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

Clinical Laboratory Improvement Amendments

26241-0007

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

Patient ID P000060
Ext ID 26241-0007

Test Patient

Sex: Female • 56yrs • 01-Jan-70

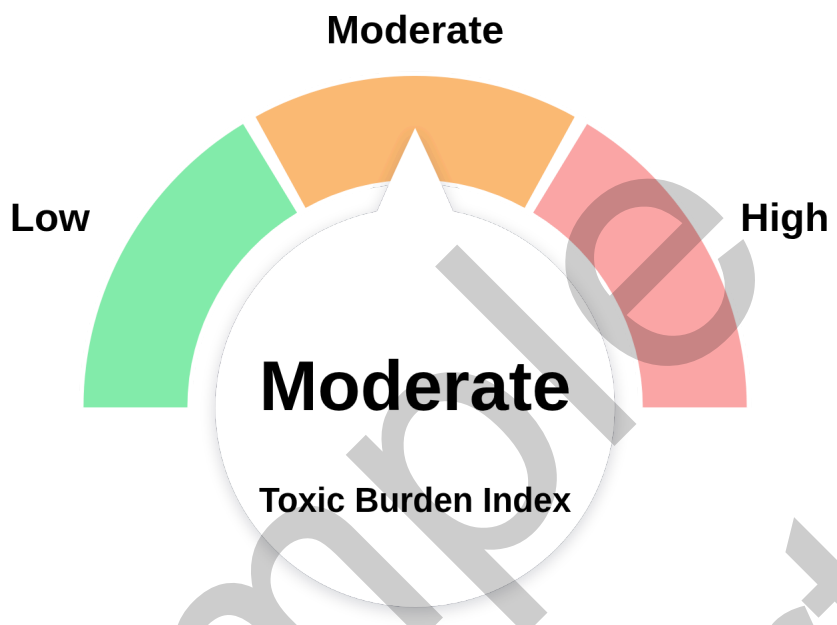
RECEIVED
29-Aug-26

Clinical Notes .

MOE-Tox Complete Environmental Toxins Profile

Specimen type - Urine, Spot

Collected
29-Aug-26



Toxic Burden Comment

Interpretation:

Moderate elevations may reflect increased exposure from environmental, dietary, or occupational sources. While concentrations are generally below levels associated with acute toxicity, cumulative effects may contribute to oxidative stress, mitochondrial dysfunction, or endocrine disruption in sensitive individuals. Management focuses on identifying and eliminating exposure sources, improving indoor air and water quality, and supporting hepatic and renal detoxification pathways. Clinical protocols may include antioxidants (vitamin C, selenium, CoQ10), glutathione or N-acetylcysteine to enhance detoxification, and binding agents such as activated charcoal or cholestyramine where indicated. Retesting after 3–6 months is advisable to confirm improvement.

Molds/Metals	Moderate Priority	Mineral Imbalance/Mitochondrial Dysfunction
Fumonisins B1	Cadmium	Manganese
Satratoxin G		Zinc
Zinc		2-Oxoisocaproic Acid
		3-Methyl-2-oxovaleric Acid
		Methylmalonic Acid (MMA) *

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

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AFLATOXINS GROUP

TEST	RESULT	H/L	REFERENCE	UNITS
Aflatoxin B1	1.33		(<3.50)	ng/gCR
Aflatoxin B2	0.62		(<5.30)	ng/gCR
Aflatoxin G1	<DL		(<2.20)	ng/gCR
Aflatoxin G2	<DL		(<6.50)	ng/gCR
Aflatoxin M1	<DL		(<4.70)	ng/gCR
Sterigmatocystin	<DL		(<0.40)	ng/gCR

GLIOTOXINS GROUP

TEST	RESULT	H/L	REFERENCE	UNITS
Gliotoxin derivative	127.00		(<180.00)	ng/gCR

OCHRATOXIN GROUP

TEST	RESULT	H/L	REFERENCE	UNITS
Ochratoxin A	0.89		(<2.20)	ng/gCR

TRICHOTHECENES GROUP - Type A

TEST	RESULT	H/L	REFERENCE	UNITS
Diacetoxyscirpenol	<DL		(<2.800)	ng/gCR
HT-2 Toxin	<DL		(<5.20)	ng/gCR
T-2 Toxin	<DL		(<0.500)	ng/gCR

TRICHOTHECENES GROUP - Type B

TEST	RESULT	H/L	REFERENCE	UNITS
Deoxynivalenol	<DL		(<15.00)	ng/gCR
Nivalenol	0.66		(<3.70)	ng/gCR

TRICHOTHECENES GROUP - Type D Macrocyclic

TEST	RESULT	H/L	REFERENCE	UNITS
Roridin A	1.12		(<5.50)	ng/gCR
Roridin E	0.34		(<3.20)	ng/gCR
Roridin H	<DL		(<3.90)	ng/gCR
Roridin L-2	<DL		(<5.70)	ng/gCR
Satratoxin G	0.46	H	(<0.30)	ng/gCR
Satratoxin H	<DL		(<0.40)	ng/gCR
Verrucarin A	0.93		(<1.60)	ng/gCR
Verrucarin J	<DL		(<2.50)	ng/gCR

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ZEARALENONE GROUP

TEST	RESULT	H/L	REFERENCE	UNITS
Zearalenone	0.84		(<1.00)	ng/gCR

MYCOTOXINS GROUP - Other

TEST	RESULT	H/L	REFERENCE	UNITS
Alternariol	0.42		(<1.50)	ng/gCR
Chaetoglobosin A	<DL		(<9.80)	ng/gCR
Citrinin	<DL		(<5.20)	ng/gCR
Enniatin B1	0.16		(<0.50)	ng/gCR
Fumonisins B1	3.15	H	(<2.20)	ng/gCR
Fumonisins B2	2.21		(<2.50)	ng/gCR
Fumonisins B3	0.60		(<4.20)	ng/gCR
Mycophenolic Acid	<DL		(<2.50)	ng/gCR
Patulin	<DL		(<7.50)	ng/gCR

