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A review of hormonal regulation of poultry adaptation to heat challenge

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Abstract

Poultry farming has become important part of animal production, providing widely available and relatively low-cost source of animal protein. At the same time, today's broilers and layers are exposed to several environmental and physiological challenges, especially high temperatures, which can disturb normal metabolic balance. This review discusses how poultry respond to heat stress through endocrine and metabolic changes, connecting basic avian physiology with responses at the cellular, tissue, and farm-management levels. Avian metabolism is characterized by high basal blood glucose, dominant hepatic control of gluconeogenesis and lipogenesis, rapid protein turnover, uric acid-based nitrogen excretion, and strong dependence on endocrine coordination. Under thermoneutral conditions, the somatotrophic, thyroid, adrenal, pancreatic, reproductive, and immune-related pathways collectively support growth, egg production, thermoregulation, and defense. During heat stress, however, birds shift from a production-oriented state toward a survival-oriented state. A key player behind that transition is corticosterone, which enhances energy substrate mobilization and acute adaptation. Yet, chronic elevation of corticosterone induces proteolysis, immunosuppression, oxidative stress and disturbance in gut-passage. Heat stress suppresses the hypothalamic-pituitary-thyroid and somatotrophic axes to trigger homeostatic metabolic reprogramming. Consequently, the performance losses are attributed to depressed feed intake which is then governed by altered hormonal regulation of muscle and liver metabolism. New transcriptomic and metabolomic studies have provided further evidence for a molecular crisis that includes overexpression of heat shock proteins, dysfunction of mitochondria, oxidative injury involving lipid peroxidation products, abnormal nutrient metabolism, and activation of catabolic pathways associated with the ubiquitin-proteasome system. Crucially, these effects can happen prior to overt clinical signs seen which suggests that identification of these changes as early events can then lead to preventative treatments undertaken. A multifaceted strategy is needed to address these challenges. Nutrition strategies that stimulate cell-resilience, such as functional amino acids, antioxidants, osmolytes and phytochemical compounds and precision feeding are necessary. At the same time, recent developments in genomic selection, nutrigenomics, and AI-based monitoring may help improve poultry productivity and resilience. Heat tolerance should not be viewed as a single trait, but as the result of several connected processes, including hormonal regulation, liver metabolism, mitochondrial activity, intestinal health, and immune function. Therefore, sustainable poultry production in the future will require an integrated approach that combines physiological knowledge, precision nutrition, and smart farming tools to support productivity, animal welfare, and adaptability under increasing heat conditions.

Keywords: Hormones, metabolism, liver, mitochondria, antioxidants, biomarkers

1. Introduction

Due to the availability of high-quality animal protein food sources [broiler meat and eggs], poultry farming has a vital role in global food security. Genetics, nutrition, housing, and management have all improved production efficiency. But these advances have also increased the metabolic costs of being a bird, and they have made birds less capable of coping with environmental stress. Heat stress is one of the biggest problems in poultry production. Birds produce a lot of heat, they are covered by a layer of feathers, their ability to lose heat through sweating is decreased and also panting (main method for cooling in birds) becomes ineffective at high temperature. Birds compensate when the ambient temperature exceeds the comfort

zone by reducing feed intake, increasing water consumption and respiration rate, altering behavior, and redistributing blood flow to dissipate heat. These responses are vital to survival of the birds, but also extract energy and nutrients away from growth, egg production, immunity and tissue repair (Attia *et al.*, 2024; Nawaz *et al.*, 2021) [10, 44].

While all of these aspects contributed to heat stress in poultry, the main focus of this review emphasizes that any type of heat stress in avian species is not just attributable to feed intake reductions or excessive exposure to environmental conditions. A re-leveling of the hormone-metabolism state whereby recruitment in terms of cellular priorities and nutrient partitioning is reorganized across hormone axes. Elevated corticosterone levels facilitated by the hypothalamic-pituitary-adrenal axis mobilize emergency energy; reduced availability of triiodothyronine via the hypothalamic-pituitary-thyroid axis decreases metabolic heat production for at least some time; and suppressed GH/IGF-1 driven anabolism is typical for the somatotrophic axis. These simultaneous changes convert the bird from a production pathway metabolic profile to one that defends thermoregulation, antioxidant defense and protein quality and gut barrier (Brugaletta *et al.*, 2022; Collier *et al.*, 2018) [13, 18].

