



Effects of Environmental Heat Stress on Reproduction and its Management in Chickens

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SUMMARY

The effects of environmental heat stress on reproduction in chickens were reviewed to find out the associated physiological changes and the interplay of the physiological events, after which the role of certain measures in the management of heat stress were identified. The normal rectal temperature for chicken is 40.6 - 43.0 °C with 45 °C as the upper limit of safety. The rectal temperature begins to increase when the ambient temperature increases, but this is prevented by panting to increase heat loss by respiratory evaporation of water. Chickens are said to be heat stressed when their body heat builds up during high ambient temperature. The physiological responses associated with it include respiratory alkalosis from panting, reduced feed intake and efficiency, reduced absorption of calcium, decrease in secretion of thyroid hormones, estrogen, progesterone and testosterone, and increased secretion of glucocorticoids, luteinising hormone and follicle stimulating hormone. Consequently, heat stress produces decrease in anabolism, increase in catabolism and insulin resistance, loss in body weight, delayed sexual maturity, reduction in number and quality of eggs, infertility in male and female chickens and reduced hatchability of fertile eggs with the greatest effect being on the number and quality of eggs produced. The impact of environmental heat stress remains a challenge as it affects reproduction in chickens and researches in this area are necessary to support increased productivity of chickens in hot semi-arid regions.

Key words: Chickens, eggs, heat stress, management, reproduction.

INTRODUCTION

Chickens are kept for meat and egg production. The normal body temperature range of chickens is 40.6- 42.4°C (Aengwanich, 2008). When the ambient temperature rises above the thermo neutral zone and is beyond the upper critical temperature limit of chickens, the chickens experience heat stress. A lot of reproductive changes are associated with heat stress in

chickens. The reproductive ability of breeder chickens is reduced (McDaniel *et al.*, 1996) and the quality of semen produced is affected by heat stress (McDaniel *et al.*, 1996; Karaca *et al.*, 2002; McDaniel *et al.*, 2004). Heat stress causes a decrease in the quality of eggs laid (Franco-Jimenez and Beck 2005; Rozenboim *et al.*, 2007) and the number of eggs hatched (Van Kampen, 1981; Peebles and Brake, 1985; Muiruri and

Harrison 1991; McDaniel *et al.*, 1995). Heat stress causes physiological changes in chickens which are responsible for the effects on reproduction (Zulkifli 1995). This review focuses on the physiological changes associated with heat stress as it affects reproduction. It further presents ways of managing heat stress.

WHAT IS HEAT STRESS?

Chickens are heat stressed if they have difficulty achieving a balance between body heat production and body heat loss. The thermo neutral zone is defined as the range of effective ambient temperatures within which the heat from normal maintenance and productive functions of the animal in non-stressful situations offsets the heat loss to the environment without requiring an increase in rate of metabolic heat production (NRC 1981; Hillman *et al.*, 1985; Ensminger *et al.*, 1999; Sahin and Kucuk, 2003). The chicken has an upper critical temperature of 45°C in Borno State (Ubosi, 1998). High environmental temperature with relative humidity is dangerous to chickens. The zone of thermal comfort for birds is between 40.6 and 43°C (Andersson and Jonasson, 1993). When the body temperature goes beyond the upper critical limit of the chicken, the chicken uses physiological means to reduce body temperature by convection, conduction and radiation. Chickens use panting as the major form of evaporative cooling since they lack sweat glands (Andersson and Jonasson, 1993). Environmental heat stress in chickens is caused by high ambient temperature and humidity. Water deficit, population density and diseases can make the effects of heat stress more severe.

PHYSIOLOGICAL RESPONSES TO HEAT STRESS

Presenting signs

The presenting signs of heat stress in chickens are panting, prostration, extension

of wings, clustering around drinkers and mortality.

Feed intake

The initial reaction of chickens to the onset of heat stress in the sub-sahelian zone of Nigeria is reduction in voluntary feed intake (Ubosi and Geidam, 1991-1992). Feed efficiency is reduced in heat stressed chickens. Chickens want to reduce metabolic heat production associated with digestion that is one of the reasons why there is a decrease in feed intake.

Water intake

Heat stressed chickens will drink water in large quantities (Borges *et al.*, 2004). Water intake is greatly increased and the birds drink to cool off and reduce their body temperature.

Endocrine response

Plasma concentration of corticosterone is high and there is an increased level of circulating glucocorticoids which stimulates gluconeogenesis, and this causes an increase in blood glucose levels (Fixter *et al.*, 1987; Franco-Jimenez and Beck, 2007). Catabolism occurs due to gluconeogenesis, resulting to muscle wasting and subsequently weight loss. Hypothyroidism was observed in heat stressed chickens by Sahin and Kucuk (2003). Hypothyroidism causes decreased metabolism, and this leads to decreased feed efficiency. Insulin resistance in heat stressed laying hens was reported by Borges *et al.* (2004). There is also a decrease in serum concentrations of vasotocin, mesotocin and prolactin (Nouwen *et al.*, 1984) and an increase in melatonin (Zulkifli and Siegel, 1995). Serum concentrations of testosterone, oestrogen and progesterone are decreased while follicle stimulating hormone (FSH) and luteinising hormone (LH) levels are increased (Sahin and Kucuk, 2003, McDaniel *et al.*, 2004). Plasma insulin

concentration decreases significantly with high temperature (Habeeb, 1987). Decreased plasma insulin concentration due to large decrease in potassium retention under heat stress has also been reported (Niles *et al.*, 1980).