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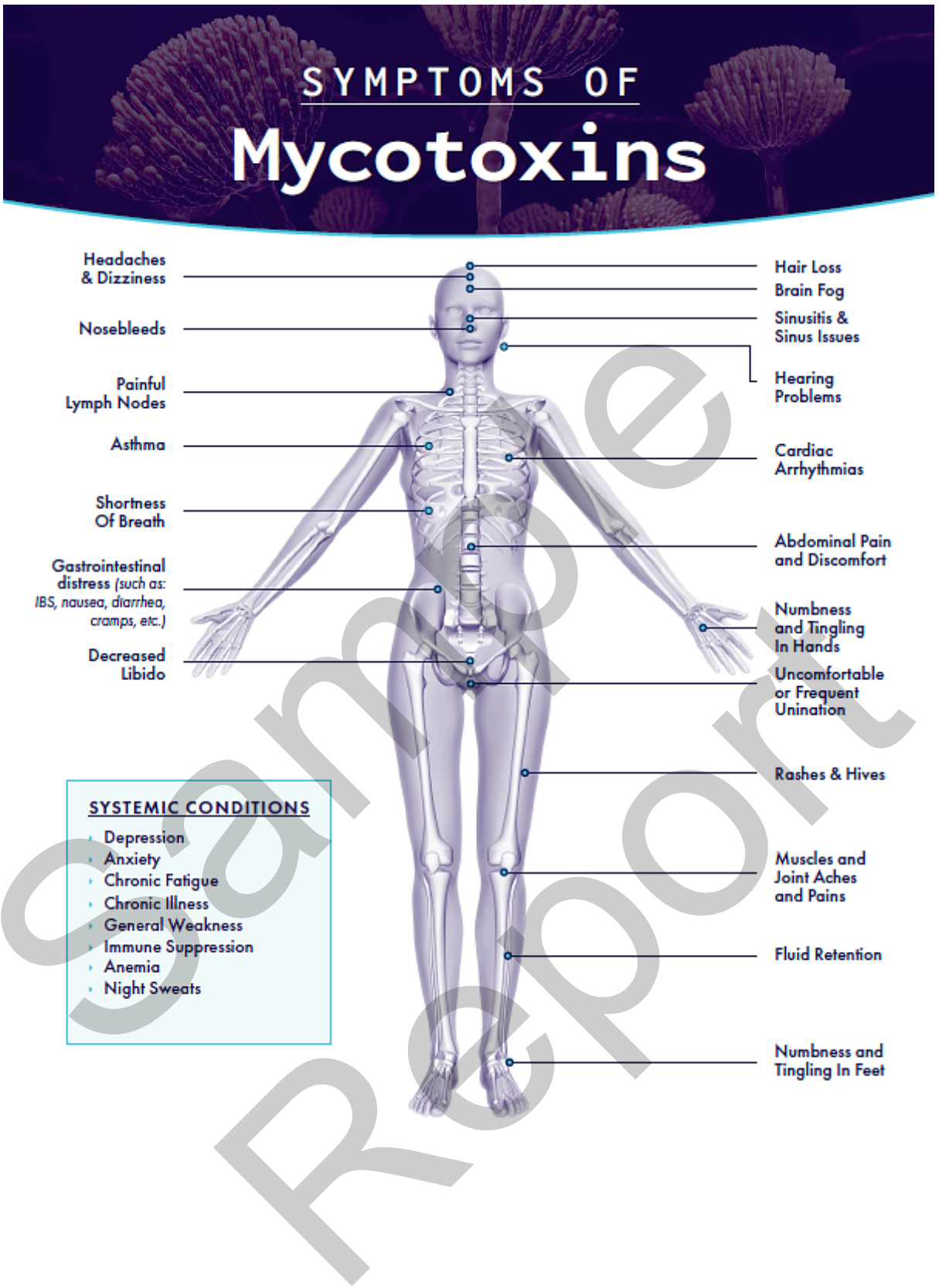

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MYCOTOXIN	Cellular Activity of Mycotoxins	Symptoms/Other	Association with a "Disease State"
AFLATOXINS			
Aflatoxin B1	Binds DNA and RNA replication	Shortness of breath, Weight loss, most potent and highly carcinogenic	Primarily attacks the liver, other organs include kidneys and lungs
Aflatoxin B2	Inhibits DNA and RNA replication	Impaired fetal growth	Affects the liver and kidneys
Aflatoxin G1	Cytotoxic, induces apoptosis in cells, DNA damage	A. flavus is a leading cause of invasive aspergillus in immune compromised patients	Cancer, Neonatal jaundice
Aflatoxin G2	Cancer, Neonatal jaundice	Aflatoxicosis in humans and animals	Malnutrition, Lung cancer
OCHRATOXINS			
Ochratoxin A	Inhibits mitochondrial ATP, potent teratogen, immune suppressor	Fatigue, Dermatitis, Irritated bowel	Kidney disease, Cancer
MACROCYCLIC	**TRICHOTHECENES**		
Satratoxin G	DNA, RNA and Protein synthesis inhibition	Fatigue	Bleeding disorders, Nervous system disorders
Satratoxin H	Inhibits protein synthesis	Fatigue	Breathing issues
Isosatratoxin F	Immunosuppression	Weakened immune system	-
Roridin A	Immunosuppression	Weakened immune system	-
Roridin E	DNA, RNA and Protein synthesis disruption	Weakened immune system	Lung and Nasal olfactory problems
Roridin H	Inhibits protein synthesis	Weakened immune system	-
Roridin L-2	Immunosuppression	Weakened immune system	-
Verrucarin A	Immunosuppression	-	-
Verrucarin J	Immunosuppression	-	-
GLIOTOXIN	**DERIVATIVE**		
Gliotoxin	Attacks intracellular function in immune system	Memory and Breathing issues	Immune Dysfunction disorders
ZEARALENONE			
Zearalenone	Estrogen mimicry	Early puberty, low sperm counts, cancer	Cancer

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PHYSIOLOGICAL MINERALS

TEST	RESULT	H/L	REFERENCE	UNITS
Calcium	127.80		(<450.00)	mg/gCR
Iron	75		(<80)	ug/gCR
Magnesium	102.94		(<290.00)	mg/gCR
Zinc	2000.00	H	(<900.00)	ug/gCR

TRACE MINERALS

TEST	RESULT	H/L	REFERENCE	UNITS
Boron	849		(<5500)	ug/gCR
Chromium	<DL		(<4.60)	ug/gCR
Cobalt	0.06		(<2.00)	ug/gCR
Copper	11.9		(<63.6)	ug/gCR
Germanium	<DL		(<1.50)	ug/gCR
Iodine	489.00		(>100.00)	ug/L
Lithium	11.04		(<130.00)	ug/gCR
Manganese	5.00	H	(<1.65)	ug/gCR
Molybdenum	14.45		(<122.00)	ug/gCR
Nickel	0.50		(<5.00)	ug/gCR
Rubidium	336		(<3000)	ug/gCR
Selenium	24.43		(10.00-111.00)	ug/gCR
Strontium	132.72		(<310.00)	ug/gCR
Vanadium	0.65		(<16.00)	ug/gCR

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TOXIC METALS

TEST	RESULT	H/L	REFERENCE	UNITS
Aluminium	10.84		(<40.00)	ug/gCR
Antimony	<DL		(<4.90)	ug/gCR
Arsenic	28.44		(<35.00)	ug/gCR
Barium	0.98		(<9.50)	ug/gCR
Beryllium	0.07		(<0.60)	ug/gCR
Bismuth	<DL		(<1.00)	ug/gCR
Bromine	807		(<7500)	ug/gCR
Cadmium	3.72	H	(<2.98)	ug/gCR
Gadolinium	1.14	H	(<0.50)	ug/gCR
Gallium	<DL		(<0.10)	ug/gCR
Lead	0.92		(<38.50)	ug/gCR
Mercury	0.2		(<17.7)	ug/gCR
Palladium	<DL		(<15.00)	ug/gCR
Platinum	2.65	H	(<1.00)	ug/gCR
Silver	<DL		(<0.10)	ug/gCR
Tellurium	<DL		(<0.80)	ug/gCR
Thallium	0.06		(<1.50)	ug/gCR
Tin	<DL		(<4.00)	ug/gCR
Titanium	<DL		(<50.00)	ug/gCR
Tungsten	<DL		(<0.50)	ug/gCR
Uranium	<DL		(<0.30)	ug/gCR
Zirconium	<DL		(<5.00)	ug/gCR

