

## Summary of Results

**REOUISITION** # **PATIENT NAME DATE OF BIRTH GENDER PRACTITIONER** 

Report Sample Mar 9, 1960 **AGE NO PHYSICIAN** 

**COLLECTION TIME COLLECTION DATE SAMPLE TYPE REPORT DATE** 

06:30 AM Apr 5, 2025 Urine Nov 14, 2025

KEY

**IMBALANCE** 



OPTIMAL



MARGINAL



SIGNIFICANT

#### Microbial Overgrowth



6 Tartaric

5 Furancarbonylglycine

21 Oxalic

Results may indicate significant microbial imbalance.





46 Methylsuccinic

48 Suberic

53 Glutaric

Results may indicate significant imbalances.

13B

11A

#### **Neurotransmitter Metabolites**

36 Dihydroxyphenylacetic (DOPAC)

54 Ascorbic

53 Glutaric

50 Methylmalonic

Results may indicate marginal imbalances in neurotransmitter metabolites.

Results may indicate significant toxic exposure.

2C

#### Toxic Exposure

5 Furancarbonylglycine

58 Pyroglutamic

6 Tartaric

21 Oxalic

9 Tricarballylic

29 Citric

24 Succinic

9D

#### Methylation/Detoxification

58 Pyroglutamic

50 Methylmalonic

53 Glutaric

Results may indicate significant detoxification and/or methylation imbalances.

11E

#### **Nutrient Needs**

46 Methylsuccinic

53 Glutaric

36 Dihydroxyphenylacetic (DOPAC)

21 Oxalic

9 Tricarballylic 58 Pyroglutamic

48 Suberic

Results may indicate multiple nutrient needs.

