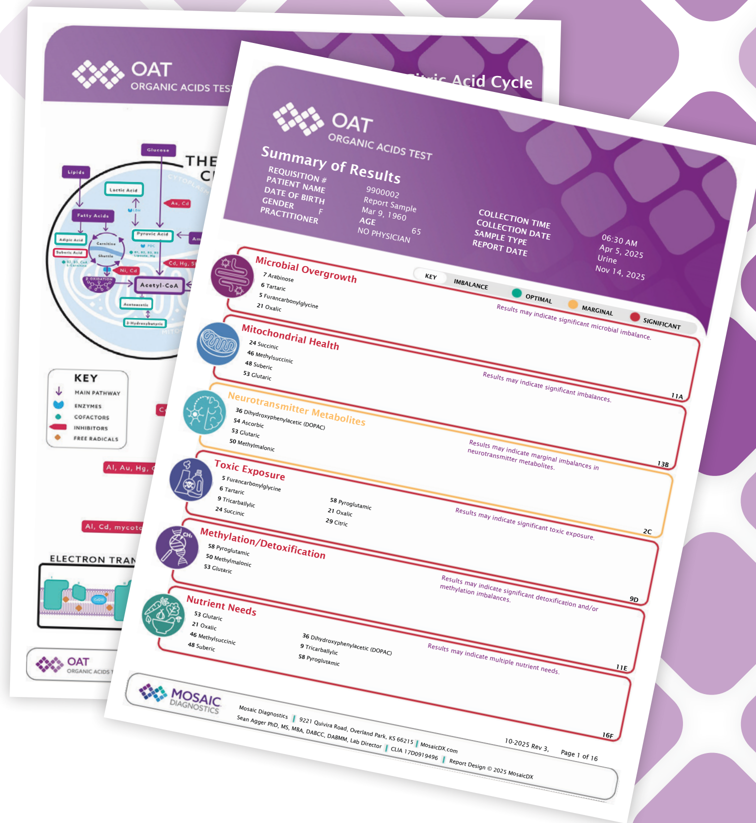
OAT
ORGANIC ACIDS TEST



The Clinical Significance of the Organic Acids Test

The Organic Acids Test (OAT) provides a comprehensive functional assessment of essential metabolic pathways that influence overall health and disease risk.

The OAT measures 76 organic acids utilizing a simple urine sample to uncover biochemical imbalances that may impact gut microbiome health, energy production, nutrient status, and detoxification. These insights help guide personalized interventions, such as microbiome re-balancing, mitochondrial support, targeted nutrient supplementation, dietary modifications, and detoxification strategies.

This makes the OAT a powerful tool for individualized clinical decision-making.

Summary of Results Page: Organizes patient findings into the **Six Key Clinical Categories.**

Microbial Overgrowth	Markers of yeast, mold, bacteria, *Clostridium* species, and their metabolic byproducts.
Mitochondrial Health	Intermediates of glucose, fatty acid, and amino acid metabolism involved in energy production.
Neurotransmitter Metabolites	Dopamine, serotonin, & norepinephrine/epinephrine pathway metabolites, providing insights into neurotransmitter imbalances.
Toxic Exposure	Environmental toxins, mycotoxins, metals, and more.
Methylation / Detoxification	Metabolites reflecting methyl group cycling, glutathione status, and nutrients involved in both methylation and detoxification.
Nutrient Needs	Direct and indirect biomarkers reflecting the functional demand for B-Vitamins, antioxidants, and other key micronutrients.

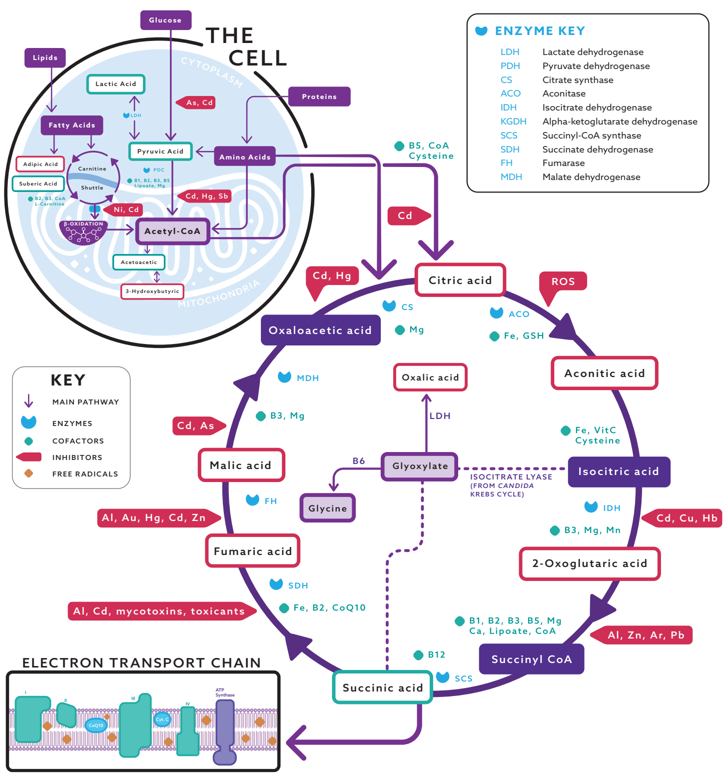
Each category is color-coded; green = unremarkable, yellow = marginal and red = significant, to reflect the level of imbalances allowing for quick prioritization of concerning areas.

Discover Our OAT and Review an OAT Sample Report

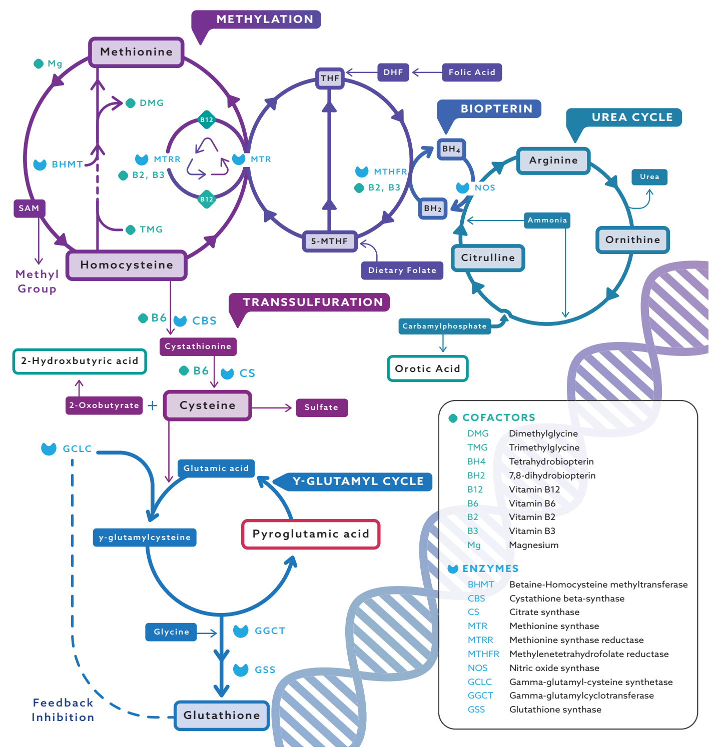
Metabolic Pathways:

