

Effects Of Natural And Artificial Daylengths On Stress Physiology In Poultry Birds

¹Amy Paul , ²Sankha Datta

^{1,2}Research Scholar, Department of Zoology, Gauhati University.

ABSTRACT

Poultry is an important element of the economies of many nations, especially poor ones, and it is also a low-cost and readily available source of animal protein. The primary goal of poultry farming is to provide a stress-free environment for the birds. High stocking density and sickness are only some of the dangers that birds face while living in captivity or captivity-like environments. An animal's genetic potential dictates how much it can produce in an ideal circumstance. It is worth noting that an animal's capacity to unleash its genetic potential is influenced by the environment in which it lives. Providing animals with optimum settings to exhibit their full genetic potential is seldom possible, according to research on how animals are raised in their natural habitats.

Keywords: Immunity, Light management, Poultry, Stress.

I. INTRODUCTION

One environmental or managerial factor influencing poultry productivity, reproduction, and welfare is light, which can be natural or artificially generated [1]. Gonadotropin-releasing hormone (GnRH), produced by the hypothalamus area and stimulates the anterior pituitary to release FSH and L.H., is stimulated by light [2]. Anabolic steroids like testosterone, estrogen, and progesterone are synthesized due to these hormones. The release of yolk precursor lipoproteins from the liver and oviduct by estrogen is critical for follicle development [3]. Lighting also stimulates metabolic hormone production (T3, T4, insulin, G.H., and IGF-1) (T3, T4, insulin, G.H., and IGF-1). These hormones develop and stimulate domestic bird productivity and reproduction [4]. Color, intensity, and photoperiod are all aspects of light. "The effects of light intensity on chicken species, including laying hens, broilers, and turkeys, have been studied extensively in a variety of areas, including productivity, behavior [5], physiology, vision [6], and welfare [7]."

In several of these studies, reduced light levels have been linked to better broiler performance [8]. In other words, low light levels may lead to less physical activity, resulting in more weight gain [9]. Reduced light levels are also thought to lessen the likelihood of cannibalism and pecking behavior. "For broiler

chickens [10], however, a scientific investigation demonstrated that light intensities between 1 and 151 lux did not influence body weight, feed intake (F.I.), or feed conversion (F.C.). Regarding egg production, chickens kept at 5 and 50 lux compared to 1 and 500 lux [11] produced more eggs". When commercial layers aged 18-45 weeks were exposed to varying light intensities, the weights of their [12] ovaries, oviducts, and ovarian large yellow follicles increased. "In contrast, no significant differences in egg production were found with light intensities ranging from 2 to 45 lux [13] and sexual maturity [14]".

The current poultry business is heavily concentrating on animal welfare concerns. Therefore, it underscores the need for research to understand and analyze welfare concerns associated with the chicken-producing business. In addition, customers' worries about poultry welfare are rising in both the meat and egg sectors. Poultry producers seeking to build a profitability program beneficial to most broiler growers must include microclimatic elements (air, temperature, humidity, and light) suggestions as they are crucial to maximizing the profitability and wellbeing of broiler chickens. Besides infections, inadequate management is a key issue that jeopardizes chicken production and wellbeing.

Light is the most critical exogenous factor in poultry production as it regulates physiological and behavioral processes in the bird. Light is important for poultry for

various reasons. Vision is the predominant sense in birds, where the largest proportion of the brain size is devoted to the visual cortex and eyes.

Sunlight is the natural source of light; it is self-generated and comes in a broad spectrum of colors compared to artificial light. The color spectrum of natural light contains the shortest wavelengths representing the ultraviolet part of the spectrum. Edwards [15] reported that ultraviolet light has a beneficial effect on skeletal tissue; this could be explained by the fact that U.V. light increases the absorption of calcium and phosphorus as it was also reported to be responsible for the synthesis of vitamins D. Moreover, birds raised under U.V. free incandescent light had less muscle growth than birds raised under U.V. light also increased fertility in broiler breeders, for sanitation of broiler hatching eggs.”

II. HEAT REGULATION IN POULTRY

Because birds lack sweat glands, they must depend on evaporative cooling and panting to keep themselves cool. Most birds' heat loss happens via breathing since they lack sweat glands. [16] The vast air-sac system in birds' lungs significantly regulates body temperature [16]. When standing, birds in tropical conditions often keep their wings apart slightly to enable air to pass through while also increasing the surface area for heat dissipation. When resting, the birds stretch out their wings to make the most of their available surface area. For heat dissipation, these behavioral reactions all work together to maximize the body surface area in touch with the environment. Temperature-stressed birds spend less time migrating and more time resting in shaded or cool areas, according to research [17]. In addition, birds regulate their body temperature by altering their food and water intake. When the temperature rises, birds tend to eat less food and drink more water. When it is cold outside, the opposite is true.

In most chicken species, chicks cannot effectively control their body temperature throughout the early and post-hatching stages. ‘In the post-hatching period, the chicks must be kept at the proper temperature to prevent them from being chilly and dying within minutes of being born. An effective brooding heat management strategy includes providing the chicks with appropriate heat (artificially). “The first week after hatching, the temperature should be 35°C, with a 3°C weekly decrease until the usual ambient temperature of 23°C - 26°C is reached. The optimal temperature range for

newborn chicks should be between 32 and 35° C, according to Lin et al. [18].” In order to prevent overheating or freezing chicks when moving them from hatcheries, it is essential to keep their temperature within a certain range. According to Abdelazeem [19], high ambient temperatures limit the chicks' development rate and feed consumption. When the temperature rises, chicks become lethargic. Due to this, feed intake is lowered, which leads to a decrease in growth potential. Feeding chicks with high nutritional density chick feeds may help alleviate this issue caused by poor feed intake.

According to research, during the hottest parts of the day, free-range birds seek refuge beneath awnings, but at night, they resume their foraging activities. It has also been seen in housed birds that cease eating during the hottest part of the day and start feeding in the evening. Keeping this in mind can help poultry farmers align their feeding plans with their flock's behavior in places with high ambient temperatures.

III. HEAT STRESS AND REPRODUCTION

According to research, research, research shows that chicken biological functions are negatively affected by high ambient temperatures outside of the thermal-neutral zone. Temperatures between 12°C to 26°C are ideal. Ebeid et al. [20] state that high temperatures significantly impact animal reproduction. When exposed to high temperatures, white leghorn chickens' reproductive activity decreases, leading to reproductive failure and low egg quality, according to the research.

