**Dr Test Doctor** NutriPath. 16 Harker Street, Burwood VIC 3125

**Lab ID**
**Patient ID** PAT-100009
**Ext ID** 26027-0297

## Test Patient

Sex: Female • 46yrs • 01-Jan-80
123 Home Street, Test Suburb Vic 3125

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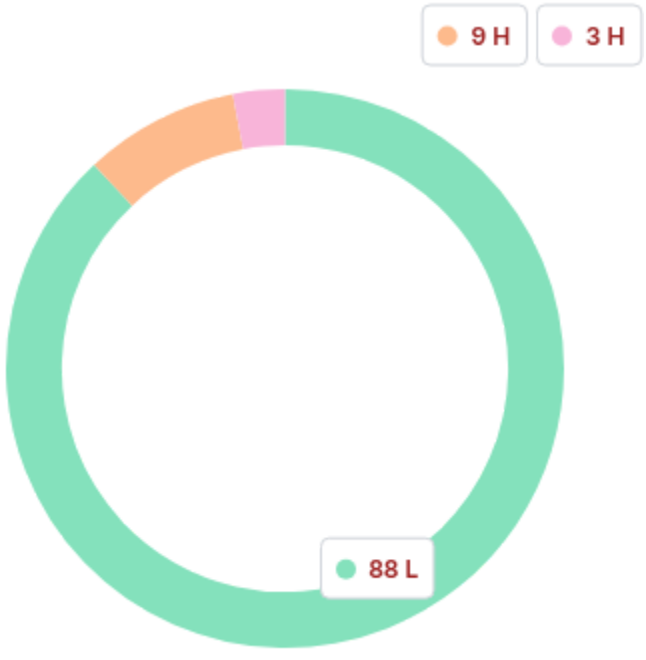
27-Jan-26

## MOE-Tox Complete Environmental Toxins

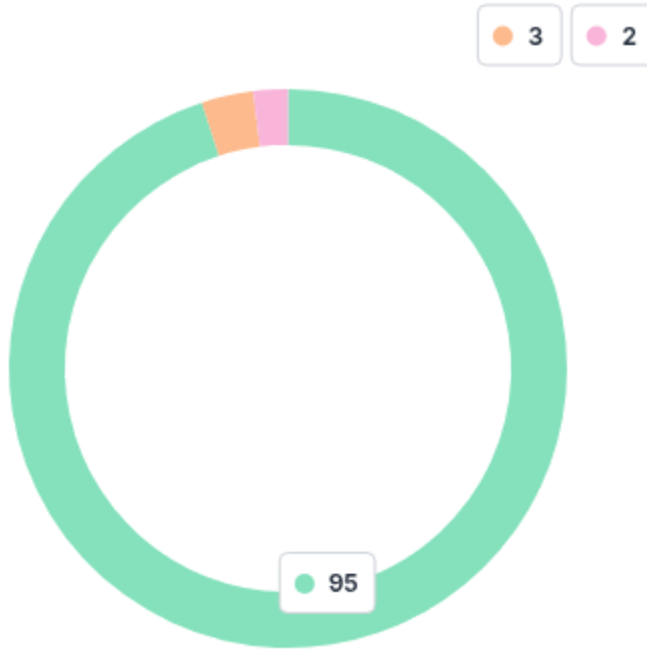
Specimen type - Urine, Spot

Collected
20-Jan-26

### TOTAL BURDEN INDEX




### HEALTHY INDEX




Low Toxic Burden  Moderate Toxic Burden  High Toxic Burden

| TEST | RESULT |
|---|---|
| Toxic Burden Legend | High |

**Interpretation:**

**Markedly elevated results** suggest significant or chronic toxicant accumulation that may carry clinical relevance. Such elevations warrant investigation of occupational, dietary, or environmental exposures and assessment of detoxification efficiency, mitochondrial status, and organ function. A structured detoxification protocol under clinical supervision is recommended, including source removal, targeted antioxidant and mitochondrial support, and medically guided use of binders or glutathione supplementation. Re-evaluation after intervention is advised to ensure toxin clearance and metabolic recovery.

| Mineral Imbalance/Mitochondrial Dysfunction | HIGH Priority | Moderate Priority |
|---|---|---|
| Iodine | Mercury | Lead |
| | Arsenic | Bisphenol A (BPA) |
| | Glyphosate | Perfluorooctanoic Acid (PFOA) |
| | Aflatoxin Group | Butylparaben |
| | Ochratoxin A | Mono-n-Butyl phthalate (mBP) |
| | Gliotoxin Derivative | |

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## OCHRATOXINS GROUP

*Ochratoxin A*

| TEST | RESULT | H/L | INTERPRETATION | REFERENCE | UNITS |
|---|---|---|---|---|---|
| Ochratoxin A | 8.390 | H | PRESENT | (<1.800) | ppb |

## AFLATOXINS GROUP

*Aflatoxin B1, Aflatoxin B2, Aflatoxin G1, Aflatoxin G2*

| TEST | RESULT | H/L | INTERPRETATION | REFERENCE | UNITS |
|---|---|---|---|---|---|
| Aflatoxin Group | 0.966 | H | EQUIVOCAL | (<0.800) | ppb |

## TRICOTHECENES GROUP

*Roridin A, Roridin E, Roridin H, Roridin L-2, Verrucarin A, Verrucarin J, Satratoxin G, Satratoxin H, Isosatratoxin F*

| TEST | RESULT | H/L | INTERPRETATION | REFERENCE | UNITS |
|---|---|---|---|---|---|
| Tricothecenes Group | 0.014 | | Not Present | (<0.070) | ppb |

## GLIOTOXINS GROUP

*Gliotoxin Derivative*

| TEST | RESULT | H/L | INTERPRETATION | REFERENCE | UNITS |
|---|---|---|---|---|---|
| Gliotoxin Derivative | 0.830 | H | EQUIVOCAL | (<0.500) | ppb |

## ZEARALENONE GROUP

*Zearalenone*

| TEST | RESULT | H/L | INTERPRETATION | REFERENCE | UNITS |
|---|---|---|---|---|---|
| Zearalenone | 0.360 | | Not Present | (<0.500) | ppb |

