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

Lab ID
Patient ID PAT-100009
Ext ID 26027-0296

Test Patient

Sex: Female • 46yrs • 01-Jan-80
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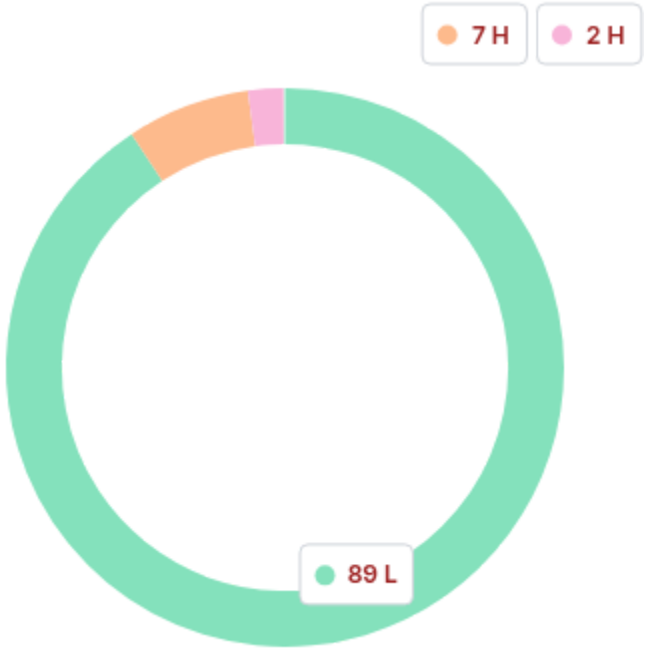
ALL-Tox Environmental Toxins

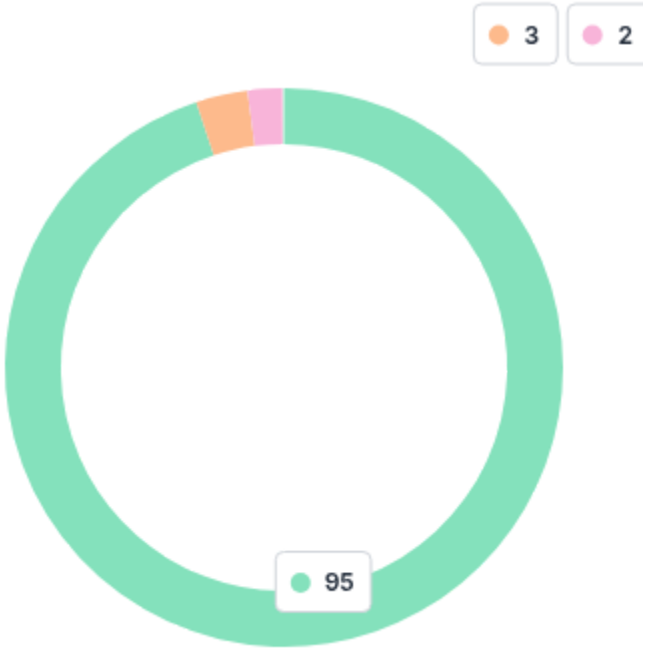
Specimen type - Urine, Spot

Collected
20-Jan-26

TOXIC BURDEN INDEX



HEALTHY INDEX



Low Toxic Burden Moderate Toxic Burden High Toxic Burden

SERVICE	RESULT
Toxic Burden Legend	High

Toxic Burden Comment

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 Arsenic Glyphosate Aflatoxin Group Ochratoxin A	Lead Bisphenol A (BPA) Perfluorooctanoic Acid (PFOA) Butylparaben Mono-n-Butyl phthalate (mBP)

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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)	0.06		(<1.00)	ug/gCR

HERBICIDES (Photosynthetic Inhibitors)

TEST	RESULT	H/L	REFERENCE	UNITS
Atrazine	0.33		(<0.50)	ug/gCR
Atrazine mercapturate	0.66	H	(<0.50)	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)	0.26		(<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

ATRAZINE MERCAPTURATE ELEVATED (URINE)

Elevated urinary atrazine mercapturate reflects confirmed internal exposure to atrazine, with evidence of hepatic phase II detoxification via glutathione conjugation. This metabolite represents the body’s biochemical processing of atrazine rather than the presence of the parent compound alone.

The detection of atrazine mercapturate indicates that atrazine has been absorbed and metabolised, typically reflecting recent exposure through drinking water, dietary intake, or environmental contact. While this metabolite itself is less biologically active than atrazine, its presence confirms systemic exposure to an endocrine-disrupting herbicide.

From a functional medicine standpoint, interpretation should consider detoxification capacity, glutathione availability, and co-exposure to other pesticides. Management focuses on reducing ongoing exposure, supporting hepatic biotransformation pathways where appropriate, and addressing cumulative environmental toxicant load rather than treating the metabolite directly.

Treatment considerations: Similar to atrazine- emphasize toxin avoidance and hepatic detoxification pathways (Phase I/II liver support), including NAC, glycine, and cruciferous vegetables.

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.

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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.

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

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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.

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.

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

Lab ID
Patient ID PAT-100009
Ext ID 26027-0296

Test Patient

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

RECEIVED
27-Jan-26

Methodology

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