DEMO DEMO

Name: DEMO DEMO
Date of Birth: 00-00-0000
Biological Sex: Male
Age: 48
Height:
Weight:
Fasting:

Telephone: 000-000-0000
Street Address:
Email:

FINAL REPORT

Accession ID: 2407077216

Provider Information

Practice Name: DEMO CLIENT, MD
Provider Name: DEMO CLIENT, MD
Phlebotomist: 0

Telephone: 000-000-0000
Address: 3521 Leonard Ct, Santa Clara, CA 95054

Specimen Information

Sample Type	Collection Time	Received Time	Report	Final Report Date
Metal Free Urine	Local 2026-07-19 11:30	2026-07-20 09:28 (PDT)	Oxidative Stress - P7 Organic Acids - P15	2026-08-03 15:33 (PDT) 2026-07-28 14:42 (PDT)

SAMPLE

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VibrantWellness

Cellular Zoomer

Cellular Zoomer - Summary Pg 2

Oxidative Stress Pg 7

Organic Acids Pg 15



SAMPLE

Cellular Zoomer

INTRODUCTION

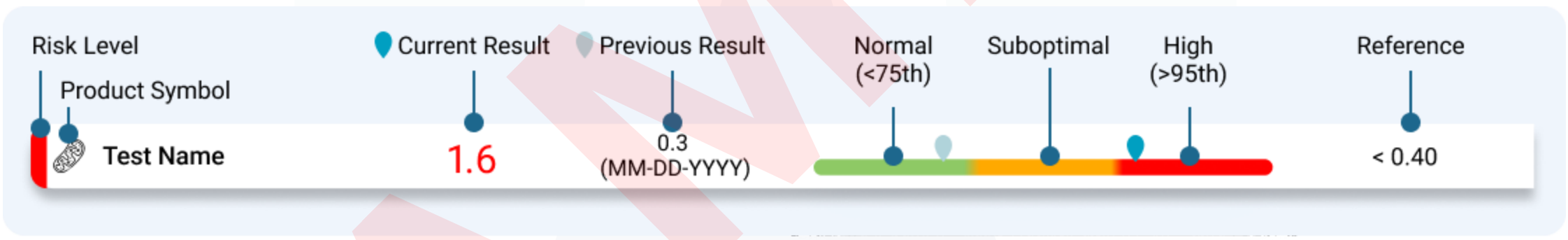
Vibrant Wellness is pleased to present Cellular Zoomer testing to support healthy lifestyle choices in consultation with your healthcare provider. The Cellular Zoomer provides quantitative assessment of cellular energy and metabolic chemistry, oxidative and nitrative stress signals, detox and clearance pathways, direct oxidative markers, neurotransmitter and stress chemistry, and microbial metabolic byproducts. Results are intended to be interpreted by healthcare providers to guide personalized strategies supporting cellular energy production, oxidative balance, and detoxification capacity.

Methodology

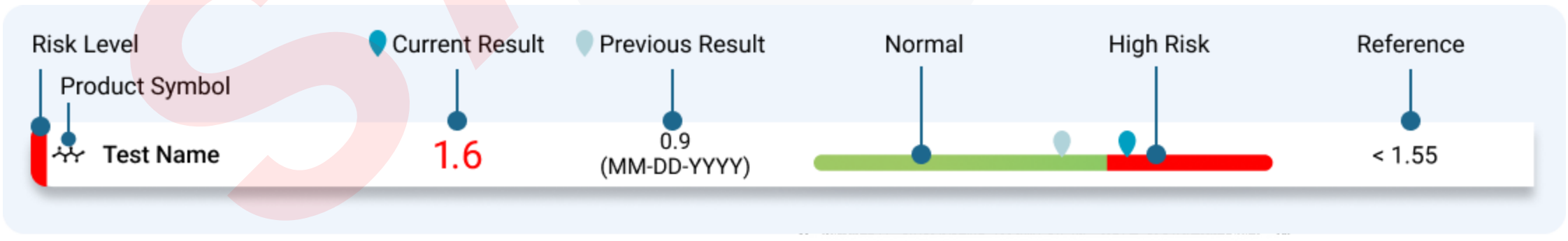
The Cellular Zoomer uses gas chromatography tandem mass spectrometry (GC-MS/MS) for quantitative detection of organic acids in urine samples. Additionally, catecholamine metabolites and serotonin and kynurenine metabolites, and oxidative stress markers are measured using liquid chromatography tandem mass spectrometry methodology (LC-MS/MS). Urine creatinine is measured using a kinetic colorimetric assay based on the Jaffé method. All organic acids and oxidative stress markers are reported as quantitative results normalized to urine creatinine to account for urine dilution variations.

Interpretation of Report

The report begins with the summary page which lists the markers whose levels are high or moderate based on the reference range. Reference ranges were established using a cohort of apparently healthy adults over 18 years of age, and pediatric reference ranges are not available. This is followed by a graphical representation of the overall oxidative stress score which is calculated using the results from all urine markers tested applied to a linear regression model. The score in green represents a normal score based on 50th percentile population, the score in yellow represents a moderate score based on 90th percentile and the score in red represent a high score based on the relatively healthy population. Additionally, the previous value is also indicated to help check for improvements every time the test is ordered. Following this section is the complete list of the markers results and their absolute levels are normalized with respect to Creatinine in a histogram format to enable a full overview along with the reference ranges. The level of the analyte with reference range is shown with three shades of color – Green, Yellow and Red. The result in green corresponds to 0 to 75th percentile indicates mild detection of the analyte. The result in yellow corresponds to 75th to 95th percentile indicates moderate detection of the analyte whereas the result in red corresponding to greater than 95th percentile indicates high detection of the analyte. The reference metric is listed to the right of the reference range. The previous and current result are listed to the left of the reference range. (result example illustration below)



The organic acids, which are represented normalized to urine creatinine, are displayed in a bar graph form to enable a full overview along with the reference ranges. The level of the organic acid has a green (normal) or red (high/low) highlight around the cell indicating the corresponding result based on the reference range of each organic acid. Additionally, the previous value (if available) is also indicated to help check for improvements every time the test is ordered. The reference metric is listed to the right of the reference range. The previous and current result are listed to the left of the reference range. (result example illustration below)



The Lab uses a “Weighted Ranking Approach” for supplement suggestions. The ranking considers both how often a supplement is recommended and its evidence-based rating. This means higher-rated supplements will appear at the top, ensuring that those with a 5-star rating come first, followed by 4-star, 3-star, and so on. This weighted ranking system for Supplement Suggestions provides helpful evidence-based suggestions for healthcare providers and patients to make informed choices. Vitamin and mineral supplement suggestions are age-matched and biological sex-matched

Continued on next page

Cellular Zoomer

INTRODUCTION

Please note: Consider all supplements in relation to medical history and symptoms. Not all recommended supplements are appropriate in all individual cases. It is important that you discuss any modifications to your diet, exercise, drug, and/or nutritional supplementation with your healthcare provider before making any changes. For urine creatinine, the reportable range is 0.05-16.02 mg/mL. Tests with urine creatinine <0.05 and >16.02mg/mL will be designated as Test Not Performed (TNP) and a retest would be requested. All tests with urine creatinine between 0.05-16.02mg/mL will be processed and included in the final patient reports.

Regulatory Disclaimer: This test was performed by Vibrant America Clinical Laboratory at 3521 Leonard Ct, Santa Clara, CA 95054 (CLIA No. 05D2078809, CAP No. 8970308). This test was developed, and its performance characteristics determined, by Vibrant America Clinical Laboratory. This test has not been cleared or approved by the U.S. Food and Drug Administration (FDA).

SAMPLE

Patient Name: DEMO DEMO
Date of Birth: 00-00-0000 Accession ID: 2407077216
Service Date: Local 9999-12-31 15:59

Cellular Zoomer - Summary

High

Oxidative Stress | Organic Acids

Oxidative Stress	Current	Previous	Result (75th / 95th)	Reference
Nε-carboxyethyllysine (CEL) (ug/g)	93.45		19.5 / 91	≤19.5
8-Hydroxy-2 - deoxyguanosine (ug/g)	11.60		1.14 / 4	≤1.14
8-Hydroxyguanosine (ug/g)	97.89		44.9 / 95.3	≤44.9
15(R)-Prostaglandin F2α (ug/g)	0.31		0.07 / 0.22	≤0.07
8-iso-prostaglandin F2α (8-iso-PGF2α) (ug/g)	0.48		0.1 / 0.26	≤0.1
Glutathione 4-hydroxynonenal (GS-HNE) (ug/g)	2.70		0.3 / 2.5	≤0.3
Malondialdehyde (ug/g)	168.40		72.87 / 163.53	≤72.87
3-Bromotyrosine (ug/g)	362.78		167.53 / 349.6	≤167.53

Organic Acids	Current	Previous	Result	Reference
2-Oxoisovaleric (mmol/mol)	3.40		0 / 2	≤2.0
3-Methyl-2-oxovaleric Acid (mmol/mol)	3.10		0 / 2.6	≤2.6
4-Hydroxyphenyllactic Acid (mmol/mol)	2.10		0 / 0.84	≤0.84
Phenyllactic Acid (mmol/mol)	0.48		0 / 0.21	≤0.21
Pyroglutamic Acid (mmol/mol)	41.00		0 / 10.13 / 32.45	10.14-32.45
2-Hydroxybutyric Acid (mmol/mol)	2.60		0 / 0.05 / 1.58	0.06-1.58
Adipic Acid (mmol/mol)	5.60		0 / 0.03 / 3.9	0.04-3.9
Ethylmalonic Acid (mmol/mol)	3.60		0 / 0.46 / 2.74	0.47-2.74
Suberic Acid (mmol/mol)	3.40		0 / 0.15 / 2.18	0.16-2.18
Lactic Acid (mmol/mol)	64.00		0 / 50.4	≤50.4
Cis-aconitic Acid (mmol/mol)	29.92		0 / 6.09 / 27.9	6.1-27.9
3-Hydroxyglutaric Acid (mmol/mol)	7.90		0 / 4.9	≤4.9
3-Methylglutaconic (mmol/mol)	8.60		0 / 6.2	≤6.2
Dihydroxyphenylpropionic Acid(DHPPA) (mmol/mol)	0.72		0 / 0.44	≤0.44

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Patient Name: DEMO DEMO
Date of Birth: 00-00-0000 Accession ID: 2407077216
Service Date: Local 9999-12-31 15:59

Cellular Zoomer - Summary

High

Organic Acids	Current	Previous	Result	Reference
Hippuric Acid (mmol/mol)	720.00		0 607	≤607.0
4-Hydroxyphenylacetic Acid (mmol/mol)	22.00		0 20.1	≤20.1
Dihydroxyphenylacetic Acid (DOPAC) (mcg/g)	1.10		0 577.29 1655	577.3-1655.5
Homovanillic Acid (HVA) (mcg/g)	4.10		0 3534 8455	3535.0-8455.0
Vanillylmandelic Acid (VMA) (mcg/g)	3.20		0 2411 5047	2411.2-5047.8
5-Hydroxyindoleacetic Acid (5-HIAA) (mcg/g)	5.20		0 1710 9788	1711.0-9788.0
Kynurenic Acid (mcg/g)	1.70		0 125.59 991.3	125.6-991.3
Quinolinic Acid (mcg/g)	3.30		0 610.29 2432	610.3-2432.9
Phosphoric Acid (mmol/mol)	31		0 999 5000	1000.0-5000.0
Glycolic Acid (mmol/mol)	0.65		0 12.59 128.7	12.6-128.7