### Reference Ranges Interpretation

| MYCOTOXIN GROUP | Not Present | EQUIVOCAL | PRESENT |
|---|---|---|---|
| Ochratoxin Group | < 1.80 ppb | 1.80 - 2.00 ppb | > 2.00 ppb |
| Aflatoxin Group | < 0.80 ppb | 0.80 - 1.00 ppb | > 1.00 ppb |
| Tricothecenes Group | < 0.04 ppb | 0.04 - 0.08 ppb | > 0.08 ppb |
| Gliotoxins Group | < 0.50 ppb | 0.50 - 1.00 ppb | > 1.00 ppb |
| Zearalenone Group | < 0.50 ppb | 0.50 - 0.70 ppb | > 0.70 ppb |

Testing performed at Real Time Labs, Carrollton, TX, USA.

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### Mycotoxins Comment

OCHRATOXINS GROUP ELEVATED (URINE):

Elevated urinary ochratoxins, most commonly ochratoxin A, indicate exposure to mould-derived nephrotoxic mycotoxins produced by Aspergillus and Penicillium species. Urinary detection reflects recent exposure and renal clearance, while ochratoxins are also known to accumulate in tissues with prolonged exposure.

Clinically, ochratoxin exposure is associated with renal tubular stress, oxidative damage, immune dysregulation, and potential endocrine disruption. Reported symptoms may include fatigue, brain fog, polyuria, increased thirst, and susceptibility to recurrent infections. Chronic exposure has been linked to nephrotoxicity and carcinogenic risk in animal and epidemiological studies.

From a functional medicine perspective, management should prioritise source identification and removal, including assessment of indoor water damage, mould contamination, and dietary exposure (e.g. grains, coffee, dried fruits). Supportive strategies commonly include optimisation of hydration to promote renal clearance, nutritional support for antioxidant capacity (e.g. glutathione pathways), and use of evidence-based binders where clinically appropriate to reduce enterohepatic recirculation. Renal function should be considered when implementing detoxification strategies.

Treatment: Antioxidants (grape seed extract, NAC), phase II detox support, avoid high-risk foods (e.g., coffee, processed meats, wine).

AFLATOXINS GROUP ELEVATED (URINE):

Elevated urinary aflatoxins indicate exposure to hepatotoxic and carcinogenic mycotoxins produced by Aspergillus species, commonly associated with contaminated grains, nuts, seeds, and improperly stored foods. Urinary aflatoxin metabolites reflect recent exposure and hepatic detoxification activity.

Clinically, aflatoxin exposure is associated with hepatic oxidative stress, impaired phase I and phase II detoxification, immune suppression, and increased long-term risk of hepatocellular carcinoma. Symptoms may include fatigue, nausea, abdominal discomfort, and heightened sensitivity to toxins.

From a functional medicine perspective, intervention focuses on strict dietary avoidance of contaminated foods, assessment of food storage practices, and support of hepatic detoxification capacity. Evidence supports the role of antioxidants (e.g. glutathione support), adequate protein intake, and micronutrients involved in liver function. Use of targeted binders may assist in reducing reabsorption. Ongoing exposure risk should be carefully assessed, particularly in vulnerable populations.

GLIOTOXINS GROUP ELEVATED (URINE):

Elevated urinary gliotoxins reflect exposure to immunosuppressive mycotoxins produced primarily by Aspergillus fumigatus. Gliotoxin detection may indicate active mould exposure or colonisation in susceptible individuals.

Clinically, gliotoxins are associated with immune suppression, increased susceptibility to infections, fatigue, and impaired inflammatory regulation. They may also interfere with macrophage and neutrophil function, contributing to chronic or recurrent illness.

From a functional medicine perspective, interpretation should include assessment of immune status, chronic inflammatory conditions, and possible ongoing environmental exposure. Management emphasises exposure control, immune system support, optimisation of antioxidant defences, and consideration of antifungal strategies where clinically indicated and appropriately supervised.

Treatment: Eliminate mold exposure, use immune modulators (zinc, vitamin D, beta-glucans), and upregulate glutathione pathways.

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## PHYSIOLOGICAL MINERALS

| TEST | RESULT | H/L | REFERENCE | UNITS |
|---|---|---|---|---|
| Calcium | 48.00 | | (<450.00) | mg/gCR |
| Iron | 2.9 | | (<200.0) | ug/gCR |
| Magnesium | 32.00 | | (<290.00) | mg/gCR |
| Zinc | 110.00 | | (<900.00) | mg/gCR |

## TRACE MINERALS

| TEST | RESULT | H/L | REFERENCE | UNITS |
|---|---|---|---|---|
| Boron | 854 | | (<5500) | ug/gCR |
| Chromium | 2.80 | | (<4.60) | ug/gCR |
| Cobalt | 0.94 | | (<1.60) | ug/gCR |
| Copper | 69.0 | H | (<55.0) | ug/gCR |
| Germanium | 0.30 | | (<1.50) | ug/gCR |
| Iodine | 88.00 | L | (>100.00) | ug/L |
| Lithium | 45.00 | | (<55.00) | ug/gCR |
| Manganese | 1.09 | | (<1.50) | ug/gCR |
| Molybdenum | 9.10 | | (<65.00) | ug/gCR |
| Nickel | 0.81 | | (<2.00) | ug/gCR |
| Rubidium | <DL | | (<3000) | ug/gCR |
| Selenium | 12.00 | | (10.00-63.00) | ug/gCR |
| Strontium | 82.00 | | (<310.00) | ug/gCR |
| Vanadium | 3.88 | | (<8.00) | ug/gCR |