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CYSTEINE DERIVATIVES

TEST	RESULT	H/L	REFERENCE	UNITS
N-Acetyl (3,4-Dihydroxybutyl) cysteine (NADB)	<DL		(<250.00)	ug/gCR
N-Acetyl (carbomoylethyl) cysteine	9.66		(<190.00)	ug/gCR
N-Acetyl phenyl cysteine (SPMA)	<DL		(<5.00)	ug/gCR
N-Acetyl (propyl) cysteine (NAPR)	0.76		(<25.00)	ug/gCR

ENVIRONMENTAL PHENOLS

TEST	RESULT	H/L	REFERENCE	UNITS
4-Nonylphenol	<DL		(<3.00)	ug/gCR
Bisphenol A (BPA)	0.48		(<4.00)	ug/gCR
Bisphenol S (BPS)	0.79		(<0.80)	ug/gCR
Triclosan (TCS)	2.64		(<50.00)	ug/gCR

HERBICIDES (Synthetic Auxins)

TEST	RESULT	H/L	REFERENCE	UNITS
2,4-Dichlorophenoxyacetic acid (2,4-D)	<DL		(<1.00)	ug/gCR

HERBICIDES (Photosynthetic Inhibitors)

TEST	RESULT	H/L	REFERENCE	UNITS
Atrazine	<DL		(<0.50)	ug/gCR
Atrazine mercapturate	<DL		(<0.50)	ug/gCR

HERBICIDES (EPSP Inhibitors)

TEST	RESULT	H/L	REFERENCE	UNITS
Aminomethylphosphonic Acid (AMPA)	<DL		(<2.00)	ug/gCR
Glyphosate	0.3		(<1.0)	ppb

METHYLTERT-BUTYL ETHER (MTBE) EXPOSURE

TEST	RESULT	H/L	REFERENCE	UNITS
2-Hydroxylsobutyric Acid	0.26		(<2.98)	mmol/molCR

MITOCHONDRIAL MARKERS

TEST	RESULT	H/L	REFERENCE	UNITS
Tiglylglycine	0.08		(<1.75)	ug/gCR

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PARABENS

TEST	RESULT	H/L	REFERENCE	UNITS
Benzylparaben	0.07		(<2.00)	ug/gCR
Butylparaben	0.08		(<1.00)	ug/gCR
Ethylparaben	0.49		(<7.00)	ug/gCR
Methylparaben	18.40		(<120.00)	ug/gCR
4-Hydroxybenzoic Acid (4-HBA)	0.08		(<0.57)	mmol/molCR
Propylparaben	0.65		(<35.00)	ug/gCR

PESTICIDES

TEST	RESULT	H/L	REFERENCE	UNITS
3-Phenoxybenzoic Acid (3PBA)	<DL		(<3.00)	ug/gCR
Diethyl Phosphate (DEP)	<DL		(<9.00)	ug/gCR
Diethyldithiophosphate (DEDTP)	0.01		(<0.20)	ug/gCR
Diphenyl phosphate (DPP)	0.33		(<2.50)	ug/gCR
Diethylthiophosphate (DETP)	<DL		(<1.00)	ug/gCR

PFA's

TEST	RESULT	H/L	REFERENCE	UNITS
Perfluorobutanoic acid (PFBA)	0.29		(<1.20)	ug/gCR
Perfluorooctanoic Acid (PFOA)	<DL		(<0.10)	ug/gCR
Perfluorooctane Sulphonic Acid (PFOS)	<DL		(<0.60)	ug/gCR

PHTHALATES

TEST	RESULT	H/L	REFERENCE	UNITS
Butyl Benzyl phthalate (BBP)	0.12		(<1.00)	ug/gCR
Mono-Benzyl phthalate (mBzP)	1.77		(<3.00)	ug/gCR
Mono-n-Butyl phthalate (mBP)	4.97		(<55.00)	ug/gCR
Mono (3-carboxypropyl) phthalate (mCPP)	<DL		(<31.00)	ug/gCR
Mono-ethyl phthalate (MEtP)	12.11		(<100.00)	ug/gCR
Mono-2-ethylhexyl phthalate (MEHP)	<DL		(<11.00)	ug/gCR
Mono-(2-ethy-5-hydroxyhexyl) phthalate (MEHHP)	<DL		(<12.00)	ug/gCR
Mono-(2-ethy-5-oxohexyl) phthalate (MEOHP)	<DL		(<27.00)	ug/gCR
Mono-n-octyl phthalate (mOP)	<DL		(<2.00)	ug/gCR
Phthalic Acid	50.31		(<111.00)	ug/gCR
Quinolinic Acid	4.40	H	(<3.74)	mmol/molCR

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VOLATILE ORGANIC COMPOUNDS

TEST	RESULT	H/L	REFERENCE	UNITS
2-hydroxyethyl-mercapturic acid (HEMA)	<DL		(<5.00)	ug/gCR
Mandelic Acid	145.0		(<267.0)	ug/gCR
Phenylglyoxylic Acid	112.7		(<287.0)	ug/gCR
Mandelic Acid + Phenylglyoxylic Acid	257.7		(<610.0)	ug/gCR

BENZENES EXPOSURE

TEST	RESULT	H/L	REFERENCE	UNITS
t,t-Muconic Acid	0.01		(<0.12)	mmol/molCR
3,4-Dimethylhippuric Acid	0.00		(<0.01)	mmol/molCR

TOLUENE EXPOSURE

TEST	RESULT	H/L	REFERENCE	UNITS
Benzoic Acid	1.43		(<9.30)	mmol/molCR
Hippuric Acid	70.6		(<603.0)	mmol/molCR

XYLENES EXPOSURE

TEST	RESULT	H/L	REFERENCE	UNITS
2-Methylhippuric Acid	0.01		(<0.10)	mmol/molCR
3-Methylhippuric Acid	0.03		(<0.11)	mmol/molCR

TEST	RESULT	H/L	REFERENCE	UNITS
Creatinine, Urine	6.80		(2.47-19.20)	mmol/L

Disclaimer: This panel employs advanced analytical technologies for accurate compound detection. Environmental compounds and organic acids in urine are quantified using tandem mass spectrometry (LC-MS/MS), while minerals and metals are measured using inductively coupled plasma mass spectrometry (ICP-MS). Urine creatinine is determined via a kinetic colorimetric assay based on the Jaffé method, and all analyte concentrations are reported as quantitative values normalized to urine creatinine to account for differences in urine concentration. Test results should always be interpreted in the context of patient symptoms, clinical history, and other laboratory data. Individuals are advised to review their results with a qualified healthcare provider.

