US BioTek LABORATORIES · **PATIENT REPORT**
16020 Linden Av N Shoreline WA, 98133 • cservice@usbiotek.com • +1 206 365 1256

Clinical Laboratory Improvement Amendments

26212-0459

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

Patient ID P000060
Ext ID 26212-0459

Test Patient

Sex: Female • 56yrs • 01-Jan-70

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31-Jul-26

Clinical Notes .

NutriSTAT EZ Collect:

	Collected
Specimen type - Urine, Spot	28-Jul-26
Specimen type - Blood-EDTA	28-Jul-26

MICROBIAL OVERGROWTH

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

YEAST/FUNGAL METABOLITES

5-Hydroxymethyl-2-furoic Acid
Arabinose

ENVIRONMENTAL EXPOSURES

3-Methylhippuric Acid
4-Hydroxybenzoic Acid (4-HBA)
5-Methylcytosine
Benzoic Acid
Quinolinic Acid
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
Quinolinic Acid
Quinolinic Acid/5-HIAA Ratio
Xanthurenic Acid

AMINO ACIDS METABOLISM

2-Oxoisocaproic Acid
Guanidinoacetic Acid
N-Acetylphenylalanine
Phenylpyruvic Acid

VITAMINS / NUTRITIONAL MARKERS

Kynurenic Acid
Quinolinic Acid
Xanthurenic Acid

DETOXIFICATION / GLUTATHIONE FUNCTION

5-Hydroxymethyl-2-furoic Acid

OXIDATIVE DAMAGE / INFLAMMATION

Quinolinic Acid

OXALATE METABOLISM

N/A

Lab Director: Jillian Harrington, PhD, HCLD (ABB). CLIA#99D2282774. Testing performed by NutriPATH, 16 Harker Street, Burwood, VIC, 3125

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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	24.50	H	(<9.30)	mmol/molCR
2	Hippuric Acid	331.0		(<603.0)	mmol/molCR
3	4-Hydroxybenzoic Acid (4-HBA)	0.65	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	2.80	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)	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	0.39		(<0.50)	mmol/molCR
	Aspergillus Markers				
22	5-Hydroxymethyl-2-furoic Acid	16.50	H	(<10.00)	mmol/molCR
23	Furan-2,5-dicarboxylic Acid	12.90		(<15.00)	mmol/molCR
24	Tartaric Acid	11.50		(<15.00)	mmol/molCR
	Fusarium				
25	Tricarballylic Acid	0.21		(<0.44)	mmol/molCR

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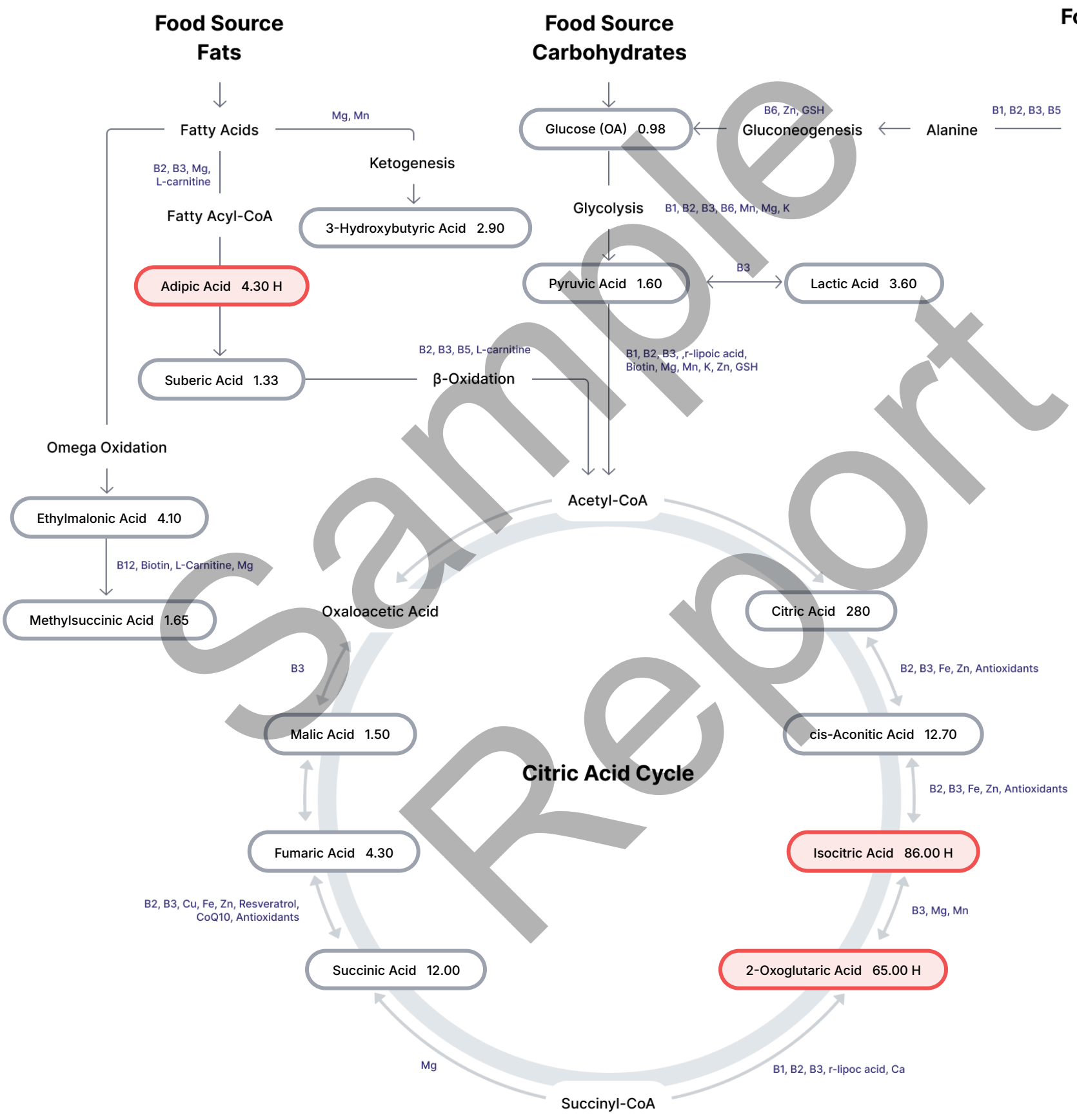
Organic Acids Pathway



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MITOCHONDRIAL & ENERGY METABOLISM

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

	TEST	RESULT	H/L	REFERENCE	UNITS
	Carbohydrate & Glycaemic Metabolism				
26	Glucose (OA)	0.98		(<1.10)	ug/mgCR
27	Pyruvic Acid	1.60		(0.50-8.70)	mmol/molCR
28	Lactic Acid	3.60		(<48.00)	mmol/molCR
29	2-Hydroxybutyric Acid **	4.10		(<6.90)	mmol/molCR
	** An elevated result may indicate methylation defects and/or toxic exposure				
	Citric Acid Cycle				
30	Citric Acid	280		(40-507)	mmol/molCR
31	cis-Aconitic Acid	12.70		(3.50-36.00)	mmol/molCR
32	Isocitric Acid	86.00	H	(5.00-65.00)	mmol/molCR
33	2-Oxoglutaric Acid	65.00	H	(2.00-45.00)	mmol/molCR
34	Succinic Acid	12.00		(1.00-15.00)	mmol/molCR
35	Fumaric Acid	4.30		(<8.60)	mmol/molCR
36	Malic Acid	1.50		(<1.80)	mmol/molCR
	Ketone/Energy Intermediates				
37	Acetoacetic Acid	8.80		(<10.00)	mmol/molCR
38	3-Hydroxybutyric Acid	2.90		(<3.10)	mmol/molCR
39	2-Hydroxybutyric Acid **	4.10		(<6.90)	mmol/molCR
	** An elevated result may indicate methylation defects and/or toxic exposure				
	Mitochondrial Markers				
40	Methylsuccinic Acid	1.65		(<10.80)	mmol/molCR
41	2-Methylglutaric Acid	0.45		(<0.76)	mmol/molCR
42	3-Methylglutaric Acid	2.90		(<8.50)	mmol/molCR
43	2-Hydroxyglutaric Acid	12.00		(<15.00)	mmol/molCR
44	3-Hydroxyglutaric Acid	4.20		(<5.50)	mmol/molCR
45	Malonic Acid	5.90		(<9.70)	mmol/molCR
46	Mevalonolactone	2.80	H	(<2.00)	mmol/molCR
47	2,4-Dihydroxybutanoic Acid	5.80		(<10.00)	mmol/molCR
48	N-Acetylaspartic Acid	10.40		(<15.00)	mmol/molCR
49	3-Methylglutaconic Acid	6.32	H	(<5.50)	mmol/molCR