The understanding of heat stress is expected to improve with advances in transcriptomics, metabolomics, proteomics, epigenomics and microbiome sciences as well as digital sensing (continuous real-time monitoring) combined with artificial intelligence methodologies thus moving the study of heat stress from quantifiable performance losses to early detection at invisible metabolic disruption. These tools suggest that modulation of gene expression, mitochondrial function, lipid metabolism, gut integrity, immune activity and neuro-hormonal feedback might occur earlier than clinical evidence. Thus, this review brings together information on endocrine responses, tissue metabolism, molecular regulation, and practical interventions to provide a clearer understanding of how poultry adapt to heat stress.

2. Avian Metabolic and Endocrine Background

2.1 Avian metabolic blueprint

Birds have a unique metabolic system that should be understood according to avian physiology rather than by direct comparison with mammals. For example, chickens normally have higher blood glucose levels than mammals, but this does not usually indicate a diabetic condition. Instead, it reflects an adaptation that helps support fast growth and tissues with high energy demands. Poultry metabolism also depends on efficient energy production, rapid use of nutrients, and close coordination between feed intake, liver function, hormonal signals, and tissue needs. These characteristics support high productivity, but they also make modern commercial birds more sensitive when heat stress, inflammation, or limited nutrient intake disturb cellular energy balance (Reyer *et al.*, 2018; Decuyper & Buyse, 2005) [19].

Part of this metabolic system is a group of hormones associated with appetite. Results of association analyses also revealed that ghrelin gene expression levels were related to blood biochemical indicators in Ross 308 broilers, including glucose, uric acid, total protein and albumin contents as well as lipid profile parameters such as triglycerides and low-density lipoprotein cholesterol levels along with the activities of liver enzymes. This indicates that the genes involved in appetite and growth may indicate metabolic status of broilers (Joody *et al.*, 2018; Piórkowska *et al.*, 2020; Rosebrough *et*

al., 2010) [30, 48, 50]. The production of the metabolically prize is expensive and part of the heat-stress problem. Fast-growing broilers and continuously egg-laying layers generated high internal heat through digestion, muscle development, fat metabolism and egg formation. These processes remain in equilibrium under normal temperature conditions to support effective production. But it is this very high metabolic activity which hampers thermoregulation during heat stress by making heat release more difficult. Thus, the endocrine and metabolic responses are diverted towards survival, hydration, acid-base balance and cell protection than growth and egg production (Geraert *et al.*, 1996; Yunianto *et al.*, 1997) [22, 59].

2.2 Hepatic regulation and nutrient partitioning

The liver is a major metabolic organ in poultry. It regulates the metabolism of carbohydrates, lipids and amino acids; promotes gluconeogenesis and de novo lipogenesis; plays a role in detoxification; helps coordinate acute-phase response Janoska & Kliment Adams A. The centrality of hepatic function, then, makes it a major driver of whether birds can preserve productive output or be dominated by a stress-driven metabolism during heat exposure (Emami *et al.*, 2020; Mei *et al.*, 2025) [20, 37].

The metabolism of amino acids is also rearranged, under stress. Birds excrete nitrogen mainly as uric acid, which supports water conservation but requires metabolic energy. During heat stress, amino acids that would normally support muscle accretion, egg protein synthesis, collagen formation, and immune function can be redirected toward gluconeogenesis, antioxidant synthesis, acute-phase proteins, and heat-shock protein production. Functional amino acids such as arginine, glutamine, sulfur amino acids, and branched-chain amino acids are therefore important not only as building blocks for protein but also as regulators of intestinal integrity, immune function, nitric oxide production, and redox balance (Castro & Kim, 2020; Chowdhury *et al.*, 2021) [15, 16].

Mitochondrial control of heat tolerance in the liver and muscle. Mitochondrial ATP production is the primary source for sustaining growth, maintenance and thermoregulation in high performance poultry. High temperature can impair electron transport, increase reactive oxygen species, decrease ATP availability and damage lipids, proteins and nucleic acids. This mitochondrial susceptibility accounts for the co-occurrence of oxidative stress, losses in muscle yield, immune dysfunction and gut barrier (Akbarian *et al.*, 2016; Tan *et al.*, 2009) during chronic heat exposure [2, 56].