Mycotoxins Comment

TRICHOTHECENES GROUP ELEVATED (URINE):

Elevated urinary trichothecenes indicate exposure to highly cytotoxic mycotoxins produced by Fusarium, Stachybotrys, and related species. These compounds are potent inhibitors of protein synthesis and are commonly associated with water-damaged buildings and contaminated grains.

Clinically, trichothecene exposure is associated with prominent inflammatory and neurological symptoms, including headaches, fatigue, cognitive dysfunction, mucosal irritation, gastrointestinal disturbance, and immune dysregulation. Trichothecenes are considered among the more clinically aggressive mycotoxins.

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From a functional medicine perspective, management requires urgent identification and removal of environmental exposure, particularly from indoor mould sources. Supportive strategies may include inflammatory modulation, antioxidant support, gut barrier protection, and carefully titrated binders under clinical supervision. Due to their potency, treatment should be individualised and paced to avoid symptom exacerbation.

FUMONISIN B1 (FB1) ELEVATED:

Description: A neurotoxic and hepatotoxic mycotoxin from Fusarium species, typically contaminating corn. It disrupts sphingolipid metabolism and cellular membranes.

Symptoms: Neurocognitive symptoms, hepatic enzyme elevations, GI disturbances.

Treatment: Avoid corn-based products, methylation support (B12, folate), liver and neurological detox support.

Nutrient Minerals Comment

MANGANESE ELEVATED (URINE):

Elevated urinary manganese suggests increased manganese exposure and renal excretion. In a first-morning urine sample, this finding reflects recent exposure or mobilisation rather than tissue accumulation.

Clinically, elevated manganese exposure may be associated with non-specific neurological symptoms, fatigue, or mood changes at higher or sustained levels, although mild elevations are often asymptomatic.

From a functional medicine perspective, this result should be interpreted in the context of occupational or environmental exposure (e.g. welding fumes, industrial dust), hepatic function, and iron status, as iron deficiency may increase manganese absorption. Correlation with clinical history and, where indicated, blood manganese may assist interpretation.

Treatment: Identify and eliminate the source of manganese exposure, often in occupational settings like welding or battery manufacturing. In some cases, iron supplementation in conjunction with chelation therapy may lead to improvements in neurological symptoms that chelation alone does not provide.

ZINC ELEVATED (URINE):

Elevated urinary zinc suggests increased zinc excretion, which commonly occurs with supplementation, physiological stress, inflammation, or altered renal handling.

Clinically, excessive zinc loss over time may contribute to copper imbalance, particularly with sustained high zinc intake, although symptoms are often non-specific.

From a functional medicine perspective, this result should be interpreted in the context of supplementation practices, inflammatory status, and copper–zinc balance rather than as an isolated indicator of zinc excess. Correlation with serum zinc and serum copper may assist in assessing systemic balance where clinically appropriate.

Treatment: Evaluate source exposure; elimination through chelation therapy with agents like CaNa2EDTA or ascorbic acid, which form complexes that are then excreted.

Toxic Metals Comment

PLATINUM ELEVATED (URINE):

Elevated urinary platinum suggests increased platinum exposure and renal excretion. Potential sources include occupational contact, environmental exposure, or prior platinum-based chemotherapy.

Clinically, elevated platinum may be associated with hypersensitivity reactions or renal effects depending on exposure context and chronicity.

From a functional medicine perspective, this result should be interpreted in the context of treatment history, occupational exposure, renal function, and cumulative metal burden rather than as an isolated marker of toxicity.

Treatment: Support renal clearance, antioxidant protection (e.g., NAC, selenium), and avoid repeat exposure if possible.

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GADOLINIUM ELEVATED (URINE):

Elevated urinary gadolinium suggests recent exposure and renal excretion, most commonly related to prior administration of gadolinium-based contrast agents used in magnetic resonance imaging (MRI). In a first-morning urine sample, this finding reflects clearance following exposure rather than endogenous accumulation.

Clinically, urinary gadolinium may remain detectable for prolonged periods after contrast exposure, particularly in individuals with reduced renal clearance. The clinical significance of low-level persistence remains an area of ongoing research.

From a functional medicine perspective, this result should be interpreted in the context of recent imaging history, renal function, and overall exposure burden, rather than as a marker of toxicity in isolation.

Treatment: Avoid repeat exposure, use binders (e.g., modified citrus pectin, zeolites), and support glutathione metabolism.

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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MICROBIAL OVERGROWTH

4-Cresol
Furancarbonylglycine

YEAST/FUNGAL METABOLITES

Arabinitol

ENVIRONMENTAL EXPOSURES

Quinolinic Acid

MITOCHONDRIA/ENERGY

N/A

FATTY ACID / KETONE METABOLISM

Ethylmalonic Acid

CARBOHYDRATE / GLYCAEMIC METABOLISM

N/A

NEUROTRANSMITTER METABOLISM

Quinolinic Acid
Quinolinic Acid/5-HIAA Ratio

AMINO ACIDS METABOLISM

2-Oxobutyric Acid
2-Oxoisocaproic Acid
3-Methyl-2-oxovaleric Acid

VITAMINS / NUTRITIONAL MARKERS

Methylmalonic Acid (MMA) *
Quinolinic Acid

DETOXIFICATION / GLUTATHIONE FUNCTION

N/A

OXIDATIVE DAMAGE / INFLAMMATION

Quinolinic Acid

OXALATE METABOLISM

N/A

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ORGANIC ACIDS INTRODUCTION

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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MICROBIAL OVERGROWTH

	TEST	RESULT	H/L	REFERENCE	UNITS
1	Benzoic Acid	1.43		(<9.30)	mmol/molCR
2	Hippuric Acid	70.6		(<603.0)	mmol/molCR
3	4-Hydroxybenzoic Acid (4-HBA)	0.08		(<0.57)	mmol/molCR
4	3,4-Dihydroxybenzoic Acid (3,4-DHBA)	8.80		(<22.10)	mmol/molCR
5	3-Hydroxyphenylacetic Acid (3-HPAA)	3.92		(<5.46)	mmol/molCR
6	3,4-Dihydroxyphenylpropionic Acid (DHPPA)	0.71		(<1.62)	mmol/molCR
7	Furancarbonylglycine	1.43	H	(<1.10)	mmol/molCR
	Amino Acid Metabolism				
8	Phenylacetic Acid (PAA)	0.88		(<2.63)	mmol/molCR
9	Phenylpropionic Acid	0.03		(<0.10)	mmol/molCR
10	3-Phenyllactic Acid (3-PLA)	1.19		(<2.00)	mmol/molCR
11	2-Hydroxyphenylacetic Acid (2-HPAA)	0.56		(0.05-0.70)	mmol/molCR
12	4-Hydroxyphenyllactic Acid (4-HPLA)	1.21		(<3.50)	mmol/molCR
	SCFA Metabolism				
13	3-Hydroxypropionic Acid (3-HPA)	2.23		(<10.92)	mmol/molCR
	Clostridial Markers				
14	4-Cresol	1.78	H	(<1.65)	ug/mgCR
15	4-Hydroxyphenylacetic Acid (4-HPAA)	2.12		(<6.56)	mmol/molCR
16	Indoleacetic Acid (IAA)	3.76		(<5.00)	mmol/molCR
17	3-(3-Hydroxyphenyl)-3-hydroxypropionic Acid (HPHPA)	39.00		(<108.00)	mmol/molCR

