

RECEIVED
29-Aug-26

Clinical Notes .

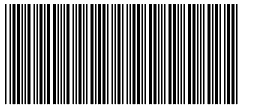
29-Aug-26

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

N/A

Kynurenic Acid
Quinolinic Acid
Xanthurenic Acid

N/A



26241-0004

Dr Test Doctor

Patient ID

Ext ID

Test Patient

Sex: Female • 56yrs • 01-Jan-70
123 Home Street, Test Suburb VIC 3125

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29-Aug-26

MICROBIAL OVERGROWTH

TEST		RESULT	H/L		REFERENCE	UNITS
1	Benzoic Acid	24.50	H	<div><div></div><div></div><div></div><div></div><div></div></div>	(<9.30)	mmol/molCR
2	Hippuric Acid	331.0		<div><div></div><div></div><div></div><div></div><div></div></div>	(<603.0)	mmol/molCR
3	4-Hydroxybenzoic Acid (4-HBA)	0.65	H	<div><div></div><div></div><div></div><div></div><div></div></div>	(<0.57)	mmol/molCR
4	3,4-Dihydroxybenzoic Acid (3,4-DHBA)	62.00	H	<div><div></div><div></div><div></div><div></div><div></div></div>	(<45.00)	mmol/molCR
5	3-Hydroxyphenylacetic Acid (3-HPAA)	12.00	H	<div><div></div><div></div><div></div><div></div><div></div></div>	(<10.00)	mmol/molCR
6	3,4-Dihydroxyphenylpropionic Acid (DHPPA)	2.90		<div><div></div><div></div><div></div><div></div><div></div></div>	(<5.30)	mmol/molCR
7	Furancarbonylglycine	2.80	H	<div><div></div><div></div><div></div><div></div><div></div></div>	(<2.00)	mmol/molCR

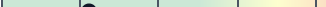
Amino Acid Metabolism

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
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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	18.50	H		(<10.00)	mmol/molCR
23	Furan-2,5-dicarboxylic Acid	12.90			(<15.00)	mmol/molCR
24	Tartaric Acid	11.80			(<15.00)	mmol/molCR

Fusarium

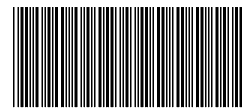
Rank	Compound	Score	Visual Scale	Threshold	Unit
25	Tricarballic Acid	0.21		(<0.44)	mmol/molCR

Patient ID P000060
Ext ID 26241-0004

Sex: Female • 56yrs • 01-Jan-70
123 Home Street, Test Suburb VIC 3125

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



26241-0004

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

Patient ID P000060
Ext ID 26241-0004

Test Patient

Sex: Female • 56yrs • 01-Jan-70
123 Home Street, Test Suburb VIC 3125

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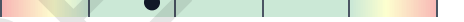
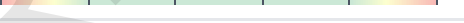
MITOCHONDRIAL & ENERGY METABOLISM

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

TEST	RESULT	H/L	REFERENCE	UNITS
Carbohydrate & Glycaemic Metabolism				
26	Glucose (OA)	0.98	<div><div></div><div></div><div></div><div></div><div></div></div> <div>(<1.10)</div>	ug/mgCR
27	Pyruvic Acid	1.60	<div><div></div><div></div><div></div><div></div><div></div></div> <div>(0.50-8.70)</div>	mmol/molCR
28	Lactic Acid	3.60	<div><div></div><div></div><div></div><div></div><div></div></div> <div>(<48.00)</div>	mmol/molCR
29	2-Hydroxybutyric Acid **	4.10	<div><div></div><div></div><div></div><div></div><div></div></div> <div>(<6.90)</div>	mmol/molCR

** An elevated result may indicate methylation defects and/or toxic exposure

Citric Acid Cycle

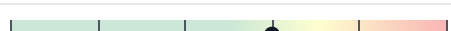
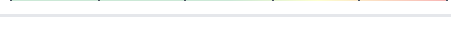
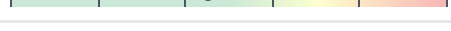
30	Citric Acid	280			(40-507)	mmol/molCR
31	cis-Aconitic Acid	12.70			(3.50-36.00)	mmol/molCR
32	Isocitric Acid	86.00	H		(5.00-65.00)	mmol/molCR
33	2-Oxoglutaric Acid	65.00	H		(2.00-45.00)	mmol/molCR
34	Succinic Acid	12.00			(1.00-15.00)	mmol/molCR
35	Fumaric Acid	4.30			(<8.60)	mmol/molCR
36	Malic Acid	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

