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Review of Heat Stress Effects on Amino Acid Composition in Cattle

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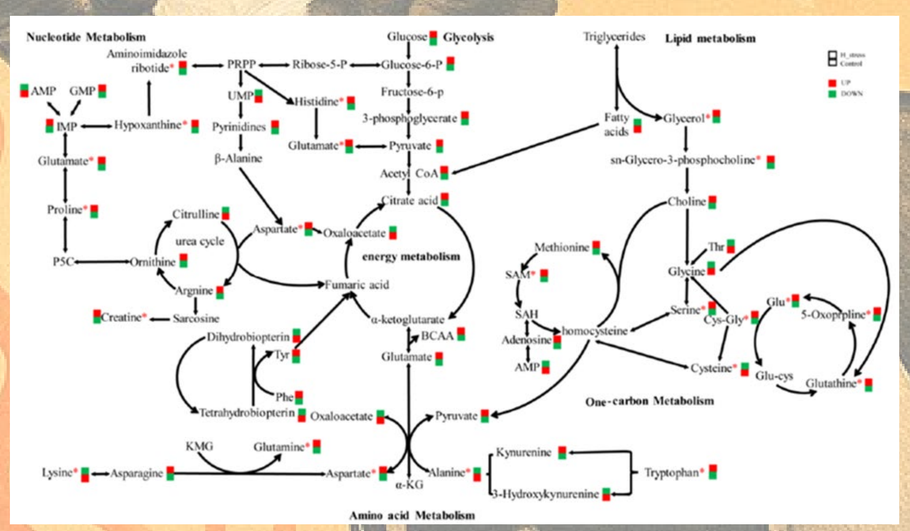
Abstract: *Heat stress (HS) disrupts physiological homeostasis in dairy cattle, impairing productivity and profoundly reshaping amino-acid (AA) metabolism. This review consolidates evidence on HS-induced shifts in intracellular, circulating, and milk AA profiles to pinpoint candidate biomarkers of thermal stress. Across studies, HS promotes skeletal-muscle proteolysis, funneling liberated AAs toward energy generation via gluconeogenesis. Intracellular pools of proline, glutamine, and valine typically rise, whereas their plasma and milk concentrations fall—indicating heightened cellular retention and use. In contrast, essential AAs such as lysine, methionine, and isoleucine uniformly decline in blood and milk, reflecting restricted systemic availability and altered mammary uptake. Stress-responsive AAs linked to immune modulation (threonine), nitrogen cycling (arginine, citrulline, ornithine), and neurotransmitter synthesis (tryptophan, tyrosine) also show compartment-specific alterations, highlighting their regulatory roles in adaptation. Consistent patterns across biological matrices position lysine, glutamine, and proline as promising indicators of HS. Targeted AA profiling of milk and blood therefore offers a practical avenue for monitoring and potentially mitigating the metabolic burden of heat stress in dairy herds.*

• Introduction

Heat stress (HS) drives a systemic metabolic shift in dairy cows, marked by accelerated skeletal-muscle proteolysis that liberates amino acids (AAs) for gluconeogenesis and energy balance. Studies report pronounced reductions in circulating AAs under HS—greater than in pair-fed, thermoneutral controls—alongside higher plasma lactate, signalling altered carbohydrate-AA interplay. Intracellular and serum profiling shows compartment-specific changes: regulators such as phenylalanine, tyrosine, and tryptophan steer catecholamine- and serotonin-mediated stress responses; threonine supports immunity; and alanine sustains glucose production. Milk metabolomics echoes these patterns, with notable declines in taurine and lysine, implicating HS in oxidative-stress defence and protein-synthesis constraints. Urea-cycle intermediates (ornithine, citrulline, arginine) and glucogenic AAs (methionine, glutamine, asparagine) also shift, reflecting heightened nitrogen turnover and TCA-cycle fueling. Overall, HS reshapes AA pools across blood and milk, highlighting lysine, taurine, phenylalanine, tyrosine, and tryptophan as promising biomarkers. This review collates current evidence to pinpoint the most reliable AA indicators for detecting and mitigating heat-stress impacts in dairy cattle.