Dynamically incorporate patient specific results

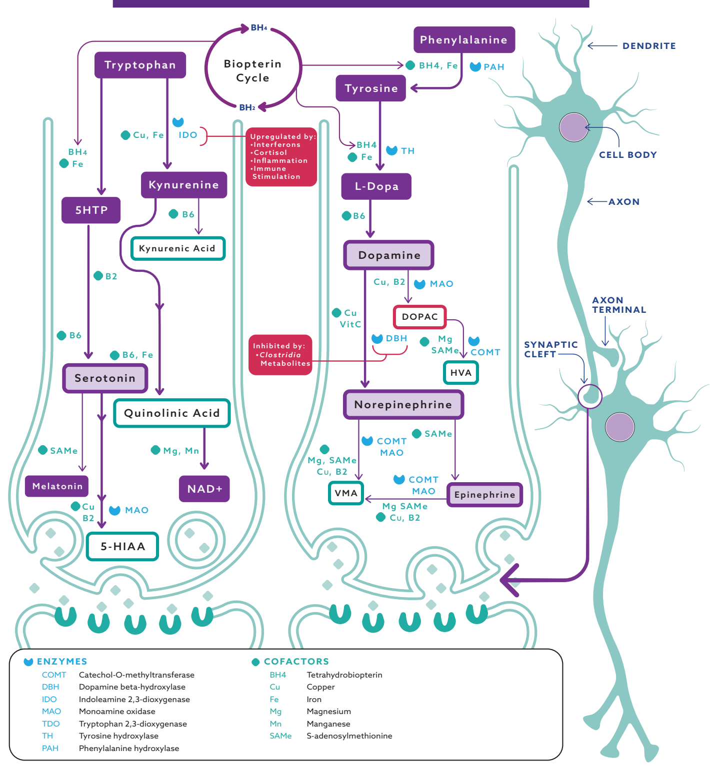
Citric Acid Cycle



Methylation and Detoxification Pathway



Neurotransmitter Pathway



The following pages list the 76 metabolite markers of the Organic Acids Test. Included is the name of the metabolic marker, the number on the report (), clinical significance, and usual initial considerations.

Intestinal Microbial Overgrowth	
Yeast and Fungal Markers	
Citramalic Acid (1)	Indicates possible fungal or mold-related dysbiosis (often Aspergillus niger) and may disrupt the Citric Acid Cycle, affecting energy production. Dietary sources like apples, tomatoes, and wine can also raise levels. Initial Considerations: Assess for fungal overgrowth, rebalance gut flora, support mitochondrial function with key nutrients, and inquire about mold and mycotoxin exposure;
5-Hydroxy-methyl-2-furoic Acid (2)	May indicate mold exposure, particularly from Aspergillus or Cladosporium species, and can also arise from dietary sources or heat-processed foods. Excess levels may subtly influence the microbiome. Initial Considerations: Assess for mold and mycotoxin exposure, review dietary contributors (coffee, dried fruit, baked goods), and support detoxification and gut balance, as needed.
3-Oxoglutaric Acid (3)	May reflect yeast or microbial overgrowth and can act as an analog of alpha-ketoglutarate, potentially disrupting the Citric Acid Cycle and mitochondrial energy production. Initial Considerations: Evaluate for yeast imbalance, support mitochondrial function (e.g., B-vitamins, alpha-lipoic acid), and rebalance gut flora, if indicated.
Furan-2,5-dicarboxylic Acid (4)	Elevations may indicate mold exposure (e.g., Aspergillus, Penicillium) or arise from dietary sources or plastics containing PEF. It can serve as a marker of environmental or microbial contribution to metabolic burden. Initial Considerations: Assess for mold or mycotoxin exposure, review dietary and environmental sources, and consider detoxification support if relevant; TOXDetect testing may be warranted with frequent plastic exposure.
Furancarbonylglycine (5)	May indicate mold exposure or overgrowth (particularly Aspergillus) and can also be influenced by high-heat processed foods like coffee. Levels often decrease with antifungal treatment, supporting its role as a fungal dysbiosis marker. Initial Considerations: Assess for mold or mycotoxin exposure, consider antifungal or gut-supportive protocols, and review dietary contributors to reduce exogenous furan intake.
Tartaric Acid (6)	Elevated tartaric acid may indicate fungal dysbiosis (Aspergillus, Penicillium, Candida, Saccharomyces) and can potentially inhibit the Citric Acid Cycle. Dietary sources, such as grapes, red wine, tamarind, and certain food additives, can also raise levels. Initial Considerations: Evaluate for fungal overgrowth, consider mycotoxin exposure, and review dietary sources; monitor related mitochondrial markers (malic and fumaric acid) if dysfunction is suspected.
Arabinose (7)	Elevated arabinose may indicate yeast overgrowth (e.g., Candida spp.) . Certain dietary sources that contain hemicellulose and pectin may also raise levels. Initial Considerations: Assess for yeast imbalance, consider antifungal or gut-supportive protocols, and review dietary sources of arabinose.
Carboxycitric Acid (8)	Elevated levels may indicate intestinal yeast or fungal overgrowth, though supporting evidence is still limited. Levels can decrease with antifungal therapy, suggesting a link to microbial dysbiosis. Initial Considerations: Assess for yeast imbalance and consider antifungal or gut-supportive interventions as indicated.
Tricarballylic Acid (9)	May indicate mycotoxin exposure from Fusarium or Aspergillus species, and can also be produced by certain gut bacteria. It can bind minerals like magnesium, calcium, and zinc, potentially affecting nutritional status. Initial Considerations: Assess for mycotoxin exposure (e.g., Fusarium, Aspergillus), support detoxification, consider gut microbiome balance, and monitor mineral status.
Bacterial Markers	
Hippuric Acid (10)	Elevations reflect gut microbial activity, dietary polyphenol intake, or environmental exposures such as toluene. Low levels may indicate dysbiosis or glycine/B5 insufficiency. Initial Considerations: Assess for gut microbial imbalances, review dietary and environmental contributors, support glycine and B-vitamin status if low. May also need to consider detoxification if exposure to solvents is suspected.
2-Hydroxyphenylacetic Acid (11)	May reflect gut bacterial overgrowth, altered phenylalanine metabolism, or dietary intake from foods like berries, tomatoes, and grapes. In rare cases, very high levels may be associated with genetic disorders such as PKU or tyrosinemia. Initial Considerations: Evaluate for microbial dysbiosis, support phenylalanine metabolism (B6 and biopterin pathways), and review dietary and supplement sources that may influence levels.

