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HEAT STRESS IMPACTS ON BROILER PERFORMANCE

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Summary

Many research have examined the negative effects of heat stress on poultry productivity since environmental stressors are commonplace globally. It has been demonstrated that heat stress has a deleterious impact on the productivity and well-being of laying hens and broilers. To better understand the fundamental mechanisms underlying the detrimental effects of heat stress on chickens and to create efficient therapies, more research is still necessary. Because of the recent detrimental effects of heat stress (HS), the most difficult environmental stressor on birds, there has been much concern over the decades-long increase in poultry production to meet the rapidly rising demand and ensure food security. In response, the chicken business has implemented a variety of environmental initiatives, involving making use of modern ventilation systems and ecologically controlled sheds. But these are not sustainable solutions, and they are extremely costly for landowners to implement. The negative consequences of HS include stunted growth, deteriorating meat quality due to decreased water-holding capacity, increased pH, and increased drip loss in meat, which alters the typical color, flavor, and texture of chicken meat. By reducing protein synthesis and increasing unwanted fat in meat, HS lowers the level of quality of meat. Previous research indicates that HS alters the effects of heat-shock proteins, insulin growth factor-1, and myogenic regulatory variables, therefore affecting the growth and development of skeletal muscle. This article concentrates on three primary areas: (1) determining the mechanism of heat stress that leads to the loss of quality and meat production in chickens; (2) discussing the physiological, metabolic, and genetic changes caused by HS that cause the global poultry industry to suffer; and (3) identifying research gaps that need to be filled in future studies. One of the most difficult environmental conditions that negatively affect the poultry industry worldwide is heat stress. The main reason broiler chicken strains are susceptible to heat anxiety is due to the fact that they lack sweat glands. The goal of the current study was to find out how heat stress affected the growth performance and bio-physiological traits of Cobb, Hubbard, and Arbor Acres broiler hybrids during the summer, when Egypt's environmental conditions were revealed. Three hundred and one day old birds, or one hundred from each hybrid, were fostered in identical circumstances with respect to water, food, and other factors on the same day. Vaccinations, medications, and breeding practices were utilized from the beginning to the moment of killing birds. The three strains—"Cobb, Hubbard, and Arboretum Acres"—were split into twelve groups by chance. Each group received two treatments—"control group and heat exposed group"—two duplicates, and twenty-five chicks. The control group experienced an environmental temperature of 32°±2 Celsius degree and a relative humidity of 50±5 percentage during housing, and the heat stressed group experienced an environmental temperature of 40°±2 Celsius degree and a relative humidity of 20±5 percentage. The weight of the body, weight gain, and edible and inedible portions of the carcass (carcass, thigh, drum, breast muscles, and giblets) and the weight of the head, legs, blood, and other inedible parts of the carcass were recorded for the heat-stressed group and the control group. The weight of lymphatic organs like the spleen, thymus, and bursa was also measured. The Cobb strain demonstrated superior growth performance and carcass characteristics under heat, according to our most recent findings. Stress situation, but the Arbor Acres strain is thought to be the strain to choose because it didn't significantly impact the user's rectal temperature like other strains that were exposed to heat. The most viable strain is the Arbor Acres strain in both the treated and control groups. In comparison to the group exposed to heat and other strains, the bursa weight of the control group and the Hubbard strain increased. In comparison to various varieties of broiler chicken, it was determined that the Cobb strain performed most effectively under heat stress.

Keywords: Heat Stress (Hs), Birds, Poultry, Chicken, Strains, Quality.

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INTRODUCTION

Animals with lightning-fast development have been produced by technological advances with the objective of improving efficiency. Nevertheless, cardiorespiratory development does not fully coincide with these developments. Due to increased body mass and metabolic rates, the outcome is a rise in body temperature and metabolic heat generation(Borges et al., 2003), especially during the growing and finishing periods when the mechanism of losing heat is slowed down(Laganá et al., 2007)

A significant amount of studies on the topic indicates that heat stress impairs broiler performance. In order to maintain homeostasis, broilers under heat stress reduce their feed intake; nevertheless, this adjustment leads to a loss in body weight gain and an increase in feed conversion(Baziz et al., 1996). A number of authors have shown that feed conversion improves when dietary fat is raised during heat stress(Dale and Fuller, 1980, Bonnet et al., 1997) and when vitamins (E and C) and minerals (Zn and Se) are supplemented(Laganá et al., 2007). However, due to the animals' reduced intake and slower pace of weight gain,(Simas, 2010) did not show any variations in broiler feed conversion at 31°C compared to thermoneutral situations. It also matters what kind of heat stress you have. Broilers are subjected to elevated temperatures on a regular basis, resulting in heat stress. Broilers are more suited to handling the diurnal environmental temperature variability that occurs during cyclic heat stress (Ribeiro et al., 2001, Borges et al., 2003, de Souza et al., 2016). (Piestun et al., 2011). Studies attempting to simulate these differences are scarce, notwithstanding considering that the harm resulting from heat stress has been previously shown.