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FATTY ACID OXIDATION & KETONE METABOLISM

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

VITAMIN & NUTRITIONAL COFACTOR MARKERS

* An elevated result may indicate nutrient deficiency

	TEST	RESULT	H/L	REFERENCE	UNITS
	B-Vitamin Functional Markers				
64	Methylmalonic Acid (MMA) *	1.65		(<1.90)	mmol/molCR
65	Formiminoglutamic Acid (FIGLU)	0.98		(<1.50)	mmol/molCR
66	Xanthurenic Acid	1.95	H	(<0.96)	mmol/molCR
67	Kynurenic Acid	3.10	H	(<2.20)	mmol/molCR
68	Quinolinic Acid	11.80	H	(<9.10)	mmol/molCR
69	Picolinic Acid	4.20		(<10.28)	mmol/molCR
70	3-Hydroxyisovaleric Acid	20.40		(<29.00)	mmol/molCR
	Vitamin-specific/Antioxidant Markers				
71	Pyroglutamic Acid *	28.80		(4.50-33.00)	mmol/molCR
72	N-Acetylcysteine (NAC)	0.05		(0.02-0.28)	mmol/molCR
73	Glutaric Acid (Vit B2) *	0.28		(0.02-0.36)	mmol/molCR
74	Pantothenic Acid (Vit B5)	3.30		(0.10-10.00)	mmol/molCR
75	Pyridoxic Acid (Vit B6)	25.90		(0.68-34.00)	mmol/molCR
76	Ascorbic Acid (Vit C)	59.00		(0.50-200.00)	mmol/molCR
77	Methylcitric Acid (Biotin/Vitamin H) *	4.40		(0.10-15.00)	mmol/molCR
78	3-Hydroxy-3-methylglutaric Acid (CoQ10) *	3.50		(0.10-5.00)	mmol/molCR

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PYRIMIDINE METABOLITES - Folate Metabolism

TEST		RESULT	H/L	REFERENCE	UNITS
79	Thymine	0.42		(<0.60)	mmol/molCR
80	Uracil	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					
81	2-Oxoisovaleric Acid	1.95		(<4.10)	mmol/molCR
82	2-Oxoisocaproic Acid	0.88	H	(<0.65)	mmol/molCR
83	3-Methyl-2-oxovaleric Acid	1.74		(<2.00)	mmol/molCR
84	3-Methylglutaric Acid	2.90		(<8.50)	mmol/molCR
85	Succinylacetone	0.41		(<0.50)	mmol/molCR
Downstream Metabolites					
86	2-Hydroxyisovaleric Acid	3.00		(<4.10)	mmol/molCR
87	2-Hydroxyisocaproic Acid	1.21		(<1.50)	mmol/molCR
88	2-Oxobutyric Acid	2.95		(<7.00)	mmol/molCR
89	2-Oxo-4-methiolbutyric Acid (KMBA)	3.20	H	(<1.50)	mmol/molCR
Amino Acid Metabolism					
90	Phenylpyruvic Acid	4.50	H	(<2.00)	mmol/molCR
91	Homogentisic Acid	1.30	H	(<1.00)	mmol/molCR
92	N-Acetylphenylalanine	6.60	H	(<5.00)	mmol/molCR
93	Mandelic Acid	188.0		(<340.0)	ug/gCR
94	Malonic Acid	5.90		(<9.70)	mmol/molCR
95	4-Hydroxyphenyllactic Acid (4-HPLA)	3.50		(<3.90)	mmol/molCR
96	2-Oxoadipic Acid	1.60		(<2.00)	mmol/molCR
97	Guanidinoacetic Acid	4.80	H	(0.50-3.00)	mmol/molCR
98	Guanidinobutyric Acid	0.88		(0.10-1.00)	mmol/molCR
99	3-Methylglutaric Acid	2.90		(<8.50)	mmol/molCR
100	N-Acetylglycine	2.90		(<5.00)	mmol/molCR

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NEUROTRANSMITTER METABOLITES

(Phenylalanine, Tyrosine)

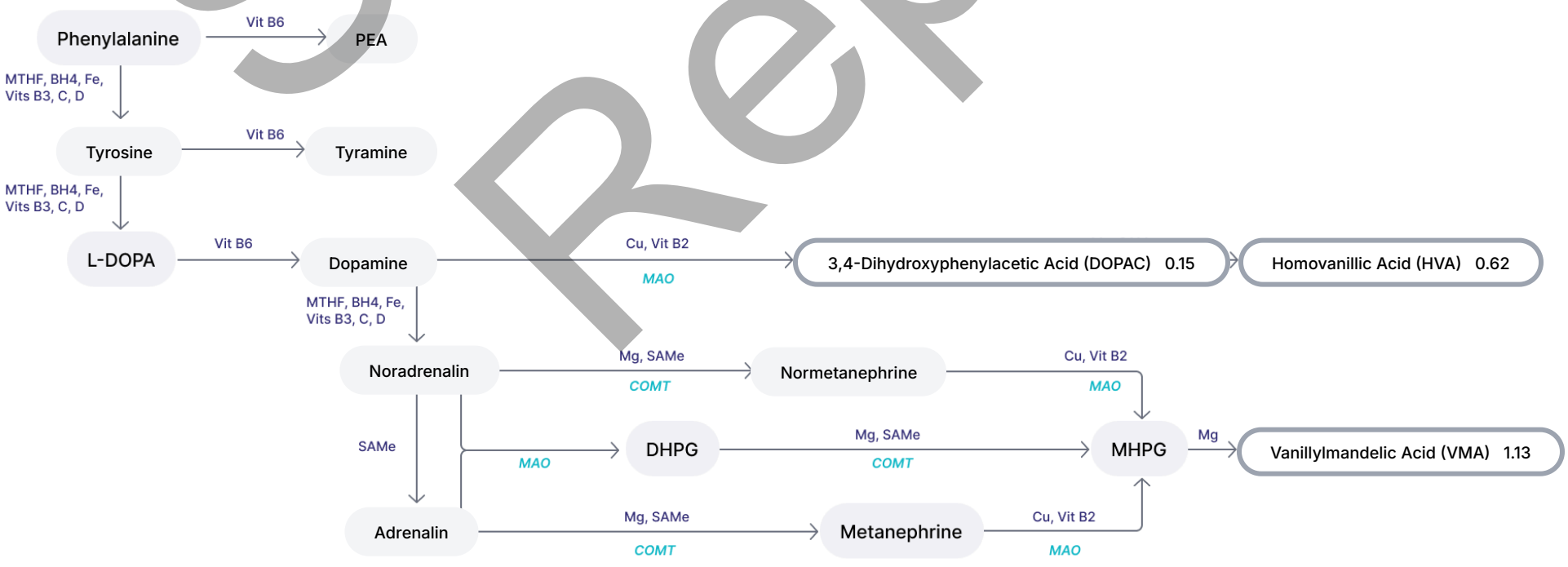
TEST	RESULT	H/L	REFERENCE	UNITS
Inhibitory (Serotonin)				
101 5-Hydroxyindoleacetic Acid (5-HIAA)	1.14		(<4.30)	mmol/molCR
Excitatory (Dopamine)				
102 3,4-Dihydroxyphenylacetic Acid (DOPAC)	0.15		(0.08-3.50)	mmol/molCR
103 3-Methyl-4-hydroxyphenylglycol (MHPG)	0.16		(0.05-0.50)	mmol/molCR
104 Homovanillic Acid (HVA)	0.62		(0.10-5.30)	mmol/molCR
105 Vanillylmandelic Acid (VMA)	1.13		(0.40-3.60)	mmol/molCR
106 HVA/DOPAC Ratio	4.1		(<10.0)	ratio
107 HVA/VMA Ratio	0.6		(<2.0)	ratio