16F



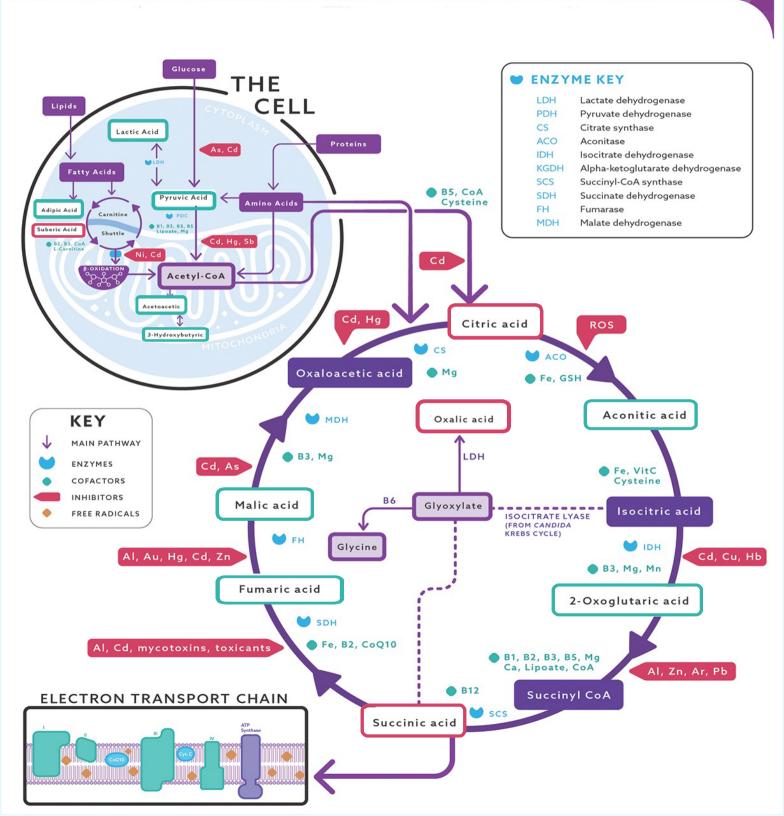


# **Test Results**

	• • ONGANIC ACIDS 1231				
	METABOLITE	REFERENCE RANGE <sup>(n</sup>		RESULTS /mol creat	
IN	TESTINAL MICROBIAL OVERGROW	тн			
YEAST AND FUNGAL	(1) Citramalic	≤3.6		1.6	1.6
	2 5-Hydroxymethyl-2-furoic (Aspergillus)	≤14		14	14
	3 3-Oxoglutaric	≤0.33		0.23	0.23
	Furan-2,5-dicarboxylic  (Aspergillus)	≤16		8.1	8.1
	5 Furancarbonylglycine (Aspergillus)	≤1.9	Н	15	15
	6 Tartaric (Aspergillus)	≤4.5	Н	6.2	6.2
	7 Arabinose	≤29	Н	69	69
	8 Carboxycitric	≤29		12	12
	9 Tricarballylic (Fusarium)	≤0.44	Н	0.55	0.55
RIAL	10 Hippuric	≤613	Н	1,340	340
	2-Hydroxyphenylacetic	0.06 - 0.66		0.53	0.53
BACTERIAL	(12) 4-Hydroxybenzoic	≤1.3		1.2	1.2
	(13) 4-Hydroxyhippuric	0.79 - 17		8.7	8.7
	DHPPA (Beneficial Bacteria)	≤0.38	Н	0.57	0.57
CLOSTRIDA BACTERIAL	4-Hydroxyphenylacetic (C. difficile, C. stricklandii & others)	≤19		15	15
	(C. sporogenes, C. botulinum & others)	≤208		162	162
	4-Cresol (C. difficile)	≤75		37	37
CLOST	3-Indoleacetic (C. stricklandii, C. subterminale & others)	≤11		2.9	2.9







