Literature Review

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Physiology, cell dynamics of small intestinal mucosa, and performance of broiler chickens under heat stress: a review¤

Fisiología, dinámica celular de la mucosa del intestino delgado y rendimiento de pollos de engorde sometidos a estrés calórico: revisión de literatura

Fisiologia, dinâmica celular da mucosa do intestino delgado e desempenho de frangos de corte submetidos ao estresse pelo calor: revisão de literatura

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Summary

High room temperature reduces production efficiency of broiler chickens. Different factors contribute to this situation: fast growth, physiological variations, and changes in the small intestine mucosa. This review aims to define the concept of heat stress and its effects on several physiological aspects related to the development of the small intestine mucosa and the performance of broiler chickens. Heat stress triggers a corticosteroid increase and a circulating triiodothyronine hormone (T_3) reduction, increases respiratory frequency, which triggers respiratory alkalosis, diminishes food intake, and leads to changes in the cellular dynamics of the small intestine mucosa. These changes depend on animal genotype, intensity, and duration of the stressor. Acute heat stress leads to a reduction in enterocyte proliferation and also to a decrease in crypt depth, without affecting villus height or villus/crypt ratio. On the other hand, chronic stress diminishes villus height and jejunum weight. These changes affect the capacity of broilers to digest and absorb the nutrients required for maintenance and production.

Keywords: *corticosteroids, enterocyte proliferation, gastrointestinal tract, microscopic findings, morphological findings.*

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Resumen

Una alta temperatura ambiente disminuye la eficiencia de la producción de pollos de engorde. Diversos factores contribuyen a esta situación: crecimiento rápido, cambios fisiológicos, y anomalías de la mucosa del intestino delgado. El objetivo de la presente revisión fue definir el concepto de estrés calórico y sus efectos sobre algunos aspectos de la fisiología, en el desarrollo de la mucosa del intestino delgado, y en el rendimiento de pollos de engorde. El estrés calórico desencadena un aumento de los niveles de corticosteroides, reducción de la hormona triyodotironina (T_3) circulante, aumento en la frecuencia respiratoria que resulta en alcalosis respiratoria, disminuye la ingesta de alimentos y provoca cambios en la dinámica celular de la mucosa del intestino delgado. Estos cambios dependen del genotipo del animal y de la intensidad y duración de la acción del factor estresante. El estrés calórico agudo causa una disminución en la proliferación de enterocitos, reducción de la profundidad de las criptas sin cambio en la altura de las vellosidades y en la relación vellosidades/cripta. Ya el estrés crónico disminuye la altura de las vellosidades y el peso del yeyuno. Estos cambios afectan la capacidad del pollo de engorde para digerir y absorber los nutrientes para su mantenimiento y producción.

Palabras clave: *corticoesteroides, hallazgos microscópicos, hallazgos morfológicos, proliferación de enterocitos*, *tracto gastrointestinal.*

Resumo

A temperatura ambiente elevada diminui a eficiência produtiva de frangos de corte. Diferentes fatores contribuem para esta situação: crescimento rápido, mudanças fisiológicas e alterações na mucosa do intestino delgado. Nesta revisão, o objetivo foi definir o conceito de estresse pelo calor e seus efeitos sobre algunos aspectos da fisiologia, no desenvolvimento da mucosa do intestino delgado e o desempenho de frangos de corte. O estresse pelo calor desencadeia aumento nos níveis de corticosteroides e redução do hormônio triiodotironina (T_3) circulante, aumenta a frequência respiratória, resultando em alcalose respiratória, diminui a ingestão de alimentos e desencadeia alterações na dinâmica celular da mucosa do intestino delgado. Essas alterações dependem do genótipo do animal e da intensidade e duração da ação do agente estressor. O estresse pelo calor agudo promove diminuição na proliferação dos enterócitos, redução na profundidade das criptas sem alteração na altura das vilosidades e na relação vilo/cripta. Já o estresse crônico diminui a altura das vilosidades e peso do jejuno. Estas alterações afetam a capacidade do frango de corte de digerir e absorver nutrientes para sua manutenção e produção.

Palavras chave: *achados microscópicos, achados morfológicos, corticosteroide, proliferação de enterócitos, trato gastrointestinal.*

Introduction

The genetic selection for fast growth and weight gain that broiler chickens have been subjected to in the last few decades has caused these breeds to become very vulnerable to environmental factors that are typical of tropical regions, such as intense solar radiation, temperature, and high air humidity. Together, these factors cause stress in birds and, consequently, discomfort.