ADRENAL STRESS (Overnight)

SERVICE	RESULT	H/L	REFERENCE	UNITS
108 Cortisol (OA)	1.0		(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)	1.14		(<4.30)	mmol/molCR
Inflammatory				
110 Kynurenic Acid	3.10	H	(<2.20)	mmol/molCR
111 Quinolinic Acid	11.80	H	(<9.10)	mmol/molCR
112 Picolinic Acid	4.20		(<10.28)	mmol/molCR
113 Xanthurenic Acid	1.95	H	(<0.96)	mmol/molCR
114 Kynurenic/Quinolinic Ratio	0.3		(<2.0)	ratio
115 Quinolinic Acid/5-HIAA Ratio	10.4	H	(<5.0)	ratio

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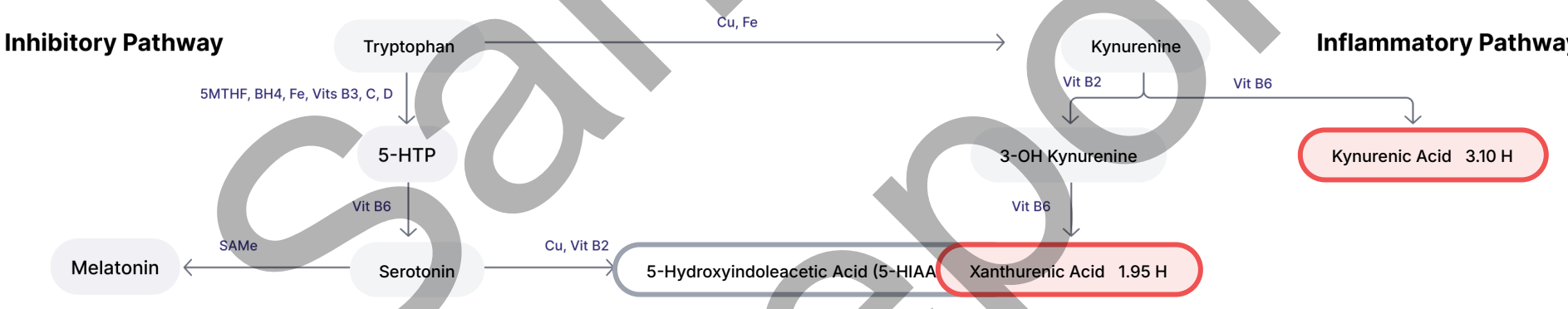

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DETOXIFICATION & GLUTATHIONE FUNCTION

(Arg, NAC, Meth, Mg, Antioxidants)

TEST		RESULT	H/L	REFERENCE	UNITS
Ammonia Metabolism					
116	Orotic Acid	1.95		(<3.20)	mmol/molCR
Glutathione Metabolism					
117	Pyroglutamic Acid *	28.80		(4.50-33.00)	mmol/molCR
118	N-Acetylcysteine (NAC)	0.05		(0.02-0.28)	mmol/molCR
119	Glucaric Acid	8.40		(<11.00)	mmol/molCR
Phase I / Xenobiotic Markers					
120	2-Hydroxyhippuric Acid	0.80		(<1.50)	mmol/molCR
121	5-Hydroxymethyl-2-furoic Acid	16.50	H	(<10.00)	mmol/molCR
122	Furan-2,5-dicarboxylic Acid	12.90		(<15.00)	mmol/molCR

OXIDATIVE STRESS & INFLAMMATION

(Vitamin C, Other Antioxidants)

TEST		RESULT	H/L	REFERENCE	UNITS
123	8-hydroxy-deoxyguanosine	1.71		(<2.70)	umol/molCR
124	Leukotriene E4	12.80		(<100.00)	pg/mgCR
125	Quinolinic Acid	11.80	H	(<9.10)	mmol/molCR
126	Pyroglutamic Acid *	28.80		(4.50-33.00)	mmol/molCR
127	Ascorbic Acid (Vit C)	59.00		(0.50-200.00)	mmol/molCR
128	Quercetin	0.87		(0.01-2.00)	mmol/molCR

OXALATE METABOLISM

TEST		RESULT	H/L	REFERENCE	UNITS
129	Glycolic Acid	20.80		(<67.00)	mmol/molCR
130	Glyceric Acid	3.80		(<6.00)	mmol/molCR
131	Oxalic Acid	42.00		(<78.00)	mmol/molCR

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ENVIRONMENTAL / XENOBIOTIC EXPOSURE

TEST	RESULT	H/L	REFERENCE	UNITS
Toluene Exposure				
132 Hippuric Acid	331.0		(<603.0)	mmol/molCR
133 Benzoic Acid	24.50	H	(<9.30)	mmol/molCR
Paraben Exposure				
134 4-Hydroxybenzoic Acid (4-HBA)	0.65	H	(<0.57)	mmol/molCR
Styrene Exposure				
135 Mandelic Acid	188.0		(<340.0)	ug/gCR
136 Phenylglyoxylic Acid	188.0		(<300.0)	ug/gCR
137 Mandelic Acid + Phenylglyoxylic Acid	284.0		(<610.0)	ug/gCR
Benzene Exposure				
138 t,t-Muconic Acid	0.20	H	(<0.12)	mmol/molCR
Phthalate Exposure				
139 Phthalic Acid	149.00		(<170.00)	ug/gCR
140 Monoethyl Phthalate	60.30		(<100.00)	ug/gCR
141 Quinolinic Acid	11.80	H	(<9.10)	mmol/molCR
MTBE Exposure				
142 2-Hydroxylsobutyric Acid	3.90		(<6.90)	mmol/molCR
Xylene Exposure				
143 2-Methylhippuric Acid	0.01		(<0.04)	mmol/molCR
144 3-Methylhippuric Acid	0.32	H	(<0.11)	mmol/molCR
145 4-Methylhippuric Acid	1.69		(<1.80)	mmol/molCR
Trimethylbenzene Exposure				
146 3,4-Dimethylhippuric Acid	0.00		(<0.01)	mmol/molCR
147 4-Hydroxyhippuric Acid	6.23		(<16.50)	mmol/molCR
Nucleotide Turnover/Methylation				
148 5-Methylcytosine	68.00	H	(10.00-50.00)	mmol/molCR
149 Uracil	8.10		(<9.00)	mmol/molCR
150 Thymine	0.42		(<0.60)	mmol/molCR

TEST	RESULT	H/L	REFERENCE	UNITS
151 Creatinine, Urine	5.50		(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	Moderate	80.0	mg
Vitamin-B3	Adequate	0.0	mg
Vitamin-B5	Mild	60.0	mg
Vitamin-B6	Moderate	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 *	Moderate	18.0	mg

* Nutritional need for Iron should be confirmed with serum Iron Studies test.

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	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

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.

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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.

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.

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.

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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.

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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.

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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.

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.

PHENYLPYRUVIC ACID ELEVATED:

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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.

Suggested Treatment Considerations:

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.

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.

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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.

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.

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.

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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.

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.

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).

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.

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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.