Semen production is affected by ambient temperatures at all phases, according to Fouad et al. [21]. Increased semen quality and quantity may be achieved by stimulating testicular development by raising the ambient temperature slightly during the early stages of semen production. Due to the reduced differentiation of seminiferous epithelial cells, the concentration and volume of semen are reduced when temperatures are high during the middle and late stages of semen production. Flock reproduction is negatively affected because of this, as is the reproductive ability of the cocks in general. Temperature stress has been shown to affect semen quality, as shown by the work of Karaca and colleagues [22].

Researchers found that heat stress adversely affects the hypothalamus and pituitary gland's function Nidamanuri et al. [23]. It has been shown that heat stress

decreases the synthesis of gonadotropin-releasing and luteinizing hormones (GnRH and L.H.), which are linked to higher prolactin levels in one research [24]. "Regression of the ovary due to low GnRH, L.H. and F.H. (stimulating follicle hormone) produces a reduction in the capacity of theca cells, resulting in decreased steroidogenesis, according to Rozenboim and colleagues [25]." Gonadotropin-releasing hormone, plasma progesterone, testosterone, and estradiol hormone levels in heat-stressed birds by Wasti et al. [26], resulting in decreased reproductive efficiency in the species overall. Due to the decreased efficacy, the flock's capacity to reproduce and hence its productivity and profitability are lowered. According to further research, eggs from chickens exposed to high temperatures decreased hatchability. High temperatures reduce the reactivity of granulosa cells, resulting in hormonal imbalances that may interfere with ovulation. Collection of summer-collected semen was used to investigate the impact of heat on spermatozoa features such as bent heads, cytoplasmic droplets, and cut mid-piece abnormalities. "Furthermore, Ayo et al. [27] found that artificial insemination with semen collected during colder intervals of the day (particularly in the morning) resulted in a greater rate of pregnancy."

Its endocrine system and environmental signals influence the animal's reproductive habits. "The length of the day, temperature, rainfall patterns (seasons), human management practises such as feed and feeding systems, animal population interactions and socialisation, and an individual's health and nutritional status are just a few examples of environmental factors that influence reproduction [28]". When one or more of these parameters is significantly altered, the reproductive system may be compromised to varying degrees, according to Taberlet et al. [29]. Environmental influences may affect reproduction in two ways: eventually or more recently, depending on when they occur. Long-term impacts are more important than short-term ones, and here is where the ultimate influencers come in. Animal feed demand and supply must be coordinated. Hence food availability is the most critical environmental element affecting breeding. The temperature of the immediate surroundings has a significant role.

Heat stress (HS) is a commonly established physical environmental stressor that may impact the performance, health, and welfare of poultry [30]. HS

occurs when heat created in the body surpasses its dissipation capabilities, and the body becomes unable to get rid of extra heat [31]. HS has been regarded as a significant environmental ingredient that affects the growth performance of chickens [32]. Various physiological changes, such as systemic immunological dysregulation, endocrine issues, respiratory alkalosis, and electrolyte imbalance are experienced by hens exposed to HS [33] and these ultimately influence their growth performance and intestinal barrier function. By altering thermoregulation, HS may boost mortality rates [34] and change the physiological and behavioural responses of birds. For example, birds spend less time on eating and walking, but more time is spent on drinking, resting, and breathing under HS demanding situations. In a study done by Branco et al. [35], birds exposed to HS were prostrate, had shorter behavioural responses such as laying down and eating. Birds sought for adaptation in HS circumstances by increasing water intake (that helps enhance heat loss), lowering meal consumption and increasing lying down to minimise heat production through movement and increase panting to promote evaporative cooling.

HS condition has been proven to boost HSP expression [36], rectal temperature, heterophil:lymphocyte ratios (H/L ratio), and plasma corticosterone [37]. An rise in corticosterone as a response to HS increases muscle protein breakdown and gives amino acid substrates to liver gluconeogenesis that is responsible for energy supply. The increase in corticosterone levels might boost oxidative stress, induce cell death in follicular cells, leading to a decline in follicle numbers and therefore reducing egg production. In layers, Allahverdi et al. [38] showed that high temperatures drastically affected egg quality, egg weight, shell weight, egg specific gravity, and shell thickness. Aside from the loss in egg quality, HS has been reported to delay sexual development in layers. Zhao et al. [39] showed that high-temperature levels impact the viability of granulosa cells in laying hens. Broilers are more sensitive to HS than layers. In the past, studies have indicated that broilers are more vulnerable to the negative effects of HS on body temperature, plasma creatine, and skeletal muscles. A higher level of oxidative stress and decreased antioxidant status in broilers have been linked to HS since it has been proven to induce lipid peroxidation and impair SOD activity. According to study, HS has been demonstrated to change the blood biochemistry of chickens. According

to Gharib et al., plasma albumin, serum calcium (Ca), and red blood cell count decreased significantly in pullets exposed to HS (40). Cyclic hypoglycemia had no impact on uric acid, total protein, albumin, globulin, or AST activity, according to a research by Bueno et al. [41].

HS has been associated to a number of changes in the intestinal morphology, gut microbiota, and intestinal integrity in poultry [42]. Tight junction relaxation occurs during HS, which compromises the intestinal barrier function and leads to increased intestinal permeability. [43] As a result, there is an increase in foodborne disease risk due to an increase in intestinal permeability. The small intestinal mucosa of HS patients showed a reduction in villous height and crypt depth. Researchers have shown that birds who have been exposed to high temperatures lose the depth of their crypts, the height of their villus, and their epithelial cell area ratio. Salmonella and E. coli may flourish in chickens given a high-sulfur diet, according to certain research findings. It has been suggested by Tsiouris and colleagues that HS may be the cause of necrotic enteritis epidemics in hens [30]. It is possible that the loss of protecting microorganisms in the digestive tract of HS-treated broilers led to decreased pH values in the digesta. According to this theory, high temperatures affect the cecum microbiota's diversity and disrupt chickens' gut bacteria's equilibrium by boosting Bacteroidetes and decreasing Euryarchaeota. GIT organs in chickens are particularly susceptible to HS [35], however the reaction depends on the digestive tract segment. "In the chicken jejunum, heat-stressed broilers demonstrated significant mRNA up-regulation of HSF3 (heat shock factor), while HS exposure increased the levels of HSF1 and HSF3 mRNA in the ileum, A greater quantity of HSF and HSP was found in the chicken ileum compared to the jejunum. HSF1 and HSF3 are two of the most important members of the HSF family in the regulation of chicken's heat-stress response (HSR). HSPs are classified based on their molecular mass. A few HSP courses are more popular than others."

