Review Article

Effect of Heat Stress on Physiological Parameters of Layers - A Review

Anand Laxmi Nidamanuri*, Shamugam Murugesan and Ram Krishna Mahapatra
ICAR-Directorate of Poultry Research, Rajendranagar, Hyderabad-500030, INDIA

***Corresponding author:** alnand44@gmail.com

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Abstract
Layer flocks are reared for egg production. In the present scenario, climate change is having important effect on agriculture and farm animal production. Rise in environmental temperature is of major concern since it leads to heat stress. Increase in demand for chicken product's protein supply also, puts challenge to the environment. Technologies are being developed for the intensification of poultry production. We have to still develop new technologies which will be viable and suit according to the climate change and become resilient to heat stress. Beyond the thermoneutral zone or at high ambient temperatures the egg production of layer decreases necessitating to manage heat stress properly. The decrease in egg productivity is dependent on different variables. In the present review, various physiological parameters in layers have been discussed which are being affected by heat stress, either naturally or under artificial conditions. Based on the physiological variables mentioned, other variables have to be explored, which if managed, may result in better egg production.

Key words: Layers, Egg Production, Heat Stress

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Introduction

Climate change, in particular global warming, will affect the health and welfare of farm animals, both directly and indirectly. Effects of global warming include temperature-related illness and death, and also the effect on animals during extreme weather conditions. Indirect effects include pathways that include those, where organisms try to adapt to higher thermal environment. Animals try to adjust to higher ambient temperatures through various means to reduce metabolic heat production. Future climate changes will be variable according to the locations, some model climate projections suggest that precipitation will increase at high latitudes, and will decrease in the tropical and subtropical land regions (IPCC, 2007). Climate changes will also influence crop production, grain yields and further availability of grains as feed for livestock production systems. Heat stress reduces the reproductive performance of laying hens by

interrupting egg production. This may be due not only by reduction in feed intake but also by disruption of hormones involved in reproduction (Donoghue *et al.,* 1989). Abiotic and biotic factors contribute to environmental stress. When the extreme changes occur in the environment, it stimulates the body responses to counteract, failing which organism experiences stress (Bijlsma and Loeschcke, 2005; Ajakaiye *et al.,* 2011). When environmental temperature exceeds the limits of thermoneutral zone, core body temperature of poultry/chicken increases. Prolonged heat stress results in dramatic physiological changes in chicken organs. These changes are used as indicators of heat stress (Melesse *et al.,* 2011).

After China and USA, India is the third largest egg producer in the world (FAO, 2014). The poultry development in India has revealed 6-7% growth rate per annum in layers. Poultry inclusive of chicken contributes significantly as meat, for human consumption. Over the years poultry industry in India has contributed approximately US$229 million to the Gross National Product (GNP) (http://www.indianmirror.com/indian-industries/poultry.html, accessed on 12/12/2016).Grain yield is affected by global warming. It is stated that 2-4 Kg of cereals is required to produce 1 Kg of chicken meat. Hence poultry is the preferred species over other farm animals for protein food production. Population growth, rising incomes and urbanization are the driving forces behind poultry sector growth. Growing demand for livestock products will also have a negative impact on environment (FAO, 2006). Occurrence of poultry diseases due to heat stress should be of the prime concern in intensive poultry production. Poultry egg production is most environmentally efficient production system. It is felt that small farms of poultry are better able to adapt to warming (Seo and Mendelsohn, 2006). Poultry houses that do not have appropriate ventilation during extreme hot conditions, results in overheating of sheds, and thus causes heat stress to birds (Akyuz, 2009). Heat stress reduction in the layer sector reduces economic loss (St. Pierre *et al.,* 2003).

To maintain normal body temperature chickens adapt to increased energy requirements or decreased energy requirements in the cold or hot environment, respectively. The nutrient composition of the diet determines the amount of heat produced during metabolism by the bird. This is referred to as heat increment of the diet. Poultry completely depends on dietary sources for all nutrients and the nutrients should be at optimal level in the diet. For each degree rise in temperature between 96-100^{o}F, a 2-3% loss of feed intake has been reported. As the temperature increases from 60^{o}F to 96^{o}F (ambient) the percentage of sensible heat loss decreases from 80% to 20% and evaporative heat loss increases from 20% to 80% (www.ces.ncsu.edu, accessed on October, 2016). In addition to high ambient temperature, relative humidity also plays an important role in bringing out stress in the chickens (Ajakaiye *et al.,* 2011). The economic losses, as calculated in the layer sector are reported as $98.1 million, if the stress due to heat load is reduced, then it has been observed that loss is also reduced to $61million annually (St. Pierre *et al.,* 2003).

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The energy obtained from the feed is utilized for maintenance and productive functions. In the process of utilization of energy, heat generated should be dissipated. If the surrounding temperature is less, then the heat dissipation is more. The humidity in the atmosphere influences the heat dissipation process; higher the humidity lower is the heat dissipation. As the relative humidity increases, temperature of the bird's body increases, which results in decrease in feed intake and less amount of heat is produced. With increase in temperature, production performance decreases (Czarick and Fairchild, 2009). The higher the air speed, the bird loses greater amount of heat. The air movement assists the bird to lose heat by respiratory moisture evaporation and at a lower speed, relative humidity also plays an important role. Under high humid conditions when air velocity is less, the bird suffers from heat stress even at lower day temperature (Simmons *et al.*, 1993). Heat stress alone and in combination with humidity increases the stress on birds and affects performance.

The response of cells or a whole organism to heat shock is extremely rapid, but it takes place slowly. This process involves the synthesis and participation of preformed heat-shock proteins within the cell as well as immediate translation of preformed messenger RNA (mRNA) into heat-shock proteins and immediate transcription of genes encoding heat-shock proteins (Yost *et al.*, 1990). In warm tropical and subtropical regions, poultry when maintained at natural environmental temperatures which bring out changes in molecular, physiological and behavioural aspects that enable chickens to cope with the flux of energy into and out of their tissues at high ambient temperatures (Pawar *et al.*, 2016).

Heat Stress and Laying Performance

Reduction in egg number, egg weight and shell thickness has been reported during heat stress. An interesting finding by Wolfenson *et al.* (2001) was that the productivity decreases only when hens experienced nocturnal heat stress but not experienced during day time. Hen-day egg production was significantly decreased through all 13 weeks for hens exposed to the constant hot temperature compared with those in the control group (Allahverdi *et al.*, 2013). India's per capita consumption of chicken meat is 4.2 kg per person and eggs is 60 per person. Poultry and egg proteins are easy to digest, cheaper and easily accessible (poulvet.com/inc/poultry India 2014.pdf, accessed on October, 2016). As the ambient temperature rises above 30^{o}C, heat stress begins, birds start to pant. Birds reared in cages are more susceptible to heat stress than those reared using deep litter systems. In layers peak production may not be achieved and the eggs produced will be soft shelled and weigh less with poor egg size quality and shell strength (Faria *et al.*, 2001). Chronic heat stress is either categorized as cyclic chronic heat stress which refers to a limited period of heat stress exposure followed by comfortable temperature for the rest of the day or constant chronic heat stress where by the bird is continuously confronted with high ambient temperature.