Bacterial Markers Cont.	
4-Hydroxybenzoic Acid (12) (4-HBA)	Reflects gut microbial activity and dietary polyphenol intake, and can be elevated with exposure to parabens. It may also support CoQ10 biosynthesis. Initial Considerations: Evaluate for gut dysbiosis, review dietary polyphenols and supplement intake, and reduce paraben exposure when relevant.
4-Hydroxyhippuric Acid (13)	Reflects gut microbial activity and polyphenol metabolism and can be elevated with paraben exposure. Low levels in the presence of high 4-HBA (12) may indicate glycine insufficiency. Initial Considerations: Evaluate for gut dysbiosis, review dietary polyphenols and supplement intake, reduce paraben exposure, and support glycine status if low.
DHPPA (dihydroxyphenylpropionic acid) (14)	Reflects gut microbial metabolism of polyphenols, primarily by Lactobacilli, Bifidobacteria, E. coli, and some Clostridium species. Elevated levels may indicate a polyphenol-rich diet or abundant beneficial bacteria, while low levels may suggest insufficient polyphenols or reduced commensal flora. Initial Considerations: Support beneficial gut bacteria, ensure adequate dietary polyphenols, and consider probiotic or prebiotic strategies if low.
Clostridia Bacterial Markers	
4-Hydroxyphenylacetic Acid (15)	Reflects Clostridia bacterial overgrowth and microbial metabolism of tyrosine and polyphenols. Elevated levels may influence dopamine/norepinephrine balance and can also be affected by polyphenol-rich diets or environmental exposures like glyphosate. Initial Considerations: Assess for Clostridia overgrowth, support gut microbiome balance, and monitor neurotransmitter-related patterns if dysbiosis is present.
HPHPA (3-(3-hydroxyphenyl)-3- hydroxypropionic acid) (16)	Produced by Clostridia bacteria via phenylalanine metabolism, HPHPA can disrupt catecholamine signaling, influencing dopamine/norepinephrine balance. Elevated levels are associated with behavioral, gastrointestinal, and neuropsychiatric conditions, and may reflect Clostridia overgrowth or environmental exposures affecting the microbiome. Initial Considerations: Support gut microbial balance, and monitor neurotransmitter-related patterns. Consider reducing environmental exposures like glyphosate or organophosphates that may favor dysbiosis.
4-Cresol (17)	Primarily produced by Clostridium difficile through tyrosine metabolism, and can inhibit dopamine beta-hydroxylase (DBH), leading to elevated dopamine and potentially toxic byproducts. Elevated levels are linked to behavioral, neuropsychiatric, cardiovascular, metabolic, and renal conditions, as well as environmental exposures like toluene, creosote, and pesticides. Initial Considerations: Support gut microbial balance, target Clostridia overgrowth, and reduce environmental toxic exposures. Monitoring dopamine/norepinephrine metabolite patterns may help guide interventions.
3-Indoleacetic Acid (IAA) (18)	Elevated levels may reflect Clostridia overgrowth, systemic inflammation, or certain genetic conditions like Hartnup's disease or PKU, and have been associated with autism, cardiovascular, kidney, and psychological disorders. IAA also exhibits beneficial effects, including reducing oxidative stress, lowering inflammation, and supporting gut mucosal immunity via the AhR pathway. Initial Considerations: Support gut microbial balance, manage Clostridia overgrowth, address environmental contributors such as glyphosate or organophosphate exposure, and monitor for tryptophan metabolic disruptions when indicated.

Oxalate Metabolites	
Glyceric Acid (19)	Elevated levels may reflect fungal or bacterial overgrowth, high fructose/glycerol intake, vitamin B3 or tryptophan insufficiency, or rare genetic disorders such as Primary Hyperoxaluria Type 2 or D-glyceric aciduria. High levels have also been linked to glucose intolerance, autoimmune conditions, and certain neuropsychiatric disorders. Initial Considerations: Support gut microbial balance, address dietary contributors (sugars, glycerol), correct nutrient insufficiencies (B3/tryptophan), and evaluate for genetic conditions if clinically indicated.
Glycolic Acid (20)	Elevated levels may result from bacterial or fungal overgrowth, toxic exposures (e.g., ethylene glycol, polyglycolate, trichloroacetic acid), B6 or glutathione deficiency, supplemental collagen intake, topical glycolic acid, or rare genetic conditions such as Primary Hyperoxaluria Type 1 or glycolic aciduria. Elevations are often associated with oxidative stress. Initial Considerations: Support gut microbial balance, address toxic exposures, optimize B6 and glutathione status, review dietary and supplement contributors, and evaluate for genetic conditions if clinically indicated.
Oxalic Acid (21)	Elevated oxalic acid can result from high-oxalate foods, microbial imbalances (yeast, mold, or low oxalate-metabolizing bacteria), certain toxins (ethylene oxide, ethylene glycol, trichloroacetic acid), nutrient insufficiencies (especially vitamin B6), fat malabsorption, or rare genetic hyperoxalurias. Excess oxalates may contribute to mineral deficiencies and have been associated with inflammation and pain across multiple body systems. Initial Considerations: Address gut microbial balance, reduce dietary oxalate intake, support B6 and fat absorption, evaluate environmental/toxin exposures, and consider genetic testing if indicated. Monitor mineral status (calcium, magnesium, iron) and adjust supplementation accordingly.

Glycolytic Cycle Metabolites

Lactic Acid (22)	Elevated lactic acid can indicate mitochondrial dysfunction, microbial overgrowth, toxin exposure, nutrient deficiencies (CoQ10, B vitamins, zinc, iron), or rare genetic/metabolic disorders. It may also rise with hypoxia, diabetes, intense exercise, or certain medications. Initial Considerations: Support mitochondrial function, address nutrient insufficiencies, evaluate microbial balance and toxin exposures, and monitor for conditions that may impair oxygen delivery or lactate clearance.
Pyruvic Acid (23)	Elevated pyruvate can indicate mitochondrial dysfunction, nutrient deficiencies (B1, B2, B3, B5, magnesium), toxin exposure, liver stress, or rare genetic/metabolic disorders. Levels often rise alongside lactic acid in impaired energy metabolism. Initial Considerations: Support mitochondrial function, correct nutrient insufficiencies, evaluate toxin exposures, and monitor for conditions affecting liver function or energy metabolism.

Mitochondrial markers – Krebs Cycle Metabolites

Succinic Acid (24)	Elevated succinic acid can indicate mitochondrial dysfunction, microbial imbalance, toxin exposure, or nutrient deficiencies (iron, riboflavin/B2, CoQ10). It may also rise with inflammation, hypoxia, or certain medications. Initial Considerations: Support mitochondrial function, correct nutrient insufficiencies, address microbial balance, evaluate toxin exposures, and monitor for inflammatory or metabolic conditions.
Fumaric Acid (25)	Elevated fumaric acid can indicate mitochondrial dysfunction, toxic exposures (metals, fungicides, mycotoxins), nutrient deficiencies (iron), or microbial influences (e.g., Aspergillus). It may also reflect metabolic changes in immune cells. Initial Considerations: Support mitochondrial function, address nutrient deficiencies, evaluate toxin and microbial exposures, and consider inflammation or immune-related metabolic shifts.
Malic Acid (26)	Elevated malic acid can indicate mitochondrial dysfunction, microbial imbalance, toxic exposures (arsenic, cadmium, mycotoxins), or nutrient deficiencies (B3/NAD). It may also reflect oxidative stress or dietary/supplement influences. Initial Considerations: Support mitochondrial function, correct nutrient deficiencies, address microbial and toxin exposures, and monitor oxidative stress and energy metabolism.
2-Oxoglutaric Acid (27)	Elevation can indicate mitochondrial dysfunction, microbial overgrowth, nutrient deficiencies (B1, B2, B3, B5, magnesium), or rare genetic disorders (fumarase or 2-ketoglutarate dehydrogenase deficiencies). It also plays a key role in nitrogen metabolism, protein synthesis, collagen production, and bone health. Initial Considerations: Support mitochondrial function, correct nutrient deficiencies, evaluate microbial balance, and consider metabolic or genetic causes if indicated.
Aconitic Acid (28)	Elevated aconitic acid can indicate mitochondrial dysfunction, toxic exposures (arsenic, aluminum, fluoride), nutrient deficiencies (iron, glutathione), or oxidative/nitrative stress. Initial Considerations: Support mitochondrial function, correct nutrient deficiencies (iron, glutathione, vitamin C, cysteine), reduce oxidative stress, and evaluate for relevant toxin exposures.
Citric Acid (29)	Elevated citric acid can indicate mitochondrial dysfunction, microbial overgrowth (fungi or yeast), toxic exposures (arsenic, aluminum, cadmium, mercury), or nutrient deficiencies (iron, glutathione). Low levels may result from impaired metabolism, hypokalemia, or iron overload, and may increase risk for oxalate stone formation. Initial Considerations: Support mitochondrial function, address nutrient deficiencies (iron, glutathione, magnesium), evaluate microbial balance, and consider toxin exposures. Dietary sources of citric acid may also influence values.