Additional dietary approaches related to electrolytic balance are recommended to preserve homeostasis and lessen the degradation brought on by elevated temperatures(Borges et al., 2003). (Mongin, 1981) identified 250 mEq/kg of diet as the optimum amount needed for preserving livestock's acid-base balance.

A meta-analysis tool, which aims to summarize and quantify studies previously conducted for the construction of new hypotheses, emerges as an alternative in light of the increasing number of publications, in addition to the challenges concerning ethical concerns related to the use of creatures for in vivo experiments (Sauvant et al., 2008). Thus, the current study sought to simulate the performance of broilers exposed to heat stress during both the first and last stages of their growth, ascertain the impact of the type of heat stress on performance, and evaluate the relationship between heat stress and the electrolytic balance of the broiler diet.

Because stress is a subjective experience, it can be challenging to identify and comprehend as a reaction to negative stimuli. "Stress is the nonspecific response of the body to any demand," according to (Selye, 1976), while "a stressor is an agent that produces stress at any time." Consequently, stress is an animal organism's biological response—that is, its reaction to a stimulus that upsets its normal physiological equilibrium, or homeostasis.

A negative balance between the net energy leaving the animal's body and entering its surroundings and the energy the animal produces as heat causes heat stress in animals. Variations in a range of natural elements, such as sunshine, thermal irradiation, air temperature,

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humidity, and movement, as well as animal features, such as species, metabolism rate, and thermoregulatory processes, could be the source of this imbalance. Environmental stresses that can be particularly harmful to animal agriculture include heat stress (Nienaber and Hahn, 2007, Nardone et al., 2010, Renaudeau et al., 2012) .Because of growing public understanding and issues, the issue of stress from the environment in animal husbandry gets quite a bit of discussion.

All species benefit from understanding how animals react to external stressors. But chickens appear to be especially vulnerable to external issues caused by temperature, notably heat stress. It has been proposed that the increased metabolic activity of contemporary poultry genotypes results in the creation of more body heat (Settar et al., 1999, Deeb and Cahaner, 2002). Understanding and managing environmental factors is essential for proficient poultry farming and well-being. So the objective of this review is to gather the most recent information and data from the scientific (peer-reviewed) literature about the significance and effects of heat stress on chicken productivity, with a particular emphasis on broilers and laying hens.

The world's temperature is currently increasing significantly and this has major consequences for the agricultural industry in the tropical and subtropical regions of the world. All living things are impacted by a steady rise in the outside temperature (Nardone et al., 2010, Sejian et al., 2018) When the temperature of a living thing rises above its regular range (the thermoneutral zone), it disrupts normal physiological processes and causes cell damage. High outside temperatures typically cause stress-related issues such as decreased productivity, altered metabolism, growth depression, and inefficient operation (Afsal et al., 2018, Renaudeau et al., 2012). Heat stress (HS) is commonly divided into two categories: chronic heat stress (CHS), which is defined as high temperatures over an extended length of time, and acute heat stress (AHS), which is intense ambient temperatures for a brief period of time. Unfortunately, AHS and CHS pose a serious threat to the biological, nutritional, medical, and organizational improvements developed by the animal cultivation industries. As a result, production declines significantly, and they stand in the way of effective livestock farming in many parts of the world(Lara and Rostagno, 2013, Pawar et al., 2016b). In contrast to acute stress caused by heat, chronic heat stress impacts barbecue chicken irreversibly by raising its fat content and weakening its muscle mass over an extended period of time. In addition to the length of heat stress, the degree of heat stress influences the extent to which productivity degradation occurs (Adu-Asiamah et al., 2021). The detrimental effects of heat exhaustion, which occur when an animal's body temperature rises beyond the thermoneutral zone and it loses its ability to control itself, may become increasingly difficult to treat in animal husbandry as a result of global warming. These days, the effects of climate change brought on by global warming are becoming more significant, particularly for the chicken meat sector (Abd El-Hack et al., 2020, Gregory, 2010). The problem of strong growth and fast metabolism of chickens makes them more susceptible to extreme temperatures, driving up production costs and negatively affecting meat quality in the grill business. In poultry raised in a highly stressful environment, especially broiler chicken metabolic alterations may develop. Resulting in a significant reduction in the grilled chicken's breast muscle size. The reduction in skeletal protein levels is also caused by HS(Zhang et al., 2012). A change in color, a drop in muscle pH, a reduction in water-holding

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capacity (WHC), and a reduction in the flavor of chicken meat are just a few of the major issues that could arise from both AHS and CHS producing a fast deterioration in the metabolic rate of chickens(Gonzalez-Rivas et al., 2020, Song and King, 2015). Numerous studies have demonstrated that the production of reactive oxygen species (ROS) at high ambient temperatures leads to oxidative stress. Since ROS cause lipid peroxidation in muscles, they have a serious impact on the growth of muscle tissue (Altan et al., 2003, Kumar et al., 2012) Therefore, resolving global food insecurity issues may benefit from an understanding of the mechanisms behind, causes, and impacts of HS in addition to the techniques that could be employed to curb or control such a worldwide menace. This review went deeply into the analysis of the information that was provided regarding the impact of HS and the methods to reduce the unintended consequences of this hazard.