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## TOXIC METALS

| TEST | RESULT | H/L | REFERENCE | UNITS |
|---|---|---|---|---|
| Aluminium | 25.00 | | (<40.00) | ug/gCR |
| Antimony | <DL | | (<1.00) | ug/gCR |
| Arsenic | 38.00 | H | (<35.00) | ug/gCR |
| Barium | 0.93 | | (<5.70) | ug/gCR |
| Beryllium | <DL | | (<0.60) | ug/gCR |
| Bismuth | <DL | | (<1.00) | ug/gCR |
| Bromine | 940 | | (<4800) | ug/gCR |
| Cadmium | 0.22 | | (<0.60) | ug/gCR |
| Cesium | 2.11 | | (<10.30) | ug/gCR |
| Gadolinium | <DL | | (<0.23) | ug/gCR |
| Gallium | <DL | | (<0.10) | ug/gCR |
| Lead | 19.00 | H | (<8.00) | ug/gCR |
| Mercury | 5.9 | H | (<3.0) | ug/gCR |
| Palladium | 0.01 | | (<15.00) | ug/gCR |
| Platinum | 0.10 | | (<1.00) | ug/gCR |
| Silver | 0.22 | H | (<0.10) | ug/gCR |
| Tellurium | <DL | | (<0.80) | ug/gCR |
| Thallium | <DL | | (<1.50) | ug/gCR |
| Tin | 0.65 | H | (<0.50) | ug/gCR |
| Titanium | 0.05 | | (<50.00) | ug/gCR |
| Tungsten | 0.01 | | (<0.50) | ug/gCR |
| Uranium | 0.01 | | (<0.10) | ug/gCR |
| Zirconium | 0.16 | | (<5.00) | ug/gCR |

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### 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.

IODINE COMMENT: Urinary iodine reflects dietary iodine intake, more than 90% of dietary iodine is excreted in the urine. WHO Guidelines: >100 ug/gCR Not Iodine deficient 50 - 100 ug/gCR Mild Iodine deficiency 20 - 49 ug/gCR Moderate Iodine deficiency < 20 ug/gCR Severe Iodine deficiency Low levels of iodine may lead to hypothyroidism and goitre and in severe cases, intellectual disability.

### 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.

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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.

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.

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).

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### CYSTEINE DERIVATIVES

| TEST | RESULT | H/L | REFERENCE | UNITS |
|---|---|---|---|---|
| N-Acetyl (3,4-Dihydroxybutyl) cysteine (NADB) | 44.00 | | (<250.00) | ug/gCR |
| N-Acetyl (carbomoylethyl) cysteine | 123.00 | | (<190.00) | ug/gCR |
| N-Acetyl phenyl cysteine (SPMA) | <DL | | (<5.00) | ug/gCR |
| N-Acetyl (propyl) cysteine (NAPR) | <DL | | (<25.00) | ug/gCR |

### ENVIRONMENTAL PHENOLS

| TEST | RESULT | H/L | REFERENCE | UNITS |
|---|---|---|---|---|
| 4-Nonylphenol | 5.50 | H | (<3.00) | ug/gCR |
| Bisphenol A (BPA) | 7.21 | H | (<4.00) | ug/gCR |
| Triclosan (TCS) | 4.30 | | (<50.00) | ug/gCR |

### HERBICIDES (Synthetic Auxins)

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

### HERBICIDES (Photosynthetic Inhibitors)

| TEST | RESULT | H/L | REFERENCE | UNITS |
|---|---|---|---|---|
| Atrazine | 0.33 | | (<0.50) | ug/gCR |
| Atrazine mercapturate | | | | ug/gCR |

### HERBICIDES (EPSP Inhibitors)

| TEST | RESULT | H/L | REFERENCE | UNITS |
|---|---|---|---|---|
| Aminomethylphosphonic Acid (AMPA) | 0.95 | | (<2.00) | ug/gCR |
| Glyphosate | 55.0 | H | (<1.0) | ppb |

### METHYLTERT-BUTYL ETHER (MTBE) EXPOSURE

| TEST | RESULT | H/L | REFERENCE | UNITS |
|---|---|---|---|---|
| alpha-HydroxyIsoButyrate | 4.66 | | (<6.90) | ug/mgCR |

### MITOCHONDRIAL MARKERS

| TEST | RESULT | H/L | REFERENCE | UNITS |
|---|---|---|---|---|
| Tiglylglycine | 3.30 | | (<10.00) | ug/gCR |

### PARABENS

| TEST | RESULT | H/L | REFERENCE | UNITS |
|---|---|---|---|---|
| Benzylparaben | 4.09 | H | (<2.00) | ug/gCR |
| Butylparaben | 1.67 | H | (<1.00) | ug/gCR |

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| TEST | RESULT | H/L | REFERENCE | UNITS |
|---|---|---|---|---|
| Ethylparaben | <DL | | (<7.00) | ug/gCR |
| Methylparaben | <DL | | (<120.00) | ug/gCR |
| ParahydroxyBenzoic Acid | 0.00 | | (<0.57) | mmol/molCR |
| Propylparaben | <DL | | (<35.00) | ug/gCR |

### PESTICIDES

| TEST | RESULT | H/L | REFERENCE | UNITS |
|---|---|---|---|---|
| 3-Phenoxybenzoic Acid (3PBA) | 1.55 | | (<3.00) | ug/gCR |
| Diethyl Phosphate (DEP) | 9.90 | H | (<9.00) | ug/gCR |
| Diethyldithiophosphate (DEDTP) | <DL | | (<0.20) | ug/gCR |
| Diphenyl phosphate (DPP) | <DL | | (<2.50) | ug/gCR |
| Diethylthiophosphate (DETP) | <DL | | (<1.00) | ug/gCR |

### PFA's

| TEST | RESULT | H/L | REFERENCE | UNITS |
|---|---|---|---|---|
| Perfluorobutanoic acid (PFBA) | 0.43 | | (<1.20) | ug/gCR |
| Perfluorooctanoic Acid (PFOA) | 0.22 | H | (<0.10) | ug/gCR |
| Perfluorooctane Sulphonic Acid (PFOS) | 0.28 | | (<0.60) | ug/gCR |

