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BIOLOGICAL ROLE OF AMINO ACIDS IN THE HUMAN BODY: METABOLIC, REGULATORY, AND CLINICAL PERSPECTIVES

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Abstract
Amino acids are commonly introduced as the structural units of proteins, but contemporary biochemistry places them at the center of metabolic integration and clinical interpretation. Beyond protein synthesis, amino acids supply carbon skeletons for energy metabolism, mediate nitrogen transfer through transamination and deamination reactions, and serve as precursors for multiple low-molecular regulators including neurotransmitters, nucleotides, nitric oxide, and antioxidants. The maintenance of amino acid homeostasis depends on coordinated digestion and absorption, inter-organ trafficking, de novo synthesis of nonessential amino acids, and regulated catabolism in liver, muscle, intestine, and kidney. Because these processes intersect with signaling networks that control growth, autophagy, immunity, and stress adaptation, disturbances of amino acid metabolism are involved in inherited enzymopathies, liver failure, chronic kidney disease, diabetes, and cancer. This narrative review summarizes key metabolic routes and clinically relevant laboratory indicators that support diagnostics and targeted nutritional or pharmacological interventions, with a brief nutritional perspective on high-quality protein sources rich in essential amino acids.

Keywords: Amino acids; nitrogen metabolism; transamination; urea cycle; mTOR signaling; glutathione; clinical biochemistry; nutritional proteins.

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1. Introduction

Amino acids constitute a regulated circulating pool that links dietary protein, endogenous proteolysis, and organ-specific anabolic and catabolic demands. In this pool, amino acids act simultaneously as substrates for protein renewal, donors and acceptors for nitrogen transfer, and providers of carbon skeletons for mitochondrial oxidation and biosynthesis. For this reason, amino acid metabolism is increasingly described as a systems-level hub that coordinates endocrine, immune, and redox responses in health and disease [1-3].

Clinical relevance follows directly from this integrative role. Disruption of amino acid handling contributes to hyperammonemia and encephalopathy in liver failure, negative nitrogen balance in critical illness, and altered plasma amino acid patterns in metabolic syndrome, diabetes, and cancer [2,12].

At the same time, selected amino acids and their metabolites function as signaling molecules, shaping mTOR-dependent growth control, autophagy, and inflammatory phenotypes [6-8].

From a nutritional perspective, essential amino acids must be obtained exogenously, while nonessential amino acids are synthesized from intermediates of central carbon metabolism. In many physiological states (growth, pregnancy, trauma, infection, or organ dysfunction), endogenous synthesis becomes insufficient, leading to conditional essentiality and clinically meaningful requirements [17]. Therefore, both the quantity and the quality of dietary proteins influence amino acid availability and the downstream metabolic response.

The aim of this article is to provide a structured synthesis of amino acid functions, emphasizing metabolic transformations, regulatory pathways, and clinically relevant laboratory indicators. A brief section also discusses whey-derived proteins as a practical example of a high-quality amino acid source that is increasingly used in food production, sports nutrition, and agricultural practice [27,28].

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2. Materials and Methods

This paper is a narrative review. A structured literature search was performed to identify English- and Russian-language publications on human amino acid metabolism, nitrogen handling (transamination, ammonia detoxification, and urea cycle function), amino acids as signaling mediators, and clinically relevant laboratory assessment.

Searches were conducted using combinations of keywords such as "amino acid metabolism", "transamination", "urea cycle", "BCAA", "mTOR", "glutamine", "hyperammonemia", and "plasma amino acid profiling". In addition, regional (CIS) sources were screened to include foundational educational and review materials commonly used in medical training. Publications were prioritized when they provided mechanistic clarity, clinical interpretation, or methodological guidance for laboratory assessment.

The final synthesis integrates the selected sources into a coherent narrative and proposes a practical set of indicators applicable to routine clinical biochemistry.

3. Results

The reviewed literature converges on three interconnected functional layers of amino acids in human physiology: structural participation in protein and peptide synthesis; metabolic participation in central carbon and nitrogen fluxes; and regulatory participation through nutrient-sensing and redox-sensitive signaling pathways. Together, these layers explain why amino acid homeostasis is simultaneously a biochemical necessity and a clinically informative phenotype.

At the metabolic level, reversible transamination transfers alpha-amino groups among carbon skeletons, allowing nitrogen to be pooled in glutamate and subsequently handled via controlled deamination. The released ammonia is rapidly detoxified, predominantly in the liver, through a mitochondrial-cytosolic pathway that yields urea.

Parallel pathways support the synthesis of key nitrogen-containing molecules such as nucleotides, heme, creatine, and polyamines, highlighting that amino acid flux is not limited to protein turnover.

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At the regulatory level, specific amino acids act as nutrient signals that coordinate anabolic growth with cellular stress adaptation.

This regulation is expressed through changes in translation initiation, autophagy, mitochondrial dynamics, and immune cell effector functions. Accordingly, altered amino acid patterns appear in catabolic stress, chronic organ disease, and inherited enzyme defects.

Clinically, amino acid and nitrogen homeostasis can be assessed indirectly by routine biochemical tests and directly by targeted amino acid profiling. The most informative approach combines markers of protein synthetic capacity, nitrogen disposal, and organ-specific injury patterns, together with context (dietary intake, inflammation, medication, and renal function).

