From Load to Capacity:

Utilizing OAT and TOXDetect to Support Chronic Conditions

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Clinical Development Manager | Mosaic Diagnostics

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Lindsay Goddard MS, RD, LD/N

- Lindsay Goddard is a Registered and Licensed Dietitian (RD, LD/N) with a Master's degree in Nutrition, a Bachelor's degree in Biology, advanced graduate-level training in Toxicology, and certification in Integrative and Functional Nutrition. She has been with Mosaic Diagnostics (formerly Great Plains Laboratory) for over seven years, where she supports and educates healthcare practitioners in the interpretation and clinical application of laboratory data while contributing to the innovation and development of clinical education.
- Prior to Mosaic, she practiced in various settings, such as hospitals, universities, and outpatient clinics, with a wide population range spanning from neonates to geriatrics. She worked within many specialties during that time, including gastroenterology, critical care, physiatry, and genetics/metabolic medicine. In addition to her work at Mosaic, Lindsay currently maintains a private practice consulting with providers and patients, serves as the Dietitian for a West Florida–based hospice, and teaches Yoga to all age groups, integrating mindfulness and coherence.

MOSAIC DIAGNOSTICS

Outline

- Prevalence and Importance of Toxicant Exposures
- Toxicant Testing
- Review Organic Acids Testing
- Connecting Organic Acids with Toxic Exposures
- Case Study
- Q&A

MOSAIC DIAGNOSTICS

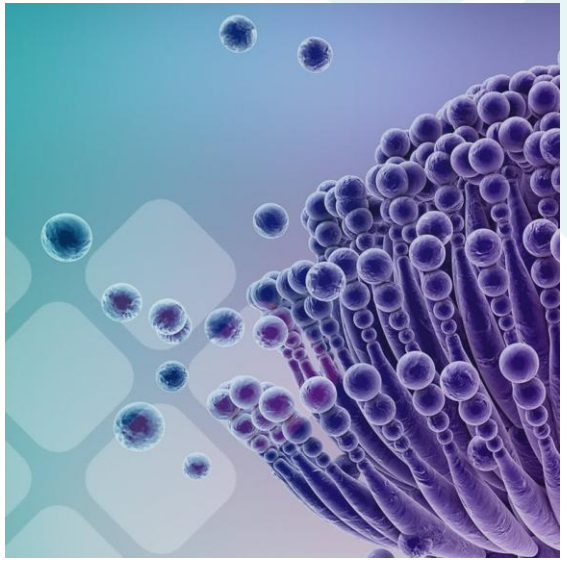
Terminology

Toxicant

- Defined as a **man-made** or **synthetic** chemical that can cause harm to a living organism.

Toxin

- Produced by living organisms e.g mycotoxins, snake venom, bacterial and plant toxins



MOSAIC DIAGNOSTICS

Prevalence of Toxicant Exposures in the EU

According to HBM4EU- biomonitoring infrastructure involved in 24 European Union Member States

Phthalate and **Phenol (BPA)** exposures are a continuous concern, and a 2020 found in 11 countries, 95% of participants were positive with some exceeding amounts beyond the safety threshold.

Associated with reproductive toxicity, ADHD, endometriosis, and general endocrine disruption

BPS, another type of phenol, continues to exceed acceptable rates.

Major endocrine disruptor

Organophosphate herbicides are estimated to be more prevalent in Europe, as compared to the US and higher amounts have been found in toddlers and children.

Impacts neurobehavioral deficits

Pyrethroids, a widely used agricultural and household insecticide, was found in 36% of European children to exceed guidance values.

Concerns for impacts on neurodevelopmental effects

Acrylamide, a genotoxic food processing contaminant was found in 100% of Spanish adults, and Southern Europeans had higher levels than Northern, and was higher in children

Classified as a probable human carcinogen

MOSAIC DIAGNOSTICS

PMID: 39693778, PMID: 27765541 PMID: 36773580, PMID: 38042944, PMID: 37236588,
doi: 10.2800/502521, https://doi.org/10.1016/j.scitotenv.2022.158368

Burden from VOCs in the EU

Europe currently does not bio monitor for VOCs

J Expo Sci Environ Epidemiol. 2026 May;36(3):490-503. doi: 10.1038/s41370-026-00838-x. Epub 2026 Jan 28.

Analysis of 18 mercapturic acids in urine samples from the German Environmental Specimen Bank–tackling the data gap in the human biomonitoring of VOCs in Europe

Nikola Pluym [1], Therese Burkhardt [2], Till Weber [3], Gerhard Scherer [2], Max Scherer [2], Marike Kolossa-Gehring [3]

Affiliations + expand

PMID: 41606230 PMCID: PMC13143828 DOI: 10.1038/s41370-026-00838-x

Abstract

Background: Human biomonitoring (HBM) plays a pivotal role in assessing exposure to toxicologically relevant chemicals, with urinary metabolites serving as key indicators. Despite the widespread implementation of HBM programs globally, certain metabolites, such as mercapturic acids (MAs) derived from volatile organic compounds (VOCs), remain understudied in non-occupationally exposed populations.

Objective: To bridge this data gap, we analyzed 18 MAs in 360 24-h urine samples collected over a time span of 21 years from 2000 to 2021 in Germany.

Methods: Two LC-MS/MS methods were utilized to quantify MAs in urine samples obtained from the Environmental Specimen Bank. Statistical analyses, including Kruskal-Wallis tests and Spearman correlations, were employed to evaluate temporal trends, sex-specific differences, and correlations between MAs.

Results: Quantification rates between 95 and 100% were obtained for 14 of the 18 MAs, with notable variations in concentrations among different metabolites. The most pronounced decrease in MA levels was observed from 2010/2015 to 2019 with a significant trend for 8 MAs, potentially reflecting changes in environmental exposures and regulations. Moreover, significant differences in urinary excretion per 24 h between males and females were observed for several MAs, highlighting the importance of considering sex in exposure assessments.

Impact statement: Comprehensive human biomonitoring (HBM) data with regard to the exposure to volatile organic compounds (VOCs) in Europe is lacking. This prompted us to quantify 18 mercapturic acids as urinary VOC metabolites in 360 samples from the Environmental Specimen Bank collected between 2000 and 2021 in Germany. Our study demonstrates the ubiquitous exposure to numerous VOCs of high toxicological relevance, albeit with a decreasing trend over time for most of the metabolites. This emphasizes the need for a broader HBM to better understand the risk of VOC exposure in Germany and more general in Europe.

A study evaluating 360 urine samples collected over a 21 year time span (2000-2021) in Germany found **95-100% samples contained metabolized analytes from VOC exposure.**

MOSAIC DIAGNOSTICS

Glyphosate in the EU

Near Universal Detection in European populations

› Environ Sci Pollut Res Int. 2022 May;29(22):32882-32893. doi: 10.1007/s11356-021-18110-0. Epub 2022 Jan 12.

Quantifiable urine glyphosate levels detected in 99% of the French population, with higher values in men, in younger people, and in farmers

Daniel Grau [1], Nicole Grau [1], Quentin Gascuel [1], Christian Paroissin [2], Cécile Stratonovitch [3], Denis Lairon [4], Damien A Devault [5], Julie Di Cristofaro [6]

Affiliations + expand

PMID: 35018595 PMCID: PMC9072501 DOI: 10.1007/s11356-021-18110-0

Abstract

France is the first pesticide-consuming country in Europe. Glyphosate is the most used pesticide worldwide and glyphosate is detected in the general population of industrialized countries, with higher levels found in farmers and children. Little data was available concerning exposure in France. Our objective was to determine glyphosate levels in the French general population and to search for an association with seasons, biological features, lifestyle status, dietary habits, and occupational exposure. This study includes 6848 participants recruited between 2018 and 2020. Associated data include age, gender, location, employment status, and dietary information. Glyphosate was quantified by a single laboratory in first-void urine samples using ELISA. Our results support a general contamination of the French population, with glyphosate quantifiable in 99.8% of urine samples with a mean of 1.19 ng/ml + / - 0.84 after adjustment to body mass index (BMI). We confirm higher glyphosate levels in men and children. Our results support glyphosate contamination through food and water intake, as lower glyphosate levels are associated with dominant organic food intake and filtered water. Higher occupational exposure is confirmed in farmers and farmers working in wine-growing environment. Thus, our present results show a general contamination of the French population with glyphosate, and further contribute to the description of a widespread contamination in industrialized countries.

French study of 6,848 participants found quantifiable urinary glyphosate in 99.8% of samples- higher in men and children.

MOSAIC DIAGNOSTICS

Patient Population

- Cardiovascular disease
- Metabolic syndromes
  - Obesity, T2DM
  - NASH, NAFLD
- Cancers
- Neurological Concerns
  - Cognitive dysfunction
  - Parkinsons
  - Migraines, Headaches
- Behavioral/Mood
  - ADD/ADHD
  - Depression, anxiety
- Fatigue
- Respiratory, Allergy
  - Asthma, wheezing, apnea, COPD
  - Eye irritation
- Immune dysregulation
  - Autoimmune disease
  - Allergies
- Endocrine Dysfunctions
  - Infertility
  - Hormone dysregulation
- GI Concerns
  - IBD, nausea, vomiting
- Osteoporosis
- Blood disorders

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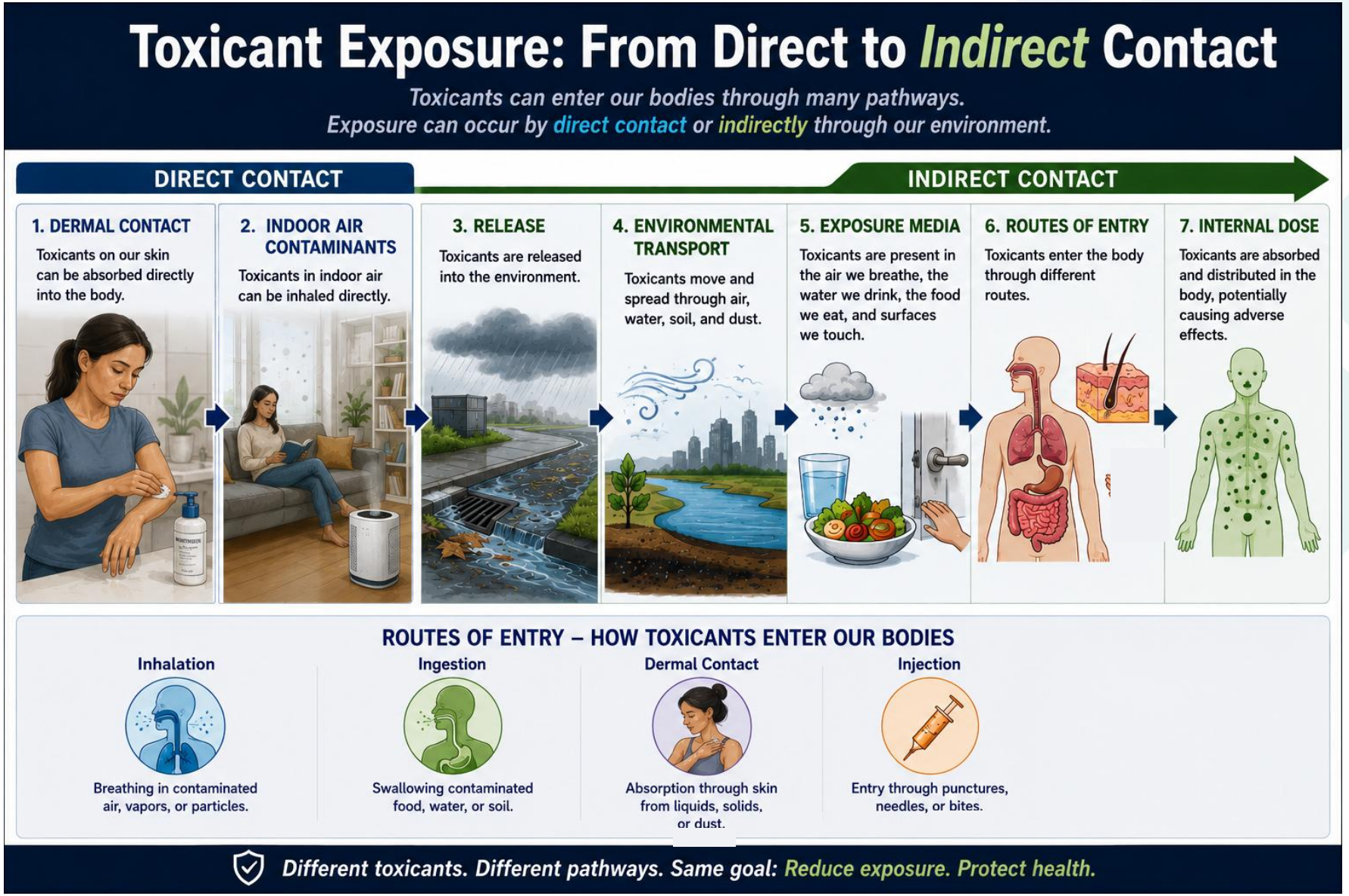
https://www.cdc.gov/exposurereport/
https://www.integrativepractitioner.com/practice-management/news/environmental-medicine-expert-shares-top-10-toxic-compound-list
https://www.cdc.gov/niosh/topics/indoorenv/moldsymptoms.html; https://www.cdc.gov/mold/faqs.htm#;
https://www.euro.who.int/__data/assets/pdf_file/0017/43325/E92645.pdf

Sources of Exposure



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Toxicant testing with TOXDetect

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TOXDetect PROFILE

The TOXDetect Profile is a **urinary assessment** of exposure to environmental toxicants. The profile measures **26 metabolites** to screen for exposure to **34 various toxicants**.

- Measure metabolites to assess parent compounds (environmental toxicants)
- Can be used alongside an OAT to evaluate metabolic impacts from the toxicants, in addition to additional toxicants.

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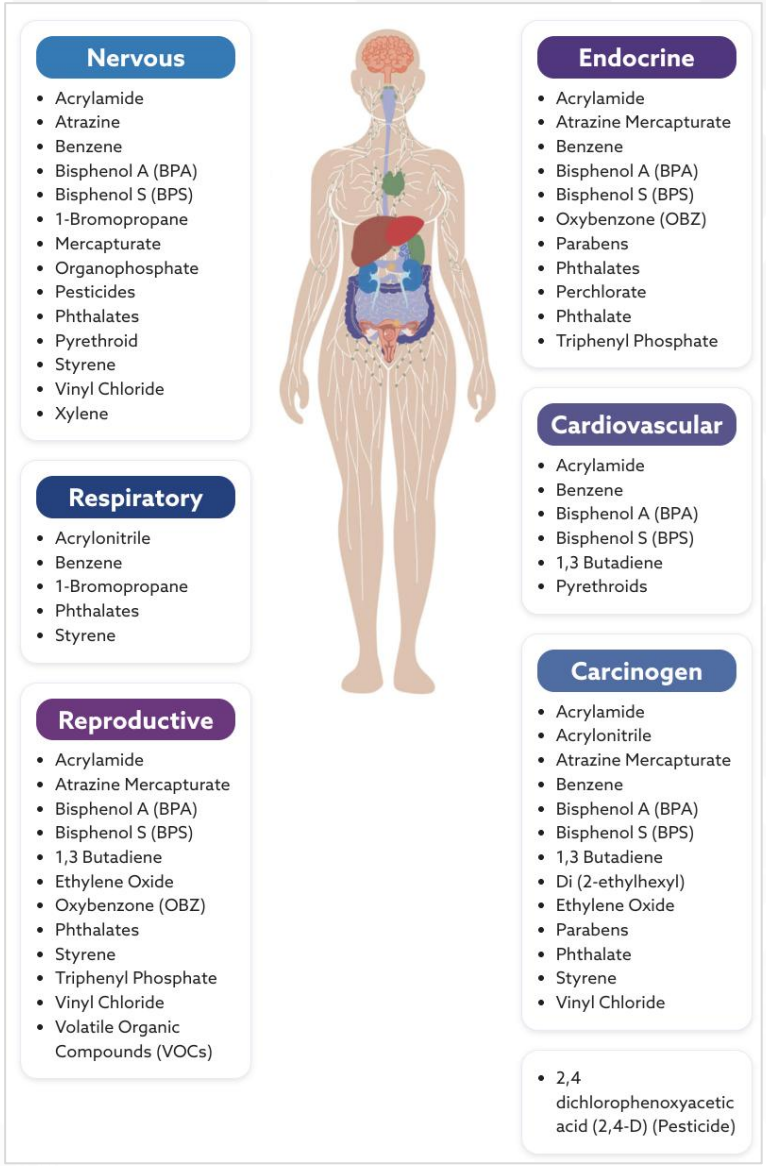
Toxicants Measured

- **Phthalates** (5)- plastics
- **Bisphenols** (BPA and BPS)
- **Volatile Organic Compounds** (VOCs) (9)
  - E.g. xylene, styrene/ethylbenzene, benzene, toluene, acrylonitrile, 1 –bromopropane, 1,3-butadiene, ethylene oxide, vinyl chloride.
- Parabens (4)- preservatives
- Pesticides (including **Organophosphates, Pyrethroids, Glyphosate**, 2,4-D, and Atrazine)
- Flame Retardants
- **Acrylamide-** cooking biproduct
- Perchlorate – bleach
- Oxybenzone - sunscreen



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https://mosaicdx.com/resource/toxdetect-profile-important-clinical-considerations/

TOXDetect PROFILE

REQUISITION #	9900001	COLLECTION TIME	
PATIENT NAME	Report Sample	COLLECTION DATE	
DATE OF BIRTH	Apr 10, 2005	SAMPLE TYPE	
GENDER	M	REPORT DATE	
PRACTITIONER	NO PHYSICIAN		

Summary of Elevated Results

The results below lists metabolites with elevated results detected in the profile. Yo
results and a more detailed description of each metabolite starting on the TOXDete
section. Please note that each value in the report needs to be considered in the cor
health and environment. Contact a qualified healthcare provider for further assista
of results.