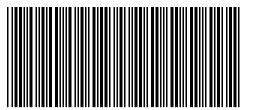
Mitochondrial Markers

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

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



26241-0004

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

Patient ID P000060
Ext ID 26241-0004

Test Patient

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PYRIMIDINE METABOLITES - Folate Metabolism

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

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NEUROTRANSMITTER METABOLITES

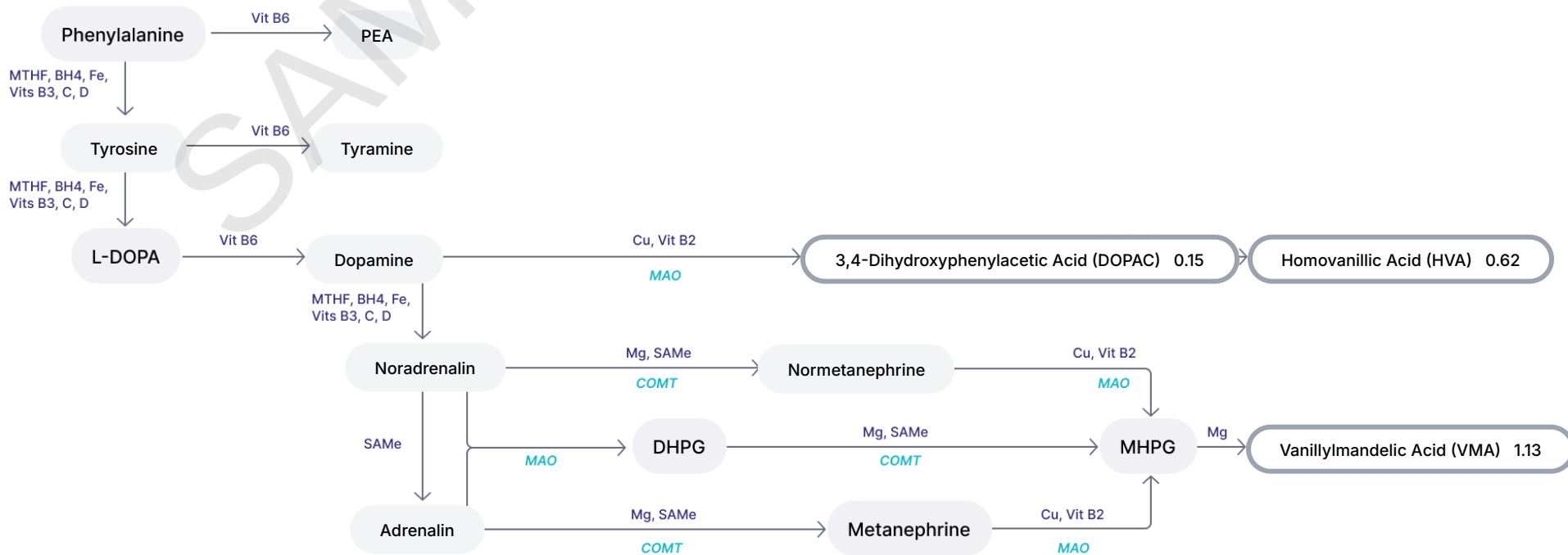
TEST		RESULT	H/L	REFERENCE	UNITS
Inhibitory (Serotonin)					
101	5-Hydroxyindoleacetic Acid (5-HIAA)	1.14	<div><div></div></div>	(<4.30)	mmol/molCR
Excitatory (Dopamine)					
102	3,4-Dihydroxyphenylacetic Acid (DOPAC)	0.15	<div><div></div></div>	(0.08-3.50)	mmol/molCR
103	3-Methyl-4-hydroxyphenylglycol (MHPG)	0.18	<div><div></div></div>	(0.05-0.50)	mmol/molCR
104	Homovanillic Acid (HVA)	0.62	<div><div></div></div>	(0.10-5.30)	mmol/molCR
105	Vanillylmandelic Acid (VMA)	1.13	<div><div></div></div>	(0.40-3.60)	mmol/molCR
106	HVA/DOPAC Ratio	4.1	<div><div></div></div>	(<10.0)	ratio
107	HVA/VMA Ratio	0.6	<div><div></div></div>	(<2.0)	ratio