3. The Major Endocrine Axes Regulating Metabolic Flexibility

3.1 Growth hormone and somatotrophic axis

Growth hormone (GH): regulates nutrient partitioning and skeletal development, promotes protein deposition, and increases lipid metabolism and hepatic insulin-like growth factor-1 production. Poultry response to GH is influenced by several parameters: receptor sensitivity; nutritional status; availability of thyroid hormones, age, genotype and hepatic responsiveness. Under conditions sufficiently rich in nutrients and endocrine signals, IGF-1 mediates many growth-promoting effects; for example, by supporting muscle-cell proliferation and protein synthesis. In comparison with ancestors, genetic selection has a great impact for the expression and responsiveness of somatotrophic genes in commercial broilers, which may differ depending on growth

rate and body composition (Jia *et al.*, 2018; Vaccaro *et al.*, 2022) [28, 57].

The somatotrophic growth program is inhibited by heat stress. Decreased feed intake leads to a smaller nutrient supply and endocrine adaptation decreases hepatic IGF-1 production and might downregulate GH sensitivity of the tissues. The reduction in GH/IGF-1 signaling limits both anabolic processes and feed efficiency, especially when the increased corticosterone additionally promotes proteolysis. This leads to a synchronous inhibition of protein deposition and muscle development instead of nutrient scarcity. In this regard, variation related to ghrelin is of interest because expression of ghrelin has been linked with growth and carcass traits in Ross 308 broilers within the respective context of this endocrine system. Despite ghrelin polymorphisms not being available for consistent prediction alone, their high expression was associated with WW, WG and carcass-component differences, supporting the use of ghrelin as a complementary marker to studies of growth regulation and nutrient partitioning. (Joody *et al.*, 2019; Zuo *et al.*, 2014; Zheng *et al.*, 2021) [31, 61, 60]

3.2 Thyroid axis, metabolic rate and thermogenesis

Thyroid hormones are central regulators of basal metabolic rate, mitochondrial respiration, thermogenesis, protein turnover, development, and productive performance. Thyroxine (T₄) is converted to the more active triiodothyronine (T₃) by deiodinases, particularly in metabolic tissues such as the liver. T₃ supports oxidative metabolism and tissue growth, but it also increases heat production. Consequently, the thyroid axis is a key mediator of the trade-off between productivity and thermal safety (Mullur *et al.*, 2014; Sejian *et al.*, 2018) [41, 53].

During heat exposure, lower T₃ activity is a common endocrine adaptation. Reduced conversion of T₄ to T₃ decreases metabolic heat production and supports survival in high ambient temperature. Yet it also blunts metabolic demand, protein turnover, growth rate and productive output in this most mundane but important of vessels. The response of the thyroid is not simply a glandularity, it is under the control of signals from the hypothalamus, feed intake (food can influence T₃ status), hepatic metabolism as well as corticosterone, cytokines and mitochondrial state. For this reason, low T₃ under heat stress should be understood as part of a coordinated neuroendocrine strategy to reduce heat load (Aldawood & Al Tamimi, 2022; Amaz & Mishra, 2024) [5, 7].

3.3 Corticosterone and the avian stress-response axis

Corticosterone is the principal avian glucocorticoid and a major indicator of hypothalamic-pituitary-adrenal activation. Thermal, nutritional, immune, and handling stressors stimulate hypothalamic corticotropin-releasing hormone, pituitary adrenocorticotrophic hormone, and adrenal corticosterone secretion. In acute stress, corticosterone supports adaptation by mobilizing glucose precursors, regulating cardiovascular and immune responses, and ensuring energy availability for immediate survival needs (Lin *et al.*, 2021; Scanes, 2016) [34, 52].

The adaptive significance of corticosterone is heavily contingent on both duration and intensity. Elevations in the long-term result in protein catabolism, impaired protein anabolism, reduced growth, altered lipid metabolism, immunosuppression of lymphoid organs and increase in oxidative stress. It further promotes intestinal barrier in addition to impairing biliary transport and is able to aggravate gut-liver and immune-metabolic burden via

inducing intestinal inflammation. So in this context corticosterone acts as a good short term adaptive signal but is disadvantageous if thermal stress becomes chronic (Mishra & Jha, 2019; Patra & Kar, 2021) [40, 46].

3.4 Integrated endocrine reprogramming under heat stress:

This heat-stressed bird has an integrated endocrine phenotype: elevated corticosterone to mobilize emergency substrates, decreased thyroid activity (i.e., and thermogenic heat production), while GH/IGF-1 signaling is restrained to prevent excessive anabolic demand. Collectively, these changes shift nutrients away from growth and reproduction toward thermoregulation, cellular repair, antioxidant protection, immune modulation and maintenance of epithelial barriers. This endocrine pattern is homeorhetic reprogramming where biological priority shifts from maximal productivity to orchestrated survival (Huang *et al.*, 2024; Sumanu *et al.*, 2022) [25, 55].