### PHTHALATES

| TEST | RESULT | H/L | REFERENCE | UNITS |
|---|---|---|---|---|
| Butyl Benzyl phthalate (BBP) | 0.50 | | (<1.00) | ug/gCR |
| Mono-Benzyl phthalate (mBzP) | 0.60 | | (<3.00) | ug/gCR |
| Mono-n-Butyl phthalate (mBP) | 65.00 | H | (<55.00) | ug/gCR |
| Mono (3-carboxypropyl) phthalate (mCPP) | <DL | | (<31.00) | ug/gCR |
| Mono-ethyl phthalate (MEtP) | 125.00 | H | (<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 | 55.00 | | (<170.00) | ug/gCR |
| Quinolinic Acid | 7.40 | | (<9.10) | mmol/molCR |

### VOLATILE ORGANIC COMPOUNDS

| TEST | RESULT | H/L | REFERENCE | UNITS |
|---|---|---|---|---|
| 2-hydroxyethyl-mercapturic acid (HEMA) | <DL | | (<5.00) | ug/gCR |
| Mandelic Acid | 27.0 | | (<340.0) | ug/gCR |
| Phenylglyoxylic Acid | 58.0 | | (<300.0) | ug/gCR |

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| TEST | RESULT | H/L | REFERENCE | UNITS |
|---|---|---|---|---|
| Mandelic Acid + Phenylglyoxylic Acid | 85.0 | | (<610.0) | ug/gCR |

### BENZENES EXPOSURE

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

### TOLUENES EXPOSURE

| TEST | RESULT | H/L | REFERENCE | UNITS |
|---|---|---|---|---|
| Benzoic Acid | 29.00 | H | (<9.30) | mmol/molCR |
| Hippuric Acid | 330.0 | | (<603.0) | mmol/molCR |

### XYLENES EXPOSURE

| TEST | RESULT | H/L | REFERENCE | UNITS |
|---|---|---|---|---|
| 2-Methylhippuric Acid | 0.02 | | (<0.04) | mmol/molCR |
| 3-Methylhippuric Acid | 0.01 | | (<0.11) | mmol/molCR |

| TEST | RESULT | H/L | REFERENCE | UNITS |
|---|---|---|---|---|
| Creatinine, Urine | 8.00 | | (2.47-19.20) | mmol/L |

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### Environmental Phenols Comment

4-NONYLPHENOL ELEVATED (URINE):

Elevated urinary 4-nonylphenol suggests increased exposure to nonylphenol compounds, which are environmental endocrine-disrupting chemicals commonly derived from the degradation of industrial surfactants and detergents.

Clinically, nonylphenol exposure has been associated with endocrine-disrupting effects in experimental models, though human symptoms are often non-specific and exposure dependent.

From a functional medicine perspective, this finding should be interpreted in the context of cumulative environmental exposure, including household cleaning products and contaminated water sources, with emphasis on reducing ongoing exposure rather than treatment of the metabolite itself.

Treatment considerations: Minimize exposure to industrial and consumer products containing nonylphenol derivatives. Support detoxification pathways with antioxidant-rich nutrition (e.g., sulforaphane, glutathione), liver support, and hydration.

BISPHENOL A (BPA) ELEVATED (URINE):

Elevated urinary bisphenol A (BPA) suggests increased exposure to BPA-containing materials, including food and beverage packaging, thermal receipt paper, and certain plastics. Urinary BPA reflects recent exposure and efficient renal elimination.

Clinically, BPA is recognised as an endocrine-disrupting chemical, and elevated exposure has been associated with hormonal dysregulation, metabolic disturbance, and reproductive effects, although symptom expression varies.

From a functional medicine perspective, this result should be interpreted in the context of dietary packaging exposure, plastic use, and overall endocrine burden, with focus on exposure identification and reduction.

Treatment considerations: Treatment focuses on reducing exposure by avoiding BPA-containing plastics and supporting the body's natural detoxification with antioxidants.

### Herbicides Comment

GLYPHOSATE ELEVATED (URINE):

Elevated urinary glyphosate suggests recent exposure and renal excretion of this widely used herbicide. Urinary glyphosate reflects short-term exposure rather than tissue accumulation.

Clinically, glyphosate exposure has been associated with gastrointestinal symptoms, oxidative stress, and potential endocrine or microbiome-related effects, though findings vary with dose and chronicity.

From a functional medicine perspective, this result should be interpreted in the context of dietary patterns, occupational or residential herbicide exposure, and cumulative pesticide burden, with emphasis on minimising ongoing exposure rather than treating the metabolite itself.

Treatment considerations: Focus on microbiome repair, liver support, antioxidant nutrients, and avoidance of glyphosate-laden foods and environments.

### Parabens Comment

BENZYLPARABEN ELEVATED (URINE):

Elevated urinary benzylparaben suggests increased exposure to paraben-containing personal care products, cosmetics, or pharmaceutical formulations. Urinary benzylparaben reflects recent exposure and effective renal elimination.

Clinically, parabens are recognised endocrine-disrupting chemicals, and elevated exposure may contribute to hormonal dysregulation, although symptom expression is variable and often subtle.

From a functional medicine perspective, this result should be interpreted in the context of cumulative personal care and cosmetic exposure, with emphasis on reducing ongoing paraben exposure rather than intervention directed at the urinary finding.

Treatment considerations: Minimize exposure to synthetic preservatives. Support detoxification and antioxidant systems. Evaluate total endocrine-disrupting chemical (EDC) burden if multiple parabens are elevated.

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BUTYLPARABEN ELEVATED (URINE):

Elevated urinary butylparaben suggests increased exposure to paraben-containing products, including cosmetics, toiletries, and some pharmaceutical preparations. Butylparaben is among the more lipophilic parabens and may exhibit greater endocrine activity.

Clinically, elevated butylparaben exposure has been associated with endocrine-related effects in experimental models, though human clinical manifestations are often non-specific.

From a functional medicine perspective, this finding should be interpreted in the context of total paraben burden and endocrine exposure load, with focus on identifying and reducing sources of exposure.

Treatment considerations: Eliminate paraben-containing products. Support liver detoxification pathways and hormone metabolism (e.g., DIM, calcium-D-glucarate, cruciferous vegetables). Monitor hormonal status if symptomatic.

