



Review Article

Thermoregulatory and Systemic Challenges in Broiler Chickens under Heat Stress

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ABSTRACT

Broiler chicken production constitutes a major component of the global poultry industry and significantly contributes to food security through the provision of affordable animal protein. However, heat stress remains one of the most severe environmental constraints affecting broiler productivity, particularly in tropical and subtropical climates. This review examines the thermoregulatory and systemic challenges associated with heat stress in broiler chickens. The thermoneutral balance of broilers is upset by high relative humidity, elevated ambient temperature, and increased radiant heat. This leads to decreased feed intake, poor growth performance, higher mortality, and degraded well-being. Heat stress triggers the hypothalamic-pituitary-adrenal axis, resulting in endocrine and metabolic changes that impair immune function and overall productivity. Broiler chickens exhibit several behavioural and physiological adaptations to dissipate excess body heat, including panting, wing spreading, increased water consumption, and reduced physical activity. The overproduction of reactive oxygen species, which causes oxidative stress when antioxidant defenses are overpowered, is one of the main effects of heat stress. Reactive oxygen species are crucial for immunological responses and cellular signaling. When they are produced in excess, they damage lipids, proteins, and nucleic acids, compromising cellular integrity and physiological processes. The antioxidant defence system, comprising enzymatic antioxidants such as superoxide dismutase, catalase, and glutathione peroxidase, as well as non-enzymatic antioxidants, plays a vital role in maintaining redox homeostasis under thermal stress. Developing efficient management and dietary methods to enhance broiler health, welfare, and productivity in demanding conditions requires an understanding of the relationships among heat stress, oxidative state, and thermoregulatory responses.

Keywords: Antioxidant defence; Broiler chickens; Heat stress; Oxidative stress; Thermoregulation

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INTRODUCTION

Broiler chicken production is a rapidly expanding sector of the global livestock industry and constitutes a major source of affordable, high-quality animal protein (Nassar, 2026). Selective breeding, improved nutritional strategies, and enhanced management practices have substantially increased growth rates, feed conversion efficiency, and carcass yield in contemporary broiler chicken strains (Tavárez and Solis de los Santos, 2016). Nevertheless, these

improvements have increased the susceptibility of broilers to environmental stressors, particularly heat stress, due to elevated metabolic heat production associated with rapid growth (Nawaz *et al.*, 2021). Climate change has intensified thermal challenges in poultry production systems worldwide. Increasing ambient temperatures, recurrent heat waves, and fluctuations in relative humidity have increased the incidence of heat stress, especially in tropical and subtropical regions where environmental conditions

frequently exceed the thermoneutral zone of broiler chickens (Nawab *et al.*, 2018). Heat stress occurs when the amount of heat produced and absorbed by the bird exceeds its capacity for heat dissipation, thereby disrupting physiological homeostasis. Due to the absence of sweat glands and limited evaporative cooling mechanisms, broiler chickens are particularly vulnerable to elevated environmental temperatures (Brugaletta *et al.*, 2022; Olayiwola and Adedokun, 2025).

Exposure to excessive heat adversely affects feed intake, growth rate, feed conversion efficiency, immune function, reproductive performance, and overall welfare (Jongbo *et al.*, 2026). Extended exposure to heat stress stimulates the hypothalamic–pituitary–adrenal (HPA) axis, triggering endocrine and metabolic changes that adversely affect the health, welfare, and productive performance of animals (Zhang *et al.*, 2025). Furthermore, heat stress promotes excessive production of reactive oxygen species (ROS), resulting in oxidative stress when the body's endogenous antioxidant defence mechanisms are unable to adequately neutralise these reactive molecules. Excessive oxidative stress disrupts cellular homeostasis by damaging lipids, proteins, and nucleic acids, ultimately compromising tissue integrity and normal physiological function (Aryal *et al.*, 2025).

Broiler chickens exhibit several behavioural and physiological adaptations to dissipate excess heat, including panting, wing spreading, increased water intake, and reduced activity. Although these responses promote thermoregulation, they are often inadequate under prolonged or severe heat exposure (Roy *et al.*, 2026). Consequently, understanding the interactions among environmental heat load, thermoregulatory mechanisms, oxidative status, and systemic physiological responses is essential for developing effective mitigation strategies (Jongbo *et al.*, 2026). This review, therefore, examines the thermoregulatory and systemic challenges associated with heat stress in broiler chickens and discusses their implications for health, welfare, and productivity.