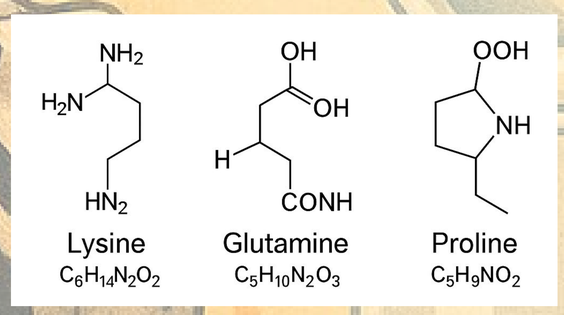
Intracellular metabolic network

• Search Strategy and Selection Criteria

This review mapped heat-stress-induced changes in bovine blood and milk amino-acid profiles through a structured literature-mining protocol. PubMed and Google Scholar were queried with Boolean combinations of "heat stress," "cows," "blood AAs," and "milk AAs," retrieving peer-reviewed studies from 2014-2024 and earlier seminal papers. Full texts were parsed with OpenAI and DeepSeek to recognize recurring metabolic patterns and generate preliminary syntheses. After exclusion of non-English papers, inaccessible articles, and studies unrelated to cattle, amino acids, or thermal stress, 46 of the 70 records met the inclusion criteria. Reference management and citation tracking were handled in Mendeley to ensure methodological transparency.

• Results

Heat stress reshapes amino-acid pools in dairy cattle in a compartment-specific manner. Non-polar, non-essential proline and alanine consistently rise in cells and blood, indicating intensified proteolysis and gluconeogenesis. Among polar uncharged amino acids, glutamine increases within mammary epithelial cells but drops in serum and milk, while taurine and serine grow in the circulation yet decline in milk, and glycine shows early vascular surges followed by systemic dips and later rebounds. Acidic glutamate and aspartate climb intracellularly but fall in plasma or milk, signaling greater mammary uptake. In the urea-cycle group, ornithine and citrulline may spike in cells or milk early during heat exposure but taper with prolonged stress, hinting at shifting nitrogen turnover. Essential amino acids display a consistent pattern of intracellular retention and systemic depletion: valine, leucine, isoleucine, and methionine build up in heat-stressed mammary cells but diminish in blood and milk; phenylalanine and tyrosine follow the same path and serve as metabolic network hubs. Threonine and histidine fluctuate with tissue and time, whereas lysine persistently falls across all compartments, underlining its heat sensitivity. Arginine rises inside mammary cells yet drops in milk, reflecting a trade-off between nitric-oxide synthesis and protein export. Tryptophan is uniformly depressed in blood and milk and down-regulated at the transcriptomic level, indicating rapid diversion into stress-responsive pathways.



• Final remarks and conclusions

HS induced both tissue-specific and amino acid-specific metabolic adaptations, with intracellular levels of several AAs (e.g., Pro, Leu, Cit) increasing in BMECs, while their systemic (blood) and secretory (milk) concentrations were often reduced. This divergence underscores a redistribution of AAs under HS, likely reflecting prioritization of local cellular demands over secretion or systemic circulation.
The results highlighted AAs such as Lys, Gln, and Pro as sensitive and differentially regulated across intracellular, systemic, and milk compartments, thereby showing that they can serve as a candidate biomarker of HS.
The complexity of heat stress-induced AA responses suggests no single universal marker, but rather a panel of metabolites—such as Lys, Val, Met, Arg, and His—may be more suitable for robust HS biomarker development. Notably, AAs involved in gluconeogenesis, immune function, and nitrogen metabolism showed the most consistent alterations, supporting their central role in HS adaptation.