This reprogramming explains why performance losses during heat stress cannot be fully corrected by simply increasing nutrient density or feed availability. Birds under thermal challenge require nutrients in different proportions and for different biological purposes. Functional amino acids, antioxidant cofactors, osmoprotective compounds, and gut-supporting ingredients become more important because they support resilience pathways rather than only growth. Effective management therefore depends on aligning diet, environment, genotype, and physiological stage with the endocrine-metabolic state of the bird (Kim *et al.*, 2025; Alagawany *et al.*, 2022) [33, 33].

4. Molecular and Tissue-Level Mechanisms of Heat-Stress Adaptation

4.1 Heat-shock response, mitochondrial function and oxidative stress:

On a cellular level, heat stress leads to increased Oxidative pressure, destabilises proteins, challenges membrane integrity and disrupts mitochondrial function. Heat-shock proteins are molecular chaperones that stabilize and re-fold denatured proteins, but the synthesis of heat-shock proteins is energetically costly and consumes precious amino acids. Extended heat increase protein chaperone capacity and pathways rot in inflammation. Such events provide a rationale for why, in many cases, performance losses precede observable clinical distress (AL-Jaryan *et al.*, 2023; Coble *et al.*, 2014) [6, 17].

Thermal-induced reactive oxygen species: Mitochondria as a source and target. High temperature negatively affects electron transport, decreases ATP synthesis, increases lipid peroxidation and reduces antioxidant enzyme capacity. Oxidative stress underlies damage to muscle quality, disturbed hepatic lipid metabolism, compromised immune competence and loss of gut integrity. This systemic metabolic shift rather than localized thermoregulatory disturbance is thereby further corroborated by molecular and metabolomic findings (Huang *et al.*, 2015; Yang *et al.*, 2009) [24, 58].

4.2 Muscle protein turnover and amino acid redistribution

Changes in muscles dependent on breast muscle yield and the growth under heat stress are consistent with altered protein turnover. Elevated plasma corticosterone followed by suppression of IGF-1 signaling, preferentially promotes proteolysis over protein deposition, and the ubiquitous ubiquitin-proteasome system (UPS) is responsible for muscle protein degradation. Excessive amino acids can be diverted into hepatic gluconeogenesis, acute-phase responses,

antioxidant systems and stress-protein synthesis. Thus, this mechanism links changes in the endocrine status directly with carcass performance, feed efficiency, carcass quality and lean tissue wasting. Redistribution of amino acids also occurs in immune and epithelial tissues [6, 7]. Though arginine plays a role in nitric oxide signalling and immune regulation, glutamine supports enterocyte metabolism and barrier function, whereas sulfur amino acids form part of the molecule necessary for lush green plants to make de novo glutathione synthesis. Notably, these functions have increased demand under heat stress, while feed intake decreases in many instances to create a mismatch between nutrient availability and molecular defense requirements. Precision amino acid formulation can therefore support resilience more effectively than crude increases in dietary protein, which may increase heat increment and nitrogen burden (Furukawa *et al.*, 2016; Ma *et al.*, 2020) [21, 36].

4.3 Gut-liver axis, microbiota and intestinal barrier

The intestine is both a significant target and an amplifier of heat-stress injury. For instance, heat exposure was shown to reduce organ blood flow, disturb epithelial tight junctions and caused microbiota dysbiosis with increased permeability by bacterial identification. Barrier dysfunction allows microbial products and inflammatory signals to translocate into the systemic circulation and liver, thus synergizing with the increased metabolic burden of detoxification and acute-phase response/immunoregulatory processes in the liver. Such intestinal injury is capable of activating hepatic oxidative stress and endocrine-metabolic dysregulation amplification through feedbacks in the gut-liver axis (Kikusato & Toyomizu, 2023; Lin and Weng, 2017) [32].

These microbiota-targeted strategies are therefore relevant to endocrine and metabolic robustness. Currently probiotics, prebiotics, synbiotics, phytochemicals and dietary approaches enhancing intestinal barrier effectiveness are being used to minimize inflammation as well as accompany nutrient use during high-temperature environments. These interventions are more effective when combined with other strategies including environmental cooling, improvements in water quality and electrolyte balance as well as diets formulated for reduced heat increment along with maintenance of functional macro- and micro-nutrients (Ahmad *et al.*, 2022; Shehata *et al.*, 2022) [1, 54].