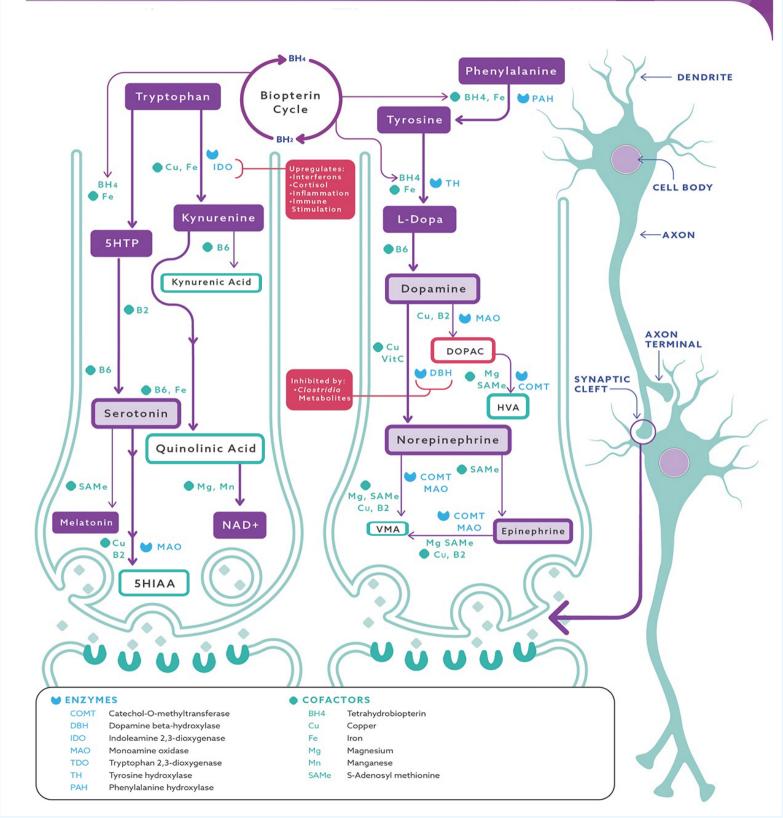


# **Test Results - continued**

				RESULTS /mol crea	SULTS REFERENCE POPULATION Females Age 13 and Over				
OXALATE METABOLITES									
	(19) Glyceric	0.77 - 7.0	Н	7.6	7.6				
	(20) Glycolic	16 - 117		89	89				
	(21) Oxalic	6.8 - 101	Н	224	<u> </u>				
GLYC	DLYTIC METABOLITES								
	22 Lactic	≤48		22	22				
	23) Pyruvic	≤9.1		2.4	2.4				
MITO	CHONDRIAL MARKERS								
	24) Succinic	≤9.3	Н	18	18				
YCLI	(25) Fumaric	≤0.94		0.82	0.82				
D C	(26) Malic	0.06 - 1.8		1.7	1.7				
CITRIC ACID CYCLE	(27) 2-Oxoglutaric	≤35		11	11				
ITRI	(28) Aconitic	6.8 - 28		14	14				
O	(29) Citric	≤507	Н	610	<u></u>				
MITOCHONDRIAL MARKERS									
0 %	(30) 3-Methylglutaric	≤0.76		0.35	0.33				
AMINO	(31) 3-Hydroxyglutaric	≤6.2		5.4	5.4				
A 4	(32) 3-Methylglutaconic	≤4.5		1.4	1.4				
NEURO	OTRANSMITTER METABOLITES								
a a	Homovanillic (HVA) (dopamine)	0.80 - 3.6		3.5	3.5				
SINE	(34) Vanillylmandelic (VMA)	0.46 - 3.7		2.5	2.5				
/LAL/	(norepinephrine, epinephrine)  (35) HVA / VMA Ratio	0.16 - 1.8		1.4	1.4				
PHENYLALANIN AND TYROSINE	(36) Dihydroxyphenylacetic (DOPAC)	0.08 - 3.5	Н	4.6	4.6				
<b>a 4</b>	(dopamine) (37) HVA/ DOPAC Ratio	0.10 - 1.8		0.77	0.77				
Z									
TRYPTOPHAN	(38) 5-Hydroxyindoleacetic (5-HIAA)	≤4.3		1.9	1.9				
/PTC	(39) Quinolinic	0.85 - 3.9		2.4	2.4				
TR	(40) Kynurenic	≤2.2		1.4	1.4				