YEASTS & FUNGAL METABOLITES

	TEST	RESULT	H/L	REFERENCE	UNITS
18	Arabinitol	15.9	H	(<12.7)	mmol/molCR
19	Arabinose	10.70		(<17.79)	mmol/molCR
20	Citramalic Acid	1.03		(<1.66)	mmol/molCR
21	3-Oxoglutaric Acid	0.21		(<0.50)	mmol/molCR
	Aspergillus Markers				
22	5-Hydroxymethyl-2-furoic Acid	9.80		(<12.30)	mmol/molCR
23	Furan-2,5-dicarboxylic Acid	7.98		(<9.61)	mmol/molCR
24	Tartaric Acid	1.87		(<4.50)	mmol/molCR
	Fusarium				
25	Tricarballylic Acid	0.11		(<0.44)	mmol/molCR

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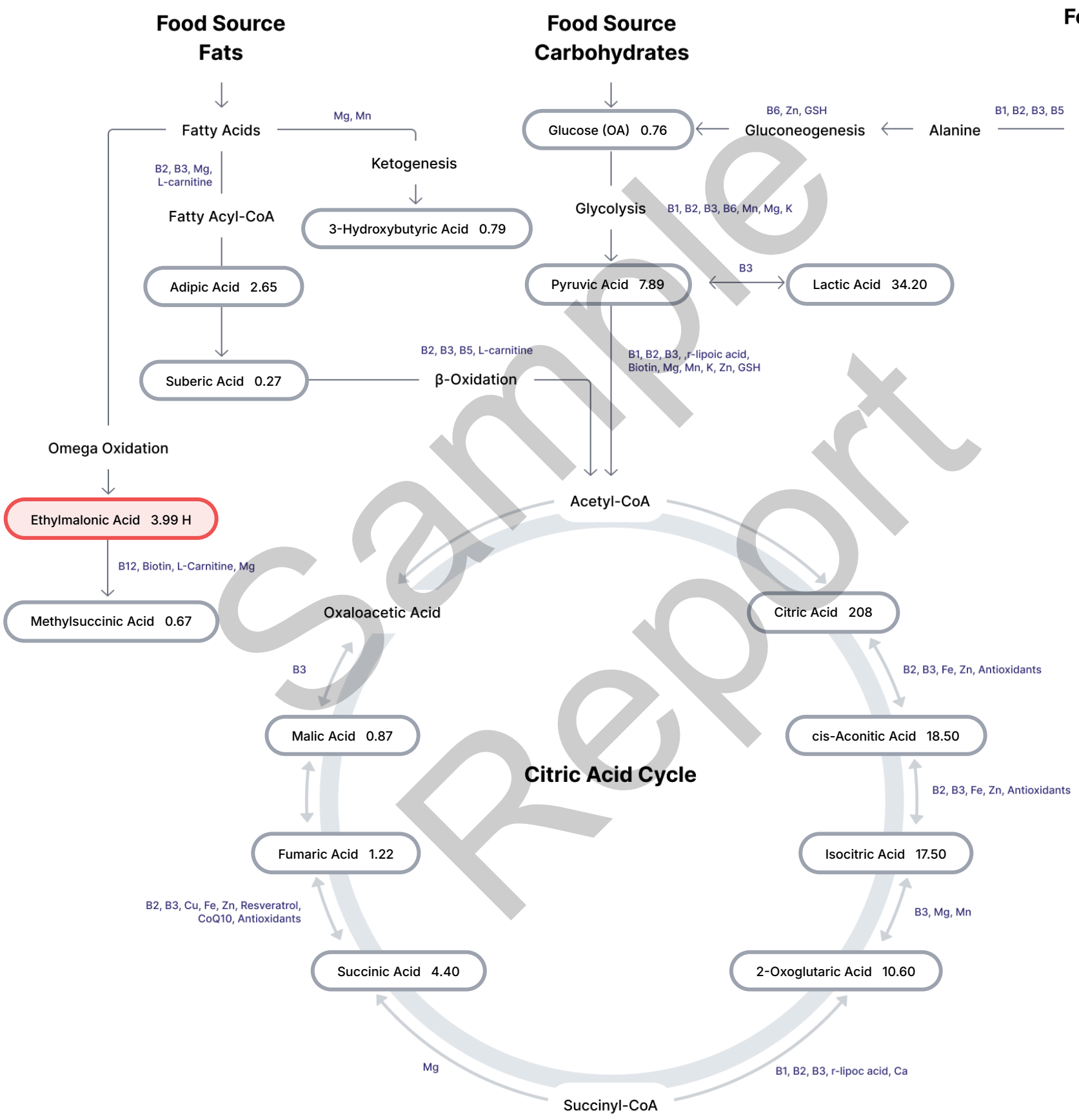
Organic Acids Pathway



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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)	0.76		(<1.10)	ug/mgCR
27	Pyruvic Acid	7.89		(0.50-9.10)	mmol/molCR
28	Lactic Acid	34.20		(<48.00)	mmol/molCR
29	2-Hydroxybutyric Acid **	3.12		(<4.06)	mmol/molCR
	** An elevated result may indicate methylation defects and/or toxic exposure				
	Citric Acid Cycle				
30	Citric Acid	208		(40-507)	mmol/molCR
31	cis-Aconitic Acid	18.50		(3.50-30.00)	mmol/molCR
32	Isocitric Acid	17.50		(5.00-49.60)	mmol/molCR
33	2-Oxoglutaric Acid	10.60		(2.00-45.00)	mmol/molCR
34	Succinic Acid	4.40		(0.70-15.00)	mmol/molCR
35	Fumaric Acid	1.22		(<4.00)	mmol/molCR
36	Malic Acid	0.87		(<1.21)	mmol/molCR
	Ketone/Energy Intermediates				
37	Acetoacetic Acid	1.23		(<4.08)	mmol/molCR
38	3-Hydroxybutyric Acid	0.79		(<3.10)	mmol/molCR
39	2-Hydroxybutyric Acid **	3.12		(<4.06)	mmol/molCR
	** An elevated result may indicate methylation defects and/or toxic exposure				
	Mitochondrial Markers				
40	Methylsuccinic Acid	0.67		(<3.50)	mmol/molCR
41	2-Methylglutaric Acid	0.12		(<0.45)	mmol/molCR
42	3-Methylglutaric Acid	1.42		(<6.00)	mmol/molCR
43	2-Hydroxyglutaric Acid	1.84		(<9.00)	mmol/molCR
44	3-Hydroxyglutaric Acid	0.54		(<2.07)	mmol/molCR
45	Malonic Acid	0.32		(<1.52)	mmol/molCR
46	Mevalonolactone	0.39		(<2.16)	mmol/molCR
47	2,4-Dihydroxybutanoic Acid	2.45		(<5.61)	mmol/molCR
48	N-Acetylaspartic Acid	3.87		(<14.20)	mmol/molCR
49	3-Methylglutaconic Acid	1.22		(<2.95)	mmol/molCR