Mitochondrial Markers – Amino Acid Metabolites

3-Methylglutaric Acid (30)	Accumulates when mitochondrial function is impaired, particularly if the electron transport chain is compromised, activating the "acetyl-CoA diversion pathway." Rare genetic disorders (HMGCL or AUH deficiencies) and certain medications or dietary factors (ketogenic diet, TPN) may also elevate levels. Initial Considerations: Support mitochondrial function, review diet and nutrient status, evaluate for potential medication effects, and consider rare metabolic disorders if clinically indicated.
3-Hydroxyglutaric Acid (31)	Produced during lysine degradation. Elevated levels can indicate mitochondrial dysfunction and may act as an acidogen or metabotoxin. Rarely, chronic elevations are linked to glutaric aciduria type I (glutaryl-CoA dehydrogenase deficiency). Dietary factors (e.g., ketogenic diet) and certain medications (e.g., valproate) can also increase levels. Initial Considerations: Support mitochondrial function, review diet and nutrient status, evaluate for potential medication effects, and consider rare metabolic disorders if clinically indicated.
3-Methylglutaconic Acid (32)	Involved in leucine metabolism and the mevalonate shunt. Elevated levels can indicate mitochondrial dysfunction and may act as an acidogen or metabotoxin. Persistent elevations are linked to rare metabolic disorders such as 3-methylglutaconic acidurias and GAMT deficiency. Dietary factors (TPN, ketogenic diet) and medications (valproate, levetiracetam, aspirin, ibuprofen, acetaminophen) can also increase levels. Initial Considerations: Support mitochondrial function, review diet and nutrient status, evaluate for potential medication effects, and consider rare metabolic disorders if clinically indicated.

Neurotransmitter Metabolism

Phenylalanine and Tyrosine Metabolites	
Homovanillic Acid (HVA) (33)	HVA is the primary urinary metabolite of dopamine and reflects dopamine turnover. Elevated levels may indicate increased dopamine metabolism due to microbial imbalances, toxic exposures (heavy metals, pesticides, mycotoxins), stress, stimulant medications, or nutrient deficiencies (B2, B6, C, copper, magnesium). Low HVA may reflect impaired dopamine synthesis, enzyme dysfunction (COMT/MAO), or deficiencies in cofactors including BH4, B2, B6, C, iron, and magnesium. Initial Considerations: Address microbial imbalances, support methylation and neurotransmitter cofactors, and evaluate for relevant dietary, lifestyle, or toxic exposures.
Vanillylmanillic Acid) (VMA (34)	VMA is the primary urinary metabolite of norepinephrine and epinephrine, reflecting catecholamine metabolism. Elevated VMA may result from increased catecholamine turnover due to stress, microbial imbalances, toxic exposures (heavy metals, mycotoxins), nutrient deficiencies (B2, B3, B6, C, copper, magnesium), or stimulant medications. Low VMA can indicate impaired catecholamine synthesis or metabolism due to enzyme dysfunction (DBH, COMT, MAO), nutrient deficiencies, or microbial/toxin inhibition. Initial Considerations: Support catecholamine metabolism with nutrients, address microbial or toxic exposures, and manage stress and lifestyle factors.
HVA:VMA Ratio (35)	Reflects the conversion of dopamine to norepinephrine and its downstream metabolism, primarily regulated by dopamine beta-hydroxylase (DBH). An elevated ratio suggests impaired DBH activity, often from microbial imbalances (Clostridia), mycotoxins, heavy metals, nutrient deficiencies (B3, copper, vitamin C),or medications. A low ratio typically indicates reduced dopamine synthesis or turnover, often related to high cortisol levels or other inhibitory factors. Initial Considerations: Focus on supporting DBH activity through nutrients, addressing microbial or toxic exposures, and managing stress or contributing medications.
Dihydrophenylacetic Acid (DOPAC) (36)	DOPAC reflects dopamine metabolism and turnover, with elevated levels indicating increased dopamine breakdown often due to DBH inhibition from microbial imbalances, toxic exposures, or nutrient deficiencies (B6, B2, magnesium, copper, vitamin C). Low levels can reflect impaired dopamine synthesis, enzyme dysfunction (MAO/COMT), or cofactor deficiencies. Initial Considerations: Focus on supporting dopamine metabolism through nutritional cofactors, addressing microbial or toxic influences, and reviewing medications or dietary contributors.
HVA:DOPAC Ratio (37)	This ratio reflects the balance between dopamine breakdown via MAO to DOPAC and conversion of DOPAC to HVA via COMT. An elevated ratio suggests increased COMT activity or reduced MAO function, while a low ratio indicates reduced COMT activity or enhanced MAO activity. Influences include genetics, methylation status, nutrient cofactors (magnesium, SAMe, B12, folate), medications, and environmental toxins. Initial Considerations: Include supporting methylation, optimizing cofactors, and addressing medications, dietary factors, or toxic exposures.
Tryptophan Metabolites	
5-Hydroxyindoleacetic Acid (5-HIAA) (38)	Main metabolite of serotonin and reflects serotonin turnover. Elevated levels suggest increased serotonin breakdown, often influenced by gut dysbiosis, high-tryptophan/serotonin foods, medications (SSRIs, 5-HTP), or toxic exposures, while low levels may indicate reduced serotonin synthesis due to microbiome imbalance, nutrient deficiencies (B6, BH4, B2, zinc, magnesium), or MAO downregulation. Initial Consideration: Focus on supporting serotonin synthesis through gut health, dietary precursors, cofactors, and addressing relevant medications or toxic exposures.
Quinolinic Acid (39) (QA)	QA is a neuroactive metabolite from tryptophan metabolism via the kynurenine pathway. Elevated levels indicate inflammation-driven excitotoxicity, neuroinflammation, or dysregulated tryptophan metabolism, influenced by microbial imbalance, toxic exposures, nutrient deficiencies (B2, B3, B6), or high cortisol. High QA is associated with neurodegenerative, psychiatric, and cardiovascular conditions, and may affect NAD+ synthesis. Initial Considerations: Lower systemic inflammation, support the kynurenine pathway with B-vitamin cofactors (B2, B3, B6), modulate gut microbiome balance, address toxic exposures, and monitor stress/cortisol levels.
Kynurenic Acid (40) (KA)	KA is the neuroactive metabolite of tryptophan via the kynurenine pathway, contributing to NAD+ production and neuroprotection, but elevations often reflect inflammation, stress, or B-vitamin deficiencies and may disrupt serotonin balance or promote excitotoxicity. Low levels can result from B6 deficiency, low protein intake, high estrogen states, or certain medications. Initial Considerations: B-vitamin repletion (B2, B3, B6), adequate dietary tryptophan, manage inflammation and stress, and review medications or hormonal factors that may influence kynurenine metabolism.