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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PHYSIOLOGICAL MINERALS

TEST	RESULT	H/L	REFERENCE	UNITS
Calcium	58.00		(<450.00)	mg/gCR
Iron	3		(<200)	ug/gCR
Magnesium	32.00		(<290.00)	mg/gCR
Zinc	0.13		(<900.00)	ug/gCR

TRACE MINERALS

TEST	RESULT	H/L	REFERENCE	UNITS
Boron	0		(<5500)	ug/gCR
Chromium	2.09		(<4.60)	ug/gCR
Cobalt	0.88		(<1.60)	ug/gCR
Copper	66.0	H	(<55.0)	ug/gCR
Germanium	<DL		(<1.50)	ug/gCR
Iodine	88.00	L	(>100.00)	ug/L
Lithium	45.00		(<55.00)	ug/gCR
Manganese	0.93		(<1.50)	ug/gCR
Molybdenum	10.60		(<65.00)	ug/gCR
Nickel	0.81		(<2.00)	ug/gCR
Rubidium	11		(<3000)	ug/gCR
Selenium	11.70		(10.00-63.00)	ug/gCR
Strontium	82.00		(<310.00)	ug/gCR
Vanadium	3.88		(<8.00)	ug/gCR

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TOXIC METALS

TEST	RESULT	H/L	REFERENCE	UNITS
Aluminium	25.00		(<40.00)	ug/gCR
Antimony	<DL		(<1.00)	ug/gCR
Arsenic	38.00	H	(<35.00)	ug/gCR
Barium	0.89		(<5.70)	ug/gCR
Beryllium	<DL		(<0.60)	ug/gCR
Bismuth	<DL		(<1.00)	ug/gCR
Bromine	190		(<4800)	ug/gCR
Cadmium	0.22		(<0.60)	ug/gCR
Cesium	2.33		(<10.30)	ug/gCR
Gadolinium	<DL		(<0.23)	ug/gCR
Gallium	<DL		(<0.10)	ug/gCR
Lead	19.00	H	(<8.00)	ug/gCR
Mercury	5.9	H	(<3.0)	ug/gCR
Palladium	0.01		(<15.00)	ug/gCR
Platinum	0.10		(<1.00)	ug/gCR
Silver	0.22	H	(<0.10)	ug/gCR
Tellurium	<DL		(<0.80)	ug/gCR
Thallium	<DL		(<1.50)	ug/gCR
Tin	0.65	H	(<0.50)	ug/gCR
Titanium	0.05		(<50.00)	ug/gCR
Tungsten	0.01		(<0.50)	ug/gCR
Uranium	0.01		(<0.10)	ug/gCR
Zirconium	0.16		(<5.00)	ug/gCR

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CHELATOR SPECIFIC ORIENTATION RANGES:

The following chelator-specific ranges have been developed based on the fact that each chelator has a specific binding capacity. The ranges were developed based on statistical calculations of a population challenged with one ampoule DMPS or 300mg DMPS.

A urine excretion value/result higher than the Orientation Ranges below, indicates a moderate to high toxic burden.

A test result higher than the normal (pre-chelation) reference range and lower than the Orientation Range indicates a low to moderate toxic burden.

ELEMENT	DMSA (ug/gCR)	EDTA (ug/gCR)	DMPS (ug/gCR)
Toxic Metals			
Aluminium (Al)	< 50	< 100	< 50
Antimony (Sb)	< 10	< 10	< 15
Arsenic (As)	< 100	< 100	< 100
Barium (Ba)	< 10	< 20	< 10
Beryllium (Be)	< 2.0	< 2.0	< 2.0
Bismuth (Bis)	< 10	< 10	< 10
Cadmium (Cd)	< 5.0	< 5.0	< 5.0
Cesium (Cs)	< 30	< 30	< 30
Cobalt (Co)	< 15	< 15	< 15
Gadolinium (Gd)	< 5.0	< 5.0	< 5.0
Lead (Pb)	< 30	< 80	<20
Mercury (Hg)	< 20	< 10	< 40
Nickel (Ni)	< 20	< 30	< 20
Palladium (Pd)	< 15	< 15	< 15
Platinum (Pt)	< 5.0	< 5.0	< 5.0
Silver (Ag)	< 5.0	< 5.0	< 5.0
Thallium (Tl)	< 10	< 10	< 10
Tin (Sn)	< 15	< 20	< 15
Titanium (Ti)	< 50	< 50	< 50
Uranium (U)	< 1.0	< 1.0	< 1.0
Essential Elements			
Manganese (Mn)	< 5.0	< 10	< 5.0
Strontium (Sr)	< 310	< 310	< 310
Tungsten (W)	< 10	< 10	< 10
Vanadium (V)	< 8.0	< 10	< 8.0
Zinc (Zn)	< 1500	< 2000	< 1500

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Nutrient Mineral Comment

COPPER ELEVATED (URINE):

Elevated urinary copper suggests increased copper excretion, which may occur with higher intake, inflammatory activity, hormonal influences, or altered hepatic copper handling.

Clinically, elevated urinary copper may be associated with fatigue, mood changes, or increased oxidative stress, although findings are non-specific.

From a functional medicine perspective, this result should be interpreted in the context of copper–zinc balance, inflammatory status, oestrogen exposure, and liver function rather than as an isolated indicator of copper excess. Correlation with serum copper and plasma zinc may assist in contextual interpretation where clinically indicated.

Toxic Metals Comment

ARSENIC ELEVATED (URINE):

Arsenic exposure may be organic (dietary, e.g. seafood) or inorganic (toxic). Urinary elevation reflects recent exposure and detoxification efficiency.

Clinically, elevated urinary levels may be associated with gastrointestinal upset, fatigue, skin changes, or neuropathic symptoms. Interpretation should consider recent exposure, occupational or environmental sources, renal clearance, and nutritional status.

From a functional medicine perspective, management focuses on differentiating organic vs inorganic sources, improving methylation capacity, ensuring folate and selenium sufficiency, and reducing exposure.

Treatment: Distinguish between organic and inorganic forms; support methylation (folate, B12, SAMe), and use chelation agents like DMSA or NAC if applicable.

LEAD ELEVATED (URINE):

Elevated urinary lead suggests increased lead exposure and/or mobilisation with renal excretion. Urinary lead reflects excretory burden rather than circulating blood lead concentration.

Clinically, elevated lead exposure may be associated with fatigue, cognitive changes, gastrointestinal symptoms, or neurological effects, depending on exposure magnitude and chronicity.

From a functional medicine perspective, this finding should be interpreted in the context of environmental and occupational exposure sources, nutritional factors influencing lead handling (e.g. iron and calcium status), and correlation with blood lead testing where clinically indicated.

Treatment: Chelation therapy (e.g., DMSA, EDTA), zinc and iron repletion, vitamin C and antioxidant support.

MERCURY ELEVATED (URINE):

Elevated urinary mercury suggests increased mercury exposure and renal excretion. Urinary mercury primarily reflects inorganic or elemental mercury exposure rather than methylmercury from seafood.

Clinically, elevated urinary mercury may be associated with neurocognitive symptoms, tremor, fatigue, or renal effects, depending on exposure magnitude and chronicity.

From a functional medicine perspective, this finding should be interpreted in the context of exposure sources (occupational, dental, environmental), renal function, and antioxidant capacity. Correlation with exposure history and, where clinically indicated, mercury speciation or blood testing may assist contextual interpretation.

Treatment: Eliminate exposure (e.g., amalgam removal by trained providers), use selenium, NAC, ALA, and chelation agents as appropriate.

TIN ELEVATED (URINE):

Tin exposure may be dietary or industrial. Elevated urinary tin reflects exposure and excretion.

Clinically, elevated urinary levels may be associated with gastrointestinal symptoms or fatigue. Interpretation should consider recent exposure, occupational or environmental sources, renal clearance, and nutritional status.

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From a functional medicine perspective, management focuses on reducing exposure and supporting renal clearance, prioritising exposure reduction and physiological resilience rather than aggressive intervention.

Treatment: Eliminate exposure, support liver detoxification, and use antioxidants (e.g., vitamin E, selenium).

SILVER ELEVATED (URINE):

Elevated urinary silver suggests increased exposure and renal excretion, commonly related to supplements (e.g. colloidal silver), occupational contact, or environmental sources.

Clinically, excessive silver exposure may be associated with skin discolouration or non-specific symptoms at higher or sustained levels, though mild elevations are often asymptomatic.

From a functional medicine perspective, this result should be interpreted in the context of supplement use and exposure history, with emphasis on exposure reduction rather than intervention.

Treatment: Identify and eliminate the source of silver exposure; possible treatment with laser therapy.

