



The Effect of Heat Stress on Meat Quality, Growth Performance and Antioxidant Capacity of Broiler Chickens: a review

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Poultry Science Journal 2022, 10(1): 1-12

Keywords

Broilers
Heat stress
Metabolism
Productivity
Oxidative damage

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Article history

Received: December 17, 2021
Revised: April 26, 2022
Accepted: May 09, 2022

Abstract

Given the escalating environmental temperature and a considerable progress in meat production capacity, broiler chickens are more sensitive to heat stress than before. The detrimental influences of heat stress include diminished feed intake and reduced average daily gain, low feed efficiency, impaired immune response, deterioration of meat quality, and disturbed gastrointestinal microflora. Generation of free radicals, reactive oxygen species, and their combined action on cell membrane leads to damage in the cell membrane integrity and alters the metabolic function of the body resulting in various physiological abnormalities. There have been numerous studies on cost-effective strategies to mitigate the harmful effect of heat stress. The use of various dietary antioxidants, vitamins, minerals, flavonoids, and probiotics, alone or in combination, has led to promising results on the growth performance of broiler chickens, thereby decreasing the unexpected losses incurred due to heat stress. This article aims to review the scientific evidence concerning the various harmful consequences of heat stress in broiler chicken, with emphasis on meat quality, carcass traits, and growth performance. We also discuss some mitigation strategies in the next section.

Introduction

Meat serves as a vital part of a mixed diet (Biesalski, 2005), due to the availability of numerous amino acids and essential micronutrients. Poultry meat production is expected to increase by 16% in 2025 compared to 2013–2015, owing to an increasing human population, widespread acceptance of chicken meat consumption, and low prices, particularly in developing countries (OECD and FAO, 2016; Dal Bosco *et al.*, 2021). Along with the growth of the poultry industry, several constraints are hindering poultry production that has led to economic loss. Previous studies have proposed that heat stress negatively affects poultry production, especially in the tropical region via deviating energy sources from production to adaptation pathways (Vandana *et al.*, 2021).

Poultry industries all around the globe, predominantly in tropical and subtropical, experience heat stress due to the compound effects of the high

ambient temperature and relative humidity, particularly during the summer season (Demir *et al.*, 2021). Temperatures between 18 and 22 °C are suitable for raising broilers, while upper temperatures might cause heat stress (Shakeri *et al.*, 2020), leading to physiological disturbance and cellular damage (Diarra and Tabuaciri, 2014; Pawar *et al.*, 2016; Vandana *et al.*, 2021). In response to heat stress, there is a drastic increase in respiration rate and a higher loss of CO₂, a phenomenon called respiratory alkalosis. and finally, there is an imbalance in the acid balance, which affects the health and performance of birds negatively (Saeed *et al.*, 2019).

As soon as the high ambient temperature surpasses the thermoneutral temperature, poultry is predisposed to detrimental effects of heat stress, inducing physiological alteration to retain homeothermy, which finally disturbs metabolism and gut health, depresses performance, cardiovascular disease, hypercholesterolemia, impairs meat quality

traits and slaughter yields, and even high mortality rate in lethal conditions (Saeed *et al.*, 2019; Shakeri *et al.*, 2020; Wasti *et al.*, 2020). The most convenient strategy to eradicate heat stress is to drop the in-house temperature to a thermoneutral temperature to eliminate the detrimental effect of heat stress and maximize productivity (Nawab *et al.*, 2018).

When poultry is exposed to ambient temperature exceeding over 26 °C, with per unit rise in temperature, appetite reduced by 1.5% and the metabolic rate increases by 20-30%; with a production of a large amount of internal metabolic heat (Zhang *et al.*, 2017). It is also reported that broiler chickens are most susceptible to the harmful effect of heat stress as they are incapable of losing high metabolic heat owing to rapid metabolism their thick feather and sweat glands in the body (Nawaz *et al.*, 2021). It was found that newly developed breeds are more prone to heat stress than the earlier genotypes of broiler (Zhang *et al.*, 2012). With intensive poultry production conjugated with the hotter and humid climate, animals are prone to heat stress affecting their overall performance. If the birds are incapable of balancing body heat production and loss, they are heat stressed.