Behavioral and Physiological Effects of Heat Stress on broiler

Birds that experience high temperatures modify their behavior and physiological equilibrium in an effort to find thermoregulation, which lowers body temperature. Most bird species respond to heat stress in a similar way, while there may be some individual differences in the strength and duration of these reactions. According to a recent study(Mack et al., 2013), birds under heat stress spend more time resting, less time moving or walking, more time with their wings raised, and less time feeding. They also spend more time drinking and panting.

When exposed to high external temperatures, animals use a variety of strategies to maintain thermoregulation and a state of home which includes enhancing radiative, convective, and evaporative heat loss by vasodilatation and sweating (Mutaf et al., 2009). The air pockets in birds provide a further means of facilitating heat exchange between their bodies and their surroundings. When panting, air sacs come in quite handy because they stimulate gas exchanges with the air and, as a result, the evaporative loss of heat by promoting air circulation on surfaces (Fedde, 1998). It is critical to remember, though, that increased panting under heat stress raises blood pH and carbon dioxide levels (alkalosis), which hinders the availability of blood bicarbonate for the calcification of eggshells and causes an increase in the availability of organic acids lowering blood levels of free calcium as well. Because it has an impact on the quality of the eggshell, this procedure is crucial for breeders and laying hens (Marder and Arad, 1989). Nevertheless, despite the fact that numerous studies have tried to define the physiological mechanisms linked to the decline in egg quality in heat-stressed birds, no clear understanding exists, and a number of plausible pathways, such as adjustments to the levels of reproductive hormones and intestinal calcium absorption, are still being researched (Elnagar et al., 2010). There are various ways in which heat stress might impact a poultry's ability to reproduce. Heat stress in females can interfere with the hypothalamus's and the ovary's normal reproductive hormone status, which can lower systemic levels and functioning(Donoghue et al., 1989, Novero et al., 1991, Rozenboim et al., 2007, Elnagar et al., 2010). Various studies have also demonstrated the adverse consequences of heat stress in guys. When males were exposed to heat stress, their sperm concentration, motility, and sperm volume all decreased (Joshi et al., 1980, MCDANIEL et al., 1995, McDaniel et al., 2004).

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Elevated ambient temperatures in poultry affect their neuroendocrine system, causing the hypothalamic-pituitary-adrenal (HPA) axis to become activated and plasma corticosterone concentrations to rise (Garriga et al., 2006, Star et al., 2008, Quinteiro-Filho et al., 2010, Quinteiro-Filho et al., 2012). Triiodothyronine (T3) and thyroxine (T4), the thyroid hormones, and their balance, control body temperature and metabolic activity. While results of heat-mediated changes on T4 concentrations are inconsistent with studies reporting a drop (Bobek et al., 1980), rise (Elnagar et al., 2010, Cogburn and Freeman, 1987), or no alteration(Mack et al., 2013, Mitchell and Carlisle, 1992), previous studies report that T3 concentrations regularly decrease under high-temperature conditions(Mack et al., 2013, Elnagar et al., 2010, Star et al., 2008, Geraert et al., 1996, Yahav and Hurwitz, 1996). Owing to the thyroid's participation in the onset of puberty and reproductive function in birds, it makes sense that heat stress would impair thyroid activity and affect the hens' ability to reproduce (Elnagar et al., 2010). Furthermore, research by (Geraert et al., 1996) shows that endocrinological alterations brought on by prolonged heat stress in broilers promote the retention of fatty acids by increasing de novo lipogenesis, decreasing lipolysis, and improving amino acid catabolism.

In conclusion, heat stress reduces the neuroendocrine constitution of bird by activating the HPA axis and decreasing the consumption of feed, which in turn reduces overall poultry and egg production. While birds' responses to heat stress are generally comparable, each bird exhibits an individual variance in response intensity and size, that may also be influenced by the heat stress event's intensity and duration. The fact that heat stress is frequently accompanied by other stressors, such as inadequate ventilation and living quarters, as well as social interactions and previous events, which have been demonstrated to influence an individual's stress response, represents another possible explanation for variances (Boissy et al., 2007, Hemsworth, 2003). Furthermore, a growing body of research suggests that a large portion of the variance in how people react to heat stress appears to have a genetic basis (Mack et al., 2013, Soleimani et al., 2011, Felver-Gant et al., 2012). Still, more research is needed in this field to expand on the limited information that is at present readily available. In general, it is essential to remember that the notion of welfare for animals is varied and depends on a variety of factors, including the animal's capacity to execute particular behaviors, be free from sickness, and adapt to its surroundings and social milieu(Lay Jr et al., 2011).