Color Key LOW MODERATE HIGH

Creatinine Value: * 100.00 mg/dl

METABOLITE	RESULTS
Parent	ug/g creatinine

HIGH RESULTS

		75th
6) 2-3-4 Methylhippuric Acid (2,-3-,4-MHA) Xylene	1,603.00	208.00
12) 2-Hydroxyethyl Mercapturic Acid (HEMA) Ethylene Oxide, Vinyl Chloride	6.20	1.86
13) 2,4-Dichlorophenoxyacetic Acid (2,4-D) 2,4-Dichlorophenoxyacetic Acid (2,4-D)	5.10	0.58
16) Diphenyl Phosphate (DPP) Triphenyl Phosphate	8.00	1.63
17) N-Acetyl (Carbomethyl) Cysteine Acrylamide	220.00	93.40
18) Perchlorate (PERC) Perchlorate	16.00	4.01

*Methodology: LC-MS/MS. *The creatinine test is performed to adjust metabolic marker results for differences in fluid intake. Urinary creatinine, from a diagnostic value due to variability as a result of recent fluid intake. The results should be interpreted in conjunction with the complete clinical picture, given patient history and presentation, and at the discretion of the*

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Mosaic Diagnostics | 9221 Quivira Road, Overland Park, KS 66215 | MosaicDX.c
Dr. L. G. Bates-Dubrow, PhD, CC(NRCC) | CLIA 17D0919496 | © 2024 Mosaic D
This test was developed and its performance characteristics determined by Mosaic Diagnostics Laboratory. It has not been cleared or approved by the U

TOXDetect PROFILE — Results - Continued

Color Key LOW MODERATE HIGH

Creatinine Value: * 100.00 mg/dl

METABOLITE	RESULTS	PERCENTI
Parent	ug/g creatinine	75% | 95

VOC - VOLITILE ORGANIC COMPOUNDS

		75th	95th
6) 2-3-4 Methylhippuric Acid (2,-3-,4-MHA) Xylene	1,603.00	256.70	1,205.00
7) Phenylglyoxylic Acid (PGO) Styrene/Ethylbenzene	120.00	306.00	509.00
8) N-Acetyl Phenyl Cysteine (NAP) Benzene	2.80	1.35	2.98

Parent Compound: Xylene
Xylene is widely used in industry and medical laboratories. Xylene is released primarily from industrial sour
One can also come in contact with xylene through automobile exhaust and a variety of consumer products
cigarette smoke, paints, varnish, rust preventives, and shellac. Literature suggests that xylene exposure ca
toxic effects on various systems of the body. Longer term effects can damage the liver and kidneys.

Parent Compound: Styrene/Ethylbenzene
Styrene is widely used to make plastics and rubber, which are used to manufacture a variety of products, su
insulation, pipes, automobile parts, printing cartridges, food containers, and carpet backing. Exposure may
through ingestion via transfer to foods, especially fatty foods heated in styrene containers, through breathi
indoor air that has styrene vapors from building materials, photocopiers, tobacco smoke, and other produc
Styrene and styrene oxide have been implicated as reproductive toxicants, neurotoxicants, and linked to an
increased risk of leukemia and lymphoma.

Parent Compound: Benzene
Benzene has been used extensively in the past as an industrial solvent; however, due to its toxicity and pot
health hazards, its use has been reduced. Exposure can occur occupationally, in the general environment a
the home as a result of the ubiquitous use of benzene-containing petroleum products, including motor fuel
solvents. Benzene exposure has been linked to respiratory, hepatic, cardiovascular, immune, nervous, and
endocrine system dysfunction.

Interpretation Continued

VOC - VOLITILE ORGANIC COMPOUNDS

Color Key LOW MODERATE HIGH

2-3-4 Methylhippuric Acid (2,-3-,4-MHA) Xylene	1,603.00	N-Acetyl Phenyl Cysteine (NAP) Benzene	2.80
Phenylglyoxylic Acid (PGO) Styrene/Ethylbenzene	120.00		

METHYLHIPPURIC ACID (2,-3-,4-MHA)

Is a metabolite generated as a result of exposure to xylene, an aromatic hydrocarbon widely used in industry and medical laboratories. It is used extensively as a solvent in the rubber, printing, and leather industries. It is also used as a thinner for paints, cleaning agents, and varnishes. Xylene is released primarily from industrial sources. One can also come in contact with xylene through automobile exhaust and a variety of consumer products such as cigarette smoke, paints, varnish, rust preventives, and shellac. Literature suggests that xylene exposure causes toxic effects on various systems of the body. Central nervous system toxicity may lead to headaches, irritability, depression, insomnia, agitation, extreme tiredness, tremors, impaired concentration, and damage to short-term memory. Longer term effects can damage the liver and kidneys. Xylene is primarily eliminated through metabolism in the liver and subsequent excretion of 70-80% of metabolites in urine within 24 hours after exposure. Xylene is metabolized in the liver by side-chain (CH3) dehydroxylation, finally forming the metabolite methylhippuric acid.

PHENYLGLYOXYLIC ACID (PGO)

Is a metabolite generated as a result of exposure to styrene/ethylbenzene widely used to make plastics and rubber, which are used to manufacture a variety of products, such as insulation, pipes, automobile parts, printing cartridges, food containers, and carpet backing. Exposure occurs through breathing indoor air that has styrene vapors from building materials, photocopiers, tobacco smoke, and other products. Styrene may also leach from polystyrene containers used for food products, especially when food is heated in these containers. Short term exposure can cause CNS depression and skin and respiratory irritation. Long term exposure can damage the reproductive system and cause problems such as infertility and birth defects, can cause neurological damage such as memory loss, difficulty concentrating, and can cause impaired motor function. Exposure to PGO has been linked to an increased risk of leukemia and lymphoma. In the liver, styrene is metabolized to styrene-7,8-oxide (SO) by cytochrome P-450 enzymes. SO can then be further metabolized to styrene glycol, mandelic acid, and phenylglyoxylic acid, which are excreted in the urine. Glutathione conjugation is also a significant pathway for detoxification.

N-ACETYL PHENYL CYSTEINE (NAP)

Is a metabolite generated as a result of the exposure to benzene, an industrial solvent. Its use has been reduced due to toxicity and potential health hazards. Exposure has been associated with a range of acute and long-term adverse health effects and diseases, including cancer and hematological effects. Exposure can occur occupationally, in the general environment and in the home as a result of the ubiquitous use of benzene-containing petroleum products, including motor fuels and solvents. Active and passive exposure to tobacco smoke is also a significant source of exposure. Benzene exposure has been linked to respiratory, hepatic, cardiovascular, immune, nervous, and endocrine system dysfunction. High exposure to benzene may cause nausea, vomiting, dizziness, poor coordination, central nervous system depression, and even death. 22,23 The metabolism of benzene is complex and involves multiple enzymatic pathways. Benzene is primarily metabolized in the liver by the cytochrome P450 enzyme system. It undergoes oxidation to form several metabolites. These metabolites can further undergo conjugation with glucuronic acid or sulfate to form more water-soluble compounds that can be excreted in urine.

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Formerly Great Plains Laboratory

HOW THE BODY METABOLIZES ENVIRONMENTAL TOXICANTS

Review 95 references at MosaicDX.com/resource/how-the-body-metabolizes-environmental-toxicants-metabolite-chart

PHTHALATES

PARENT COMPOUND	METABOLISM OF PARENT COMPOUND	LIPOPHILIC? YES/NO	HALF-LIFE	METABOLISM IN HUMANS	ADDITIONAL DETAILS
1-5. Phthalates	1-5. Diethylphthalates Di-n-butyl Phthalate (DBP) Di(2-ethylhexyl) Phthalate (DEHP)	Yes	**1.9-3.5 hours** for most metabolites.[1]	***Phase I:*** Undergoes hydrolysis to form monoester phthalates, catalyzed by CES1 and CES2.[2] Then, it undergoes oxidative metabolism in the liver via CYP2C9, CYP2C19, and CYP3A4.[2] ***Phase II:*** Metabolites are conjugated with glucuronic acid and excreted via urine.[3]	**Sweating** can help facilitate elimination.[3] May negatively impact **mitochondrial** function and the human **microbiome**[4,5] (may consider **Organic Acid Test (OAT)** and **Comprehensive Stool Analysis (CSA)**). Supporting microbiome with **probiotics** may decrease toxicity.[5]

BISPHENOLS

PARENT COMPOUND	METABOLISM OF PARENT COMPOUND	LIPOPHILIC? YES/NO	HALF-LIFE	METABOLISM IN HUMANS	ADDITIONAL DETAILS
6. Bisphenol A (BPA)	6. Bisphenol A (BPA)	Yes	Oral exposure: 4.4-8.4 hrs [6] Dermal exposure: 11.1-30.2 hrs [7]	***Phase I:*** involves CYP450, specifically SPY3A4 [11] ***Phase II:*** Primarily involves conjugation reactions, including glucuronidation (UGTs) and sulfation (SULTs).[9,10]	
7. Bisphenol S (BPS)	7. Bisphenol S (BPS)	Yes	6.8-7.9 hours	***Phase I:*** CYP3A4 and 2C9 are involved in hydroxylation but phase 2 is the primary metabolic pathway of BPS.xi ***Phase II:*** Glucuronidation and sulfation of BPS facilitate detoxification and excretion via urine.[12] These metabolites are generally considered inactive (related to endocrine disruption) compared to the parent compound.[13]	

VOC'S

PARENT COMPOUND	METABOLISM OF PARENT COMPOUND	LIPOPHILIC? YES/NO	HALF-LIFE	METABOLISM IN HUMANS	ADDITIONAL DETAILS
8. Xylene	8. 2-3-4-Methylhippuric acid (2,3,4-MHA)	Yes	**Rapid initial phase: about 1 hour. Slower phase: 20-58 hours depending on the tissue.**	**Phase I:** Involves methyl hydroxylation in the liver via CYP2E1 to form MHA.[14] Then, excreted via urine.[15]	Absorbed primarily through **inhalation,** with about **60% retained in the lungs.**[16] Can cause **mitochondria damage** and deplete **glutathione** (may consider **OAT**).[17]
9.Styrene/ Ethylbenzene	9. Phenylglyoxylic Acid (PGO)	Yes	**1-10 hours**	***Styrene Phase I:*** Oxidation via **CYP2E1** to form **styrene-7,8-oxide (SO).**[18] Then converted by **mEH** to form **phenylglyoxylic acid, mandelic acid**, and **hippuric acid.** ***Styrene Phase II:*** SO undergoes **glutathione (GSH)** conjugation to form **PGO.**[19] ***Ethylbenzene:*** Hydroxylation by **CYP2E1,** mainly, but can also go through **CYP1A2** and **CYP2B6** to a lesser degree.[20]	Variability in **CYP2E1** activity for ethylbenzene metabolism may be due to **genetic polymorphisms** and environmental factors.[9] **Mandelic** and **Hippuric** acids are intermediates of Styrene's breakdown in the body. (may consider OAT).[21]
10. Benzene	10. N-Acetyl Phenyl Cysteine (NAP)	Yes	**8 hours**	***Phase I:*** Oxidation of benzene to benzene oxide (BO) via CYP2E1. BO can be further metabolized through several pathways.[22] ***Phase II:*** GSH conjugation results in formation of NAP.[23]	**Green tea** can decrease oxidative stress from exposure.[22] **Broccoli sprouts** can enhance detoxification.[23] Can be stored in bone marrow, causing a decrease in **red blood cells and anemia.**[24]
11. Acrylonitrile (ACN)	11. N-Acetyl (2-Cyanoethyl) Cysteine (NACE)	Yes	**3-18 hours**	***Phase I:*** Oxidation through ACN to CNEO via CYP2E1 and then further degrade to cyanide.[25] ***Phase II:*** ACN conjugates with glutathione to form NACE and excreted via urine.[26]	**Fasting** may enhance CYP2E1-mediated oxidative metabolism of ACN and decrease liver GSH levels, potentially increasing the toxicity of exposures.[27]
12. 1-bromopropane (1-BP)	12. N-Acetyl (Propyl) Cysteine (NAPR)	Yes	**6-8 hours**	**1-BP** Undergoes two pathways: CYP450-mediated oxidation and GSH conjugation. ***Phase I:*** CYP2E1 oxidation leads to formation of 1-bromo-2-propanol and bromoacetone. ***Phase II:*** Conjugate with GSH to form various mercapturic acids including NAPR.[20]	Elimination can be accelerated with **GSH (reduced)** oral, IV, transdermal or **N-acetylcysteine (NAC)** supplementation. Observed effects on neurotransmitter metabolites (**DOPAC, HVA,** and **5HIAA**)[32] (may consider **OAT**).
13. 1,3 butadiene	13. N-Acetyl (3,4-Dihydroxybutyl) Cysteine (NADB)	Yes	**Variable; few minutes - few hours**	***Phase I:*** Oxidation by CYP2E1 and 2A6 (at increased concentrations) to form epoxide, diepoxybutane, and epoxybutane diol. ***Phase II:*** Several transformations involving GSH conjugation, forming mercapturic acids to be excreted via urine.[31]	Inhalation is the predominant route of exposure; with **half exhaled** and the other half metabolized via liver and excreted in urine.[32] **GSTT1 polymorphisms** influence metabolism and detoxification.[33]
14. Ethylene Oxide (EO), Vinyl Chloride (VC)	14. 2-Hydroxyethyl Mercapturic Acid (HEMA)	EO: somewhat more hydrophilic, VC: Yes	**EO: 42-84 minutes; VC: 1-2 days**	***EO Phase I:*** Metabolized by CYP2E1 in liver, then undergoes hydrolysis by epoxide hydrolase. ***EO Phase II:*** Conjugation with GSH mediated by Glutathione S-transferase (GST).[34] ***VC Phase I:*** Also metabolized by CYP2E1 to form CEO and CAA, and undergoes hydrolysis. ***VC Phase II:*** GSH conjugation.[35,36]	**EO** can convert to ethylene glycol, then to **oxalic acid** (may consider **OAT**).[27,28] **VC** can cross the placenta and enter the **fetus.**[30]