# Neurotransmitter Pathway



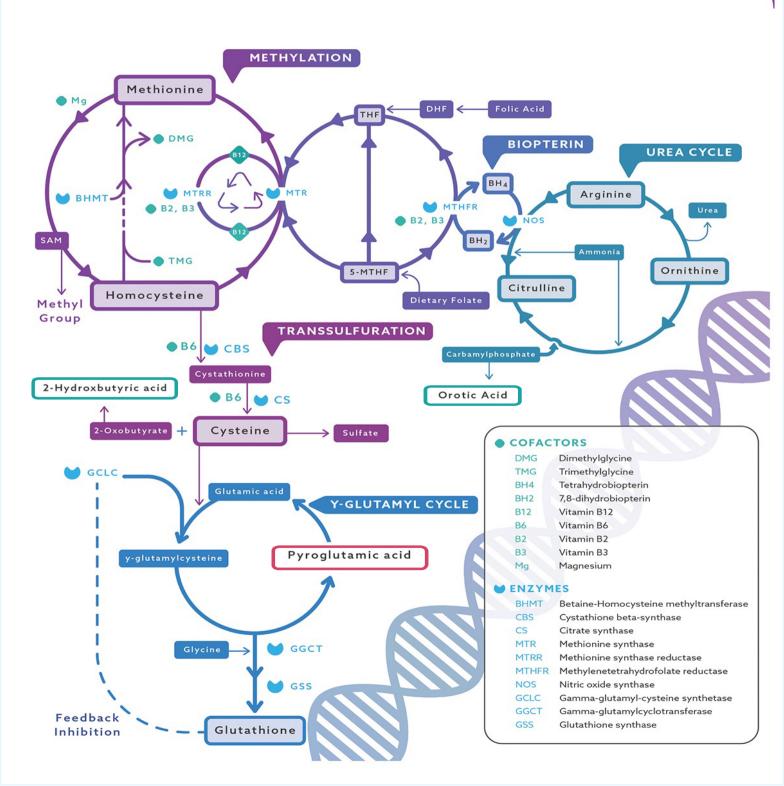


# **Test Results - continued**

METABOLITE	REFERENCE RANGE (n		RESULT /mol cre	REFERENCE POPULATION eatinine) Females Age 13 and Over				
PYRIMIDINE METABOLITES - FOLATE METABOLISM								
41) Uracil	≤9.7		4.5	4.5				
(42) Thymine	≤0.56		0.19	0.19				
KETONE AND FATTY ACID OXIDATION								
(43) 3-Hydroxybutyric	≤3.1		1.7	1.7				
44 Acetoacetic	≤10		1.3	1.3				
45 Ethylmalonic	0.44 - 2.8		2.1	2.1				
(46) Methylsuccinic	0.10 - 2.2	Н	4.1	4.1				
47) Adipic	0.04 - 3.8		2.0	2.0				
(48) Suberic	0.18 - 2.2	Н	3.2	3.2				
(49) Sebacic	≤0.24		0.21	0.21				
NUTRITIONAL MARKERS								
Vitamin B12 (50) Methylmalonic *	≤2.3	н	2.8	2.8				
Vitamin B6 (51) Pyridoxic	≤34		3.7	3.7				
Vitamin B5 (52) Pantothenic	≤10	н	23	23				
Vitamin B2 (Riboflavin)  (53) Glutaric *	0.04 - 0.36	н	0.89	0.89				
Vitamin C (54) Ascorbic	10 - 200	L	0.56	0.56				
Vitamin Q10 (CoQ10)  (55) 3-Hydroxy-3-methylglutaric *	0.17- 39		29	29				
Glutathione Precursor and Chelating A  (56) N-Acetylcysteine (NAC)	agent ≤0.28		0.04	0.00				
Biotin (Vitamin H)  (57) Methylcitric *	0.19- 2.7		1.1	1.1				
* A high value for this marker may indicate a deficiency of this vitamin.								



# Methylation and Detoxification Pathway





\*Creatinine

## **Test Results - continued**

**METABOLITE** REFERENCE RESULTS REFERENCE POPULATION (mmol/mol creatinine) Females Age 13 and Over **RANGE** INDICATORS OF DETOXIFICATION Glutathione (58) Pyroglutamic \* 10 - 33 43 Methylation, Toxic exposure (59) 2-Hydroxybutyric \*\* 0.03 - 1.81.4 **Ammonia Excess** (60) Orotic 0.06 - 0.540.48Aspartame, Salicylates, or GI Bacteria (61) 2-Hydroxyhippuric ≤1.3 0.39 \* A high value for this marker may indicate a Glutathione deficiency.\*\* High values may indicate methylation defects and/or toxic exposure. **AMINO ACID METABOLITES** 2-Hydroxyisovaleric ≤2.0 0 2-Oxoisovaleric ≤2.1 0.57 63) (64) 3-Methyl-2-oxovaleric ≤2.0 0.54 65) 2-Hydroxyisocaproic ≤2.0 0.10 (66) 2-Oxoisocaproic ≤2.0 0.12 67) 2-Oxo-4-methiolbutyric ≤2.0 0.09 68) Mandelic ≤2.0 0.38 69) Phenyllactic ≤2.0 0.12 70) Phenylpyruvic ≤2.0 0.14 Homogentisic ≤2.0 0.02 4-Hydroxyphenyllactic 0.35 ≤2.0 N-Acetylaspartic < 38 2.4 74) Malonic ≤9.7 5.3 3.7 4-Hydroxybutyric ≤4.8 Low values are not necessarily associated with inadequate protein intake and have not been demonstrated to indicate specific amino acid deficiencies. MINERAL METABOLISM (76) Phosphoric 1,000 - 5,000 2.493 INDICATOR OF FLUID INTAKE

10

\* The urine sample has a creatinine concentraction <20mg/dl Low creatinine may indicate sample dilution, potentially leading to skewed values when normalized

to creatine. Consider a repeat specimen collection if results are inconsistent with clinical presentation.