Suboptimal

Oxidative Stress Organic Acids

Oxidative Stress	Current	Previous	Result (75th / 95th)	Reference
11-β-Prostaglandin F2α (ug/g)	0.29		0.11 0.4	≤0.11
Dityrosine (ug/g)	2.20		1.31 5	≤1.31

Creatinine

No markers are outside the normal reference range

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Oxidative Stress

INTRODUCTION

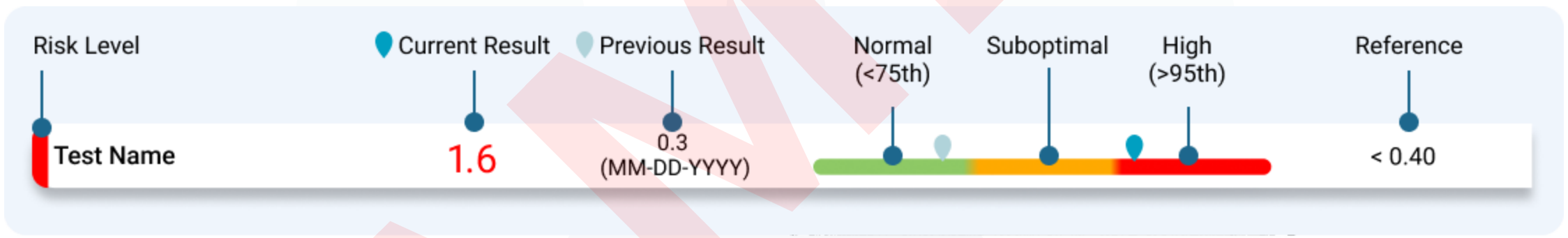
Vibrant Wellness is pleased to present the Oxidative Stress Panel to support healthy lifestyle choices in consultation with your healthcare provider. The Oxidative Stress Panel provides quantitative assessment of lipid peroxidation markers, DNA damage indicators, RNA damage markers, protein oxidation products, and advanced glycation end-products. Results are intended to be interpreted by healthcare providers to guide personalized wellness strategies informed by insights into oxidative health status and markers of cellular damage.

Methodology

The Oxidative Stress panel uses liquid chromatography tandem mass spectrometry methodology (LC-MS/MS) for quantitative detection of damage markers in urine samples. Urine creatinine is measured using a kinetic colorimetric assay based on the Jaffé method. All damage markers are reported as the quantitative result normalized to urine creatinine to account for urine dilution variations.

Interpretation of Report

The report begins with the summary page which lists the damage markers whose levels are high or moderate based on the reference range. Reference ranges were established using a cohort of apparently healthy adults over 18 years of age, and pediatric reference ranges are not available. This is followed by a graphical representation of the overall oxidative damage score which is calculated using the results from all urine damage markers tested applied to a linear regression model. The score in green represents a normal score based on 50th percentile population, the score in yellow represents a moderate score based on 90th percentile and the score in red represent a high score based on the relatively healthy population. Additionally, the previous value is also indicated to help check for improvements every time the test is ordered. Following this section is the complete list of the damage markers results and their absolute levels are normalized with respect to Creatinine in a histogram format to enable a full overview along with the reference ranges. The level of the analyte with reference range is shown with three shades of color – Green, Yellow and Red. The result in green corresponds to 0th to 75th percentile indicates mild detection of the analyte. The result in yellow corresponds to 75th to 95th percentile indicates moderate detection of the analyte whereas the result in red corresponding to greater than 95th percentile indicates high detection of the analyte. The reference metric is listed to the right of the reference range. The previous and current result are listed to the left of the reference range. (result example illustration below)

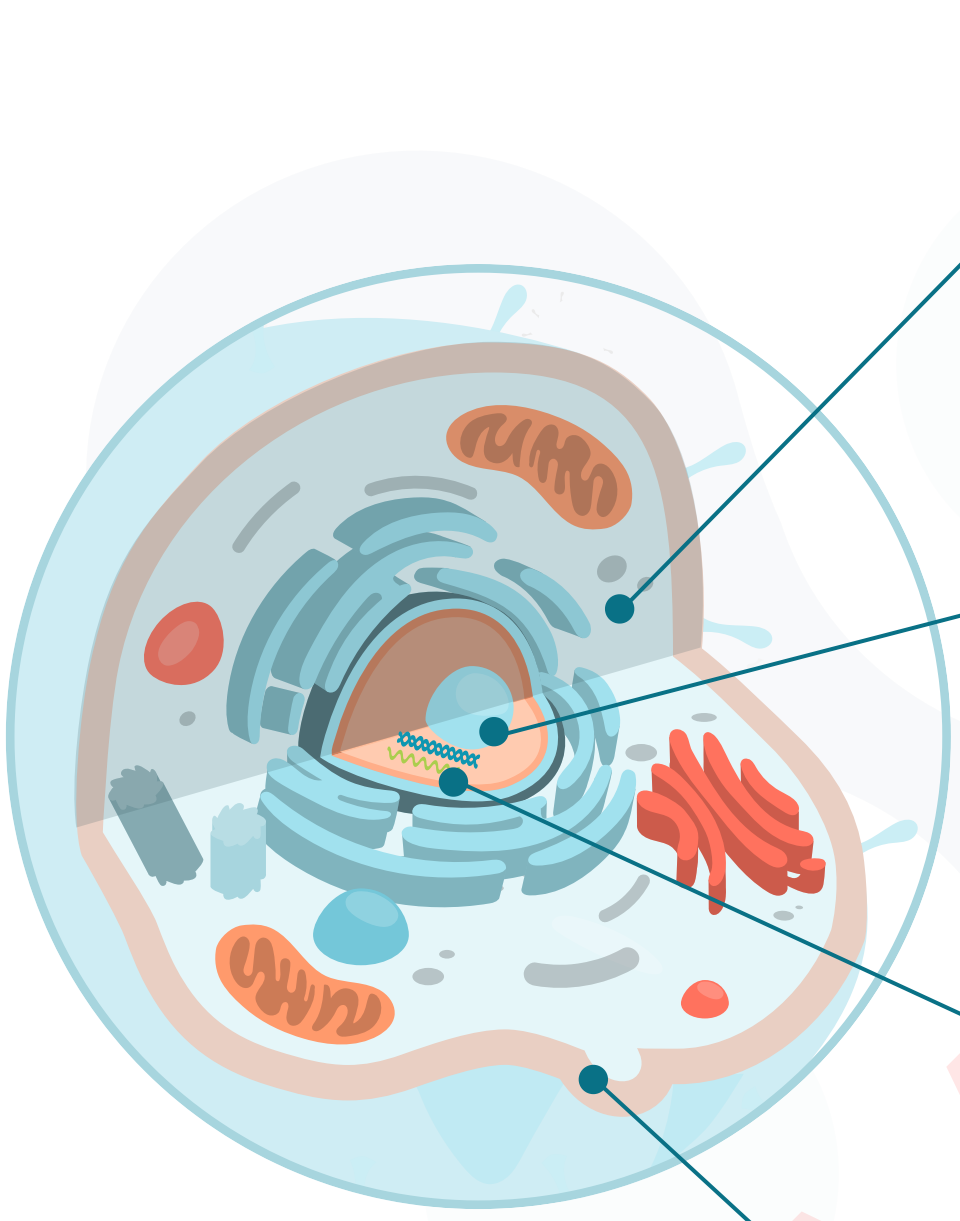


Please note: It is important that you discuss any modifications to your diet, exercise, and nutritional supplementation with your healthcare provider before making any changes.

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Oxidative Stress - Summary

Oxidative Stress Profile Summary



Protein Oxidation

- 3-Bromotyrosine
- Dityrosine
- Nε-carboxyethyllysine (CEL)

DNA Damage

- 8-Hydroxy-2 - deoxyguanosine
- 8-Hydroxyguanosine

RNA Damage

No markers out of range

Lipid Peroxidation

- 11-β-Prostaglandin F2α
- 15(R)-Prostaglandin F2α
- 8-iso-prostaglandin F2α (8-iso-PGF2α)
- Glutathione 4-hydroxynonenal (GS-HNE)
- Malondialdehyde

Oxidative Stress Biomarkers

Lipid Peroxidation	Current	Previous	Result (75th / 95th)	Reference
15(R)-Prostaglandin F2α (ug/g)	0.31		0.07 / 0.22	≤0.22

Lipid peroxidation is a degenerative process wherein free radicals attack and break down lipids under oxidative stress. The process affects cell membranes, lipoproteins, and other lipid-containing structures. 15(R)-Prostaglandin F2α (15-F2t-IsoP) is an isoprostane generated through the non-enzymatic peroxidation of arachidonic acid in membrane phospholipids. This biomarker serves as an indicator of oxidative stress and can reliably reflect lipid peroxidation in chronic diseases. Elevated levels of 15-F2t-IsoP can lead to DNA oxidation and subsequent structural DNA damage. 15-F2t-IsoP is thereby, valuable in assessing oxidative damage to DNA and understanding its implications for cellular health and disease development. Studies have shown that increased levels of 15-F2t-IsoP contribute to heightened oxidative stress associated with aging, hypertension, diabetes mellitus, hypercholesterolemia, smoking, and coronary artery disease.

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Oxidative Stress - Summary

Oxidative Stress Biomarkers

Lipid Peroxidation	Current	Previous	Result (75th / 95th)	Reference
8-iso-prostaglandin F2α (8-iso-PGF2α) (ug/g)	0.48		0.1 / 0.26	≤0.26

8-iso-prostaglandin F2α (8-iso-PGF2α) is an isoprostane generated through the non-enzymatic peroxidation of arachidonic acid in membrane phospholipids. It is found in human plasma and excreted in urine. This biomarker serves as an indicator of oxidative stress and can reliably reflect lipid peroxidation in chronic diseases. Elevated levels of 8-iso PGF2α can lead to DNA oxidation and subsequent structural DNA damage. 8-iso PGF2α is thereby, valuable in assessing oxidative damage to DNA and understanding its implications for cellular health and disease development. Studies have shown that increased levels of 8-iso PGF2α contribute to heightened oxidative stress associated with aging, hypertension, diabetes mellitus, hypercholesterolemia, smoking, and coronary artery disease.

Glutathione 4-hydroxynonenal (GS-HNE) (ug/g)	2.70		0.3 / 2.5	≤2.5

Glutathione-conjugated 4-HNE (GS-HNE) serves as a marker of oxidative stress due to its formation as a consequence of lipid peroxidation, a prominent outcome of oxidative stress. 4-HNE, a toxic and stable end product of lipid peroxidation, is produced when cells are exposed to oxidative stressors. In response to elevated levels of oxidative stress, cells mount a defense mechanism to counteract the damaging effects of 4-HNE. One of the predominant mechanisms involves the conjugation of 4-HNE to glutathione (GSH) catalyzed by enzymes known as glutathione S-transferases (GSTs). This reaction results in the formation of GS-HNE. The presence of GS-HNE in cells signifies not only the occurrence of oxidative stress but also the cell's efforts to neutralize the harmful effects of 4-HNE. As such, the measurement of GS-HNE levels can provide valuable insights into the extent of oxidative stress and the cellular response to it, making it a useful biomarker for assessing oxidative stress-related conditions and their potential impact on cellular health.