Heat Stress and Factors Affecting Broiler Chicken Production

Decades of intensive genetic selection aimed at accelerating growth rate and increasing body weight have enhanced the productivity of modern broiler chickens but have also increased their susceptibility to environmental stressors, particularly adverse climatic conditions. Therefore, losses from heat stress, as well as pain and a lack of well-being, are widespread in tropical regions with high temperatures, high humidity, and intense sun

radiation (Nawaz *et al.*, 2021; Oke *et al.*, 2024). The production environment plays a pivotal role in modern broiler production systems, which are designed to achieve maximum growth performance and production efficiency within limited space and over a relatively short production cycle (Averós and Estevez, 2018). Broiler housing facilities should provide a well-regulated thermal environment that enables birds to fully express their genetic potential for growth, health, and productive performance. The facility's thermal environment and performance measures are now more variable due to increased automation and modern technology (George and George, 2023).

Thermoneutral Zone in Broiler Chickens

The thermoneutral zone refers to the environmental temperature range within which homeothermic animals, including birds and mammals, can maintain body temperature without requiring adjustments in metabolic heat production or evaporative cooling mechanisms (Krishnan *et al.*, 2023). Broiler chickens maintained within their thermoneutral zone experience minimal thermal stress and are able to achieve optimal productive performance (Mangan and Siwek, 2024). In poultry, the thermoneutral range varies according to climatic conditions, with reported values of approximately 12–26°C in temperate environments and 18–24°C in tropical regions. As homeothermic animals, broiler chickens attain a stable body temperature of about 42°C by the fourth week of life, while the surrounding temperature required to maintain thermal comfort is approximately 24°C (Vandana *et al.*, 2021; Ayo and Ogbuagu, 2021). Pereira and Naas (2008) found that the thermal comfort temperature of female broiler breeders in the sixth week was approximately 21°C. Heat stress is often caused by ambient temperature variations at twilight in the tropics that exceed the thermoneutral zone of the chicken (Ayo and Ogbuagu, 2021). In the tropics, broilers typically surpass the top limit of the thermoneutral zone, which may be caused by genetics, age, or nutrition (Vandana *et al.*, 2021). Thermoneutral zones are 18–24°C for broilers aged five weeks, and 29–33°C, 30–29.5°C, 26–28.5°C, and 23–27°C for broilers aged one to four weeks. Regardless of age, ambient temperatures outside of their thermoneutral zone may have a detrimental impact on birds' energy balance and fitness (Aluwong *et al.*, 2017). While higher temperatures will reduce production performance, lower temperatures may increase feed consumption. High temperatures have a detrimental effect on livestock, especially broiler chickens, in

terms of their productivity, ability to reproduce, immune responses, and overall health (Lara and Rostagno, 2013). Exposure of broiler chickens to temperatures exceeding the optimal thermal comfort range during the first week of life may predispose them to metabolic disturbances, including ascites and sudden death syndrome (Ayo *et al.*, 2014).

Negative Impacts of Thermal Environmental Stressors on Broiler Chickens

Climatic conditions exert a substantial influence on the health, welfare, and productive performance of broiler chickens, with broader implications for the efficiency and sustainability of poultry production systems (Attia *et al.*, 2024). Global warming and associated climatic changes have adversely affected broiler chicken productivity and overall production performance. The output of broiler chicken production is directly affected by meteorological factors such as relative humidity and ambient temperature (Oke *et al.*, 2024). During the hot-dry season, Nigeria experiences high ambient temperatures, low relative humidity, and significant radiant energy. It makes it harder for broiler chickens to expel heat from their bodies, which leads to heat stress and a high death rate from hyperthermia (Ogbuagu *et al.*, 2023b). At three weeks of age, the mortality rate may rise to 20%, and the broiler chickens will remain very vulnerable until they reach market age at five to seven weeks (Beckford *et al.*, 2020). Thus, for broiler chicks exposed to high temperatures and humidity, body weight is an important factor in determining thermal sensitivity, which has a positive correlation (Apalowo *et al.*, 2024). The physiological and performance traits of broiler chickens are negatively affected by high ambient temperatures in numerous ways. The majority of poultry species have a thermoneutral range of 18 to 24 °C. The rate of growth, productivity, and other biochemical markers are all affected when the ambient temperature goes above this threshold (Nawaz *et al.*, 2021; Vandana *et al.*, 2021). The hot-humid season is characterised by elevated relative humidity and high dry-bulb temperatures, conditions that predispose broiler chickens to heat stress (Hemanth *et al.*, 2024). In broiler chicks, exposure to increased dry-bulb temperatures has been reported to negatively affect performance by reducing feed consumption, limiting body weight gain, and compromising feed conversion efficiency (Ogbuagu *et al.*, 2025). Furthermore, increased ambient temperature may induce heat stress in broiler chickens by elevating cloacal and core body temperatures (Ogbuagu *et al.*, 2023b).