Table 1. Selected laboratory indicators for assessing amino acid and nitrogen metabolism in clinical practice (typical adult reference intervals are approximate and laboratory-dependent).

Indicator (specimen, unit)	What it reflects	Approximate reference interval	Frequent interpretive patterns
Urea / BUN (serum, mmol/L or mg/dL)	Global nitrogen disposal; protein intake vs catabolism; hepatic ureagenesis and renal excretion	Urea ~2.5-7.8 mmol/L (BUN ~7-20 mg/dL)	High: dehydration, high protein intake, catabolic stress, renal dysfunction; Low: severe liver failure, low intake
Ammonia (plasma, µmol/L)	Detoxification capacity and urea cycle flux	~11-35 µmol/L	High: liver failure/portosystemic shunting, urea cycle disorders, some drugs; correlates with encephalopathy risk
ALT/AST (serum, U/L)	Hepatocellular injury affecting amino acid handling and ureagenesis	Method-dependent; often ALT <35-45, AST <35-40	High: hepatocellular injury; interpret with bilirubin/INR to estimate synthetic function
Albumin (serum, g/L)	Hepatic protein synthetic capacity; chronic inflammation and nutritional status	~35-50 g/L	Low: chronic liver disease, inflammation, nephrotic loss, malnutrition; slower to change than prealbumin
Prealbumin / transthyretin (serum, mg/dL)	Short-term protein status and hepatic synthesis (in context of inflammation)	~15-36 mg/dL	Low: acute catabolic states or inflammation; rises with effective nutritional repletion

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Indicator (specimen, unit)	What it reflects	Approximate reference interval	Frequent interpretive patterns
Creatinine and eGFR (serum, mg/dL; mL/min/1.73 m²)	Renal capacity to excrete urea and regulate amino acid/acid-base balance	eGFR >90 (normal); creatinine depends on sex/muscle mass	Low eGFR: retention of nitrogenous waste; altered amino acid patterns and dose adjustment for protein/AA therapy
Plasma amino acid profile (EDTA plasma, µmol/L)	Direct phenotype of amino acid homeostasis; detects inherited defects and secondary disturbances	Panel-specific; laboratory-defined	Characteristic patterns in PKU, MSUD, urea cycle defects, liver disease, critical illness; interpret with clinical context
Homocysteine (plasma, µmol/L)	One-carbon and sulfur amino acid metabolism; B-vitamin status	~5-15 µmol/L	High: folate/B12/B6 deficiency, renal dysfunction, genetic variants; associated with vascular risk
Glutathione status (whole blood or plasma; ratio GSH/GSSG)	Redox buffering capacity linked to cysteine supply and oxidative stress	Method-dependent	Lower ratio: oxidative stress and depleted antioxidant reserve; may accompany chronic disease and inflammation

4. Discussion

The integrated view emerging from the reviewed sources supports the interpretation of amino acids as both metabolites and signals. Nutrient-sensing pathways, particularly those centered on mTOR, connect amino acid availability to translational control and cellular growth programs, while coupling nutrient excess or deprivation to autophagy and stress adaptation [6].

This mechanistic framework helps explain why plasma amino acid signatures can reflect both nutritional intake and disease-driven reprogramming of metabolism [2,3].

Branched-chain amino acids (leucine, isoleucine, and valine) are a representative example. They serve as oxidative fuels in muscle and act as regulatory cues that influence anabolic signaling, yet their circulating elevations have also been associated with insulin resistance in some metabolic contexts [7,8].

Similarly, glutamine occupies a dual position: it is a major nitrogen carrier and an intestinal fuel, but it also intersects with immune cell function and redox regulation through glutathione synthesis [10,11].

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Clinically, the indirect markers summarized in Table 1 are most informative when interpreted as a constellation rather than in isolation. For example, hyperammonemia requires integration of ammonia with liver synthetic function and neurological status; in suspected inherited disorders, targeted amino acid profiling and confirmatory genetic testing are decisive [15,16].

In chronic kidney disease, the balance between adequate protein supply and nitrogenous waste accumulation requires individualized nutritional planning based on renal function and inflammatory state.

Whey-derived proteins provide a practical nutritional context for amino acid biology. Dry whey and whey protein fractions are characterized by a high biological value and a favorable essential amino acid pattern, which supports their use in formulated foods and specialized nutrition. Recent regional publications describe technological and applied perspectives of dry whey production and use in food systems and agriculture [27,28].

At the mechanistic level, whey protein intake can influence hepatic nitrogen metabolism through modulation of transamination flux and urea cycle function, linking dietary amino acid delivery to detoxification capacity and metabolic adaptation [29]. Such observations are consistent with the broader concept that protein source quality affects both amino acid availability and downstream regulatory responses.

5. Conclusion

Amino acids should be viewed as an integrated metabolic network rather than only as protein building blocks. Their involvement in carbon and nitrogen fluxes, redox homeostasis, and nutrient-sensing regulation helps explain their diagnostic relevance in liver and kidney disease, inherited metabolic disorders, catabolic stress, chronic inflammation, and related conditions.

In clinical practice, the most informative evaluation is typically achieved by combining routine biochemical indicators with targeted amino acid profiling when clinically justified. From a nutritional perspective, high-quality proteins such as whey-derived products can support the delivery of essential amino acids

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and may influence nitrogen metabolism, highlighting the importance of dietary protein quality in both preventive and therapeutic approaches.

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