

Investigation	Observed Value	Unit	Biological Reference Interval
Amino Acid Quantitative Analysis by LC MSMS (EDTA PLASMA)			
Alanine	169.65	µmol/L	111-518
Arginine	29.94	µmol/L	18.4-102
Asparagine	68.64	µmol/L	28.4-102
Aspartic acid	51.45	µmol/L	4.6-60.3
Citrulline	26.3	µmol/L	8.4-40.7
Cystine	6.82	µmol/L	0.9-39.1
Ethanolamine	20.17	µmol/L	4.1-21.9
Glutamic acid	99.01	µmol/L	35-288
Glutamine	482.1	µmol/L	300-688
Glycine	283.48	µmol/L	164.7-402
Isoleucine	<u>23.1</u>	µmol/L	24.7-94.7
Leucine	66.14	µmol/L	44.5-158
Lysine	<u>234.04</u>	µmol/L	75.5-228
Methionine	12.96	µmol/L	9.3-31
Ornithine	63.74	µmol/L	23.4-165
Phenylalanine	95.14	µmol/L	27.7-95.8
4-OH-Proline	<u>Not Detected</u>	µmol/L	8.7-31.9
Proline	164.07	µmol/L	89.2-286
Serine	<u>59.02</u>	µmol/L	61.8-230
Taurine	90.88	µmol/L	34.9-266
Threonine	85.88	µmol/L	51-167
Tryptophan	26.43	µmol/L	15.1-58.4
Tyrosine	<u>109.12</u>	µmol/L	27.3-92.1
Valine	252.87	µmol/L	94.8-261
IMPRESSION	Elevated levels of Lysine & Tyrosine were observed in present sample.		
	Kindly correlate clinically.		

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Sr. No.	Amino Acids	Low	High	
1	Arginine (Arg)	Often reflects a diet poor in high quality protein, causing arginine to be poorly absorbed. Because arginine is required for nitric oxide production, deficiencies have wide-ranging effects on cardiovascular and other systems		May indicate a functional block in the urea cycle. Manganese activates an arginase enzyme, so supplementing with manganese may help.
2	Histidine (His)	Check dietary protein or malabsorption if other essential AAs are low. Low histidine is associated with rheumatoid arthritis, folate deficiency, and/or salicylate/steroid use.		May indicate excessive protein intake. If high 3-Methylhistidine, muscle protein breakdown is indicated.
3	Isoleucine (Ile)	A chronic deficiency of this AA can cause hypoglycemia and related problems and loss of muscle mass or inability to build muscle.		Large intake of this AA or incomplete metabolism of it. If other BCAAs are high, add vitamin B6 to aid metabolism.
4	Leucine (Leu)	Potential catabolism of skeletal muscle. Check 3-Methylhistidine to confirm this.		Large intake of this AA or incomplete metabolism of it. If other BCAAs are high, add vitamin B6 to aid metabolism.
5	Lysine (Lys)	Either poor dietary intake or too high intake of arginine. Low levels can inhibit transamination of AA collagen synthesis. If concurrent weakness or high		Impaired metabolism of lysine. Add vitamin C, niacin, vitamin B6, α -ketoglutarate and iron to enhance utilization of lysine.
6	Methionine (Met)	triglycerides, add carnitine. Possible poor-quality protein diet. Adverse effects on sulfur metabolism. Improve dietary methionine intake or supplement.		Excessive intake of methionine-rich protein or inefficient metabolism. If other sulfur-containing AAs are low, then enhance methionine utilization by adding the necessary cofactors.
7	Phenylalanine (Phe)	Can result in altered thyroid function and catecholamine deficits including symptoms of depression, cognitive disorders, memory loss, fatigue, and autonomic dysfunction. Reduce lifestyle stressors and supplement phenylalanine.		magnesium and vitamin B6. High protein intake or a block in the conversion of phenylalanine to tyrosine. Iron, vitamin C, and niacin are necessary for this enzymatic step. Check tyrosine level and, if low, supplement tyrosine and iron.

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8	Threonine (Thr)	Can result in hypoglycemic symptoms, particularly if glycine or serine is also low. Supplement threonine/BCAAs.	Excessive dietary intake or possible insufficient metabolism of threonine. The initial step here requires (vitamin B6) and zinc is needed to phosphorylate vitamin B6 to its active coenzyme form, so supplementation with vitamin B6 and zinc can be helpful.
9	Tryptophan (Trp)	Commonly correlated with depression, insomnia, and schizo-phrenia. Supplementation with 5-Hydroxytryptophan (5-HTP) may help. 5-HTP is one enzymatic step away from serotonin.	Possibly inadequate metabolism of tryptophan. Required nutrients for this process include niacin and vitamin B6.
10	Valine (Val)	Deficiency in this or other BCAAs indicates potential muscle loss. If several essential AAs are low, check for adequate stomach acid. Supplement the BCAAs.	Excessive intake or vitamin B6 functional deficit. If other BCAAs are high, vitamin B6 should be given.
11	Glycine (Gly)	Possible generalized tissue loss, glycine being part of the nitrogen pool and important in gluconeogenesis. Supplement glycine.	Supplement vitamin B5, folic acid, and vitamins B6, and B2 for the efficient metabolism of glycine to pyruvic acid for oxidation and for glutathione synthesis or gluconeogenesis.
12	Serine (Ser)	Can lead to disordered methionine metabolism and deficits in acetylcholine synthesis. If simultaneous high threonine or phosphoserine, then need for vitamin B6, folate, and manganese is indicated.	When accompanied by low threonine, indicates glucogenic compensation and catabolism. Supplement threonine and BCAAs.
13	Taurine (Tau)	May increase risk for oxidative stress, fat malabsorption, high cholesterol, atherosclerosis, angina, arrhythmias, and seizure disorders. Supplement taurine or cysteine and vitamin B6, even if fresh fish or lean meat is eaten. Females do not synthesize taurine as easily as males.	May be due to excessive inflammation in the body or to supplementation of other amino acids.