Heat stress and Meat quality

There is evidence that heat stress could affect the meat quality. For example, it could increase fat deposition and reduce the protein content of meat (De Antonio *et al.*, 2017). Similarly, it also could increase the occurrence of pale, soft, exudative (PSE) meat conditions (Janisch *et al.*, 2011; Vandana *et al.*, 2021). In the next section, some important carcass traits affected by heat stress are briefly discussed.

pH value

The pH value of meat is a direct measurement of muscle acid content, and which is linked to meat tenderness, colour, shelf life and water holding capacity (Janisch *et al.*, 2011). The pH of living muscle tissue is almost neutral (7.1 to 7.2). Post-slaughter, glycogen is a primary source of energy leading to the formation of lactic acid. Thus, produced lactic acid lowers the pH value of meat until cells are deprived of glycogen, and the ultimate pH is reached (Pighin *et al.*, 2014; Zaboli *et al.*, 2019). The variation in muscle pH is closely associated with glycogen reserves and the conversion ratio of glycogen to lactic acid in the muscle tissue (Wang *et al.*, 2009). The adverse effects of heat stress are prominently noticeable in adult birds. Lu *et al.*, (2017) conducted experiment where birds were subjected to chronic heat stress (32 °C for 14 days), showed significantly lower meat pH after 45 min and 24 h of slaughter, but did not show any pH change after 7 days of heat exposure. Another experiment revealed a decrease in pH when one-day-old broilers

male Ross 308 were exposed to heat stress (32 ± 1 °C from 9:00 AM to 5:00 PM) during the finisher period (Baghban Kanani *et al.*, 2017). Similarly, Zhang *et al.*, (2012) conducted an experiment on male broiler chickens exposed to cyclic heat stress (36 °C from 10:00-16:00, 23 °C from 16:00-10:00) and constant heat stress (34 °C). They demonstrated low initial and final pH in breast and thigh meat in birds under constant heat stress whereas cyclic heat stress reduced only breast meat initial and final pH. Additionally, Tang *et al.*, (2013), who reared one-day-old male broiler chickens at 35 °C during the first week followed by 3 °C reduction per week to reach 22 °C at d 30 and then reach 38 °C. After 1 and 2 hours of heat stress exposure, the level of both immediate pH (pHi) and ultimate pH (pHu) in the pectoralis of heat-stressed chickens was lower than that of control. Pre-slaughter heat stress results in elevated secretion of catecholamine, hasten the breakdown of glycogen, declined pH and reduced meat quality (Bozzo *et al.*, 2018). Pre-slaughter acute heat stress exposure, particularly in the broiler, provokes adequate adrenaline release and exerts a detrimental effect on meat quality (Lowe *et al.*, 2002).

On the contrary, Peña *et al.*, (2008) did not find any variation in pH among the birds reared at cyclic heat stress (32 °C for 5 h per day for 19 d). The following result is in harmony with another independent test carried out by Goo *et al.*, (2019), which reveals all pH values within a standard range of general meat quality. In addition, Liu *et al.*, (2019) also found no significant change in pH of breast and thigh muscles following 45 minutes and 24 hours post-slaughter. An experiment by Zeferino *et al.*, (2016) showed the increase in the meat pH value 24 hours post-slaughter, when birds are subjected to heat stress (32 °C for 15 days) over the finisher period. The higher ultimate pH in meat may be accredited to pre-slaughter exhaustion of glycogen reserve and the lowest residual glucose level following the stressful condition (Bray *et al.* 1989). These inconsistent results may be attributed to the differences in breed, slaughtering age, experimental condition, and adaptive capacity of birds, or exposure of birds to heat stress may not be enough to make a significant change in meat quality.

Meat color (lightness, redness and yellowness)

Meat color is one of the decisive factors for determining meat quality, which is interrelated to shear force, pH, and water holding capacity (Ružic *et al.*, 2020). Myoglobin, along with cytochrome C and hemoglobin are responsible for exhibiting natural meat color, (Mancini and Hunt, 2005). The meat color is also affected by the degree of oxidation of the myoglobin. Upon exposure to oxygen, pink-color myoglobin (Mb) is oxidized to a red-color

intermediate product known as oxy-myoglobin (MbO_2), which finally yields dull-brown met-myoglobin (Met-Hb). The iron atom present in fresh meat is in a ferrous state (Fe^{++}), and upon oxidation, it is converted to a ferric state (Fe^{+++}). The pigments concentration and their oxidation state are influential factors for altering meat color (Brewer, 2004; Mancini and Hunt, 2005; Suman *et al.*, 2016). The myoglobin concentration is lower in chicken breast meat than in chicken thigh meat, thus imparting lighter color to breast meat. Additionally, feed grains such as corn and wheat contain numerous pigments responsible for determining broiler meat color (Smith *et al.*, 2002).