MOSAIC DIAGNOSTICS

PARABENS

PARENT COMPOUND	METABOLISM OF PARENT COMPOUND	LIPOPHILIC? YES/NO	HALF-LIFE	METABOLISM IN HUMANS	ADDITIONAL DETAILS
15-18. Parabens	**15-18. Methylparaben (MeP) Ethylparaben (EtP) Propylparaben (PrP) Butylparaben (BuP)**	Yes	Depends on the specific type of paraben but ranges from **7.7-20.3 hrs**[41] **MeP: 12.2 hrs, EtP: 12.0 hrs, Prp: 9.3 hrs.**[42]	**Phase I:** Parabens undergo hydrolysis by carboxylesterases (CES1 mainly in the liver and CES2 is more active in the skin).[43,44] **Phase II:** Primarily undergo glucuronidation via UGTs.[45]	

PESTICIDES

PARENT COMPOUND	METABOLISM OF PARENT COMPOUND	LIPOPHILIC? YES/NO	HALF-LIFE	METABOLISM IN HUMANS	ADDITIONAL DETAILS
19. Atrazine Mercapturate	**19. Atrazine Mercapturate**	Yes	**12 hrs**[46]	***Phase I:*** Involves CYP450, particularly CYP1A2, leading to the formation of various metabolites.[47] ***Phase II:*** Key enzymes in phase II are GST, specifically the pi class GST (hGSTP1-1).[48]	hGSTP1-1 catalyzes the conjugation of atrazine with glutathione, forming atrazine mercapturate, which is then excreted.[48]
20. 2,4-Dichlorophenoxyacetic Acid (2,4-D)	**20. 2,4-Dichlorophenoxyacetic Acid (2,4-D)**	No, but chemical modifications can increase its lipophilicity	**11.6 hours from plasma and 17.7 hours for urinary excretion.**	Metabolism of **2,4-D** is minimal and largely excreted as the unchanged parent compound – about **82% unchanged** and **12% is excreted as a conjugate.**[49]	**2,4-D** can form reactive metabolites, such as **2,4-D-CoA**, which can covalently bind to proteins, potentially contributing to **hepatotoxicity**. This binding occurs through the formation of **2,4-D-S-acyl-glutathione (2,4-D-SG)** thioester and covalent adducts with human serum albumin.[50]
21. Pyrethroids	**21. 3-Phenoxybenzoic Acid (3PBA)**	Yes	**5-8 hours with 88% excreted in urine within 24 hrs of exposure.**	***Phase I:*** Undergo extensive metabolism in humans through hydrolysis and oxidation. **CES1** and **CES2** play a role in hydrolysis.[51] Many **CYP450** enzymes, but especially **CYP2C19**, exhibit highest intrinsic clearance of pyrethroid metabolites.[52] ***Phase II:*** Metabolites formed in Phase I reactions, such as **3PBA,** may undergo further conjugation with amino acids, sulfates, and sugars.[53]	**Pyrethroids** have the ability to influence **glucose metabolism** and lipid oxidation.[54] (may consider **OAT**)
22. Organophosphates (OPs)	**22. Diethylphosphate (DEP)**	Yes	**Depends on type of organophosphate but can vary between 18 hours - 7 days.**	**Bioactivation:** Involves oxidative desulfuration to form **oxon metabolites** – potent AChE inhibitors. Key CYP enzymes involved are **CYP2B6, 2C19, and 1A2.**[55] Detoxification: Involves dearylation of OPs to form inactive metabolites. **CYP2C19 and 3A4** significantly contribute to this pathway, but **2C19** is involved in detoxification of **chlorpyrifos** and **diazinon**, converting them to non-toxic metabolites.[56]	**Lactobacillus rhamnosus** may help decrease toxic OP pesticide exposure by passive binding.[57] **Lactobacillus casei** may decrease OP-induced cytotoxicity.[58] **Paraoxonase 1 (PON1)** is involved in the hydrolysis of certain OPs. Polymorphisms in this enzyme can increase susceptibility to toxicity.[59] **PON1** can be upregulated by **pomegranate juice.**[60]

OTHER

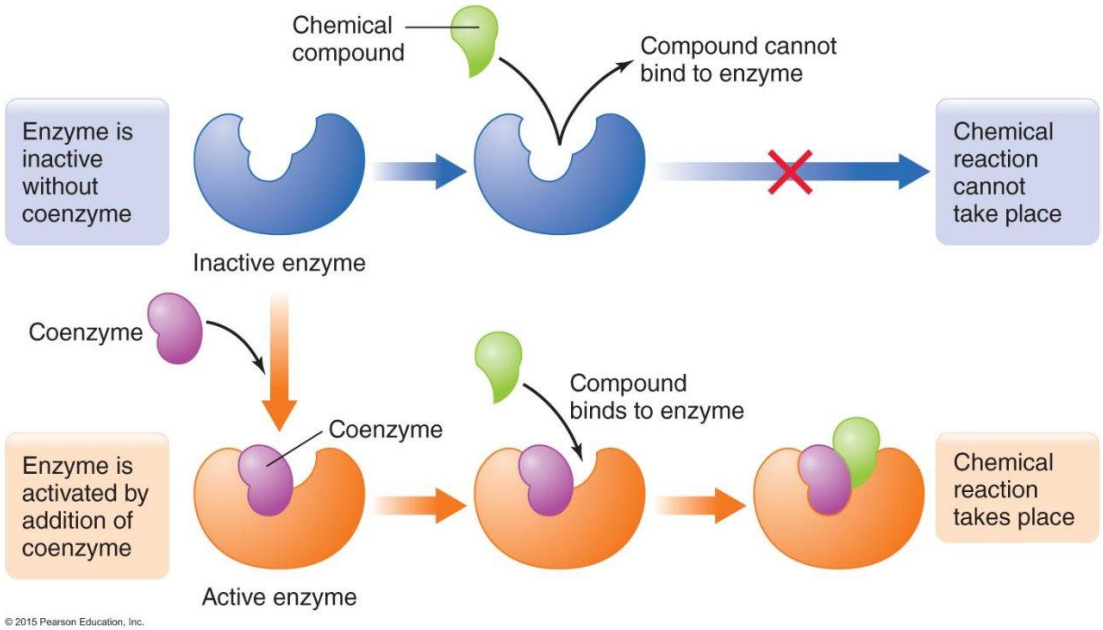
PARENT COMPOUND	METABOLISM OF PARENT COMPOUND	LIPOPHILIC? YES/NO	HALF-LIFE	METABOLISM IN HUMANS	ADDITIONAL DETAILS
23. Triphenyl Phosphate (TPHP)	**23. Diphenyl Phosphate (DPP)**	Yes	**7.6 days**	***Phase I:*** TPHP undergoes O-dearylation to form DPP and hydroxylation to produce mono- and dihydroxylated metabolites with CYP1A2 and 2E1 playing a significant role.[61] ***Phase II:*** hydroxylated metabolites undergo conjugation, such as glucuronidation, to form glucuronide conjugates to be more water-soluble.[62]	
24. Acrylamide	**24. 2N-Acetyl (Carbomethyl) Cysteine (NAE)**	No	**2.5 hours for initial phase and longer second phase extending up to several days.**	**Glutathione Conjugation:** acrylamide + GSH forms NAE, which is then excreted in the urine. This accounts for the majority of acrylamide metabolism (75-86%).[63] **Oxidation to Glycidamide:** this is done via CYP2E1. Glycidamide is considered a more reactive and potentially genotoxic metabolite. [64]	The relative contribution of these pathways can vary based on individual factors such as genetic polymorphisms in **CYP2E1** and **GSTs**, as well as external factors like diet and exposure levels.[65] GSH precursors such as NAC and methionine have been shown to protect against the cytotoxicity of AA and other metabolites.[66]
25. Perchlorate	**25. Perchlorate (PERC)**	No	**8 hours**	Perchlorate is not metabolized in the human body. Instead, it is absorbed and excreted largely unchanged. [65] It competitively inhibits the sodium-iodide symporter in the thyroid gland, blocking iodide intake (which is essential for thyroid hormone synthesis). [66]	In breastfed infants, there is evidence suggesting **bifidobacteria** in the gut may reduce perchlorate via perchlorate reductase.[69]
26. Oxybenzone	**26. Oxybenzone (OBZ)**	Yes	**1.5 - 2 days**[70]	***Phase I:*** oxybenzone (BP-3) is primarily metabolized by CYP450 enzymes through hydroxylation and demethylation, producing BP-1 and BP-8.[71] ***Phase II:*** the hydroxylated metabolites are conjugated with glucuronic acid or sulfate, making them more water-soluble for excretion.[72]	Oxybenzone is a common ingredient in sunscreens and other personal care products. Its lipophilicity facilitates its penetration through skin and subsequent systemic absorption.[73]

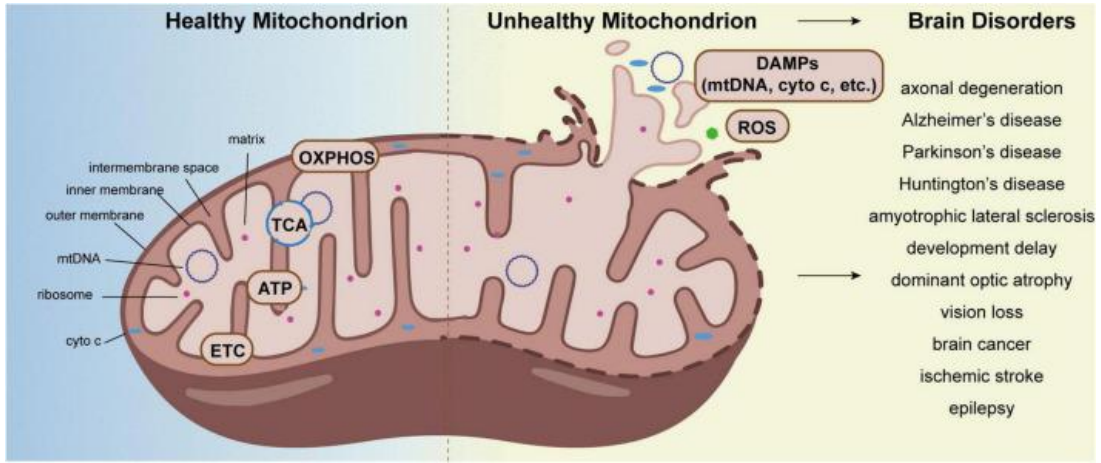
MOSAIC DIAGNOSTICS

Organic Acids

MOSAIC DIAGNOSTICS

What are Organic Acids?





- **Acidic byproducts of cellular metabolism** that are produced by living organisms—including humans, bacteria, and fungi—and are typically **excreted in urine.**

- Downstream products of various **metabolic pathways**, that give insight into microbial activity, energy production, neurotransmitter metabolism, toxic exposure, methylation and detoxification, and nutrient needs.

Adapted from: Mitochondrial quality control in the brain: The physiological and pathological roles. - Abstract - Europe PMC

6 Key Clinical Categories

01.
Microbial Overgrowth

Markers of yeast, mold, bacteria, Clostridium species, and their metabolic byproducts.

02.
Mitochondrial Health

Intermediates of glucose, fatty acid, and amino acid metabolism involved in energy production.

03.
Neurotransmitter Metabolites

Dopamine, serotonin, and norepinephrine/epinephrine pathway metabolites, providing insights into neurotransmitter imbalances.

04.
Toxic Exposure

Provides insights into metabolites influenced by mold toxicant, heavy metal exposures, and associated oxidative stress and glutathione depletion.

05.
Methylation/Detoxification

Metabolites reflecting methyl group cycling, glutathione status, and nutrients involved in both methylation and detoxification.

06.
Nutrient Needs

Direct and indirect biomarkers reflecting the functional demand for B-vitamins, antioxidants, and other key micronutrients.

MOSAIC DIAGNOSTICS

OAT + Toxic Exposures

MOSAIC DIAGNOSTICS

Impacts from Toxicants

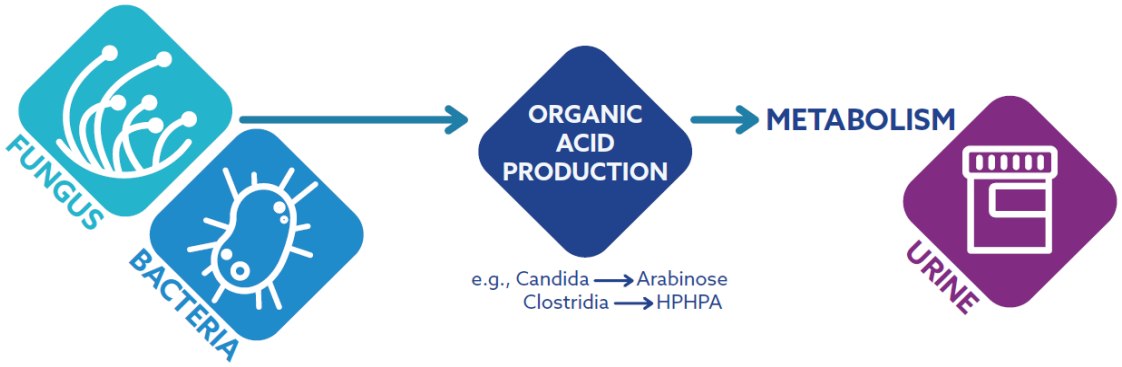
- There can be numerous effects from toxic exposure that can be revealed
  - Microbiome, mitochondria, neurotransmitters, methylation and detoxification.