The immune and digestive systems of birds are weakened by HS, making them more vulnerable to sickness in the first place. A link between the TLR4-NF-B signalling system and E. coli-induced intestinal inflammation in chickens was discovered by HS. "Quinteiro-Filho et al. [46] discovered that HS activated

the hypothalamus-pituitary-adrenal (HPA) axis, increased Salmonella enteritidis infection, and weakened the chicken immune system". The HPA axis, a part of the nervous system, governs biological functioning as well as the body's reaction to stressful situations. In response to increased synthesis of the hormone corticosterone, which is produced when the HPA axis is stimulated by stress, lower levels of Toll-like receptors and IgA are seen [46]. A decrease in B lymphocytes and an increase in T-cytotoxic suppressors and T-helpers in broiler chicken blood were two additional effects of HS discovered by Honda et al. It's possible that chickens' immune systems may also be altered by the HS's impacts. "According to Quinteiro-Filho et al., it was shown that HS decreased the relative weight of numerous immune organs (bursa, spleen, and thymus)." Reduced feed intake and development, lower feed efficiency, decreased egg quality and quantity are all linked to HS in hens, as is an increase in oxidative stress. Poultry oxidative stress may be exacerbated by stressors, such as nutritional or high-stress environments.

IV. DEVELOPMENT OF IMMUNE RESPONSE IN POULTRY

Immunocompetence develops just a few days after hatching, although the birds' defensive mechanisms begin to develop throughout embryonic life. "Embryos and chicks are temporarily protected against microbial infection by maternal antibodies present in the yolk during incubation and immediately after hatching". Because it does not stimulate antibody production, immunizing a 1-day-old chick is ineffective. When vaccinating chicks, the secondary lymphoid tissues have developed to their full extent by one-week post-hatch, allowing for a successful immune response with specific antibody production [47]. The post-hatching phase, which lasts for about a week, is essential due to the lack of maternal immunity, which may be found in abundance in mammalian colostrum.

Immunocompetence undergoes various modifications due to growth, reproduction, nutritional changes, and stress. This may be due to immune cell receptors that react differently to endocrine signaling molecules such as sex and metabolic hormones. Some effector processes may be increased or decreased due to these hormones' immunomodulation (i.e., immunosuppression or immunostimulation). Birds'

immune systems may be rebalanced to adapt to environmental changes and life phases.

V. STRESS TYPES AND THEIR IMPACT ON THE IMMUNE SYSTEM OF BIRDS

An adaptive reaction to dangers that threatens a bird's homeostatic equilibrium might be characterized as "stress" in this context. Stimuli may come from the outside world or the inside, and the bird's reaction varies depending on the stressor's intensity and duration and the bird's physiological state. Temperature, air quality, mycotoxins, pesticides, general cleanliness, and food composition changes are some stressors that might affect a plant's ability to produce food [48].

A. Temperature stressors

Animals such as birds are homoeothermic in nature. If they are reared at a temperature between 21 and 28 degrees Celsius, they can keep their internal organs at a stable temperature. Animals are subjected to heat or cold stress when the temperature of the surroundings exceeds the thermoneutral zone. The quality of chicken products (meat or eggs), fertility, and disease resistance are all significantly impacted by heat stress. As a result, birds cannot display their high-productive genotypes under high temperatures and relative humidity.

Additionally, the quality of the meat is lowered after slaughter due to heat stress, which affects feed intake, feed conversion ratio, and body weight. "Both their feather covering and the lack of sweat glands make birds more susceptible to heat stress than other

household animals [49]." As a result, chicken strains range in their ability to withstand heat stress, with birds with a rapid development rate being the least tolerant. Commercial broilers are especially susceptible to heat stress because of their fast development and high metabolic rate. As a result, choosing a broiler strain for usage in hot and warm climates requires careful consideration of its capacity to regulate temperature [50].

Lymphocyte-mediated responses are often inhibited by very high temperatures, resulting in various modifications in the bird's [51] humoral immune response. Aside from decreasing immune system function, exposure to high temperatures also depresses IgM and IgG levels in the blood as well as the number of lymphocytes in the body. "As a consequence of the altered macrophage activity produced by heat stress, inflammation of the jejunal epithelium and increased Salmonella enteritidis pathological lesions were also seen" [51, 52].

When the body is exposed to heat stress, the levels of adrenocorticotropin-tropic hormone (ACTH) and the brand name hormone (CRH) rise, affecting the GIT and causing an increase in corticosteroid tiers, which in turn promotes lipogenesis, fat deposition, and protein catabolism. "This is Fig (Fig. 1), The formation of heat shock protein (HSP) in immune cells such as heterophils, monocytes, and lymphocytes is also influenced by heat stress".