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Clinical Laboratory Improvement Amendments

26241-0007

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FATTY ACID OXIDATION & KETONE METABOLISM

	TEST	RESULT	H/L	REFERENCE	UNITS
50	Adipic Acid	2.65		(<3.40)	mmol/molCR
51	Pimelic Acid	0.95		(<2.96)	mmol/molCR
52	Suberic Acid	0.27		(<1.16)	mmol/molCR
53	Azelaic Acid	1.52		(<3.98)	mmol/molCR
54	Sebacic Acid	0.09		(<0.33)	mmol/molCR
55	Ethylmalonic Acid	3.99	H	(<3.38)	mmol/molCR
56	Propionylglycine	0.46		(<0.82)	mmol/molCR
57	N-Butyrylglycine	0.42		(<1.27)	mmol/molCR
58	Isovalerylglycine	0.18		(<0.81)	mmol/molCR
59	N-(2-Methylbutyryl)glycine	0.09		(<0.69)	mmol/molCR
60	3-Methylcrotonylglycine	0.41		(<1.10)	mmol/molCR
61	Tiglylglycine	0.22		(<1.75)	mmol/molCR
62	Hexanoylglycine	0.71		(<5.40)	mmol/molCR
63	Suberylglycine	0.83		(<1.86)	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) *	1.88	H	(<1.60)	mmol/molCR
65	Formiminoglutamic Acid (FIGLU)	0.92		(<1.21)	mmol/molCR
66	Xanthurenic Acid	0.88		(<0.94)	mmol/molCR
67	Kynurenic Acid	1.34		(<1.57)	mmol/molCR
68	Quinolinic Acid	4.40	H	(<3.74)	mmol/molCR
69	Picolinic Acid	1.65		(<8.42)	mmol/molCR
70	3-Hydroxyisovaleric Acid	3.80		(<20.00)	mmol/molCR
	Vitamin-specific/Antioxidant Markers				
71	Pyroglutamic Acid *	11.00		(4.50-25.80)	mmol/molCR
72	N-Acetylcysteine (NAC)	0.13		(0.02-0.28)	mmol/molCR
73	Glutaric Acid (Vit B2) *	0.11		(0.02-0.36)	mmol/molCR
74	Pantothenic Acid (Vit B5)	2.02		(0.10-10.00)	mmol/molCR
75	Pyridoxic Acid (Vit B6)	5.89		(0.68-34.00)	mmol/molCR
76	Ascorbic Acid (Vit C)	17.20		(0.50-200.00)	mmol/molCR
77	Methylcitric Acid (Biotin/Vitamin H) *	2.90		(0.10-17.50)	mmol/molCR
78	3-Hydroxy-3-methylglutaric Acid (CoQ10) *	2.55		(0.10-5.00)	mmol/molCR

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PYRIMIDINE METABOLITES - Folate Metabolism

TEST		RESULT	H/L	REFERENCE	UNITS
79	Thymine	0.29		(<0.47)	mmol/molCR
80	Uracil	5.12		(<8.09)	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	0.71		(<3.55)	mmol/molCR
82	2-Oxoisocaproic Acid	0.92	H	(<0.46)	mmol/molCR
83	3-Methyl-2-oxovaleric Acid	1.31	H	(<1.22)	mmol/molCR
84	3-Methylglutaric Acid	1.42		(<6.00)	mmol/molCR
85	Succinylacetone	0.05		(<0.20)	mmol/molCR
Downstream Metabolites					
86	2-Hydroxyisovaleric Acid	1.12		(<3.10)	mmol/molCR
87	2-Hydroxyisocaproic Acid	0.32		(<1.21)	mmol/molCR
88	2-Oxobutyric Acid	3.05	H	(<2.98)	mmol/molCR
89	2-Oxo-4-methiolbutyric Acid (KMBA)	0.22		(<1.23)	mmol/molCR
Amino Acid Metabolism					
90	Phenylpyruvic Acid	0.66		(<1.54)	mmol/molCR
91	Homogentisic Acid	1.03	H	(<0.50)	mmol/molCR
92	N-Acetylphenylalanine	0.26		(<2.50)	mmol/molCR
93	Mandelic Acid	145.0		(<267.0)	ug/gCR
94	Malonic Acid	0.32		(<1.52)	mmol/molCR
95	4-Hydroxyphenyllactic Acid (4-HPLA)	1.21		(<3.50)	mmol/molCR
96	2-Oxoadipic Acid	0.32		(<1.50)	mmol/molCR
97	Guanidinoacetic Acid	1.09		(0.50-2.91)	mmol/molCR
98	Guanidinobutyric Acid	0.67		(0.10-1.00)	mmol/molCR
99	3-Methylglutaric Acid	1.42		(<6.00)	mmol/molCR
100	N-Acetylglycine	1.81		(<4.34)	mmol/molCR

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NEUROTRANSMITTER METABOLITES

(Phenylalanine, Tyrosine)