Exposures of poultry to pre-slaughter heat stress significantly influence the meat color. Studies have shown that heat-stressed chicken meat exhibits an increase in lightness and yellowness as well as a decrease in redness level (Suman *et al.*, 2016). Peña *et al.*, (2008) reported standard meat quality with normal lightness (L^*), redness (a^*), and yellowness (b^*) values within the usual range. In this study, the meat sample obtained after freezing yields yellowish and dark red color meat. Zeferino *et al.*, (2016) and Lu *et al.*, (2017) in two independent experiments revealed a significant increase in L^* but decreased in a^* without affecting yellowness. Increased L^* as well as reduced a^* was associated with depressed feed intake due to heat stress. Other numerous studies such as Lu *et al.*, (2007) have revealed increased meat lightness, as an effect of chronic heat stress, along with the inconsistent result on yellowness and redness. Zhang *et al.*, (2012) stated unchanged yellowness and decreased redness under chronic heat stress, but Lu *et al.*, (2007) could not detect any changes in either of the color measurement in Arbor Acres breast muscle under constant heat stress conditions. The decreased redness value in meat indicated the presence of a surplus amount of oxidized myoglobin in heat-stressed birds (Mancini and Hunt, 2005). Baghban Kanani *et al.*, (2017) under heat stress conditions documented an increase in L^* but a^* and b^* are unaffected. Similar experiments by Petracci *et al.*, (2004), in contrast, improved lightness but reduced yellowness and redness in breast muscle. The development of pallor meat in the experiment is related to a drop in pH due to the anaerobic glycolysis under heat stress conditions yielding PSE meat. Wang *et al.*, (2009) acknowledged that lightness and pH are negatively correlated. Petracci *et al.*, (2004) explained the development of yellowness in meat is associated with the production of reactive oxygen species (ROS) and lipid peroxidation. Imik *et al.*, (2012) showed a significant effect on the L^* , a^* and b^* values of chicken drumsticks and breast meat such that L^* value increased, a^* value fluctuated with decreasing trend and b^* value also fluctuated when breast meat was stored for 8 days. In the

synchronization to these results, Petracci and Fletcher (2002) documented the effect of storage on color parameters (L^* , a^* , and b^*) in breast meat of chicken, such that lightness increases with prolonged storage of 8 days, a^* value follow decreasing trend along with fluctuated b^* value. These findings established that meat quality and storage time are inversely proportional to each other as well as heat stress exerts negative and detrimental effects on meat color parameters. In an experiment by Goo *et al.*, (2019), heat stress had no significant impact on meat color. The breast muscle meat quality had standard color with no significant impact, when broiler are subjected to heat stress conditions.

Cooking loss (%)

In general terms, the cooking loss is the loss of moisture during the cooking of meat. Cooking loss is an excellent index for moisture and fat loss due to protein denaturation and membrane disintegration when heated (Cheng and Sun, 2008). There are three different forms of muscle water content: a low percentage of bound water, free water, and the highest rate of immobilized water (Bowker and Zhuang, 2013). Except for the bound water, other types of water could be driven out either by lower pH value or by conventional cooking/heating, resulting in myofibril shrinkage and considerable water loss (Honikel, 1998).

Muscle constitutes a wide variety of proteins, categorized mainly as connective tissues, sarcoplasmic and myofibrillar proteins. These proteins are prone to denaturation during heating. The shrinkage in meat occurs, followed by fibrillar protein denaturation due to oozing of meat water yielding firmer meat with low moisture under higher temperatures (Tornberg, 2005; King and Whyte, 2006). Cooking meat under specific conditions (time and temperature) develops some characteristics such as color, flavor, odor, and appearance that increase palatability and acceptability for consumers (Domínguez *et al.*, 2015). Apart from killing pathogenic microbes, the cooking method should hold two basic parameters: higher water holding capacity (water retention) and sensory attributes like juiciness, flavor, and tenderness should be within an acceptable range (Chiavaro *et al.*, 2009).