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ESSENTIAL AMINO ACIDS

SERVICE		RESULT	H/L	REFERENCE	UNITS
1	Histidine	40.8		(15.0-300.0)	umol/gCR
2	Isoleucine	46.0	H	(0.0-6.0)	umol/gCR
3	Leucine	75.3	H	(0.0-10.0)	umol/gCR
4	Lysine	65.0	H	(2.0-50.0)	umol/gCR
5	Methionine	6.3	H	(0.0-5.0)	umol/gCR
6	Phenylalanine	40.6	H	(0.0-20.0)	umol/gCR
7	Threonine	63.8		(1.0-80.0)	umol/gCR
8	Tryptophan	37	H	(2-18)	umol/gCR
9	Valine	138.0	H	(0.5-40.0)	umol/gCR
10	Total Branched Chain AAs	259	H	(1-70)	umol/gCR

CONDITIONALLY ESSENTIAL AMINO ACIDS

TEST		RESULT	H/L	REFERENCE	UNITS
11	Arginine	3.8		(0.0-8.0)	umol/gCR
12	Cysteine	11.8		(1.0-25.0)	umol/gCR
13	Glutamine	299.0	H	(2.0-100.0)	umol/gCR
14	Glycine	175.0		(40.0-800.0)	umol/gCR
15	Proline	119	H	(0-20)	umol/gCR
16	Taurine	11.8	L	(15.0-350.0)	umol/gCR
17	Tyrosine	35	H	(0-14)	umol/gCR

NON-ESSENTIAL AMINO ACIDS

SERVICE		RESULT	H/L	REFERENCE	UNITS
18	Alanine	188	H	(7-95)	umol/gCR
19	Asparagine	41.2		(2.0-70.0)	umol/gCR
20	Aspartate	4.3		(0.2-10.0)	umol/gCR
21	Glutamate	309	H	(1-20)	umol/gCR
22	Serine	40.7		(0.0-120.0)	umol/gCR
23	Large Neutral Amino Acids (LNAA)	335	H	(3-85)	umol/gCR

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INTERMEDIARY & FUNCTIONAL METABOLITES

(Functional markers of B-Vitamin status, Urea Cycle, & one-carbon metabolism)

SERVICE		RESULT	H/L	REFERENCE	UNITS
24	alpha-Aminoadipic Acid	0.0		(0.0-7.0)	umol/gCR
25	alpha-Aminobutyric Acid	14.0	H	(0.0-5.0)	umol/gCR
26	beta-Aminoisobutyric Acid	0.9		(0.0-5.0)	umol/gCR
27	Cystathionine	0.11		(0.00-20.00)	umol/gCR
28	Citrulline	23.20	H	(0.00-3.00)	umol/gCR
29	GABA	0.88		(<15.00)	umol/gCR
30	Ornithine	0.9		(0.0-7.0)	umol/gCR
31	Phosphoserine	0.4		(0.0-120.0)	umol/gCR
32	Sarcosine	4.0		(0.0-8.0)	umol/gCR

DIETARY PEPTIDE RELATED MARKERS

SERVICE		RESULT	H/L	REFERENCE	UNITS
33	1-Methyl Histidine	4.78	L	(5.00-300.00)	umol/gCR
34	3-Methyl Histidine	12.9		(5.0-200.0)	umol/gCR
35	beta-Alanine	1.4		(0.0-8.0)	umol/gCR
36	Anserine	3.8		(0.0-7.0)	umol/gCR
37	Hydroxyproline	6.2	H	(0.0-3.0)	umol/gCR
38	Hydroxylysine	0.4		(0.0-7.0)	umol/gCR
39	Carnosine	0.56		(0.00-10.00)	umol/gCR

AMINO ACID FUNCTIONAL RATIOS

SERVICE		RESULT	H/L	REFERENCE	UNITS
40	Phenylalanine/Tyrosine	1.2		(<25.0)	ratio
41	Glutamate/Glutamine	1.03		(0.10-4.00)	ratio
42	Hydroxyproline/Proline	0.1		(0.0-2.0)	ratio
43	a-Aminobutyrate/Leucine	0.2		(0.0-2.0)	ratio
44	Tryptophan/LNAAs	0.11		(0.02-1.00)	ratio

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Amino Acids Comment

LEUCINE ELEVATED:

Description: Elevated urinary leucine indicates either overflow from markedly elevated circulating leucine exceeding tubular reabsorption capacity, or defective B0AT1-mediated reabsorption of neutral amino acids at the proximal tubule.

Clinical Significance: Elevated urine leucine as part of a combined BCAA elevation (isoleucine + leucine + valine) alongside their branched-chain keto acid derivatives is characteristic of MSUD. Isolated leucinuria beyond the BCAA pattern may suggest isovaleric acidaemia (IVA, OMIM #243500, isovaleryl-CoA dehydrogenase deficiency) where leucine catabolism is specifically blocked urine isovalerylglycine is the confirmatory marker and produces a characteristic 'sweaty feet' odour. Broad neutral aminoaciduria including leucine elevation indicates Hartnup disorder.

Suggested Treatment: Perform urine organic acid analysis to identify keto acid derivatives. If MSUD, refer urgently to metabolic specialist. For IVA, supplement glycine (to conjugate isovaleric acid) and carnitine. For Hartnup disorder, supplement nicotinamide and optimise dietary protein. Review BCAA supplementation for mild overflow elevations.

LYSINE ELEVATED:

Description: Elevated urinary lysine (lysinuria) most significantly occurs as part of a dibasic aminoaciduria pattern where the shared cationic amino acid transporter is defective, resulting in urinary wasting of lysine alongside arginine and ornithine.

Clinical Significance: Dibasic aminoaciduria (elevated urine lysine + arginine + ornithine) with low or normal circulating levels is the hallmark of lysinuric protein intolerance (LPI, OMIM #222700, SLC7A7 mutations). In LPI, the intestinal and renal basolateral cationic transporter is defective causing post-prandial hyperammonaemia, protein intolerance, hepatosplenomegaly, and pulmonary complications. Cystinuria (SLC3A1/SLC7A9) also causes dibasic aminoaciduria but without hyperammonaemia and with prominent urinary cystine elevation. Isolated lysinuria with elevated circulating lysine suggests hyperlysinaemia (AASS deficiency, OMIM #238700), typically benign.

Suggested Treatment: Quantify urine arginine, ornithine, and cystine to characterise the pattern. Test SLC7A7 for LPI or SLC3A1/SLC7A9 for cystinuria. LPI is managed with low-protein diet, citrulline supplementation (2-8 g/day), and carnitine. Cystinuria requires high fluid intake (>3L/day) and alkalinisation.

METHIONINE ELEVATED:

Description: Elevated urinary methionine (methioninuria) occurs when circulating methionine is markedly elevated and exceeds tubular reabsorptive capacity, or when neutral amino acid transport at the proximal tubule is broadly impaired.

Clinical Significance: Methioninuria is a key urinary finding in homocystinuria due to CBS deficiency (OMIM #236200), where both methionine and homocysteine accumulate and overflow into urine the urine cyanide-nitroprusside test historically detected urinary homocystine. It also occurs in methionine adenosyltransferase I/III deficiency (MAT1A, OMIM #250850) and citrin deficiency. In Hartnup disorder, methionine may be among the broadly elevated neutral amino acids in urine. High dietary methionine from excessive supplementation or very high animal protein intake can also produce methioninuria.

Suggested Treatment: Quantify urine homocystine/homocysteine and full amino acid panel. If CBS deficiency is suspected, test CBS gene and assess B6 responsiveness. Review methionine supplementation and dietary protein load. Ensure adequate B-vitamin cofactors for methionine cycle processing.

PHENYLALANINE ELEVATED:

Description: Elevated urinary phenylalanine (phenylalaninuria) occurs when circulating phenylalanine exceeds the renal tubular transport maximum. In untreated or poorly controlled PKU, characteristic urinary metabolites including phenylpyruvic acid, phenyllactic acid, and phenylacetic acid also accumulate, producing the musty urine odour historically associated with the condition.

Clinical Significance: Phenylalaninuria with urinary phenylpyruvate (giving a green colour with ferric chloride reagent) is the classic urinary finding in phenylketonuria (PKU, OMIM #261600) and its variants. Urinary phenylalanine screening using the Guthrie bacterial inhibition assay was historically used for newborn screening before blood-spot methods became standard. Elevated urinary phenylalanine without urinary metabolite accumulation may occur in mild hyperphenylalaninaemia. Broad neutral aminoaciduria including phenylalanine is also seen in Hartnup disorder (B0AT1 deficiency).

Suggested Treatment: Confirm with quantitative urine amino acid analysis and urine organic acids. Refer urgently to a metabolic specialist if PKU spectrum is confirmed. Initiate dietary phenylalanine restriction, medical formula supplementation, and BH4 responsiveness testing.

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Screen for BH4 deficiency disorders (PTPS, DHPR) which require additional neurotransmitter precursor therapy.