The concept of stress, often poorly understood, can be defined as "a force of any kind that acts in the body causing a strain state" (Silva, 2000). Therefore, a strain state in living beings results from stress and depends on the physiological characteristics of each organism (Silva, 2000). When different environmental factors act alone or together on a living being, they become stress factors that may cause tension on the body (Silva, 2008). Therefore, the symptoms presented by animals suffering from the action of environmental factors show that they are under strain or tension (Silva and Maia, 2012). When birds are in a state of strain, neuroendocrine as well as physiological and behavioral responses occur that enable the birds to acclimate to the adverse environmental conditions they are being raised in, in order to maintain homeostasis (Van Borelli, 1995).

The strain generated by heat stress may also trigger changes in intestinal development by reducing the small intestine weight, the number of villi, and the proliferation rate of enterocytes (Uni *et al.*, 2001). It directly affects the bird capacity to digest and absorb the nutrients required for maintenance and

production (Quintero-Filho *et al.*, 2010). The strain caused by heat stress may reduce growth rate, food intake, final body weight, and weight gain, as well as feed conversion ratio. Thus, performance of birds subjected to heat stress is compromised by deficient intestinal development and diminished food intake. Furthermore, the strain caused by heat stress may affect broiler metabolism, cause oxidative stress, and increase superoxide production and malondialdehyde levels (Azad *et al*., 2010).

This review aims to define the concept of heat stress and its consequences on several physiological aspects of the development of the small intestine mucosa and the performance of broiler chickens.

Heat stress

The glossary of the International Union of Physiological Science (IUPS) Thermal Commission (2001) defines "thermal comfort zone" as "the range of ambient temperatures, associated with specified mean radiant temperature, humidity, and air movement, within which a human in specified clothing expresses indifference to the thermal environment for an indefinite period."

A bird maintains its body temperature in the thermal comfort zone with minimal use of thermoregulatory mechanisms; i.e., the metabolic rate is minimum, constant, and independent of the room temperature (Abreu *et al.*, 1998). Homeothermy is maintained with lower energy expenditure under these conditions and the bird is able to express its maximum production potential (Furlan, 2006).

According to the IUPS Thermal Commission (2001), "thermal stress is any change in the thermal relationship between a temperature regulatory mechanism and the environment which, if not compensated, will result in hyper- or hypothermia."

The effects of heat stress on bird performance were extensively studied in past decades (Gross and Siegel, 1981; Arjona *et al.*, 1988; Donkoh, 1989; Cooper and Washburn, 1998), at a time when most sheds were open and thus there was little climatic control in the production environment.

In recent years, there has been a considerable increase in the number of studies on heat stress and its effects on animal production, both for economic reasons and for animal welfare (Quintero-Filho *et al.*, 2010), and because heat stress is an important problem affecting the poultry industry in tropical and subtropical regions (Sabah *et al.*, 2008).

Organisms react to a stressor with physiological or behavioral adjustments that allow them to better manage an adverse environment. These adjustments are an attempt to maintain homeostasis, i.e., the relative constancy of chemical-physical properties in an organism internal environment that is maintained by regulatory mechanisms (IUPS Thermal Commission, 2001). The Thermal Commission (2001) considered such facts as evidence that animals develop response mechanisms when their homeostasis is threatened.

It is necessary to emphasize that an animal responses to stressors depend mostly on the genotype (Havenstein *et al*., 2003a), intensity and duration of the stressor (Mitchell and Lemme, 2008). The stressor effects on physiological functions may be sufficiently intense to compromise the capacity of birds to grow, reproduce and produce efficiently (Medeiros *et al.*, 2005; Silva *et al.*, 2007).

Heat stress may be acute or chronic. Acute stress refers to sudden and short periods of high temperature for less than seven days, whereas chronic stress refers to prolonged periods of high temperature for more than seven days (Gonzalez-Esquerra and Leeson, 2006).

Physiological changes induced by heat stress in broilers

Birds are homoeothermic animals that maintain a relatively constant internal body temperature under a wide range of room temperatures (Dawson and Whittow, 1998). The body temperature of adult domesticated broilers is approximately 41.1 °C (Marchini *et al*., 2007). When air temperature and humidity exceed the thermal comfort range, the ability to dissipate heat is reduced and animals suffer the effects of heat stress (Dawson and Whittow, 1998).

