Chapter 3
Heat Stress Impact on Livestock Production

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Abstract The effects of heat stress on several aspects of animal production are well documented. Heat stress results from the animal's inability to dissipate sufficient heat to maintain homeothermy. High ambient temperature, relative humidity, and radiant energy compromise the ability of animals to dissipate heat. As a result, there is an increase in body temperature, which in turn initiates compensatory and adaptive mechanisms to re-establish homeothermy and homeostasis. Heat stress could affect animal production and well-being, especially because of increase in air temperature. Heat stress is very common and on the increase particularly in the tropics. There is considerable research evidence that shows significant decline in animal performance when subjected to heat stress. Heat stress inflicts heavy economic losses on livestock production. The effects of heat stress is evident in feed consumption, production efficiency in terms of milk yield or weight gain per unit of feed energy, growth rate, egg production, and reproductive efficiency. The physiologic mechanisms underlying the action of heat stress on the decline of production performance of domestic animals have not been fully investigated. Heat stress requires further investigation, and the elucidation of the mechanisms may facilitate adoption of comprehensive preventive and control measures to combat heat stress in domestic animals. This chapter examines heat stress and its negative impacts on livestock production. It elucidates the general negative effects of heat stress on physiologic and production parameters of domestic livestock. The mechanisms involved when animals are subjected to heat stress and impacts of heat stress on domestic animals are emphasized. An understanding of these mechanisms may result in the development of improved techniques for enhancing livestock productivity in tropical environments.

Keywords Heat Stress · Livestock · Production

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3.1 Introduction

Domesticated animals, contribute to agricultural and economic development through provision of much needed protein of a nation; in particular, those countries in which the populations are growing faster than the economy. Animal protein requirements are met through domestic animal sources. The present supply of protein from domestic animals is currently inadequate to meet the protein requirement of the world's growing population because of many factors. One paramount and currently overriding factor is heat stress affecting the performance of domestic animals. It has been reported that domestic animals kept and managed in harsh environments are exposed to stress (Adeloye and Daramola 2004). Under extreme climate conditions, reduced ability of the animal to dissipate environmental heat results in significant heat stress during at least part of the year. Animals that are managed in these states loose condition, and often fail to perform when compared to animals not undergoing heat stress. Heat stress is one pressure placed upon an animal resulting in an increased need for the animal to dissipate excess body heat, requiring the animal to modify it's behavior to reduce this added stress. Figure 3.1 describes the details of the stress response in animals.

Physiologic changes do occur when animals are stressed but paramount and fundamental are loss of weight, reduced production, and death (Adeloye and Daramola 2004). Changes occurring in the animal as a result of heat stress include elevated body temperature, respiration rates, increased maintenance energy requirement, decreased efficiency of nutrient utilization, decreased dry matter (DM) intake, decreased milk production, and reproductive performance. The severity of heat stress experienced by an animal depends on actual temperature and humidity, length of the heat stress period, degree of night cooling that occurs, ventilation and air flow and level of production. The numerous physiologic mechanisms for coping with heat stress have been reported (Blackshaw and Blackshaw 1994). Sweating, elevated respiratory rates, vasodilation with increased

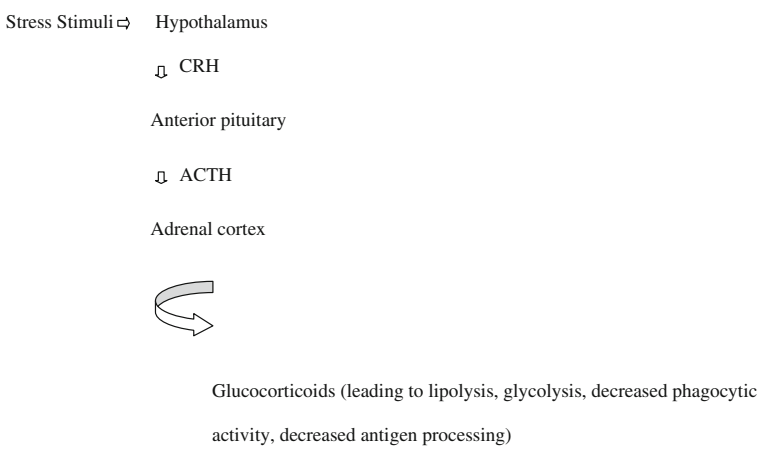

Fig. 3.1 Description of the stress cycle. The figure describes the stress cycle starting from the stress stimulus on hypothalamus to release CRH. CRH acts on the anterior pituitary and release ACTH. ACTH acts on adrenal cortex to release glucocorticoids to elicit the biologic actions to relieve stress (*Source* Adeloye and Daramola 2004)

blood flow to skin surface, reduced metabolic rate, decreased DM intake, and altered water metabolism are the physiologic responses that have negative impact on the production performance of livestock (West 1999). This review intends to integrate information documented on impacts of heat stress on the performance of domestic animals. The review identifies the mechanisms involved when animals are subjected to heat stress and knowledge of animal responses to heat stress may be used to evaluate the impacts of heat stress in animal production. Figure 3.2 describes the impact of heat stress on the productive parameters in livestock.