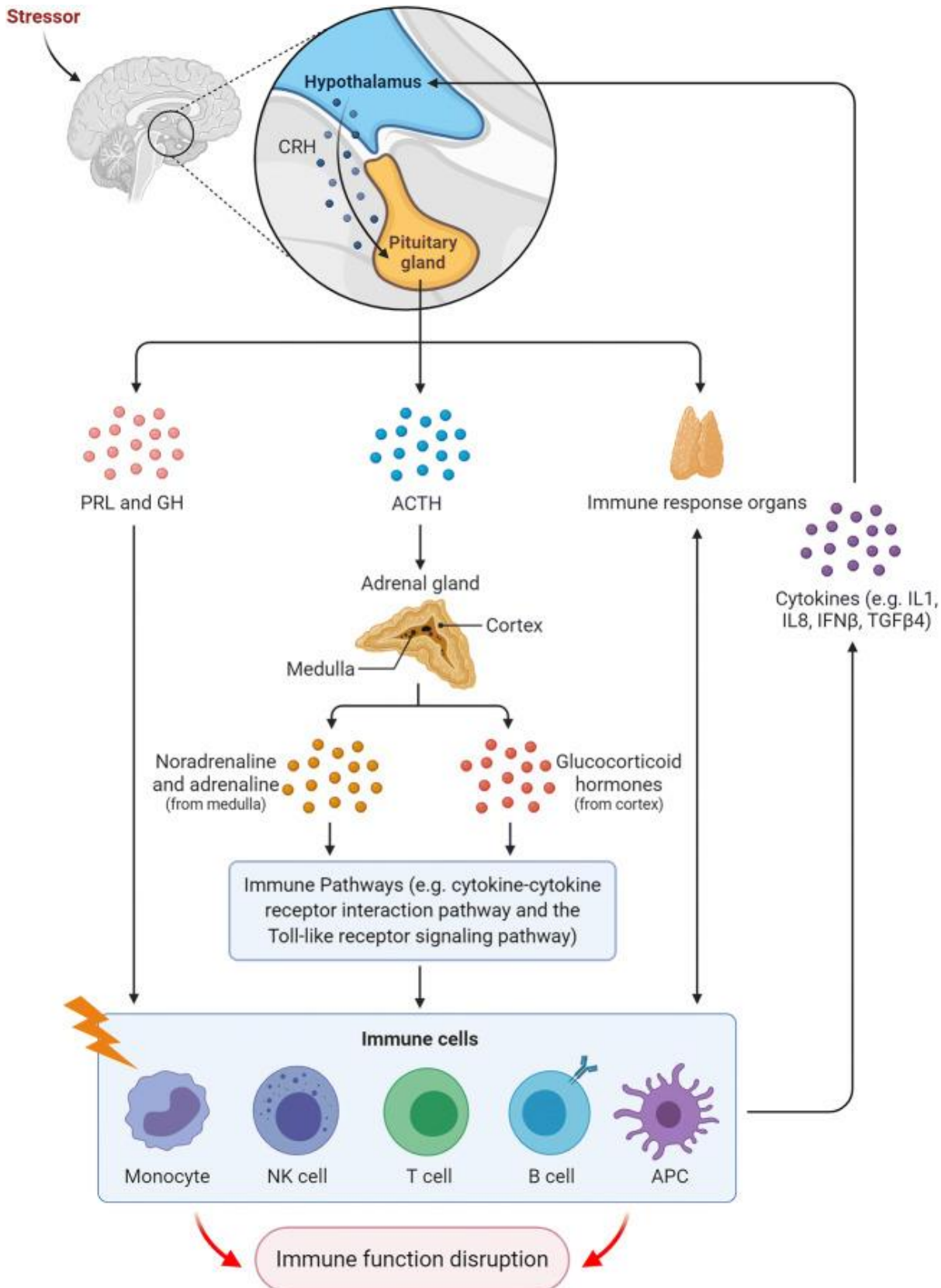


Fig. 1: Shock-induced central nervous system controls the hypothalamic-pituitary-adrenal system and immunological responses. Hormones are released as a result of stress. For example, stress-induced corticosteroid overdose activates several immunological mechanisms (e.g., cytokine-cytokine receptor interaction and the Toll-like receptor signaling pathways). “Interleukins are immunological mediators produced by the body in response to inflammatory stimuli. The adrenocorticotropic hormone, the corticotropin-releasing hormone, the growth

hormone, the interferon, the interleukin, and the prolactin hormone all fall under the umbrella term of adrenocorticotrophic hormone" (ACTH).

Exposure to colder temperatures (cold stress) might alter the immune response and affect the severity of infectious infections. A bird's performance may be impacted by cold stress in one of two ways: reduced body mass or decreased egg output. As a result, stress hormones are released by the hypothalamus-pituitary-adrenal (HPA) axis, which is responsible for restoring homeostasis. Due to prolonged exposure to low temperatures and increasing corticosteroid levels, birds are more susceptible to illnesses, such as respiratory infections [53] and metabolic abnormalities [54]. Examples include necrotic enteritis in broiler chickens, pulmonary hypertension, and colibacillosis in turkey poults [55], Corticosterone levels are reduced, and T3 levels are increased as a result of cold stress in order to create more metabolic heat to assist in maintaining body

temperature in colder conditions [56]. "iNOS (inducible nitric oxide synthase) mRNA and free Nrf2 (nuclear factor (erythroid-derived 2)-like 2; NFE2L2) protein are overexpressed in hens exposed to cold stress, increasing oxidative stress." The Nrf2/ARE (antioxidant response element) signaling pathway (Fig. (Fig.2),2) induces the antioxidative stress response through Nrf2. It upregulates the production of the fatty acid-binding protein (L-FABP), which inhibits membrane lipid peroxidation and increases fatty acid storage (Fig. As a result, chickens' humoral and cell-mediated immunity is reportedly reduced by cold stress. Cold stress increased the synthesis of IL10 [57] and IL2 (Jansk et al. 1996) while decreasing phagocytic activity, tumor necrosis factor (TNF), and IL6 production [58].