Most of the experiments conducted in different settings had acknowledged the similar effect of heat stress on the cooking loss. These experiments documented the considerable rise in cooking loss under heat stress conditions. An investigation by Zeferino *et al.* (2016) found a significant increase in cooking loss when birds were subjected to heat stress conditions. The results were concurrent with the findings of Lu *et al.*, (2017), who also found a significant increasing trend of drip loss in the first week and significantly higher cooking loss in the

second week of the heat stress period. Similar findings by Zhang *et al.* (2012), demonstrated a greater cooking loss in breast meat and thigh meat of male broiler chicken under constant heat stress of 34 °C). The results are credited as a result of prominent protein denaturation under heat stress conditions, thereby reducing the water-binding capacity of meat. Surprisingly, Liu *et al.*, (2019) did not notice any significant effect on cooking loss of breast and leg muscle in heat-stressed birds, which may be due to the difference in experiment setting and duration as well as the extent of heat stress, incapable of producing the expected results.

Water holding capacity (%)

Water-holding capacity is linked to the softness and flavor, color, nutrient content, and fragrance of broiler meat (Hu *et al.*, 2016). Palatability and consumer acceptability are increased for the meat with higher water holding capacity and low shear force (good tenderness). Water holding capacity (WHC) is the competence of fresh meat to withhold the moisture and determine both the quantity and quality of yield and subsequently affect the economic worth of products (Cheng and Sun, 2008; Huff Lonergan *et al.*, 2010). Broiler meat possesses poor water holding capacity regardless of heat stress. Hashizawa *et al.* (2013) reported poor WHC in the broiler meat kept under heat stress conditions (Hashizawa *et al.*, 2013). Similarly, another study showed a reduction in the water holding capacity when broilers were raised under heat stress conditions (T: 34 ± 1°C, RH: 65%–75%) (Hu *et al.*, 2016). Exposure of broiler to heat stress steered the alteration in pH value, thereby producing the PSE meat. Wilhelm *et al.*, (2010) claimed that the PSE meat maintains poor WHC (Wilhelm *et al.*, 2010). In contrast, Peña *et al.* (2008) showed a higher WHC for heat-stressed broiler meat.

Shear force (Newton)

Shear force value (SFV) is an indicator of the meat stiffness. Lyon *et al.* (2004) showed that shear force value is the key determinant of the meat tenderness. Alteration in shear force caused by heat stress could be perceptible by individuals (Lyon *et al.*, 2004). More tenderness is a sign of superior meat quality, and consumers shows great interest in tender meat with a high. Experiments conducted by Lu *et al.* (2017) and Hashizawa *et al.* (2013) showed a significant drop in SFV of meat under heat stress conditions. The lower SFV obtained in those trials could be connected to alteration observed in the centesimal and chemical conformation of muscle, which varies with bird genetic makeup and age. Breakdown of sarcomere structure and decline in SFV occur in birds subjected to a pre-slaughter heat stress, (Wilhelm *et al.*, 2010; Hashizawa *et al.*, 2013).

Furthermore, a trial performed by Zhang *et al.* (2012) reported a significantly higher shear force value in the meat of heat-stressed broilers. Also, Zeferino *et al.* (2016) did not notice any change in SFV.

Growth performance and carcass quality

Heat stress has detrimental effects on the productivity of chickens. In other words, it is responsible for the greater secretion of the stress hormone, which has a deleterious impact on the growth performance through undesirable alteration in nutrient metabolism in the small intestine leading to economic loss (Wang *et al.*, 2021). Also, the negative effects of high ambient temperature on birds include low slaughter weight, heavy mortality, and ultimately reduced meat quality and meat productivity of broiler chicken (Zhang *et al.*, 2012).

Growth performance

High ambient environmental temperature is a significant threat for poultry enterprises due to its negative consequences on meat quality, feed consumption, and body weight gain (Akşit *et al.*, 2006). Animal attempts to get acclimatized and adapted to heat stress via metabolism alteration. Utilization of feedstuff promptly got diverted into survival and health in the heat stress condition. Heat-stressed broilers get adapt to heat stress at the expense of productive energy causing an energy deficit for growth (Nawab *et al.*, 2018).