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The physiological, endocrine and productive responses are adversely affected by heat stress (Khan *et al.,* 2012). There is acid-base (Imik *et al.,* 2013), electrolytes imbalance (Borges *et al.,* 2004) and these disturbances are due to increased respiration rate (Renaudeau *et al.,* 2011). When such physiological disturbances take place, it even leads to increased mortality (Sandhu *et al.,* 2012). Increased panting under heat stress conditions leads to decreased blood carbon dioxide levels and higher blood pH (alkalosis) that in turn hampers blood bicarbonate availability for egg shell mineralization. Osmotic change and dehydration are due to the result of water imbalance which in turn affects the cell performance negatively (Sahin *et al.,* 2002).

Heat Stress and Immune Functions

The response to stress is exhibited in many ways and the effect on immune system is dependent on the extent to which one is able to cope up with changes associated with stress. Usually, farm animals under stress adapt themselves to maintain homeostasis of the functions of the body (McEwen and Seeman, 1999). The neuroendocrine system gets stimulated under the effect of stress. The hypothalamic neurons perceive the increases in body temperature and exert an inhibiting influence on cells that are responsible for controlling feed intake. The two major systems are hypothalamic pituitary axis and the sympathetic nervous system. The hormones corticosterone, catecholamines like epinephrine and norepinephrine secreted by the above mentioned systems, act on the immune cells. It is well known that immune cells have receptors for these hormones (El-Lethay *et al.,* 2003). The down regulation of immune response to stress reduces resistance of the body to microbes (Dohms and Metz, 1991). Studies by El-Lethey *et al.* (2003) in White Leghorns revealed that there are stress resistant and stress susceptible antibody responses. Heterophils, granulocytes in chicken exhibit different functions when stimulated such as phagocytosis (Genovese *et al.,* 2013; 2006), degranulation (He *et al.,* 2005) and produce extracellular traps (Chuammitri *et al.,* 2009). The extracellular traps are formed by decondensed chromatin fibres, contain antimicrobial peptides released by granules of neutrophils (Donis-Maturano *et al.,* 2015) which trap and kill microorganisms. But if this process continues it kills antigen presenting cells such as macrophages and dendritic cells. This is for controlling the inflammatory process (Donis-Maturano *et al.,* 2015).

Corticosterone concentration is responsible for quantitative and qualitative changes in immune functions. The most reported effect of stress is the suppression of immune organs and immune cells (Shini, 2004; Shini *et al.,* 2008a). The hormone whose concentration gets elevated in plasma due to stress is corticosterone, is well documented in birds resulting in increase in the number of heterophils and decrease in the number of lymphocytes thus, favoring higher ratio for heterophils which has been well studied by Shini *et al.* (2008 a, b; 2009). It is also reported that acute heat stress may enhance immune system

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(Dhabhar, 2009) function and prepare the immune system for subsequent challenges. The immediate response to stress or stressor is to increase the circulatory level of glucose (Assenmacher, 1973). Elevation in blood glucose level may be attributed to increase in glucocorticoids secretion which plays a major role in glucose metabolism. The decline in blood glucose concentration during heat stress may be due to a decrease in concentration of thyroxine, which is closely associated with energy metabolism during heat stress. Reduction in serum thyroxine is probably due to lower metabolic rate for thermoregulation and to prevent hyperthermia. Chronic stress system may cause immune suppression by involution of immune organs (Bursa of Fabricius, thymus and spleen) (Puvadolpirod and Thaxton, 2000; Shini, 2004; Shini *et al.,* 2008 a, b). Stress with respect to elevated level of corticosterone brings about up regulation of proinflammatory chemokines of both heterophils and lymphocytes. It also significantly up regulates proinflammatory cytokines IL-6 and IL-18. This was due to acute stress experience. Similar results were observed by Shini *et al.* (2010) as early as 3 h post exposure to stress. Chronic heat stress in 26 week old layer hens resulted in a decrease in antibody titer to sheep red blood cells (Mashalay *et al.,* 2004). Macrophages and heterophils in addition to phagocytic function produce derivatives of oxygen, reactive oxygen species which attack targets along with NADPH oxidase by respiratory burst and kill micro organisms (Lin *et al.,* 1992). Studies by Muir and Craig (1998), Cheng and Jefferson (2008) revealed that laying hens of selected breeds, through genetic selection for egg production or productivity parameters has led to the development of successful technology, resulting in the improved response of the animal to stress. Berthelot-Herault *et al.* (2003) reported that IgG and IgA levels were higher in layer line (B13) and Leghorn line (L2) compared to another Leghorn line (PAI2) and meat type line (YII) when subjected to infections. It was observed that immune response is different in different layer lines or breeds.

The suppression or decrease in the weight of spleen is contributed to the decrease in the physiological functions of leucocytes. It acts as an important organ for assessing immune suppression (Dietert *et al.,* 1994). Not only the number of immune cells, but their products like cytokines and chemokine's expression is also affected. Redmond *et al.* (2011) reported that heterophils from Leghorns expressed higher level of CXCLi2 mRNA than those from Fayoumis. Felver and Gant (2012) reported higher level of heat shock protein HSP70 in the liver of White Leghorn hens (KGB) selected for productivity than the White Leghorn hens selected for higher egg production (DXL).

Heat Stress and Enzyme Activity

Melesse *et al.* (2011) studied levels of different metabolic enzymes like Lactate dehydrogenase, Creatine kinase, Glutamic pyruvic transaminase etc. in different breeds and he observed that WL-dw (dwarf White Leghorn) was the most heat tolerant one. From all these studies it was inferred that distinct genetic lines

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respond differently to stressors. Heat stress has detrimental effect on egg production parameters (Rozenbiom *et al.*, 2007; Faria *et al.*, 2001), which has been shown to be not related to feed intake (Smith and Oliver, 1972). All studies indicate that high temperatures reduce the efficiency of feed energy utilization for productive purposes. Layers not only eat less, even their production is less at high temperatures, but also produce less heat per unit of intake, especially at temperatures above 30°C even when exposed to three days (Attia *et al.*, 2016).