Pyrimidine Metabolites – Folate Metabolism	
Uracil (41)	Accumulation often reflects impaired methylation or folate cycle dysfunction, as its conversion to thymine depends on active folate (B9) and related B vitamins (B2, B6, B12). Elevations may also occur with liver dysfunction, certain medications (e.g., 5-fluorouracil), or genetic variants affecting thymidylate synthase or DPD activity. Initial Considerations: Assess and support methylation cofactors (B2, B3, B6, B9, B12, magnesium), evaluate folate status and homocysteine levels, and address potential liver or genetic factors influencing pyrimidine metabolism.
Thymine (42)	DNA nucleobase synthesized from uracil through a folate-dependent methylation process, reflecting overall methylation efficiency and folate cycle integrity. Elevated thymine may indicate disruptions in folate or B-vitamin metabolism or reduced DPD enzyme activity, which also affects uracil and 5-fluorouracil clearance. Initial Considerations: support methylation with cofactors (B2, B3, B6, B9, B12), evaluate folate and homocysteine status, and consider possible genetic variants (e.g., MTHFR, DPD) if pyrimidine metabolism appears dysregulated.

Ketone and Fatty Acid Oxidation	
3-Hydroxybutyric Acid (43)	Key ketone body produced during fatty acid oxidation and serves as an alternative energy source during fasting, low-carbohydrate intake, or impaired glucose metabolism. Elevated levels suggest increased ketogenesis from fasting, ketogenic diets, diabetes, or mitochondrial dysfunction affecting fatty acid utilization. Initial Considerations: Assess diet, glucose regulation, and mitochondrial health, ensure adequate B3 (niacin) to support NADH balance, and evaluate for possible fatty acid oxidation disorders (e.g., carnitine or MCAD deficiency) when markedly elevated.and pulmonary infections.
Acetoacetic Acid (44)	Key ketone body produced from fatty acid oxidation within the mitochondria and serves as an alternative energy source during fasting, carbohydrate restriction, or impaired glucose metabolism. Elevated levels suggest increased ketogenesis due to fasting, ketogenic diets, diabetes, mitochondrial dysfunction, or fatty acid oxidation defects (e.g., carnitine or MCAD deficiency). Initial Considerations: Assess diet, glucose control, and mitochondrial function, with support for redox balance through niacin (B3) and evaluation of related ketone and fatty acid oxidation markers (43, 45–49).
Ethylmalonic Acid (45)	Reflects fatty acid oxidation efficiency and mitochondrial function. Elevated EMA may result from riboflavin (B2) or carnitine insufficiency, high-fat or vegan diets, or impaired mitochondrial beta-oxidation (e.g., MCAD or SCAD deficiencies). Initial Considerations: Support mitochondrial function with riboflavin and carnitine, moderating high-fat intake or MCT supplementation, and evaluating related fatty acid oxidation markers (46–49) for broader pathway insights.
Methylsuccinic Acid (46)	Reflects fatty acid oxidation and mitochondrial function, particularly in isoleucine metabolism. Elevated MSA may indicate mitochondrial dysfunction, carnitine or riboflavin deficiency, or high-fat/low-carb diets, and may be seen in conditions like MCAD deficiency, ethylmalonic encephalopathy, or glutaric acidemias though rare. Initial Considerations: support mitochondrial function with riboflavin and carnitine, review dietary fat intake, and evaluate related fatty acid oxidation markers (45, 47–49) for broader pathway context.

Ketone and Fatty Acid Oxidation Cont.	
Adipic Acid (47)	Reflects impaired fatty acid oxidation and mitochondrial stress, often rising when beta-oxidation is compromised, and omega-oxidation becomes more active. Elevations may indicate mitochondrial dysfunction, carnitine or riboflavin deficiency, or high-fat/low-carb dietary patterns. Initial Considerations: support mitochondrial function with carnitine, riboflavin, and magnesium, optimize macronutrient balance, and review related fatty acid oxidation markers (45–49) for broader metabolic context.
Suberic Acid (48)	Marker of impaired beta-oxidation, reflecting a compensatory shift toward omega-oxidation and mitochondrial stress. Elevations may suggest mitochondrial dysfunction, carnitine or riboflavin deficiency, or dietary influences such as high-fat or low-carb intake. Initial Considerations: support mitochondrial function with carnitine, riboflavin, and magnesium, moderate excessive fat intake, and assess related fatty acid oxidation markers (45–49) for broader metabolic insight.
Sebacic Acid (49)	Reflects impaired fatty acid oxidation and mitochondrial inefficiency, often secondary to carnitine or riboflavin insufficiency, high-fat or MCT-rich diets, or mitochondrial stress. It may also provide contextual clues for potential toxicant exposure from industrial compounds or cosmetic ingredients. Initial Considerations: Support mitochondrial beta-oxidation with carnitine and riboflavin, moderate excessive fat or MCT intake, and evaluate related fatty acid oxidation markers (45–48) for broader pathway assessment.

Nutritional Markers **A high value for this marker may indicate a deficiency of this vitamin.*	
***Methylmalonic Acid (Vitamin B12) (50)**	A sensitive functional marker for vitamin B12 deficiency and impaired propionic acid metabolism, linking amino acid and fatty acid oxidation to the Citric Acid Cycle. Elevations may reflect B12 or biotin insufficiency, gut microbial imbalance, or mitochondrial dysfunction. Initial Considerations: Assess and replete vitamin B12 (preferably as adenosylcobalamin and methylcobalamin), ensure adequate biotin levels, and investigate factors affecting B12 absorption such as GI health or medication use (e.g., metformin, PPIs).
Pyridoxic Acid (Vitamin B6) (51)	Reflects vitamin B6 status, which is essential for neurotransmitter synthesis, methylation, detoxification, and mitochondrial energy production. Elevated levels may indicate high B6 intake, medication effects, or renal stress, while low levels can reflect inadequate intake, gut dysbiosis, or impaired activation due to low riboflavin. Initial Considerations: Optimize vitamin B6 status , asses for B2 cofactor sufficiency, review medication use (e.g., isoniazid, oral contraceptives, antiepileptics), and support gut and mitochondrial health.
Pantothenic Acid (Vitamin B5) (52)	Essential for Coenzyme A synthesis, supporting energy production, fatty acid metabolism, and mitochondrial function. High levels may reflect recent dietary intake, supplementation, or rare genetic conditions like PANK2 mutations, while low levels can indicate insufficient intake or microbial insufficiency (e.g., low Lactobacillus production). Initial Considerations: Ensure adequate dietary B5, support mitochondrial energy pathways, review supplement use, and evaluate microbial contributions.
***Glutaric Acid (Vitamin B2) (53)**	A key marker of riboflavin (B2)-dependent amino acid and fatty acid metabolism. Elevated levels may indicate B2 deficiency, mitochondrial dysfunction, or, rarely, genetic disorders such as glutaric acidemia type I or II. Initial Considerations: Evaluate B2 status, support mitochondrial energy and neurotransmitter pathways, assess methylation/detoxification support, and review dietary intake or medications that may influence B2 levels.
Ascorbic Acid (Vitamin C) (54)	Essential for antioxidant protection, collagen synthesis, neurotransmitter metabolism, and mitochondrial function. Low levels may indicate increased oxidative stress, nutritional insufficiency, or higher demand from illness, smoking, or metabolic stress. Initial Considerations: Evaluate dietary intake, supplementation, and cofactor needs such as iron and zinc. Also monitor mitochondrial and neurotransmitter-related markers.
***3-Hydroxy-3-methylglutaric Acid (Coenzyme Q10) (55)**	Linked to low CoQ10 and mitochondrial dysfunction. Elevations may indicate deficiencies in COQ10, impaired HMG-CoA lyase activity, rare inborn errors of metabolism, or secondary effects from mitochondrial stress and statin use. Initial Considerations: Evaluate CoQ10 status, mitochondrial function, and potential influences from medications, such as statins. This marker also connects to pathways impacting cholesterol and CoQ10 synthesis, so broader metabolic assessment may be warranted.
N-Acetylcysteine Acid (56)	NAC is a key precursor to glutathione, supporting antioxidant defense and detoxification. Elevated levels typically reflect supplementation or, rarely, enzymatic acylation issues, while low levels are usually expected when NAC supplementation is not being utilized. Initial Considerations: Evaluate glutathione status, oxidative stress, and potential interactions with NAC supplementation. When combined with pyroglutamic acid (58), elevations may indicate impaired conversion to cysteine, influencing antioxidant capacity.
***Methylcitric Acid (Biotin, Vitamin H) (57)**	Methylcitric acid is a key biomarker for biotin (B7) sufficiency, reflecting the activity of propionyl-CoA carboxylase. Elevated levels indicate impaired propionyl-CoA metabolism, which can result from biotin deficiency, B12 deficiency, microbial imbalances, or rare genetic conditions such as biotinidase deficiency or methylcitric acidemia. Initial Considerations: Assess biotin status and consider supplementation if levels are insufficient, evaluate for microbial dysbiosis or overgrowth of propionic-acid-producing bacteria. Also consider dietary influences and review B12 status.