Average daily gain and average daily feed intake were significantly lower in heat-stressed broilers than that in thermoneutral conditions (Wang *et al.*, 2021). Zeferino *et al.*, (2016) showed the decrease of daily weight gain (by 32%) and feed intake (by 20%), and increase of feed conversion ratio (by 16%) in heat-stressed broiler chickens. The results were in agreement with Liu *et al.*, (2019), Harsini *et al.*, (2012) and Habibian *et al.*, (2016), who found a decrease in daily weight gain and feed intake, and an increase in feed conversion ratio. The lower daily weight gain was the consequence of a decrease in feed intake, whereas a drop in feed intake was taken to diminish the metabolic heat increment (Song *et al.*, 2013) under the heat stress condition. Similarly, a reduction in daily weight gain and feed intake was demonstrated in the experiment by (Goo *et al.*, 2019) and Imik *et al.*, (2012). The effects of heat stress were noticed largely in male broilers in later experiments due to its rapid growth rate in comparison to females. Likewise, Toghiani *et al.*, (2012) revealed a decrease in feed intake in heat-stressed broilers. The results resonate with the findings of Dai *et al.*, (2009), who had presented a significant decline in feed intake and body weight gain by 28% and 8%, respectively.

However, Peña *et al.*, (2008) investigated no significant differences in live body weight gain, and feed intake, which may be due to the adaptation of

birds to heat stress such that heat stress effects are more pronounced during the early stages of acclimatization and effects are unnoticed at later stages.

Slaughter and Carcass characteristics

In the case of fast-growing commercial broiler chickens, the average slaughter weight ranges from 2.5 to 3.0 kg, reaching within four to seven weeks of age. The decline in live body weight and carcass yield was observed under heat-stressed conditions (Toghyani *et al.*, 2012). The decline in the weight of the birds occurs as a result of a drastic drop in the feed intake of broilers exposed to a heat stress, in an attempt to minimize metabolic heat production and to maintain homeothermy (Ryder *et al.*, 2004). Similarly, Imik *et al.* (2012) and Liu *et al.* (2019) displayed the decline in carcass yield of heat-stressed birds in two different experimental results. The results were reinforced by Lu *et al.* (2018) report in which the undesirable effect of heat stress on carcass traits was confirmed. In contrast, Goo *et al.* (2019) found no significant differences in live body weight. Similarly, Habibian *et al.* (2016) and Lu *et al.* (2007) observed no significant effect of cyclic heat stress in carcass weight of broiler, and are supported by result of Two different experiments conducted by Zeferino *et al.* (2016) and Zhang *et al.* (2012), showed differences in breast and thigh muscle weight under chronic heat stress conditions. Zeferino *et al.* (2016) observed a decrease in the weight of the breast, whereas thigh weight remained unchanged, while Zhang *et al.* (2012) reported a reduction in breast muscle weight and an increase in thigh muscle (Zhang *et al.*, 2012; Zeferino *et al.*, 2016). Other findings also supported the earlier findings of Zhang *et al.* (2012) who showed an increase in thigh muscle and a decrease in breast muscle weight (Liu *et al.*, 2019). The weight variation between the thigh and breast muscle could be due to the difference in myofibril metabolism between these muscles. Breast muscle contains a plentiful amount of white fast contracting glycolytic fiber, in which metabolism and hypertrophy rely upon the supply of glycogen, whereas in the leg, red slow contracting oxidative fiber is abundant (Ismail and Joo, 2017). Decline in feed consumption results in depletion of glycogen sources, and diminished protein synthesis in breast muscle (Temim *et al.*, 2000). In Zeferino *et al.*, (2016) and Zhang *et al.* (2012) experiments, after a feed restriction period, the relative weight of proventriculus, heart, and liver were reduced in heat-stressed birds which was regarded as a physiological adjustment.