Maintaining homeostasis requires thermoregulation, which is regulated by the endocrine, metabolic, and central nervous systems. Basal metabolic rate is correlated with body mass, confirmation, and morphological parameters like fur color. These factors can be affected by behavioral changes(Canals et al., 1989, Cooper and Geiser, 2008). Animals' thermoregulatory systems are adaptive, helping them endure harsh environments. Birds adjust their physiological and behavioral responses to maintain body temperature through thermoregulation in high-temperature environments. Under heat stress, birds spend more time drinking, panting, elevating their wings, and moving towards cooler surfaces than they do consuming(Mack et al., 2013). Under harsh weather conditions, birds use a variety of heat-exchange mechanisms to maintain their homeostasis, including convection, evaporative heat loss, perspiration, and vasodilation (Mutaf et al., 2009, Fedde, 1998)

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Automatic reactions to a stimulus are termed physiological responses. Physiological reactions are the first line of defense against HS in broiler chickens. Broiler chickens use a variety of techniques for the purpose of reducing their body temperature. The body's featherless areas seem to function as "thermal windows," or places where heat dissipation is most effective, helping broilers regulate their body temperature at high temperatures (Yalcin et al., 1997). In the context of thermoregulation, feather ruffling, dust-bathing, and maintaining the wings away from the body are examples of plumage-related adaptive behaviors. In addition, they consume less food and drink more water when they sleep (Shakeri et al., 2020). And relocate to a cool spot to prevent absorbing additional heat from the surroundings. Even though these early reactions aid in controlling body temperature, they might not be sufficient in hot surroundings, enabling the chicken's body temperature to remain above the ideal range. If so, their body temperature must be lowered by other biological processes.

Chickens under heat stress exhibit different behaviors, including less eating, drinking, and sweating, more time spent with their wings raised, less walking or moving, more sleeping, and a lethargic demeanor (Mack et al., 2013). The internal generated heat and reduction within the bird are out of equilibrium as a result of heat stress. Birds may control the speed at which they lose temperature by altering their activities within the thermoneutral zone. They have no intention of going through heat stress at this point, and their body temperature is stable. When temperatures approach or surpass the upper critical temperature, the birds begin to enthusiastically expel body heat through panting.

Effect of Heat Stress on the Immune Response

Many investigations have been carried out to clarify the various manners in which traumatic stress influences an animal's immunological response. The central nervous system (CNS) modulates the immune response through a complex network that functions in both directions across the immunological, endocrine, and neurological systems. The primary mechanisms via which the immune response can be modified are the sympathetic–adrenal medullar (SAM) and hypothalamic–pituitary–adrenal (HPA) axes. Numerous neuroendocrine products of the HPA and SAM axes, including cortisol and catechol amines, have been demonstrated to have receptors on lymphocytes, monocytes or macrophages, and granulocytes. These compounds may influence cellular trafficking, proliferation, cytokine secretion, antibody synthesis, and cytolytic activity. Numerous in-depth reviews have been written about this topic (Downing and Miyan, 2000, Padgett and Glaser, 2003, Butts and Sternberg, 2008, Marketon and Glaser, 2008). Still, new information is being created that sheds more light on how the immunological, endocrine, and neurological systems interact.

The detrimental effect of extreme temperature on the immunological response in chickens has been the subject of numerous studies in recent years. Even though various metrics are used in each study, heat stress generally has an immunosuppression adverse impact on broilers and laying hens. For example, heat stress in laying hens has been observed to result in lower relative weights of the thymus and spleen(Ghazi et al., 2012) ; heat stress in broilers has likewise been found to result in lowered weights of lymphoid organs (Quinteiro-Filho et al., 2010, Bartlett and Smith, 2003, Niu et al., 2009). In addition, laying hens exposed to prolonged heat stress

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showed decreased liver weights, according to (Felver-Gant et al., 2012) Detected lower liver weights in laying chicks whose livers were subjected to chronic heat stress.(Bartlett and Smith, 2003) found that during primary and secondary humoral reactions, broilers exposed to heat stress had reduced levels of specific IgM and IgG, as well as decreased levels of total circulating antibodies. They also saw noticeably lower liver, spleen, bursa, and thymus weights. Additionally,(Aengwanich, 2010) showed that broilers under heat stress experienced a drop in bursa weight as well as a reduction in the number of lymphocytes in the bursa's cortex and medulla.