3.2 Feed Intake and Nutrient Utilization

Temperature levels outside the thermal comfort zone will increase body maintenance requirements for animals. Thermal comfort zone is a range in ambient temperature in which body temperature is possible and an animal need not change the metabolic rate. Consequently, efficiency of utilization of energy for production is decreased. Lowered efficiency occurs because animals eat less, thereby requiring more time to achieve a given level of weight and an appreciable amount of the energy consumed is siphoned off for the labor exerted to mitigate heat stress. The effect of heat stress on nutrient intake and utilization are well-documented in literature (Shanklin 1963; Attebery and Johnson 1969; Warren et al. 1974; Igono et al. 1985; Mallonee et al. 1985; Holter et al. 1996;). Studies have found that there is a significant negative correlation between Temperature-Humidity Index and DM intake (Shanklin 1963). However, differences exist for heat tolerance of different

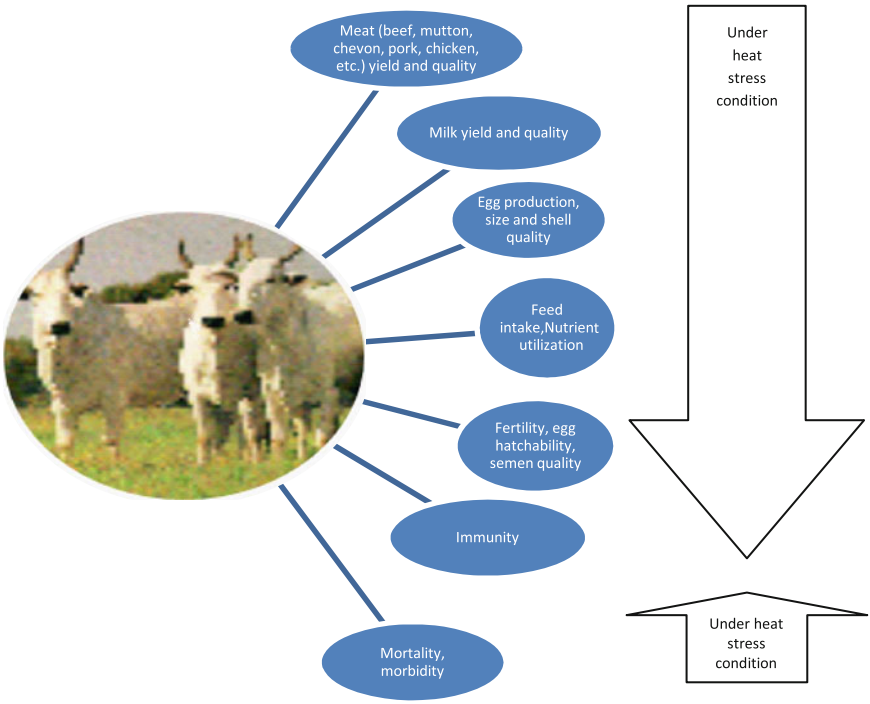

Fig. 3.2 Impact of heat stress on livestock production parameters. The figure describes the effect of heat stress on meat production, milk yield and quality, egg production, feed intake, nutrition utilization, fertility, semen quality, egg hatchability, immunity, mortality, and morbidity

breeds; and sweating capacity and metabolic rates are implicated in these variations (Blackshaw and Blackshaw 1994). Holter et al. (1997) reported a reduction in DM intake in Jersey cows when minimum Temperature-Humidity Index exceeded 56 and further reduced until Temperature-Humidity Index reached 72. During heat stress, DM intake is reduced to 22% for multiparous and 6% for primiparous dairy cows because of smaller body size and lower metabolic rate in primiparous cows (Igono et al. 1985). Although reduced DM intake and heat generated during ruminal fermentation and body metabolism aid in maintaining heat balance, heat stress caused by increased environmental temperature elevates the respiratory rate and water intake (Mallonee et al. 1985), reduces the gut motility, rumination, ruminal contractions (Attebery and Johnson 1969) and depresses appetite (Warren et al. 1974) by directly affecting the appetite center of the hypothalamus (Baile and Forbes 1974).

Feed consumption, feed quality, nutrient composition, rates of passage of digesta and volumes of ruminal and postruminal digestive organs are various factors that affect digestibility (Ellis et al. 1984). At high temperature, decreased feed intake evokes increased digestion by decreasing the passage of digesta and increasing the ruminal volume (Lippke 1975). These physiologic alterations are more helpful for animals consuming higher forage diets. Peripheral vasodilation

and central vasoconstriction cause reduced blood flow to the ruminant forestomach (Engelhardt et al. 1977). This in turn decreases the portal vein blood flow hence, reducing nutrient absorption (McGuire et al. 1989).