In contrast, relatively low ambient temperatures characterise the Harmattan (cold-dry) season, decreased relative humidity, reduced solar radiation, and the prevalence of dust-laden winds originating from the Sahara Desert and moving towards the Gulf of Guinea. This season is considered the most thermally challenging period in the Northern Guinea Savannah zone (Minka and Ayo, 2014). During the harmattan season, ambient temperatures may fluctuate markedly, ranging from approximately 9°C at night to 35°C during the day. Such thermal fluctuations have been shown to adversely affect the health and performance of broiler chickens (Abioja and Abiona, 2021).

Both hot-humid and cold-dry conditions impose considerable thermal stress on broiler chickens, thereby compromising productivity and welfare. Consequently, the identification, modification, and effective management of environmental factors are essential for minimising thermal stress and optimising broiler performance (Oke *et al.*, 2020) (Figure 1).

Mechanisms Underlying Heat Stress

Heat stress occurs when the amount of heat generated and absorbed by broiler chickens exceeds their capacity for heat dissipation, resulting in a disruption of thermal homeostasis (Brugaletta *et al.*, 2022). This imbalance arises when environmental conditions, particularly elevated ambient temperature and relative humidity, impair the efficiency of sensible and evaporative heat loss mechanisms. Due to their rapid growth rate, high metabolic activity, and absence of sweat glands, broiler chickens are especially susceptible to heat stress (Wasti *et al.*, 2020; Khan *et al.*, 2023).

Exposure to elevated environmental temperatures triggers a series of neuroendocrine and metabolic responses to maintain body temperature. Thermal stress activates the HPA axis, leading to increased secretion of corticotropin-releasing hormone, adrenocorticotrophic hormone, and corticosterone (Krishnan *et al.*, 2023). Sustained activation of the HPA axis alters nutrient metabolism, suppresses immune function, and compromises growth performance. Concurrently, heat stress reduces feed intake as an adaptive response to minimise metabolic heat production, thereby negatively affecting nutrient utilisation and productivity (Prates, 2025).

At the cellular level, heat stress impairs mitochondrial activity and promotes excessive generation of ROS, including superoxide anions (O_2^-), singlet oxygen ($^1O_2$), hydroxyl radicals ($HO^\cdot$), and hydrogen peroxide (H_2O_2) (Belhadj Slimen *et al.*, 2014). Under normal physiological conditions, ROS are produced as part of

cellular metabolism and are efficiently regulated by endogenous antioxidant defence mechanisms. However, during heat stress, increased ROS accumulation can exceed the capacity of these antioxidant systems, leading to an imbalance between oxidant production and antioxidant protection, thereby inducing oxidative stress (Akbarian *et al.*, 2016).

Oxidative stress represents a critical pathological mechanism underlying the adverse effects of heat stress in broiler chickens. Excessive ROS initiates lipid peroxidation of cellular and organelle membranes, leading to loss of membrane fluidity, impaired membrane integrity, and dysfunction of membrane-bound enzymes (Akbarian *et al.*, 2016; Bhattacharjee, 2019). Furthermore, ROS-induced oxidative damage affects proteins, nucleic acids, and mitochondrial components, thereby disrupting cellular signalling, energy metabolism, and gene expression. Severe oxidative injury may trigger apoptosis or necrosis, culminating in tissue dysfunction and reduced physiological efficiency (Juan *et al.*, 2021; Hong *et al.*, 2024).