ADRENAL STRESS (Overnight)

SERVICE	RESULT	H/L	REFERENCE	UNITS
108 Cortisol (OA)	8.0	<div><div></div></div>	(1.0-63.8)	ug/gCR

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Excitatory Pathway

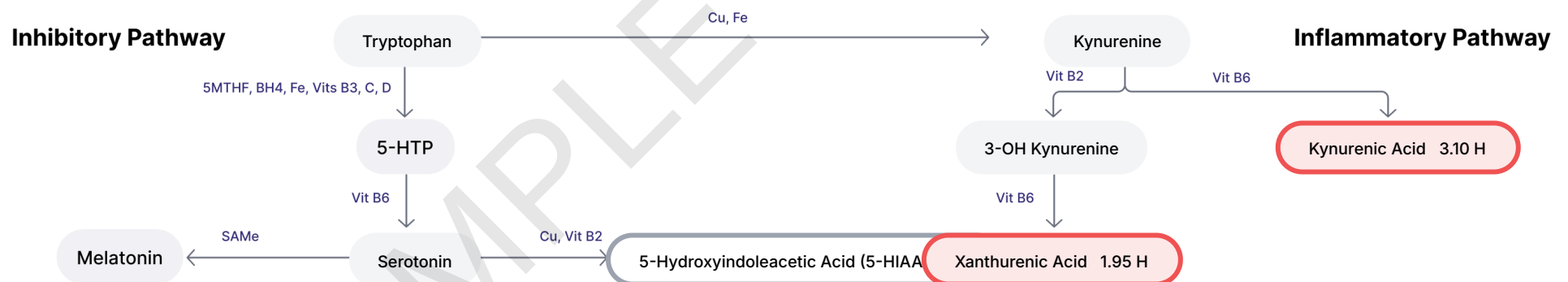


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(Tryptophan, B6, Antioxidants)

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

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



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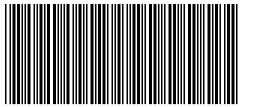
(Arg, NAC, Meth, Mg, Antioxidants)

OXIDATIVE STRESS & INFLAMMATION

(Vitamin C, Other Antioxidants)

OXALATE METABOLISM

[illegible]



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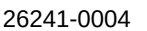
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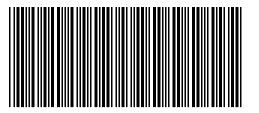
ENVIRONMENTAL / XENOBIOTIC EXPOSURE