In periods of heat stress, the risks of acidosis are increased. Factors that can contribute to rumen acidosis problems are: decreased DM intake with lower proportion of forage and higher levels of fermentable carbohydrates, decreased rumination, and decreased saliva to the gut—a source of bicarbonate—with a reduction of its buffering power due to increased carbon dioxide (CO_2) expelled (Bach et al. 2007). Rumen pH also decreases which impairs fiber digestion efficiency: rumen fibrolytic bacteria are the most affected when rumen pH drops below 6.0 (Bach et al. 2007). All of these factors contribute to decreasing feed utilization during heat stress.

In poultry, reduction in feed intake consequentially results in deficiency of essential nutrients. Growth rate is reduced in broilers when environmental temperature rises because energy obtained from the small amount of feed consumed is expended in panting. The result is that birds have lower final body weight after normal feeding period with higher environmental temperatures. Often, the broiler chickens reach market weight of less than 4 kg at day 84 instead of day 42. Ain Baziz et al. (1990) observed that in heat-stressed birds, body weight gain reduced more than feed intake, because part of the metabolizable energy intake is used for heat dissipation, resulting in impaired feed conversion ratio. Heat stress, also leads to lower feed efficiency. More feed is required to produce a unit weight of chicken. Consequently, longer times are required to reach market weight in broiler chickens reared under heat stress conditions. These negative effects are found to be greater in poultry with a high potential for growth rate (Cahaner et al. 1998; Settar et al. 1999). High thermal loads may depress enterocyte proliferation, reduction in villus mass and dry weight per unit length of jejunum (Sahin et al. 2001). A number of morphologic and physiologic changes occur in the gastrointestinal tracts of chickens exposed to heat stress. There is a reduction in gut mobility and depression of gastrointestinal blood flow (Wolfenson et al. 2000). Combinations of all these effects, lead to reduction in digestibility of feed and nutrient absorption from the intestines. It is not known whether high ambient temperature has impacts on the secretion and efficiency of digestive enzymes. However, Sahin et al. (2001) showed that plasma triiodothyronine (T_3) and thyroxine (T_4), which are important growth promoters in animals, are adversely affected in heat-stressed broiler chickens. Heat stress in domestic birds elicits secretion and increase in plasma levels of corticosteroids, while that of plasma proteins decreases with marked increase in blood glucose concentrations. Therefore, the inhibition of growth and production in heat-stressed broiler birds may be engineered by stress hormones, especially corticosteroids. As suggested by these observations, it is likely that combined effects of reduced feed consumption, increased energy cost required for heat dissipation, altered metabolic and gut physiologic processes cause the reduction in weight gains.

The consequences of heat exposure during rearing and during transportation to slaughterhouses generally involve an increase in mortality, reduced meat yield, and quality. Seasonal heat stress has been reported to accelerate postmortem glycolytic metabolism leading to biochemical changes in muscle and the

production of pale, soft exudative meat characteristics in chickens. These detrimental effects are exacerbated in older birds (Sandercock et al. 2001). Ante and postmortem muscle metabolism is affected by stress reactions prior to slaughter. The rate and extent of glycogen breakdown, pH decline, and drip loss are also influenced. Terlouw (2004) stated that the effect is principally due to variations in adenosine-triphosphatase (ATPase) activity and muscle glycogen reserve. But acute heat stress appeared to have no effect upon breast meat color in broilers (Sandercock et al. 2001). No change in pH of broiler meat of acutely heat-stressed chickens was also reported. Terlouw (2004) indicated that production of meat with normal pH does not necessarily mean that animals have not been stressed. Lu et al. (2007) attributed high mortality and decreased growth, carcass, and breast muscle yield during heat exposure and indicated that heat stress did indeed cause physiologic stress in broilers, even though the resulting meat had normal pH.

3.3 Body Growth

Growth, the increase in live body mass, is under genetic and environmental regulation. The environmental factors that influence body growth include available nutrients, hormones, and enzymes as well as ambient temperatures (Bar and Radde 2009). Adequate and organized interaction of vascular growth factors and their receptors are required for placental development. When subjected to environmental heat stress at early stages of placental development, impaired placental vascular development occurs in part, due to lower levels of vascular growth factors in the tissues. Exposure to chronic heat stress has been observed to lower the circulating placental hormone concentrations due to impaired trophoblast cell development (Regnault et al. 2000).

The noticeable effects of heat stress on growth performance are the results of a decrease in anabolic activity caused by reduced voluntary feed intake, and increase in tissue catabolism. It is well-established that heat stress negatively impacts growth rate in swine. Although reduced feed intake undoubtedly plays a significant role in this reduction, studies in laboratory animals and other non-swine species indicate that muscle growth also is affected by heat stress—related alterations in muscle physiology (Kamanga-Sollo et al. 2011). Cattle, goats, and sheep are less sensitive to the effects of temperature than swine and poultry. Provided feeding is adequate, growth rate is not appreciably affected until the ambient temperature increases above the thermal comfort zone. When animal is subjected to temperatures outside of the comfort zone for a given animal size, the amount of feed required per unit of gain rises markedly. From the time of hatching until about 4 weeks of age, chicks have a narrow comfort range (32–34°C), efficiency of gain is reduced if the chicks are kept at a high temperature, growth rate will be almost ceased by the 7 or 8th week. While cold is a limiting factor in early stages of animal development, heat is most limiting in latter stages.