Increase in plasma concentration of enzymes lactate dehydrogenase, glutamic oxaloacetic transaminase and creatine kinase reflect tissue damage, in liver (Mikami *et al.*, 2004) and muscle (Sandercock *et al.*, 2006). In layers, it is reported that acute heat stress does not lead to tissue damage (Lin *et al.*, 2006). The damage is caused by oxidative injuries. The same author reported an increase in plasma MDA level as an indicator of lipid peroxidation. Oxidative damage may be minimised by antioxidant defence mechanisms that protect the cell against cellular oxidants and repair systems that prevent the accumulation of oxidatively damaged molecules. Antioxidant enzymes (CAT, SOD, GPx) play a vital role in protecting cells or tissues from harmful effects of Reactive Oxygen Species. Activities of different enzymes in plasma like glutamic pyruvic transaminase (GPT), glutamic oxalo acetate transaminase (GOT), creatine kinase (CK) and lactate dehydrogenase (LDH) when estimated in chickens (layers) belonging to four different genotypes mainly dwarf White Leghorn, White Leghorn with improved feed efficiency, New Hampshire Lohmann White and Lohmann Brown; (the control group was maintained at 18-20^{o}C and experimental groups were maintained at 30-32^{o}C), control groups of the respective genotypes did not exhibit any significant difference with respect to activity of enzymes when compared between the groups. However, under high temperature conditions, the activities of the different enzymes were significantly different from their respective control groups of chicken. In experiments conducted by Khan and Sardar (2005) in Layers, namely Desi Fayoumi and Commercial White Leghorn revealed that when these birds were reared under high ambient temperatures, egg production, feed consumption, egg weight and egg shell thickness decreased. The blood levels of enzymes alkaline phosphatase, glutamate pyruvate transaminase and glutamate oxalate transaminases increased. In contrast, the ascorbic acid levels decreased. It has been reported by Cheng *et al.* (1990), Chakraborty and Sadhu (1983), Takeda and Hare (1985), the increase in the level/activities of the enzymes is due to leakage of enzymes under stressful conditions (Bhatti *et al.*, 2003; Bhatti and Dil, 2005).

Heat Stress and Gut

When subjected to heat stress the tight junctions between epithelial cells of the intestine become leaky, which has an effect on absorption of nutrients and pathological bacteria can go in to systemic circulation leading to inflammatory response. One of the main toxin is the lipo-polysaccharide which acts as an

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endotoxin and elicits immune response after heat stress (Slawinska *et al.,* 2016).It has been observed that expression of ghrelin gene in hypothalamus of HY-line layers was upregulated whereas, those of orexigenic genes was not affected. Central (hypothalamus) and peripheral (duodenum and jejunum) CCK mRNA levels in heat-exposed laying hens decreased intestine mobility and, hence, a lower passage rate, which allowed intestine enzymes more time to digest nutrients (Song *et al.,* 2012). Results of the studies of the same authors indicated that some hypothalamic and gastrointestinal tract peptides were involved in the appetite regulation in heat-exposed laying hens. In addition to this, heat exposure resulted in a significant decrease in body weight gain, feed intake, laying rate, average egg mass, egg production and shell thickness. Similar results were reported by Anjum *et al.* (2014).

Heat Stress and Egg Shell Quality

Heat stress affects biological defence mechanisms including immunological and metabolic functions. Higher ambient temperature can have negative impact on the productivity of birds. It has been reported in heat stressed commercial layers that when they have been exposed to high temperature at 36^{o}C, plasma calcium concentration decreased and pH increased. Layers exposed to high ambient temperature results in decreased egg production and quality (Allahverdi *et al.,* 2013; Aberra *et al.,* 2010). The calcium binding protein calbindin localization in intestinal segments and egg shell gland is decreased when exposed to high temperature (Ebeid *et al.,* 2012). During summer, stress can result in smaller eggs and reduced shell quality via a number of physiological processes occurring within the bird (Usayran *et al.,* 2001; Allahverdi *et al.,* 2009). Heat stress also reduces the activity of carbonic anhydrase, an enzyme which results in the formation of bicarbonate which contributes carbonate to the formation of egg shell (Balnave and Muherreza, 1997). Albumen height and Haugh units decrease with storage time and this decrease occurs more quickly at higher temperatures.

Heat Stress Hormones and Reproduction

It is suggested that reduced feed consumption may account for impairment in reproduction. It has been suggested that heat stress may modulate the activity of hypothalamus and pituitary (Braganza and Wilson, 1978; Jeronen *et al.,* 1978). It has been reported by Johnson (1981) in laying hens, that heat stress decreased circulatory prolactin and gonadotropins, which affect the reproductive function. Studies in White Leg Horn, layers revealed that when birds were subjected to heat stress 42±3^{o}C for 15d, egg production dropped by 20% and there was a decrease in egg weight and follicles with a concomitant decrease in the expression level of cytochrome P 450, 17α hydroxylase enzymes. Further, non significant decrease in the expression level of cytochrome P 450 aromatase and plasma estradiol level has also been reported (Rozenboim *et al.,* 2007). But, the high ambient temperature did not affect yolk or plasma

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concentration of corticosterone. Similarly Hester *et al.* (1996) reported difference in concentration of plasma corticosterone between control and heat stress treated groups. Heat stress reduces luteinizing hormone and hypothalamic gonadotropin – releasing hormone levels in Leghorns due to increased prolactin levels, which in turn causes ovarian regression (Donoghue *et al.,* 1989; Rozenboim *et al.,* 1993; You *et al.,* 1995).

Thyroid hormones play an important role in regulating metabolic and thermogenesis functions in chicken (Lam and Harvey, 1990; He *et al.,* 2000; Tao *et al.,* 2006).The level of thyroxine (T_3) in blood plasma was reduced in all the heat stressed different layer genotypes. Similar results have been observed in layers (Decuypere and Kahn, 1988; Maak *et al.,* 2003; Tao *et al.,* 2006; Melesse *et al.,* 2011). Circulatory levels of T_3 have been linked to the size of the bird. Small size chickens produce less T_3 (Tixier-Boichard *et al.,* 1990; Zeman *et al.,* 1996). Higher level of plasma T_3 has been attributed to decreased ability to lose heat or increased heat production. Thyroid hormones viz. thyroxin and triodothyronine provide a major mechanism of acclimatization. The role of thyroid gland hormone in adaptation to heat stress is related to the role of hormones in regulation of metabolic rate of birds (Mc Naff, 1988). Corticotrophin Releasing Hormone from the hypothalamus influences the decreased production of T_4 and T_3 and it has been used as an indicator of stress in laying birds (Melesse *et al.,* 2011).

A decrease in egg production may also be due to decrease in uptake of gut calcium or enzyme production related to maintenance of calcium homeostasis (Franco-Jimenez *et al.,* 2007). The level of circulatory T_3 denotes or is related to egg productivity and they are directly related to each other (Elnagar *et al.,* 2010). Klandorf and Harvey (1985) reported that reduced feed intake reduces circulatory T_3 concentrations, but, reports of Mack *et al.* (2013) stated that, the decrease in T_3 concentration was irrespective of feed intake.

The effect of hot environment has been found to vary among different breeds of chicken (Deeb and Cahaner, 2002; Melesse *et al.,* 2011; Mashaly *et al.,* 2004). Some have reported that acute heat stress does not have effect on plasma T_3 or T_4 concentration (Bowen and Washburn, 1985; May *et al.,* 1986). The higher level of circulatory T_3 causes increased metabolic rate and reduced thermotolerance (Bowen and Washburn, 1985). This is associated with changes in sizes of thyroid gland and its secretion. Heat stress caused hypothyroid effect, decreased plasma progesterone concentration and increased plasma prolactin concentration in Hy-Line laying hens (Elnagar *et al.,* 2010). Estrogen, T_3 and T_3/T_4 ratio was observed to decrease under heat stressed conditions. Decrease in thyroid hormone activity has been reported under heat stress conditions (Sinurat *et al.,* 1987).