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14	Tyrosine (Tyr)	Implicated in depression, hypothyroidism, and blood pressure disorders. If phenylalanine is normal or high (barring PKU), iron, vitamin C, and niacin supplementation might be indicated to help convert phenylalanine to tyrosine.	Inadequate utilization of tyrosine. Supplement the cofactors needed here including iron, copper, vitamin B6, and ascorbate.
15	Asparagine (Asn)	Can reflect functional need for magnesium in the conversion from aspartic acid.	Can indicate problems with purine (therefore protein) synthesis.
16	Aspartic Acid (Asp)	inhibits ammonia detoxification in the urea cycle. Can be converted to oxaloacetate using B6 and α-KG and thus enter the Krebs cycle. Low levels can reflect decreased cellular energy generation, seen as fatigue. Citric and aspartic acids can drive the Krebs (citric acid) cycle, when	Sometimes seen in epilepsy and stroke. Magnesium and zinc may counteract high aspartic acid levels.
17	Citrulline (Cit)	combined with B6 and α-KG. *	Can indicate a functional enzyme block in the urea cycle, leading to an ammonia buildup. Supplement magnesium and aspartic acid to drive the cycle. Lower protein intake is suggested in ammonia toxicities.
18	Glutamic Acid (Glu)	Can suggest mild hyperammonemia, especially if high glutamine. Low protein, high complex carbohydrate and B6, α-KG and BCAA's suggested to correct ammonia toxicity.	Possible underconversion to α-KG in liver for use in citric acid cycle. Supplement niacin and B6.
19	Glutamine (Gln)	Deficient intake or absorption of essential amino acids (glutamine is derived from histidine). Check overall amino acid level of diet.	Marker of vitamin B6 deficiency. Ammonia accumulation suspected, if low or low normal glutamic acid. Extra α-KG needed to combine with ammonia and to make up for energy deficit caused by overutilization of α-KG to deal with toxic ammonia levels.

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20	Ornithine (Orn)	Possibly due to low arginine, as it is synthesized from arginine. As a source of regulatory polyamines, a low can affect cellular metabolism.	A possible metabolic block in urea cycle, causing excess ammonia burden. Confirm by checking for high glutamine, low glutamic acid.
21	α -Amino-N-Butyric Acid	Possible increased need for the nutrients which aid in threonine metabolism from which this AA is derived. These include α -KG and B6.	Inadequate utilization of this for cellular energy generation. Alpha-ABA is converted to succinyl Co-A for use in the citric acid cycle via mechanisms requiring biotin and B12.
22	Alanine (Ala)	May point to hypoglycemic conditions because of its role in gluco-neogenesis. Supplement with alanine and the branched chain amino acids leucine, isoleucine and valine.	Possible inadequate cellular energy substrates. Check for hypoglycemia or for exercise prior to blood draw. Chronic use of alanine for energy can lead to muscle wasting. Supplement the branched-chain amino acids.
23	Proline (Pro)	Tissue levels probably low. As proline is a major component of collagen, low plasma level can mean defective connective tissue synthesis. Proline metabolized to α -KG. Check intake of high quality protein.	Can demonstrate poor utilization. Add vitamin C to aid collagen synthesis if symptoms present. Niacin (cofactor precursor) helps oxidize proline to glutamate.

Note :

- Results to be evaluated in the context of clinical findings and or additional test results
- It is presumed that the specimen used to perform the test belongs to the patient specified above, such verification having been carried out at the level of sample collection.
- Low or undetected levels of amino acids in plasma are also suspected during protein malabsorption, detoxification, oxidative stress, low protein diet or inadequate nutrition
- Increase in concentration of some amino acids could also be due to artifacts arising during sample handling (sample clotting or storage temperature)
- Change in methodology and reference range

References:

- Teitz Textbook of Clinical Chemistry, 2nd edition.
- Filee Romain , al. 2014 Evaluation of physiological amino acids profiling by Tandem Mass Spectrometry; JIMD Rep 13: 119 – 128, Published online 2013 Nov 5. Biochemical Genetics Laboratory, Human Genetics, CHU Liege, University of Liege, Belgium

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