The response to stressor agents involves a series of neuroendocrine and behavioral responses that seek to maintain the balance of vital functions (Barnett and Hemsworth, 1990; Van Borelli, 1995) by activating the hypothalamic-pituitary-adrenal (HPA) axis. The stressor agent causes a nerve stimulation that reaches the hypothalamus and activates the HPA axis, resulting in the production of corticotropin-releasing hormone (CRH). This hormone acts on the adenohypophysis, which will produce and secrete adrenocorticotropic hormone (ACTH) and β-endorphins. Blood-stream transported ACTH stimulates glucocorticoid secretion in the adrenal cortex as well as adrenalin and noradrenalin release in the sympathetic nerve endings and in the adrenal medulla (Dukes, 2004). Plasma corticosteroid levels rapidly increase as a consequence of severe heat stress and reach a peak after one hour of heat exposure (Siegel, 1980).

When the stressor action persists on an animal, there is prolonged secretion of hormones such as catecholamines and corticosteroids. Numerous harmful consequences will be triggered if corticosterone levels remain high (Ribeiro *et al.*, 2008), including redistribution of body reserves—including energy and proteins—thus leading to reduced growth, reproduction, and health (Braude *et al*., 1999; Oliveira *et al.,* 2006) because corticosterone is the glucocorticoid responsible for glucose formation from body reserves of carbohydrates, lipids, and proteins (Freeman, 1987).

HPA regulation is accomplished by the negative feedback mechanism of corticosterone, which acts on the hypothalamus by inhibiting CRH release in the adenohypophysis, and thereby inhibiting ACTH secretion (Dukes, 2004).

Thyrotropin releasing hormone (TRH), produced in the hypothalamus, acts as a thyroid stimulating hormone (TSH) stimulator. TSH acts in the thyroid gland to produce thyroxin (T_4), which is transformed into triiodothyronine (T_3). Serum T_3 concentration is reduced as heat stress persists (Yahav *et al.*, 1995; Yahav, 1999). Thyroid hormone synthesis and secretion are regulated by the thyroid hormones themselves in a self-regulatory manner. These events demonstrate that there is "interaxial" hypothalamic control of TSH release in the chicken (Debonne *et al.*, 2008).

Furthermore, heat stress causes important changes in the electrolytic balance of broilers (Borges *et al.*, 2004). The respiratory frequency of birds increases in order to reduce body temperature during heat stress (Marchini *et al.*, 2007), and panting results in changes of the acid-base balance and in respiratory alkalosis from greater elimination of CO_2 and from pCO_2 reduction in the blood. The body responds with metabolic acidosis, an H^+ excretion decrease, and an HCO_3^- excretion increase by the kidneys to compensate for this disturbance, and thus such changes contribute to blood acidification (Borges *et al.*, 2004). Energy reserve reduction takes place and the homeostatic efficiency balance of the body diminishes as the acclimatization mechanisms begin to fail.

Different physiological responses may occur during heat stress and they depend on the intensity, severity, and duration of heat stress (Azad *et al*., 2010), thereby causing higher or lower levels of glucocorticoid release (Gonzalez-Esquerra and Leeson, 2006). These physiological responses are: changes in the functional integrity of the intestinal mucosa (Horn *et al.*, 2009), body temperature increase (Marchini *et al.*, 2007; Mitchell and Lemme, 2008), muscle degradation (Zuo *et al.,* 2015), and increase in mortality rate (Estrada-Pareja *et al.*, 2007) when high environmental temperatures coincide with the age to market (Arjona *et al.*, 1988).

Effects of heat stress on cell dynamics of broiler chicken small intestinal mucosa

Small intestine mucosa development results from two associated cytological events: cell renewal — due to proliferation and differentiation of columnar cells in the crypt and along the villi (Uni *et al.*, 1998) — and the natural loss of cells by extrusion at the villous apices (Pelicano *et al.*, 2003) after they have undergone apoptosis (Renehan *et al.*, 2001). Enterocyte proliferation in broiler chicken small intestine occurs in the crypt and along the villi (Uni *et al.*, 1998). At four days of age, these cells migrate to the duodenum and jejunum (Geyra *et al.*, 2001).