Less intraepithelial lymphocytes and IgA-secreting cells have also been seen in the intestinal tract of laying hens under heat stress(Deng et al., 2012), despite reports of a reduced systemic humoral immune response(Bozkurt et al., 2012). Reductions in the phagocytic capability of monocytes and the antibody response in broilers under heat stress have also been documented by others(Bartlett and Smith, 2003, Niu et al., 2009).

Furthermore, heat-stressed broilers showed decreased phagocytosis-performing macrophages, as well as decreased basal and oxidative burst macrophages (Quinteiro-Filho et al., 2010, Quinteiro-Filho et al., 2012). Additionally, recent research has shown that heat stress may alter the number of circulating cells.

Heat stress has been demonstrated to increase the heterophil: lymphocyte ratio because it diminishes the amount of circulating lymphocytes and increases the amount of heterophils. (Felver-Gant et al., 2012, Prieto and Campo, 2010)

Reactive oxygen species (ROS) levels rise in adverse environmental conditions as the bird's body attempts to maintain thermal homeostasis. As a result, the body experiences oxidative stress and begins releasing and generating heat shock proteins (HSP) as an attempt at defense against the harmful effects of reactive oxygen species on cells (Dröge, 2002). Indeed, broilers and laying hens subjected to heat stress produced higher amounts of HSP70 (Gu et al., 2012, Felver-Gant et al., 2012).

Heat stress negatively impacts a bird's health by causing alterations to their physiology, metabolism, hormones, and immune system. In a warm environment, it reduces the production of T and B lymphocytes and inhibits the phagocytic activity of leukocytes in the blood (Kadymov and Aleskerov, 1988). According to Bartlett and Smith (2003), reduced total circulating antibody levels and reduced amounts of particular IgM and IgG in broilers in a hot environment. (BEN and PEREK, 1976) revealed a decline in total WBC and actions of leukocytes after exposure to heat exposure, which (Zulkifli et al., 2000) likewise endorsed the amount of heat stress had dramatically decreased the production of antibodies. Reversal of the immune system the central nervous system's reaction

(CNS), and is carried out via a sophisticated network of the immune, endocrine, and neurological systems.

Poultry industry performance is affected by high temperatures and their ability to fight infection. Stress suppresses the immune system and lowers the weight of the lymphoid organs,

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spleen, liver, bursa of Fabricus, and thymus in chickens. Heat stress reduces the amount of antibodies (IgG and IgM) in the blood, and this in return decreases the humoral response, both primary and secondary (Lara and Rostagno, 2013, Aengwanich, 2010, Tirawattanawanich et al., 2011)

According to(Kala et al., 2017) and (Papadopoulou et al., 2017), heat stress lowers macrophage phagocytic capacity and reduces the antimicrobial response in broilers. Furthermore, heat stress can alter the blood vessel cells ratio. Increased heterophil to lymphocyte ratio at high temperatures because there are more heterophils and fewer lymphocytes(Lara and Rostagno, 2013, Prieto and Campo, 2010). Although poultry birds render attempts to maintain homeostasis, stressful situations cause a rise in reactive oxygen species production.

Toll-like receptors play a role during heat stress because they detect various microbial patterns. TLR2, TLR4, TLR5, and TLR9 detect distinct bacteria by identifying lipoproteins, lipopolysaccharides, flagellin, and DNA, in that order (de Zoete et al., 2010). Heat stress activates the TLR4 pathway. Upon TLR activation, adapter proteins My88 are involved in a signaling cascade. This results in the induction of pro-inflammatory cytokines while suppressing immunity due to the stimulation of transcription factors in the nucleus (Karnati et al., 2015).

Impact of Heat Stress on Poultry Production

This effect of heat stress on grill output efficiency has been the subject of numerous published studies. As was previously shown, pigeons that endure exposure to high ambient temperatures experience physiological, immunological, and behavioral reactions that negatively impact their productivity. An estimated $1.69 to $2.36 billion in yearly economic losses are attributed to heat stress in the U.S. animal production sector; of this amount, the poultry industry bears the brunt, accounting for $128 to $165 million of losses(St-Pierre et al., 2003).

According to a recent study (Sohail et al., 2012), at 42 days of age, broilers exposed to prolonged heat stress had a considerably reduced body weight (−32.6%), a significantly lower feed intake (−16.4%), and a significantly higher feed conversion ratio (+25.6%). Multiple other investigations have demonstrated that broilers exposed to heat stress exhibit a reduced rate of development (Deeb and Cahaner, 2002, Ghazi et al., 2012, Niu et al., 2009, Attia et al., 2011, Imik et al., 2012b). Though the negative consequences of heat stress on broilers appear to be fairly constant, it's crucial to remember that stocking density plays a significant role as a potential aggravating factor, from the perspectives of welfare and productivity(Estevez, 2007).