Malondialdehyde (ug/g)	168.40		72.87 / 163.53	≤163.53

Malondialdehyde (MDA) is generated from the lipid peroxidation process (free radicals attack and breakdown lipids) by the peroxidation of membrane polyunsaturated fatty acids, including omega-3 (n-3) and omega-6 (n-6) fatty acids. Reactive oxygen species can cause the peroxidative breakdown of phospholipids that lead to the accumulation of MDA, thus, making MDA a biomarker of oxidative stress. MDA is also produced in the process of prostaglandin synthesis. Elevated MDA levels are seen to be associated with conditions like diabetes, sepsis, and coronary heart disease. MDA is also involved in the pathogenesis of conditions such as Alzheimer's, Parkinson's disease, cancer, diabetes, cardiovascular and liver diseases.

11-β-Prostaglandin F2α (ug/g)	0.29		0.11 / 0.4	≤0.4

Lipid peroxidation is a degenerative process wherein free radicals attack and break down lipids under oxidative stress. The process affects cell membranes, lipoproteins, and other lipid-containing structures. The non-enzymatic oxidation of arachidonic acid leads to the production of 11-β-prostaglandin F2α (11-PGF2α). It is irreversibly produced from prostaglandin D2 via the enzyme prostaglandin-F synthase. Thus, elevated levels of 11-PGF2α are indicative of the increased oxidation of arachidonic acid by free radicals. Its increased levels can lead to inflammation. It may contribute to cardiac diseases owing to their involvement in vasoconstriction and cardiomyocyte hypertrophy (thickening of heart muscles). 11-PGF2α is also regarded as a marker of oxidative stress linked to inflammation.

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Oxidative Stress - Summary

Oxidative Stress Biomarkers

DNA Damage	Current	Previous	Result (75th / 95th)	Reference
8-Hydroxy-2 -deoxyguanosine (ug/g)	11.60		1.14 / 4	≤4

Reactive oxygen species produced either endogenously or exogenously can attack nucleic acid in living cells. The guanine molecule which is one of the four main nucleobases found in the nucleic acids in DNA, gets oxidized to produce the modified 8-hydroxy-2'-deoxyguanosine (8-OHdG) which acts as one of the predominant forms of free radical-induced lesions of DNA. Thus, 8-OHdG can indicate the effect of oxidative damage in DNA. The marker is also used to estimate the risk for atherosclerosis, diabetes, and various cancers. Additionally, 8-OHdG is helps estimate the damage in the DNA after exposure to cancer-causing agents, such as tobacco smoke, asbestos fibers, heavy metals, and polycyclic aromatic hydrocarbons.

8-Hydroxyguanosine (ug/g)	97.89		44.9 / 95.3	≤95.3

Free radicals produced either endogenously or exogenously can attack nucleic acid in living cells. Reactions of reactive oxygen species (ROS) and reactive nitrative species (RNS) with RNA yield 8-hydroxyguanosine (8-HdG). Among the known oxidative lesions in nucleic acids, 8-HdG is abundant and appears to be most deleterious due to its high mutagenic potential. This implies that 8-HdG is capable of inducing genetic mutation. RNA dysfunction caused by oxidative damage may contribute to the development of various degenerative diseases. Urinary levels of 8-HdG have risen as indicators of oxidative damage of RNA by ROS.

Protein Oxidation Products	Current	Previous	Result (75th / 95th)	Reference
3-Bromotyrosine (ug/g)	362.78		167.53 / 349.6	≤349.6

3-Bromotyrosine (Br-Tyr) is a compound generated by the halogenation of tyrosine residues in plasma or tissue proteins. Br-Tyr synthesis is primarily catalyzed by the enzyme eosinophil peroxidase (EPO). Eosinophils, specialized white blood cells, play a central role in immune responses. When eosinophils become activated in response to various immune challenges, they release enzymes like eosinophil peroxidase (EPO). EPO is an enzyme that can generate reactive oxygen species (ROS) as part of its normal enzymatic activities during this process a subsequent production of Br-Tyr as a stable by-product occurs. Elevated levels of Br-Tyr in biological samples serve as a crucial marker of oxidative stress, particularly associated with eosinophil-driven oxidative processes. This unique characteristic positions Br-Tyr as a valuable indicator, shedding light on the extent of oxidative stress induced by eosinophil activation and its potential implications in various physiological and pathological conditions.

Dityrosine (ug/g)	2.20		1.31 / 5	≤5

Post-translational modification (PTM) is a normal protein modification process occurring in cells. Many PTMs are dependent on environmental factors. Changes in the structure of proteins can occur when a cell is exposed to oxidative/nitrative stress. One such PTM is the change in protein–protein cross-linkage between two tyrosine molecules leading to the formation of dityrosine. This can be mediated by the oxidative species, hydroxyl radical and nitrative species, peroxynitrite and nitrosoperoxycarbonate leading to increased hydroxylation and nitration tyrosine. Thus, dityrosine is produced due to oxidative/nitrative stress under a variety of biological conditions. It may participate in lipid peroxidation which includes attacking and breaking down fats. Elevated dityrosine levels have been linked with pathologies such as eye cataracts, atherosclerosis, acute inflammation, and Alzheimer's disease. Thus, dityrosine has risen as an important biomarker for oxidative stress, especially for oxidatively modified proteins during UV and gamma-irradiation, aging, and exposure to oxygen free radicals, nitrogen dioxide, peroxynitrite, and lipid hydroperoxides.

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Patient Name: DEMO DEMO
Date of Birth: 00-00-0000 Accession ID: 2407077216
Service Date: Local 2026-07-19 11:30

Oxidative Stress - Summary

Oxidative Stress Biomarkers

Advanced Glycation Products	Current	Previous	Result (75th: 19.5, 95th: 91)	Reference
Nε-carboxyethyllysine (CEL) (ug/g)	93.45			≤91

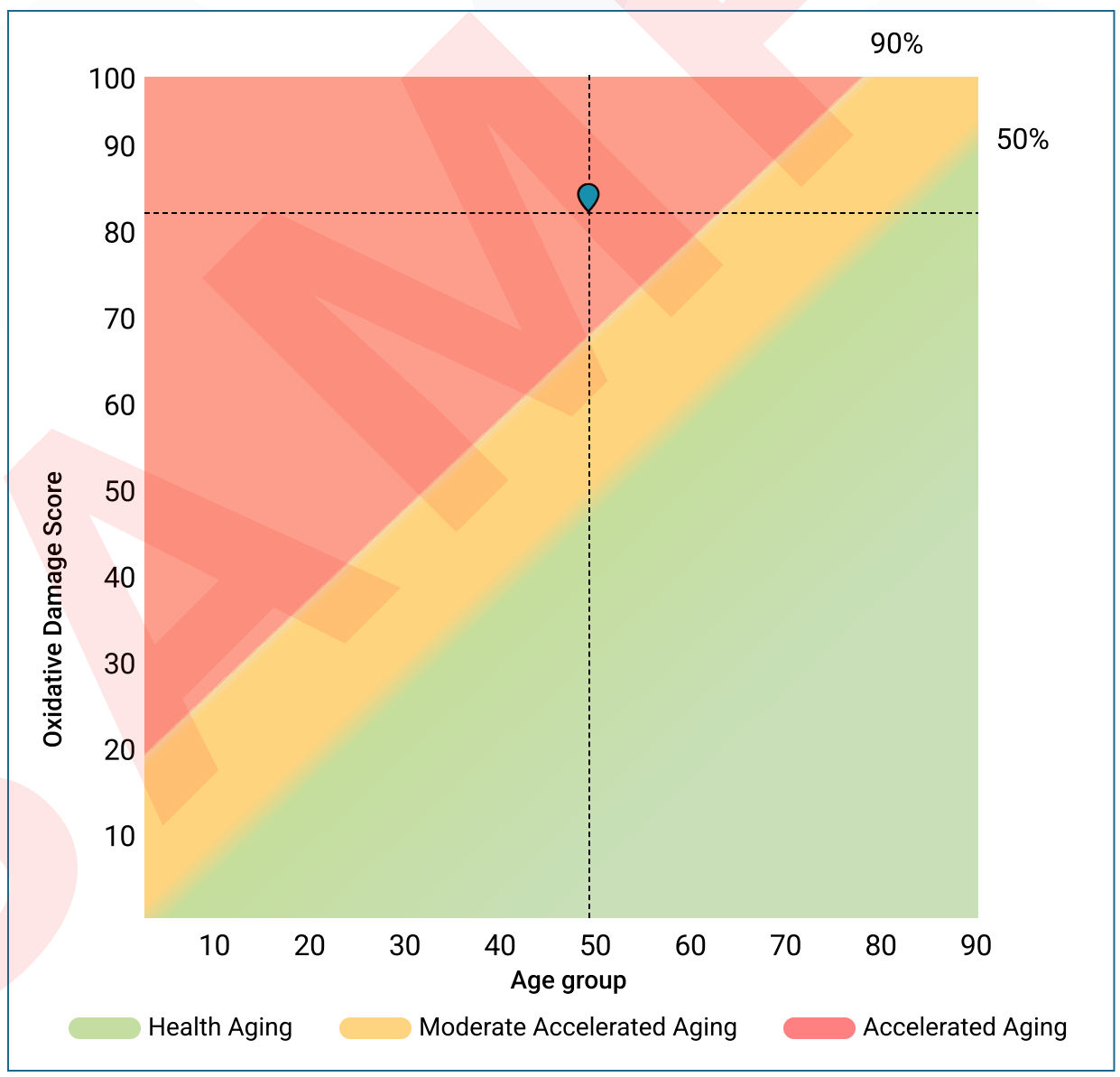
Glycation is a spontaneous non-enzymatic reaction wherein free reducing sugars bind to free amino groups of proteins, DNA, and lipids. This results in the formation of advanced glycation end-products (AGE). Glycation and oxidative stress are closely linked, and they are together referred to as "glycoxidation". All steps of glycoxidation generate free radicals, some of them being common with the lipid peroxidation pathway. Owing to this, AGE has been considered a urinary biomarker of oxidative stress. The AGE product, Nε -carboxyethyllysine (CEL) is formed when methylglyoxal (formed from the oxidation of lipids and sugars) reacts with lysine. CEL interacts with AGE receptors (RAGEs) which may give rise to oxidative stress. This may even induce cellular dysfunction. Urinary levels of CEL can be used to monitor the degree of oxidative stress in the body system.

Creatinine

Test Name	Current	Previous	Result (0, 0.24, 2.16)	Reference
Urine Creatinine (mg/ml)	0.88			0.25-2.16

Oxidative Stress Score

Current Result Previous Result



Result

Your Given Age: 48

Your Oxidative Stress Profile looks similar to a ***83.66*** year old.