### Pesticides Comment

DIETHYL PHOSPHATE (DEP) ELEVATED (URINE):

Elevated urinary diethyl phosphate (DEP) suggests exposure to organophosphate pesticides. DEP is a non-specific dialkyl phosphate metabolite reflecting exposure to multiple organophosphate compounds.

Clinically, organophosphate exposure may be associated with neurological, gastrointestinal, or autonomic symptoms at higher levels, depending on exposure intensity and duration.

From a functional medicine perspective, this finding should be interpreted in the context of dietary, occupational, or residential pesticide exposure, with emphasis on exposure mitigation rather than intervention directed at the metabolite.

Treatment considerations: Avoid exposure to OP-containing pesticides. Support acetylcholine metabolism and nerve function (e.g., choline, B5, magnesium). Use nutrients that enhance detoxification and methylation (B-vitamins, folate, SAMe).

### PFAS Comment

PERFLUOROOCTANOIC ACID (PFOA) ELEVATED:

Elevated PFOA in urine is primarily from exposure and can be linked to potential health effects on the kidneys, hyperuricemia (high uric acid), cancer, endocrine, reproductive.

There is no medically approved treatment to remove PFOA from the body, but exposure can be reduced by avoiding certain foods and products, and some medical interventions may help lower levels.

### Phthalates Comment

MONO-N-BUTYL PHTHALATE (mBP) ELEVATED (URINE):

Elevated urinary mono-n-butyl phthalate (mBP) suggests exposure to dibutyl phthalate (DBP), commonly found in personal care products, cosmetics, fragrances, and some plastics.

Clinically, DBP exposure has been associated with endocrine-disrupting effects, particularly affecting reproductive hormone pathways.

From a functional medicine perspective, this finding should be interpreted in the context of personal care product use and cumulative phthalate exposure, with emphasis on reducing ongoing sources.

Treatment considerations: Avoid DBP-containing products. Use glutathione, selenium, and zinc to support detoxification and hormone metabolism. Consider endocrine evaluation.

MONO-ETHYL PHTHALATE (MEtP) ELEVATED (URINE):

Elevated urinary mono-ethyl phthalate (MEtP) suggests exposure to diethyl phthalate (DEP), commonly used in fragrances, cosmetics, and personal care products.

Clinically, DEP exposure is generally associated with endocrine modulation rather than acute toxicity.

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**Lab ID**
**Patient ID** PAT-100009
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From a functional medicine perspective, this finding highlights fragrance-related exposure and should be interpreted in the context of total phthalate burden.

Treatment considerations: Switch to phthalate-free personal care products. Support liver Phase II detox with amino acids (glycine, glutamine), fiber, and methylation nutrients. Consider endocrine and oxidative stress evaluation.

### Environmental Toxins 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.

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.

MONO-ETHYL PHTHALATE (MEtP) ELEVATED (URINE):

Elevated urinary mono-ethyl phthalate (MEtP) suggests exposure to diethyl phthalate (DEP), commonly used in fragrances, cosmetics, and personal care products.

Clinically, DEP exposure is generally associated with endocrine modulation rather than acute toxicity.

From a functional medicine perspective, this finding highlights fragrance-related exposure and should be interpreted in the context of total phthalate burden.

Treatment considerations: Switch to phthalate-free personal care products. Support liver Phase II detox with amino acids (glycine, glutamine), fiber, and methylation nutrients. Consider endocrine and oxidative stress evaluation.

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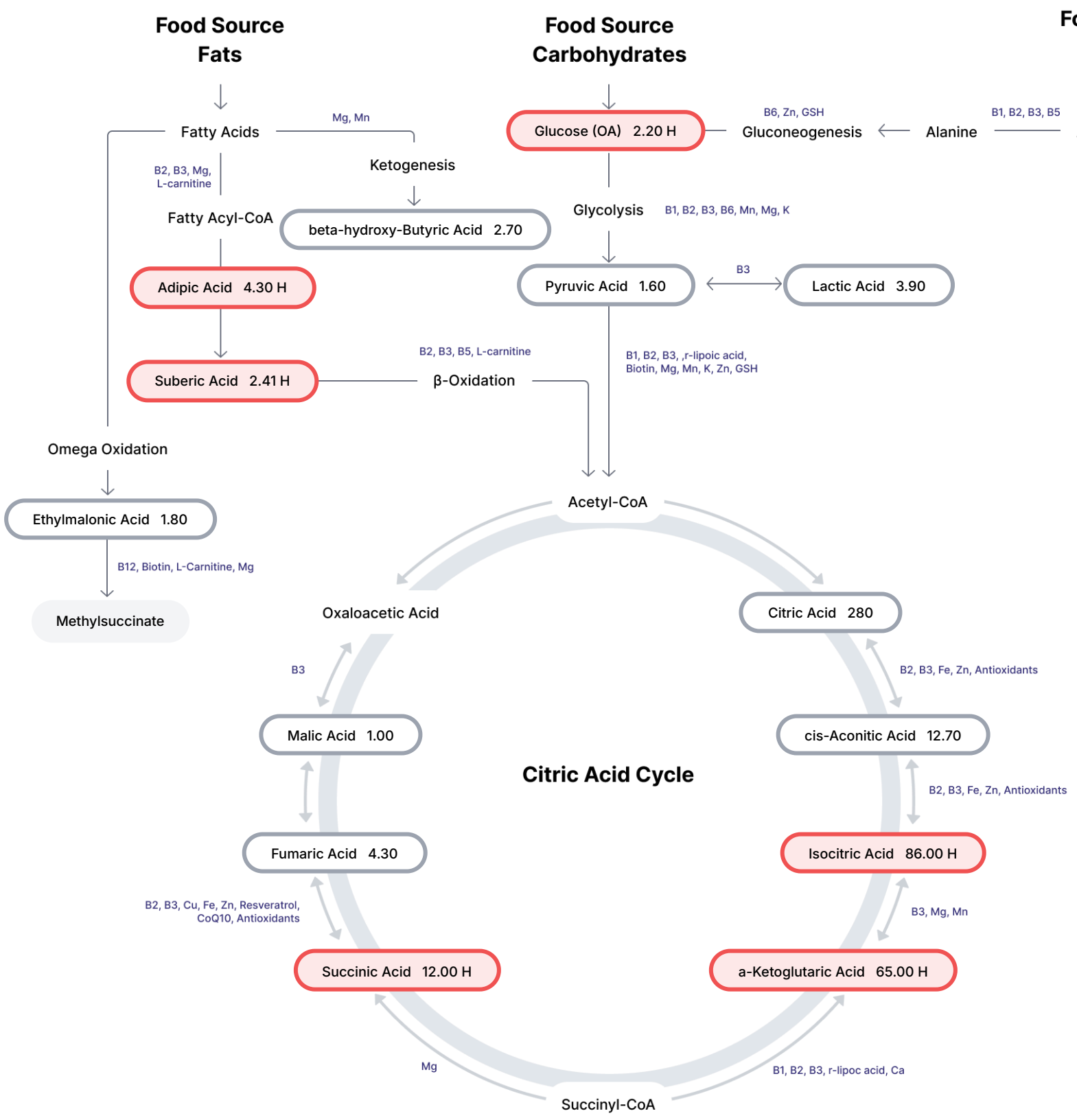
**Legend** Not Tested | Within Range | Out of Range — L = Low, LL = Critically Low — H = High, HH = Critically High