It is well-established that heat stress negatively impacts growth rate in animals. Although reduced feed intake undoubtedly plays a significant role in this reduction, studies in laboratory animals indicate muscle growth also is affected by heat stress-related alterations in muscle physiology. There is now emerging evidence that heat shock proteins (hsp), produced in response to heat stress and other types of cellular stress, may play important roles in regulating rate and efficiency of muscle growth. Stress, whether thermal or otherwise causes denaturing of proteins in the cells. During heat stress, the synthesis of hsp is increased while synthesis of other proteins is downregulated. So, the proteins that should have helped as building blocks are denatured or not synthesized at all. Because muscle satellite cells play crucial roles in postnatal muscle growth, the effects of heat stress on rates of satellite cell proliferation, protein synthesis, and protein degradation play an important role in determining the rate and extent of muscle growth.

Chronic exposure of growing pigs to a high ambient temperature is associated with enhanced lipid metabolism in the liver and the adipose tissue (Kouba et al. 2001). As a consequence, plasma triglyceride uptake and storage is facilitated in the adipose tissues, which results in greater fatness (Kouba et al. 2001). Increased fatness in long-term heat-exposed pigs was accompanied by the changes in the distribution of adipose tissues: a shift of body fat toward internal sites (Le Dividich et al. 1998), an increased weight of flare fat, and increased ratio of flare fat:back fat + flare fat (Kouba et al. 2001). The change in fat distribution in these heat-exposed pigs would appear to increase heat loss and represents an adaptation to high ambient temperature (Le Dividich et al. 1998; Kouba et al. 2001). Heat-exposed chickens also exhibit enhanced fat deposition (Ain Baziz et al. 1990).

Feed efficiency under hot conditions differs somewhat between mammals and birds. Feed to weight gain ratio is enhanced in hot conditions in chickens (Ain Baziz et al. 1990). On the other hand, an improvement in feed efficiency is often observed in rats and pigs under heat exposure (Rinaldo and Le dividich 1991). Stress reactions prior to slaughter may influence ante and postmortem muscle metabolism, and consequently, the rate and extent of glycogen breakdown, pH decline, and drip loss. Seasonal heat stress accelerates postmortem metabolism and biochemical changes in the muscle, which produces a faster pH decline, lower ultimate pH, and higher lightness values in turkey meat (McKee and Sams 1997). McKee and Sams (1997) and Lu et al. (2007) showed that chronic heat stress increased the lightness in muscle. The impact of stress response on meat quality is not inevitable. Terlouw (2004) indicated that production of meat with normal ultimate pH does not necessarily mean that animals have not been stressed.

3.4 Milk Production

During heat stress cows exhibit reduced feed intake, decreased activity, increased respiratory rate, and increased peripheral blood flow in sweating. When heat stress is experienced close to calving, an additional negative side effect is reduced cow's

ability to produce high quality colostrum and impaired transfer of maternal IgG's to colostrum (West 2003). Levels of milk yield are sensitive to temperature conditions because temperature affects the feed intake of lactating cow. Temperature level at which significant depressions in milk yield occur depends on the humidity conditions, level of production, size of animal, and the breeds involved. At higher temperature above the comfort range, milk yield is depressed.

Heat stress is most detrimental to dairy cattle. Consequently reductions in feed consumption, milk production, and reproductive performance have been reported (Cavestany et al. 1985; Sharma et al. 1988; Bernabucci et al. 1999). Heat stress is a major factor contributing to low milk production and low fertility in lactating dairy cows. Various tissues are affected and their functions disrupted under heat stress (Wolfenson et al. 2000). High milk production is associated with high metabolic heat production. As a result, cows have to dissipate larger amounts of heat in order to maintain normothermia. At air temperatures of 27°C, under humid climates, the body temperature of lactating cows rises above normothermic values, and severe hyperthermia develops as air temperature rises (Wolfenson et al. 2000). The ambient temperatures rise to levels that induce hyperthermia in lactating cows under heat stress. The impact of heat stress is compounded by relatively low sweating rate in cattle (Berman and Wolfenson 1992).

The severity of heat stress is correlated to both ambient temperature and humidity level (Bach et al. 2007). The animal comfort is optimal, with a body temperature between 38.4 and 39.1°C. Above 25°C, and even 20°C for some authors, the cow suffers from heat stress: its health status and production performance are affected. Cows have ways to maintain thermal balance and regulate body temperature under high heat conditions. This involves favoring heat dispersion, in particular through evaporation, by increasing subcutaneous blood flow, panting, drooling, etc. These activities increase the maintenance energy needs of the animal by an estimated 20% at 35°C (Bach et al. 2007). In the case of the dairy cow, this means that part of its production energy will be redirected to thermal regulation. Also, rumination, which produces heat, decreases dramatically. Cows will tend to eat less during the day, but more often and in small quantities. They will tend to consume more feed at night when it is cooler, slug feed, sort feed and tend to choose feeds that produce less heat during digestion, choosing grains and proteins over forages.