TEST		RESULT	H/L		REFERENCE	UNITS
Toluene Exposure						
132	Hippuric Acid	331.0		<div><div></div><div></div><div></div><div></div><div></div><div></div><div></div></div>	(<603.0)	mmol/molCR
133	Benzoic Acid	24.50	H	<div><div></div><div></div><div></div><div></div><div></div><div></div><div></div></div>	(<9.30)	mmol/molCR
Paraben Exposure						
134	4-Hydroxybenzoic Acid (4-HBA)	0.65	H	<div><div></div><div></div><div></div><div></div><div></div><div></div><div></div></div>	(<0.57)	mmol/molCR
Styrene Exposure						
135	Mandelic Acid	188.0		<div><div></div><div></div><div></div><div></div><div></div><div></div><div></div></div>	(<340.0)	ug/gCR
136	Phenylglyoxylic Acid	106.0		<div><div></div><div></div><div></div><div></div><div></div><div></div><div></div></div>	(<300.0)	ug/gCR
137	Mandelic Acid + Phenylglyoxylic Acid	294.0		<div><div></div><div></div><div></div><div></div><div></div><div></div><div></div></div>	(<610.0)	ug/gCR
Benzene Exposure						
138	t,t-Muconic Acid	0.20	H	<div><div></div><div></div><div></div><div></div><div></div><div></div><div></div></div>	(<0.12)	mmol/molCR
Phthalate Exposure						
139	Phthalic Acid	149.00		<div><div></div><div></div><div></div><div></div><div></div><div></div><div></div></div>	(<170.00)	ug/gCR
140	Monoethyl Phthalate	60.30		<div><div></div><div></div><div></div><div></div><div></div><div></div><div></div></div>	(<100.00)	ug/gCR
141	Quinolinic Acid	11.80	H	<div><div></div><div></div><div></div><div></div><div></div><div></div><div></div></div>	(<9.10)	mmol/molCR
MTBE Exposure						
142	2-Hydroxylsobutyric Acid	3.90		<div><div></div><div></div><div></div><div></div><div></div><div></div><div></div></div>	(<6.90)	mmol/molCR
Xylene Exposure						
143	2-Methylhippuric Acid	0.01		<div><div></div><div></div><div></div><div></div><div></div><div></div><div></div></div>	(<0.04)	mmol/molCR
144	3-Methylhippuric Acid	0.32	H	<div><div></div><div></div><div></div><div></div><div></div><div></div><div></div></div>	(<0.11)	mmol/molCR
145	4-Methylhippuric Acid	1.69		<div><div></div><div></div><div></div><div></div><div></div><div></div><div></div></div>	(<1.80)	mmol/molCR
Trimethylbenzene Exposure						
146	3,4-Dimethylhippuric Acid	0.00		<div><div></div><div></div><div></div><div></div><div></div><div></div><div></div></div>	(<0.01)	mmol/molCR
147	4-Hydroxyhippuric Acid	6.23		<div><div></div><div></div><div></div><div></div><div></div><div></div><div></div></div>	(<16.50)	mmol/molCR
Nucleotide Turnover/Methylation						
148	5-Methylcytosine	68.00	H	<div><div></div><div></div><div></div><div></div><div></div><div></div><div></div></div>	(10.00-50.00)	mmol/molCR
149	Uracil	8.10		<div><div></div><div></div><div></div><div></div><div></div><div></div><div></div></div>	(<9.00)	mmol/molCR
150	Thymine	0.42		<div><div></div><div></div><div></div><div></div><div></div><div></div><div></div></div>	(<0.60)	mmol/molCR
TEST		RESULT	H/L		REFERENCE	UNITS
151	Creatinine, Urine	5.40		<div><div></div><div></div><div></div><div></div><div></div><div></div><div></div></div>	(2.47-19.20)	mmol/L



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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



26241-0004

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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.

Important Disclaimer

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.

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

Nutritional Guide Practitioner Notes

BENZOIC ACID ELEVATED (URINE):

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

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

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.



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

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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.

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:

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

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



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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.

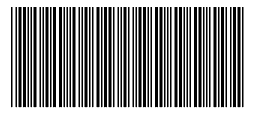
2-OXO-4-METHYLTHIOBUTYRIC ACID ELEVATED:

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.



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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.

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.

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 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.

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 (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.

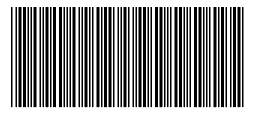
Elevated urinary GAA may indicate impaired conversion of guanidinoacetate to creatine, which can occur in the context of guanidinoacetate methyltransferase (GAMT) deficiency, a rare inherited metabolic disorder. More commonly, elevated levels may reflect relative inefficiency in methylation-dependent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

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.

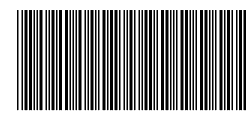
Clinically, elevated quinolinic acid may be associated with neurocognitive symptoms such as brain fog, mood disturbance, irritability, sleep disruption, or heightened pain sensitivity, although symptom expression varies.

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):



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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.

5-METHYLCYTOSINE ELEVATED:

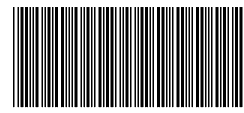
Urinary 5-methylcytosine (5-MC) is a modified nucleoside derived from DNA methylation and reflects global DNA methylation turnover and nucleic acid metabolism. Elevated levels may indicate increased DNA turnover, enhanced methylation flux, or increased degradation of methylated cytosine residues.

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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TOXIC METALS

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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.

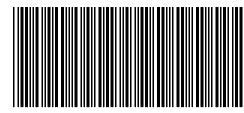
CADMIUM ELEVATED (URINE):

Elevated urinary cadmium suggests increased cadmium exposure and/or mobilisation with renal excretion. Urinary cadmium is considered a marker of chronic body burden rather than acute exposure.

Clinically, elevated cadmium exposure may be associated with renal tubular stress and bone mineral effects at higher or sustained levels. Smoking remains a common non-occupational source.

From a functional medicine perspective, this finding should prompt assessment of exposure sources (smoking, diet, occupation) and consideration of renal health and antioxidant status, with emphasis on exposure reduction rather than intervention unless clinically indicated.