4.4 Omics and epigenetic signatures of resilience

Systems biology of heat-stress adaptation using omics technologies. Genomics, transcriptomics, proteomics, metabolomics and microbiome profiling each offer perspectives on thermogenesis genomic identifies inherited variation in thermotolerance and immune competence; transcriptomic captures change in gene expression; proteomic tracks functional protein responses; metabolomic defines biochemical state; microbiome profiling describes intestinal ecology and epigenomics clarifies long-term regulation. Although these approaches showed that heat tolerance is a network phenotype involving several systems physiology functions including, but not limited to, endocrine feedback, mitochondrial function and hepatic metabolism following endurance exercise, the effects of cellular repair mechanisms are only recently been demonstrated (Hubbard *ET AL.*, 2019; Perini *et al.*, 2020) [47].

Epigenetic regulation could modulate thermal resilience at a given developmental stage. Therefore, they may be suitable to regulate epigenetic marks triggered due to early-life thermal

conditioning (TC) and nutritional programming or as regulators that modulate transcriptional regulation response via heat challenge later in life through DNA methylation and histone modification. Although the response is context-dependent on timing, intensity of exposure, genotype and management practices in ornamental birds, this developmental plasticity may allow them to adapt to thermal exposure. We utilize the convergence of epigenetic biomarkers with transcriptomic and metabolomic data to assign birds and production systems with enhanced adaptability (Loyau *et al.*, 2016; Carvalho *et al.*, 2021) [35, 14].

5. Mitigation Strategies and Applied Interventions

5.1 Environmental and management control

Environmental Control: The first line of heat-stress mitigation Everyone is accountable for leveraging the aspects that directly impact heat load and bird comfort, namely: ventilation, evaporative cooling, shade, stocking-density management, water access, litter quality and feeding schedules. Thermoregulation can help lighten their metabolic load by feeding at cooler times of day, avoiding unnecessary demanding work & reducing humidity. Such interventions are essential, because biological compensations will not fully counterbalance the most severe and persistent thermal environments (Melki *et al.*, 2024; Saeed *et al.*, 2019) [38, 41].

5.2 Precision nutrition and functional nutrients

Precision nutrition under heat stress aims to reduce heat increment while sustaining essential metabolic and protective functions. Diets can be adjusted by balancing digestible amino acids, moderating crude protein, using highly digestible ingredients, optimizing energy source, and supporting electrolyte and acid-base balance. Functional amino acids are particularly important because they contribute to gut integrity, immune modulation, collagen synthesis, antioxidant defense, nitric oxide production, and maintenance of cellular stability.

Nutrigenomics reinforces the notion that nutrients are both biological signals and substrates. Certain nutrients can affect expression of genes, pathways involved and the modulation of inflammatory outcomes as well as other responses relying on redox systems, mitochondrial function and microbial ecology. Such a perspective reinforces the transition from fixed formulation towards age, genotype, heat load, production stage and biomarker-based adaptive feeding. This level of precision feeding is potentially most useful in transition periods, peaks of seasonal heat and reproductive/finishing phases where metabolic demand runs high (Berenjian *et al.*, 2023) [11].

5.3 Antioxidants, osmolytes and gut-directed strategies

Antioxidative therapeutic targets to reduce oxidative stress burden caused by mitochondrial dysfunction and inflammation due to heat; Well-designed antioxidants such as vitamin E and C, selenium carotenoids, polyphenols, phytochemicals to have a positive redox balance and reduction of lipid peroxidation. Osmoprotective compounds and proper electrolyte management may also provide additional cellular hydration, acid-base balance, as well as thermoregulatory stability (Amevor *et al.*, 2025; Ringseis & Eder, 2022) [8, 49].

Gut-directed strategies stabilize gut structure and inhibit inflammatory spillover from the gut-liver axis, in synergy with antioxidant nutrition. All these substances comprise probiotics, prebiotics, synbiotics and phytochemical locus that

were carried over to sustain microbial balance/proliferation, enhance tight junctions function and along with immune regulatory support. Their value is increased when they are combined with environmental control, and diets that limit unnecessary heat production during digestion while providing functional amino acids and micronutrients necessary for epithelial repair. Gut-liver resilience might also be aided by fermented feed ingredients. Hence, a feed efficient and functional continues availability of animal model is key to understand that ameliorative effect of kiwi juice addition in whole barb (Joodi *et al.*, 2025; Al-Baidhani *et al.*, 2025) [29, 4].