The regulatory mechanisms for the reduced reproductive efficiency in the hens when subjected to high ambient temperature is modulated at the level of hypothalamus and pituitary (Braganza and Wilson, 1978 a,b; Jeronen *et al.,* 1978). When ovaries are affected by heat stress, functions of granulosa and theca cells are delayed and hence steroidogenesis. When reproductive hormones are affected, it leads to decrease in

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reproductive efficiency (Rozenboim, 2007). *In Vitro* study by Hrabia *et al.* (2004) indicated that prolactin affects steriodogenesis by ovarian follicle and type of follicular layer. The change in ovarian steroid production is due to heat stress which is due to decreased ovarian blood flow in laying hens (Wolfenson *et al.*, 1981). Stress has been shown to affect the pathway for PRL, FSH and LH in laying hens. Circulatory plasma prolactin and gonadotropin levels decline (Johnson, 1981). Hens subjected to acute thermal stress had decreased circulating LH levels and was attributed to reduced LH releasing ability of the hypothalamus (Donoghue *et al.*, 1989).

The layer hens, 16 week old Ross Brown, when subjected to high ambient temperature ranging between 25 to 35^{o}C, it was observed that T_3, T_4 and TSH concentrations in the serum was less and serum ACTH concentration was higher (Sahin and Kacuk, 2001). Further, under heat stress, albumin, triglycerides and cholesterol concentration in serum decreased. In the same species/breed effect of high ambient temperature (30-40^{o}C) on sixteen week old hens for sixteen weeks was studied, it was observed that level of Vitamin A and E in serum and liver was less when compared with the control counterparts. Similarly, activity of glutathione peroxidase and reduced glutathione in serum, liver and muscle had decreased; whereas the TBARS level in liver and muscle had increased significantly (Naziroglu *et al.*, 2000).

Heat Stress and Oxidative Stress

In layers metabolic heat production is high as a result of high rate of egg production (Blem, 2000). Heat stress causes effects on mineral balance, plasma electrolyte concentrations (Deyhim and Teeter, 1990; Belay and Teeter, 1993) and has an impact on disease resistance vis a vis with an effect on immune system. Studies by Kutlu and Forber (1993) and Berrong and Washburn (1998), indicated that heat stress also affects, plasma protein concentration. Further, decrease in concentration of plasma minerals like calcium and phosphorus has also been reported (Usayran *et al.*, 2001). When there is prolonged heat stress, it leads to decreased ATP synthesis, cellular calcium homeostasis and cell permeability which predispose cells to apoptosis. Mujahid *et al.* (2007) reported that when chickens were exposed to acute heat conditions, concentration of plasma and mitochondrial MDA increased significantly. When stress is experienced during hot conditions, ROS when generated, they trigger and modulate cell signalling activities. The activation of transcription factors AP-I, NF-KB and Nrf2 have been shown to take place in heat stressed chickens. AP-I is a modulator of genes which gets stimulated by environmental stimuli. NF-kB expression regulates pro-inflammatory responses. When the heat stress is severe or prolonged, over production of ROS in mitochondria damages proteins, lipids and DNA , which in turn leads to decreased ATP synthesis and calcium dyshomeostasis. Finally it leads to cell apoptosis and necrosis (Akbarian *et al.*, 2016, review).

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Heat Stress and Osmoregulation

The monovalent minerals Na^+, K^+ and Cl^- exert characteristic effects on hen's acid-base homeostasis, regulation of osmotic pressure and absorption of amino acids. The Na^+ and K^+ are mainly alkalogenic causing shift towards alkaline pH of the body fluids (Borges *et al.,* 2003). Minerals such as copper and magnesium affect the structural composition of the egg shell (Roberts, 2004; Balnave, 2004; Vecerek *et al.,* 2002). High ambient temperature conditions cause decrease in concentration of blood serum electrolytes (Franco-Jimenez and Beck, 2007; Pavlik *et al.,* 2009). The physiological mechanisms that are invoked by birds exposed to high temperatures must meet the opposing demands of thermoregulation and respiratory alkalosis. Dissipation of heat by evaporative cooling causes an increase in respiration, while respiratory alkalosis causes decrease in respiration. Laying hens within the thermoneutral zone consume approximately 200 ml of water per day, whereas hens maintained at 40°C will consume approximately 500 ml per day (North and Bell, 1990). Arad *et al*. (1983) observed that even without subjecting birds to heat stress, water deprivation for 48 h, which included 24 h without food, of fowls maintained at 25°C was associated with an increase in serum concentrations of sodium and in serum osmolality.

Heat Stress and Different Genotypes

Study by Mack *et al.* (2013) revealed that heat stress caused reduction in productivity and different behavioral changes were observed in laying hens, but it was observed that the response was varying depending on the genotype of the bird. The two strains of the White Leghorn hens used were Dekalb XL and Kind gentle bird. In these breeds heat stress did not affect body weight of the birds. Results of Fox (1951) and Gowe *et al.* (2008) revealed that differences in genetic background, may contribute to difference in responses. The differences in egg quality have been observed in hens under heat stress whose response varied with respect to difference in strains (Melesse *et al.,* 2010). Reason given by Zhang *et al.* (2005), Wright *et al.* (2006) and Goraga *et al.* (2010) was that the difference in genotypes may result in affecting physiological functions differently, which in turn might influence quality of the egg differently. The behavioural changes associated with heat stress, has been reported as reduced time spent in walking and spent more time in walking and also in keeping the wings elevated. All these behavioral adaptations have been attributed for causing reduction in body temperature (Gowe *et al.,* 2008; Gerken *et al.,* 2006). Reports of Mack *et al.* (2013) stated that panting was the only response observed in hens, subjected to heat stress. Photoperiod length and light intensity have been also reported as factors affecting sexual maturity and behavioural responses (Koelkebeck, 2001). Studies by Koelkebeck *et al.* (1992, 1993) revealed that under heat stress, layer's bone characteristics were affected in commercial caged layer flocks. Koelkebeck *et al*. (1993), in an experiment on heat-stressed laying hens, found that when birds were provided with carbonated drinking water it improved tibia breaking strength. They

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suggested that carbonated drinking water during heat stress may reduce bone breakage during the processing of spent hens. Mortality due to osteoporosis is less, but accounts up to one third of mortality. It has been indicated that it is due to deficiency of calcium, phosphorus and Vitamin D (Webster, 2003).Cross bred naked neck and frizzle phenotypes produced more eggs when compared with their normal feathered sibs. Similar were the results obtained with the other production parameters. It showed that the cross bred strains mentioned had better tolerance to hot and humid environments (Hagan *et al.*, 2011, 2013, 2014). These studies clearly indicated that by introducing heat tolerant genes, some of the cross breeds obtained had better capacity to withstand the higher ambient temperatures (Aberra *et al.*, 2013). Studies by Mahrous *et al.* (2008) suggested that selection of genotypes with naked neck and frizzle genes, had better disease resistance ability and breeds may be more suitable for rearing under unfavourable conditions. In recent times, newer methods like genetic marker assisted selection of poultry breeds are being explored, for increased heat tolerance and disease resistance. Application of modern molecular techniques in poultry breeding has great potential to improve poultry productivity through understanding various cellular and molecular mechanisms involved in production, physiological and immunological aspects of the poultry birds.