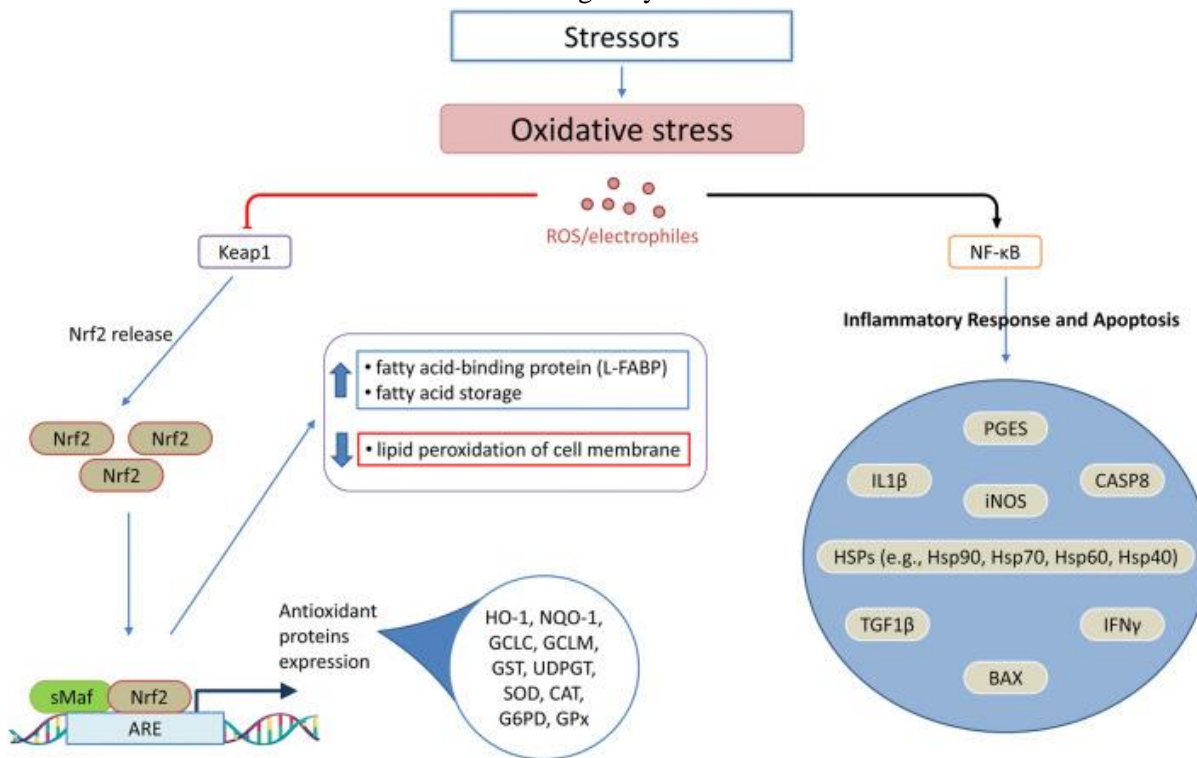


Fig. 2: "Stressors such as cold or heat may cause oxidative stress. In response to oxidative stress, Nrf2/ARE signalling and NF-B are activated, leading to the production of antioxidant proteins, inflammation, and apoptosis. Catalase; G6PD dehydrogenation; GCLC and GCLM, glutamate-cysteine-ligase subunits; G6PD dehydrogenation; ARE, antioxidant response element; CASP8, caspase-8; CAT, catalase; G6PD dehydrogenation IFN, interferon, I.L., interleukin, iNOS, kelch-like ECH-associated protein 1, NF-B, nuclear factor kappa light-chain enhancer of activated B cells, NAD(P)H quinone oxidoreductase-1, PGES, prostaglandin E synthase, reactive oxygen species, and ROS, reactive oxygen species are all abbreviations for glutathione peroxidase"

A reduced temperature environment also impacts the formation of antioxidants. Cold stress reduced SOD and

GPx activity and raised malondialdehyde (MDA) levels while decreasing antioxidant enzyme activities. "High

expression of antioxidants such as Hsp27 (HSP40), HSPR60 (HSPR70), and HSPR90 (HSPR90) was shown to be a protective response against cold-inducible stress-induced oxidative damage". These modulations are influenced by the duration and severity of the stressor.

B. Other environmental stressors

Changing rearing procedures, stocking density, farm location, and other physical variables may induce a stress reaction and regulate the immunological response. The cutaneous basophil hypersensitivity reactions were shown to be affected by slatted flooring and high breeding density, increasing the heterophil-to-lymphocyte (H/L) ratio and duration of tonic immobility, and lowering antibody titer. Larking hens were allowed to perch naturally, and the H/L ratio decreased. Variations in immunological responses occur dependent on the species and the geographic region of the farm. Growing birds like sparrows (*Passer domesticus*) in a temperate setting have raised baseline corticosterone levels because of changes in photoperiod and increasing parasite burdens.

Overpopulation drastically impacts poultry's immunological response by limiting the development of immune organs. [59] A predisposing factor for human disease [60], a decrease in nutritional uptake, and a drop in growth performance are all associated with it. The plasma IgG levels and macrophage cell lysis of macrophages were likewise lowered by higher stocking density. Corticosterone levels are elevated due to the interaction between the hypothalamic-pituitary-adrenal axis. "Antibody and cell-mediated responses are generally reduced under unfavorable settings". Other pituitary and hypothalamus "ACTH-like" and "-endorphin-like" peptides created by cells of the immune system may operate as initiators of the immune response and connect with the central route (the nerve system), as well as the central pathway. "Exposure to pollutants or pathogenic stress during development may also magnify the impact of other stresses considerably."

VI. DISCUSSION

Natural or artificial light is one of the most significant management elements, which plays a crucial role in controlling poultry production and reproduction. "This happens by altering many behavioral and physiological processes of birds". The findings of the current experiment showed a non-significant effect of the access of natural daylight on the activity of broiler

chickens, where the birds in the two different experimental treatments showed similar rates of standing, walking, and laying down behaviors over the other observation weeks and the different periods of the day [61, 62]. This indicates that introducing natural daylight into poultry houses was a non-effective environmental modification in increasing the physical activity of broilers. However, there was a significant increase in the rates of expression of sleeping behavior in the 5th week of age in birds kept entirely under artificial light compared to those with access to natural light during the day. "These results agree with Ruis et al. [63] who reported no difference in activity between birds kept under artificial light and those allowed access to an outside run with natural light."

The results of this study showed that natural daylight had a strong effect on the expression of ingestive behaviors by broiler chickens. Birds with access to natural light showed higher rates of eating behavior in the 4th week of age and day session and drinking behavior in the 2nd week of age and night session. The light could explain that natural light provided a better vision and a higher motivation to feed and drink than birds kept entirely under artificial light. Chickens are known to have a well-developed sense of vision; they are originally designed to be used in bright light environments. This suggests that natural light is required by broiler chickens to use their sense of vision to its full potential and to maximize their ability to express natural behavioral patterns.

Moreover, this data showed that birds with access to natural light had higher rates of preening in the 4th week of age and leg stretching in the 5th week. This effect may be due to the greater illuminance and the broader wavelength spectrum of daylight. This is in agreement with Khalil et al. [65], Who found that birds kept in the natural light group spent more time preening when compared to other different artificial light intensities.

The measured production parameters were unaffected by the introduction of natural daylight into poultry houses. However, birds exposed to natural light recorded significantly heavier weights in the 5th week of age. Moreover, food intake tended to be significantly higher in birds in natural light groups. This indicates a distinctive preference by broiler chickens for natural daylight. Moreover, incorporating natural light into light regimes was more optimal for broilers' performance when compared to regimes depending

entirely on artificial light. However, the real benefit of this advantage can be taken only during summertime. A previous study reported that the color of cold white light, which contains wavelengths from the blue part of the spectrum, resembles the color temperature of natural daylight. Riber et al. [66] Reported that birds showed higher body weights when reared under the cold white light than neutral white light at the slaughter age.