5.4 Genomic selection and resilient breeding

Breeding programmes thus have to increasingly balance productivity against robustness. High levels of selection under thermoneutral conditions can also increase metabolic fragility and vulnerability when challenged by heat, disease or nutritional variability. We argue that heat-tolerant birds may not show the lowest optimal performance in optimal conditions but instead exhibit more consistent performance in the face of challenge. This makes resilience a desirable selection target with more practical applications in areas of recurrent high temperature and humidity (Bettridge *et al.*, 2018; Arango & Fulton, 2025) [12, 9].

Conventional traits like survival and feed efficiency, egg output, growth along with molecular correlates of endocrine stability, mitochondrial function, immune competence, oxidative status and gut barrier integrity can be integrated in genomic selection. Abstract There is an opportunity, for research aimed at defining how to more efficiently breed poultry to adapt to the climate change and feed sustainability challenges of tomorrow, by potentially incorporating multiscale data on genomic, transcriptomic, metabolomic, microbiome and epigenetic profiles (Jastrebski *et al.*, 2017; Perini *et al.*, 2020) [27, 47].

6. Future Perspectives: Biomarkers, AI and Heat-Resilient Systems:

Subsequently, future management of heat-stress will shift from reactive treatment toward predictive control. Biomarkers originating from endocrine profiles, oxidative status, intestinal integrity, gene expression, metabolite patterns and microbiome structure are able to detect predisposition to severe panting, growth depression, egg losses or mortality before they take place. Biomarkers that capture the biologically distinct modes of failure will need to be quantifiable, repeatable and scalable at commercial scale. Technologies linked to artificial intelligence and the Internet-of-Things will help translate biological understanding into decisions made on the farm. Machine-learning systems can incorporate cameras, microphones, thermal sensors, wearables, environmental sensors (such as temperature and humidity), feed and water meters (FVs), and weather data to identify abnormal behaviour, predict heat-stress risk, provide advice on mitigation strategies. Such systems can vary ventilation, cooling, lighting, feeding schedule, water management and alerts before a decrease in productivity occurs. They are only valuable when digitally assimilated with physiology, not when viewed in isolation as management outputs (Ojo *et al.*, 2022; Naeem *et al.*, 2025; Natsir *et al.*, 2025) [45, 42, 43].

A climate-resilient system of poultry production will utilize biotechnologies including climate-adapted genetics, precision feeding, microbiome support and welfare monitoring, as well as data-driven control of environmental factors. An aim these days is not only preventing mortality but also maintaining

growth rate, the efficiency of reproduction, immunity to any illness or disease and quality of meat & animal comfort. Within this framework, resilience is a biological and sustainability trait because homeostasis-maintaining birds exposed to heat use feed, water, housing energy and health interventions more efficiently (Hossain *et al.*, 2026; Idowu *et al.*, 2026; Melki *et al.*, 2024) [23, 26, 38].

7. Conclusion

An integrative biological system of hormonal regulation and metabolic adaptation in poultry that coordinates growth, reproduction, thermoregulation, immunity and cellular protection. Historical and comparative perspectives on avian productivity are informed by similar features of the metabolic blueprint, including elevated glucose availability, liver-dominant metabolism, rapid protein turnover, and endocrine control of nutrient partitioning. When under heat stress, this system is rearranged: corticosterone is released to give energy in an emergency situation, thyroidal activity falls so less heat production happens producing a loss of mass and the GH/IGF-1 axis conveys the signal to decrease demand by making fewer structures in tissues. Shifts are protective for survival and detrimental for growth, feed efficiency, egg production, muscle deposition and tissue quality.

Mechanistically, heat stress includes dysfunction of mitochondria, oxidative damage, heat-shock response, redistribution of amino acid metabolism, disruption of intestinal barrier integrity and gut microbiota and the activation of inflammation via gut-liver axis. These responses are indicative of an endocrine-metabolic reprogramming in response to the performance loss, not just a feed-intake issue. Consequently, effective mitigation is contingent on a holistic systems approach encompassing environmental control; precision nutrition including functional amino acids, antioxidants and probiotics as well genomic selection, nutrigenomics/omics biomarker and AI-enabled monitoring capabilities.

In warming climates, sustainable future poultry production will be contingent on the ability to predict stress prior to apparent deterioration and targeted mitigation strategies tailored by genotype, age, stage of production and ambient loads. Integration of systems-based approaches that combine endocrine physiology, molecular biomarkers and smart-farm technologies can provide transformative potential to enhance productivity, welfare and sustainability in poultry systems while maintaining biological resilience under increasing thermal challenge.

Conflict of Interest

Not available

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Not available

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