3.5 Semen Production and Sperm Characteristics

Some physiologic traits that have direct bearing on quality of ejaculate are known to be affected when male animals are stressed due to handling, methods of ejaculation and elevated temperature during the time of semen collection (Marai et al. 1997). Among all climatic elements, temperature is the most important

parameter affecting spermatogenesis. Skinner and Louw (1966) reported that high ambient temperature causes a sharp reduction in semen quality with many abnormal sperm cells. Exposure to heat stress is registered by the temperature-humidity index that includes both ambient temperature and relative humidity (LPHSI 1990; Marai et al. 2000). Heat stress is known to cause temporary interruption of sperm production, sperm motility, and secondary defects (Moreira et al. 2001).

Heat stress affects all phases of semen production in breeder cocks as reported in other species (Banks et al. 2005). Although limited high temperature stimulates testicular growth in the early phase and promotes increased semen volume and concentration, a subsequent rise lead to decreased semen quality and quantity with time (Obidi et al. 2008; McDaniel et al. 1996; Edens 1983). Serum calcium and phosphorus levels were observed to be significantly lowered in heat-stressed birds (McDaniel et al. 1995, 1996). Transient inward calcium ion currents whose density increased during spermatogenesis, from spermatogonia to early spermatids, have been observed (Hagiwara and Kawa 1984). The decrease in spermatogenesis due to inhibition of calcium and potassium ion exchange (Hagiwara and Kawa 1984; Schreiber et al. 1998), implies that distinct expression and non-inhibition of ion channels during spermatogenesis may enhance the excitation and differentiation of seminiferous epithelium (Hagiwara and Kawa 1984; Schreiber et al. 1998), as is characteristic of excitable tissues. Some of the ion channels regulating ion exchange during the preliminary stages of germinal cell differentiation end up in mature spermatozoa, determining their physiologic properties (Schreiber et al. 1998. The decrease in calcium ion level due to heat stress causes deleterious multiple effects on testicular function through inhibition of intracellular ion exchange (McDaniel et al. 1996).

In a study conducted to investigate the changes that might occur in spermiograms, blood and physiologic indices following successive electroejaculation (EE) during cold and hot periods of the day, progressive sperm motility, sperm concentration, and mass activity followed similar trend and the values deteriorated with respect to elevated temperatures during semen collection periods (Daramola and Adeloye 2010). Primary abnormalities increased with respect to elevated temperatures during semen collection periods (Daramola and Adeloye 2010). The authors observed that reduced ejaculate quality reflects stress stimuli arising from increase ambient temperature and physiologic traits in West African Dwarf (WAD) goat and reported it as the adaptive mechanism evolved to cope with stress arising from elevated temperatures. The proportion of the sperm abnormalities increased concurrently in response to increased ambient temperature intensity (Daramola and Adeloye 2010). The increase in morphologic abnormalities at high ambient temperature indicates that periods of collection have deleterious effect on the testes or epididymis, such as testicular degeneration (Daramola and Adeloye 2010). Skinner and Louw (1966) reported that high ambient temperature causes a sharp reduction in semen quality with many abnormal sperm cells. The rise in primary abnormalities therefore indicates that elevated ambient temperature results in the rapid release of immature spermatozoa (Skinner and Louw 1966; Daramola and Adeloye 2010).

The reproductive performance of the rooster is greatly depressed during environmental stress. In a simulated study on the effects of heat stress on fertility in broiler breeder roosters, McDaniel et al. (1996) showed that the broiler breeder contributed more to heat-induced infertility than the female. When the male broiler breeder was exposed to a temperature of 32°C, male fertility declined to 42% and in vivo sperm-egg penetration declined to 52%, compared to values obtained from males that were maintained at 21°C. This observation demonstrated a significant inhibition of the rooster's spermatozoa viability through qualitative and quantitative depression in semen characteristics, such as spermatozoa motility.