The results of the current study showed that natural light was effective in reducing leg abnormalities commonly manifested by broiler chickens. Gait score was significantly lower in birds exposed to natural light; none of these birds manifested a gait score that could impair their mobility or walking ability. While some of the birds in the artificial light groups manifested a high gait score, greatly impacting their walking ability, some experienced complete lameness, which is considered a strong indication of inadequate welfare. It has been shown that it is possible to improve lameness in broilers with high gait scores by treating them with analgesics and anti-inflammatory drugs. This has proved the assumption that leg disorders in broilers are painful.

VII. CONCLUSION

The poultry business in the tropics and the rest of the globe are dealing with the difficulty of heat stress. Global warming has caused temperatures to rise in recent years. The output and quality of chicken products are negatively impacted by heat stress. In addition, the advancements in poultry welfare are hampered by this practice. However, there is still a need for more study into the effects of heat stress on free-range chickens in the tropics and the best ways to mitigate it.

REFERENCES

- [1]. Prescott, N.B., H.H. Kristensen, and C.M. Watches, 2004. Light, In *Measuring and Auditing Broiler Welfare*. Weeks, C. and A. Butterworth (Eds.), CAB Int., Wallingford, UK, pp: 101-116
- [2]. Chen, K.L., W.T. Chi and P.W.S. Chiou, 2005. Caponization and testosterone implantation affect male chickens' blood lipid and lipoprotein profiles. *Poult. Sci.*, 84: 547-552
- [3]. Dawson, A., 2005. Seasonal differences in the secretion of luteinizing hormone and prolactin in response to N-methyl-DL-aspartate in starlings (*Sturnus vulgaris*). *J. Neuroendocrinol.*, 17: 105-110.

- [4]. Mateescu, R.G. and M.L. Thonney, 2005. Effect of testosterone on insulin-like growth factor-I, androgen receptor, and myostatin gene expression in splenius and semitendinosus muscles in sheep. *J. Anim. Sci.*, 83: 803-809.
- [5]. Lewis, P.D., P.J. Sharp, P.W. Wilson, and S. Leeson, 2004. Changes in light intensity can influence age at sexual maturity in domestic pullets. *Br. Poult. Sci.*, 45: 123-132
- [6]. Ahmad, F., A.U. Haq, M. Ashraf, G. Abbas, and M.Z. Siddiqui, 2011. Effect of different light intensities on the production performance of broiler chickens. *Pak. Vet. J.*, 31: 203-206
- [7]. Yahav, S., S. Hurwitz and I. Rozenboim, 2000. The effect of light intensity on growth and development of turkey toms. *Br. Poult. Sci.*, 41: 101-106.
- [8]. Lien, R.J., J.B. Hess, S.R. McKee and S.F. Bilgili, 2008. Effect of light intensity on live performance and processing characteristics of broilers. *Poult. Sci.*, 87: 853-857
- [9]. Alvino, G.M., R.A. Blatchford, G.S. Archer, and J.A. Mench, 2009. Light intensity during rearing affects broiler chickens' behavioral synchrony and resting patterns. *Br. Poult. Sci.*, 50: 275-283
- [10]. Lewis, P.D., N. Ciccone, P.W. Wilson and S. Leeson, 2005. The light intensity can influence plasma FSH and age at sexual maturity in domestic pullets. *Br. Poult. Sci.*, 46: 506-509.
- [11]. Prescott, N.B., C.M. Watches and J.R. Jarvis, 2003. Light, vision, and the welfare of poultry. *Anim. Welfare*, 12: 269-288.
- [12]. Deep, A., K. Schwan-Lardner, T.G. Crowe, B.I. Fancher and H.L. Classen, 2010. Effect of light intensity on broiler production, processing characteristics, and welfare. *Poult. Sci.*, 89: 2326-2333.
- [13]. Hill, J.A., D.R. Charles, H.H. Specter, R.A. Bailey, and A.J. Ballantyne, 1988. Effects of multiple environmental and nutritional factors in laying hens. *Br. Poult. Sci.*, 29: 499-511
- [14]. Renema, R.A. and F.E. Robinson, 2001. Effects of light intensity from photostimulation in four strains of commercial egg layers: 1. Ovarian morphology and carcass parameters. *Poult. Sci.*, 80: 1112-1120.
- [15]. Edwards, H. M. (2003). Effects of UVB irradiation of very young chickens on growth