mg/dL



## Explanation of Report Format

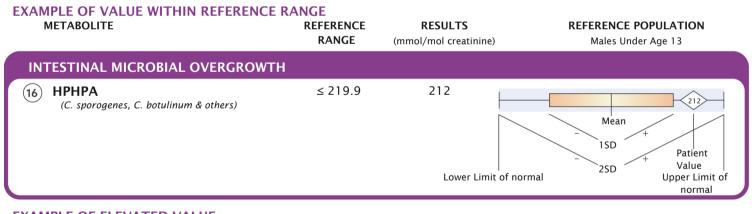
The reference ranges for organic acids were established using samples collected from typical individuals of all ages with no known physiological or psychological disorders. The ranges were determined by calculating the mean and standard deviation (SD) and are defined as + 2SD of the mean. Reference ranges are age and gender specific, consisting of Male Adult ( $\ge 13$  years), Female Adult ( $\ge 13$  years), Male Child (< 13 years), and Female Child (< 13 years).

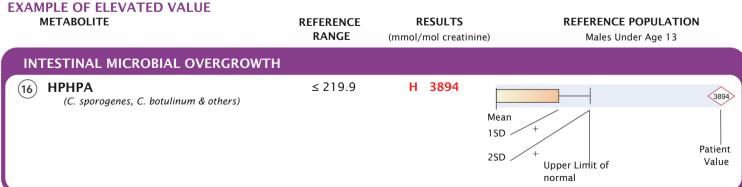
There are two types of graphical representations of patient values in the report format of both the standard Organic Acids Test and the Microbial Organic Acids Test.

The first graph will occur when the value of the patient is within the reference (normal) range, defined as the mean plus or minus two standard deviations.

The second graph will occur when the value of the patient exceeds the upper limit of normal. In such cases, the graphical reference range is "shrunk" so that the degree of abnormality can be appreciated at a glance. In this case, the lower limits of normal are not shown, only the upper limit of normal is shown.

In both cases, the value of the patient is given to the left of the graph and is repeated on the graph inside a diamond. If the value is within the normal range, the diamond will be outlined in black. If the value is high or low, the diamond will be outlined in red.









For more extensive interpretations, refer to the support guide







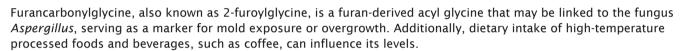








## 5 Furancarbonylglycine



## 6 Tartaric



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

## 7) Arabinose



Arabinose is an aldopentose sugar that can be metabolized by various organisms through pathways involving conversion into intermediates of the pentose phosphate pathway. It has been studied as a marker for intestinal yeast overgrowth and shown clinically to respond to antifungal therapies. Arabinose is also a naturally occurring sugar compound found in numerous plants, such as beets and grains.











Tricarballylic acid is often associated with the fumonisin class of mycotoxins produced mainly by various *Fusarium* species and *Aspergillus nigri* (black aspergilli). These mold exposures can come from indoor environments and contaminated dietary sources such as numerous grain products. This metabolite may also be produced from certain bacteria. Tricarballylic acid has been shown to bind to various minerals, influencing nutritional needs.

## (10) Hippuric







Hippuric acid is a conjugate of glycine and benzoic acid, and may be influenced by gut bacteria, diet, and environmental exposures. It can be produced during the breakdown of benzene-type aromatic compounds from either microbial activity in the gut, exposure to toluene, or naturally produced in the liver from dietary intake of polyphenol-rich foods. Low levels can indicate poor microbial activity, glycine or B5 insufficiencies, and has also been associated with numerous chronic conditions. In rare cases, extreme elevations may be linked to genetic metabolic disorders.

## 14) DHPPA



3,4-Dihydroxyphenylpropionic acid (DHPPA), also known as dihydrocaffeic acid, is a metabolite produced by gut microbiota through the breakdown of certain dietary polyphenols. DHPPA is primarily associated with *Lactobacilli*, *Bifidobacteria*, *E. coli*, and some *Clostridium* species often identified as commensal. Elevated levels may indicate a polyphenol-rich diet or an abundance of these flora, while low levels suggest insufficient polyphenols or potentially reduced beneficial bacteria.