Add Additional Insight into specific toxins and toxicants

- Certain organic acids can be produced from the metabolism of specific toxicants.
  - Hippuric acid (marker 10) = Toluene
- Specific ones can directly influence it.
  - Quinolinic acid (Marker 39)and phthalates

Microbial Overgrowth

METABOLITE	REFERENCE RANGE	RESULTS (mmol/mol creatinine)	REFERENCE POPULATION Females Under Age 13
INTESTINAL MICROBIAL OVERGROWTH			
YEAST AND FUNGAL			
1 Citramalic	≤ 3.6	1.6	
2 5-Hydroxymethyl-2-furoic *(Aspergillus)*	≤ 14	14	
3 3-Oxoglutaric	≤ 0.33	0.23	
4 Furan-2,5-dicarboxylic *(Aspergillus)*	≤ 16	8.1	
5 Furancarbonylglycine *(Aspergillus)*	≤ 1.9	H 15	
6 Tartaric	≤ 4.5	H 6.2	
7 Arabinose	≤ 29	H 69	
8 Carboxycitric	≤ 29	12	
9 Tricarballyic *(Fusarium)*	≤ 0.44	H 0.55	
BACTERIAL			
10 Hippuric	≤ 613	H 1,340	
11 2-Hydroxyphenylacetic	0.06-0.66	0.53	
12 4-Hydroxybenzoic	≤ 1.3	1.2	
13 4-Hydroxyhippuric	0.79-17	8.7	
14 DHPPA *(Beneficial Bacteria)*	≤ 0.38	H 0.57	
CLOSTRIDIA BACTERIAL			
15 4-Hydroxyphenylacetic *(C. difficile, C. stricklandii, & others)*	≤ 19	15	
16 HPHPA *(C. sporogenes, C. botulinum & other)*	≤ 208	162	
17 4-Cresol *(C. difficile)*	≤ 75	37	
18 3-Indoleacetic *(C. stricklandii, C. subterminale & others)*	≤ 11	2.9	
OXALATE METABOLITES			
19 Glyceric	0.77-7.0	H 7.6	
20 Glycolic	16-117	89	
21 Oxalic	6.8-101	H 224	



Microbial Overgrowth Section

- **Yeast Markers (*Candida*)**
  - Numerous toxicants impact the microbiome, allowing for dysbiosis and opportunistic organisms to thrive, in particular *Candida*. PMID: 36274693

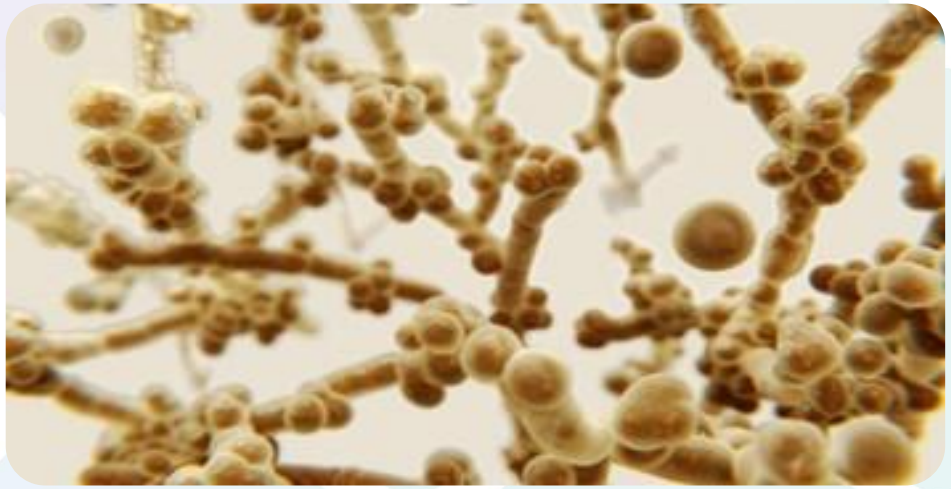


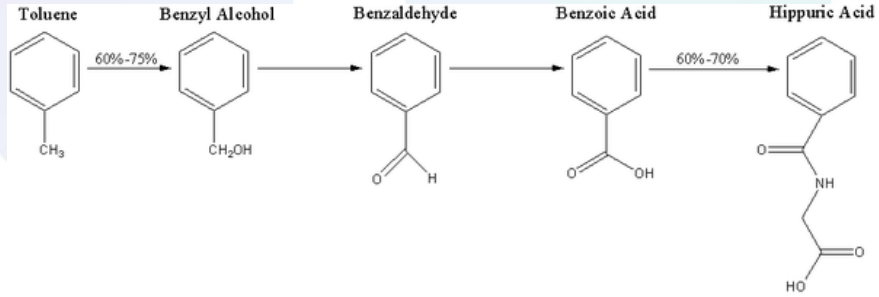
Image used from: Candida Albicans Stock Photos

METABOLITE		REFERENCE RANGE		RESULTS (mmol/mol creatinine)	REFERENCE POPULATION Females Under Age 13
INTESTINAL MICROBIAL OVERGROWTH					
YEAST AND FUNGAL	1 Citramalic	≤5.3		5.3	5.3
	2 5-Hydroxymethyl-2-furoic *(Aspergillus)*	≤30	H	68	68
	3 3-Oxoglutaric	≤0.52		0.51	0.51
	4 Furan-2,5-dicarboxylic *(Aspergillus)*	≤22	H	27	27
	5 Furancarbonylglycine *(Aspergillus)*	≤3.6		1.4	1.4
	6 Tartaric *(Aspergillus)*	≤3.9		1.2	1.2
	7 Arabinose	≤56	H	105	105
	8 Carboxycitric	≤34		0.54	0.54
	9 Tricarballylic *(Fusarium)*	≤0.86		0.16	0.16

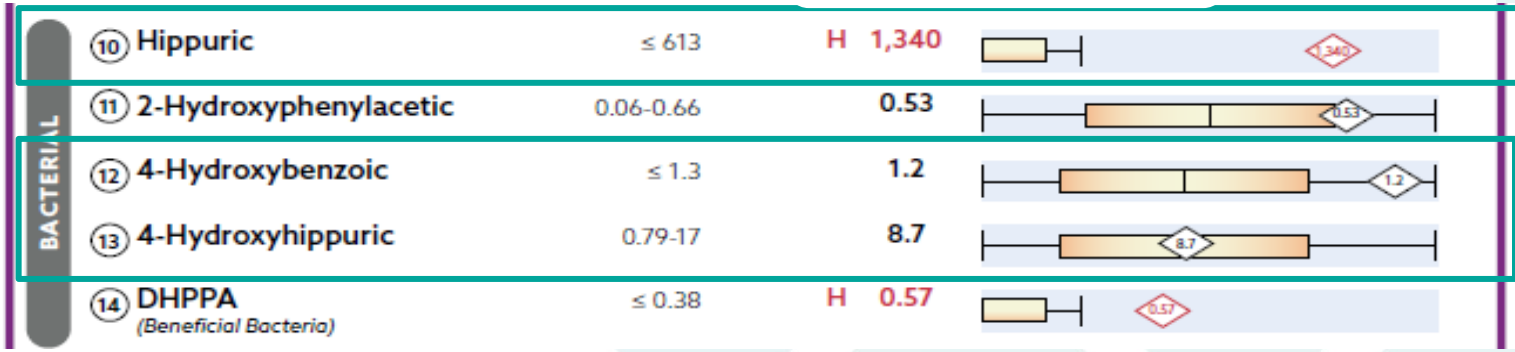
Microbial Overgrowth Section

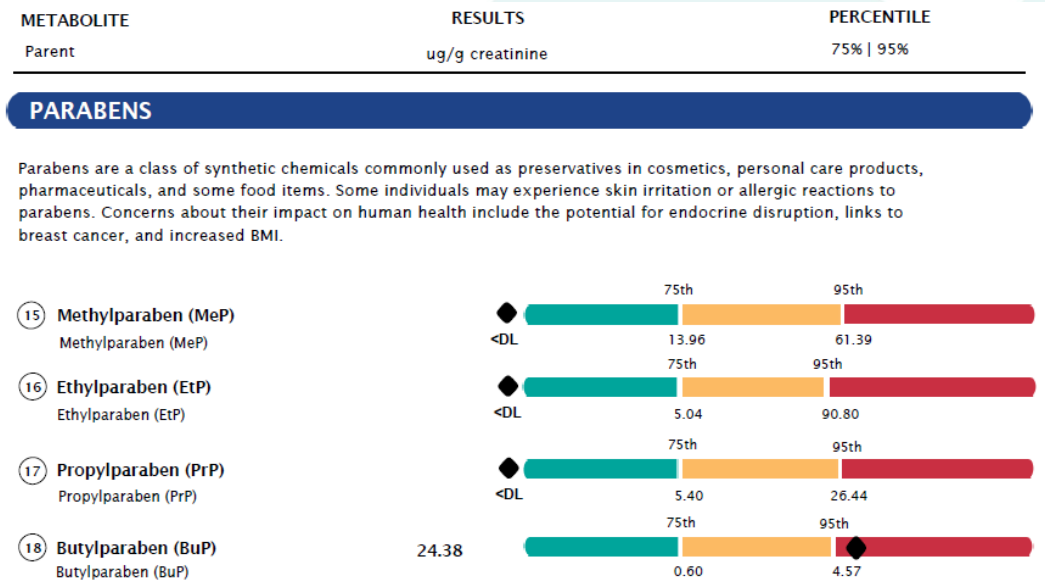
- **Bacterial Markers**
  - Hippuric acid (10) = **Toluene**
    PMID: 34696600
  - 4-Hydroxybenzoic and 4-Hydroxyhippuric acids (12,13) = **Parabens**

PMID: 20930423



Source: Techno Science



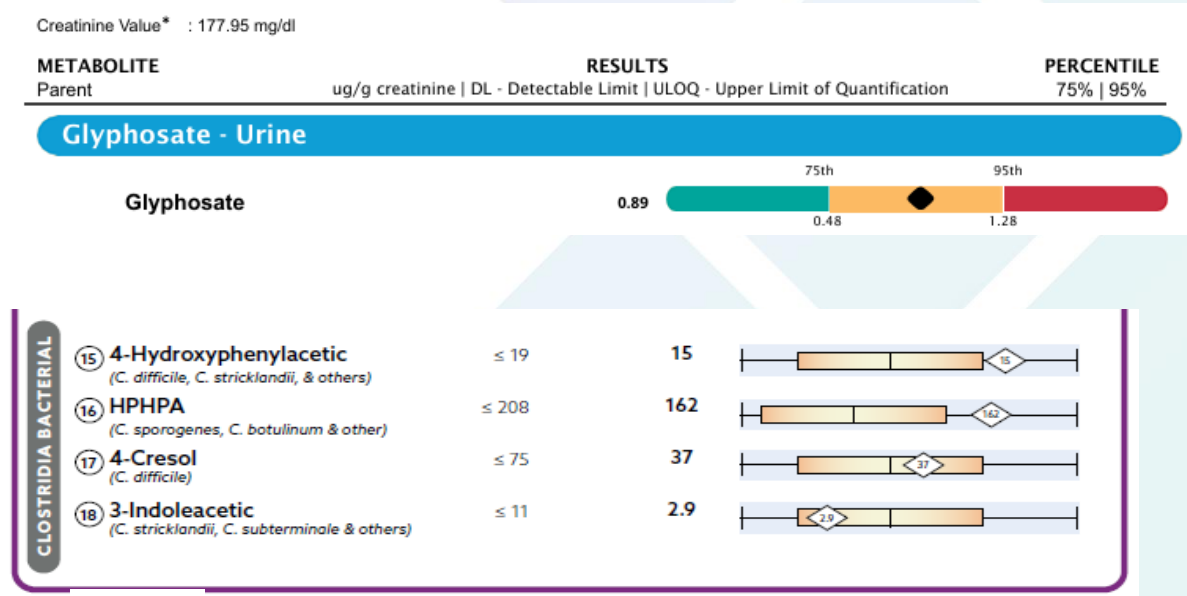


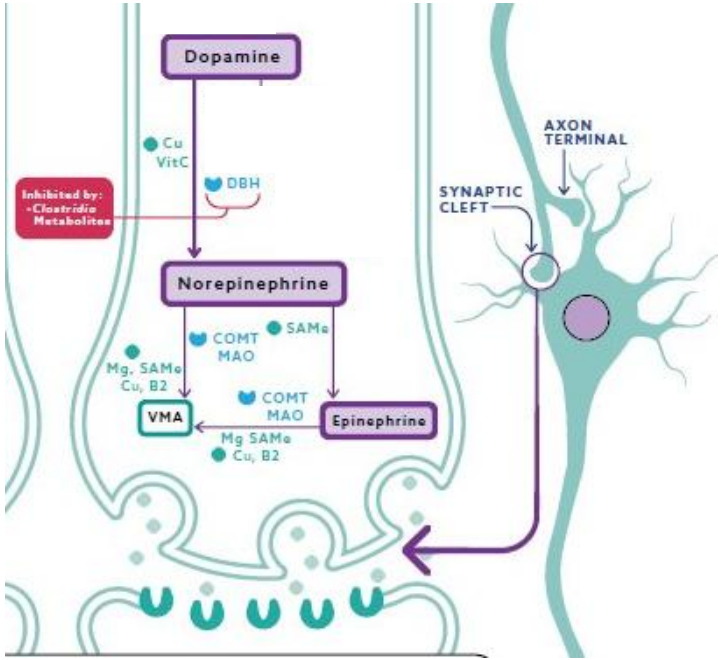
Microbial Overgrowth Section

- **Clostridium** Bacterial Markers
  - Impacted by Glyphosate
    - Glyphosate inhibits 5-enolpyruvylshikimate-3-phosphate synthase (EPSPS), selectively suppressing beneficial gut bacteria while Clostridia remain relatively resistant.

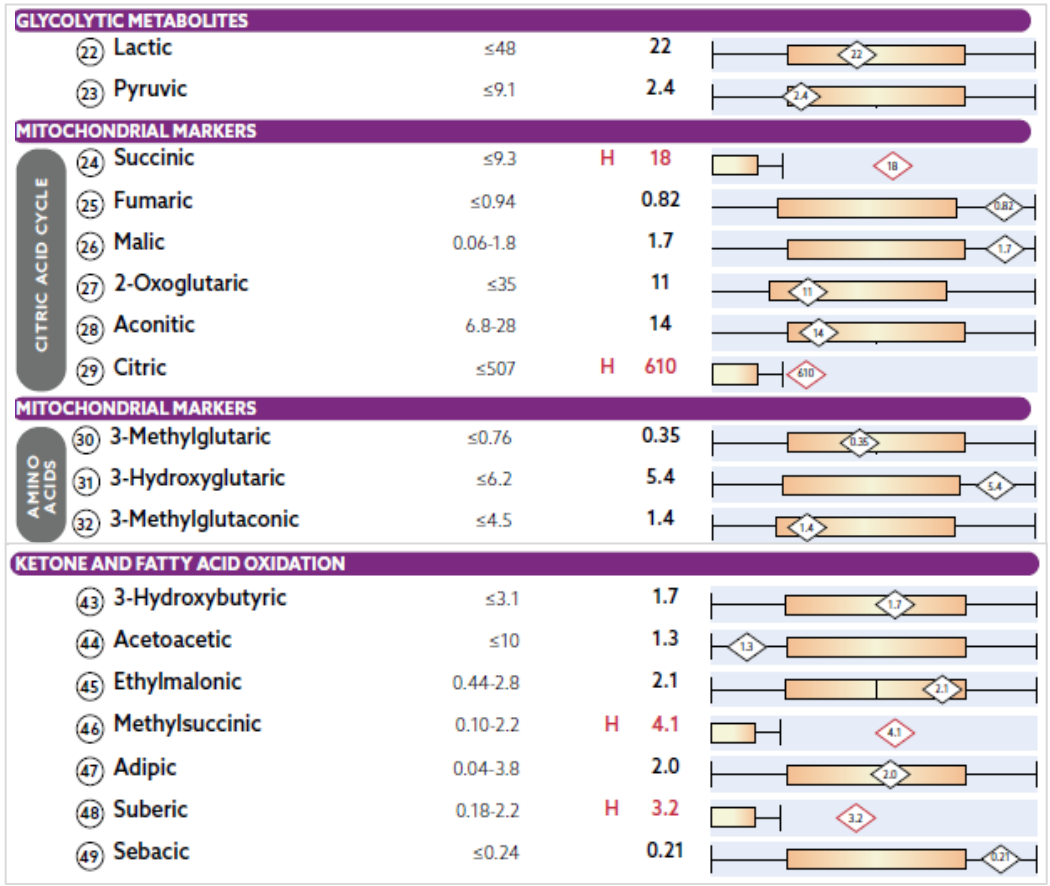
PMID: 38099711 , PMID: 38244513, PMID: 31442459

Clostridia metabolites (15-18) impact Neurotransmitters by blocking dopamine beta hydroxylase (DBH). PMID: 35323033, PMID:37363147





Mitochondrial Health



GLYCOLYTIC METABOLITES				
22	Lactic	≤48		22
23	Pyruvic	≤9.1		2.4
MITOCHONDRIAL MARKERS				
CITRIC ACID CYCLE				
24	Succinic	≤9.3	H	18
25	Fumaric	≤0.94		0.82
26	Malic	0.06-1.8		1.7
27	2-Oxoglutaric	≤35		11
28	Aconitic	6.8-28		14
29	Citric	≤507	H	610
MITOCHONDRIAL MARKERS				
AMINO ACIDS				
30	3-Methylglutaric	≤0.76		0.35
31	3-Hydroxyglutaric	≤6.2		5.4
32	3-Methylglutaconic	≤4.5		1.4
KETONE AND FATTY ACID OXIDATION				
43	3-Hydroxybutyric	≤3.1		1.7
44	Acetoacetic	≤10		1.3
45	Ethylmalonic	0.44-2.8		2.1
46	Methylsuccinic	0.10-2.2	H	4.1
47	Adipic	0.04-3.8		2.0
48	Suberic	0.18-2.2	H	3.2
49	Sebacic	≤0.24		0.21