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Oxidative Stress - Summary

Suggestions

Nutrients	Dosage	Purpose
Vitamin C	90 mg/day	Vitamin C, as an antioxidant, reduces MDA (malondialdehyde) levels by scavenging reactive oxygen species (ROS) and preventing lipid peroxidation, thus limiting the formation of MDA. Vitamin C supplementation decreases 8-iso-prostaglandin F2α (8-iso-PGF2α) levels by acting as a powerful antioxidant, scavenging free radicals and inhibiting lipid peroxidation, thereby reducing oxidative stress. Vitamin C decreases 8-Hydroxy-2'-deoxyguanosine (8-OHdG) by donating electrons to neutralize reactive oxygen species, reducing oxidative stress and DNA damage. It also regenerates other antioxidants like glutathione, enhancing their capacity to repair and prevent DNA oxidation.
Omega-3 fatty acids	950 mg/day	Omega-3 fatty acid (n-3 FA) supplementation decreases 8-iso-prostaglandin F2α (8-iso-PGF2α) levels by reducing inflammation and oxidative stress, inhibiting lipid peroxidation and promoting overall cardiovascular health. Omega-3 fatty acids reduce 11-β-Prostaglandin F2α levels by inhibiting the activity of cyclooxygenase (COX) enzymes, which are responsible for the conversion of arachidonic acid into prostaglandins, including 11-β-Prostaglandin F2α. Studies show Omega-3 fatty acids reduce Prostaglandin F2α levels by inhibiting the activity of cyclooxygenase (COX) enzymes, which are responsible for the conversion of arachidonic acid into prostaglandins, including Prostaglandin F2α.
Vitamin E	22 IU/day	Vitamin E, a potent antioxidant, reduces MDA (Malondialdehyde) levels by neutralizing free radicals that would otherwise lead to lipid peroxidation, preventing the formation of MDA as a byproduct. Vitamin E supplements reduce 8-iso-prostaglandin F2α (8-iso-PGF2α) levels by acting as an antioxidant, neutralizing reactive oxygen species that would otherwise promote the formation of 8-iso-PGF2α.
Beta-carotene	6 -15 mg/day	Beta-carotene supplements reduce MDA levels by acting as an antioxidant, scavenging free radicals and preventing lipid peroxidation, which reduces the formation of MDA in cells. Beta-carotene supplements act as antioxidants, scavenging free radicals and reducing oxidative stress, thereby lowering 8-iso-prostaglandin F2α (8-iso-PGF2α) levels, a marker of oxidative damage.
Selenium	55 mcg/day	Selenium supplements reduce MDA levels by enhancing the activity of antioxidant enzymes, particularly glutathione peroxidase, which helps neutralize free radicals and prevent lipid peroxidation. Selenium reduces 8-iso-prostaglandin F2α (8-iso-PGF2α) levels by acting as a cofactor for the enzyme glutathione peroxidase, which helps neutralize reactive oxygen species responsible for the formation of 8-iso-PGF2α.

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Oxidative Stress - Summary

Suggestions

Nutrients	Dosage	Purpose
Curcumin	250 mg/day	Curcumin, a potent antioxidant, reduces MDA levels by scavenging free radicals and inhibiting lipid peroxidation, thereby decreasing the formation of MDA as a byproduct of oxidative stress. Curcumin supplements decrease 8-hydroxy-2'-deoxyguanosine by inhibiting oxidative stress-induced DNA damage, as curcumin's antioxidant properties neutralize free radicals and enhance the activity of DNA repair enzymes, reducing the formation of 8-hydroxy-2'-deoxyguanosine.

SAMPLE

Patient Name: DEMO DEMO
Date of Birth: 00-00-0000 Accession ID: 2407077216
Service Date: Local 2026-07-19 11:30

Oxidative Stress - All Markers

Oxidative Stress Biomarkers

Lipid Peroxidation	Current	Previous	Result (75th – 95th)	Reference
11-β-Prostaglandin F2α (ug/g)	0.29		0.11 – 0.4	≤0.4
15(R)-Prostaglandin F2α (ug/g)	0.31		0.07 – 0.22	≤0.22
8-iso-prostaglandin F2α (8-iso-PGF2α) (ug/g)	0.48		0.1 – 0.26	≤0.26
Glutathione 4-hydroxynonenal (GS-HNE) (ug/g)	2.70		0.3 – 2.5	≤2.5
Malondialdehyde (ug/g)	168.40		72.87 – 163.53	≤163.53

DNA Damage	Current	Previous	Result (75th – 95th)	Reference
8-Hydroxy-2 -deoxyguanosine (ug/g)	11.60		1.14 – 4	≤4
8-Hydroxyguanine (ug/g)	5.10		16 – 49.4	≤49.4
8-Hydroxyguanosine (ug/g)	97.89		44.9 – 95.3	≤95.3

RNA Damage	Current	Previous	Result (75th – 95th)	Reference
8-Nitroguanine (ug/g)	2.10		33.45 – 107.47	≤107.47
8-Nitroguanosine (ug/g)	2.40		778.1 – 2608.9	≤2608.9

Protein Oxidation Products	Current	Previous	Result (75th – 95th)	Reference
3-Bromotyrosine (ug/g)	362.78		167.53 – 349.6	≤349.6
3-Chlorotyrosine (ug/g)	<1.6		3.43 – 9.92	≤9.92
Dityrosine (ug/g)	2.20		1.31 – 5	≤5
Nitrotyrosine (ug/g)	<4.55		91.32 – 285.69	≤285.69

Advanced Glycation Products	Current	Previous	Result (75th – 95th)	Reference
Nε-(carboxymethyl)lysine (CML) (ug/g)	12.60		15.87 – 70.3	≤70.3
Nε-carboxyethyllysine (CEL) (ug/g)	93.45		19.5 – 91	≤91

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Organic Acids

INTRODUCTION

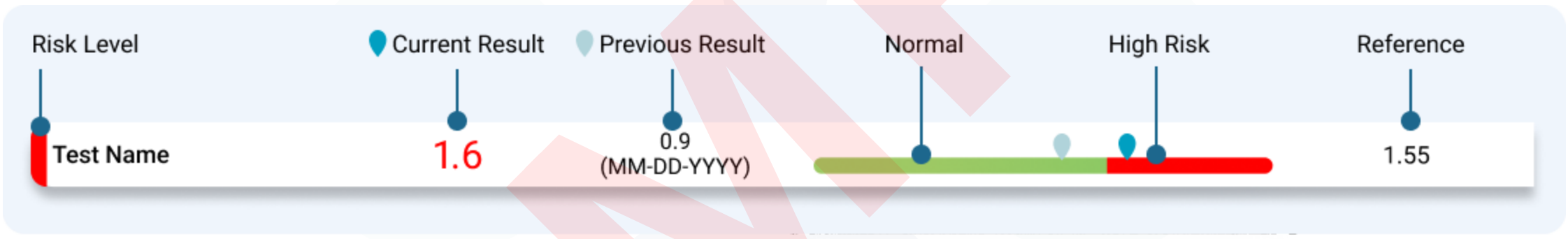
Vibrant Wellness is pleased to present the Organic Acids Panel to support healthy lifestyle choices in consultation with your healthcare provider. The Organic Acids Panel provides quantitative assessment of amino acid metabolites, detoxification and oxidative stress markers, mitochondrial function indicators, microbial metabolites, neurotransmitter and stress hormone metabolism, and nutritional status markers. Results are intended to be interpreted by healthcare providers to guide personalized wellness strategies informed by insights into metabolic function, biochemical balance, and cellular health.

Methodology

The Organic Acids panel uses gas chromatography tandem mass spectrometry (GC-MS/MS) for quantitative detection of organic acids in urine samples. Additionally, catecholamine metabolites and serotonin & kynurenine metabolites are measured using liquid chromatography tandem mass spectrometry methodology (LC-MS/MS). Urine creatinine is measured using a kinetic colorimetric assay based on the Jaffé method. All organic acids markers are reported as the quantitative result normalized to urine creatinine to account for urine dilution variations.

Interpretation of Report

The report begins with the summary page which lists only the organic acids whose levels are outside the normal reference range. Reference ranges have been established using a cohort of 1000 apparently healthy adults over 18 years of age, and pediatric reference ranges are not available . Following this section is a graphical representation of the AXON terminal and a summary of Krebs cycle including the results for the relevant analytes. This is followed by a complete list of the organic acids which are represented normalized to urine creatinine, in a bar graph form to enable a full overview along with the reference ranges. The level of the organic acid has a green (normal) or red (high/low) highlight around the cell indicating the corresponding result based on the reference range of each organic acid. Additionally, the previous value (if available) is also indicated to help check for improvements every time the test is ordered. The reference metric is listed to the right of the reference range. The previous and current result are listed to the left of the reference range. (result example illustration below)



Please note: It is important that you discuss any modifications to your diet, exercise, and nutritional supplementation with your healthcare provider before making any changes.

Regulatory Disclaimer: This test was performed by Vibrant America Clinical Laboratory at 3521 Leonard Ct, Santa Clara, CA 95054 (CLIA No. 05D2078809, CAP No. 8970308). This test was developed, and its performance characteristics determined, by Vibrant America Clinical Laboratory. This test has not been cleared or approved by the U.S. Food and Drug Administration (FDA).

Organic Acids - Summary

Amino Acid Metabolites

Amino Acid Metabolites	Current	Previous	Result	Reference
2-Oxoisovaleric Acid (mmol/mol)	3.40		0 — 2	≤2.0

2-oxoisovaleric acid is a product of valine catabolism. It is also called It is used as a marker for maple syrup urine disease, which is a condition that involves a mutation in the branched chain a-keto acid dehydrogenase complex (BCKDH), that's responsible for breaking down all branched chain amino acids. This enzyme requires Vitamin B1, Vitamin B2 (FAD), Vitamin B3 (NAD), Vitamin B5 (Coenzyme A) and Lipoic acid as nutrient cofactors. Elevated levels of stress can increase breakdown of muscle/proteins and result in higher levels of BCAA metabolites. 2-oxoisovaleric acid can then undergo a series of metabolic reactions to form succinyl CoA, which can enter the kreb's cycle and affect energy metabolism[ii]. Considerations may include sufficient intake of nutrient cofactors, including vitamin B1, B2, B3, B5 and alpha lipoic acid and limit intake of high amounts of branched chain amino acid in the diet or via supplementation.

3-Methyl-2-oxovaleric acid (mmol/mol)	3.10		0 — 2.6	≤2.6

3-Methyl-2-oxovaleric acid is a metabolite produced in the muscles, liver, and nervous system during the metabolism of isoleucine, an essential amino acid. It is a crucial marker of how efficiently the body processes isoleucine for muscle protein synthesis, energy production, hemoglobin formation, blood sugar regulation, and immune function. Elevated levels of 3-Methyl-2-oxovaleric acid suggest impairments in isoleucine metabolism, indicating maple syrup urine disease, thiamine deficiency, biotin deficiency, vitamin B6 deficiency, mitochondrial dysfunction, or oxidative stress. Measuring this metabolite helps assess isoleucine metabolism, monitor for metabolic disorders like MSUD, evaluate B-vitamin and biotin status, and guide decisions on nutritional supplementation, providing mitochondrial support, antioxidant therapy, and moderate adjustment of dietary protein.