Chronic heat exposure has been demonstrated to have a breed-dependent deleterious impact on grill fat accumulation and meat quality(Lu et al., 2007). Indeed, as recent research has shown, heat stress in broilers is linked to a decline in the chemical content and quality of meat(Dai et al., 2012, Imik et al., 2012a). A different recent study(Zhang et al., 2012) showed that prolonged heat stress in broilers reduced the overall amount of nipple tissue whilst boosting the percentage of thigh muscle. Additionally, the research revealed that birds under heat stress had reduced protein contents and larger amounts of fat accumulation.

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When being transported from the production farms to the processing facilities, broilers may encounter a number of stressors, such as the transport microenvironment's temperature problems, acceleration, vibration, motion, impacts, fasting, water withdrawal, social disruption, and noise (Mitchell and Kettlewell, 1998, Warriss et al., 2005). A crucial aspect of this intricate mix of variables is heat stress, specifically thermal stress. The effectiveness of the bird's physiological and behavioral thermoregulatory systems is diminished by the restricted space conditions under shipping containers (Warriss et al., 2005). As a result, the negative consequences of these elements alone or in combination might cause anything from slight discomfort to death. As a matter of fact, research has linked heat stress during transportation to increased death rates, lower-quality meat, and inferior welfare status(Mitchell and Kettlewell, 1998). (Warriss et al., 2005) showed a seasonal influence throughout the course of three-year research, with peak mortality rates occurring in the summer. Additionally, the study demonstrated a consistent, noticeable rise in grill mortality with rising ambient temperatures. The percentage of bruises was correlated with the season, the time of transport, and the ambient temperature in a study to identify the factors influencing bruises and broiler mortality at harvest (Nijdam et al., 2004). These same factors were also linked to higher mortality, along with body weight and stocking density, transport, and labor time. Interestingly, it was also discovered that a bird's weight has a direct effect on mortality during transportation between producing farms and processing facilities (bigger birds = higher mortality risk)(Drain et al., 2007). It is indisputable that public concern regarding the health and well-being of grill producers is increasing, both with regard to the production phase in general and the harvest procedure in particular. It's clear that this field hasn't received enough attention, so more research is desperately needed.

Many variables, including environmental stress (like heat stress), which is likely one of the most frequent problems in many production systems worldwide, can also have an impact on the productivity of flocks of laying hens. The majority of the negative impacts of heat stress on production are most likely caused by decreased feed intake, which lowers body weight, feed efficiency, egg output, and egg quality (Mashaly et al., 2004, Deng et al., 2012). Heat stress, however, has also been linked to lower calcium and plasma protein levels, as well as poor nutritional digestibility (Mahmoud et al., 1996, Bonnet et al., 1997, Zhou et al., 1998). This is in addition to decreased feed intake.

A 12-day heat stress phase reduced daily feed intake by 28.58 g/bird in a recent study(Deng et al., 2012), which led to a 28.8% drop in egg production. According to(Star et al., 2009), laying hens exposed to heat stress saw reductions in feed conversion of 31.6%, egg production of 36.4%, and egg weight of 3.41%. In a different study(Lin et al., 2004) heat stress led to lower eggshell thickness, increased egg breakage, and decreased production performance. In addition, it has been demonstrated that heat stress significantly reduces eggshell weight (−9.93%), eggshell percent (−0.66%), egg weight (−3.24%), and eggshell thickness (−1.2%) (Ebeid et al., 2012). (Mack et al., 2013) also noted lower egg output, egg weight, and eggshell thickness in laying hens exposed to heat stress. An intriguing set of tests(Farnell et al., 2001) revealed the increasing adverse effects that persistent stress from heat possesses on the growth and development of embryos. In these tests, the egg production of laying hens exposed to heat

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stress for 8–14 days, 30–42 days, and 43–56 days decreased by 13.2%, 26.4%, and 57%, respectively. In a different study (Mashaly et al., 2004), laying hens exposed to prolonged heat stress for five weeks showed a significant drop in body weight (19.3%), feed intake (34.7%), and egg production (28.8%).

Even though there is much variety in the effects among the numerous published research, it is noteworthy that heat stress consistently has a major impact on egg production and quality. The use of birds with varying ages or genetic backgrounds, along with the varying lengths of time and intensity of the heat stress treatments administered, may readily account for the heterogeneity of the results recorded.

Due to the rising demand for meat and eggs, both commercial businesses and small-scale backyard farmers in rural areas have made poultry farming a top priority. Globally, decreased growth and egg production are believed to be a result of warmer temperatures as time passes in poultry production systems. The negative effects on the poultry industry are expected to get worse at a period in which heat stress is considered to be becoming an international issue and is anticipated to have a substantial impact on the worldwide temperature increase due to climate change. A thorough analysis is conducted to examine the effects of heat stress on various aspects of poultry cultivation and development, which involves egg production, physiology, reproduction, immunity, and disease incidence. The development of heat-tolerant breeds of poultry may result from the use of sophisticated biotechnology tools to select appropriate molecular markers and create deserving breeding programs. In addition, appropriate mitigation techniques have been proposed to lessen the negative effects of these stressful environments on poultry in order to reduce the monetary harm to poultry breeders.