Heat stress also compromises intestinal integrity by disrupting tight junction proteins and increasing gut permeability. This facilitates the translocation of luminal antigens and pathogens, promotes systemic inflammation, and exacerbates immune dysfunction (Patra and Kar, 2021). Consequently, broiler chickens subjected to prolonged heat stress exhibit impaired health, reduced growth performance, compromised welfare, and increased mortality (Apalowo *et al.*, 2024). Therefore, oxidative damage, endocrine disturbances, and cellular dysfunction collectively underpin the detrimental impacts of heat stress in broiler chickens (Akbarian *et al.*, 2016).

Adverse Effects of Heat Stress on the Production of Broiler Chicken

Reduced Feed Intake and Growth Performance

A significant and immediate physiological response to heat stress is a pronounced decline in feed consumption. Broiler chickens voluntarily decrease feed consumption to minimise the metabolic heat generated during digestion and nutrient metabolism (Ayo and Ogbuagu, 2021). Although this adaptive response reduces endogenous heat production, it also limits nutrient availability for growth. Consequently, heat-stressed broilers exhibit reduced body weight gain, impaired feed efficiency, and lower final market weights (Ogbuagu *et al.*, 2022). Prolonged exposure to elevated environmental temperatures can lead to considerable economic losses in poultry production by reducing productivity

and increasing operational and management costs (Apalowo *et al.*, 2024).

Impaired Nutrient Digestibility and Metabolism

Heat stress adversely influences gastrointestinal function, leading to alterations in digestive enzyme secretion, intestinal morphology, and nutrient absorption (Patra and Kar, 2021). High ambient temperatures reduce intestinal integrity by causing villus atrophy, crypt hyperplasia, and increased intestinal permeability. These changes compromise nutrient digestibility and absorption, thereby reducing the efficiency of protein, lipid, and carbohydrate utilisation (Patra and Kar, 2021; Ogbuagu *et al.*, 2022). Furthermore, heat stress alters endocrine and metabolic processes, shifting energy utilisation from growth towards maintenance and thermoregulatory mechanisms (Vandana *et al.*, 2021).

Deterioration of Carcass Characteristics and Meat Quality

Chronic heat exposure negatively affects carcass yield and meat quality in broiler chickens. Heat-stressed birds generally exhibit reduced breast muscle development, lower dressing percentage, and increased abdominal fat deposition (Nawaz *et al.*, 2021; Ogbuagu *et al.*, 2022). Thermal stress-induced alterations in muscle metabolism can negatively influence meat quality attributes, resulting in reduced water-holding capacity, increased drip loss, changes in muscle pH, development of pale, soft, exudative (PSE) meat, and diminished tenderness (Ogbuagu and Ayo, 2023). Oxidative damage to muscle tissues further contributes to deterioration in meat quality and shelf life (Gray *et al.*, 1996).

Oxidative Stress and Cellular Damage

Increased ambient temperatures stimulate the excessive generation of ROS, thereby inducing oxidative stress when endogenous antioxidant defence systems become insufficient to counteract their accumulation (Garcia-Caparrós *et al.*, 2021). An overproduction of ROS promotes oxidative damage, including lipid peroxidation, protein degradation, and nucleic acid injury, ultimately resulting in impaired cellular function and tissue damage (Afzal *et al.*, 2023). Organs with high metabolic activity, including the liver, intestine, heart, and skeletal muscles, are particularly vulnerable to oxidative damage. Persistent oxidative stress ultimately impairs physiological functions and reduces productive performance (Ogura and Shimosawa, 2014).

Suppression of Immune Function

Heat stress exerts profound immunosuppressive effects in broiler chickens (Liu *et al.*, 2025). Exposure

to high temperatures reduces the development and functional capacity of primary lymphoid organs, such as the thymus and bursa of Fabricius, and secondary lymphoid tissues, including the spleen (Hirakawa *et al.*, 2020). Additionally, heat stress impairs both humoral and cell-mediated immune responses by reducing lymphocyte proliferation, antibody production, and cytokine secretion (Imberti *et al.*, 2025). Consequently, broiler chickens become more susceptible to infectious diseases and exhibit reduced responsiveness to vaccination programmes (Kőrösi Molnár *et al.*, 2021).

Increased Morbidity and Mortality

Severe or prolonged heat stress significantly increases morbidity and mortality rates in broiler flocks (Wasti *et al.*, 2020). Excessive heat load may lead to panting, respiratory alkalosis, dehydration, electrolyte imbalance, and circulatory failure. In extreme cases, birds may succumb to heat exhaustion or heat stroke (Khan *et al.*, 2023). Increased mortality not only reduces flock performance but also imposes substantial economic burdens on poultry producers (Apalowo *et al.*, 2024).