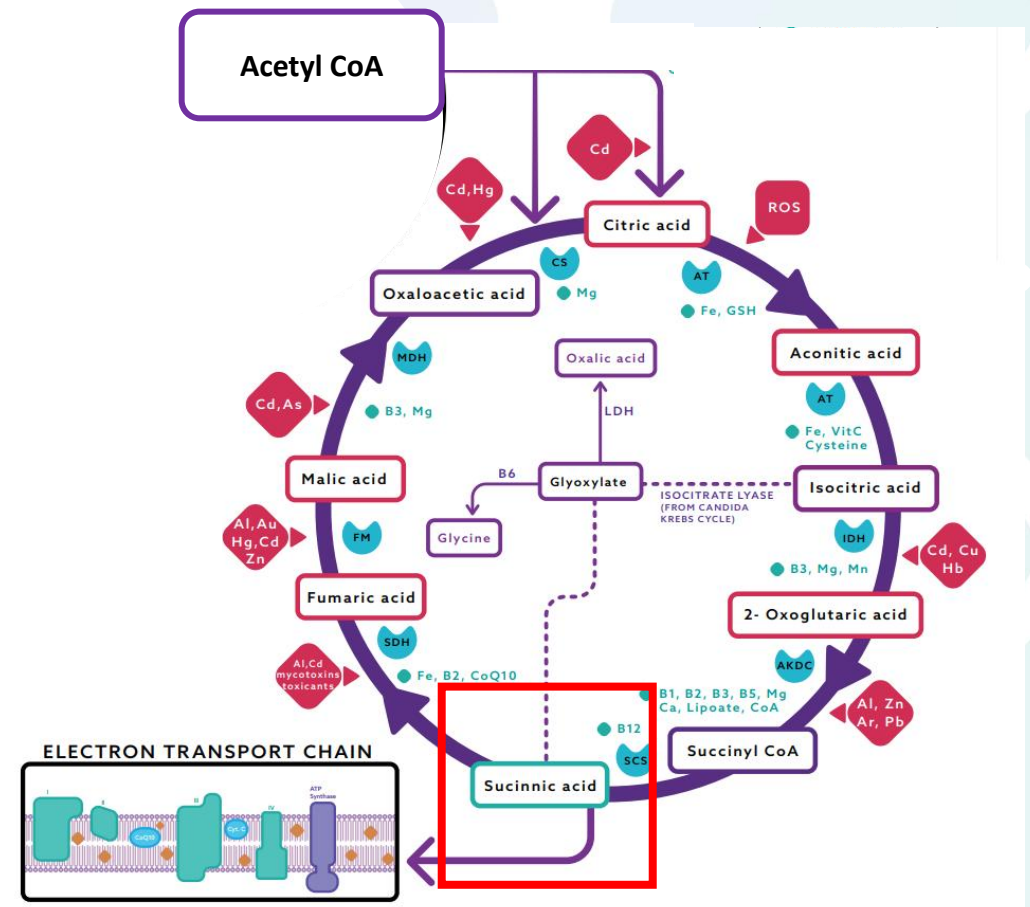
MOSAIC DIAGNOSTICS

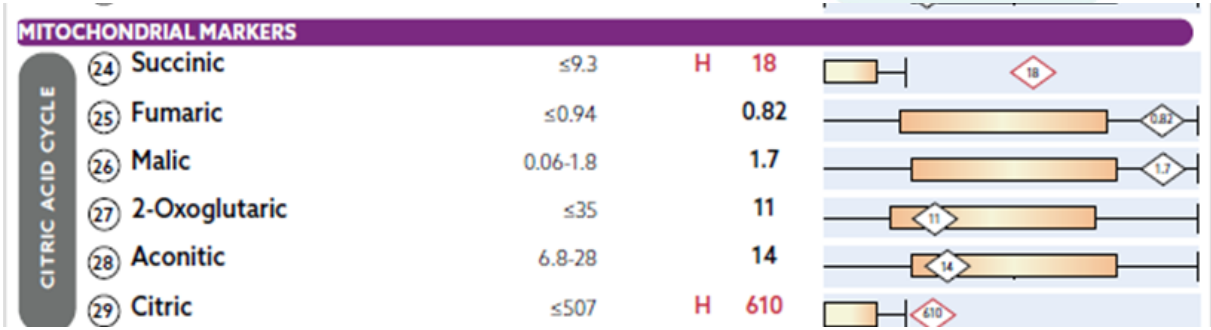
Mitochondrial Impacts

Succinic acid

- Succinate Dehydrogenase enzyme is incredibly sensitive to toxicants.
  - There are certain fungicides designed to directly inhibit it.

- Elevations can allude to environmental **toxicant exposure**. PMIDs: 24250680, 36835457

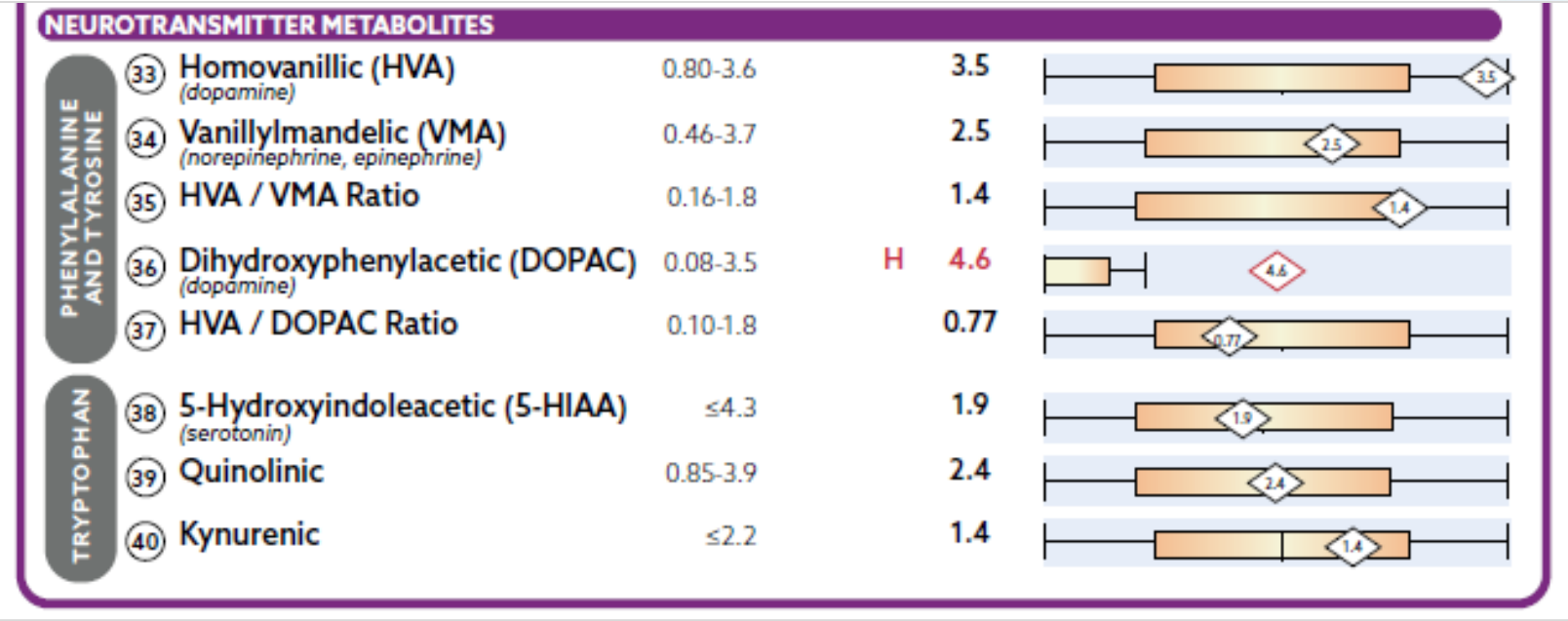




MOSAIC DIAGNOSTICS

https://doi.org/10.1016/j.jscs.2010.06.006,
https://doi.org/10.1016/j.envint.2023.108219.

Neurotransmitter Metabolites



NEUROTRANSMITTER METABOLITES					
PHENYLALANINE AND TYROSINE	33	Homovanillic (HVA) *(dopamine)*	0.80-3.6		3.5
	34	Vanillylmandelic (VMA) *(norepinephrine, epinephrine)*	0.46-3.7		2.5
	35	HVA / VMA Ratio	0.16-1.8		1.4
	36	Dihydroxyphenylacetic (DOPAC) *(dopamine)*	0.08-3.5	H	4.6
	37	HVA / DOPAC Ratio	0.10-1.8		0.77
TRYPTOPHAN	38	5-Hydroxyindoleacetic (5-HIAA) *(serotonin)*	≤4.3		1.9
	39	Quinolinic	0.85-3.9		2.4
	40	Kynurenic	≤2.2		1.4

Neurotransmitter Impacts

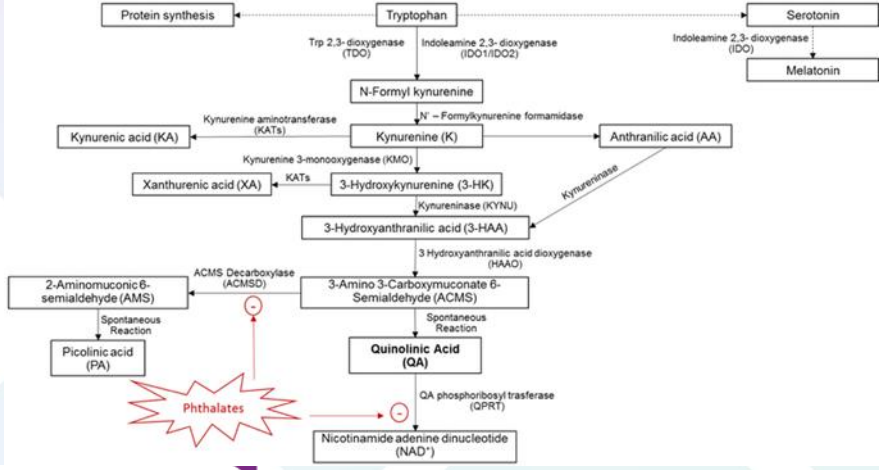
- Numerous toxicants can have direct and indirect impacts NT levels

- **Pesticides**
  - Oxidative stress & Dopaminergic neurodegeneration.

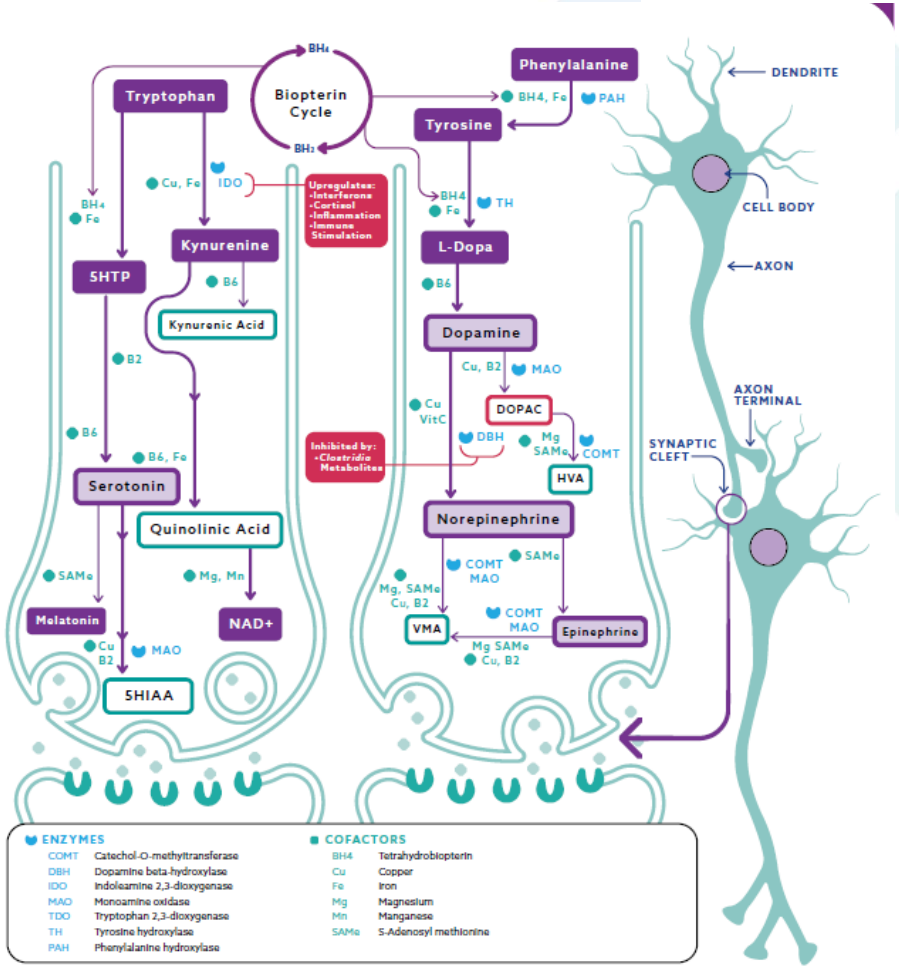
  PMID: 16797138

- **Phthalates**
  - Inhibits alpha-amino-beta-carboxymuconate-epsilon-semialdehyde decarboxylase (ACMSD) and quinolate phosphoribosyltransferase (QPRT) enzyme, causing **Quinolinic Acid** to elevate.
    - Quinolinic acid can contribute to neuroinflammation and is implicated in many neurological disorders.

      PMID: 15229365, PMID: 30826665



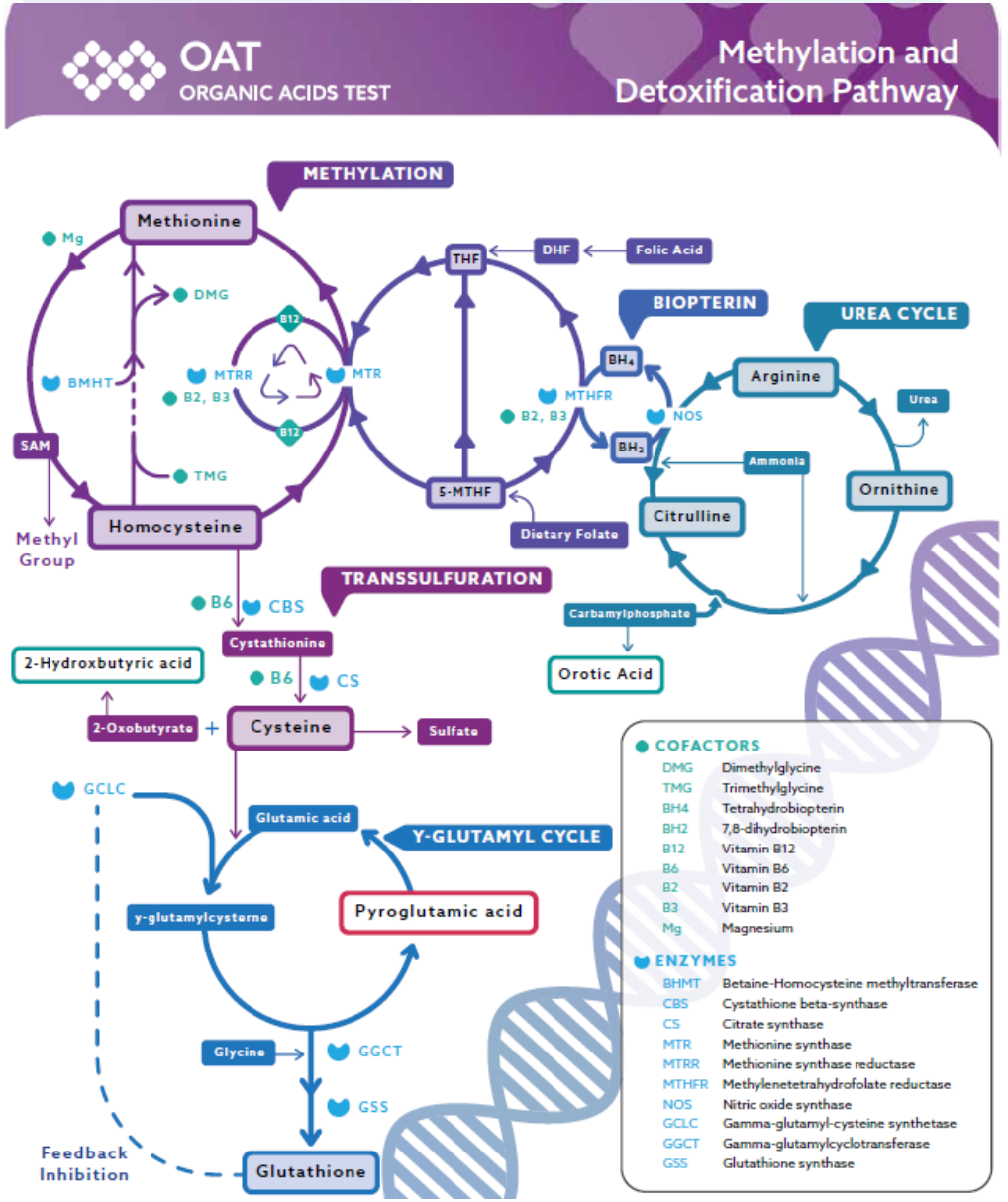
Source: **High phthalate exposure increased urinary concentrations of quinolinic acid, implicated in the pathogenesis of neurological disorders: Is this a potential missing link?**