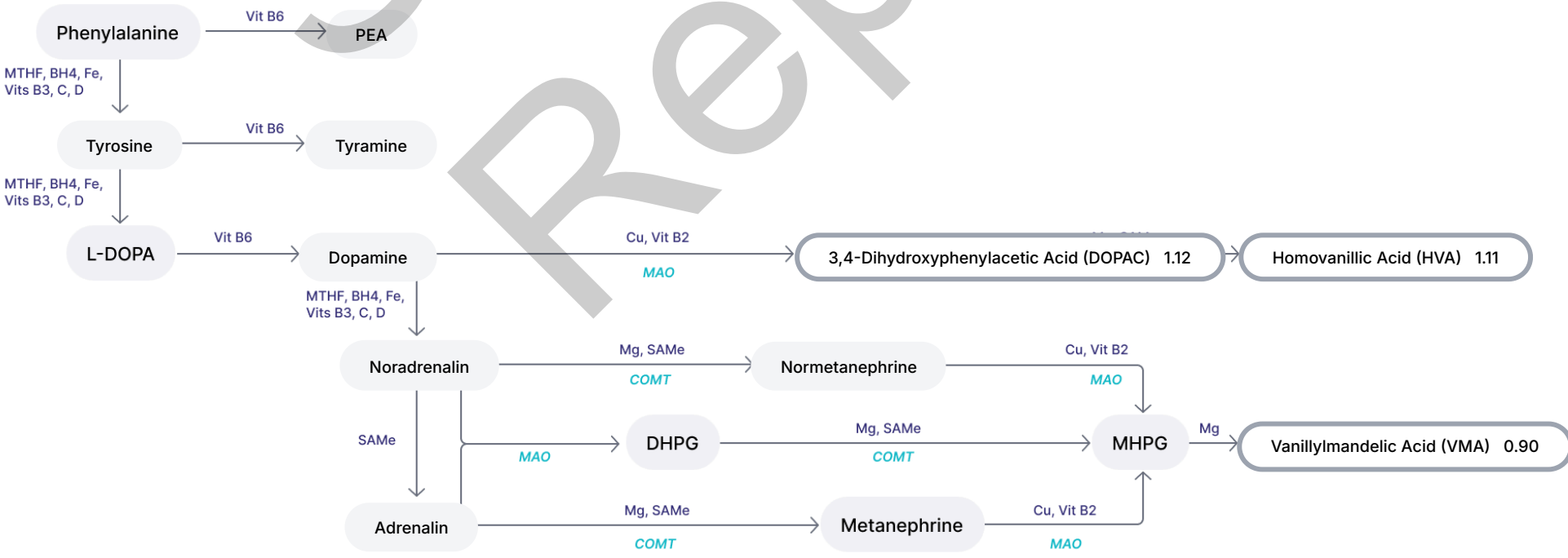
TEST	RESULT	H/L	REFERENCE	UNITS
Inhibitory (Serotonin)				
101 5-Hydroxyindoleacetic Acid (5-HIAA)	0.72		(<4.30)	mmol/molCR
Excitatory (Dopamine)				
102 3,4-Dihydroxyphenylacetic Acid (DOPAC)	1.12		(0.08-2.07)	mmol/molCR
103 3-Methyl-4-hydroxyphenylglycol (MHPG)	0.23		(0.05-0.50)	mmol/molCR
104 Homovanillic Acid (HVA)	1.11		(0.10-2.70)	mmol/molCR
105 Vanillylmandelic Acid (VMA)	0.90		(0.40-3.45)	mmol/molCR
106 HVA/DOPAC Ratio	1.0		(<4.1)	ratio
107 HVA/VMA Ratio	1.2		(<1.4)	ratio

ADRENAL STRESS (Overnight)

SERVICE	RESULT	H/L	REFERENCE	UNITS
108 Cortisol (OA)	18.4		(1.0-66.1)	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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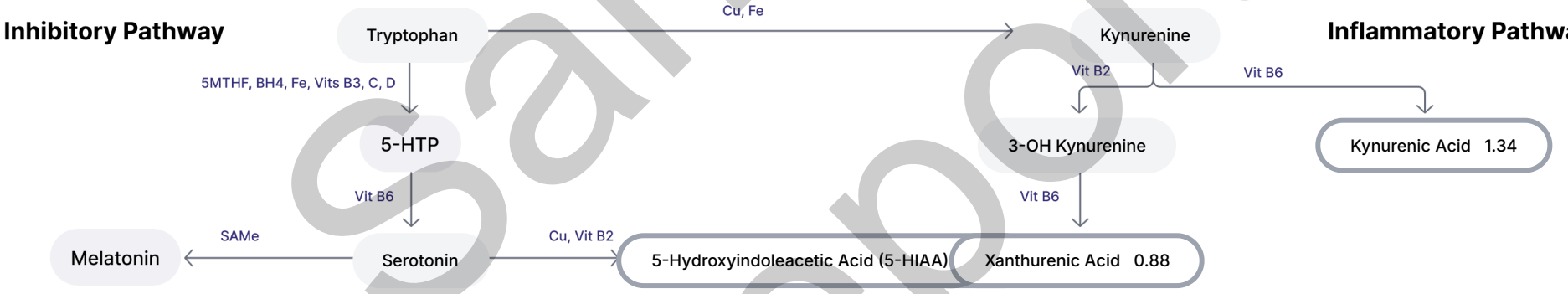
TRYPTOPHAN/KYNURENINE PATHWAY

(Tryptophan, B6, Antioxidants)

TEST	RESULT	H/L	REFERENCE	UNITS
Inhibitory				
109 5-Hydroxyindoleacetic Acid (5-HIAA)	0.72		(<4.30)	mmol/molCR
Inflammatory				
110 Kynurenic Acid	1.34		(<1.57)	mmol/molCR
111 Quinolinic Acid	4.40	H	(<3.74)	mmol/molCR
112 Picolinic Acid	1.65		(<8.42)	mmol/molCR
113 Xanthurenic Acid	0.88		(<0.94)	mmol/molCR
114 Kynurenic/Quinolinic Ratio	0.3		(<1.3)	ratio
115 Quinolinic Acid/5-HIAA Ratio	6.1	H	(<2.8)	ratio

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



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DETOXIFICATION & GLUTATHIONE FUNCTION

(Arg, NAC, Meth, Mg, Antioxidants)

TEST		RESULT	H/L	REFERENCE	UNITS
Ammonia Metabolism					
116	Orotic Acid	1.75		(<3.15)	mmol/molCR
Glutathione Metabolism					
117	Pyroglutamic Acid *	11.00		(4.50-25.80)	mmol/molCR
118	N-Acetylcysteine (NAC)	0.13		(0.02-0.28)	mmol/molCR
119	Glucaric Acid	1.02		(<4.06)	mmol/molCR
Phase I / Xenobiotic Markers					
120	2-Hydroxyhippuric Acid	0.95		(<1.38)	mmol/molCR
121	5-Hydroxymethyl-2-furoic Acid	9.80		(<12.30)	mmol/molCR
122	Furan-2,5-dicarboxylic Acid	7.98		(<9.61)	mmol/molCR

OXIDATIVE STRESS & INFLAMMATION

(Vitamin C, Other Antioxidants)

TEST		RESULT	H/L	REFERENCE	UNITS
123	8-hydroxy-deoxyguanosine	1.99		(<2.39)	umol/molCR
124	Leukotriene E4	3.60		(<10.00)	pg/mgCR
125	Quinolinic Acid	4.40	H	(<3.74)	mmol/molCR
126	Pyroglutamic Acid *	11.00		(4.50-25.80)	mmol/molCR
127	Ascorbic Acid (Vit C)	17.20		(0.50-200.00)	mmol/molCR
128	Quercetin	0.54		(0.01-2.00)	mmol/molCR

OXALATE METABOLISM

TEST		RESULT	H/L	REFERENCE	UNITS
129	Glycolic Acid	12.90		(<39.50)	mmol/molCR
130	Glyceric Acid	0.42		(<1.10)	mmol/molCR
131	Oxalic Acid	39.60		(<103.00)	mmol/molCR

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ENVIRONMENTAL / XENOBIOTIC EXPOSURE