Alterations in Behaviour and Welfare

Heat stress induces several behavioural modifications aimed at dissipating excess body heat. Affected birds typically increase water consumption, reduce physical activity, spread their wings, and exhibit panting and gular fluttering (Ogbuagu *et al.*, 2023a). Although these responses facilitate heat dissipation, prolonged expression of such behaviours is indicative of compromised welfare (Oke *et al.*, 2025). Chronic exposure to elevated temperatures is associated with discomfort, distress, reduced activity, and poor overall well-being, thereby raising important animal welfare concerns (Joseph *et al.*, 2023; Oke *et al.*, 2025).

Endocrine and Physiological Disturbances

Exposure to high ambient temperatures disrupts endocrine homeostasis in broiler chickens. Heat stress alters circulating concentrations of corticosterone, thyroid hormones, insulin, and other metabolic regulators (Geraert *et al.*, 1996; Khan *et al.*, 2023). Elevated corticosterone concentrations promote protein catabolism and suppress immune function, whereas reduced thyroid hormone secretion lowers metabolic rate as an adaptive mechanism to decrease heat production (Habeeb *et al.*, 2018). These endocrine alterations contribute to reduced growth performance and impaired physiological functions (Mehaisen *et al.*, 2017).

Economic Consequences

The cumulative effects of reduced growth rate, poor feed efficiency, increased disease susceptibility, deteriorated carcass quality, and elevated mortality culminate in significant economic losses for the poultry industry (Apalowo *et al.*, 2024). Additional costs associated with cooling systems, health management, and nutritional interventions further increase production expenses (Mangan and Siwek, 2024). Therefore, effective mitigation strategies are essential for sustaining broiler productivity under conditions of elevated environmental temperature.

Thermoregulatory Adaptation of Broiler Chickens under Heat Stress

Thermoregulation is a neurophysiological process that allows animals to preserve internal thermal stability despite fluctuations in environmental conditions by integrating signals from internal and external temperature sensors and initiating appropriate physiological responses, such as vasoconstriction, feather positioning adjustments, and panting (Krishnan *et al.*, 2023).

Like the majority of animals, broiler chickens are homeothermic, meaning that they can withstand a wide variety of ambient temperatures without losing much of their deep body temperature. An adult broiler chicken's body temperature in the tropics typically ranges from 41 to 42 °C (Abioja and Abiona, 2021). Broiler chickens possess both metabolic (chemical) and physical thermoregulatory mechanisms that enable them to maintain a relatively constant body temperature under varying environmental conditions (Krishnan *et al.*, 2023). However, in many parts of the world, especially tropical regions characterised by high temperatures and humidity, broilers are often exposed to environmental conditions that exceed their thermoneutral range, thereby challenging their capacity for effective thermal regulation (Oke *et al.*, 2024). Since birds do not possess functional sweat glands, they depend primarily on evaporative cooling through the respiratory system, involving water loss from the lungs and air sacs, to dissipate excess heat as environmental temperatures increase (Whittow, 1986). During periods of heat stress, particularly in the hot-dry season, broiler chickens exhibit behavioural adaptations that help restore and maintain body temperature within the thermoneutral range (Ogbuagu *et al.*, 2023a). Walking, standing, and feed intake are all decreased during heat stress, whereas sitting, drinking, elevating the wings, and panting are increased to release extra heat. Drinking water replenishes the water lost due to evaporative cooling caused by panting. Additionally, wing

spreading allows heat to escape into the surroundings by exposing the skin of the apteria, or the area beneath the wings (do Amaral Vercellino *et al.*, 2025). Further evidence that temperature stress reduces the welfare of broiler chickens is provided by these behavioural changes, which show that exposed chicks are upset (Wasti *et al.*, 2020).