## Organic Acids Pathway



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## CARBOHYDRATES METABOLISM/Glycolysis

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

| | TEST | RESULT | H/L | REFERENCE | UNITS |
|---|---|---|---|---|---|
| 1 | Pyruvic Acid | 1.60 | | (0.50-8.70) | mmol/molCR |
| 2 | Lactic Acid | 3.90 | | (<48.00) | mmol/molCR |
| 3 | Glucose (OA) | 2.20 | H | (<1.10) | ug/mgCR |

## KETONE/FATTY ACIDS METABOLISM

*(Carnitine & B2)*

| | TEST | RESULT | H/L | REFERENCE | UNITS |
|---|---|---|---|---|---|
| 4 | Adipic Acid | 4.30 | H | (<3.80) | mmol/molCR |
| 5 | Suberic Acid | 2.41 | H | (<2.20) | mmol/molCR |
| 6 | Ethylmalonic Acid | 1.80 | | (<5.80) | mmol/molCR |
| 7 | Methyl-Succinic Acid | 1.60 | | (<10.80) | mmol/molCR |
| 8 | Pimelic Acid | 2.30 | | (<4.00) | mmol/molCR |
| 9 | alpha-hydroxy-Butyric Acid | 2.80 | | (<6.90) | mmol/molCR |
| 10 | beta-hydroxy-Butyric Acid | 2.70 | | (<3.10) | mmol/molCR |

## B-COMPLEX VITAMINS/AMINO ACID MARKERS

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

| | TEST | RESULT | H/L | REFERENCE | UNITS |
|---|---|---|---|---|---|
| 11 | alpha-Ketoisovaleric Acid | 2.30 | | (<4.10) | mmol/molCR |
| 12 | alpha-Ketoisocaproic Acid | 0.40 | | (<0.65) | mmol/molCR |
| 13 | alpha-keto-beta-Methylvaleric Acid | 0.80 | | (<2.00) | mmol/molCR |
| 14 | Xanthurenic Acid | 3.30 | H | (<0.96) | mmol/molCR |
| 15 | beta-Hydroxyisovaleric Acid | 7.20 | | (<29.00) | mmol/molCR |
| 16 | Methylmalonic Acid | 3.90 | H | (<1.90) | mmol/molCR |
| 17 | Formiminoglutamic Acid | 1.70 | H | (<1.50) | mmol/molCR |

## CITRIC ACID CYCLE METABOLISM

*(B Comp, CoQ10, Amino Acids, Mg)*

| | TEST | RESULT | H/L | REFERENCE | UNITS |
|---|---|---|---|---|---|
| 18 | Citric Acid | 280 | | (40-507) | mmol/molCR |
| 19 | cis-Aconitic Acid | 12.70 | | (3.50-36.00) | mmol/molCR |
| 20 | Isocitric Acid | 86.00 | H | (5.00-65.00) | mmol/molCR |
| 21 | a-Ketoglutaric Acid | 65.00 | H | (4.00-52.00) | mmol/molCR |
| 22 | Succinic Acid | 12.00 | H | (1.00-9.70) | mmol/molCR |
| 23 | Fumaric Acid | 4.30 | | (<8.60) | mmol/molCR |
| 24 | Malic Acid | 1.00 | | (<1.80) | mmol/molCR |

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| TEST | RESULT | H/L | REFERENCE | UNITS |
|---|---|---|---|---|
| 25 3-Methylglutaric Acid | 3.60 | | (<8.50) | mmol/molCR |

## NEUROTRANSMITTER METABOLISM

*(Tyrosine, Tryptophan, B6, Antioxidants)*

| TEST | RESULT | H/L | REFERENCE | UNITS |
|---|---|---|---|---|
| 26 Homovanillic Acid (HVA) | 2.90 | | (0.10-5.30) | mmol/molCR |
| 27 Vanillylmandelic Acid (VMA) | 3.70 | H | (0.40-3.60) | mmol/molCR |
| 28 5-Hydroxyindoleacetic Acid (5HIAA) | 3.00 | | (<4.30) | mmol/molCR |
| 29 Kynurenic Acid | 2.70 | H | (<2.20) | mmol/molCR |
| 30 Quinolinic Acid | 7.40 | | (<9.10) | mmol/molCR |
| 31 Picolinic Acid | 3.50 | | (<10.28) | mmol/molCR |
| 32 Cortisol (OA) | 44.0 | | (7.0-63.8) | ug/mgCR |

## OXIDATIVE DAMAGE/ANTIOXIDANT MARKERS

*(Vitamin C, Other Antioxidants)*

| TEST | RESULT | H/L | REFERENCE | UNITS |
|---|---|---|---|---|
| 33 Parahydroxyphenyllactic Acid | 4.60 | | (<14.60) | mmol/molCR |
| 34 8-hydroxy-deoxyguanosine | 2.90 | H | (<2.70) | mmol/molCR |