TRYPTOPHAN ELEVATED:

Description: Elevated urinary tryptophan alongside characteristic indole compounds in urine (indolylacrylic acid, indican/indoxyl sulphate, indoleacetic acid, indolelactic acid) is the diagnostic urinary pattern of Hartnup disorder (OMIM #234500, SLC6A19/B0AT1 deficiency).

Clinical Significance: In Hartnup disorder, intestinal tryptophan absorption via B0AT1 is impaired, allowing large amounts of tryptophan to reach the colon where bacterial fermentation converts it to indole compounds. These are absorbed, partially conjugated, and excreted in urine as indican and indole derivatives, while renal tubular tryptophan reabsorption is simultaneously defective causing tryptophanuria. The clinical consequence is functional niacin deficiency (tryptophan is the endogenous niacin precursor via the kynurenine pathway), producing pellagra-like photosensitive dermatitis, cerebellar ataxia, and psychiatric symptoms. Elevated urinary indican without tryptophanuria may also reflect small intestinal bacterial overgrowth (SIBO).

Suggested Treatment: If Hartnup disorder is confirmed (SLC6A19 gene), supplement nicotinamide to compensate for impaired tryptophan-to-NAD+ conversion. High-protein diet supports adequate tryptophan intake via peptide transporters (PEPT1), which remain intact in Hartnup. Investigate and treat SIBO if indican is elevated without tryptophanuria.

VALINE ELEVATED:

Description: Elevated urinary valine occurs when circulating valine is markedly elevated due to impaired BCKDH complex catabolism, or when neutral amino acid tubular reabsorption is defective causing broad aminoaciduria.

Clinical Significance: Elevated urinary valine alongside leucine and isoleucine with branched-chain keto acids in urine is the hallmark of maple syrup urine disease (MSUD, OMIM #248600). The characteristic sweet/caramel urine odour in MSUD is produced by the excreted branched-chain keto acid derivatives. Isolated valinuria with markedly elevated circulating valine (without leucine and isoleucine elevation) points to valinemia (OMIM #277100, valine aminotransferase deficiency), an extremely rare disorder. Broad neutral aminoaciduria including valine occurs in Hartnup disorder (B0AT1 deficiency).

Suggested Treatment: Perform urine organic acids to identify branched-chain keto acids. If MSUD pattern confirmed, refer urgently to a metabolic specialist for dietary BCAA restriction and monitoring. For isolated valinemia (rare), specialist management is required. For Hartnup disorder, supplement nicotinamide and optimise protein intake.

TOTAL BRANCHED CHAIN AMINO ACIDS ELEVATED:

Description: Elevated total urinary BCAAs occur when circulating BCAA concentrations are markedly elevated exceeding tubular reabsorption capacity or when B0AT1-mediated neutral amino acid reabsorption is impaired, causing broad aminoaciduria.

Clinical Significance: Pan-BCAA elevation in urine alongside keto acid derivatives (detectable on urine organic acids) is the hallmark of MSUD. In MSUD the characteristic sweet/maple syrup odour of urine is produced by the excreted keto acids. In Hartnup disorder (B0AT1/SLC6A19 deficiency), all neutral amino acids including BCAAs are elevated in urine due to defective tubular reabsorption here urine tryptophan and indole compounds additionally distinguish Hartnup. Mild overflow BCAA-uria from excessive supplementation is less common but possible.

Suggested Treatment: Perform urine organic acids to identify BCAA-derived keto acids. If MSUD confirmed, refer urgently to metabolic specialist. For Hartnup disorder, supplement nicotinamide and ensure adequate protein intake. Review BCAA supplementation for isolated mild elevations without metabolic disorder features.

GLUTAMINE ELEVATED:

Description: Elevated urinary glutamine occurs when glutamine accumulates in the circulation and exceeds renal tubular reabsorptive capacity, or when renal tubular reabsorption is defective as part of Fanconi syndrome.

Clinical Significance: Elevated urinary glutamine as part of a broad aminoaciduria is characteristic of renal Fanconi syndrome (from Wilson's disease, cystinosis, tyrosinaemia type I, heavy metal toxicity, or antiretroviral therapy), where multiple proximal tubular transport functions are impaired. High-dose L-glutamine supplementation is the most common cause of elevated urinary glutamine in clinical practice excess glutamine simply overflows renal reabsorption. Glutamine synthetase gain-of-function mutations may elevate circulating glutamine with secondary glutaminuria.

Lab Director: Jillian Harrington, PhD, HCLD (ABB). CLIA#99D2282774. Testing performed by NutriPATH, 16 Harker Street, Burwood, VIC, 3125

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Suggested Treatment: Assess for Fanconi syndrome markers (glucosuria, phosphaturia, uricosuria, broad aminoaciduria) if elevated urinary glutamine is unexplained. Review L-glutamine supplement dose. Investigate Wilson's disease and cystinosis in appropriate clinical contexts. Avoid high-dose glutamine supplementation in multi-organ failure contexts.

PROLINE ELEVATED:

Description: Elevated urinary proline (prolinuria) occurs as overflow from markedly elevated circulating proline in the hyperprolinaemias, or as part of iminoglycinuria where the shared imino acid tubular transporter is defective, causing urinary excretion of proline, hydroxyproline, and glycine without circulating elevations.

Clinical Significance: Prolinuria with elevated circulating proline is characteristic of hyperprolinaemia type I (OMIM #239500, PRODH deficiency) and type II (OMIM #239510, P5CDH/ALDH4A1 deficiency). Type II also produces elevated urinary pyrroline-5-carboxylate (P5C) and is associated with intellectual disability and seizures. Iminoglycinuria (OMIM #242600, SLC36A2 or SLC6A20 defects) causes elevated urinary proline, hydroxyproline, and glycine without circulating elevation and is generally benign. Broad aminoaciduria from Fanconi syndrome will also include proline wasting.

Suggested Treatment: If prolinuria accompanies elevated circulating proline, confirm PRODH or ALDH4A1 gene defects. Type II hyperprolinaemia requires specialist management. For iminoglycinuria (isolated proline, hydroxyproline, and glycine in urine without circulating elevation), confirm the benign nature and no specific treatment is required. Investigate Fanconi syndrome if broad aminoaciduria accompanies the finding.

TAURINE LOW:

Increase dietary intake from seafood, meat, and poultry. Assess B6 (P5P) status as the cofactor for cysteine sulphinic acid decarboxylase (CSAD), the enzyme producing taurine from cysteine. Evaluate cardiovascular function in confirmed long-term depletion states.

TYROSINE ELEVATED:

Description: Elevated urinary tyrosine (tyrosinuria) occurs as overflow from markedly elevated circulating tyrosine when hepatic tyrosine catabolism is impaired, or as part of broad neutral aminoaciduria from tubular transport defects.

Clinical Significance: Tyrosinuria is a key urinary finding in all three tyrosinaemia syndromes. In tyrosinaemia type I (OMIM #276700, FAH deficiency), urinary succinylacetone is the pathognomonic metabolite more diagnostically specific than tyrosinuria. In type II (OMIM #276600, TAT deficiency), marked tyrosinuria accompanies corneal erosions and palmoplantar keratosis. In Hartnup disorder, tyrosine is among the broadly elevated neutral amino acids in urine. Transient neonatal tyrosinaemia in premature infants produces benign, self-resolving tyrosinuria. Severe liver disease from any cause impairs tyrosine catabolism, producing secondary tyrosinuria.

Suggested Treatment: Quantify urinary succinylacetone urgently for tyrosinaemia type I (NTBC/nitisinone is the primary treatment under specialist care). Assess liver function. Evaluate Hartnup disorder if broad neutral aminoaciduria accompanies the finding. Document prematurity and neonatal status as a confounder.

ALANINE ELEVATED:

Description: Elevated urinary alanine indicates overflow from markedly elevated circulating alanine due to impaired mitochondrial energy metabolism, or generalised aminoaciduria from renal Fanconi syndrome where tubular reabsorption of multiple solutes is defective.

Clinical Significance: Elevated urine alanine alongside elevated urinary lactate and pyruvate, often accompanied by metabolic acidosis, is a characteristic urinary finding in pyruvate dehydrogenase (PDH) deficiency and mitochondrial respiratory chain disorders. In these conditions, pyruvate accumulation drives excess alanine production via ALT transamination. Renal Fanconi syndrome (from Wilson's disease, cystinosis, tyrosinaemia type I, heavy metal toxicity, or antiretroviral therapy) causes broad urinary wasting of amino acids including alanine, alongside glucose, phosphate, and urate. Iminoglycinuria characteristically elevates urine alanine alongside glycine and proline.