Furthermore, broiler chickens exposed to heat stress may exhibit several clinical manifestations, including altered thoracoabdominal posture, panting, widened leg stance, body stretching, respiratory distress, increased restlessness, and general weakness (Lara and Rostagno, 2013). Prostration, convulsions, and even heat exhaustion can be symptoms of extreme stress in broilers (Saad *et al.*, 2023). Heat-stressed birds exhibit a variety of adaptive behaviours, such as raising their wings and squatting, slowing down and closing their eyes, moving away from other birds and toward cooler areas, and diverting blood from internal organs to the skin, which darkens the skin and increases pecking. These responses and reactions of broiler chickens to thermally induced stress suggest that heat stress has a significant impact on poultry's daily behaviour and that it is crucial to investigate how heat stress affects growing birds' behaviour (Li *et al.*, 2015; Cartoni Mancinelli, 2023).

Reactive Oxygen Species: Roles in Cellular Homeostasis and Oxidative Damage

Reactive oxygen species are oxygen-derived free radicals characterised by the presence of one or more unpaired electrons, which enables them to exist independently and exhibit high chemical reactivity. ROS are produced when oxygen is taken in and used in various physiological processes (Krumova and Cosa, 2016). Reactive oxygen species comprise a range of oxygen-derived molecules, including hydrogen peroxide, singlet oxygen, superoxide radicals, hydroxyl radicals, and other reactive oxygen-containing compounds, some of which may not possess unpaired electrons but retain strong oxidative potential. They are essential for maintaining tissue oxygen homeostasis (Noguchi and Niki, 2019). A dynamic balance between oxidant and antioxidant systems is sustained through a sequence of spontaneous reduction–oxidation (redox) reactions involving electron transfer between interacting chemical species. Electron-gaining compounds undergo reduction, whereas electron-losing compounds undergo oxidation. When an organism's antioxidant system is unable to neutralise or remove ROS, oxidative stress results (Sies *et al.*, 2024). According to Farag and Alagawany (2018), oxidative stress is a significant source of biological harm and is

believed to be the root cause of a number of conditions that impact the growth and development of chickens. Overproduction of ROS can damage cellular proteins, lipids, or DNA, impeding normal physiological processes and signal transduction pathways. Cancer, heart disease, neurological disorders (Alzheimer's and Parkinson's diseases, multiple sclerosis), diabetes, rheumatoid arthritis, and other conditions have all been connected to cellular damage caused by ROS (Juan *et al.*, 2021; Rauf *et al.*, 2024).

The biological roles of ROS are multifaceted, exhibiting both beneficial and detrimental effects. Physiologically, ROS contribute to immune defence by promoting innate immune responses through their production in phagocytic cells, including neutrophils and macrophages, where they facilitate phagocytosis and the subsequent destruction of invading microorganisms (Andrés *et al.*, 2022). Furthermore, ROS participate in the regulation of adaptive immunity by activating signalling pathways, such as extracellular signal-regulated kinases and p38 mitogen-activated protein kinase pathways, which are involved in lipopolysaccharide-induced cytokine synthesis by macrophages (Bassoy *et al.*, 2021). In addition, ROS are implicated in the regulation of inflammatory processes through the stimulation of pro-inflammatory cytokines, including tumour necrosis factor- α (TNF- α), interleukin-6 (IL-6), interleukin-1 (IL-1), and interleukin-8 (IL-8) (Cantet *et al.*, 2021).

Additionally, ROS are involved in cell signalling by controlling apoptosis and acting as a second messenger in signalling pathways. The cellular response to oxidative stress and growth-promoting stimuli depends on the maintenance of a delicate equilibrium among signalling pathways that regulate cell proliferation, growth suppression, and programmed cell death (Martindale and Holbrook, 2002; Zhang *et al.*, 2016). The essential connections in the signal transduction pathways are second messengers. A brief rise in their concentration within the cell in reaction to an external stimulation is a crucial requirement for second messengers (Newton *et al.*, 2016). Every cell has an internal redox buffer that quickly eliminates ROS and an enzyme system that produces ROS in response to a particular stimulus. As their concentrations in the cell can both rise and fall quickly following the effect of physiological stimuli, ROS may therefore meet the criteria for the second messenger. Numerous growth agents, cytokines, and hormones work by raising the

intracellular ROS concentration (Bindoli and Rigobello, 2013; Checa and Aran, 2020).