Indicators of Detoxification **A high value for this marker may indicate a Glutathione deficiency.* ***High values may indicate methylation defects and/or toxic exposures.*	
***Pyroglutamic Acid (58)**	Elevated pyroglutamic acid may indicate impaired glutathione synthesis, often due to oxidative stress, mitochondrial dysfunction, microbial imbalance, toxic exposures, or nutritional deficiencies (B6, magnesium, glycine, cysteine, glutamine). It reflects disruptions in the gamma-glutamyl cycle, impacting antioxidant capacity and detoxification. Initial Considerations: Support glutathione production through nutrient repletion, address oxidative stress and microbial imbalances, and review medications or supplements that may deplete glutathione.
****2-Hydroxybutyric Acid (59)**	Elevated 2-hydroxybutyric acid is a byproduct of the breakdown of the sulfur amino acid metabolite, cystathionine, which may be formed in excess during oxidative stress or when toxic exposures increase the need for detoxification. When glutathione is depleted by excessive toxic exposure, pyroglutamic acid may also be elevated. May also be elevated due to certain genetic SNPs in the methylation pathway or deficiencies of methyl tetrahydrofolate, methyl B12, or betaine. Initial Considerations: Support nutrients associated with methylation and transsulfuration, reduce oxidative stress, investigate potential exposures and/or genetic SNPs.
Orotic Acid (60)	Elevated orotic acid often reflects urea cycle dysfunction or hyperammonemia, where excess carbamoyl phosphate is shunted into pyrimidine synthesis. It may also rise with microbial imbalances (e.g., E. coli, Candida), nutrient insufficiencies (arginine, B12, magnesium, zinc), or liver dysfunction. Methylation defects and BH4 insufficiency can further contribute by impairing ammonia detoxification and neurotransmitter synthesis. Initial Considerations: Assess urea cycle cofactors (arginine, B12, B2, magnesium), methylation status, gut dysbiosis, and liver function.
2-Hydroxyhippuric Acid (61)	Elevated 2-hydroxyhippuric acid often reflects high salicylate or aspartame exposure, microbial overgrowth (especially Clostridia), or increased metabolism of aromatic compounds. Gut bacteria can convert phenolic and aromatic amino acids into benzoate derivatives that conjugate with glycine to form this metabolite. Environmental toxicants and salicylate-containing foods or medications (e.g., aspirin, topical salicylic acid) may further raise levels. Initial Considerations: Assess salicylate and aspartame intake, glycine status, microbiome balance, and potential toxin or medication influences.

Amino Acid Metabolites	
2-Hydroxyisovaleric Acid (62)	2-Hydroxyisovaleric acid is a valine metabolite reflecting branched-chain amino acid (BCAA) breakdown and mitochondrial energy metabolism. Elevated levels can indicate mitochondrial dysfunction, BCAA metabolism impairment, or thiamine (B1) and related nutrient deficiencies, and may also suggest rare genetic disorders like maple syrup urine disease or other inborn errors of metabolism. Initial Considerations: Assess BCAA intake, mitochondrial cofactors (B1, B2, B3, B5, lipoic acid, magnesium), and evaluate for potential metabolic or genetic contributors. Low values are generally not clinically significant.
2-Oxoisovaleric Acid (63)	2-Oxoisovaleric acid is a valine-derived keto acid that reflects branched-chain amino acid (BCAA) metabolism and mitochondrial energy production. Elevated levels can indicate impaired BCKDH enzyme activity, nutrient deficiencies (B1, B2, B3, B5, lipoic acid, magnesium), or genetic disorders such as maple syrup urine disease (MSUD). Initial Considerations: Evaluate BCAA intake, mitochondrial cofactors, and potential metabolic or genetic contributors. Low values are typically not clinically significant.
3-Methyl-2-oxovaleric Acid (64)	3-Methyl-2-oxovaleric acid is an isoleucine-derived branched-chain keto acid that reflects BCKDH enzyme activity and mitochondrial energy metabolism. Elevated levels can indicate impaired mitochondrial function, nutrient deficiencies (B1, B2, B3, B5, lipoic acid, magnesium), excessive BCAA intake, or rare genetic disorders such as MSUD, propionic, methylmalonic, or isovaleric acidemias. Initial Considerations: Evaluate BCAA intake, cofactors supporting BCKDH, medications affecting metabolism, and potential metabolic or genetic contributors. Low values are generally not clinically significant.
2-Hydroxyisocaproic Acid (65)	2-Hydroxyisocaproic acid is a leucine-derived metabolite reflecting branched-chain amino acid (BCAA) metabolism and mitochondrial function. Elevated levels can result from impaired BCKDH enzyme activity due to nutrient deficiencies (B1, B2, B3, B5, B6, lipoic acid, magnesium), microbial production (fungi, lactic acid bacteria, Clostridium), high intake of fermented foods or probiotics, or rare genetic conditions such as MSUD or E3 deficiency. Initial Considerations: Evaluate nutrient cofactors for BCAA metabolism, dietary and microbial influences, and potential metabolic or genetic contributors. Low values are not clinically significant.
2-Oxoisocaproic Acid (66)	2-Oxoisocaproic acid is a leucine-derived keto acid reflecting branched-chain amino acid (BCAA) metabolism and mitochondrial energy production. Elevated levels can result from BCKDH dysfunction due to nutrient deficiencies (B1, B2, B3, B5, B6, lipoic acid, magnesium), high-protein or leucine-rich diets, metabolic stress, microbial dysbiosis, certain medications, or rare genetic conditions such as MSUD or isovaleric acidemia. Initial Considerations: Assess mitochondrial health, nutrient cofactors, dietary and microbial influences, and potential metabolic or genetic contributors. Low values are not clinically significant.
2-Oxo-4-methiolbutyric Acid (67)	An intermediate in methionine metabolism that reflects transamination pathway activity and methylation balance. Elevated levels can indicate vitamin B6 deficiency, excessive methionine intake, or genetic variations affecting sulfur amino acid metabolism, and may contribute to accumulation of potentially toxic byproducts. Initial Considerations: Evaluate methylation status, B6 and related nutrient status, dietary methionine intake, and possible metabolic or genetic contributors. Low values are not clinically significant.
Mandelic Acid (68)	Mandelic acid is a metabolite of phenylalanine and a key biomarker for environmental exposure to styrene or ethylbenzene. Elevated levels may reflect impaired dopamine metabolism, cofactor deficiencies (iron, B2, BH4), or, rarely, genetic conditions like PKU. Initial Considerations: Assess environmental exposures, evaluate neurotransmitter and phenylalanine metabolism, and check relevant nutrient status. Low values are not clinically significant.
Phenyllactic Acid (69)	Formed when phenylalanine is diverted from tyrosine synthesis, typically due to impaired phenylalanine hydroxylase (PAH) activity or BH4 deficiency. Elevated levels may reflect nutrient insufficiencies (iron, B2, folate, vitamin C), gut microbial contributions (Clostridium species), or, rarely, genetic conditions such as PKU. Initial Considerations: Support PAH function via cofactors, assess BH4 status, evaluate neurotransmitter metabolites, and reviewing potential microbial imbalances. Low values are not clinically significant.
Phenylpyruvic Acid (70)	Phenylpyruvic acid is produced when phenylalanine is diverted from tyrosine synthesis, typically due to reduced phenylalanine hydroxylase (PAH) activity or impaired BH4 function. Elevated levels may indicate nutrient deficiencies (iron, B2, folate, vitamin C), genetic conditions like PKU, or excessive dietary phenylalanine. Initial Considerations: Support PAH and BH4 activity, evaluate phenylalanine and tyrosine related neurotransmitter metabolites, and address potential nutrient or microbial factors. Low values are not clinically significant.