Suggested Treatment: Perform urine organic acids (elevated lactate/pyruvate suggests mitochondrial dysfunction). Assess for Fanconi syndrome markers (glucosuria, phosphaturia, uricosuria) if broad aminoaciduria is present. Investigate Wilson's disease and cystinosis in appropriate clinical contexts. Refer to specialist for mitochondrial disorder evaluation.

GLUTAMIC ACID ELEVATED:

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Description: Elevated urinary glutamate alongside aspartate constitutes dicarboxylic aminoaciduria from defective EAAT3 (SLC1A1) renal tubular reabsorption. Isolated elevated urinary glutamate without aspartate elevation may reflect dietary glutamate excess or generalised Fanconi aminoaciduria.

Clinical Significance: Dicarboxylic aminoaciduria (elevated urine glutamate + aspartate) from SLC1A1/EAAT3 deficiency is generally considered benign. In renal Fanconi syndrome, glutamate is among the broadly wasted amino acids alongside glucose, phosphate, and urate. High dietary monosodium glutamate (MSG) consumption can transiently increase urinary glutamate as a dietary confounder. Glutamate dehydrogenase gain-of-function (hyperinsulinism-hyperammonaemia syndrome) causes elevated circulating glutamate with secondary glutamateuria.

Suggested Treatment: Quantify urine aspartate concurrently. Assess for Fanconi syndrome (glucosuria, phosphaturia) if broad aminoaciduria is present. Review dietary MSG intake as a confounder. Assess for hyperinsulinism-hyperammonaemia if clinically relevant. No treatment required for benign dicarboxylic aminoaciduria.

LARGE NEUTRAL AMINO ACIDS (Isol+Leu+Val+Phen+Tyr) ELEVATED:

Description: Elevated total urinary LNAAs indicate either overflow from markedly elevated circulating LNAAs exceeding tubular transport capacity, or defective B0AT1 (SLC6A19)-mediated reabsorption producing broad neutral aminoaciduria.

Clinical Significance: Broad neutral aminoaciduria elevated urinary excretion of multiple neutral amino acids simultaneously including all or most LNAAs is the hallmark of Hartnup disorder (OMIM #234500, SLC6A19/B0AT1 deficiency). The pellagra-like rash, cerebellar ataxia, and psychiatric symptoms of Hartnup result specifically from impaired intestinal tryptophan absorption (not renal wasting) reducing niacin synthesis. Individual LNAA elevations indicate specific disorders: elevated urinary phenylalanine points to PKU; elevated urinary BCAAs with keto acids indicates MSUD; elevated urinary tyrosine indicates tyrosinaemia.

Suggested Treatment: Characterise individual amino acid elevations within the LNAA group to identify the specific disorder. If broad neutral aminoaciduria confirms Hartnup disorder, supplement nicotinamide (50-300 mg/day) and ensure adequate high-protein dietary intake using intact peptide sources.

ALPHA-AMINO-N-BUTYRATE ELEVATED:

Description: Elevated urinary AANB reflects overflow from accumulation of this threonine and methionine catabolism intermediate when B6-dependent transaminase activity is insufficient to adequately catabolise it. The excess is then renally excreted.

Clinical Significance: Elevated urinary AANB, mirroring a raised circulating AANB/Leucine ratio, is a functional marker of B6 (pyridoxal-5-phosphate) and B2 (riboflavin) insufficiency. B6-dependent aminotransferases are responsible for AANB catabolism; when cofactor availability is limiting, AANB accumulates and overflows into urine. This functional deficit impairs neurotransmitter synthesis (aromatic amino acid decarboxylase requires P5P), homocysteine processing, and overall amino acid nitrogen metabolism. Elevated urinary AANB provides corroborating evidence for the circulating AANB/Leucine ratio finding.

Suggested Treatment: Supplement pyridoxal-5-phosphate (P5P) and riboflavin-5-phosphate (R5P) as activated B-vitamins. Reassess both urinary AANB and the circulating AANB/Leucine ratio after 6-8 weeks of supplementation. Evaluate dietary B-vitamin intake and gastrointestinal absorptive capacity.

CITRULLINE ELEVATED:

Description: Elevated urinary citrulline (citrullinuria) occurs as overflow from markedly elevated circulating citrulline when argininosuccinate synthetase (ASS1) activity is deficient, or from excessive L-citrulline supplementation overwhelming tubular reabsorption capacity.

Clinical Significance: Citrullinuria is the hallmark urinary finding in citrullinaemia type I (OMIM #215700, ASS1 deficiency), a urea cycle disorder where citrulline cannot be condensed with aspartate. Markedly elevated urinary citrulline alongside elevated urinary orotic acid and hyperammonaemia confirms this diagnosis. Citrin deficiency (citrullinaemia type II, OMIM #603471) in adults presents with elevated circulating citrulline and citrullinuria with characteristic dietary preferences for protein and fat over carbohydrate. In OTC deficiency (which causes low urinary citrulline), urinary orotic acid is massively elevated — useful for distinguishing these disorders. Mild citrullinuria without hyperammonaemia most commonly reflects L-citrulline or watermelon extract supplementation.

Suggested Treatment: Urgently quantify urinary orotic acid and ammonia. Distinguish citrullinaemia (high citrulline + elevated orotic acid) from OTC deficiency (low citrulline + very high orotic acid). Confirm with ASS1 or SLC25A13 gene testing. Review citrulline supplementation dose. Manage confirmed citrullinaemia with protein restriction and arginine supplementation under specialist metabolic care.

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1-METHYLHISTIDINE LOW:

Description: Urinary 1-methylhistidine is derived exclusively from the hydrolysis and urinary excretion of dietary anserine (from poultry and fish). It is quantitatively excreted in urine and not metabolised further, making it a highly specific and quantitative biomarker of poultry and fish consumption.

Clinical Significance: Low urinary 1-methylhistidine confirms low or absent recent consumption of chicken, turkey, or fish. It is a dietary biomarker only, with no metabolic or pathological significance. Low values in conjunction with clinical assessment of dietary adequacy may highlight insufficient poultry and fish intake, which also means lower anserine availability for intracellular pH buffering and antioxidant protection.

Suggested Treatment: No clinical intervention required. If increased carnosine-type dipeptide benefits are desired, increase poultry and fish consumption. Note timing of last poultry or fish meal when interpreting. Beta-alanine supplementation can provide some of the carnosine precursor benefits.

HYDROXYPROLINE ELEVATED:

Description: Elevated urinary hydroxyproline is a classical marker of increased bone resorption and collagen catabolism, reflecting release of hydroxyproline from collagen triple helix degradation by matrix metalloproteinases (MMPs) and osteoclast-derived cathepsins.

Clinical Significance: Markedly elevated urinary hydroxyproline occurs in Paget's disease of bone (reflecting extremely high bone turnover), hyperparathyroidism (primary or secondary), hyperthyroidism, osteoporosis with high bone resorption, inflammatory arthritis (rheumatoid arthritis, psoriatic arthritis), and malignancy with skeletal metastases. High dietary gelatine or bone broth/collagen hydrolysate intake significantly elevates urinary hydroxyproline and must be excluded before interpreting the result as endogenous collagen catabolism. The hydroxyproline-to-creatinine ratio corrects for urine concentration.

Suggested Treatment: Evaluate bone turnover markers (CTX, P1NP), parathyroid hormone, calcium, thyroid function, and skeletal imaging. Restrict dietary gelatine and collagen supplements for 48 hours before repeat collection. Treat underlying bone or inflammatory pathology identified.