Can Heat Stress Impact Food Safety?

Abnormal meat traits and quality loss have been linked to heat stress in broiler maturing periods(Lu et al., 2007, Zhang et al., 2012). Furthermore, it has been demonstrated that grill transportation at high temperatures from farms to processing facilities reduces the overall quality of the meat (Mulder, 1995, Debut et al., 2005, Dadgar et al., 2010). It has been demonstrated that heat stress in laying hens has a negative impact on egg quality and production(Bozkurt et al., 2012, Mashaly et al., 2004). In recent times, food safety has emerged as a significant concern for the global chicken and egg-producing sector. In reality, the philosophy of contemporary culinary excellence has started incorporating food security as an essential aspect.

In poultry and egg production, colonization of birds by foodborne viruses like Salmonella and Campylobacter and their subsequent spread throughout the human food chain pose a serious threat to public health and the poultry industry's bottom line.

As a matter of fact, one of the most frequently discovered causes of foodborne disease is the handling and ingestion of undercooked poultry products (Gantois et al., 2009, Newell et al., 2010, Eisenberg et al., 2012, Domingues et al., 2012)

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Stress can significantly impair food safety through a number of possible routes, as evidenced by the growing body of research. The processes behind this effect have not yet been completely understood, despite evidence connecting stress with pathogen carriage and shedding in agricultural animals (Rychlik and Barrow, 2005, Humphrey, 2006, Rostagno, 2009). It has been demonstrated that environmental stress can result in pathogen colonization of farm animals, increased fecal shedding and horizontal transmission, and ultimately an increased risk of animal product contamination (Humphrey, 2006, Rostagno, 2009, Verbrugghe et al., 2012). These features of infections in animals have long been linked to the impact of stress-related hormones and mediators on the immune system (mostly as a consequence of immunosuppression). However, a fresh viewpoint known as "microbial endocrinology" has emerged recently, based on the direct impact of stress-related hormones and mediators on bacterial pathogens (Lyte, 2004). Numerous recent studies have shown that bacteria, including Salmonella and Campylobacter, can use the host's neuroendocrine changes brought on by the stress response to their benefit to continue to multiply and become more harmful (Verbrugghe et al., 2012, Freestone et al., 2008). It is crucial to understand that environmental stressors, like heat stress, possess a tendency to change the host-pathogen relationship.

Stressors can modify the protective microbiota and reduce the integrity of the intestinal epithelium, two alterations that can occur in the gastrointestinal tract that are particularly sensitive to them(Collins et al., 2012, Dinan and Cryan, 2012). As was previously said, there is also a lot of evidence to suggest that adapting to and managing environmental stressors can change biological defense mechanisms like cell-mediated immunity and antibodies, making a person more vulnerable to infections. A complex and dynamic microbial community, known as the microbiome, is present in the chicken digestive tract and is influenced by a number of variables(Wei et al., 2013). The effects of environmental stresses, especially heat stress, on the intestinal microbial community of chickens are not currently extensively investigated in the scientific community. The rumen, a far more complicated microbial system than the intestinal microbiome of chicken, has been shown in studies to be affected by heat stress in terms of both microbial composition and short-chain fatty acid content(Tajima et al., 2007, Uyeno et al., 2010). Consequently, it seems sensible to believe that heat stress would further have an influence on the microbial populations in poultry's gastrointestinal tract. This knowledge gap still needs further investigation and understanding, though. In particular, it has been documented that heat stress raises the permeability of the intestinal barrier in broilers (Garriga et al., 2006). There have been reports of altered shape and modifications in the composition of the microbial community in the intestinal tract of broilers exposed to heat stress(Burkholder et al., 2008). Furthermore, the same study (Burkholder et al., 2008)demonstrated by an ex vivo technique that Pseudomonas Enteritidis's mucosal adhesion rose in tissues derived from heat-stressed birds. Further investigations(Bozkurt et al., 2012, Deng et al., 2012) also found morphological changes in the intestinal tract of laying hens exposed to heat anxiety, which included a drop in villus height and a decrease in the ratio of villus height to crypt depth. These findings corroborated the changes in gastrointestinal morphology seen in the earlier study(Burkholder et al., 2008)

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The abnormal process of intestinal permeability begins with oxidative stress. Reactive oxygen species (ROS) concentrations rise in heat stress situations, increasing intestinal permeability and facilitating the translocation of microorganisms from the digestive tract. In fact, it has been documented that broilers exposed to heat stress experience enhanced inflammation and translocation of Salmonella Enteritidis (Quinteiro-Filho et al., 2010, Quinteiro-Filho et al., 2012), which raises the pathogen's levels in spleen samples.