- and bone development. *Br. J. Nutr.* 90: 151-160.
- [16]. Donald DB, William DW. Commercial Chicken Meat and Egg Production. In *Commercial Chicken Meat and Egg Production*. Springer US. 2002
- [17]. Mack LA, Felver-Gant JN, Dennis RL, Cheng HW. Genetic variations alter production and behavioral responses following heat stress in 2 strains of laying hens. *Poultry Science*, 2013;92:285-294
- [18]. Lin H, Jiao HC, Buyse J, Decuypere E. Strategies for preventing heat stress in poultry. In *World's Poultry Science Journal*. 2006;62:1
- [19]. Abdelazeem MH. Effect of chronic heat stress on broiler chicks' performance and immune system. 2007
- [20]. Ebeid TA, Suzuki T, Sugiyama T. High ambient temperature influences eggshell quality and calbindin-D28k localization of eggshell gland and all intestinal segments of laying hens. *Poultry Science*. 2012;91:2282-2287
- [21]. Fouad AM, Chen W, Ruan D, Wang S, Xia WG, Zheng CT. Impact of Heat Stress on Meat, Egg Quality, Immunity, and Fertility in Poultry and Nutritional Factors That Overcome These Effects: A Review. *International Journal of Poultry Science*. 2016;15: 81-95
- [22]. Karaca AG, Parker HM, Yeatman JB, McDaniel CD. The effects of heat stress and sperm quality classification on broiler breeder male fertility and semen ion concentrations. *British Poultry Science*. 2002;43:621-628
- [23]. Nidamanuri A, Murugesan S, Mahapatra R. Effect of Heat Stress on Physiological Parameters of Layers: A Review. *International Journal of Livestock Research*. 2017; 1
- [24]. Hester PY, Muir WM, Craig JV, Albright JL. Group Selection for Adaptation to Multiple-Hen Cages: Production Traits During Heat and Cold Exposures. 1996; 1:2
- [25]. Rozenboim I, Tako E, Gal-Garber O, Proudman JA, Uni Z. The Effect of Heat Stress on Ovarian Function of Laying Hens
- [26]. Wasti S, Sah N, Mishra B. Impact of heat stress on poultry health and performances, and potential mitigation strategies. *Animals*, 2020;10:1-19
- [27]. Ayo JO, Obidi JA, Rekwot PI. Effects of Heat Stress on the Well-Being, Fertility, and Hatchability of Chickens in the Northern Guinea Savannah Zone of Nigeria: A Review. *ISRN Veterinary Science*, 2011; 1-10
- [28]. King LM, Brillard JP, Bakst MR, Donoghue AM. Segregation of spermatozoa within sperm storage tubules of fowl, turkey hens, and turkey hens. 2002
- [29]. Taberlet P, Coissac E, Pansu J, Pompano F. Conservation genetics of cattle, sheep, and goats. *Comptes rendus biologiques* 2011;334:247-254
- [30]. Tsiouris, V., Georgopoulou, I., Batzios, C., Pappaioannou, N., Ducatelle, R., and Fortomaris, P. (2018). Heat stress as a predisposing factor for necrotic enteritis in broiler chicks. *Avian Pathol.* 47:24574. doi: 10.1080/03079457.2018.1524574
- [31]. Lara, L.J., and Rostagno, M.H. (2013). Impact of heat stress on poultry production. *Animals*. 3, 356–369. doi: 10.3390/ani3020356
- [32]. Goo, D., Kim, J. H., Park, G. H., Delos Reyes, J. B., and Kil, D. Y. (2019). Effect of heat stress and stocking density on growth performance, breast meat quality, and intestinal barrier function in broiler chickens. *Animals*. 9:107. doi: 10.3390/ani9030107
- [33]. Teeter, R. G., Smith, M. O., Owens, F. N., Arp, S. C., Sangiah, S., and Breazile, J. E. (1985). Chronic heat stress and respiratory alkalosis: Occurrence and treatment in broiler chicks. *Poult. Sci.* 64, 1060–1064. doi: 10.3382/ps.0641060
- [34]. Saiz del Barrio, A., Mansilla, W. D., Navarro-Villa, A., Mica, J. H., Smeets, J. H., den Hartog, L. A., et al. (2020). Effect of mineral and vitamin C mix on growth performance and blood corticosterone concentrations in heat-stressed broilers. *J. Appl. Poultry Res.* 29, 23–33. doi: 10.1016/j.japr.2019.11.001
- [35]. Branco, T., Moura, D. J., de de Alencar Nääs, I., da Silva Lima, N. D., Klein, D. R., Oliveira, S. R., et al. (2021). The sequential behavior pattern analysis of broiler chickens exposed to heat stress. *AgriEng.* 3, 447–457. doi: 10.3390/agriengineering3030030
- [36]. Vinoth, A., Thirunalasundari, T., Tharian, J. A., Shanmugam, M., and Rajkumar, U. (2015).

- Effect of thermal manipulation during embryogenesis on liver heat shock protein expression in chronic heat stressed colored broiler chickens. *J. Therm. Biol.* 53, 162–171. doi: 10.1016/j.jtherbio.2015.10.010
- [37]. Soleimani, A. F., Zulkifli, I., Omar, A. R., and Raha, A. R. (2011). Physiological responses of 3 chicken breeds to acute heat stress. *Poult. Sci.* 90, 1435–1440. doi: 10.3382/ps.2011-01381
- [38]. Allahverdi, A., Feizi, A., Takhtfooladi, H.A., and Nikpiran, A. (2013). Effects of heat stress on acid-base imbalance, plasma calcium concentration, egg production and egg quality in commercial layers. *Glob. Veterinaria.* 10, 203–207. doi: 10.5829/idosi.gv.2013.10.2.7286
- [39]. Zhao, Q., Xue, W., Zhang, S., Guo, Y., Li, Y., Wu, X., et al. (2021). The functions of Patchouli and Elsholtzia in the repair of hen follicular granular cells after heat stress, *Poultry Science* 21:101306, doi: 10.1016/j.psj.2021.101306
- [40]. Gharib, H. B. A., El- Menaway, M. A., Attalla, A. A., and Stino, F. K. R. (2005). Response of commercial layers to housing at different cage densities and heat stress conditions. 1-Physiological indicators and immune response. *Egypt. J. Anim. Product.* 42, 47–70. doi: 10.21608/ejap.2005.93011
- [41]. Bueno, J. P. R., Nascimento, M. R. B., de, M., Martins, J. M. da, S., Marchini, C. F. P., Gotardo, L. R. M., et al. (2017). Effect of age and cyclical heat stress on the serum biochemical profile of broiler chickens, *Semina: Ciências Agrárias, Londrina.* 38, 1383–1392. doi: 10.5433/1679-0359.2017v38n3p1383
- [42]. Song, J., Xiao, K., Ke, Y. L., Jiao, L. F., Hu, C. H., Diao, Q. Y., et al. (2014). Effect of a probiotic mixture on intestinal microflora, morphology, and barrier integrity of broilers subjected to heat stress. *Poult. Sci.* 93, 581–588. doi: 10.3382/ps.2013-03455
- [43]. Gupta, A., Chauhan, N.R., Chowdhury, D., Singh, A., Meena, R. C., Chakrabarti, A., and Singh, S. B. (2017). Heat stress modulated gastrointestinal barrier dysfunction: Role of tight junctions and heat shock proteins. *Scand. J. Gastroentero.* 52, 1315–1319 doi: 10.1080/00365521.2017.1377285
- [44]. Santos, R. R., Awati, A., Roubos-van den Hil, P. J., Tersteeg-Zijderfeld, M. H., Koolmees, P. A., and Fink-Gremmels, J. (2015). Quantitative histo-morphometric analysis of heat-stress-related damage in the small intestines of broiler chickens. *Avian Pathol. J. W.V.P.A.*, 44, 19–22. doi: 10.1080/03079457.2014.988122
- [45]. Akinyemi, F. T., Bello, S.F., Uyanga, V.A., Oretomiloye, C., and Meng, H. (2020). Heat Stress and gut microbiota: effects on poultry productivity. *Int. J. Poult. Sci.* 19, 294–302 doi: 10.3923/ijps.2020.294.302
- [46]. Quinteiro-Filho, W. M., Calefi, A. S., Cruz, D., Aloia, T., Zager, A., Astolfi-Ferreira, C. S., et al. (2017). Heat stress decreases expression of the cytokines, avian β -defensins 4 and 6 and Toll-like receptor 2 in broiler chickens infected with *Salmonella Enteritidis*. *Vet. Immunol. Immunopathol.* 186, 19–28. doi: 10.1016/j.vetimm.2017.02.006
- [47]. Honda, B. T., Calefi, A. S., Costola-de-Souza, C., Quinteiro-Filho, W. M., da Silva Fonseca, J. G., de Paula, V. F., et al. (2015). Effects of heat stress on peripheral T and B lymphocyte profiles and IgG and IgM serum levels in broiler chickens vaccinated for Newcastle disease virus. *Poult. Sci.* 94, 2375–2381. doi: 10.3382/ps/pev192
- [48]. Yasuda M, Kajiwara E, Ekino S, Taura Y, Hirota Y, Horiuchi H, Matsuda H, Furusawa S. Immunobiology of chicken germinal center: I. Changes in surface Ig class expression in the chicken splenic germinal center after antigenic stimulation. *Dev Comp Immunol.* 2003;27:159–166. doi: 10.1016/s0145-305x(02)00066-6.
- [49]. Dietert RR, Golemboski KA, Austic RE. Environment-immune interactions. *Poult Sci.* 1994;73:1062–1076. doi: 10.3382/ps.0731062
- [50]. El-Naggar K, El-Kassas S, Abdo SE, Kirrella AAK, Al Wakeel R.A. Role of gamma-aminobutyric acid in regulating feed intake in commercial broilers reared under normal and heat stress conditions. *J Therm Biol.* 2019;84:164–175. doi: 10.1016/j.jtherbio.2019.07.004.
- [51]. Wang Y, Saelao P, Chanthavixay K, Gallardo R, Bunn D, Lamont SJ, Dekkers JM, Kelly T, Zhou H. Physiological responses to heat stress