Methylation/ Detoxification Impacts

INDICATORS OF DETOXIFICATION

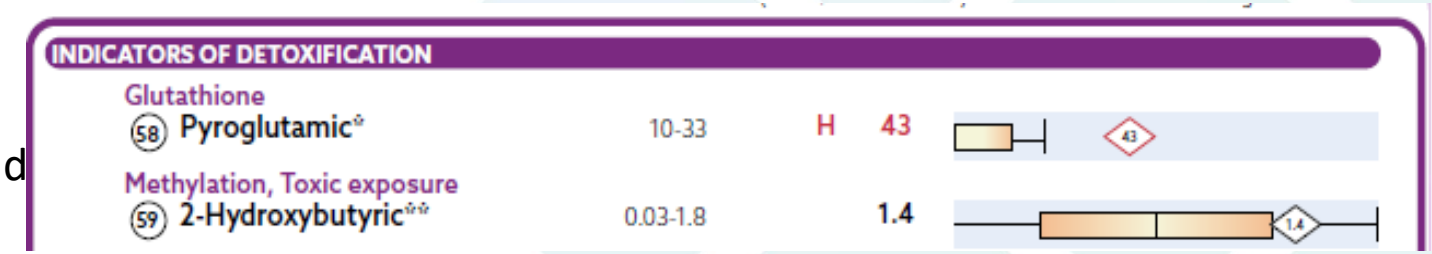
Glutathione				
(58) Pyroglutamic*	10-33	H	43	
Methylation, Toxic exposure				
(59) 2-Hydroxybutyric**	0.03-1.8		1.4	
Ammonia Excess				
(60) Orotic	0.06-0.54		0.48	



MOSAIC DIAGNOSTICS

Methylation/ Detoxification Impacts

- **Pyroglutamic Acid**- functional marker for glutathione (GSH) status.
  - GSH is important for the biotransformation of many toxins and toxicants.
  - Increased oxidative stress → increased demand
- **2 Hydroxybutyric Acid**- pathway Cysteine support for GSH vs Methylation impacts



Nutritional Needs

PYRIMIDINE METABOLITES - FOLATE METABOLISM			
(41) Uracil	≤9.7		4.5
(42) Thymine	≤0.56		0.19

NUTRITIONAL MARKERS			
Vitamin B12			
(50) Methylmalonic*	≤2.3	H	2.8
Vitamin B6			
(51) Pyridoxic	≤34		3.7
Vitamin B5			
(52) Pantothenic	≤10	H	23
Vitamin B2 (Riboflavin)			
(53) Glutaric*	0.04-0.36	H	0.89
Vitamin C			
(54) Ascorbic	10-200	L	0.56
Vitamin Q10 (CoQ10)			
(55) 3-Hydroxy-3-methylglutaric*	0.17-39		29
Glutathione Precursor and Chelating Agent			
(56) N-Acetylcysteine (NAC)	≤0.28		0.04
Biotin (Vitamin H)			
(57) Methylcitric*	0.19-2.7		1.1

*A high value for this marker may indicate a deficiency of this vitamin.

MINERAL METABOLISM			
(76) Phosphoric	1,000-5,000		2,493

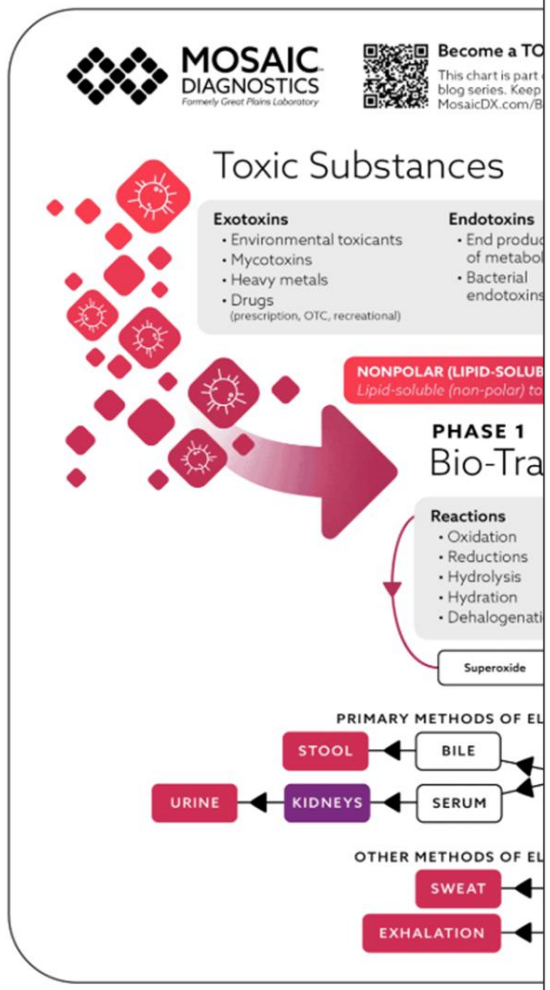
MOSAIC DIAGNOSTICS

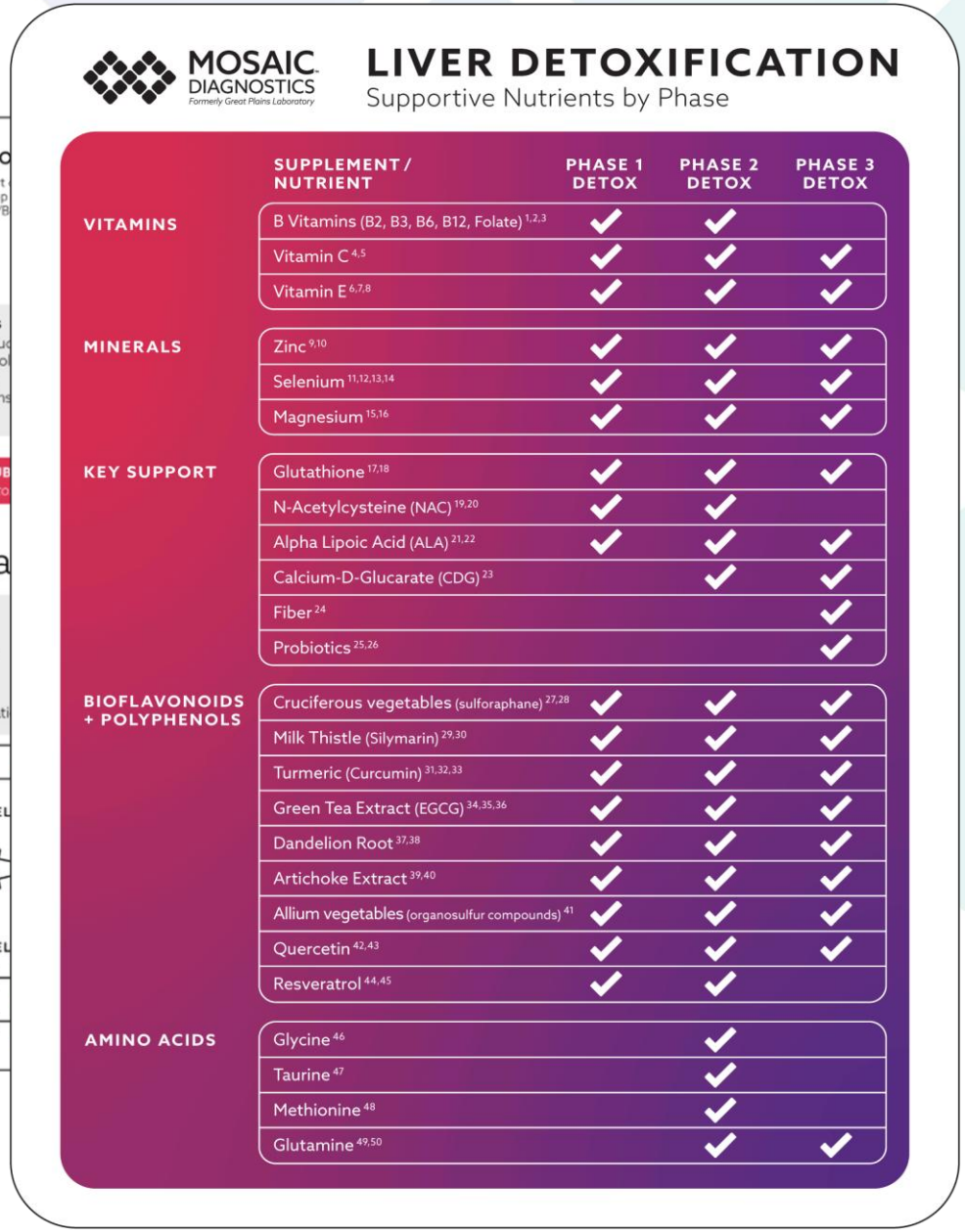
Nutritional Impacts

- **B Vitamins, Antioxidants, Minerals**

  Toxic exposures can influence the need for nutrients involved in various metabolic processes

  - Specific nutrient to the OAT:
    - B Vitamins
    - Vitamin C
    - GSH
    - CoQ10
  - Other nutrients from patterns:
    - Mg
    - Zinc
    - Iron
    - Glycine





MOSAIC DIAGNOSTICS

PMID: 1778274, PMID: 22357258, PMID: 33011353

Other Markers

Other metabolized byproducts of exposure.

- Mandelic acid (68) = **Styrene**

PMID: 18203133

- Malonic acid (74)= **Acrolein :** forms with the oxidation of PUFAs-linolenic acid (canola, soybean, corn, sunflower, flax, peanuts)

PMID: 30635735

AMINO ACID METABOLITES

(68) Mandelic	≤2.0	0.13	0.13
(74) Malonic	≤9.7	4.8	4.8

METABOLITE	RESULTS	PERCENTILE
Parent	ug/g creatinine	75% | 95%
VOC - VOLATILE ORGANIC COMPOUNDS		
(9) Phenylglyoxylic Acid (PGO)	352.88	75th 306.00 / 95th 509.00
Styrene/Ethylbenzene		

Parent: Styrene is widely used to make plastics and rubber, which are used to manufacture a variety of products, such as insulation, pipes, automobile parts, printing cartridges, food containers, and carpet backing. Exposure may occur through ingestion via transfer to foods, especially fatty foods heated in styrene containers, through breathing indoor air that has styrene vapors from building materials, photocopiers, tobacco smoke, and other products. Styrene and styrene oxide have been implicated as reproductive toxicants, neurotoxicants, and linked to an increased risk of leukemia and lymphoma.

MOSAIC
DIAGNOSTICS

FUNDAMENTAL DETOX STRATEGIES *See Disclaimer on Page 154

1. Reduce ongoing exposure by identifying and eliminating sources (e.g., moldy environments, contaminated water, occupational chemicals) and prioritizing organic foods to reduce pesticide intake, using HEPA air filtration and remove dust to limit airborne pollutants and mold spores, and avoiding plastics, synthetic fragrances, and other common environmental toxic exposures in the home and workplace.[1109-1116]
2. Support biotransformation pathways, especially glutathione conjugation, glucuronidation, sulfation, and methylation, using nutrients such as NAC, glycine, selenium, B6, B12, folate, and magnesium. More detailed information can be found in the following blog: The Liver: Supportive Nutrients in Detoxification - MosaicDX.
3. Enhance glutathione status via liposomal glutathione, N-acetyl cysteine (NAC), or other precursors like glycine
4. Target mitochondrial repair with CoQ10, acetyl-L-carnitine, alpha-lipoic acid, and B complex.
5. Bind and eliminate toxins and toxicants using agents like activated charcoal, zeolite clay, chlorella, humic and fulvic acids, fibers, or prescription binders (e.g., cholestyramine), depending on toxic exposure type.
6. Support Phase II detoxification with sulforaphane, calcium-D-glucarate, taurine, and cruciferous vegetables. Additional information can be found in the following blog: The Liver: Its Important Role in Detoxification - MosaicDX
7. Address secondary dysbiosis or fungal overgrowth, which may be exacerbated by toxin-induced immune suppression or antimicrobial activity, using targeted botanicals and gut repair strategies.
8. Replenish key vitamins (e.g. B vitamins, C, etc.) and minerals (e.g., zinc, magnesium, molybdenum) displaced by toxic exposure to restore enzyme function.
9. Consider adjunct therapies such as sauna, lymphatic support, and detox protocols based on individual tolerance and clinical presentation.

CLINICAL TIPS *See Disclaimer on Page 154

- **Elevated pyroglutamic acid** often signals glutathione depletion—consider supporting glutathione synthesis via NAC, glutathione, or precursors.
- Elevations in **succinic acid** and related Citric Acid Cycle markers warrant assessment for toxic exposures that influence mitochondria (heavy metals, toxicants, mycotoxins).
- **Fungal and Mycotoxin-Linked Markers to Screen for Mold Exposure**: Elevated levels of **5-Hydroxymethyl-2-furoic acid**, **Furan-2,5-dicarboxylic acid**, **Furancarbonylglycine**, **Citramalic**, **Tartaric**, and **Tricarballylic acid** can indicate mold exposure (especially *Aspergillus* or *Fusarium* species). Evaluate these in the context of clinical history and potential environmental exposure.
- **Identify Environmental and Heavy Metal Toxicity Through Patterned Marker Changes**: Look for elevated **HVA**, **VMA**, **pyruvic**, **citric**, **quinolinic**, **mandelic**, **hippuric**, **oxalic**, and **malonic acids** as possible indicators of toxic exposures from **heavy metals** (**arsenic**, **mercury**, **cadmium**), **pesticides**, **phthalates**, **solvents**, **parabens**, and **combustion byproducts**. Assess

Elevations and Association with **Specific** Toxic Substances

ORGANIC ACIDS	ASSOCIATION WITH TOXIC SUBSTANCE	RELATED TESTS
7. Arabinose	Indicator of *Candida* overgrowth.[36,37] Mycotoxins and toxicants disrupt the microbiome and intestinal epithelial barrier, and influence the immune system, influencing candida growth.[38-41]	MycoTOX PROFILE and/or TOXDetect PROFILE *Depending on clinical assessment; Comprehensive Stool ANALYSIS WITH PARASITOLOGY; IgG Food MAP WITH CANDIDA + YEAST
24. Succinic Acid	Succinate dehydrogenase enzyme, which converts succinate to fumarate, can be directly inhibited by chemical toxicants, mycotoxins, and heavy metals.[42-46]	MycoTOX PROFILE; TOXDetect PROFILE; Metals TOXIC + NUTRIENT *Depending on clinical assessment
33. Homovanillic Acid (HVA)	Both metabolites of Dopamine.[15,22] Various pesticides can affect dopamine metabolism causing elevated HVA and DOPAC.[22]	TOXDetect PROFILE; Metals TOXIC + NUTRIENT
36. DOPAC (3,4-dihydroxy-phenylacetic acid)	Heavy metals have been associated with elevated HVA.[15] Certain mycotoxins from *Aspergillus* and *Fusarium* can influence dopamine metabolism.[6,7]	MycoTOX PROFILE
34. Vanillylmandelic Acid (VMA)	Elevations may result from heavy metal exposure.[16]	Metals TOXIC + NUTRIENT
58. Pyroglutamic Acid	Reflects glutathione (GSH) status. Elevations can indicate insufficient levels or high demand.[48] Exposures to toxicants, heavy metals, and mycotoxins all influence glutathione levels.[49-51]	MycoTOX PROFILE; TOXDetect PROFILE; Metals TOXIC + NUTRIENT *Depending on clinical assessment
59. 2-Hydroxybutyric Acid	Biproduct of homocysteine to cystathionine pathway. Elevations can allude to high demand for cysteine for GSH or methylation defects related to various toxic exposures.[52,53]	MycoTOX PROFILE; TOXDetect PROFILE; Metals TOXIC + NUTRIENT *Depending on clinical assessment

Are you ready to clean up your world?