Elevated glyceric acid can result from microbial imbalances, nutrient deficiencies, diet, or genetic mutations. Elevated glyceric acid from fungal or microbial overgrowth, may be through the glyoxylate pathway, where excess glyoxylate is converted to glyceric acid or from direct production. Insufficiencies in vitamin B3 or tryptophan, which are cofactors for glyoxylate reductase/hydroxypyruvate reductase (GRHPR), the enzyme regulating this pathway, may also contribute to increased levels. Additionally, high fructose intake, glycerol metabolism, and various genetic conditions can also lead to excess glyceric acid. Elevated levels have also been associated with conditions such as glucose intolerance, rheumatoid arthritis, and psychiatric illnesses.

## (21) Oxalic



Oxalates, the anionic form of oxalic acid, are naturally occurring compounds that can form crystalline structures when combined with a cation (e.g. calcium, lead, etc). Various organisms such as plants and fungi produce them for structure, defense, and function, but they are also a normal part of human metabolism. Molds and yeasts can produce oxalates directly and indirectly, respectively, while beneficial bacteria such as *Oxalobacter formigenes* help break them down. Additionally, exposure to certain toxicants such as ethylene oxide, ethylene glycol, and trichloroacetic acid can increase levels in the urine. When consumed from oxalate-containing plants (e.g. spinach, soy, beets, tea. etc.), soluble and unbound oxalates are either absorbed in the colon or bind to minerals like calcium and form insoluble compounds in the intestinal lumen, followed by excretion in feces. Oxalates' ability to bind essential minerals may contribute to deficiencies, while factors like vitamin B6 insufficiency, fat malabsorption, high-oxalate food consumption, collagen supplementation, and certain medications can further impact oxalate excretion. Regardless of source, in humans, excessive oxalates can potentially cause systemic problems and have been associated with numerous health conditions involving pain and inflammation.

## (24) Succinic







Succinic acid plays a key role in the Krebs cycle and the electron transport chain, where it is oxidized to fumarate (fumaric acid) via succinate dehydrogenase (SDH), also known as mitochondrial Complex II. SDH is the only enzyme that participates in both the Citric Acid Cycle and the Electron Transport Chain (ETC), giving insights into both pathways. Elevated levels can indicate mitochondrial dysfunction, microbial imbalance, or toxic exposures, such as heavy metals or certain fungicides. Deficiencies in nutrients such as iron, riboflavin (B2), and CoQ10 can also contribute to increased succinic acid levels.







Citric acid is a key component of the Krebs cycle, formed from acetyl-CoA and oxaloacetate by the enzyme citrate synthase. The enzyme aconitase, which requires iron for activity, converts citric acid to isocitrate, and its function is essential for proper citric acid metabolism. Elevated citric acid levels can indicate microbial overgrowth, mitochondrial dysfunction, or toxic exposure to substances such as arsenic or aluminum. Nutrient deficiencies, particularly in iron, can reduce aconitase activity, leading to increased citric acid levels, while oxidative stress can also inhibit the enzyme. Low citric acid levels may result from conditions such as hypokalemia, impaired metabolism, or iron overload, and can increase the risk of oxalate stone formation due to greater free calcium availability.

## 36) Dihydroxyphenylacetic (DOPAC)



Dihydroxyphenylacetic acid (DOPAC) is a key dopamine metabolite formed through the oxidative deamination of dopamine by monoamine oxidase (MAO), resulting in a neurotoxic byproduct, 3,4-dihydroxyphenylacetaldehyde (DOPAL), which is further metabolized into DOPAC by aldehyde dehydrogenase enzyme (ALDH2). DOPAC, along with HVA and VMA, is used to assess dopamine and norepinephrine metabolism with fluctuations in these metabolites reflecting alterations in catecholamine turnover and balance. DOPAC levels can be elevated due to disruptions in dopamine metabolism, which can be caused by DBH inhibition via clostridia or toxic exposures such as mycotoxins or heavy metals. Nutritional deficiencies in vitamin B6, B2, magnesium, copper, and vitamin C, along with genetic variations in COMT or DBH, can also impact DOPAC levels. Low precursors like phenylalanine and tyrosine, as well as deficiencies in cofactors like tetrahydrobiopterin and other enzymatic cofactors, can impair dopamine synthesis and metabolism, leading to lower DOPAC levels. Additionally, dietary factors, medications, and certain pharmaceuticals can further affect dopamine metabolism and lead to either elevated or reduced DOPAC levels.