## DETOXIFICATION INDICATORS

*(Arg, NAC, Meth, Mg, Antioxidants)*

| TEST | RESULT | H/L | REFERENCE | UNITS |
|---|---|---|---|---|
| 35 2-Methylhippuric Acid | 0.02 | | (<0.04) | mmol/molCR |
| 36 Orotic Acid | 2.55 | | (<3.20) | mmol/molCR |
| 37 Glucaric Acid | 4.60 | | (<11.00) | mmol/molCR |
| 38 Pyroglutamic Acid | 15.90 | | (4.50-33.00) | mmol/molCR |

## BACTERIAL DYSBIOSIS MARKERS

| TEST | RESULT | H/L | REFERENCE | UNITS |
|---|---|---|---|---|
| 39 Benzoic Acid | 29.00 | H | (<9.30) | mmol/molCR |
| 40 Hippuric Acid | 330.0 | | (<603.0) | mmol/molCR |
| 41 Phenylacetic Acid | 2.40 | | (<3.90) | mmol/molCR |
| 42 Phenylpropionic Acid | 0.60 | H | (<0.40) | mmol/molCR |
| 43 ParahydroxyBenzoic Acid | 0.00 | | (<0.57) | mmol/molCR |
| 44 p-HydroxyPhenylacetic Acid | 3.90 | H | (<3.90) | mmol/molCR |
| 45 Indoleacetic Acid | 6.80 | | (<11.00) | mmol/molCR |
| 46 Tricarballylic Acid | 0.38 | | (<0.44) | mmol/molCR |

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## CLOSTRIDIAL SPECIES

| TEST | RESULT | H/L | REFERENCE | UNITS |
|---|---|---|---|---|
| 47 DiHydroxyPhenylPropionic Acid | 2.98 | | (<5.30) | mmol/molCR |
| 48 4-Cresol | 0.55 | | (<3.00) | ug/mgCR |
| 49 3-hydroxy-Propionic Acid | 3.98 | | (<17.00) | mmol/molCR |

## YEAST/FUNGAL DYSBIOSIS MARKERS

| TEST | RESULT | H/L | REFERENCE | UNITS |
|---|---|---|---|---|
| 50 Arabinitol | 33.0 | | (<36.0) | mmol/molCR |
| 51 Citramalic Acid | 3.10 | | (<3.60) | mmol/molCR |
| 52 Tartaric Acid | 4.90 | | (<15.00) | mmol/molCR |

## OXALATE METABOLITES

| TEST | RESULT | H/L | REFERENCE | UNITS |
|---|---|---|---|---|
| 53 Oxalic Acid | 13.80 | | (<78.00) | mmol/molCR |
| 54 Glyceric Acid | 4.10 | | (<6.00) | mmol/molCR |
| 55 Glycolic Acid | 20.30 | | (<67.00) | mmol/molCR |

## NUTRITIONAL MARKERS

| TEST | RESULT | H/L | REFERENCE | UNITS |
|---|---|---|---|---|
| 56 Pyridoxic Acid (Vit B6) | 5.70 | | (0.68-34.00) | mmol/molCR |
| 57 Pantothenic Acid (Vit B5) | 0.80 | | (0.10-10.00) | mmol/molCR |
| 58 Glutaric Acid (Vit B2) | 0.13 | | (0.02-0.36) | mmol/molCR |
| 59 Ascorbic Acid (Vit C) | 28.00 | | (0.50-200.00) | mmol/molCR |
| 60 CoEnzyme Q10 (CoQ10) | 1.10 | | (0.10-5.00) | mmol/molCR |
| 61 N-Acetylcysteine (NAC) | 0.08 | | (0.02-0.28) | mmol/molCR |
| 62 Biotin (Vit H) | | | | mmol/molCR |

| TEST | RESULT | H/L | REFERENCE | UNITS |
|---|---|---|---|---|
| Creatinine, Urine | 8.00 | | (2.47-19.20) | mmol/L |

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## NUTRITIONAL GUIDE

| TEST | Adult RDI | UNITS | Clinical Notes |
|---|---|---|---|
| Vitamin-E | 200.0 | U | |
| Vitamin-B1 | 15.0 | mg | |
| Vitamin-B2 | 17.0 | mg | |
| Vitamin-B3 | 13.0 | mg | |
| Vitamin-B5 | 10.0 | mg | |
| Vitamin-B6 | 5.0 | mg | |
| Glycine | 5.0 | mg | |
| Glutamine | 0.0 | mg | |
| Glutathione | 50.0 | mg | |
| Taurine | 6.0 | mg | |
| Tyrosine | 0.0 | mg | |
| Tryptophan | 8.0 | mg | |
| L-Arginine | 0.0 | mg | |
| Aspartic Acid | 0.0 | mg | |
| Acetyl-L-Carnitine | 20.0 | mg | |
| Biotin | 0.0 | ug | |
| Chromium | 3.0 | ug | |
| Coenzyme Q10 | 400.0 | mg | |
| Calcium-D-glucurate | 0.0 | mg | |
| EPA/DHA | 0.0 | mg | |
| Iron | 0.0 | mg | |
| Folinic Acid | 0.0 | ug | |
| D-Lactate-free probiotics | 1.0 | billion CFU | |
| Magnesium | 140.0 | mg | |
| Manganese | 0.0 | mg | |
| Malic Acid | 0.0 | ug | |
| Methionine | 6.0 | mg | |
| N-Acetylcysteine | 100.0 | mg | |
| Ornithine | 10.0 | mg | |
| Vanadium | 0.0 | ug | |
| alpha-Lipoic Acid | 200.0 | mg | |
| Lysine | 0.0 | mg | |
| Lactobacillus | 1.0 | billion CFU | |
| 5-hydroxy-Tryptophan (5-HTP) | 0.0 | mg | |
| Serine | 5.0 | mg | |
| Probiotics (Multistrain) | 100.0 | billion CFU | |