Heat stress may be responsible for the inhibition of osmotic equilibrium and ionic channels that are key elements in the interplay between spermatozoa, its environment, and the egg, thus disrupting spermatozoa cellular homeostasis, distorting spermatozoa behavior, and metabolic machinery (Darszon et al. 1999). In mammals, excessive levels of reactive oxygen species have been correlated with decreased sperm motility (Agarwal et al. 1994; Armstrong et al. 1999). The report of McDaniel et al. (1996) showed that semen characteristics, such as consistency, spermatozoa concentration, and seminal volume were depressed as a result of a decrease in seminiferous epithelial cell differentiation, which is manifested by environmental temperatures outside the zone of thermal comfort. Heat-induced infertility is mediated through any compromise in the fluidity and integrity of spermatozoa cell membranes as well as acrosomal and deoxyribonucleic acid damage (Surai 2000, 2002, 2010), and the inhibition of expression of hyaluronic acid binding sites as well as acrosomal integrity (Shamsuddin and Rodriguez-Martinez 1994; Morrell and Rodriguez-Martinez 2011). The differences found when breeder cocks were exposed to elevated ambient temperatures were not evident when the female birds alone were exposed to the same high ambient temperatures (McDaniel et al. 1996; Abd-Ellah 1995). Edens (1983) reported significant effects of ambient temperature on male fertility, which were evident within 12 h of challenge at a typical summer temperature of 29°C, although semen characteristics, such as semen volume, spermatozoa concentration, and percentage dead spermatozoa were unaffected by the heat treatment. This apparent lack of observable depreciation in semen characteristics obtained in the study of Edens (1983) suggests that roosters can adapt to short-term exposure to thermal stress. Thus, physiologic changes inimical (injurious/harmful) to testicular functions may not occur in short-term exposure to heat stress. The finding of Edens (1983) disagreed with those of McDaniel et al. (1995, 1996), who subjected roosters to a long-term heat exposure. The depression in in vivo sperm-egg penetration and fertility in heat-stressed roosters reported by McDaniel et al. (1995, 1996) may be due to a decrease in number of spermatozoa stored in the sperm host glands in the hen's reproductive tract (Bakst et al. 1994; Brillard 2003). In other words, a decrease in oviductal spermatozoa storage results in fewer spermatozoa cells available to bind, penetrate, and fertilize the egg in the infundibulum of the hen as documented by King et al. (2002).

In mammals, spermatozoa's binding with uterine epithelial cells is a strong index of spermatozoa viability and fertilizing capacity, implying that spermatozoa

attachment to uterine epithelial cells is indicative of normal ultrastructure and mitochondrial membrane potential (Mburu et al. 1996; Taylor et al. 2008; Taylor et al. 2009). It is known that spermatozoa that have been bound temporarily to uterine epithelial cells can pass along the oviduct for fertilization (Taylor et al. 2008). In this context, it is reasonable to conclude that heat stress in the rooster retards or even prevents important physiologic mechanisms, such as sperm-uterine epithelial cells interaction, capacitation, acrosome reaction, and zonal vesicle binding, resulting in depressed fertility. This is, apparently, due to a depletion of endogenous antioxidant milieu in semen, leading to speedy exhaustion of spermatozoa energy reserves. On the other hand, it is likely that exposed spermatozoa are properly stored in the hen's oviduct, but their release was inhibited; thus, the spermatozoa were unable to bind and penetrate the ovum (Abd-Ellah 1995; King et al. 2002; Brillard 2003). It is worthy to note that roosters in pen-mated (natural mating) breeding system are known to reduce mating activity, and sexual arousal behavior (libido) is strongly impaired during heat stress, presumably through dehydration and alteration in secretion of sex hormones.

3.6 Immune Response and Endocrine System

Several studies have been conducted on the effects of heat stress due to high temperature on the immune responses of chickens, with variable results. Thaxton et al. (1968) demonstrated that high environmental temperatures (44.4–47.8°C) affect the development of specific immune responses in young chickens. These effects include the suppression of circulating white blood cells (Nathan et al. 1976; Heller et al. 1979) and an increase in the heterophil/lymphocyte ratio (H/L ratio) (Mogenet and Youbicier-Simo 1998), which are indicators of stress (Gross and Siegel 1983). Heat stress also reportedly causes a reduction in antibody production in young chickens (Zulkifi et al. 2000). On the other hand, Donker et al. (1990) found that heat exposure did not reduce antibody production, rather, significantly increased antibody titers were observed following heat exposure (Heller et al. 1979). The difference in these findings could be associated with age and breed. Regnier et al. (1980) suggested that heat-induced immunosuppression may depend on breed of bird and Kelley (1983) reported that effects on immune responses may depend on the length and intensity of the heat exposure.

Heat stress can negatively affect an animal's growth performance and the immune competence to some bacterial or viral infections (Goligorsky 2001). It has been reported that heat stress results in decrease of both primary and secondary lymphoid organs, profiles of circulating leukocytes, T cell in the blood, and antibody response to sheep red blood cells or against Newcastle disease (Davison et al. 1988; Liew et al. 2003).

The endocrine system involved in coordination of metabolism is substantially altered because of thermal stress (Beede and Collier 1986). The hormones associated with adaptation to heat stress are prolactin, growth hormone, thyroid hormones, glucocorticoids, mineralocorticoids, catecholamines, and antidiuretic hormone. Prolactin is vital for mammogenesis (Buttle et al. 1979), lactogenesis (Akers et al. 1981). Growth hormone is a calorigenic hormone produced from the anterior pituitary gland and does not function through a target gland but exerts its effects on almost all tissues of the body. Igono et al. (1988) reported that Growth hormone content in milk of low, medium, and high production groups declined when Temperature-Humidity Index exceeded 70. Plasma Growth hormone reductions that occurred with heat-stressed cows did not occur in thermoneutral conditions for cows fed restricted intakes that were similar to those consumed during heat stress (McGuire et al. 1989).