Conclusion

In conclusion, scientists in different fields, must work together to combat difficulties arising from heat stress by developing new methods of cooling systems, newer indices to evaluate climate change effects, selection of better genotypes getting adapted to higher temperature effects and above all conservation of natural resources for alleviating heat stress in future.

References

1. Aberra M, Maak S, Pingel H , LengerkenG . Assessing the thermo-tolerance potential of five commercial layer chicken genotypes under long-term heat stress environment as measured by their performance traits. J. Anim. Prod. Adv. 3: 254-264. 2013.
2. Aberra M, Maak S, von Lengerken G . Effect of long-term heat stress on egg quality traits of Ethiopian naked neck chickens and their F1 crosses with Lohmann White and New Hampshire chicken breeds. Livestock Research for Rural Development 22:071. 2010.
3. AjakaiyeJJ, Perez-Bello A and Mollineda TA . Impact of heat stress on egg quality in layer hens supplemented with l- ascorbic acid and dl- tocopherol acetate. Veterinary Archives, 81:119-132. 2011.
4. Akbarian A, Michiels J, Degroote J, Majdeddin M, Golian A, and De Smet S. Association between heat stress and oxidative stress in poultry; mitochondrial dysfunction and dietary interventions with phytochemicals. Journal of Animal Science and Biotechnology, 7: 3. 2016.
5. Akyuz, A. Effects of some climates parameters of environmentally uncontrollable broiler houses on broiler performance. Journal of Animal Veterinary Advances 8:2608-2612.2009.
6. Allahverdi A, Feizi A, Takhtfooladi HA, Nikpiran H. 2013. Effects of heat stress on acid-base imbalance, plasma calcium concentration, egg production and egg quality in commercial layers. Global Vet. 10:203-207. 2009.

DOI 10.5455/ijlr.20170312042324

7. Allahverdi A , Feizi A , Takhtfooladi H A and Hosein Nikpiran . Effects of heat stress on acid-base imbalance, plasma calcium concentration, egg Production and egg quality in commercial layers. Global Veterinaria 10: 203-207. 2013.
8. Anjum MS, Sandhu MA, Nasir M and Rahman Z . Upgrade of egg quality through different heat-combating systems during high environmental temperature. Tropical Animal Health and Production, 46: 1135–1140. 2014.
9. Arad Z, Marder J and Eylath U. Serum electrolyte and enzyme responses to heat stress and dehydration in the fowl (*Gallus domesticus*). Comparative Biochemistry and Physiology 74A, 449–453. 1983.
10. Assenmacher I. The peripheral endocrine glands. Pages 183–286 in Avian Biology. 3rd ed. D. S. Farmer and J. R. King, ed. Academic Press, New York, NY. 1973.
11. Attia Y A, Abd EI-Hamid A E, Ellakany H F, Bovera F, AI-Harthi M A and Ghazaly S A. Laying performance, digestibility and plasma hormones in laying hens exposed to chronic heat stress as affected by betaine, vitamin C, and/or vitamin E supplementation. Springer Plus. 5:1619. 2016.
12. Balnave D and Muherreza S K. Improving eggshell quality at high temperatures with dietary sodium bicarbonate. Poultry Science, 76:588–593.1997.
13. Balnave D. Challenges of accurately de fining the nutrient requirements of heat-stressed poultry. In: World's Poultry Science Association Invited Lecture. Poultry Science, 83, 5-14.2004.
14. Belay T and Teeter RG. Broiler water balance and thermo balance during thermoneutral and high ambient temperature exposure. Poultry Science. 72:116-124. 1993.
15. Berrong S L and Washburn K W. Effects of genetic variation on total plasma protein, body weight gains, and body temperature responses to heat stress. Poultry Science, 77:379–385. 1998.
16. Berthelot-Herault, F, Mompart F, Zygmunt M S, Dubray G and Duchet-Suchaux M. Antibody reponses in the serum and gut of chicken lines differing in ceacal carriage of Salmonella enteritidis . Veterinary Immunology and Immunopathology, 96:43-52. 2003.
17. Bhatti B, Sardar M, Siddique RB and Talat T. Effect of serum enzyme in chicken exposed to hydropericardium virus. Pakistan Journal of Veterinary Research. 1:17-19. 2003.
18. Bhatti BM and Dil S. Effect of Vitamin C on immune response in Desi chicken against New Castle disease. Pakistan Journal of Veterinary Research, 2:48-49. 2005.
19. Bijlsma R and Loeschcke V. Environmental stress, adaptation and evolution: an overview. Journal of Evolutionary Biology, 18:744–749. 2005.
20. Blem C R. Energy balance. Pages 327-341 in: Sturkie's Avian Physiology. 5th ed. (Ed. G. C. Whittow). Academic Press, New York, NY. 2000.
21. Borges SA, Fischer da Silva AV, Ariki J, Hooge DM and Cummings KR. Dietary electrolyte balance for broiler chickens under moderately high ambient temperatures and relative humidities. Poultry Science, 82: 301-308. 2003.
22. Borges S A, Da Silva A V F, Majorka A, Hooge D M, and Cummings K R. Physiological responses of broiler chickens to heat stress and dietary electrolyte balance (sodium plus potassium minus chloride, milliequivalents per kilogram). Poultry Science, 83: 1551-1558. 2004.
23. Bowen SJ and Washburn KW. Thyroid and Adrenal Response to Heat Stress in Chickens and Quail Differing in Heat Tolerance. Poultry Science, 64:149-154. 1985.
24. Braganza A and Wilson WO. Effect of acute and chronic elevated air temperature, constant (34°C) and cyclic (10 to 34°C), on brain and heart norepinephrine of male Japanese quail. General and Comparative Endocrinology, 30: 233–237. 1978a
25. Braganza A and Wilson WO. Elevated temperature effects on catecholamines and serotonin in brains of male Japanese quail. Journal of Applied Physiology, 45:705–708. 1978b.
26. Chakraborty AK and Sadhu DP. Effect of acute heat stress and its modifications by adrenaline and adrenolytic drugs in pigeons. Indian Journal of Animal Science, 53: 575-578. 1983.
27. Cheng TK, Coon CN and Hamre ML. Effect of environmental stress on the ascorbic acid requirement of laying hens. Poultry Science, 69: 774–785. 1990.