Start here!

Detoxify Your World

With a few simple steps, we can minimize toxic exposure and improve health.

Explore how you can become aware of the toxicants around us and detoxify your world.

Common Toxicant Sources ↓

Organic Foods ↓

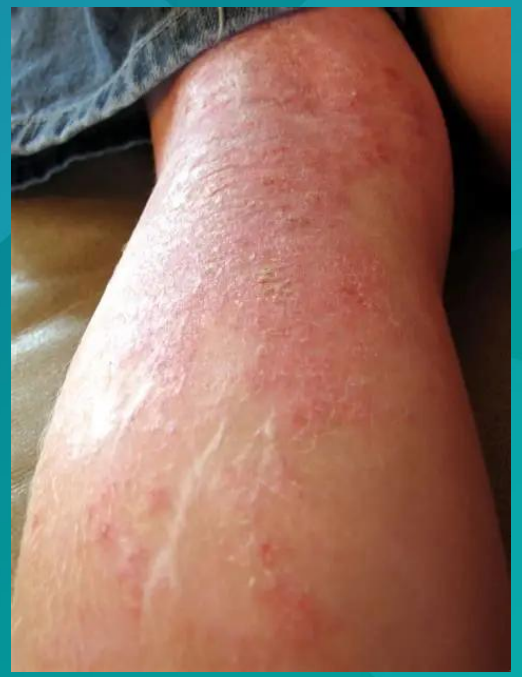
Education ↓

Household Products ↓

Air & Water ↓

Case Study:

Pediatric Eczema



MOSAIC DIAGNOSTICS

Case overview:

10 year old Male

Chief Concerns:

- Hx of Eczema that has been controlled, but now has returned and is more severe.
- Extremely picky eating
- Was potty trained at 5, now with bed wetting
- Difficulty sleeping, even when itching is under control
- Headaches, mainly behind the eyes
- Increasing fatigue and muscle weakness

- **PMH:**
  - Vaginal delivery, eczema
- **Medications/Supplements:**
  - MV, digestive enzymes
- **Diet:**
  - Prefers GF spaghetti w/ tomato sauce, some fruit, and GF corn chips, DF/GF pizza.
- **Social Hx:**
  - Attends a private school in the downtown area
  - Recently moved to an apartment close to school
  - Lives with both parents, one younger sibling, no pets.
- **Other Tests**
  - Environmental IgE : allergies to Aspergillus, various Tree Pollen, and cats
  - Food IgG: sensitivities to casein and gluten
  - Stool Test: Elevated IgA, lower pancreatic elastase

MOSAIC DIAGNOSTICS

Color Key LOW MODERATE HIGH

Creatinine Value: * 85.24 mg/dl

METABOLITE Parent	RESULTS ug/g creatinine	PERCENTILE 75% | 95%

HIGH RESULTS

14	**2-Hydroxyethyl Mercapturic Acid (HEMA)** Ethylene Oxide, Vinyl Chloride	11.20	75th 1.86 | 95th 4.83
18	**Butylparaben (BuP)** Butylparaben (BuP)	24.38	75th 0.60 | 95th 4.57

MODERATE RESULTS

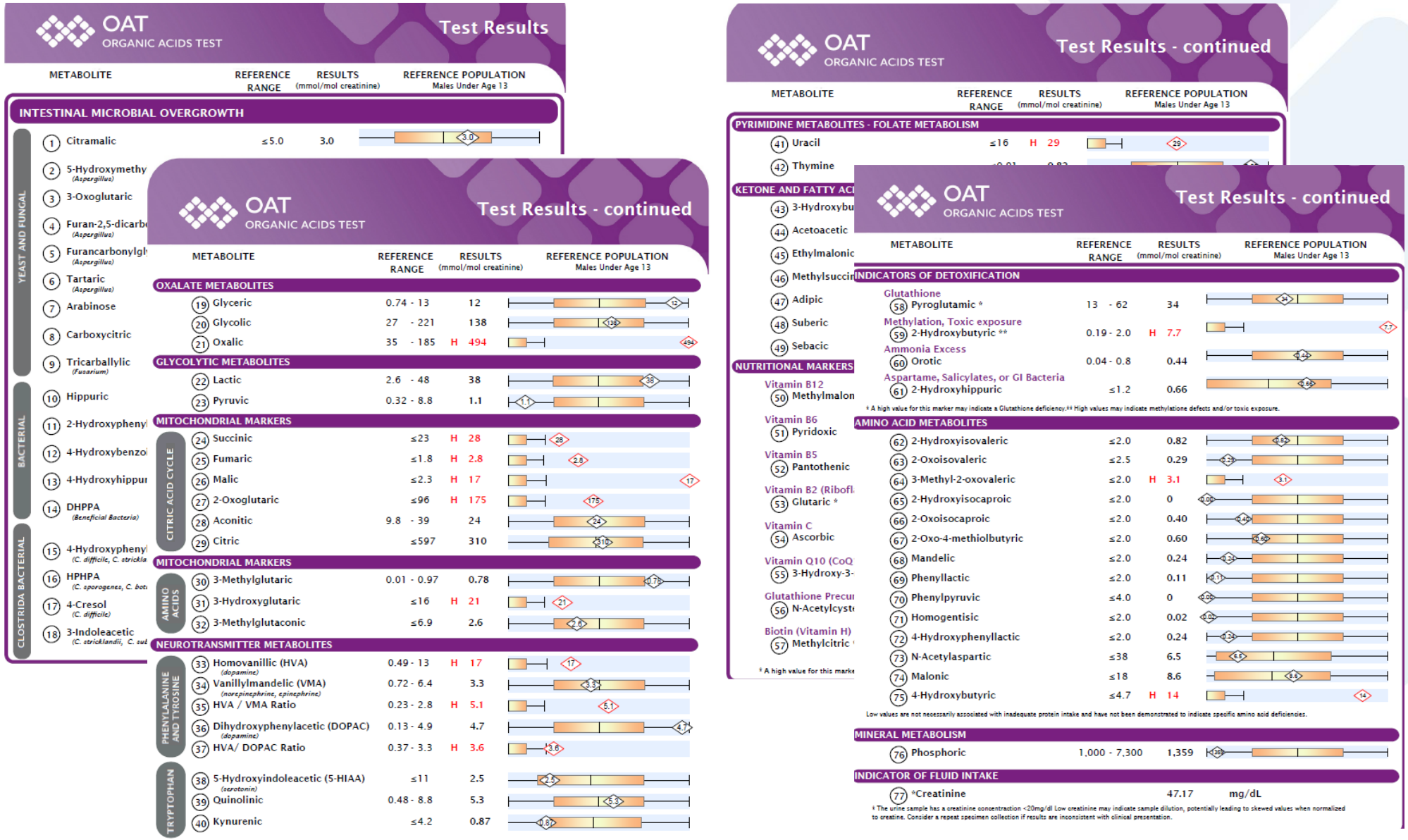
1	**Monoethylphthalate (MEP)** Diethylphthalates	57.26	75th 51.50 | 95th 275.00
3	**Mono-2ethylhexyl phthalate (MEHP)** Di(2-ethylhexyl) Phthalate (DEHP)	4.11	75th 1.92 | 95th 5.16
4	**Mono-(2-ethyl-5-oxohexyl) phthalate (MEOHP)** Di(2-ethylhexyl) Phthalate (DEHP)	6.20	75th 5.19 | 95th 14.40
9	**Phenylglyoxylic Acid (PGO)** Styrene/Ethylbenzene	352.88	75th 306.00 | 95th 509.00
12	**N-Acetyl (Propyl) Cysteine (NAPR)** 1-bromopropane	46.10	75th 11.10 | 95th 47.80
20	**2,4-Dichlorophenoxyacetic Acid (2,4-D)** 2,4-Dichlorophenoxyacetic Acid (2,4-D)	0.67	75th 0.58 | 95th 1.60
22	**Diethylphosphate (DEP)** Organophosphates	7.63	75th 4.61 | 95th 15.00
24	**N-Acetyl (Carbomethyl) Cysteine (NAE)** Acrylamide	131.17	75th 93.40 | 95th 212.00
25	**Perchlorate (PERC)** Perchlorate	4.41	75th 4.01 | 95th 9.39

Creatinine Value* : 57.77 mg/dl

METABOLITE Parent	RESULTS ug/g creatinine | DL - Detectable Limit | ULOQ - Upper Limit of Quantification	PERCENTILE 75% | 95%

Glyphosate - Urine

Glyphosate	1.28	75th 0.48 | 95th 1.28



OAT
ORGANIC ACIDS TEST

Test Results

METABOLITE	REFERENCE RANGE	RESULTS (mmol/mol creatinine)	REFERENCE POPULATION Males Under Age 13
INTESTINAL MICROBIAL OVERGROWTH			
YEAST AND FUNGAL			
1 Citramalic	≤5.0	3.0	
2 5-Hydroxymethy (Aspergillus)			
3 3-Oxoglutaric			
4 Furan-2,5-dicarb (Aspergillus)			
5 Furancarbonylgl (Aspergillus)			
6 Tartaric (Aspergillus)			
7 Arabinose			
8 Carboxycitric			
9 Tricarballylic (Fusarium)			
BACTERIAL			
10 Hippuric			
11 2-Hydroxypheny			
12 4-Hydroxybenzoi			
13 4-Hydroxyhippur			
14 DHPPA (Beneficial Bacteria)			
CLOSTRIDIA BACTERIAL			
15 4-Hydroxyphenyl (C. difficile, C. strickla			
16 HPHPA (C. sporogenes, C. bot			
17 4-Cresol (C. difficile)			
18 3-Indoleacetic (C. stricklandii, C. sub			

OAT
ORGANIC ACIDS TEST

Test Results - continued

METABOLITE	REFERENCE RANGE	RESULTS (mmol/mol creatinine)	REFERENCE POPULATION Males Under Age 13
OXALATE METABOLITES			
19 Glyceric	0.74 - 13	12	
20 Glycolic	27 - 221	138	
21 Oxalic	35 - 185	H 494	
GLYCOLYTIC METABOLITES			
22 Lactic	2.6 - 48	38	
23 Pyruvic	0.32 - 8.8	1.1	
MITOCHONDRIAL MARKERS			
CITRIC ACID CYCLE			
24 Succinic	≤23	H 28	
25 Fumaric	≤1.8	H 2.8	
26 Malic	≤2.3	H 17	
27 2-Oxoglutaric	≤96	H 175	
28 Aconitic	9.8 - 39	24	
29 Citric	≤597	310	
MITOCHONDRIAL MARKERS			
AMINO ACIDS			
30 3-Methylglutaric	0.01 - 0.97	0.78	
31 3-Hydroxyglutaric	≤16	H 21	
32 3-Methylglutaconic	≤6.9	2.6	
NEUROTRANSMITTER METABOLITES			
PHENYLALANINE AND TYROSINE			
33 Homovanillic (HVA) (dopamine)	0.49 - 13	H 17	
34 Vanillylmandelic (VMA) (norepinephrine, epinephrine)	0.72 - 6.4	3.3	
35 HVA / VMA Ratio	0.23 - 2.8	H 5.1	
36 Dihydroxyphenylacetic (DOPAC) (dopamine)	0.13 - 4.9	4.7	
37 HVA/ DOPAC Ratio	0.37 - 3.3	H 3.6	
TRYPTOPHAN			
38 5-Hydroxyindoleacetic (5-HIAA) (serotonin)	≤11	2.5	
39 Quinolinic	0.48 - 8.8	5.3	
40 Kynurenic	≤4.2	0.87	

OAT
ORGANIC ACIDS TEST

Test Results - continued

METABOLITE	REFERENCE RANGE	RESULTS (mmol/mol creatinine)	REFERENCE POPULATION Males Under Age 13
PYRIMIDINE METABOLITES - FOLATE METABOLISM			
41 Uracil	≤16	H 29	
42 Thymine			
KETONE AND FATTY ACI			
43 3-Hydroxybu			
44 Acetoacetic			
45 Ethylmalonic			
46 Methylsuccin			
47 Adipic			
48 Suberic			
49 Sebacic			
NUTRITIONAL MARKERS			
Vitamin B12			
50 Methylmalon			
Vitamin B6			
51 Pyridoxic			
Vitamin B5			
52 Pantothenic			
Vitamin B2 (Ribofl			
53 Glutaric *			
Vitamin C			
54 Ascorbic			
Vitamin Q10 (CoQ			
55 3-Hydroxy-3-			
Glutathione Precu			
56 N-Acetylcyst			
Biotin (Vitamin H)			
57 Methylcitric			

* A high value for this marke

OAT
ORGANIC ACIDS TEST

Test Results - continued

METABOLITE	REFERENCE RANGE	RESULTS (mmol/mol creatinine)	REFERENCE POPULATION Males Under Age 13
INDICATORS OF DETOXIFICATION			
Glutathione			
58 Pyroglutamic *	13 - 62	34	
Methylation, Toxic exposure			
59 2-Hydroxybutyric **	0.19 - 2.0	H 7.7	
Ammonia Excess			
60 Orotic	0.04 - 0.8	0.44	
Aspartame, Salicylates, or GI Bacteria			
61 2-Hydroxyhippuric	≤1.2	0.66	
AMINO ACID METABOLITES			
62 2-Hydroxyisovaleric	≤2.0	0.82	
63 2-Oxoisovaleric	≤2.5	0.29	
64 3-Methyl-2-oxovaleric	≤2.0	H 3.1	
65 2-Hydroxyisocaproic	≤2.0	0	
66 2-Oxoisocaproic	≤2.0	0.40	
67 2-Oxo-4-methiolbutyric	≤2.0	0.60	
68 Mandelic	≤2.0	0.24	
69 Phenyllactic	≤2.0	0.11	
70 Phenylpyruvic	≤4.0	0	
71 Homogentisic	≤2.0	0.02	
72 4-Hydroxyphenyllactic	≤2.0	0.24	
73 N-Acetylaspartic	≤38	6.5	
74 Malonic	≤18	8.6	
75 4-Hydroxybutyric	≤4.7	H 14	
MINERAL METABOLISM			
76 Phosphoric	1,000 - 7,300	1,359	
INDICATOR OF FLUID INTAKE			
77 *Creatinine		47.17 mg/dL	

* A high value for this marker may indicate a Glutathione deficiency.** High values may indicate methylation defects and/or toxic exposure.