It is conceivable that elevated temperatures could impact not just the amount of bacteria present in bird droppings but also the length of time and degree of contamination in the surrounding area, which could result in more extensive spread. A short study (Traub-Dargatz et al., 2006) found no evidence that heat stress changed the quantity of Pseudomonas shed in feces or prolonged its life. However, seasonal variations in the prevalence of Salmonella and Campylobacter in broiler and laying hen flocks and retail chicken products have been documented by a number of epidemiological investigations(Patrick et al., 2004, Wales et al., 2007, Van Der Fels-Klerx et al., 2008, Jorgensen et al., 2011, Zdragas et al., 2012).

Food safety is considered a crucial component of the current nutritional value idea. The poultry industry faces numerous challenges pertaining to food safety on a global scale because of harsh conditions.

According to (Pawar et al., 2016a) stress can have a negative impact on food safety through a variety of potential processes, such as the shedding of pathogens on farm animals under stressful conditions. High temperatures in broilers lead to undesirable meat characteristics and quality loss [60]. Furthermore, due to the elevated temperatures during transit from chicken farms to processing facilities, meat quality is lost (Dadgar et al., 2010). Heat stress in laying hens is linked to a reduction in egg production and quality (Pawar et al., 2016a, Bozkurt et al., 2012). A significant public health and economic issue in the poultry and egg industry is the colonization of birds by foodborne pathogens like Salmonella and Campylobacter, which then spread through the human food chain.

In fact, eating and handling undercooked poultry products is one of the most frequently mentioned causes of foodborne illness (Gantois et al., 2009, Domingues et al., 2012). Elevated ambient temperatures have the potential to modify not only the bacterial counts in bird droppings, but also the length of time and degree of contamination in the surrounding area, which could result in increased dispersion. Heat stress, however, did not result in higher concentrations or longer survival of Salmonella excreted in feces (Traub-Dargatz et al., 2006). Seasonal variations in the incidence of Campylobacter and Salmonella in flocks of laying hens and broilers. As well as in chicken products sold in retail establishments, have been noted in a number of epidemiological studies (Patrick et al., 2004, Zdragas et al., 2012)

Therefore, this topic represents a critical knowledge gap that needs to be filled because of the enormous implications for our comprehension of the ecology and epidemiology of diseases in poultry flocks under high temperature or heat stress conditions.

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CONCLUSION

One of the biggest environmental stressors affecting chicken production globally is heat stress. Reduced growth and egg production, as well as lower poultry and egg quality and safety, are some of the detrimental impacts of heat stress on broilers and laying hens. On the other hand, the detrimental effects of heat stress on poultry welfare have to be a top priority.

Numerous studies have been conducted on the impact of heat stress on poultry's immune system and productivity, as this overview demonstrates (broilers and laying hens). On the other hand, there is a dearth of knowledge regarding the fundamental mechanisms underlying the consequences that have been observed and confirmed, as well as the behavior and welfare of birds under heat stress.

The poultry business is finding the HS issue to be increasingly difficult over time. Because of genotype selection for faster growth rates in order to meet the ever-increasing demand for food, grilled chickens are now susceptible to HS. Unfortunately, if selection for only production traits is prioritized against heat tolerance and climate adaptation in light of current trends in global warming, the detrimental effects of heat stress on poultry health and production are likely to persist and be inherited by the next generation during gestation.

Because high producers and commercial grill breeds are unable to survive HS, the industry suffers significant financial losses that lead to problems with food security. The only long-term way to reduce the harmful effects of HS is through genetic selection for heat tolerance in chickens.

Recognizing this threat to food security, industry and scientists must work together to find a solution. As part of these efforts, heat-tolerant breeds' genotypes should be profiled, and thorough research on the relationship between genotype and phenotype in susceptible and heat-tolerant grill breeds should also be conducted. (b) Investigating the entire molecular mechanism of muscle growth and development during the middle school environment. (c) Creating a miniature chicken breed from frizzled feathers may provide a clearer picture of the chemical and genetic processes underpinning heat resistance. Adopting contemporary management and environmental techniques could reduce the negative effects of heat on meat production and quality along with breeding strategies.

Lastly, it is noteworthy that numerous published studies have concentrated on intervention strategies to address heat stress conditions. These strategies employ a variety of techniques, such as environmental management (facilities design, ventilation, sprinkling, shading, etc.), nutritional manipulation (diet formulation based on the metabolic condition of the birds), and the addition of feed additives (antioxidants, vitamins, minerals, probiotics, prebiotics, essential oils, etc.) to the diet.

However, the majority of the therapies have had uneven or erratic effectiveness. In recent times, two novel strategies have been investigated: early-life training, also known as perinatal heat acclimation, and genetic selection of breeds which have a higher ability to tolerate high temperatures (also known as higher heat tolerance).

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