DOI 10.5455/ijlr.20170312042324

28.Cheng H W and Jefferson L. Different behavioral and physiological responses in two genetic lines of laying hens after transportation. Poultry Science, 87: 885–892. 2008.
29.Chuammitri P, Ostojić J, Andreasen C B, Redmond S B, Lamont S J, Palić D. Chicken heterophil extracellular traps (HETs): Novel defense mechanism of chicken heterophils. Veterinary Immunology and Immunopathology, 129: 126–131. 2009.
30.Czarick, M. and Fairchild B. Without air movement evaporative cooling pads can increase bird heat stress. Poultry Housing Tips 21:1-5. 2009. Univ. of Georgia Cooperative Extension.
31.Decuypere E and Kuhn ER. Thyroid hormone physiology in Galli formes: Age and strain related changes in physiological control. American Zoology, 28: 401-415. 1988.
32.Deeb N and Cahaner A. Genotype-by-environment interaction with broiler genotypes differing in growth rate. 3. Growth rate and water consumption of broiler progeny from weight-selected versus non selected parents under normal and high ambient temperatures.Poultry Science, 81: 293-301. 2002
33.Deyhim F and Teeter RG. Acid base balance and plasma corticosterone of heat distressed broilers consuming KCl and NaCl supplemental drinking water. Poultry Science, 69: 1990.
34.Dhabhar F S. Enhancing versus Suppressive Effects of Stress on Immune Function: Implications for immunoprotection and immunopathology. Neuroimmunomodulation, 16: 300–317. 2009.
35.Dietert R R, Golemboski K A and Austic R E. Environment-immune interactions. Poultry Science, 73:1062–1076.1994.
36.Dohms JE, Metz A. Stress–mechanisms of immunosuppression. Veterinary Immunology and Immunopathology, 30: 89–109. 1991.
37.Donis-Maturano L, Sánchez-Torres LE, Cerbulo-Vázquez A, Chacón-Salinas R, García-Romo GS, Orozco-Uribe MC, Yam-Puc JC, González-Jiménez MA, Paredes-Vivas YL, Calderón-Amador J, Estrada-Parra S, Estrada-García I, Flores-Romo L. Prolonged exposure to neutrophil extracellular traps can induce mitochondrial damage in macrophages and dendritic cells. Springer plus, 4:161. 2015.
38.Donoghue DJ, Krueger BF, Hargis B M, Miller AM and El. Halawani M E. Thermal stress reduces serum LH and bioassayable hypothalamic content of LHRH in the hen. Biology of Reproduction, 41: 419-424. 1989.
39.Ebeid TA, Suzuki T and Sugiyama T. High ambient temperature influences eggshell quality and calbindin-D28k localization of eggshell gland and all intestinal segments of laying hens. Poultry Science, 91: 2282–2287. 2012.
40.El-Lethey H, Huber-Eicher B and Jungi TW. Exploration of stress-induced immunosuppression in chickens reveals both stress-resistant and stress-susceptible antigen responses. Veterinary Immunology and Immunopathology, 95: 91-101. 2003.
41.Elnagar SA, Scheideler SE and Beck MM. Reproductive hormones, hepatic deiodinase messenger ribonucleic acid, and vasoactive intestinal polypeptide-immunoreactive cells in hypothalamus in the heat stress-induced or chemically induced hypothyroid laying hen. Poultry Science, 89: 2001-2009. 2010.
42.FAO (2006).The state of food insecurity in the world.Published by FAO, UN. Rome, Italy
43.FAO (2014). Food Outlook. Food and Agricultural Organisations of the United Nations. Rome, Italy http://www.fao.org/3/a-i4136e.pdf
44.Faria, DE, Junqueira OM, Souza PA, Titto EAL. Performance, body temperature and egg quality of laying hens fed vitamins D and C under three environmental temperatures. Brazilian Journal of Poultry Science, 3:49-56. 2001.
45.Felver–Gant JN, Mack LA, Dennis RL, Eicher SD, Cheng HW. Genetic variations alter physiological responses following heat stress in 2 strains of laying hens. Poultry Science, 91: 1542–1551. 2012.
46.Fox TW. Studies on heat tolerance in the domestic fowl. Poultry Science, 30:477–483. 1951.
47.Franco-Jimenez DJ and Beck MM . Physiological changes to transient exposure to heat stress observed in laying hens. Poultry Science, 86: 538-44. 2007.
48.Genovese K J, He H, Swaggerty C L, and Kogut M H. The avian heterophil. Developmental and Comparative Immunology, 41:334–434.2013.

DOI 10.5455/ijlr.20170312042324

49. Genovese KJ, He H, Lowry VK, Swaggerty CL and Kogut MH. Comparison of heterophil functions of modern commercial and wild-type Rio Grande turkeys. Avian Pathology, 35: 217–223. 2006.
50. Gerken M, Afnan R and Dörl J. Adaptive behaviour in chickens in relation to thermoregulation. Archive Geflüglk. 70:199–207. 2006.
51. Goraga Z, Nassar M, Schramm GP and BrockmannGA . Phenotypic characterization of chicken inbred lines that differ extremely in growth, body composition and egg production traits. Archive Tierzucht , 53: 337–349. 2010.
52. Gowe RS, Fairfull RW and DaghirNJ . Breeding for resistance to heat stress. pp 11–30 in Poultry Production in Hot Climates. N. J. Daghir, ed. CABI, Oxfordshire, UK. 2008.
53. Hagan JK, Adomako K and Olympio SO . Evaluating the egg production performance of crossbred naked-neck and frizzle chickens under intensive system, Ghanaian Journal of Animal Science, 5: 49–55. 2011.
54. Hagan JK, Adomako K and Olympio SO . Effect of incorporation of heat-tolerant genes into Lohmann brown layers on egg production and quality under hot and humid environments, Indian Journal of Animal Sciences, 83: 725–729. 2013.
55. Hagan JK, Adomako K and Olympio SO . Effect of incorporation of thermo-regulatory genes into exotic layers on egg production and quality under tropical environment. Tropical Animal Health Production, 46: 107. 2014.
56. He J, Ohtsuka A and Hayashi K . Selenium influences growth via thyroid hormone status in broiler chickens. British Journal of Nutrition, 84: 727-732. 2000.
57. He H, Lowry VK, Swaggerty CL, Ferro PJ, Kogut MH. In vitro activation of chicken leukocytes and in vivo protection against Salmonella enteritidis organ invasion and peritoneal S. enteritidis infection-induced mortality in neonatal chickens by immunostimulatory CpG oligodeoxynucleotide. FEMS Immunology and Medical Microbiology, 43, 81–89. 2005.
58. Hester PY, Muir WM, Craig JV and Albright JL. Group selection for adaptation to multiple hen cages: Hematology and adrenal function. Poultry Science,75: 1295–1307. 1996.
59. Hrabia A, Paczoska-Eliasiewicz H and Rzasa J . Effect of prolactin on estradiol and progesterone secretion by isolated chicken ovarian follicles. Folia Biol (Krakow). , 52:197-203. 2004.
60. Imik H, Kaynar O, Ozkanlar S, Gumus R, Polat H and Ozkanlar Y. Effects of vitamin C and α-lipoid acid dietary supplementations on metabolic adaptation of broilers to heat stress. Revue de Médecine Vétérinaire, 164:52-59. 2013.
61. IPCC (Intergovernmental Panel on Climate Change: AR4), 2007. The Intergovernmental Panel on Climate Change 4th Assessment Report. Jackson Institute, University College, London.www.ipcc.ch/
62. Jeronen E, Peura M L and Hissa R . Effect of temperature stress on brain monoamine content in the pigeon. Journal of Thermal Biology, 3:25–30. 1978.
63. Johnson AL. Comparison of three serial blood sampling techniques on plasma hormone concentrations in laying hens. Poultry Science, 60:2322–2327. 1981.
64. Khan SH and Sardar R. Effect of vitamin C supplementation on the performance of desi, Faoumi and commercial white leghorn chicken exposed to heat stress. Pakistan Veterinary Journal, 25: 136-166. 2005.
65. Khan R U, Nikousefat Z, Selvaggi M, Laudadio V, and Tufarelli V. Effect of ascorbic acid in heat-stressed poultry. World's Poultry Science Journal, 68: 477-490. 2012.
66. Klandorf H and Harvey S. Food intake regulation of circulating thyroid hormones in domestic fowl. General and Comparative Endocrinology, 60:162–170. 1985.
67. Koelkebeck KW, Harrison PC and Madindou T. Research note: effect of carbonated drinking water on production performance and bone characteristics of laying hens exposed to high environmental temperatures. Poultry Science, 72: 1800-1803. 1993.
68. Koelkebeck KW, Parsons CM, Leeper R W and Moshtaghian J . Effect of duration of fasting on post molt laying hen performance. Poultry Science, 71:434-439. 1992.