4-Hydroxyphenyllactic acid (mmol/mol)	2.10		0 — 0.84	≤0.84

4-Hydroxyphenyllactic acid is a metabolite produced primarily in the liver during the incomplete metabolism of tyrosine, an amino acid important for neurotransmitter production. It is a crucial marker of how efficiently the body processes tyrosine for dopamine, norepinephrine, and thyroid hormone synthesis, vitamin C status, and antioxidant status. Elevated levels of 4-Hydroxyphenyllactic acid suggest impairments in tyrosine metabolism, indicating vitamin C deficiency, oxidative stress, liver dysfunction, alkaptonuria, or rarely, hereditary tyrosinemia. Measuring this metabolite helps assess tyrosine metabolism efficiency, detect vitamin C deficiency, evaluate antioxidant status and liver function, and guide decisions on nutritional supplementation, providing antioxidant support and liver support nutrients, and potentially moderating dietary tyrosine and phenylalanine intake.

Phenyllactic acid (mmol/mol)	0.48		0 — 0.21	≤0.21

Phenyllactic acid is a metabolite produced primarily in the liver and other tissues during metabolism of phenylalanine, an essential amino acid. It is a crucial marker of how efficiently the body processes phenylalanine for tyrosine synthesis, neurotransmitter production, and protein formation. Elevated levels of phenyllactic acid suggest impairments in phenylalanine metabolism, commonly associated with phenylketonuria (PKU) or liver disease. Measuring this metabolite helps diagnose PKU and other phenylalanine metabolism disorders early, monitor treatment effectiveness in diagnosed patients, assess liver function, and guide decisions on strict dietary restriction of phenylalanine, nutritional supplementation, and regular monitoring of phenylalanine levels.

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Organic Acids - Summary

Detoxification & Oxidative Stress

Glutathione	Current	Previous	Result	Reference
Pyroglutamic acid (mmol/mol)	41.00		0 10.13 32.45	10.14-32.45

Pyroglutamic acid (5-oxoproline) is produced during glutathione metabolism and reflects the body's glutathione synthesis and detoxification capacity. Elevated levels indicate impaired glutathione recycling, often due to genetic defects in the gamma-glutamyl cycle, oxidative stress, acetaminophen toxicity, or nutritional deficiencies. Measuring this metabolite helps assess glutathione status, identify rare metabolic disorders, evaluate detoxification capacity, monitor chronic oxidative stress, and guide decisions on supporting antioxidant pathways, supplementing glutathione precursors, addressing medication-induced toxicity, and optimizing cellular redox balance.

Oxidative Stress	Current	Previous	Result	Reference
2-Hydroxybutyric acid (mmol/mol)	2.60		0 0.05 1.58	0.06-1.58

2-Hydroxybutyric acid is produced in the liver during the metabolism of methionine, threonine, and glutathione, serving as an early marker of insulin resistance and oxidative stress. Elevated levels suggest impaired glucose metabolism, increased oxidative stress, hepatic glutathione depletion, or early metabolic syndrome. It can also indicate alpha-ketoacid dehydrogenase dysfunction or disturbances in amino acid metabolism. Measuring this metabolite helps assess metabolic health, detect early insulin resistance before diabetes develops, evaluate oxidative burden, and guide decisions on implementing dietary interventions for blood sugar control, providing antioxidant support, addressing glutathione status, and monitoring cardiometabolic risk factors.

Metabolism & Mitochondrial Function

Fat & Ketones	Current	Previous	Result	Reference
Adipic acid (mmol/mol)	5.60		0 0.03 3.9	0.04-3.9

Adipic acid is a metabolite produced in the liver and other tissues during the breakdown of medium-chain fatty acids and certain amino acids. It is a crucial marker of how efficiently the body oxidizes medium-chain fatty acids for energy and processes specific amino acids through their degradation pathways. Elevated levels of adipic acid suggest impaired fatty acid oxidation, indicating MCAD deficiency (a genetic disorder affecting fat breakdown), other fatty acid oxidation disorders, carnitine deficiency, vitamin B2 deficiency, mitochondrial dysfunction, or periods of prolonged fasting or metabolic stress. Measuring this metabolite helps detect fatty acid oxidation disorders (particularly MCAD deficiency), assess mitochondrial function and energy production capacity, evaluate carnitine and riboflavin status, monitor metabolic stress and fasting tolerance, and guide decisions on considering nutritional supplements.

Ethylmalonic acid (mmol/mol)	3.60		0 0.46 2.74	0.47-2.74

Ethylmalonic acid is a metabolite produced in various tissues during the metabolism of short-chain fatty acids such as butyric acid from gut bacteria and through certain amino acid breakdown pathways. It is a crucial marker of mitochondrial fatty acid oxidation efficiency, neurotoxicity, short-chain acyl-CoA dehydrogenase (SCAD) deficiency, and ethyl malonic encephalopathy. Elevated levels of ethylmalonic acid suggest impaired short-chain fatty acid metabolism. Measuring this metabolite helps detect fatty acid oxidation disorders and mitochondrial dysfunction, assess the impact of gut microbiome activity on metabolism, and guide decisions on considering nutritional supplementation.

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Organic Acids - Summary

Metabolism & Mitochondrial Function

Fat & Ketones

	Current	Previous	Result	Reference
Suberic acid (mmol/mol)	3.40		0 0.15 2.18	0.16-2.18

Suberic acid is a metabolite produced in the liver and peroxisomes during the breakdown of medium-chain fatty acids through omega-oxidation, an alternative pathway that becomes important when normal mitochondrial fatty acid oxidation is compromised. It is a crucial marker of the body's adaptive response to impaired primary fat oxidation processes. Elevated levels of suberic acid suggest increased reliance on omega-oxidation pathways. Measuring this metabolite helps identify fatty acid oxidation disorders and peroxisomal dysfunction, assess the functional status of alternative metabolic pathways, evaluate carnitine and vitamin B2 adequacy, monitor metabolic stress and adaptation during energy demands, and guide decisions on maintaining optimal nutritional health.

Glycolysis

	Current	Previous	Result	Reference
Lactic acid (mmol/mol)	64.00		0 50.4	≤50.4

Lactic acid is a metabolite produced primarily in muscles, red blood cells, and other tissues during anaerobic metabolism when cells break down glucose for energy without sufficient oxygen. It is a crucial marker of cellular oxygen availability, anaerobic energy production, mitochondrial function, tissue perfusion, and the balance between aerobic and anaerobic metabolism. Elevated levels of lactic acid suggest increased anaerobic metabolism or impaired lactate clearance. Measuring this metabolite helps assess tissue oxygenation and perfusion status, detect mitochondrial dysfunction and metabolic disorders, evaluate the severity of metabolic stress or critical illness, monitor exercise intensity and recovery capacity, and guide decisions on nutritional supplementation.

Kreb's Cycle

	Current	Previous	Result	Reference
Cis-aconitic acid (mmol/mol)	29.92		0 6.09 27.9	6.1-27.9

Cis-aconitic acid is a metabolite produced in the mitochondria as an intermediate in the Krebs cycle, formed during the conversion of citric acid to isocitric acid by the enzyme aconitase. It is a crucial marker of Krebs cycle function. Elevated levels of cis-aconitic acid suggest impaired aconitase enzyme function or Krebs cycle disruption, indicating iron deficiency, oxidative stress, or genetic disorders affecting Kreb's cycle enzymes. Measuring this metabolite helps detect iron deficiency and its impact on mitochondrial metabolism, identify potential environmental toxin exposure, and guide decisions on maintaining nutritional health.

Mitochondrial Function

	Current	Previous	Result	Reference
3-Hydroxyglutaric acid (mmol/mol)	7.90		0 4.9	≤4.9

3-Hydroxyglutaric acid (3HGA) is an organic acid that’s elevated in the neurometabolic disorder involving a glutaryl-CoA dehydrogenase deficiency, leading to type 1 glutaric aciduria. This is an autosomal recessive defect in the metabolic pathway of lysine, hydroxylysine, and tryptophan. Clinical presentations early in life for this condition include macrocephalus, mild hypotonia, jittery, irritability and feeding difficulties with progression to more significant neurological symptoms over time. 3HGA has been shown to provoke a moderate impairment in brain energy metabolism[xvi]. It can also induce excitotoxic cellular injury by activating N-methyl-d-aspartate receptors. Intervention consideration may include a low-protein diet to restrict glucogenic amino acids (lysine, tryptophan, and hydroxylysine). Other considerations may include carnitine supplementation and aggressive treatment during acute episodes of intercurrent illnesses[xvii].

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Organic Acids - Summary

Metabolism & Mitochondrial Function

Mitochondrial Function	Current	Previous	Result	Reference
3-Methylglutaconic (mmol/mol)	8.60		0 — 6.2	≤6.2

3-Methylglutaconic acid is a metabolite produced during the metabolism of leucine and is also associated with mitochondrial dysfunction affecting various metabolic pathways. It is a crucial marker of mitochondrial membrane integrity, energy production and a sensitive indicator of mitochondrial disorders. Elevated levels of 3-methylglutaconic acid suggest aciduria, cardiolipin synthesis defects, and some enzyme deficiencies. Measuring this metabolite helps diagnosis of primary mitochondrial disorders and specific genetic syndromes, monitor mitochondrial health in complex neurological and cardiac conditions, and guide decisions on dietary modifications while sustaining nutritional health.

Microbial Metabolites

Bacterial Metabolites	Current	Previous	Result	Reference
Dihydroxyphenylpropionic acid(DHPPA) (mmol/mol)	0.72		0 — 0.44	≤0.44

Dihydroxyphenylpropionic acid (DHPPA) is a gut microbiota–derived organic acid formed from the bacterial metabolism of dietary polyphenols and aromatic amino acids, particularly through clostridial species. It serves as a urinary marker of intestinal microbial activity and is commonly associated with dysbiosis-related metabolic patterns. Under physiological conditions, it reflects normal fermentation of dietary flavonoids and quinolone compounds by colonic bacteria. Elevated levels may indicate Clostridium overgrowth or increased microbial conversion of polyphenol substrates, suggesting intestinal dysbiosis.

Hippuric acid (mmol/mol)	720.00		0 — 607	≤607.0

Hippuric acid is a glycine-conjugated metabolite formed in the liver from benzoic acid, which originates largely from gut microbial metabolism of dietary polyphenols and phenylalanine-derived compounds. It serves as a key host–microbiome co-metabolite and is widely used as a urinary marker of gut microbial activity, dietary polyphenol intake, and hepatic phase II glycine conjugation capacity. Elevated levels may reflect increased microbial fermentation of aromatic substrates, higher dietary intake of fruits and plant phenolics, occupational exposure to compounds such as toluene, or increased benzoate burden requiring detoxification. Conversely, reduced hippuric acid levels have been associated with gut microbiota dysbiosis, obesity, hypertension, and type 2 diabetes, suggesting diminished microbial diversity or impaired aromatic metabolism.

Clostridia Metabolites	Current	Previous	Result	Reference
4-Hydroxyphenylacetic acid (mmol/mol)	22.00		0 — 20.1	≤20.1

4-Hydroxyphenylacetic acid is a gut microbiota–derived metabolite produced from bacterial metabolism of tyrosine and represents a measurable fraction of dietary tyrosine intake. It is absorbed from the intestine, further processed by the host, and excreted in urine, reflecting intestinal microbial activity and host–microbe metabolic interaction. Elevated urinary levels may indicate increased anaerobic bacterial fermentation or small intestinal bacterial overgrowth, and are considered a marker of gut microbiota dysbiosis. Persistent elevation can reflect excessive aromatic amino acid fermentation and altered intestinal microbial balance.