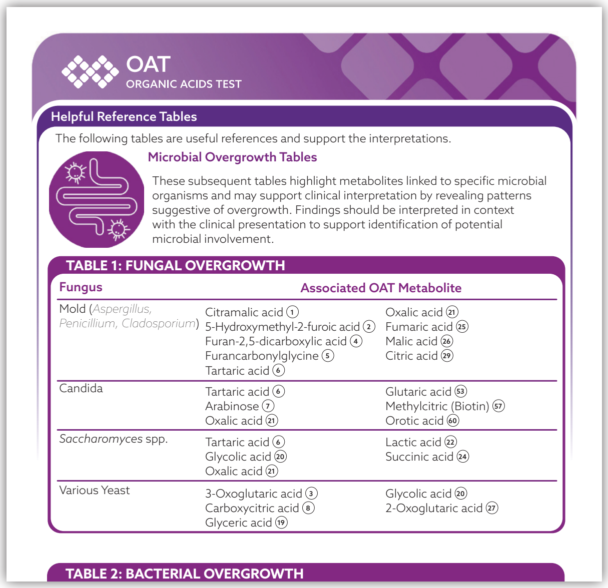
Amino Acid Metabolites Cont.	
Homogentisic Acid (71)	Homogentisic acid (HGA) is an intermediate in tyrosine breakdown, formed when tyrosine metabolism diverges from dopamine and catecholamine synthesis. Elevated levels often reflect impaired activity of homogentisate 1,2-dioxygenase (HGD), as seen in alkaptonuria, or may be influenced by metabolic stress such as uncontrolled hyperglycemia. HGA feeds into the citric acid cycle via maleylacetoacetate and fumarate, linking tyrosine metabolism to mitochondrial energy production. Initial Considerations: Genetic testing may be considered if elevated and corresponding clinical symptoms are present. Low values have no known clinical significance.
4-Hydroxyphenyllactic Acid (72)	4-Hydroxyphenyllactic acid (4-HPLA) is formed from tyrosine via transamination to 4-hydroxyphenylpyruvic acid followed by reduction. Elevations can result from microbial activity (Bifidobacteria, Lactobacillus, Clostridium), excessive dietary tyrosine, or rare genetic defects in HPD leading to tyrosinemia. High 4-HPLA may indicate dysbiosis and can influence neurotransmitter synthesis by altering large neutral amino acid transport into the brain. Initial Considerations: Assess microbiome, and review tyrosine realted neurotransmitters for further insights. Low values have no known clinical significance.
N-Acetylaspartic Acid (73)	N-Acetylaspartic acid is synthesized in neurons from aspartate and acetyl-CoA and supports fluid balance, myelin synthesis, and neurotransmitter function. Elevated NAA is primarily linked to Canavan disease, a rare autosomal recessive disorder caused by mutations in the ASPA gene, resulting in aspartoacylase deficiency. Initial Considerations: Genetic testing may be considered if elevated and corresponding clinical symptoms are present. Low values have no known clinical significance.
Malonic Acid (74)	Malonic acid is involved in fatty acid metabolism and energy production. Elevations may result from impaired mitochondrial function or acrolein exposure from polluted air or burning cooking oils past their smoke point. Markedly high levels are associated with malonyl-CoA decarboxylase deficiency, often alongside elevated methylmalonic acid (50). Initial Considerations: Review mitochondria section and support as needed. Also consider reducing exposures to acrolein, and supporting detoxification, if warranted. Low values have no known clinical significance.
4-Hydroxybutyric Acid (75)	4-Hydroxybutyric acid is formed from GABA degradation in the brain and liver, influencing inhibitory and excitatory neurotransmission via GABA and GHB receptors. Elevated 4-HBA may result from microbial production (e.g., Clostridium, Pseudomonas, Saccharomyces), GHB ingestion, or rare metabolic disorders such as SSADH deficiency. NAD , derived from vitamin B3, is required for its metabolism. Initial Considerations: Assess microbial overgrowth, support B3, evaluate neurotransmitters, and if extremely elevated, may consider genetic testing or intake of GHB. Low values have no known clinical significance.

Mineral Metabolites	
Phosphoric Acid (76)	Phosphoric acid reflects phosphate status, essential for ATP production, bone health, and cellular signaling. Elevated levels may result from high dietary phosphate or vitamin D intake, phosphate additives, heavy metal exposure (e.g., lead, uranium), or conditions causing increased bone resorption or renal phosphate loss. Low levels may indicate poor intake, absorption, or kidney/liver dysfunction. Initial Considerations: review dietary and supplement phosphorus sources, vitamin D status, renal function, parathyroid, and potential toxicant exposures.

Provider Support Guide

A helpful tool to assist interpretation and application of test results

Reference Tables – key metabolites that support interpretation and reveal patterns of imbalance.



OAT
ORGANIC ACIDS TEST

Helpful Reference Tables

The following tables are useful references and support the interpretations.