#### (46) Methylsuccinic



Methylsuccinic acid (MSA), is a fatty acid metabolite involved in isoleucine metabolism. It is associated with mitochondrial dysfunction from dysfunction of fatty acid utilization, and has been associated with type 2 diabetes, and various inborn errors of metabolism.









Suberic acid is a dicarboxylic acid that serves as a key marker of fatty acid metabolism that undergoes omega-oxidation when beta oxidation is impaired. High suberic acid may indicate mitochondrial dysfunction, carnitine or Vitamin B2 (Riboflavin) insufficiencies, metabolic stressors, dietary intake, or in rare cases, genetic abnormalities.

## (50) Methylmalonic











Vitamin B12 is essential for propionic acid metabolism, as it enables the conversion of methylmalonyl-CoA to succinyl-CoA through the B12-dependent enzyme methylmalonyl-CoA mutase (MMUT). When B12 is deficient, this process is disrupted, causing the accumulation of methylmalonic acid (MMA). Propionic acid metabolism connects branched-chain amino acid and odd-chain fatty acid oxidation to the Citric Acid Cycle, and disturbances in this pathway can have widespread metabolic consequences. Vitamin B12 not only plays a role in mitochondrial function, but it can also disrupt the methylation processes, which can have cascading effects, particularly in neurotransmitter metabolism, and detoxification pathways.

#### 2) Pantothenic







Pantothenic acid (B5) plays a crucial role in metabolism, primarily through its involvement in Coenzyme A (CoA) synthesis, which is essential for energy production. Levels of B5 can be influenced by dietary intake, microbial activity, supplementation including products containing B5 derivatives or royal jelly. Rare genetic mutations, such as those in the PANK2 gene linked to PKAN, can lead to significantly elevated levels due to impaired conversion to CoA. Additionally, low B5 levels may have a significant influence on mitochondrial dysfunction, potentially affecting the Citric Acid Cycle and energy metabolism.







Glutaric acid metabolism relies on riboflavin (B2) as a cofactor for glutaryl-CoA dehydrogenase, and a deficiency can lead to its accumulation, impacting amino acid and fatty acid metabolism. Riboflavin is essential for mitochondrial energy production, neurotransmitter synthesis, and detoxification through its role in FAD-dependent enzymes. Insufficient B2 levels can affect ATP synthesis, methylation, and oxidative stress balance, potentially contributing to metabolic dysfunction. Dietary restrictions, medications such as oral contraceptives, and inadequate riboflavin intake may further influence these processes, increasing the risk of deficiency-related complications.

## (54) Ascorbic



Ascorbic acid, also known as vitamin C, is a water-soluble nutrient found in citrus fruits and vegetables, essential for collagen synthesis, various biological functions, and antioxidant protection. Ascorbic acid levels are influenced by factors such as microbial imbalance, mitochondrial health, neurotransmitter metabolism, and nutritional needs such as iron or zinc deficiency. Foods rich in vitamin C and supplements, as well as medications, can increase its levels, while conditions such as critical illness, infections, and smoking raise the body's demand for it. Vitamin C levels are commonly low, due to its ability to be rapidly metabolized and excreted as well as its short half-life.

#### 58) Pyroglutamic



Pyroglutamic acid (5-oxoproline) is formed from glutamate, glutamine, and gamma-glutamylated peptides through gamma-glutamylcyclotransferase and is involved in glutathione metabolism. When intracellular glutathione synthesis is impaired, pyroglutamic acid levels rise, prompting increased cystine uptake to support restoration and reduce its accumulation. Factors such as microbial imbalance, mitochondrial dysfunction, toxic exposures, methylation, and nutritional deficiencies (e.g., magnesium, vitamin B6, precursor amino acids) can elevate pyroglutamic acid levels by disrupting glutathione metabolism and related cycles. Conversely, genetic mutations, severe nutritional deficiencies, and metabolic disorders may influence low levels, though this is mainly theoretical.





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