- in two genetically distinct chicken inbred lines. *Poult Sci.* 2018;97:770–780. doi: 10.3382/ps/pex363.
- [52]. Yalcin S, Özkan S, Türkmüt L, Siegel PB. Responses to heat stress in commercial and local broiler stocks. 1. Performance traits. *Br Poult Sci.* 2001;42:149–152. doi: 10.1080/00071660120048375.
- [53]. Dohms JE, Metz A. Stress—mechanisms of immunosuppression. *Vet Immunol Immunopathol.* 1991;30:89–109. DOI: 10.1016/0165-2427(91)90011-z.
- [54]. Quinteiro-Filho WM, Gomes AVS, Pinheiro ML, Ribeiro A, Ferraz-de-Paula V, Astolfi-Ferreira CS, Ferreira AJP, Palermo-Neto J. Heat stress impairs performance and induces intestinal inflammation in broiler chickens infected with *Salmonella Enteritidis*. *Avian Pathol.* 2012;41:421–427. doi: 10.1080/03079457.2012.709315. [[PubMed](#)] [[CrossRef](#)] [[Google Scholar](#)]
- [55]. Quinteiro-Filho WM, Rodrigues MV, Ribeiro A, Ferraz-de-Paula V, Pinheiro ML, Sá LRM, Ferreira AJP, Palermo-Neto J. Acute heat stress impairs performance parameters and induces mild intestinal enteritis in broiler chickens: role of acute hypothalamic-pituitary-adrenal axis activation. *J Anim Sci.* 2012;90:1986–1994. doi: 10.2527/jas.2011-3949. [[PubMed](#)] [[CrossRef](#)] [[Google Scholar](#)]
- [56]. Siegel HS. Immunological responses as indicators of stress. *Worlds Poult Sci J.* 1985;41:36–44. DOI: 10.1079/WPS19850003
- [57]. Sato T, Tezuka K, Shibuya H, Watanabe T, Kamata H, Shirai W. Cold-induced ascites in broiler chickens and its improvement by temperature-controlled rearing. *Avian Dis.* 2002;46:989–996. doi: 10.1637/0005-2086(2002)046[0989:CIAIBC]2.0.CO;2.
- [58]. Tsiouris V, Georgopoulou I, Batzios C, Pappaioannou N, Ducatelle R, Fortomaris P. The effect of cold stress on the pathogenesis of necrotic enteritis in broiler chicks. *Avian Pathol.* 2015;44:430–435. doi: 10.1080/03079457.2015.1083094
- [59]. Hangalapura BN, Nieuwland MG, de Vries RG, Heetkamp MJ, Van den Brand H, Kemp B, Parmentier HK. Effects of cold stress on immune responses and body weight of chicken lines divergently selected for antibody responses to sheep red blood cells. *Poult Sci.* 2003;82:1692–1700. doi: 10.1093/ps/82.11.1692.
- [60]. Sesti-Costa R, Ignacchiti M, Chedraoui-Silva S, Marchi L, Mantovani B. Chronic cold stress in mice induces a regulatory phenotype in macrophages: correlation with increased 11β -hydroxysteroid dehydrogenase expression. *Brain Behav Immun.* 2012;26:50–60. DOI: 10.1016/j.bbi.2011.07.234
- [61]. Campo J, Gil M, Davila S, Munoz I. Influence of perches and footpad dermatitis on tonic immobility and heterophil to lymphocyte ratio of chickens. *Poult Sci.* 2005;84:1004–1009. doi: 10.1093/ps/84.7.1004.
- [62]. Heckert RA, Estevez I, Russek-Cohen E, Pettit-Riley R. Effects of density and perch availability on the immune status of broilers. *Poult Sci.* 2002;81:451–457. doi: 10.1093/ps/81.4.451.
- [63]. Tsiouris V, Georgopoulou I, Batzios C, Pappaioannou N, Ducatelle R, Fortomaris P. High stocking density as a predisposing factor for necrotic enteritis in broiler chicks. *Avian Pathol.* 2015;44:59–66. doi: 10.1080/03079457.2014.1000820.
- [64]. Ruis, M. A. W., Coenen, E., Harn, J. V. and Rodenburg, B. (2004). Effect of an outdoor run and natural light on the welfare of fast-growing broilers. In: *Proceedings 38th ISAE-congress*. Helsinki, Finland. Page: 255.
- [65]. Khalil, H. A., Hanafy. A. M. and Hamdy, M. M. (2016). Effect of artificial and natural daylight Intensities on some behavioral activities, plumage conditions, effective and physiological changes for Japanese quail. *Asian. J. Poult. Sci.* 10: 52-63.
- [66]. Riber, A. B. (2015). Effects of light color on preferences, performance, and welfare in broilers. *Poult. Sci.* 8: 1721- 1728