Heat stress on glucose metabolism

Pyruvate, Lactic acid, and fat content

Under the aerobic condition, muscle glycogen enters

into glycolysis and yields two pyruvate molecules as an end-product. After the slaughter of animals, the circulatory system collapse leading to a deprived oxygen supply. Under anaerobic conditions, pyruvate undergoes lactic acid fermentation yielding two lactic acids, which ionize to form lactate at the expense of 2 molecules of NADH. This reduction of pyruvate to lactic acid is essential for transforming muscle to meat (Pösö and Puolanne, 2005; Huff-Lonergan, 2009). The study by Lu *et al.* (2017) found no significant differences in lactic acid production during the first week of heat stress but increased in the second week (Lu *et al.*, 2017). Birds under chronic heat stress revealed the elevated mRNA expression of PDHK-4, LDH activity, and depressed CS activity that lead to a reduced supply of glucose into Krebs's cycle, and cells depend upon anaerobic glycolysis. Furthermore, the breast meat of chronic heat-stressed broiler chickens had more pyruvate transformed into lactate, with the subsequent decline in muscle pH, thus spoiling meat quality (Song and King, 2015). Under constant heat stress, higher lactic acid content could be found in the breast muscle while diurnal cyclic heat stress results in higher pyruvate content. Similarly, in the thigh muscle, pyruvate and lactic acid content are increased under constant heat stress and no significant difference was observed in diurnal high temperature (Zhang *et al.*, 2012). Post slaughtering of the animal, the collapse of the circulatory system leads to deficient of oxygen and nutrient to muscle such that spare glycogen and glucose undergoes anaerobic degradation, resulting in excessive production of lactic acid (Pösö and Puolanne, 2005). In the following trial, the higher lactic acid content in heat stress birds suggested that the higher amount of anaerobic degradation of glycogen occurred during the conversion of muscle to meat.

Environment is recognized as one of the crucial and influencing factors for the deposition of fat in birds and incidence of higher intramuscular, abdominal, and cutaneous fat deposition is associated with chronic exposure to heat stress (De Antonio *et al.*, 2017; Lu *et al.*, 2019). Furthermore, an experiment by Lu *et al.* (2017) revealed the chronic heat stress in broiler causes a significantly higher intramuscular fat deposit in breast muscle which was associated with an increase in mRNA expression of acetyl Co-A carboxylase (ACC) and fatty acid synthase (FAS). ACC and FAS are two key enzymes associated with the metabolism of fat (Lu *et al.*, 2017). Chronic heat-stressed chicken had low protein but high fat deposition in skeletal muscle (Zhang *et al.*, 2012; Zuo *et al.*, 2015). Similarly, the enhanced fat deposit could also be associated with reducing physical activity and basal metabolism. The elevation of abdominal fat deposits may be an adaptive mechanism to heat stress conditions. The excess

nutritional energy deposited as fat decrease the incremental heat, and thus, a smaller amount of heat is lost (Dai *et al.*, 2009). However, the enhanced abdominal fat deposition in broilers is unproductive in the poultry industry and hold significant loss in consumer acceptability and market value. In conflict with the result, Zeferino *et al.* (2016) presented a lower abdominal fat percentage in heat-stressed broilers, probably due to partial feed restriction imposed upon them (Zeferino *et al.*, 2016). The variations in results could be linked to the model of heat stress (cyclic or constant), age of the animal, chicken breed, and the technique used to calculate the fat index.

Metabolic enzyme activity

Lactate dehydrogenase (LDH), Pyruvate kinase (PK), Citrate synthase (CS), and Hexokinase (HK) are the major metabolic enzymes that significantly affect glucose metabolism. Lu *et al.* (2017) found the different levels of LDH, CS, PK activity for the heat-stressed birds (Lu *et al.*, 2017). In the first week of heat stress, no differences were noticed in LDH and PK activities, but CS activity was significantly increased. Similarly, in the second week, the activity of LDH and PK were substantially higher, but CS activity was lower. Similarly, in the second week, the activity of LDH and PK were significantly higher but CS activity was lower. Similarly, another experiment, (Zhang *et al.*, 2012) reported that, in constant high or diurnal cyclic heat stress conditions, the activity of LDH and PK was more elevated in breast muscle, whereas the thigh muscle showed lower LDH activity. Also, the activity of HK did not have significant differences in breast muscle at constant high temperature, whereas HK activity was higher in the thigh of constant high or diurnal cyclic heat-stressed broiler. The higher activity of LDH indicates more lactate production, a drop in ultimate pH, and the production of pale, soft, exudative meat. Similarly, the higher HK activity indicates the efficient glucose mobilization and feeding of more glucose substrate to the glycolytic process. Likewise, the muscle of broilers subjected to heat stress revealed higher PK activity indicative of enormous pyruvate production for providing energy.