Treatment: Remove exposure, support zinc and selenium (compete with cadmium), and consider chelation (e.g., EDTA or DMSA).



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ESSENTIAL AMINO ACIDS

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

CONDITIONALLY ESSENTIAL AMINO ACIDS

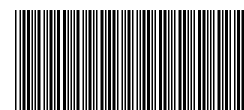
TEST		RESULT	H/L		REFERENCE	UNITS
11	Arginine	3.8		<div><div></div><div></div><div></div><div></div><div></div></div>	(0.0-8.0)	umol/gCR
12	Cysteine	11.8		<div><div></div><div></div><div></div><div></div><div></div></div>	(1.0-25.0)	umol/gCR
13	Glutamine	299.0	H	<div><div></div><div></div><div></div><div></div><div></div></div>	(2.0-100.0)	umol/gCR
14	Glycine	175.0		<div><div></div><div></div><div></div><div></div><div></div></div>	(40.0-800.0)	umol/gCR
15	Proline	119	H	<div><div></div><div></div><div></div><div></div><div></div></div>	(0-20)	umol/gCR
16	Taurine	177.0		<div><div></div><div></div><div></div><div></div><div></div></div>	(15.0-350.0)	umol/gCR
17	Tyrosine	35	H	<div><div></div><div></div><div></div><div></div><div></div></div>	(0-14)	umol/gCR

NON-ESSENTIAL AMINO ACIDS

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

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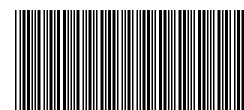
SERVICE		RESULT	H/L		REFERENCE	UNITS
40	Phenylalanine/Tyrosine	1.2		<div><div></div><div></div><div></div><div></div><div></div><div></div><div></div><div></div><div></div><div></div></div>	(<25.0)	ratio
41	Glutamate/Glutamine	1.03		<div><div></div><div></div><div></div><div></div><div></div><div></div>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



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TRYPTOPHAN ELEVATED:

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 BOAT1-mediated neutral amino acid reabsorption is impaired, causing broad aminoaciduria.

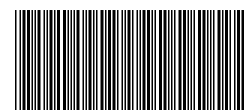
Clinical Significance: Pan-BCAA elevation in urine alongside keto acid derivatives (detectable on urine organic acids) is the hallmark of MSUD. In MSUD the characteristic sweet/maple syrup odour of urine is produced by the excreted keto acids. In Hartnup disorder (BOAT1/SLC6A19 deficiency), all neutral 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.

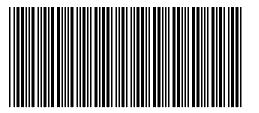
Clinical Significance: Elevated urinary glutamine as part of a broad aminoaciduria is characteristic of renal Fanconi syndrome (from Wilson's disease, cystinosis, tyrosinaemia type I, heavy metal toxicity, or antiretroviral therapy), where multiple proximal tubular transport functions are impaired. High-dose L-glutamine supplementation is the most common cause of elevated urinary glutamine in clinical practice