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Organic Acids - Summary

Neurotransmitters & Stress Hormones

Catecholamine Metabolites & Ratios	Current	Previous	Result	Reference
Dihydroxyphenylacetic acid (DOPAC) (mcg/g)	1.10		0 577.29 1655	577.3-1655.5

3,4-Dihydroxyphenylacetic acid (DOPAC) is a catechol-derived organic acid and major urinary metabolite of the neurotransmitter dopamine, formed via monoamine oxidase (MAO) oxidation of 3,4-dihydroxyphenylacetaldehyde in the brain, peripheral tissues, and gut. Urinary DOPAC reflects dopamine turnover and catecholamine metabolism, with levels influenced by neural activity, stress, diet, or microbial interactions. High DOPAC suggests increased dopamine turnover from stress, anxiety, ADHD, or gut dysbiosis (e.g., bacterial decarboxylation of tyrosine), potentially linking to oxidative stress via quinone formation or mitochondrial interference; paired with low HVA/DOPAC ratios, it may signal poor dopamine conversion to norepinephrine. Reduced excretion points to dopamine deficiency, possibly from impaired synthesis, Parkinson's-like states, chronic fatigue, or COMT/MAO inhibition; clinically, it correlates with low motivation, depression, or poor reward signaling, warranting nutritional support.

Homovanillic acid (HVA) (mcg/g)	4.10		0 3534 8455	3535.0-8455.0

Homovanillic acid (HVA) is the major downstream metabolite of dopamine, formed when dopamine is first broken down by MAO and then further processed by COMT. Elevated HVA reflects increased activity or presence of dopamine-modulating gut commensals such as Clostridium species, which enhance dopamine production and accelerate its metabolism. Excessive dopamine turnover can contribute to stress-driven behaviors. Clinically, high HVA is associated with anxiety, behavioral instability, and dysregulated gut–brain communication due to excessive dopaminergic activity. Mild elevations in HVA levels may result from certain dopaminergic medications, such as NRDIs and dopamine agonists, as well as from dietary factors, including bananas, oranges, tangerines, avocado, tomatoes, plums, prunes, pineapple, chocolate, and walnuts. Supplements that may contribute to mild elevations of HVA include tyrosine, phenylalanine, bitter orange, yohimbe, and Mucuna pruriens. In rare cases, significant elevations may warrant further evaluation for disorders of catecholamine metabolism, including dopamine-producing neuroendocrine tumors.

Vanillylmandelic acid (VMA) (mcg/g)	3.20		0 2411 5047	2411.2-5047.8

Vanillylmandelic acid (VMA) is the main breakdown product of the catecholamines norepinephrine and epinephrine, formed through metabolism by monoamine oxidase (MAO) and catechol-O-methyltransferase (COMT). High VMA levels generally indicate increased catecholamine production and breakdown, which may occur when Clostridium species are more abundant or more active, enhancing microbial stimulation of catecholamine pathways. This accelerates norepinephrine and epinephrine turnover and raises VMA levels. Clinically, high VMA is associated with heightened stress responses, anxiety, elevated blood pressure, headaches, and rapid gut motility, reflecting amplified catecholamine activity and increased gut–brain metabolic signaling. Mild elevations in VMA levels may result from certain medications, including TCAs, MAOIs, and SNRIs, as well as dietary factors such as bananas, oranges, tangerines, avocado, tomatoes, plums, prunes, pineapple, chocolate, and walnuts. Supplements that may contribute to mild elevations include tyrosine, bitter orange, yohimbe, and Mucuna pruriens. Markedly elevated VMA levels may be associated with a catecholamine-secreting tumor and should be evaluated in conjunction with the patient's clinical presentation and overall diagnostic assessment. Additional testing may include measurement of plasma free metanephrines and/or 24-hour urinary fractionated metanephrines and catecholamines.

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Organic Acids - Summary

Neurotransmitters & Stress Hormones

Serotonin & Kynurenine Metabolites & Ratios	Current	Previous	Result	Reference
5-Hydroxyindoleacetic acid (5-HIAA) (mcg/g)	5.20		0 1710 9788	1711.0-9788.0

5-Hydroxyindoleacetic acid (5-HIAA) is the principal end-product of serotonin (5-hydroxytryptamine) metabolism. In the body, serotonin synthesized from tryptophan is degraded primarily by monoamine oxidase (MAO) to 5-HIAA, which is water-soluble and excreted in urine . Because it reflects overall serotonin turnover, urinary 5-HIAA serve as established biomarkers of serotonergic activity. Under normal physiological conditions, 5-HIAA is present in low, stable concentrations, corresponding to balanced serotonin synthesis and degradation. Elevated 5-HIAA levels indicate increased serotonin production and breakdown and are classically associated with carcinoid tumors and active neuroendocrine disease, often correlating with symptoms such as flushing and diarrhea. Persistently high levels may also reflect heightened serotonergic turnover in certain inflammatory or metabolic states and can contribute to systemic effects such as carcinoid heart disease in severe cases.

Kynurenic acid (mcg/g)	1.70		0 125.59 991.3	125.6-991.3

Kynurenic acid (KynA) is a biologically active metabolite formed in the kynurenine pathway of tryptophan degradation. Tryptophan, an essential amino acid, is converted via indoleamine-2,3-dioxygenase (IDO) into kynurenine, which is subsequently metabolized to kynurenic acid. This pathway is strongly activated during systemic inflammation, oxidative stress, immune activation, and tissue injury. Kynurenic acid is produced in multiple tissues, including immune cells and the kidney, and is eliminated primarily through renal excretion. Under physiological conditions, urinary KynA is present at low concentrations; however, increased urinary excretion reflects enhanced activation of the inflammatory tryptophan–kynurenine pathway and/or altered renal handling. Higher urinary KynA levels correlated with reduced glomerular filtration rate (eGFR), prolonged acute kidney injury duration.

Quinolinic acid (mcg/g)	3.30		0 610.29 2432	610.3-2432.9

Quinolinic acid (QA) is a downstream metabolite of the kynurenine pathway of tryptophan metabolism and an intermediate in de novo NAD^+ biosynthesis. Under inflammatory or physiological stress states, activation of indoleamine-2,3-dioxygenase (IDO) increases QA production, while impaired conversion by quinolinate phosphoribosyltransferase (QPRT) promotes its accumulation. QA is water-soluble and excreted in urine, where levels generally reflect systemic pathway activation. Elevated urinary QA is consistently associated with acute illness, kidney injury, autoimmune disease, infections, cancer, and psychiatric disorders, and often correlates with disease severity. Excess levels indicate inflammatory activation and disrupted NAD^+ metabolism and may contribute to cytotoxic and neurotoxic effects. Low urinary levels are typically seen in healthy individuals and are not considered clinically harmful, but may reflect reduced pathway flux.

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Organic Acids - Summary

Nutrition & Oxalates

Nutrients	Current	Previous	Result	Reference
Phosphoric acid (mmol/mol)	31		0 999 5000	1000.0-5000.0

Phosphoric acid is the inorganic end-product of phosphate metabolism and reflects systemic phosphate balance, cellular energy turnover, and bone mineral dynamics. In the body, phosphate is a structural component of ATP, nucleic acids, phospholipids, and intracellular signaling molecules, and excess circulating phosphate is filtered by the kidneys and excreted in urine. Thus, urinary phosphoric acid primarily represents renal handling of dietary phosphate and bone–mineral homeostasis. Elevated urinary phosphoric acid may indicate increased phosphate intake, enhanced bone resorption, hyperparathyroidism, or renal phosphate wasting, and can be associated with disturbances in mineral metabolism and potential bone demineralization over time. Persistently high urinary losses may reflect dysregulated calcium–phosphate balance and increased risk of skeletal fragility. Low urinary phosphoric acid may reflect inadequate dietary phosphate, impaired renal excretion, reduced bone turnover, or altered energy metabolism. Chronically reduced phosphate availability can impair ATP production, neuromuscular function, and skeletal mineralization. Overall, abnormal urinary phosphoric acid, either elevated or reduced, signals disruption in mineral metabolism, cellular energy processes, or renal phosphate regulation, and should be interpreted in the context of kidney function, vitamin D status, and overall metabolic balance.

Oxalates	Current	Previous	Result	Reference
Glycolic acid (mmol/mol)	0.65		0 12.59 128.7	12.6-128.7

Urinary glycolic acid (glycolate) is a small organic acid that serves as an intermediate in glyoxylate metabolism and is closely linked to oxalate production, a key factor in calcium oxalate kidney stone formation. Elevated urinary glycolic acid is particularly significant in primary hyperoxaluria type I, where it reflects increased endogenous glyoxylate production and impaired metabolic conversion, often accompanied by high oxalate and increased glycolate/creatinine ratio. Markedly high levels therefore suggest metabolic oxalate overproduction and increased nephrolithiasis risk. Mild elevations may also be seen with dietary factors or mild hyperoxaluria.

Creatinine

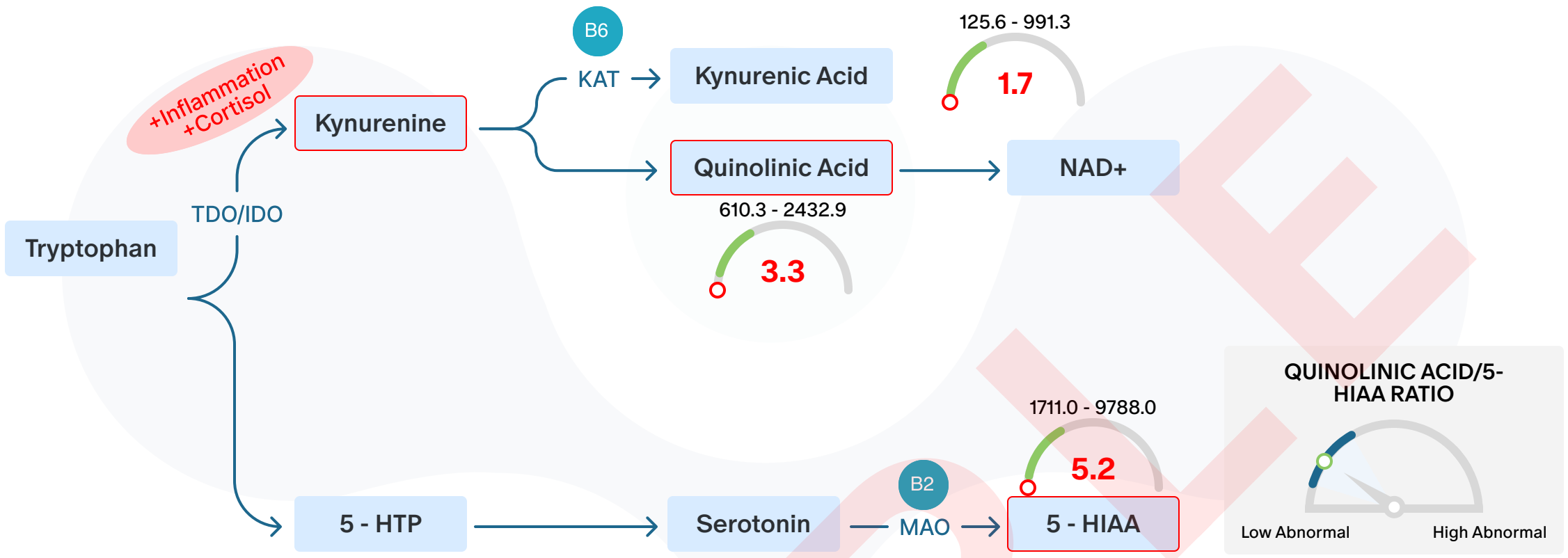
Urine Creatinine	Current	Previous	Result	Reference
Urine Creatinine (mg/ml)	0.88		0 0.24 2.16	0.25-2.16