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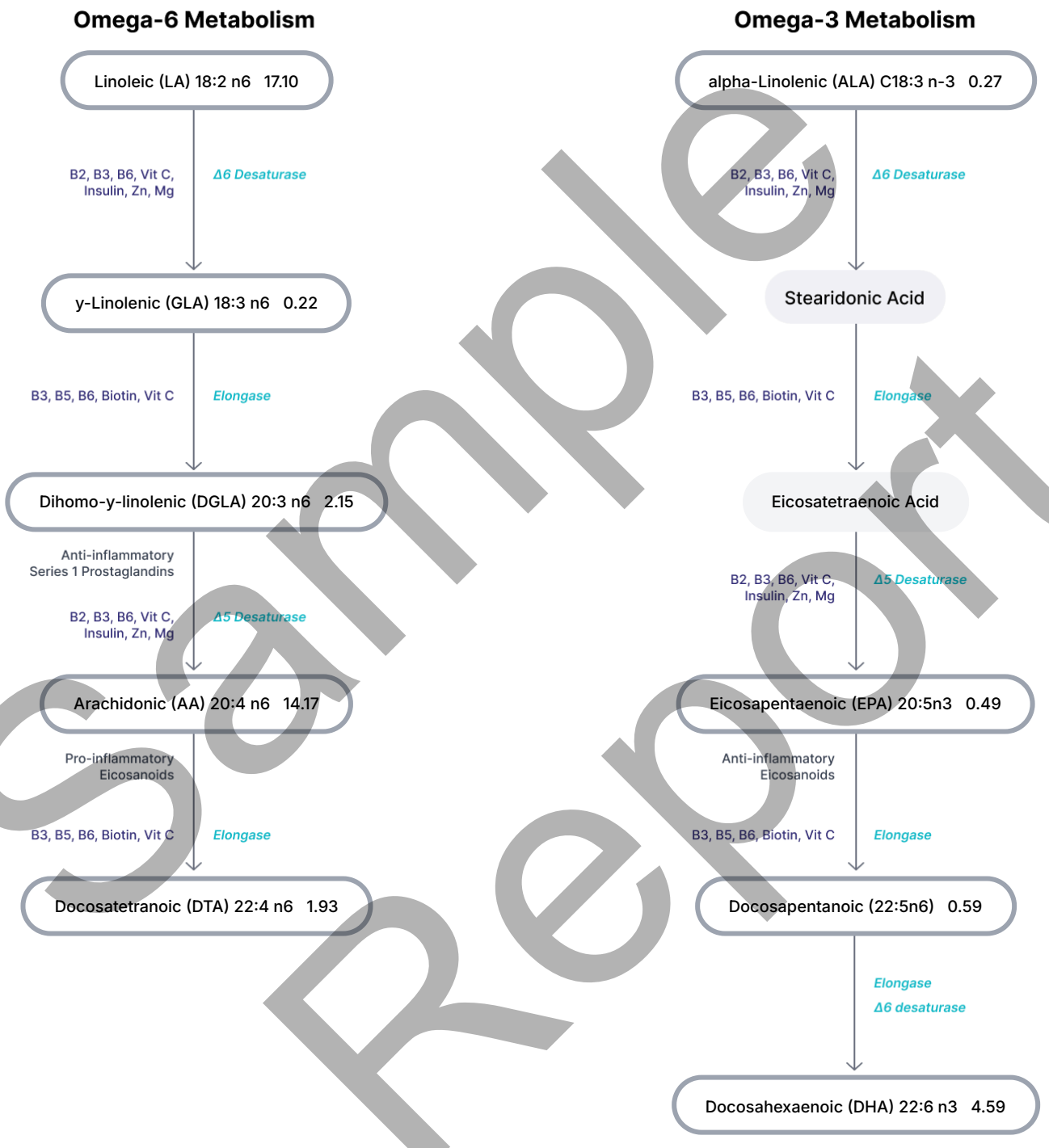
Legend Not Tested | Within Range | Out of Range | L = Low, LL = Critically Low | H = High, HH = Critically High | Regulator | Enzyme

Essential Fatty Acids Pathway



Analysed essential fatty acids, with molecular structure	Analyte with molecular structure shorthand	Analyte with molecular structure shorthand
	Linoleic acid (18:2 n-6)	α-Linolenic acid (18:3 n-3)
	γ-Linolenic acid (18:3 n-6)	Eicosatetraenoic acid (20:4 n-3)
	Dihomo-γ-linolenic acid (20:3 n-6)	Eicosapentaenoic acid (20:5 n-3)
	Arachidonic acid (20:4 n-6)	Docosapentaenoic acid (22:5 n-3)
	Docosatetraenoic acid (22:4 n-6)	Docosahexaenoic acid (22:6 n-3)

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Clinical Laboratory Improvement Amendments

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RED CELL FATTY ACIDS SUMMARY

TEST	RESULT	H/L	REFERENCE	UNITS
Saturated Fats, Total	37.36		(29.89-42.10)	%
Monounsaturated Fats, Total	18.57		(15.65-31.82)	%
Omega 3 Fatty Acids, Total	7.00		(2.57-15.15)	%
Omega 6 Fatty Acids, Total	36.36		(24.85-44.15)	%
Omega 3/Omega 6 Ratio	0.19		(0.07-5.30)	ratio
AA/EPA Ratio	28.92		(1.10-30.00)	ratio
Omega 3 Index	6.89		(>4.00)	%
Delta 6 Desaturase Activity (LA/DGLA)	7.95		(6.00-25.00)	ratio

OMEGA 3 FATTY ACIDS

TEST	RESULT	H/L	REFERENCE	UNITS
alpha-Linolenic (ALA) C18:3 n-3	0.27		(0.10-1.90)	%
Eicosapentaenoic (EPA) 20:5n3	0.49		(0.14-6.92)	%
Docosapentaenoic (DPA) 22:5 n3	1.65		(0.53-2.81)	%
Docosahexaenoic (DHA) 22:6 n3	4.59		(1.00-6.50)	%
Total Omega 3 Fatty Acids	7.00		(2.57-15.15)	%

OMEGA 6 FATTY ACIDS

TEST	RESULT	H/L	REFERENCE	UNITS
Linoleic (LA) 18:2 n6	17.10		(14.00-31.30)	%
y-Linolenic (GLA) 18:3 n6	0.22		(0.05-0.72)	%
Eicosadienoic 20:2 n6	0.20		(0.10-0.43)	%
Dihomo-y-linolenic (DGLA) 20:3 n6	2.15		(0.50-2.50)	%
Arachidonic (AA) 20:4 n6	14.17		(5.00-14.80)	%
Docosatetranoic (DTA) 22:4 n6	1.93		(0.30-2.50)	%
Docosapentanoic (22:5n6)	0.59		(0.08-0.83)	%
Total Omega 6 Fatty Acids	36.36		(24.85-44.15)	%

MONOUNSATURATED FATTY ACIDS

TEST	RESULT	H/L	REFERENCE	UNITS
Palmitoleic 16:1 n7	0.69		(0.13-2.90)	%
Vaccenic 18:1 n7	0.53		(0.00-1.50)	%
Oleic 18:1 n9	15.58		(14.20-29.50)	%
Gondoic 20:1 n9	0.18		(0.10-0.77)	%
Nervonic 24:1 n9	1.59		(0.50-3.00)	%
Total Monounsaturated Fatty Acids	18.57		(15.65-31.82)	%
Total Omega 9 Fatty Acids	17.35		(16.00-27.00)	%

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SATURATED FATTY ACIDS

TEST	RESULT	H/L	REFERENCE	UNITS
Myristic C14:0	0.62		(0.10-2.45)	%
Pentadecylic C15:0	0.16		(0.05-0.30)	%
Palmitic C16:0	20.99		(17.50-27.10)	%
Margaric C17:0	0.35		(0.14-0.45)	%
Stearic C18:0	12.86		(8.40-15.00)	%
Arachidic C20:0	0.18		(0.10-0.53)	%
Behenic C22:0	0.57		(0.20-1.59)	%
Lignoceric C24:0	1.63		(0.20-1.92)	%
Total Saturated Fatty Acids	37.36		(29.89-42.10)	%

TRANS FATTY ACIDS

TEST	RESULT	H/L	REFERENCE	UNITS
Trans Palmitoleic 16:1 n-7t	0.12		(0.02-0.55)	%
Trans Oleic 18:1t	0.31		(0.00-0.51)	%
Trans Linoleic 18:2n6t	0.24		(0.07-0.92)	%
Trans Fatty Acids, Total	0.67		(0.30-2.02)	%
Trans Fat Index	0.55		(0.22-1.99)	%

Methodology

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

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