TEST	RESULT	H/L	REFERENCE	UNITS
Toluene Exposure				
132 Hippuric Acid	70.6		(<603.0)	mmol/molCR
133 Benzoic Acid	1.43		(<9.30)	mmol/molCR
Paraben Exposure				
134 4-Hydroxybenzoic Acid (4-HBA)	0.08		(<0.57)	mmol/molCR
Styrene Exposure				
135 Mandelic Acid	145.0		(<267.0)	ug/gCR
136 Phenylglyoxylic Acid	112.7		(<287.0)	ug/gCR
137 Mandelic Acid + Phenylglyoxylic Acid	257.7		(<610.0)	ug/gCR
Benzene Exposure				
138 t,t-Muconic Acid	0.01		(<0.12)	mmol/molCR
Phthalate Exposure				
139 Phthalic Acid	50.31		(<111.00)	ug/gCR
140 Monoethyl Phthalate	28.00		(<167.00)	ug/gCR
141 Quinolinic Acid	4.40	H	(<3.74)	mmol/molCR
MTBE Exposure				
142 2-Hydroxylsobutyric Acid	0.26		(<2.98)	mmol/molCR
Xylene Exposure				
143 2-Methylhippuric Acid	0.01		(<0.10)	mmol/molCR
144 3-Methylhippuric Acid	0.03		(<0.11)	mmol/molCR
145 4-Methylhippuric Acid	0.01		(<1.74)	mmol/molCR
Trimethylbenzene Exposure				
146 3,4-Dimethylhippuric Acid	0.00		(<0.01)	mmol/molCR
147 4-Hydroxyhippuric Acid	1.10		(<13.10)	mmol/molCR
Nucleotide Turnover/Methylation				
148 5-Methylcytosine	13.10		(10.00-56.50)	mmol/molCR
149 Uracil	5.12		(<8.09)	mmol/molCR
150 Thymine	0.29		(<0.47)	mmol/molCR

TEST	RESULT	H/L	REFERENCE	UNITS
151 Creatinine, Urine	6.80		(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 *	Adequate	0.0	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

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Bacterial Dysbiosis Comment

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.

4-CRESOL ELEVATED (URINE):

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

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

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

Yeast Dysbiosis Comment

D-ARABINITOL ELEVATED (URINE):

D-arabinitol is a polyol produced predominantly by yeast species such as Candida. Elevation suggests increased yeast or fungal activity within the gastrointestinal tract.

Clinically, elevations may be associated with bloating, sugar cravings, fatigue, brain fog, or recurrent fungal symptoms. Results should be interpreted alongside bowel habits, dietary intake, medications, and the broader clinical context.

From a functional medicine perspective, management focuses on reducing fermentable sugars, addressing yeast overgrowth, supporting gut barrier integrity, and restoring microbial balance, addressing upstream gastrointestinal and metabolic contributors rather than the marker in isolation.

Consider: Anti-fungal therapy for Candida overgrowth; a microbiome assessment to determine overall fungal overgrowth.

Fatty Acid Oxidation & Ketone Metabolism

ETHYLMALONIC ACID ELEVATED (URINE):

Elevated urinary ethylmalonic acid suggests altered fatty acid metabolism, particularly involving short-chain and branched-chain fatty acid oxidation. Ethylmalonic acid accumulation may reflect mitochondrial inefficiency or altered electron transport activity.

Clinically, elevated ethylmalonic acid may be associated with fatigue, muscle weakness, reduced exercise tolerance, or non-specific metabolic symptoms, although symptom severity varies widely.

From an organic acid pattern perspective, elevated ethylmalonic acid may be observed alongside other markers of impaired fatty acid oxidation or mitochondrial stress, such as elevations in adipic or suberic acids, indicating reduced efficiency of beta-oxidation pathways.

From a functional medicine perspective, this finding should be interpreted in the context of mitochondrial function, riboflavin (vitamin B2) availability, and overall metabolic stress rather than as an isolated abnormality.

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Supplementation Recommendations: B complex (B2, B5), CoQ10L-Carnitine (may be contraindicated in patients on thyroid medications), L-Lysine (precursor to L-Carnitine), Other nutrients involved in Carnitine synthesis (Mg, SAMe, Vit B6, ascorbic acid, iron, niacin) Also: Glycine, Avoid medium chain fatty acids such as coconut oil.

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.

3-METHYL-2-OXOVALERIC ACID ELEVATED (URINE):

Elevated urinary alpha-keto-beta-methylvaleric acid suggests impaired isoleucine catabolism and increased metabolic stress on branched-chain amino acid pathways.

Clinically, elevated levels may be associated with fatigue, muscle symptoms, and reduced exercise tolerance.

From an organic acid pattern perspective, this elevation often clusters with other BCAA ketoacids, indicating shared cofactor insufficiency or mitochondrial overload.

From a functional medicine perspective, this finding should be interpreted alongside related BCAA markers and overall metabolic demand.

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

2-OXOBUTYRIC ACID ELEVATED:

α-Ketobutyric acid (α-KB) is an organic acid intermediate formed during the catabolism of amino acids, particularly threonine, methionine, and cystathionine, and is also linked to glutathione metabolism and transsulfuration pathways. It can be further metabolised to propionyl-CoA and subsequently succinyl-CoA, entering the tricarboxylic acid (TCA) cycle.

Elevated urinary α-ketobutyric acid may reflect increased amino acid catabolism, particularly involving methionine and threonine pathways, and may be observed in the context of metabolic stress or increased protein turnover. In addition, elevations may be associated with altered transsulfuration pathway activity, including increased flux related to glutathione synthesis or turnover.

From a biochemical perspective, accumulation of α-ketobutyric acid may also be seen in association with oxidative stress, where increased demand for glutathione influences pathway dynamics. However, this finding is non-specific and should be interpreted in conjunction with other markers of amino acid metabolism, oxidative stress, and mitochondrial function.

Suggested Treatment Considerations:

Consider correlation with other markers of transsulfuration and glutathione metabolism. Assessment of nutritional status, including amino acid intake and relevant cofactors, may be appropriate where clinically indicated. Evaluation of metabolic stressors and oxidative balance may also be considered. Management should be guided by the overall clinical context and associated laboratory findings.

Vitamin & Nutritional Cofactor Markers

METHYLMALONIC ACID ELEVATED (URINE):

Elevated urinary methylmalonic acid suggests reduced vitamin B12–dependent enzymatic activity or increased metabolic demand for B12. Accumulation reflects impaired conversion of methylmalonyl-CoA to succinyl-CoA.

Clinically, elevated methylmalonic acid may be associated with fatigue, neurological symptoms, cognitive changes, or impaired red blood cell function, although presentations vary.

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From an organic acid pattern perspective, elevated methylmalonic acid may be observed alongside elevations in odd-chain fatty acid markers and disruptions in TCA cycle intermediates, particularly reduced succinic acid availability due to impaired succinyl-CoA entry.

From a functional medicine perspective, elevated methylmalonic acid should be interpreted in the context of vitamin B12 status, gastrointestinal absorption, and overall mitochondrial efficiency rather than concentration alone.

Neurotransmitter Metabolism Comment

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.

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.

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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).

Methodology

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

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