DOI 10.5455/ijlr.20170312042324

69. Koelkebeck, KW. Physiological Responses of Poultry to the Environment. Poultry Illinois livestock trail. Wednesday, June 13, 2001. www.livestocktrail.illinois.edu/poultry net).
70. Kutlu HR and Forbes JM. Changes in growth and blood parameters in heat-stressed broiler chicks in response to dietary ascorbic acid. Livestock Production Science, 36: 335-350. 1993.
71. Lam SK and Harvey S. Thyroid regulation of body temperature in anaesthetised chickens. Comparative Biochemistry and Physiology, 95A: 435-439. 1990.
72. Lin HK, Bloom SE and Dietert RR. Macrophage antimicrobial functions in a chicken MHC chromosome dosage model. Journal of Leukocyte Biology, 52: 307–314.1992.
73. Lin H, Decuypere E and Buyse J. Acute heat stress induces oxidative stress in broiler chickens. Comparitive Biochemistry and Physiology, 144:11–17. 2006.
74. Maak S, Melesse A, Schmidt R and Von Lengerken G. Effect of long-term heat exposure on peripheral concentrations of heat shock protein 70 (Hsp70) and hormones in laying hens with different genotypes. British Poultry Science, 44: 133-138. 2003.
75. Mack LA, Felver-Gant JN, Dennis RL and Cheng HW. Genetic variations alter production and behavioral responses following heat stress in 2 strains of laying hens. Poultry Science, 92 :285–294. 2013.
76. Mahrous M, Galal A, Fathi MM and Zein El-Dein A. Impact of naked-neck (Na) and frizzle (F) genes on growth performance and immunocompetence in chickens, International Journal of Poultry Science, 7: 45–54. 2008.
77. Mashaly MM, Hendricks GL, Kalama MA, Gehad AE, Abbas AO and Patterson PH . Effect of heat stress on production parameters and immune responses of commercial laying hens. Poultry Science, 83:889–894. 2004.
78. May JD, Deaton JW, Reece FN and Branton SL. Effect of acclimation and heat stress on thyroid hormone concentration. Poultry Science, 65: 1211–1213.1986.
79. McEwen B S and Seeman T. Protective and damaging effects of mediators of stress: Elaborating and testing the concepts of allostasis and allostatic load. Annals of the New York Academy of Sciences, 896: 30-47. 1999.
80. Melesse A, Tiruneh W and Negesse T. Effects of feeding *Moringastenopetala* leaf meal on nutrient intake and growth performance of Rhode Island Red chicks under tropical climate. Tropical Subtropical Agroecosystem, 14: 485-492. 2011.
81. Melesse A, Maak S, Schmidt R and von Lengerken G. Effect of long-term heat stress on key enzyme activities and T_3 levels in commercial layer hens. International Journal of Livestock Production, 2: 107-116. 2011.
82. Mikami TS, Ishibashi S Y, and Ohta S. Endurance exercise training inhibits activity of plasma GOT and liver caspase-3 of mice [correction of rats] exposed to stress by induction of heat shock protein 70. Journal of Applied Physiology, 96:1776–1781. 2004.
83. Muir W M, and Craig J V. Improving animal well-being through genetic selection. Poultry Science, 77:1781–1788. 1998.
84. Mujahid A, Pumford NR, Bottje W, Nakagawa K, Miyazawa T, Akiba Y and Toyomizu M. Mitochondrial oxidative damage in chicken skeletal muscle induced by acute heat stress. Journal of Poultry Science, 44:439–445. 2007.
85. Naziroglu M, Sahin K, Simsek H, Aydilek N and Ertas ON . The effects of food withdrawal and darkening on lipid peroxidation of laying hens in high ambient temperatures. DtschTierarztlWochenschr, 107:199-202. 2000.
86. North, M.O. and Bell, D.D. (1990) Commercial Chicken Production Manual, 4th edn. Van Nostrand Reinhold, New York, 262 pp.
87. Pavlik A, Lichovnikova M and Jelinek P. Blood plasma mineral profile and qualitative indicators of the eggshell in laying hens in different housing systems. Acta Veterinaria Brno, 78: 419–429. 2009.