Effective food metabolism is directly related to the integrity of the digestive system, especially the small intestine, and part of the digestive process,

as well as the absorption of nutrients, occurs in the enterocytes (Smith *et al.*, 1990). The functional integrity of the epithelial intestinal mucosa depends on coordinated processes, such as metabolic regulation of epithelial cells and regulation of the mucus layer. The epithelium of intestinal mucosa is covered by a mucus layer composed of glycoproteins synthesized by the goblet cells. This mucus acts as a protection to help maintain epithelium integrity, lubrication, nutrient absorption (Gibson *et al.*, 1996), and molecule transport between the luminal contents and the epithelial cells during digestion and absorption of nutrients from the diet (Uni *et al.*, 1998). Thus, mucus is the first barrier the nutrients have to interact with; they have to diffuse into the mucus to be absorbed and gain access to the circulatory system and their target organs (Bansil and Turner, 2006). Environmental factors able to induce changes in the dynamics of mucins have the potential to affect viscosity and integrity of the mucus layer and nutrient transportation (Horn *et al*., 2009). Therefore, interruption of intestinal homeostasis leads to changes in the mucus barrier that protects the enteric mucosa. An increase in the enteric mucosa permeability may result in inflammatory processes and injuries to mucosa cells (Dharmani *et al.*, 2009).

One of the main features that make the broiler chicken so productive is the great capacity of its intestinal epithelium to absorb nutrients (Oliveira *et al.*, 2000). According to Cera *et al.* (1988), the absorption capacity of the small intestine is proportional to the number and size of the intestinal villi and the surface area available for absorption. Thus, adequate and fast weight gain is related to the morpho-functional integrity of the intestinal mucosa, especially in the small intestine (Furlan *et al.*, 2001). Such integrity is crucial to enable the bird to improve its digestion and absorption processes and, therefore, its performance potential.

Damage to intestinal morphology is one of the first consequences of stress (Cosen-Binker *et al.*, 2004) and it depends on the stressor intensity and duration. Acute stress reduces enterocyte proliferation in chicks (Uni *et al.*, 2001) and reduces ileum crypt depth without changing villus height or the villus/crypt ratio (Burkholder *et al.*, 2008). Nevertheless, Marchini *et al.* (2011) found no change in enterocyte proliferation in broiler chickens subjected to daily cyclic heat stress (from 12-13 h at 38 °C between the 1st and the 27th day and at 40 ºC between the 28th and the 42nd day). Chronic heat stress led to a 19% reduction in villus height and 26% reduction in jejunum weight (Mitchell and Carlisle, 1992). However, no changes were observed in villus and crypt structures when birds were subjected to cyclic heat stress (36 °C for 12 h, between the 35th and the 41st day of age), a fact that could be attributed to the fast re-epithelialization of intestinal mucosa (Quintero-Filho *et al.*, 2010). Actually, it demonstrated that the epithelial structure is replaced in less than 36 h after the stress condition is removed (Burkholder *et al.*, 2008).

Reduction of feed intake as a consequence of heat stress (Quintero-Filho *et al.*, 2010) is one of the factors that may change intestinal morphology, for instance, by reducing villus height and crypt depth (Burkholder *et al.*, 2008) and by reducing enterocyte proliferation in the small intestine, both of which would directly affect the capacity to digest and absorb nutrients necessary for maintenance and production (Uni *et al.*, 2001). According to Sell *et al*. (1985), crypt depth reduction along with increased mucus secretion could reduce absorption capacity of the intestine.

It is important to highlight that feed intake reduction is not the only cause of growth rate decrease in broilers reared under high room temperature. Geraert *et al*. (1996) subjected broilers to heat stress and fed other birds, kept under thermoneutral conditions, with the same feed intake as those reared under heat stress. They concluded that feed intake reduction by broilers kept under high temperatures was responsible for approximately 50% of growth reduction. These authors also reported that broilers reared under high temperature showed changes in protein metabolism (a decrease in protein synthesis or an increase in its catabolic rate), which can also contribute to a reduction of the growth rate.

With regard to lower enterocyte proliferation in broilers reared under high temperatures, Gao *et al*. (2013) reported that heat stress induces programmed cell death (apoptosis) in the small intestine of rats, and this is mediated by the AKT signalling pathway. The AKT is a serine-threonine kinase first described as an oncogene within the mouse leukaemia virus

(Bellacosa *et al*., 1991) and it is implied in cell growth and survival (Franke *et al.*, 2008). It inhibits the pro-apoptosis effect (Dasari *et al*., 2008) induced by heat stress and promotes cell survival (Datta *et al*., 1997). It represses apoptosis by inhibiting the activities of pro-apoptotic proteins (Gao *et al*., 2013). Gao *et al*. (2013) reported that during heat-induced apoptosis, more AKTs were activated, showing increased phosphorylation. These authors observed that two hours after heat exposure there was a higher level of AKT phosphorylation in rat IEC-6 cell line that coincided with a marked reduction of apoptosis. The authors also suggested an effect of AKT on suppressing apoptosis triggered by heat stress and therefore protects villi epithelial cells from apoptosis at certain points in the apoptotic process, promoting cell survival. When they replicated their study *in vitro,* the results were similar to the *in vivo* ones. Therefore, the authors concluded that high temperature induces intestinal cell damage regardless of feed intake.