Although stress isn't the only reason that cortisol is secreted into the bloodstream, it has been termed "the stress hormone" because it's also secreted in higher levels during the body's response to stress, and is responsible for several stress-related changes in the body. Daramola and Adeloye (2010) and Daramola et al. (2011) reported increased cortisol level with respect to the semen collection periods and indicated that the increase observed in cortisol concentrations in the hot period of the day reflects stress stimuli due to elevated ambient temperature (Daramola and Adeloye 2010; Daramola et al. 2011). Apparently, the higher cortisol levels observed was attributed to stress caused by increase in ambient temperature (Daramola and Adeloye 2010), similar to the reports of Ortiz-de-Montellano et al. (2007) and therefore reflects stress stimuli due to elevated ambient temperature.

3.7 Blood Parameters

There is a great variation in the hematologic and biochemical parameters as observed between breeds of goats (Azab and Abdel-Maksoud 1999; Tambuwal et al. 2002). Seasonal variations in hematologic parameters of domestic chickens have also been reported (Oladele et al. 2003). Lowered packed cell volumes in domestic chickens have been observed during the hot-dry season, a period associated elevated ambient temperature as compared to other season (Oladele et al. 2003). Oladele et al. (2001) attributed the low values of hemoglobin and packed cell volume during the hot-dry season to heat and nutritional stress, which impairs the production of blood cells in birds and further observed significant correlation between the hot-dry season meteorological elements and packed cell volume, hemoglobin, and total protein in the chicken. Hemoglobin therefore seems to be highly responsive to fluctuations in ambient temperature, with a significant negative response to the deleterious effect of heat stress. Total protein values also demonstrate a significant and negative relationship with elevated ambient temperature (Oladele et al. 2003). These observations support the findings of Sahin et al. (2001), who demonstrated significant negative effects of heat stress on total

serum proteins in broiler chickens. Sahin et al. (2001) also reported significant negative effects of heat stress (32°C) on serum concentration of some metabolites and minerals in broilers. Serum levels of thyroxine (T_4) and triiodothyronine (T_3) were significantly reduced due to high levels of adrenocorticotropic hormone in unsupplemented (vitamins E and A) broiler chickens, when compared to values obtained in birds that received antioxidant feed supplements (vitamins E and A). Serum calcium and phosphorus levels were observed to be significantly lowered in heat-stressed birds (McDaniel et al. 1995, 1996).

3.8 Egg Production

The effect of heat stress in laying birds is chronologic: laying flocks typically have a reduction in egg size, followed by lowered egg production, and reduced egg shell quality (Grieve 2003). Ambient temperatures influence reproductive function through alteration of feed intake. Reduction in feed intake resulting from heat stress is observed in layer chickens during hot-dry seasons (Simon 2003; Ayo et al. 1999) which ultimately leads to reduction in hen-day production. The observed decreases in voluntary feed intake by birds is attributed to physiologic responses to heat stress, aimed at reducing the excessive endogenous heat generated in the body due to feed metabolism (Simon 2003). The depression is attributed to an imbalance in calcium-estrogen relationship and lowered Haugh unit of the ovalbumin (Mahmoud et al. 1996). This implies that high environmental temperature depresses yolk size, ovalbumin consistency, and optimum calcium deposition within the egg shell.

Various authors reported that increased environmental temperatures affect egg production. Smith (2000) stated that the effect of ambient temperature on average egg weight appears to be cumulative. Thus, when birds are kept at 26°C, the mean egg weight increases by 1 g per week whereas when kept at 35°C, the average egg weight remains constant for a period of six months. North (1984) illustrated the effect of poultry house temperature on egg production, egg weight and feed consumed per egg as percentage of the optimum of 16°C. The author did not observe any change in egg production until a temperature of 24°C was reached. However a gradual decline in egg production was recorded as the temperature increased to 32°C.

Egg shell weight, shell thickness and specific gravity significantly declined in laying hens heat-stressed for 5 weeks (Mashaly et al. 2004). The heat-stressed laying flock often lays eggs with thinner shells because of acid–base disturbances in the blood (respiratory alkalosis). The higher blood pH caused by decreases in blood CO_2 concentrations reduce the amount of ionized calcium and bicarbonate in the blood. Ionized calcium is the form of calcium utilized by the shell gland in producing the egg shell (Grieve 2003). Increasing the amount of calcium in the diet does not correct this problem.