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| TEST | Adult RDI | UNITS | Clinical Notes |
|---|---|---|---|
| **Phenylalanine** | **0.0** | mg | |
| **Vitamin-C** | **400.0** | mg | |

**Disclaimer:**

*Supplement recommendations are based on the Organic Acid test results. The prescribing health practitioner must take into consideration the age, weight, sex, and pregnancy or lactation state. In addition, consider clinical state, medication regime, associated drug-nutrient depletion and allergies.*

*The doses listed above are considered optimal, based on lab results and do not apply to specific disease conditions where doses may need to be altered. The vitamins, minerals or amino acids listed are elemental quantities. Use clinical discretion when choosing the right salt with the guidance of your compounding health professional. For example, Magnesium may be prescribed as a glycinate for its calming effect or threonate may be used for a Magnesium that crosses the blood-brain-barrier.*

References:

Laboratory Evaluations for Integrative and Functional Medicine by Richard Lord.

J.Alexander Bralley; Textbook of Nutritional Medicine by Alan Gaby.

## Carbohydrate Metabolism Comment

GLUCOSE ELEVATED (URINE):

Elevated urinary glucose suggests that circulating blood glucose levels have exceeded the renal threshold for reabsorption, resulting in glucose spill into the urine. This most commonly reflects impaired glucose regulation or reduced renal glucose handling.

Clinically, elevated urinary glucose may be associated with increased thirst, frequent urination, fatigue, blurred vision, or unexplained weight changes, although symptoms may be mild or absent in early stages.

From an organic acid pattern perspective, elevated urinary glucose may be observed alongside alterations in glycolytic and tricarboxylic acid (TCA) cycle intermediates, such as elevated pyruvic or lactic acid and disrupted citric or succinic acid patterns, reflecting impaired carbohydrate utilisation and metabolic inefficiency.

From a functional medicine perspective, elevated urinary glucose should be interpreted in the context of overall glycaemic control, insulin signalling, dietary carbohydrate load, and metabolic flexibility rather than as an isolated urinary finding.

Supplementation Recommendations: Chromium, Vanadium, Insulin, Diabetic medication.

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### Ketone/Fatty Acids Comment

ADIPIC ACID ELEVATED (URINE):

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

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

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

SUBERIC ACID ELEVATED (URINE):

Suberic acid reflects reliance on omega-oxidation pathways due to incomplete mitochondrial fatty-acid oxidation, commonly seen with metabolic stress or carnitine deficiency.

Clinically, elevations may be associated with low energy, hypoglycaemic symptoms, cognitive slowing, or poor stress tolerance. Interpretation should consider dietary intake, medications, and the broader metabolic context.

From a functional medicine perspective, management focuses on repletion of mitochondrial cofactors, assessment of carnitine status, macronutrient (Vitamin B2) balancing, and addressing inflammatory or infectious stressors, addressing underlying contributors rather than isolated suppression of the marker.

### B-Complex Vitamins/Amino Acids Comment

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.

### Citric Acid Cycle Comment

ISOCITRIC ACID ELEVATED (URINE):

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

Clinically, symptoms are non-specific.

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

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

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

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

ALPHA-KETOGLUTARIC ACID ELEVATED (URINE)

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

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

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

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

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

SUCCINIC ACID ELEVATED (URINE):

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

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

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

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

Elevated succinate may indicate a deficiency of Riboflavin and CoQ10.

### Neurotransmitter Metabolism Comment

VANILMANDELLIC ACID (VMA) ELEVATED (URINE):

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

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

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

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

KYNURENIC ACID ELEVATED (URINE):

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

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

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

Consider: Supplementation with Vitamin B6.

### Methylation Cofactors Comment

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.

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.

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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.

FORMIMINOGLUTAMIC ACID ELEVATED (URINE):

Elevated urinary formiminoglutamic acid suggests reduced folate-dependent metabolic activity, most commonly reflecting functional folate insufficiency or increased folate demand. Accumulation occurs when histidine metabolism cannot proceed efficiently through one-carbon transfer pathways.

Clinically, elevated FIGLU may be associated with fatigue, anaemia, cognitive changes, or impaired methylation capacity, although symptoms are often non-specific.

From an organic acid pattern perspective, elevated FIGLU may be observed alongside other markers of impaired methylation or B-vitamin imbalance, including potential interactions with vitamin B12-dependent pathways such as methylmalonic acid.

From a functional medicine perspective, elevated FIGLU should be interpreted in the context of folate status, vitamin B12 interplay, and overall one-carbon metabolic balance rather than as an isolated abnormality.

### Oxidative Damage/Detoxification Comment

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

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

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

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

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

### Bacterial Dysbiosis Comment

BENZOIC ACID ELEVATED (URINE):

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

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

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

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

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

PHENYLPROPIONIC ACID ELEVATED (URINE):

Phenylpropionic acid reflects microbial metabolism of dietary polyphenols and aromatic amino acids. Elevation may indicate dysbiosis or increased microbial fermentation.

Clinically, elevations may be associated with digestive discomfort, bloating, or fatigue. Results should be interpreted alongside diet, environmental exposures, medications, and overall clinical context.

From a functional medicine perspective, management focuses on dietary review, gut microbiome optimisation, and reduction of excessive fermentable substrates, with emphasis on reducing exposure and supporting metabolic clearance pathways.

P-HYDROXYPHENYLACETIC ACID ELEVATED (URINE):

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Elevated urinary p-hydroxyphenylacetic acid suggests increased microbial metabolism of tyrosine or altered aromatic amino acid handling.

Clinically, elevated levels may be associated with gastrointestinal symptoms, fatigue, or neurocognitive complaints such as brain fog, although symptoms are non-specific.

From an organic acid pattern perspective, elevation often clusters with other tyrosine- and phenylalanine-derived metabolites, supporting a gut microbial contribution.

From a functional medicine perspective, elevated p-hydroxyphenylacetic acid should be interpreted in the context of gut microbiome composition, protein digestion, and bowel function.

### Methodology

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