Redirection of blood flow takes place during exposure to heat stress. Heat stress significantly reduces digestive system capillary blood flow to inner organs such as the upper respiratory tract, to the brain of laying hens (Wolfenson *et al.,* 1981) and to the skin (Cronje, 2007). It can result in gut ischemia and increased ROS production due to insufficient oxygen supply (Cronje, 2007), which may induce excessive epithelial cell apoptosis, shedding of intestinal epithelial cells (Gao *et al*., 2013), and reduced nutrient absorption (Kohn *et al*., 1993).

It has also been demonstrated that heat stress increases lipid peroxidation. Bagchi *et al.* (1999) reported that acute and chronic stress exposure increases mucosal lipid peroxidation, cytochrome c reduction, and hydroxyl radical production in rat intestinal mucosa. Such results indicate that oxygen free radicals, including superoxide anions and hydroxyl radicals, are involved in the pathogenesis of acute and chronic stress-induced gastrointestinal injury, and can result in lipid peroxidation.

High body temperature due to heat stress (Marchini *et al*., 2007) can induce metabolic changes involved with oxidative stress induction. According to Ismail *et al*. (2013), heat stress increases lipid peroxidation — high values of malondialdehyde (MDA) — and depletes the bird antioxidant capacity, as evidenced by decreased catalase (CAT), glutathione-S-transferase (GST), and superoxide dismutase (SOD) activity in broilers. Altan *et al*. (2003) concluded that heat stress increased lipid peroxidation due to increased free radical generation, as indicated by the increased MDA concentration in broilers exposed to heat stress. According to Altan *et al*. (2000), oxidative stress can be considered part of the broiler chicken response to heat exposure.

Effects of heat stress on broiler chicken performance

Between 1956 and 2000, selective breeding of broiler chickens for fast growth and more efficient food conversion resulted in significant productivity gains for the poultry industry. The most remarkable changes observed in the modern broiler chicken when compared to its ancestors are better growth rates, heavier carcass weight, and greater yield of breast meat. In 2000, broiler chickens reached 1.82 Kg body weight at 32 days of age. In 1966, a chicken needed approximately 60 days to attain the same weight, and it took 84 days in 1956 (Havenstein *et al.*, 2003a; 2003b). Therefore, improvements in genetics, nutrition, and breeding management over the last few decades have resulted in approximately one-third of the time and three times less food for broilers to reach 1.85 Kg. Genetic increase was responsible for 85 to 90% increase in growth weight (Havenstein *et al.*, 2003b).

However, fast growth comes along with a disproportionate development of the cardiovascular and respiratory organs (Havenstein *et al.*, 2003a). This impairs the ability to deal with heat stress challenges (Yahav *et al.*, 2005), since birds selected for high growth rates also present higher metabolic rates and generate more endogenous heat (Thiruvenkadan *et al.*, 2011).

The changes introduced by domestication and genetic selection — to improve broiler response to acute heat stress (36 °C for 3 hours) — were described by Soleimani *et al.* (2011). Those authors reported a greater susceptibility to heat stress in the fast growing, genetically improved Cobb breed when compared to the unimproved Red Jungle Fowl (RJF) and Village Fowl (VF) breeds. They investigated birds of the

same age (30 days old) weighing 150 g (RJF), 354 g (VF) and 1432 g (Cobb), and also birds with the same body weight (930 g) at 150 (RJF), 90 (VF), and 22 (Cobb) days of age. The Cobb chickens exhibited greater heterophil: lymphocyte ratio, greater body temperature, lower Hsp70 levels in the brain, and lower corticosterone concentrations. They concluded that domestication and genetic improvement for greater weight gain and faster development resulted in physiological changes in the chicken breeds currently used in the poultry industry. These changes led to more susceptible and less resistant individuals when compared with unimproved breeds.

During heat stress, there is blockage of the appetite center located in the hypothalamus. Consequently, there is feed consumption reduction (Akşit *et al.*, 2006) and the birds suffer deficiencies in many, if not all, nutrients essential to their performance (Donkoh, 1989).