3.9 Incubation, Embryonic Development and Hatchability

The modern incubator is a simulated artificial design that mimics the mother-hen's role of providing fertile eggs with optimum environmental conditions (temperature and humidity) to stimulate embryonic development until hatching (French 1997). Deleterious effects of heat stress on the incubation of the avian embryo, hatchability, post-hatch development of chicks are well-documented (Romanoff 1972; Deeming and Ferguson 1991; French 1997; Hill 2001; Moraes et al. 2003, 2004; Lourens et al. 2007). Optimum environmental conditions are synonymous with incubation temperatures, which determine the efficiency of embryonic and post-hatch development of chicks (Lin et al. 2006; Romanoff 1972). In line with observation of Romanoff (1972), French (1997), Hill (2001) and Lourens et al. (2007) reported deleterious effects of heat stress on the incubation of the avian embryo, and showed extensive influences of temperature on chicks' embryo development, and that environmental temperature is the most critical factor in incubation efficiency. Wilson (1991), Lourens et al. (2005, 2007) and Moraes et al. (2003) confirmed the adverse effects of temperature on post-hatch development of chicks. It has been shown that a constant incubation temperature of 37.8°C, established as thermal homeostasis in the chick embryo (Lourens 2001), gave the best embryo development and hatchability (Lourens et al. 2007; Wilson 1991). Any marginal deviation from this fragile balance is detrimental to the developing embryo (Lourens et al. 2007). Thus, a constant high temperature of 38.9°C during incubation initially accelerates embryonic growth, utilization of nutrients and energy from the yolk and albumen reserves, but later decreases embryonic development as a result of limited metabolic process by insufficient exchange of oxygen (Lourens et al. 2005; Rahn et al. 1974).

Heat stress in the incubation process has been shown to have diverse detrimental influences on embryos. French (1994) observed deleterious effect of heat stress on embryo survival and showed that dead embryos occurred soon after subjecting them to heat stress, especially on days 7 and 19. This implies that embryos at these stages of development may be very sensitive to all types of stress, including heat stress, which could be related to the chorioallantoic membrane susceptibility to environmental stress. Increased embryonic death is, apparently, due to increased endogenous (metabolic) heat production (French 1994). The observation is in line with the findings of Lourens et al. (2005), who reported significant embryo mortality and, hence, lower hatchability in chicken eggs, when they were subjected to a high incubation temperature of 38.9°C. Apart from embryonic mortality, the quality of chicks from heat-stressed embryos has been reported to be adversely affected. Lourens et al. (2005) established depressed chick quality, lower percentage of first-grade chicks due to adverse effects of heat stress on chick quality and production.

Temperature is the most important factor in the incubation of any avian egg. An egg can, to a certain degree, compensate for various insults to its well-being except for temperature extremes. A 0.5°F change in incubator temperature can

have a profound effect in overall performance of a group of eggs that incubate (Jeffrey et al. 2007). Extremes in temperature (high or low) can cause problems with embryo growth, or in many cases death. Eggs that are hatching early or very small chicks at hatch are an indication of incubator temperatures being too high (Jeffrey et al. 2007).

As water evaporates during incubation, it is replaced by gas molecules that form the air cell, which should occupy about 15% of the egg volume prior to hatching (Rahn et al. 1977) Increasing shell porosity and permeability will increase the oxygen uptake of the embryo until a maximum rate is reached, after which further increase in shell porosity and permeability have little effect (Burton and Tullett 1982). Therefore, oxygen availability is not a limiting factor for embryos within highly permeable, porous shells, but rather there is the danger of dehydration caused by elevated temperature (Burton and Tullett 1984). During incubation, eggs should ideally lose a quantity of water equal to 12% of their initial mass (Davis and Ackerman 1987). Water loss exceeding 20% of initial egg mass causes increased mortality and subsequent dehydration of the embryo, thus decreasing hatching success (Davis and Ackerman 1987).

Heat-stressed embryos have been observed to exhibit shorter face length and low lung weight, resulting in weaker chicks with high incidence of culled-out birds due to unsteady gait (Yalcin and Siegel 2003). An increase in environmental temperature may cause metabolizable energy to be diverted from growth and development to functions involved in homeothermy. High environmental temperatures reduce thyroid function and, consequently, metabolic rate, oxygen consumption, and growth rate (Romanoff et al. 1938; Moraes et al. 2003). Christensen et al. (2002) showed that the chick embryonic thyroid plays a major role in maturation of vital tissues during the final stages of in ovo life; the authors reiterated that the embryonic thyroid had a significant control of hatching times and survival rates of neonates. These reports support the fact that lower egg fertility and hatchability, retarded embryonic and post-hatch chick developments are due to consistent heat stress (Abioja 2010).

3.10 Conclusion

Heat stress occurs at the point where the animals cannot dissipate an adequate quantity of heat to maintain body thermal balance. High temperature, high humidity, and radiant energy (sunlight) are the major environmental factors that contribute to heat stress. Heat stress is very common and on the increase particularly in the tropics. There is considerable research evidence that shows significant decline in animal performance when subjected to heat stress. It inflicts heavy economic losses on livestock production. The effects of heat stress is evidenced in feed consumption, gross efficiency in terms of milk yield or weight gain per unit of feed energy, growth rate, milk production, egg production, and reproductive efficiency. The effects should be considered in future experiments, designed to

elucidate the mechanism of heat stress on production efficiency in the domestic animals reared in the tropical and subtropical regions of the world. The physiologic mechanisms underlying the action of heat stress on the decline of production performance of domestic animals have not been fully investigated. This requires further investigation, and the elucidation of the mechanisms may facilitate the adoption of comprehensive preventive and control measures of combating heat stress in domestic animals.

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