Microbial Overgrowth Tables

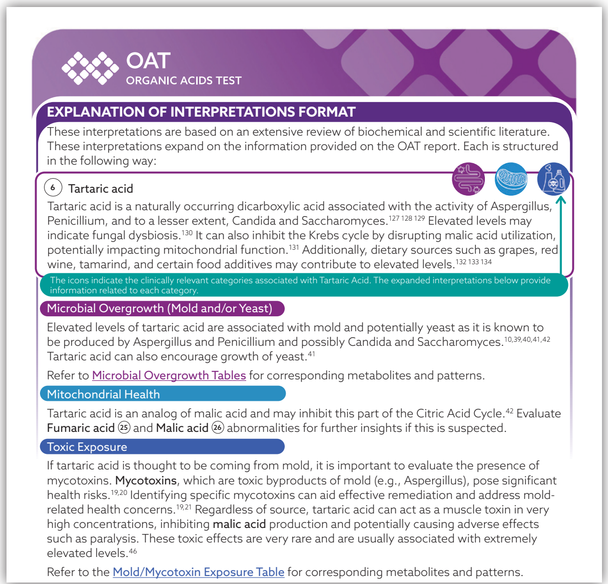
These subsequent tables highlight metabolites linked to specific microbial organisms and may support clinical interpretation by revealing patterns suggestive of overgrowth. Findings should be interpreted in context with the clinical presentation to support identification of potential microbial involvement.

TABLE 1: FUNGAL OVERGROWTH

Fungus	Associated OAT Metabolite	
Mold (*Aspergillus, Penicillium, Cladosporium*)	Citramalic acid ① 5-Hydroxymethyl-2-furoic acid ② Furan-2,5-dicarboxylic acid ④ Furancarbonylglycine ⑤ Tartaric acid ⑥	Oxalic acid ㉑ Fumaric acid ㉕ Malic acid ㉖ Citric acid ㉙
Candida	Tartaric acid ⑥ Arabinose ⑦ Oxalic acid ㉑	Glutaric acid ㊸ Methylcitric (Biotin) ㊼ Orotic acid ㊿
Saccharomyces spp.	Tartaric acid ⑥ Glycolic acid ⑳ Oxalic acid ㉑	Lactic acid ㉒ Succinic acid ㉔
Various Yeast	3-Oxoglutaric acid ③ Carboxycitric acid ⑧ Glyceric acid ⑲	Glycolic acid ⑳ 2-Oxoglutaric acid ㉗

TABLE 2: BACTERIAL OVERGROWTH

Detailed Interpretations – organized into the clinical categories the analyte may impact.



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ORGANIC ACIDS TEST

EXPLANATION OF INTERPRETATIONS FORMAT

These interpretations are based on an extensive review of biochemical and scientific literature. These interpretations expand on the information provided on the OAT report. Each is structured in the following way:

⑥ Tartaric acid

Tartaric acid is a naturally occurring dicarboxylic acid associated with the activity of Aspergillus, Penicillium, and to a lesser extent, Candida and Saccharomyces.[127 128 129] Elevated levels may indicate fungal dysbiosis.[130] It can also inhibit the Krebs cycle by disrupting malic acid utilization, potentially impacting mitochondrial function.[131] Additionally, dietary sources such as grapes, red wine, tamarind, and certain food additives may contribute to elevated levels.[132 133 134]

The icons indicate the clinically relevant categories associated with Tartaric Acid. The expanded interpretations below provide information related to each category.

Microbial Overgrowth (Mold and/or Yeast)

Elevated levels of tartaric acid are associated with mold and potentially yeast as it is known to be produced by Aspergillus and Penicillium and possibly Candida and Saccharomyces.[10,39,40,41,42] Tartaric acid can also encourage growth of yeast.[41]

Refer to Microbial Overgrowth Tables for corresponding metabolites and patterns.

Mitochondrial Health

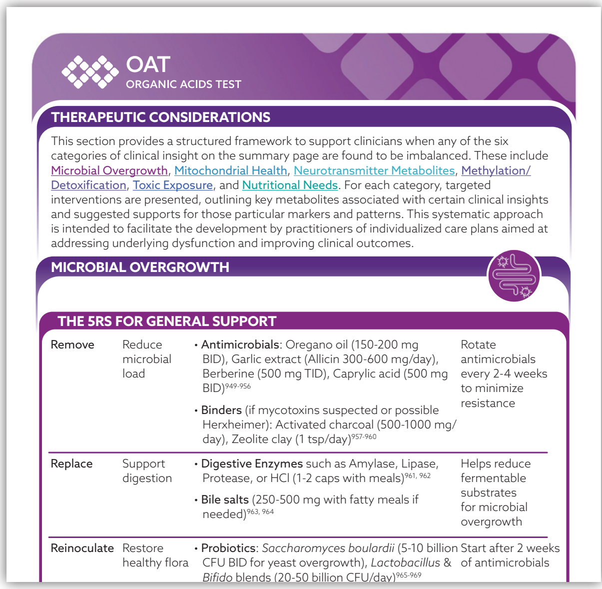
Tartaric acid is an analog of malic acid and may inhibit this part of the Citric Acid Cycle.[42] Evaluate **Fumaric acid** ㉕ and **Malic acid** ㉖ abnormalities for further insights if this is suspected.

Toxic Exposure

If tartaric acid is thought to be coming from mold, it is important to evaluate the presence of mycotoxins. **Mycotoxins**, which are toxic byproducts of mold (e.g., Aspergillus), pose significant health risks.[19,20] Identifying specific mycotoxins can aid effective remediation and address mold-related health concerns.[19,21] Regardless of source, tartaric acid can act as a muscle toxin in very high concentrations, inhibiting **malic acid** production and potentially causing adverse effects such as paralysis. These toxic effects are very rare and are usually associated with extremely elevated levels.[46]

Refer to the Mold/Mycotoxin Exposure Table for corresponding metabolites and patterns.

Therapeutic Considerations – guides treatment addressing imbalances within clinical categories.



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THERAPEUTIC CONSIDERATIONS

This section provides a structured framework to support clinicians when any of the six categories of clinical insight on the summary page are found to be imbalanced. These include Microbial Overgrowth, Mitochondrial Health, Neurotransmitter Metabolites, Methylation/Detoxification, Toxic Exposure, and Nutritional Needs. For each category, targeted interventions are presented, outlining key metabolites associated with certain clinical insights and suggested supports for those particular markers and patterns. This systematic approach is intended to facilitate the development by practitioners of individualized care plans aimed at addressing underlying dysfunction and improving clinical outcomes.

MICROBIAL OVERGROWTH

THE 5RS FOR GENERAL SUPPORT

Remove	Reduce microbial load	• **Antimicrobials:** Oregano oil (150-200 mg BID), Garlic extract (Allicin 300-600 mg/day), Berberine (500 mg TID), Caprylic acid (500 mg BID)[949-956] • **Binders** (if mycotoxins suspected or possible Herxheimer): Activated charcoal (500-1000 mg/day), Zeolite clay (1 tsp/day)[957-960]	Rotate antimicrobials every 2-4 weeks to minimize resistance
Replace	Support digestion	• **Digestive Enzymes** such as Amylase, Lipase, Protease, or HCl (1-2 caps with meals)[961, 962] • **Bile salts** (250-500 mg with fatty meals if needed)[963, 964]	Helps reduce fermentable substrates for microbial overgrowth
Reinoculate	Restore healthy flora	• **Probiotics:** *Saccharomyces boulardii* (5-10 billion CFU BID for yeast overgrowth), *Lactobacillus* & *Bifido* blends (20-50 billion CFU/day)[965-969]	Start after 2 weeks of antimicrobials

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