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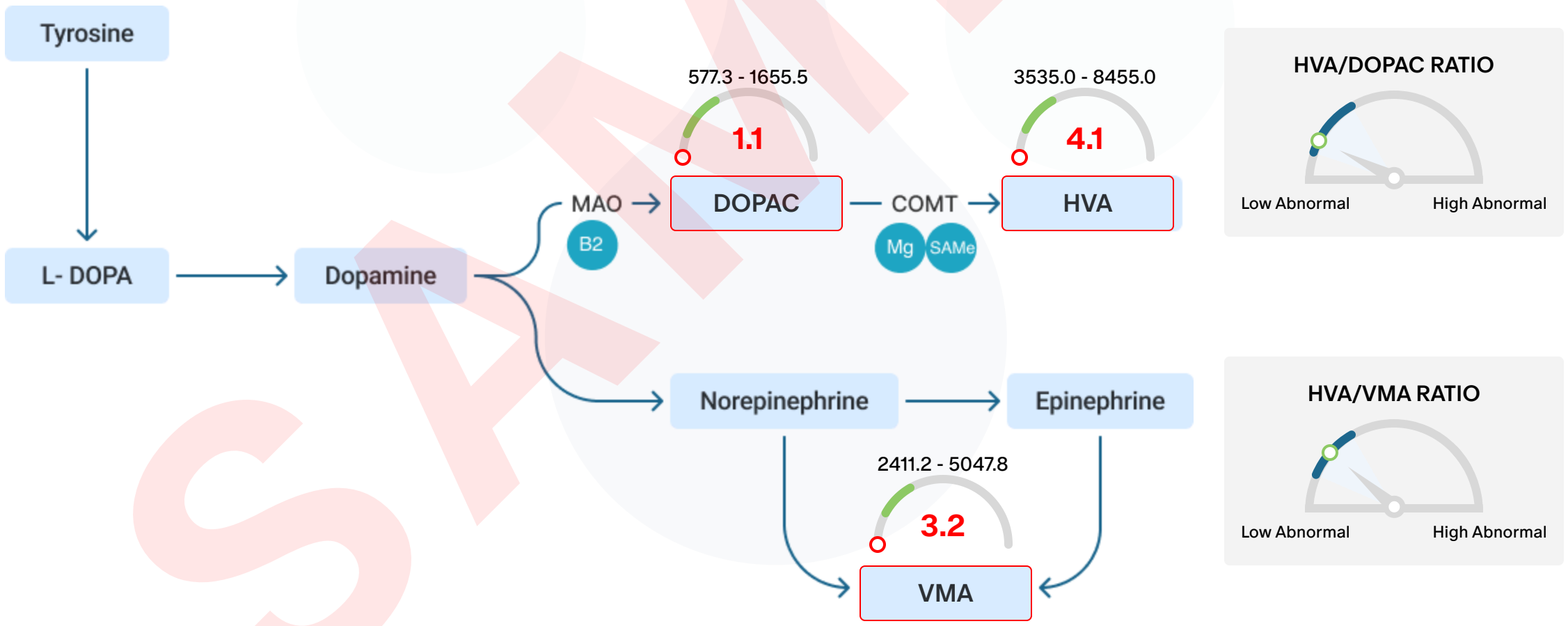
Organic Acids - Summary

Axon Terminal

Tryptophan metabolites & ratios



Catecholamine Metabolites & Ratios



Legend

High/Low | In control | Current Value

NOTE

Inflammation and cortisol are inducers of the kynurenine pathway

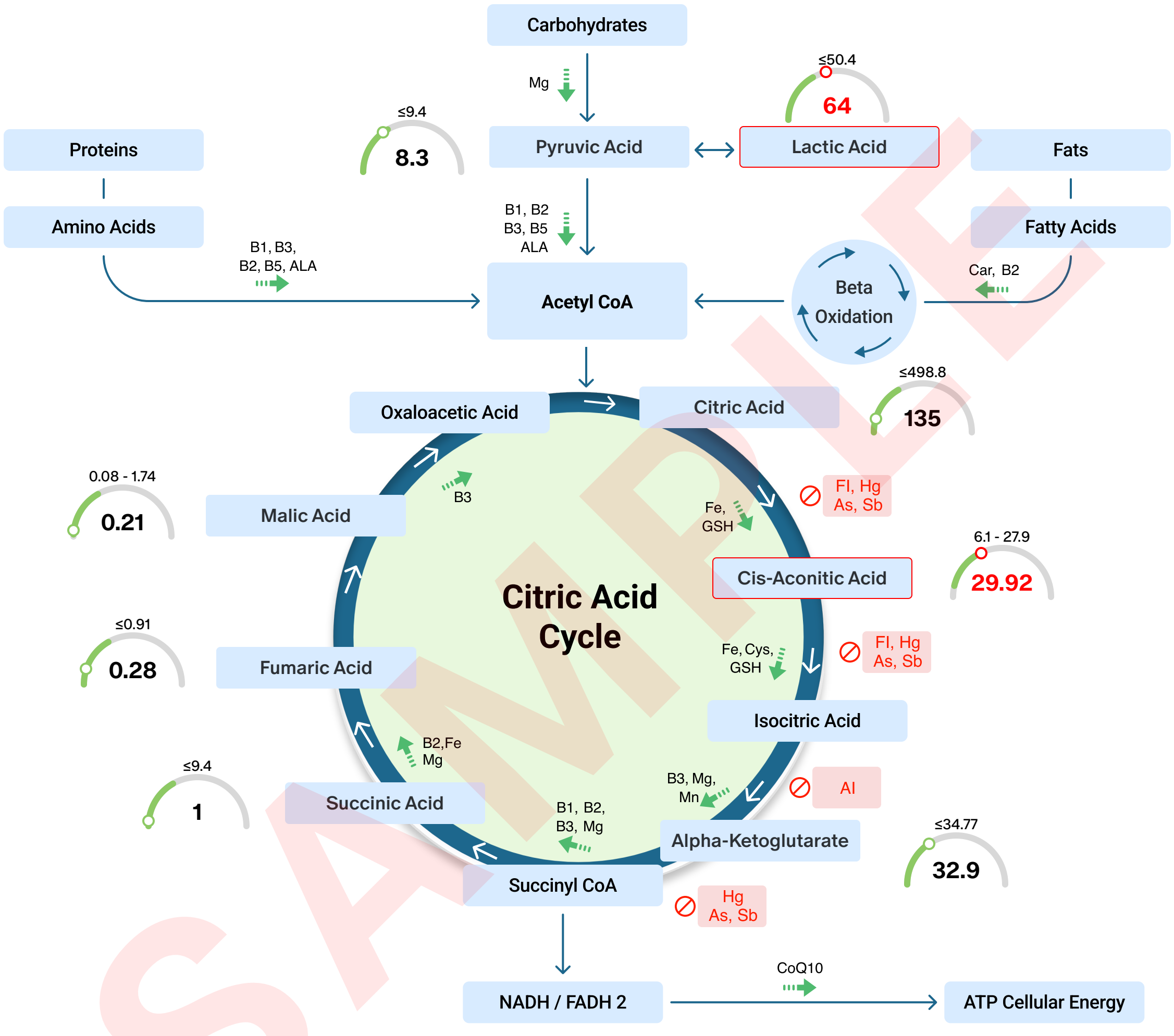
ABBREVIATION KEY

5-HTP	*5-hydroxytryptophan*	**HVA**	*Homovanillic acid*	**MAO**	*Monoamine oxidase*
5-HIAA	*5-hydroxyindoleacetic acid*	**IDO**	*Indoleamine 2,3-dioxygenase*	**TDO**	*Tryptophan-2,3-dioxygenase*
COMT	*Catechol-O-methyltransferase*	**KAT**	*Kynurenine aminotransferase*	**VMA**	*Vanillylmandelic acid*
DOPAC	*3,4-dihydroxyphenylacetic acid*	**L-DOPA**	*L-3,4-dihydroxyphenylalanine*	**DBH**	*Dopamine Beta Hydroxylase*

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Organic Acids - Summary

Krebs Cycle At-A-Glance



Legend

High/Low | In control | Current Value | Main Pathway | Cofactors | Inhibitors

ABBREVIATION KEY

Al	*Aluminum*	**B2**	*Riboflavin*	**GSH**	*Glutathione*
As	*Arsenic*	**B3**	*Niacin*	**Hg**	*Mercury*
ALA	*Alpha lipoic acid*	**B5**	*Pantothenic acid*	**Mg**	*Magnesium*
Car	*Carnitine*	**FAD**	*Flavin adenine dinucleotide*	**Mn**	*Manganese*
CoQ10	*Co Enzyme Q10*	**FADH2**	*Flavin adenine dinucleotide*	**NADH**	*Nicotinamide adenine dinucleotide*
Cys	*Cysteine*	**Fl**	*Fluoride*	**Sb**	*Antimony*
B1	*Thiamine*	**Fe**	*Iron*		

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Patient Name: DEMO DEMO
Date of Birth: 00-00-0000 Accession ID: 2407077216
Service Date: Local 2026-07-19 11:30

Organic Acids - All Markers

Amino Acid Metabolites

Amino Acid Metabolites	Current	Previous	Result	Reference
2-Hydroxyisocaproic acid (mmol/mol)	0.62		0 0.88	≤0.88
2-Hydroxyisovaleric acid (mmol/mol)	0.29		0 0.4	≤0.4
2-Oxo-4-methiolbutyric acid (mmol/mol)	0.11		0 0.18	≤0.18
2-Oxoisocaproic acid (mmol/mol)	0.34		0 0.41	≤0.41
2-Oxoisovaleric Acid (mmol/mol)	3.40		0 2	≤2.0
3-Methyl-2-oxovaleric acid (mmol/mol)	3.10		0 2.6	≤2.6
4-Hydroxyphenyllactic acid (mmol/mol)	2.10		0 0.84	≤0.84
Homogentisic acid (mmol/mol)	0.24		0 0.35	≤0.35
Malonic acid (mmol/mol)	7.20		0 9.8	≤9.8
N-Acetylaspartic acid (mmol/mol)	1.40		0 3.9	≤3.9
Phenyllactic acid (mmol/mol)	0.48		0 0.21	≤0.21
Phenylpyruvic acid (mmol/mol)	0.91		0 0.22 2.2	0.23-2.2

Detoxification & Oxidative Stress

Ammonia	Current	Previous	Result	Reference
Orotic acid (mmol/mol)	0.24		0 0.07 0.52	0.08-0.52

Glutathione	Current	Previous	Result	Reference
N-Acetylcysteine acid (mmol/mol)	0.20		0 0.26	≤0.26
Pyroglutamic acid (mmol/mol)	41.00		0 10.13 32.45	10.14-32.45

Oxidative Stress	Current	Previous	Result	Reference
2-Hydroxybutyric acid (mmol/mol)	2.60		0 0.05 1.58	0.06-1.58