DOI 10.5455/ijlr.20170312042324

88. Pawar SS, Sajjanar B, Lonkar VD, Nitin KP, Kadam AS, Nirmale AV, Brahmane MP, Bal SK . Assessing and mitigating the impact of heat stress in poultry. Advances in Animal and Veterinary Science, 4: 332-341. 2016.
89. Puvadolpirod S and Thaxton J P. Model of physiological stress in chickens. 1. Response parameters. Poultry Science, 79: 363-369. 2000.
90. Redmond S B, Chuammitri P, Andreasen C B, Palić D, and Lamont S J. Proportion of circulating chicken heterophils and CXCLi2 expression in response to Salmonella enteritidis are affected by genetic line and immune modulating diet. Veterinary Immunology and Immunopathology, 140: 323–328.2011.
91. Renaudeau D, Collin A, Yahav S, de Basilio V, Gourdine J L, and Collier R J. Adaptation to hot climate and strategies to alleviate heat stress in livestock production. Animal, 1: 1-22. 2011.
92. Roberts JR . Factors affecting egg internal quality and egg shell quality in laying hens. The Journal of Poultry Science, 41: 161-177. 2004.
93. Rozenboim I, Tabibzadeh C, Silsby J L, and El Halawani M E. The effect of ovine prolactin (oPRL) administration on hypothalamic vasoactive intestinal peptide (VIP), gonadotropin-releasing hormone I and II (GnRH-I, II) content and anterior pituitary VIP receptors in laying turkey hens. Biology of Reproduction, 48:1246–1250. 1993.
94. Rozenboim I, Tako E, Gal-Garber O, Proudman JA and Uni Z . The effect of heat stress on ovarian function of laying hens. Poultry Science, 86:1760–1765. 2007.
95. Sahin K and Kucuk O. Effects of vitamin C and vitamin E on performance, digestion of nutrients, and carcass characteristics of Japanese quails reared under chronic heat stress (34°C). Journal of Animal Physiology and Animal Nutrition, 85: 335–342. 2001.
96. Sahin K , Sahin N and Yaralioglu S (2002) Effects of vitamin C and vitamin E on lipid peroxidation, blood serum metabolites, and mineral concentrations of laying hens reared at high ambient temperature. Biological Trace Element Research 85: 35-45.2002.
97. Sandercock D A, Hunter, R R, Mitchell M A and Hocking P M. Thermoregulatory capacity and muscle membrane integrity are compromised in broilers compared with layers at the same age or body weight. British Poultry Science, 47:322–329. 2006.
98. Sandhu M A, Mirza F Q, Afzal F, and Mukhtar N. Effect of heat stress on cellular and humoral immunity and its cure with α-tocopherol in meat type birds. Livestock Science, 148: 181-188. 2012.
99. Seo SN and Mendelsohn RO 2006. The Impact of Climate Change on Livestock Management in Africa: A Structural Ricardian Analysis. World Bank Publications, USA., Pages: 48.
100. Shini S, Shini A and Kaiser P. Cytokine and chemokine gene expression signatures in heterophils of chicken exposed to corticosterone in drinking water. Stress, 13: 185-194. 2010.
101. Shini S. Physiological response of laying hens to management practices. PhD Thesis, University of Queensland, Brisbane, Queensland, Australia. 2004.
102. Shini S, Shini A and Huff GR . Effects of chronic and repeated corticosterone administration in rearing chickens on physiology, the onset of lay and egg production of hens. Physiological Behavior, 98: 73–77. 2009.
103. Shini S, Kaiser P, Shini A and Bryden W L . Biological response of chickens exposed to corticosterone and a bacterial endotoxin. Comparative Physiology Biochemistry and Molecular Biology, 149:324–333. 2008a.
104. Shini S, Kaiser P, Shini A and Bryden WL. Differential alterations in ultrastructural morphology of chicken heterophils and lymphocytes induced by corticosterone and lipopolysaccharide. Veterinary Immunology and Immunopathology, 122:83–93. 2008b.
105. Simmons JD, Lotland BD and May JD. Heat loss from broiler chickens subjected to various air speeds and ambient temperatures. Applied Engineering in Agriculture, 13:665-669. 1993.
106. Sinurat AP, Balnave D and McDowell GH .Growth performance and concentrations of thyroid hormones and growth hormone in plasma of broilers at high temperatures. Australian Journal of Biological Sciences, 40:443-50. 1987.

DOI 10.5455/ijlr.20170312042324

107. Slawinska A, Hsieh JC, Schmidt CJ, Lamont SJ. Heat Stress and Lipopolysaccharide Stimulation of Chicken Macrophage-Like Cell Line Activates Expression of Distinct Sets of Genes. Shelden EA, ed. *PLoS ONE*. 2016; 11(10):e0164575. doi:10.1371/journal.pone.0164575.
108. Smith A J and Oliver L. Some nutritional problems associated with egg production at high environmental temperatures. I. The effect of environmental temperature and rationing treatments on the productivity of pullets fed on diets of differing energy content. Rhodasian Journal of Agricultural Science, 10:3–10. 1972.
109. Song Z, Liu L, Sheikhahmadi A, Jiao H, and Lin H. Effect of heat exposure on gene expression of feed intake regulatory peptides in laying hens. BioMed Research International 2012(4-5):484869
110. St. Pierre NR, Cobanov B and Schnitkey G. Economic losses from heat stress by US livestock industries. Journal of Dairy Science, 86:E52-E77. 2003.
111. Takeda Yand Hara M. Significance of ferrous ion and ascorbic acid cycle. Journal of Bio chemistry, 214: 657.1985.
112. Tao X, Zhang ZY, Dong H, Zhang H and Xin H. Responses of thyroid hormones of market-size broilers to thermoneutral constant and warm cyclic temperatures. Poultry Science, 85: 1520-1528. 2006.
113. Tixier-Boichard M, Decuypere E, Huybrechts L, Kühn E, Mérat P. Effects of dietary T3 on growth parameters and hormone levels in normal and sex-linked dwarf chickens. Domestic Animal Endocrinology, 7:573-85. 1990.
114. Usayran N, Farran M T, Awadallah H H O, Al-Hawi I R, Asmar R J, and Ashkarian V M. Effects of added dietary fat and phosphorus on the performance and egg quality of laying hens subjected to a constant high environmental temperature. Poultry Science, 80: 1695–1701. 2001.
115. Vecerek V, Strakova E, Suchy P and Voslarova E . Influence of high environmental temperature on production and haematological and biochemical indexes in broiler chickens. Czechoslovak Journal of Animal Science, 47: 176-182. 2002.
116. Webster AB. Physiology and behavior of the hen during induced molt. Poultry Science, 82:992-1002. 2003.
117. Wolfenson D, Frei YF, Snapir N, Berman A. Heat stress effects on capillary blood flow and its redistribution in the laying hen. Pflugers Archive, 390: 86-93. 1981.
118. Wolfenson D, Bachrach D, Maman M, Graber Y, and Rozen- boim I. Evaporative cooling of ventral regions of the skin in heat stressed laying hens. Poultry Science, 80:958–964. 2001.
119. Wright D, Kerje S, Lundström K, Babol J, Schütz K, Jensen P and Andersson L. Quantitative trait loci analysis of egg and meat production traits in a red junglefowl × White Leghorn crosses. Animal Genetics, 37:529–534. 2006.
120. Yost HJ, Petersen RB and Lindquist S. RNA metabolism: strategies for regulation in the heat shock response.Trends in Genetics, 6, 223–227.1990.
121. You S, Foster L K, Silsby J L, M E El Halawani, and D N Foster. Sequence analysis of the turkey LH-subunit and its regulation by gonadotropin-releasing hormone and prolactin in cultured pituitary cells. Journal of Molecular Endocrinology, 14:117–129. 1995.
122. Zeman M, Buyse J, Minvielle F, Bordas A, Merat P, Decuypere E . Effect of the sex-linked dwarf gene on plasma somatotropic and thyroid hormone levels and on energy metabolism of Leghorn and brown egg-type lying hens and their reciprocal crosses. Archive Gefluegelkd, 61: 66-71. 1996.
123. Zhang L, Ning Z, Xu G, HouZ, Yang N. Heritabilities and genetic and phenotypic correlations of egg quality traits in brown-egg dwarf layers. Poultry Science, 84:1209–1213. 2005.

DOI 10.5455/ijlr.20170312042324