Heat stress on antioxidant activity

During the heat stress condition, there is prompt production of reactive oxygen species, disturbing the physiological and biochemical mechanism occurring in the cell (Mujahid *et al.*, 2007; Slimen *et al.*, 2014). A surge in ROS production occurs due to an impaired electron transport chain within the membrane. Elevated ROS production or lower quenching capacity of antioxidant elements disrupt the equilibrium between oxidant and antioxidant defence mechanisms, thereby causing lipid peroxidation, and

oxidative damage to DNA and protein (Tepe *et al.*, 2006; Marí *et al.*, 2010). Mitochondria, play an essential role in energy metabolism, including substrate oxidation and oxidative phosphorylation. When an animal is exposed to heat stress, oxidative stress is induced, causing surplus production of reactive oxygen species, which leads to mitochondrial dysfunction (Rhoads *et al.*, 2013; Montgomery and Turner, 2015; Slimen *et al.*, 2016). Damaged and malfunctioning mitochondria, as the direct effect of heat stress, resulting in an adjustment in energy substance metabolism, lead to reduced oxidative degradation of carbohydrates and lipids (Montgomery and Turner, 2015). The resultant alterations in energy substance metabolism significantly impact meat quality. Similarly, heat stress results in producing free radicals (Mujahid *et al.*, 2009; Azad *et al.*, 2010). Free radicals exert negative influences by intensifying the adenosine monophosphate-activated protein kinase (AMPK) activity in myofibril (Wang *et al.*, 2009) and lipid peroxidation (Imik *et al.*, 2010). One of the vital factors, which affect the meat organoleptic characteristics, is lipid oxidation. The undesirable changes in texture, odor, flavor, and nutritive value occurs due to lipid oxidation (Love and Pearson, 1971; Demir *et al.*, 2022).

It is well established that antioxidants are responsible for quenching free radicals and reactive oxygen species produced inside cells (Lanari *et al.*, 2004). Upon exposure to a heat stress, the potentiality of antioxidant activity is impaired and causes regression in in-vivo antioxidant status (Sahin *et al.*, 2002). Hyperthermia decreases the plasma concentration of antioxidants such as vitamins A, E, and C, folic acid, and minerals like selenium, iron, and zinc (Harsini *et al.*, 2012). The body develops various enzymatic and non-enzymatic antioxidant defense systems against detrimental effects of reactive oxygen species (Irato and Santovito, 2021). The enzymatic antioxidant defense mechanisms are delivered by glutathione peroxidase, superoxide dismutase, glutathione reductase, and catalase in synchronization with copper, magnesium, selenium, and zinc. Similarly, direct free scavengers like vitamin A, vitamin C, vitamin E, and β -Carotene provide the non-enzymatic antioxidant defense mechanism to the organism (Vaisi-Raygani *et al.*, 2007; Irato and Santovito, 2021).

Furthermore, when the animal is subjected to high ambient temperature, lipid oxidation occurs that yielding soluble products known as malondialdehyde (MDA) or Thiobarbituric acid reactive substance (TBARS). The production rates of MDA / TBARS directly correspond with the lipid peroxidation rate and indirectly with the level of cell injury (Gan *et al.*, 2014). Higher heat stress, higher the oxidative stress, higher the production of TBARS, and greater the deterioration of meat quality. Various experiments

conducted at different times show multiple results, which exhibit a significant increase in the TBARS value. Broiler imperilled to heat stress condition displayed a 1 to 4 fold increment in TBARS/MDA value and decline in selenium content (Wang *et al.*, 2009; Azad *et al.*, 2010). Furthermore, Harsini *et al.* (2012) also investigated a significant rise in MDA content in skeletal muscle of heat-stressed chickens (Harsini *et al.*, 2012). The following results are attributed to the impaired muscle membrane integrity (Sandercock *et al.*, 2001) due to high ambient temperature, leading to high lipid peroxidation rate in skeletal muscle (Wang *et al.*, 2009). The factors influencing the production rate of TBARS/MDA are

storage temperature and storage duration (Kim *et al.*, 2010). The MDA/TBARS level of serum concentration truly picturizes the in vivo antioxidant capacity of the body.

Strategies to mitigate heat stress

Understanding the roles of various supplements and changing feeds seem to be potential ways to reduce the adverse effects of heat stress in chickens. Numerous management strategies (Figure 1) are outlined to mitigate the effect of heat stress on broiler chickens (Renaudeau *et al.*, 2012; Saeed *et al.*, 2019; Shakeri *et al.*, 2020; Goel, 2021; Khan *et al.*, 2021).