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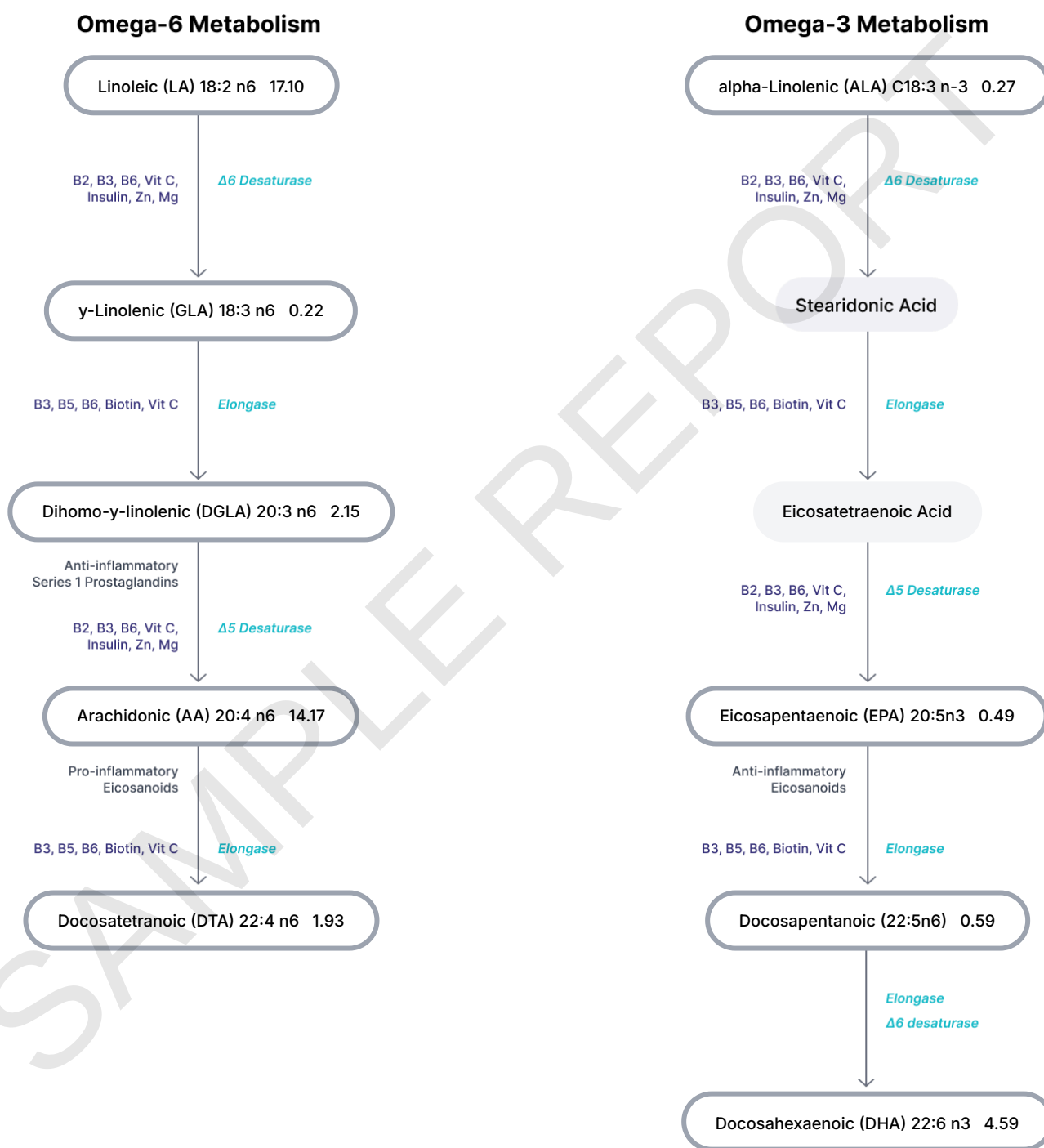
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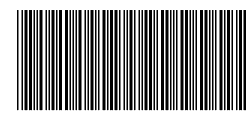
Legend Not Tested Within Range Out of Range L = Low, LL = Critically Low H = High, HH = Critically High Regulator Enzyme



- α-Linolenic acid (18:3 n-3)
- Eicosatetraenoic acid (20:4 n-3)
- Eicosapentaenoic acid (20:5 n-3)
- Docosapentaenoic acid (22:5 n-3)
- Docosahexaenoic acid (22:6 n-3)

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



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RED CELL FATTY ACIDS COMMENTS

LOW C18:1 n-9 (OLEIC ACID):

Oleic acid (C18:1 n-9) is a monounsaturated fat commonly found in olive oil, avocado, and many nuts. It is one of the most well-known healthy fats and has been associated with heart health. Oleic acid helps lower levels of LDL (bad) cholesterol while maintaining or increasing HDL (good) cholesterol. It also has anti-inflammatory properties and supports healthy cell function. Including sources of oleic acid in your diet is a good way to promote heart health and overall well-being.

ELEVATED TOTAL OMEGA 9 FATTY ACIDS:

Total omega-9 fatty acids, such as oleic acid, gondoic acid, and nervonic acid, are important monounsaturated fats that contribute to cardiovascular health, inflammation regulation, and cell membrane structure. Omega-9 fatty acids have been shown to lower LDL cholesterol and increase HDL cholesterol, reducing the risk of heart disease. Additionally, these fatty acids play key roles in metabolic health, supporting insulin sensitivity, and reducing inflammation. An elevated intake of omega-9 fatty acids is beneficial for long-term health and may help prevent chronic conditions such as cardiovascular disease, diabetes, and cognitive decline.

Methodology

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