Antioxidant Defence System and Stress Responses in Broiler Chickens

In broiler chickens, exposure to heat stress increases the generation of ROS and reactive nitrogen species (RNS), which may exert harmful effects on cellular components. Antioxidants function as protective agents by neutralising free radicals and reducing oxidative damage within cells (Surai *et al.*, 2019). However, when endogenous antioxidant defence mechanisms become overwhelmed, excessive accumulation of ROS/RNS occurs in blood and tissues, promoting lipid peroxidation and subsequent cellular injury. This can harm biological membranes, causing cellular damage and dysfunction, decreasing productivity, and leading to illnesses (Checa and Aran, 2020). The antioxidant defence system in broiler chickens consists of a diverse group of endogenous and dietary antioxidants, including lipid-soluble compounds such as vitamins A and E, carotenoids, and ubiquinones; water-soluble antioxidants including ascorbic acid, uric acid, taurine, and carnitine; as well as enzymatic antioxidants such as superoxide dismutase (SOD), glutathione peroxidase (GPx), and catalase (CAT) (Surai, 2016). By removing precursors or deactivating catalysts, the first line of defence is in charge of stopping the generation of free radicals. This complex consists of three antioxidant enzymes: SOD, GPx, and CAT. The most crucial element of the cell's initial line of antioxidant defence is believed to be SOD (Jomova *et al.*, 2024). It needs a metal cofactor to operate since it is a metalloenzyme. Depending on the type of metal ion it needs as a cofactor, SOD can take many different forms. Superoxide dismutase is a metalloenzyme that commonly incorporates essential metal cofactors, including iron (Fe), zinc (Zn), copper (Cu), and manganese (Mn), depending on its isoform (Ighodaro and Akinloye, 2018). It facilitates the dismutation of superoxide radicals into hydrogen peroxide, which, when excessively accumulated, can induce oxidative damage to cellular structures and biological tissues. In the presence of Fe^{2+} , the Fenton reaction also converts it to the dangerous hydroxyl radical (OH). Therefore, eliminating superoxide radicals can lessen the production of hydroxyl radicals (Lu *et al.*, 2010). Catalase, another antioxidant enzyme that is abundant in peroxisomes, reduces damage caused by free radicals by breaking down H_2O_2 into water and molecular oxygen. The enzyme is primarily localised within peroxisomes, where it participates in the antioxidant defence system. It utilises iron or

manganese as a cofactor to complete the detoxification process initiated by SOD, thereby contributing to the elimination of ROS (Anwar *et al.*, 2024). Glutathione Peroxidase is an enzyme found mostly in the mitochondria but also in the cytoplasm that converts lipid peroxides to their corresponding alcohols and H_2O_2 to water (Ighodaro and Akinloye, 2018). It converts lipid hydrogen peroxides (ROOH) produced from PUFA oxidation into stable, non-toxic hydroxyl fatty acids (Vaya and Aviram, 2001).

The second line of antioxidant defence is activated when the initial defence mechanisms become insufficient. It comprises chain-breaking antioxidants, including vitamins E and A, ubiquinol, carotenoids, ascorbic acid, uric acid, and other antioxidant compounds (Umber *et al.*, 2023). Additionally, the glutathione and thioredoxin systems play essential roles in this phase by limiting oxidative chain reactions. These antioxidants inhibit lipid peroxidation by scavenging peroxy radical intermediates, thereby reducing the propagation of oxidative damage (Surai, 2021).

When the second line of antioxidant defence is insufficient to prevent oxidative damage to lipids, proteins, and DNA caused by ROS and RNS, a third level of defence is activated. This phase involves repair and removal mechanisms that eliminate damaged cellular components. It includes enzymes such as lipases, proteases, DNA repair enzymes, methionine sulfoxide reductase, ligases, nucleases, and polymerases, together with molecular chaperones such as heat shock proteins that restore cellular integrity and function (Surai *et al.*, 2019; Gouda *et al.*, 2024).

Management Strategies for Mitigating Heat Stress in Broiler Chickens

Effective management of heat stress in broiler chickens requires an integrated approach involving environmental, nutritional, and husbandry interventions (Onagbesan *et al.*, 2023). Environmental management should focus on improving ventilation and air movement, reducing stocking density, providing adequate shade, and maintaining suitable house conditions (Holik, 2009; Apalowo *et al.*, 2024). Continuous access to clean and cool drinking water is essential to support thermoregulation and prevent dehydration (Arsenopoulos *et al.*, 2026). Feeding during cooler periods of the day and minimizing handling, transportation, and other stressful activities during periods of high ambient temperature can further reduce the physiological burden associated with heat exposure (Prates, 2025).