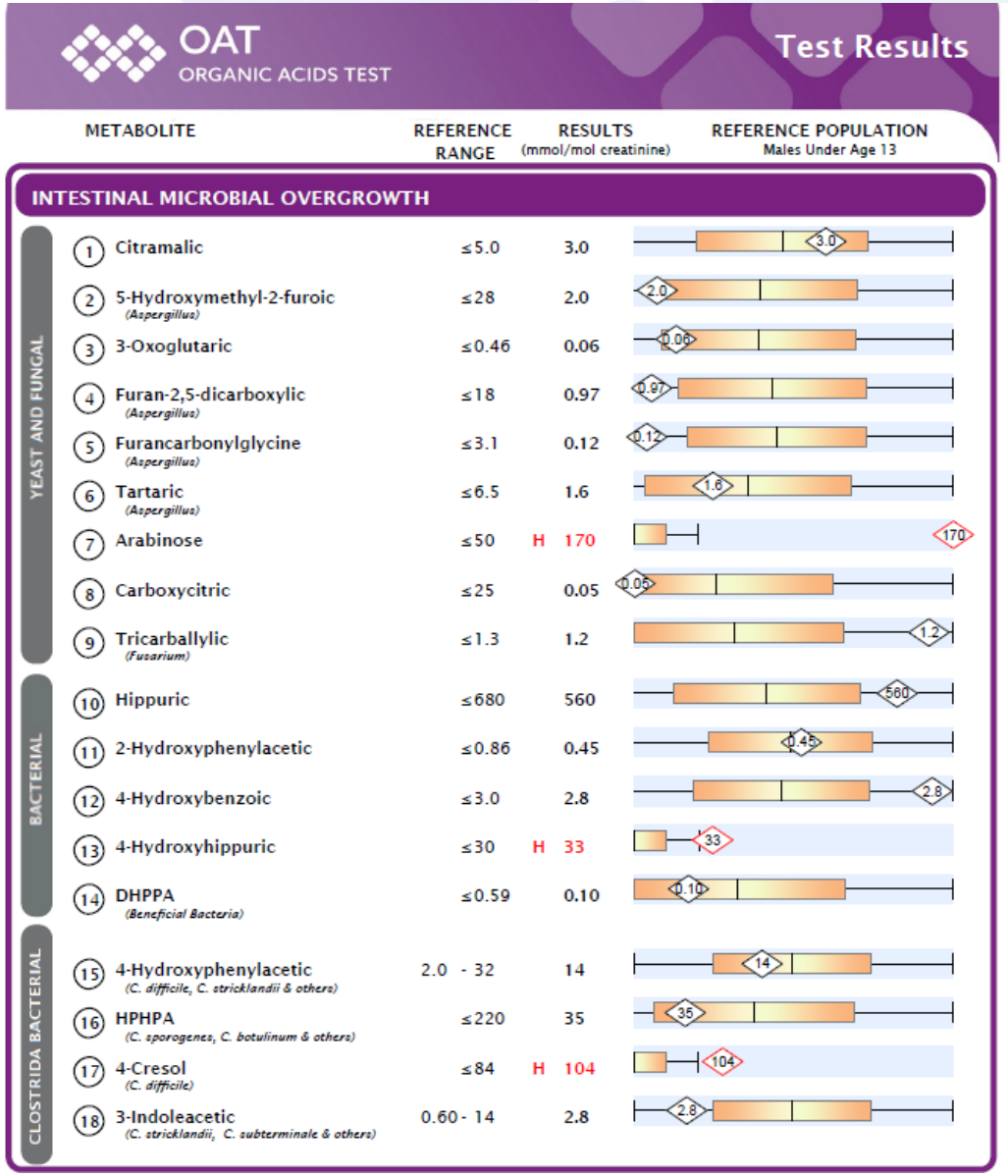
Low values are not necessarily associated with inadequate protein intake and have not been demonstrated to indicate specific amino acid deficiencies.

* The urine sample has a creatinine concentration <20mg/dl Low creatinine may indicate sample dilution, potentially leading to skewed values when normalized to creatine. Consider a repeat specimen collection if results are inconsistent with clinical presentation.

MOSAIC DIAGNOSTICS

Results

Microbial Imbalance	Mechanism	Possible Symptom (s)	Category
Yeast	Environmental? Diet?	Likely influencing eczema, picky eating, and headaches	
General Bacteria	Influenced by Fungus and Clostridia? Parabens?		
Clostridia	Pesticide exposure?	Influence on eczema, bed wetting, sleep, fatigue, pick eating?	



MOSAIC DIAGNOSTICS

Results

Oxalates	Mechanism	Possible Symptom(s)	Category
Oxalic acid	From fungus – diet is not high in plant-based foods.	Influencing headaches? Urinary incontinence?	

Multiple mitochondrial impacts	Mechanism	Possible Symptom(s)	Category
Citric acid cycle , amino acid utilization for energy	Toxic exposure Additional impacts; fungus, nutrient insufficiencies (B2), oxalates, elevated dopamine (next slide).	Fatigue and muscle weakness?	

OAT ORGANIC ACIDS TEST

Test Results - continued

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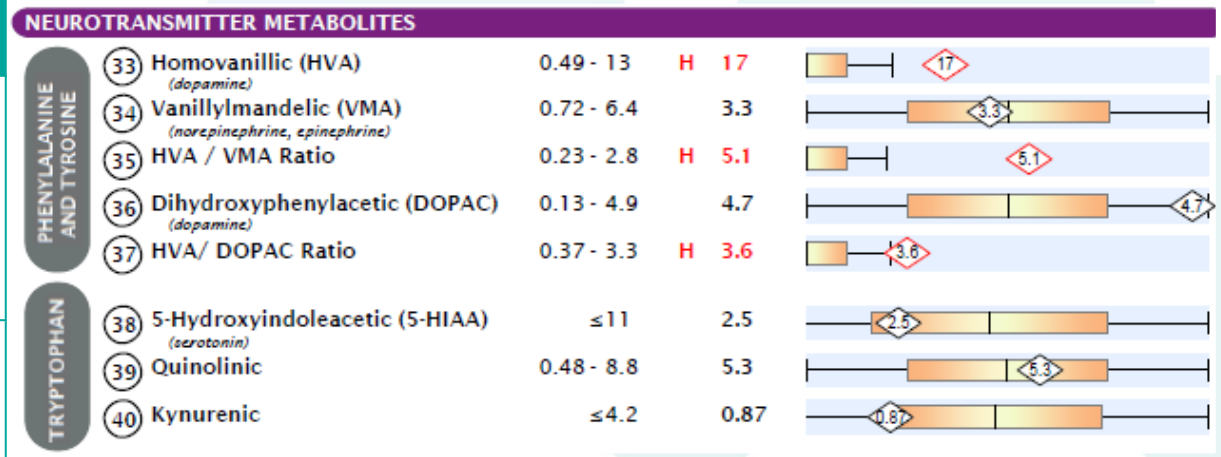
MOSAIC DIAGNOSTICS

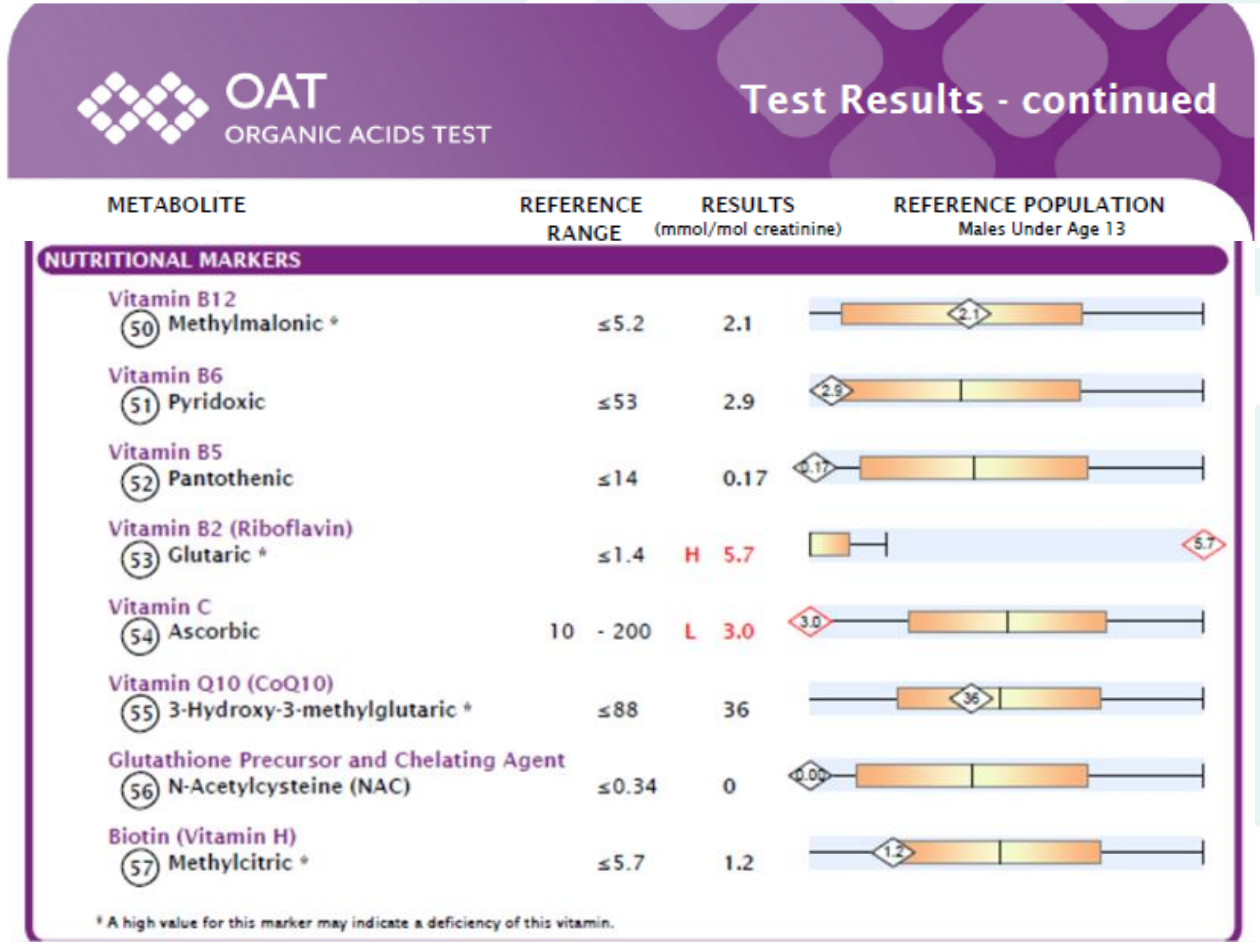
Results

Neurotransmitter Metabolites	Mechanism	Possible Symptom(s)	Category
HVA, HVA/VMA ratio	Clostridia influencing Dopamine	sleep (dopamine) and bed wetting (norepinephrine influences urethral sphincter and tone)	
HVA/DOPAC ratio	Upregulation of COMT	May be why it's not influencing his behavior	



Results

Nutrient Needs	Mechanism	Category
Low B6	Influences NTs, Methylation, Detoxification	
Low B5	Influences Mitochondria, NTs	
Low B2	Influences Mitochondria (Succinic acid), NTs, Methylation, Detoxification	



OAT
ORGANIC ACIDS TEST

Test Results - continued

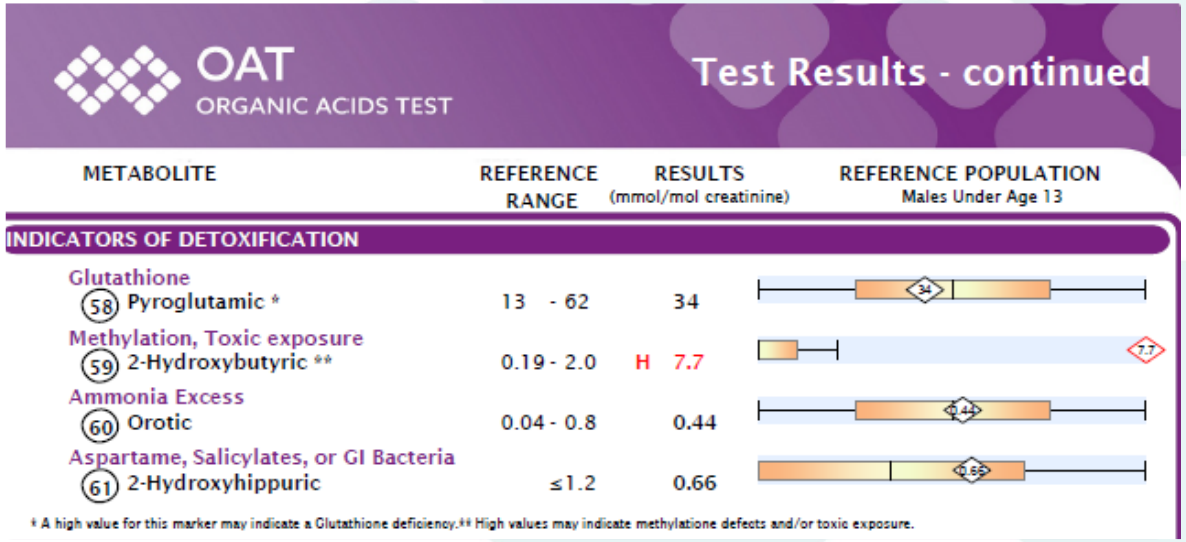
METABOLITE	REFERENCE RANGE		RESULTS (mmol/mol creatinine)	REFERENCE POPULATION Males Under Age 13
NUTRITIONAL MARKERS				
Vitamin B12 (50) Methylmalonic *	≤5.2		2.1	2.1
Vitamin B6 (51) Pyridoxic	≤53		2.9	2.9
Vitamin B5 (52) Pantothenic	≤14		0.17	0.17
Vitamin B2 (Riboflavin) (53) Glutaric *	≤1.4	H	5.7	5.7
Vitamin C (54) Ascorbic	10 - 200	L	3.0	3.0
Vitamin Q10 (CoQ10) (55) 3-Hydroxy-3-methylglutaric *	≤88		36	36
Glutathione Precursor and Chelating Agent (56) N-Acetylcysteine (NAC)	≤0.34		0	0.00
Biotin (Vitamin H) (57) Methylcitric *	≤5.7		1.2	1.2

* A high value for this marker may indicate a deficiency of this vitamin.

MOSAIC DIAGNOSTICS

Results

Methylation/ Detoxification	Mechanism	Category
2-Hydroxybutyric acid	Supports cysteine synthesis for GSH vs Methylation dysfunction	



OAT
ORGANIC ACIDS TEST

Test Results - continued

METABOLITE	REFERENCE RANGE	RESULTS (mmol/mol creatinine)	REFERENCE POPULATION Males Under Age 13
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Glutathione			
(58) Pyroglutamic *	13 - 62	34	
Methylation, Toxic exposure			
(59) 2-Hydroxybutyric **	0.19 - 2.0	H 7.7	
Ammonia Excess			
(60) Orotic	0.04 - 0.8	0.44	
Aspartame, Salicylates, or GI Bacteria			
(61) 2-Hydroxyhippuric	≤1.2	0.66	

* A high value for this marker may indicate a Glutathione deficiency.** High values may indicate methylation defects and/or toxic exposure.

Interventions

Botanical regmin 6-8 weeks, then retest.

- Combination oregano oil, caprylic acid and garlic
- High dose Spore-based Probiotics (Bacillus)
- Ca/Mg citrate with any meals high in Oxalates, Epsom salt baths in the evening, cream on forehead.

Mitochondrial Health

- MV w/ high dose B vitamins (B1,B2, B3, B6, B5, B12)

Neurotransmitter Support

- Address clostridia

Detoxification/Methylation Support

- Vitamin C
- Curcumin to support Glucaronidation
- NAC
- Humic + Fulvic Acids
- Soluble Fiber

Additional Nutrient Needs

- Zinc for picky eating
- Omega 3s

Additional Testing

Retest OAT in 8-12 weeks post-therapy, depending on symptoms.

Toxicant in 3 months after interventions.

MOSAIC DIAGNOSTICS

Thank you for your time!

Questions?

Contact Us

If you have any questions, please reach out to the appropriate team and/ or visit our website to learn more.

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MOSAIC DIAGNOSTICS