Feed consumption reduction is a change in dietary behavior associated with production mechanisms and heat loss. The change is necessary to dissipate the heat generated by digestion and energy metabolism (Veldkamp *et al.*, 2005). Thus, feed consumption of chickens subjected to heat stress between 21 and 40 days of age was inversely proportional to the increase in the room temperature as the temperature was raised from 25 to 31 °C (Estrada-Pareja *et al.*, 2007).

As the stress period persists, its negative impact on feed intake gets worse. Birds raised at a room temperature of 32 °C had a feed intake decrease of 14.7% between 1 and 21 days of age, and a decrease of 22% between 1 and 49 days. Therefore, there is 2.2% decrease in feed intake per degree centigrade increase in room temperature above the thermal comfort zone (Oliveira *et al.*, 2006).

In addition to the prolonged stress period, intensity of the high temperatures also affects feed intake. Al-Fataftah and Abu-Dieyeh (2007) subjected broilers daily to three constant temperatures: 25 (thermoneutrality), 30, and 35 °C. According to these authors, there was 12.4% feed consumption reduction in birds kept at 30 °C and 28% reduction in those kept at 35 °C when compared with birds kept at thermoneutrality (25 °C). Therefore, as broilers were challenged by higher room temperatures, greater feed intake reduction was observed.

Temperature increase and heat stress duration may also cause negative responses in body weight gain, and these responses also depend on broiler breed. Weight gain losses may range from 13.9% in broiler chickens kept at 32 °C for 24 h between 1 and the 49 days of age (Oliveira *et al.*, 2006), to 44% in Isa Vedette chickens kept at 35 °C for 24 h between 28 and 56 days of age (Al-Fataftah and Abu-Dieyeh, 2007). These authors concluded that lower weight gain at high temperatures result from feed intake reduction, digestion inefficiency, and impaired metabolism. Azad *et al.* (2010) also concluded that either chronic or cyclic heat stress could reduce body weight gain due to reductions in both feed consumption and feed efficiency.

Reports about feed conversion and mortality in birds subjected to heat stress are controversial, and responses depend on stressor intensity and duration. Estrada-Pareja *et al.* (2007) found no changes in feed conversion of broiler chickens subjected to heat stress (31 °C) for 24 h between 21 and 40 days of age when compared to those raised in thermoneutrality (25 °C), despite a 6.9% reduction in weight gain. However, Akşit *et al.* (2006) reported poorer feed conversion in broiler chickens kept at 34 °C and 60% relative humidity from 21 to 49 days of age, when compared to those kept in thermoneutrality. Cooper and Washburn (1998) reported that mortality did not change in Ross male broilers subjected to 32 °C for 24 h between 28 and 49 days of age. However, when Al-Fataftah and Abu-Dieyeh (2007), evaluated performance of broilers subjected to heat stress, they verified a significant increase in mortality of Isa Vedette chickens raised at 35 °C between 4 and 8 weeks of life. Losses by mortality in Brazil due to excessive heat in chicken raising sheds corresponds to 10% of the total production through the hottest months of the year (Aradas *et al.*, 2005).

Exposure to high room temperature is a cause decline in broiler production efficiency and meat yield. High environmental temperatures also harm breast, thigh, and drumstick meat yield. This negative effect is worsen by an increase in relative air humidity (Oliveira *et al.*, 2006). These changes take place

because heat stress can stimulate reactive oxygen species (ROS) production, particularly superoxide formation and oxidative damage. Heat-induced ROS formation may be the cause of damages in the skeletal muscle (Azad *et al*., 2010). High temperatures also cause changes in energy, protein, and fat retention in the body, and cause physiological changes in the development of organs and carcass (Baldwin *et al.*, 1980).

Final considerations

Our current understanding of the effects of heat stress on broiler chicken performance shows that production efficiency declines under heat stress when compared to production efficiency under thermoneutral conditions.

According to the existing evidence, it is reasonable to assume that broilers under heat stress develop an acid-base imbalance and increased plasma corticosteroid levels. Furthermore, heat stress can generate oxidative stress and gut flow impairment, and can damage the intestinal epithelium.

Our challenge is to document the link between these concepts and broiler performance. Understanding the effects of heat stress on the physiology of broiler chickens has been the purpose of recent studies. Further investigations into this subject will be valuable not only for tropical regions, but also because of climate changes caused by global warming.

Conflicts of interest

The authors declare they have no conflicts of interest with regard to the work presented in this report.

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