Toxins	Current	Previous	Result	Reference
Mandelic acid (mmol/mol)	0.10		0 0.24	≤0.24

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Organic Acids - All Markers

Metabolism & Mitochondrial Function

Amino Acid Metabolites	Current	Previous	Result	Reference
4-Hydroxybutyric acid (mmol/mol)	1.90		0 4.57	≤4.57

Fat & Ketones	Current	Previous	Result	Reference
3-Hydroxybutyric acid (mmol/mol)	2.40		0 3.5	≤3.5
Acetoacetic acid (mmol/mol)	1.70		0 9.6	≤9.6
Adipic acid (mmol/mol)	5.60		0 0.03 3.9	0.04-3.9
Ethylmalonic acid (mmol/mol)	3.60		0 0.46 2.74	0.47-2.74
Methylsuccinic acid (mmol/mol)	1.40		0 0.12 2.14	0.13-2.14
Sebacic acid (mmol/mol)	0.20		0 0.23	≤0.23
Suberic acid (mmol/mol)	3.40		0 0.15 2.18	0.16-2.18

Glycolysis	Current	Previous	Result	Reference
Lactic acid (mmol/mol)	64.00		0 50.4	≤50.4
Pyruvic acid (mmol/mol)	8.30		0 9.4	≤9.4

Kreb's Cycle	Current	Previous	Result	Reference
Citric acid (mmol/mol)	135.00		0 498.8	≤498.8
Cis-aconitic acid (mmol/mol)	29.92		0 6.09 27.9	6.1-27.9
Alpha-ketoglutarate (mmol/mol)	32.90		0 34.77	≤34.77
Succinic acid (mmol/mol)	1.00		0 9.4	≤9.4
Fumaric acid (mmol/mol)	0.28		0 0.91	≤0.91
Malic acid (mmol/mol)	0.21		0 0.07 1.74	0.08-1.74

Mitochondrial Function	Current	Previous	Result	Reference
3-Hydroxyglutaric acid (mmol/mol)	7.90		0 4.9	≤4.9
3-Methylglutaconic (mmol/mol)	8.60		0 6.2	≤6.2
3-Methylglutaric acid (mmol/mol)	0.42		0 0.75	≤0.75

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Organic Acids - All Markers

Microbial Metabolites

Bacterial Metabolites	Current	Previous	Result	Reference
2-Hydroxyphenylacetic acid (mmol/mol)	0.41		0 0.04 0.69	0.05-0.69
4-Hydroxybenzoic acid (mmol/mol)	0.55		0 1.3	≤1.3
4-Hydroxyhippuric acid (mmol/mol)	12.40		0 0.73 16.98	0.74-16.98
Dihydroxyphenylpropionic acid(DHPPA) (mmol/mol)	0.72		0 0.44	≤0.44
Hippuric acid (mmol/mol)	720.00		0 607	≤607.0

Clostridia Metabolites	Current	Previous	Result	Reference
3-Indoleacetic acid (IAA) (mmol/mol)	3.40		0 12.67	≤12.67
4-Cresol (mmol/mol)	0.12		0 74.88	≤74.88
4-Hydroxyphenylacetic acid (mmol/mol)	22.00		0 20.1	≤20.1
3-(3-hydroxyphenyl)-3-hydroxypropionic acid(HPHPA) (mmol/mol)	0.22		0 227	≤227.0

Fungal Metabolites	Current	Previous	Result	Reference
3-Oxoglutaric acid (mmol/mol)	0.14		0 0.31	≤0.31
Arabinose (mmol/mol)	24.00		0 30	≤30.0
Carboxycitric acid (mmol/mol)	0.12		0 30	≤30.0
Citramalic acid (mmol/mol)	0.22		0 3.8	≤3.8
Tartaric acid (mmol/mol)	3.10		0 4.47	≤4.47
5-Hydroxymethyl-furoic acid (mmol/mol)	0.30		0 13.4	≤13.4
Furan-2,5-dicarboxylic acid (mmol/mol)	0.18		0 16.7	≤16.7
Furancarbonylglycine (mmol/mol)	0.21		0 1.82	≤1.82
Tricarballyic acid (mmol/mol)	0.06		0 0.5	≤0.5

Neurotransmitters & Stress Hormones

Catecholamine Metabolites & Ratios	Current	Previous	Result	Reference
Dihydroxyphenylacetic acid (DOPAC) (mcg/g)	1.10		0 577.29 1655	577.3-1655.5

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Organic Acids - All Markers

Neurotransmitters & Stress Hormones

Catecholamine Metabolites & Ratios	Current	Previous	Result	Reference
Homovanillic acid (HVA) (mcg/g)	4.10		0 3534 8455	3535.0-8455.0
Vanillylmandelic acid (VMA) (mcg/g)	3.20		0 2411 5047	2411.2-5047.8
HVA/DOPAC Ratio	3.73		0 2.59 8.3	2.6-8.3
HVA/VMA Ratio	1.28		0 0.73 1.88	0.74-1.88

Serotonin & Kynurenine Metabolites & Ratios	Current	Previous	Result	Reference
5-Hydroxyindoleacetic acid (5-HIAA) (mcg/g)	5.20		0 1710 9788	1711.0-9788.0
Kynurenic acid (mcg/g)	1.70		0 125.59 991.3	125.6-991.3
Quinolinic acid (mcg/g)	3.30		0 610.29 2432	610.3-2432.9
Quinolinic Acid/5-HIAA Ratio	0.63		0 0.31 1.1	0.32-1.1

Nutrition & Oxalates

Nutrients	Current	Previous	Result	Reference
3-Hydroxy-3-methylglutaric (mmol/mol)	0.28		0 0.13 38.95	0.14-38.95
Ascorbic acid (Vitamin C) (mmol/mol)	36.00		0 12.19 179.25	12.2-179.25
Glutaric acid (Vitamin B2) (mmol/mol)	0.34		0 0.02 0.38	0.03-0.38
Methylcitric acid (Biotin) (mmol/mol)	0.22		0 0.14 2.96	0.15-2.96
Methylmalonic acid (Vitamin B12) (mmol/mol)	0.19		0 2.21	≤2.21
Pantothenic acid (Vitamin B5) (mmol/mol)	1.80		0 9.91	≤9.91
Pyridoxic acid (Vitamin B6) (mmol/mol)	5.20		0 34	≤34.0
Phosphoric acid (mmol/mol)	31		0 999 5000	1000.0-5000.0

Oxalates	Current	Previous	Result	Reference
Glyceric acid (mmol/mol)	2.20		0 0.73 7.4	0.74-7.4
Glycolic acid (mmol/mol)	0.65		0 12.59 128.7	12.6-128.7
Oxalic acid (mmol/mol)	28.00		0 6.16 110.52	6.17-110.52

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Organic Acids - All Markers

Nutrition & Oxalates

Pyrimidines	Current	Previous	Result	Reference
Thymine (mmol/mol)	0.11		0 — 0.63	≤0.63
Uracil (mmol/mol)	0.32		0 — 9.4	≤9.4

Salicylates	Current	Previous	Result	Reference
2-Hydroxyhippuric acid (mmol/mol)	0.41		0 — 1.42	≤1.42

SAMPLE

Cellular Zoomer

RISK AND LIMITATIONS

Test results reflect biological and analytical findings at the time of specimen collection and may vary between individuals. Reference ranges for most of laboratory-developed tests (LDT) were established using a healthy adult population and may not be representative of other specific populations (e.g. pediatric, pregnant, individuals with chronic conditions or from all ethnic backgrounds). They do not provide absolute levels at which the symptoms may occur and hence clinical correlation by the provider is recommended.

Results may be affected by pre-analytical variables related to specimen collection, handling, transport, storage, and inherent biological variability. Specimens including urine, saliva, stool, and blood-based samples (serum, plasma, EDTA whole blood, TES, and dried blood spots) may be impacted by improper collection technique, contamination, insufficient sample volume, delayed shipment or processing, temperature excursions, or improper storage conditions. Additional factors such as hemolysis; anticoagulant effects; clotting, centrifugation, or mixing parameters; incomplete mixing with transport media; and variability in dried blood spot application or saturation may further affect analyte stability or result accuracy. Specimen-specific factors, including urine dilution or concentration, variability in saliva composition or flow rate, and intermittent microbial shedding in stool, may also contribute to result variability. For stool-based testing, one negative specimen does not rule out the possibility of parasitic infection or the presence of other pathogens. These pre-analytical variables may impact result accuracy and, in some cases, lead to a Test Not Performed (TNP). When clinically appropriate, repeat testing may be recommended; however, repeat testing may still fail to produce a reportable result if the underlying limitations persist.

All laboratory testing methodologies are subject to inherent analytical limitations related to instrument performance, assay design, methodological variability, and the specifications of FDA-approved and laboratory-developed analytes included in a test panel. As with all clinical laboratory testing, there is a small possibility of incorrect results due to technical errors, sample misidentification, contamination, rare genetic variants, or software-related issues.

Genetic testing is helpful in analyzing risks to various diseases. However, it is important to note that genetic risk determinants are neither necessary nor sufficient for the development of disease. Environmental and lifestyle risk factors could also affect the risk of disease development. Genetic risk does not indicate how common a health condition or variant is within the population; a risk-associated variant may be common or uncommon. Interpretation of genetic results should consider individual health context, as population-based reference frameworks may not fully represent all age groups, ethnic backgrounds, or health profiles. Genetic testing evaluates only the genotypes indicated and does not assess other genetic abnormalities found elsewhere in the genome. Different laboratories may test different variants when evaluating genetic risk for a given condition; therefore, genetic risk results may not be directly comparable between laboratories.

Some individuals may experience anxiety related to their genetic test results. Vibrant encourages any concerned individual to consult with a qualified healthcare professional prior to sample collection for a genetic test. Users of the test are encouraged to discuss their test results with a genetic counselor, board-certified clinical molecular geneticist, or equivalent health care professional. In some cases, the identification of risk-associated genetic variants may prompt discussion with a healthcare provider about additional testing or follow-up.

The reported analytes, SNPs, and associated informational content are informed by scientific knowledge at the time of reporting, including peer-reviewed scientific publications, publicly available research, and guidance from recognized scientific and public health organizations. Interpretive content may be updated as scientific knowledge continues to evolve. The informational content included in this report is derived from publicly available scientific literature and is provided for educational and informational purposes only. This content does not replace medical advice from a qualified healthcare professional. Any wellness, nutritional, or dietary recommendations, diagnoses of medical conditions, or treatment decisions based on these results are made at the discretion and responsibility of the ordering healthcare professional.

Vibrant does not diagnose, treat, or cure medical conditions and does not replace the care of a licensed medical practitioner or counselor, nor does Vibrant recommend self-diagnosis or self-medication. Depending on the nature of testing, individuals who receive moderate- or high-risk results may be advised to pursue confirmatory testing and appropriate medical follow-up. Vibrant assumes no liability for any loss, injury, or damages arising from the procurement, compilation, interpretation, delivery, or reporting of information contained in this report, nor from any decisions made or actions taken based on these results.

The supplement recommendations and dosage guidelines provided are intended for general informational purposes only and should not replace professional medical advice; final dosage decisions must be made in consultation with your healthcare provider. Vibrant disclaims any liability for adverse effects, outcomes, or consequences arising from the use of these suggestions.

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