Nutritional interventions may complement environmental management by improving the capacity of broiler chickens to withstand thermal stress (He *et al.*, 2018). Supplementation with appropriate electrolytes, vitamins, minerals, antioxidants, and selected feed additives may help maintain fluid and electrolyte balance, reduce oxidative stress, and support physiological and immune functions (Ogbuagu *et al.*, 2023a; 2025). Regular monitoring of ambient temperature, relative humidity, temperature–humidity index, and behavioural or physiological indicators of heat stress is also recommended to facilitate timely management interventions (Onagbesan *et al.*, 2023). Collectively, these strategies can reduce the adverse effects of heat stress on feed intake, growth performance, physiological homeostasis, health, and overall productivity of broiler chickens (Wasti *et al.*, 2020).

CONCLUSION

Heat stress constitutes a significant challenge to broiler chicken production by disrupting thermoregulatory balance and inducing physiological, metabolic, immunological, and oxidative disturbances. Excessive production of reactive oxygen species during thermal stress overwhelms endogenous antioxidant defence mechanisms, resulting in oxidative damage and impaired productivity. Although broiler chickens exhibit behavioural and physiological adaptations to mitigate heat load, these responses are often inadequate under prolonged exposure. Therefore, effective environmental management and nutritional strategies aimed at enhancing antioxidant capacity are essential for improving broiler health, welfare, and productivity under conditions of thermal stress.

Conflict of Interest

Authors declare no conflict of interest.

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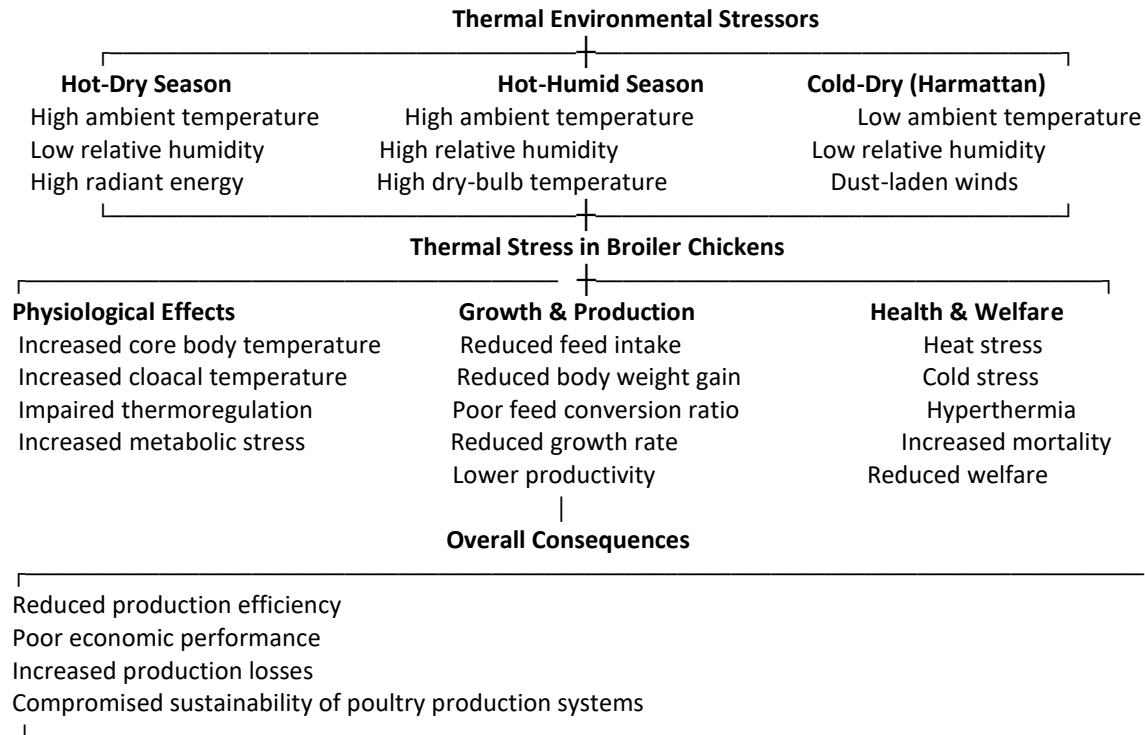


Figure 1: Negative Impacts of Thermal Environmental Stressors on Broiler Chickens