATED (URINE):

Elevated urinary 4-hydroxybenzoic acid suggests increased exposure to parabens or altered metabolism of aromatic compounds. Parabens are commonly used as preservatives in cosmetics, personal care products, and some pharmaceuticals.

Clinically, elevated 4-hydroxybenzoic acid may be associated with non-specific symptoms such as fatigue, headaches, or endocrine-related concerns, although many individuals remain asymptomatic.

From an organic acid pattern perspective, elevated 4-hydroxybenzoic acid may be seen alongside other aromatic or phenolic metabolites, reflecting cumulative preservative or polyphenol exposure.

From a functional medicine perspective, this finding should be interpreted in the context of environmental and personal care exposures, hepatic conjugation capacity, and cumulative endocrine-disrupting burden, with emphasis on reducing ongoing exposure.

Treatment: Treatments focus on improving gut health through dietary changes like increasing plant-based foods, fermented foods, and prebiotics while reducing artificial sweeteners and potentially eliminating paraben-containing products.

5-METHYLCYTOSINE ELEVATED:

Urinary 5-methylcytosine (5-MC) is a modified nucleoside derived from DNA methylation and reflects global DNA methylation turnover and nucleic acid metabolism. Elevated levels may indicate increased DNA turnover, enhanced methylation flux, or increased degradation of methylated cytosine residues.

This pattern may be observed in the context of increased cellular turnover, oxidative stress, inflammatory processes, or enhanced DNA repair activity, and may also be influenced by environmental exposures, including certain xenobiotics.

While 5-MC provides insight into methylation turnover, it does not directly assess methylation capacity. Interpretation should be made in conjunction with clinical findings and, where appropriate, other markers of one-carbon metabolism and oxidative stress.

Suggested Treatment Considerations:

Review potential contributors to increased oxidative stress and environmental exposures. Consider assessment of one-carbon metabolism and methyl donor status (e.g. folate, vitamin B12, choline) where clinically indicated. Supportive strategies may include optimisation of nutritional status and antioxidant capacity. Further evaluation should be guided by the overall clinical context and associated laboratory findings.

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Lab ID
Patient ID P000065
Ext ID 26096-0001

Test Patient

Sex: Female • 56yrs • 01-Jan-70

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Methodology

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