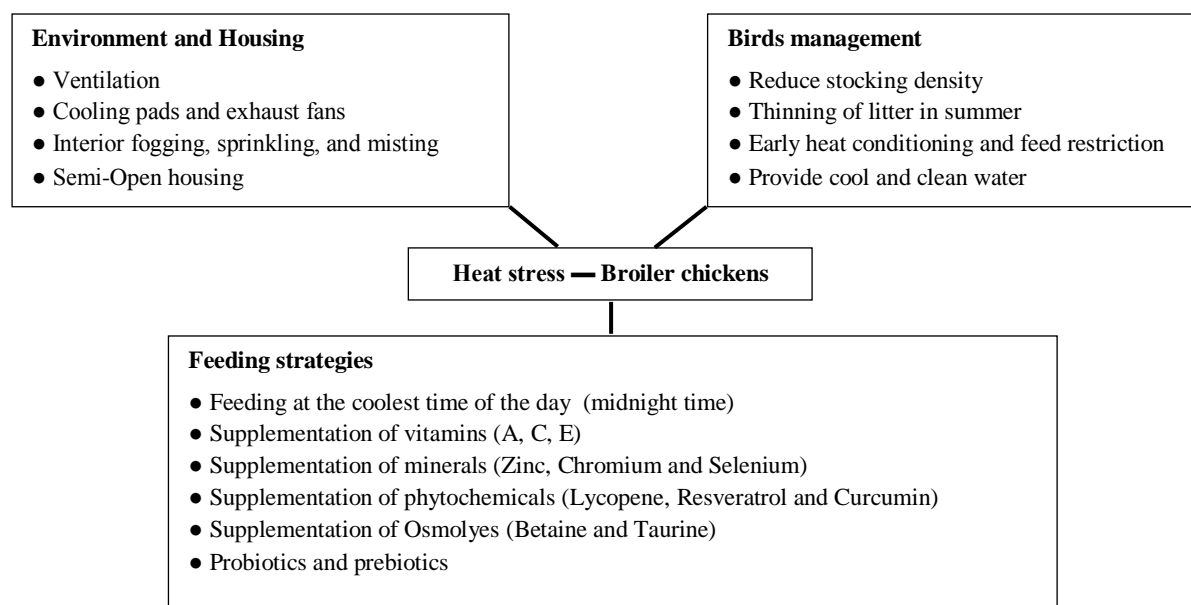


Figure 1. Methods to mitigate heat stress in broilers

In brief, several micronutrients, including vitamins and minerals supplements improve growth performance in broiler chickens subjected to acute heat stress through diverse routes (Shakeri *et al.*, 2020). Similarly, East-west oriented, open style, naturally ventilated poultry buildings with exhaust fans, cooling pads, or air conditioning have been recommended in the tropics, but they are expensive (Saeed *et al.*, 2019; Wasti *et al.*, 2020; Goel, 2021). Water sprinkling is also recommended in chicken buildings to reduce heat load (Khan *et al.*, 2021). In addition, thermal conditioning can be utilized to improve heat tolerance in chickens at an early age (Goel, 2021). Supplementation of herbal and phytochemicals, sodium carbonate, probiotics and prebiotics, and electrolyte along with the use of fats and synthetic ration can help to mitigate heat stress in broilers (Saeed *et al.*, 2019).

Conclusion

Environmental factors, particularly high ambient

temperature, play an essential role in influencing the birds' meat quality, carcass traits, and growth performance. Exposure to hyperthermia negatively impacts growth performances and carcass traits, thereby spoiling the meat quality. Negative influences of heat stress on the performance and productivity significantly impact the market value of raw and processed meat products. Therefore, minimizing the indoor high ambient temperature to the thermoneutral temperature is only the cost-effective effort that producers can introduce to reduce the detrimental effects of heat stress on the productivity of the animals. The effects of heat stress conditions should be assessed as early as possible to formulate a strategic plan for reducing the stress conditions; else, it will result in lethal, life-threatening diseases. Formulation of the feedstuffs enriched with antioxidants, proper ventilation, sprinkling water over the shed, appropriate housing with the installation of the fans and air conditioner, good stocking density, and proper handling during transportation are

effective countermeasures for reducing the heat stress condition.

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