Electrolyte imbalance

Fixter *et al.* (1987) and Borges *et al.* (2004) reported that hemodilution in acute heat stress caused a decrease in sodium ions concentration and an increase of potassium ions in the blood stream due to altered membrane permeability. Prolonged panting results in respiratory alkalosis because of excessive loss of water and carbon dioxide which results in loss of bicarbonate ions (Teeter *et al.*, 1985; Teeter and Smith, 1986).

Denaturation of vitamins

Vitamins are oxidised and denatured in heat stressed chickens (McDaniel *et al.*, 2004). The levels of vitamin A, B, C, D, and E are significantly reduced. Vitamin A is needed for the maintenance of epithelial tissues, vitamin B is needed for metabolism of amino acid and breakdown of glycogen to glucose, and vitamin C is required for the synthesis of testosterone, oestrogen and progesterone. Vitamin D is required for absorption of calcium from the gut. Vitamin E is required for normal reproduction and musculature development.

Release of heat shock protein

Heat shock protein (Hsp) is a small subset of proteins that is detected only when living cells are responding to an exposure to metabolic stress (Morimoto *et al.*, 1997; Givisiez *et al.*, 1999). Heat shock protein; Hsp 70 is undetected under normal conditions but is highly induced in cells experiencing stress (Smith, 1993; Deyric *et al.*, 1999; Franco-Jimenez and Beck, 2007).

REPRODUCTION

Male reproduction

Hormones

Testosterone is responsible for maintaining spermatogenesis and ascorbic acid is required for the synthesis of testosterone (McDowell, 1989; Kutiv and Forbes, 1993). Thornton (1961) reported that ascorbic acid levels in plasma are inversely proportional to environmental temperatures. As environmental temperature rises, the ascorbic acid levels in plasma decreases. A decline in seminal ascorbic acid has been associated with a decrease in fertility in humans (Jungck *et al.*, 1947). In heat stressed cocks, the synthesis of testosterone is defective and reduced because of decreased serum concentration of ascorbic acid. Serum concentration of testosterone is low and because of negative feedback, FSH and LH are secreted in large quantities (McDaniel *et al.*, 2004).

Spermatogenesis

Spermatogenesis takes place in the seminiferous tubules within the testes. When body temperature is increased, the three stages of spermatogenesis are distorted. During spermatocytogenesis, the DNA construction is altered due to abnormal mitotic divisions (McDaniel *et al.*, 2004). Certain meiotic stages are skipped which results in formation of defective spermatids (McDowell, 1989). During spermiogenesis, a lot of mishapened spermatozoa are produced; some have no heads, some have no tails while some have unusually large heads. Acrosome formation does not take place in some spermatozoa (Joshi *et al.*, 1980 and McDaniel *et al.*, 2004). Semen quality is reduced in heat stress (Joshi *et al.*, 1980).

Storage of spermatozoa

Spermatozoa are stored in the testes until during spermiation, when the spermatozoa move through the deferent duct to the

cloaca. Heat stress damages the stored spermatozoa (McDaniel *et al.*, 2004).

Transport of spermatozoa

Heat stress can destroy the spermatozoa as they move through the deferent duct. Spermatozoa that were produced and stored before exposure to heat stress are damaged during transportation (McDaniel *et al.* 2004).

Mating behaviour

Cocks are reluctant to mate during heat stress because they avoid contact with any warm surface or body to prevent heat gain by conduction (McDaniel and Parker, 2004). The decrease in sexual drive may occur due to reduction in serum concentration of testosterone. This change in mating behaviour leads to a decrease in the number of fertilised eggs laid by hens kept for breeding.

Female reproduction

Hormones

Oestrogen is responsible for the development of the oviduct and sexual maturity. Progesterone and testosterone stimulate the formation of albumen. Progesterone also signals the release of LH from the pituitary gland. Yolk development is initiated by FSH (Lucas and Ostagno, 2013). Acute and chronic heat stress cause significant reduction in the concentration of progesterone and testosterone in serum of laying hens due to denaturation of ascorbic acid which is required for their synthesis but no significant change in LH and FSH concentration (Rozenboim *et al.*, 2007).

Folliculogenesis

A sexually matured hen has only the left ovary functioning. The ovary is activated as the time of egg laying approaches. In heat stressed hens, the concentration of reproductive hormones is decreased, and follicles do not grow well. Few follicles are

stimulated to start growing due to decreased levels of oestrogen, progesterone and testosterone (Smith and Oliver, 1972; De Andrade *et al.*, 1977; Wolfenson *et al.*, 1979; Dankoh, 1989; Mills *et al.*, 1999; Sahin and Kucuk, 2003; Rozenboim *et al.*, 2007).

Ovulation

A hen is said to ovulate when the yolk is released from the matured follicle. Nervous and hormonal factors are responsible for the initiation of ovulation (Lucas and Ostagno, 2013). If a hen is heat stressed, the initiation of ovulation is delayed due to decrease number of matured follicles and serum concentration of progesterone and testosterone levels (Rozenbiom *et al.*, 2007). The time of previous egg passing through the vent (egg lay) also triggers the next ovulation (Lucas and Ostagno, 2013).

Egg formation

The infundibulum engulfs the yolk that is released from the matured follicle and fertilization of the blastodisc (blastoderm) takes place in the infundibulum (Eastin and Spaziani, 1978). Oestrogen that is responsible for development of the oviduct is decreased in heat stress. An improperly developed infundibulum fails to engulf the released yolk (Lucas and Ostagno, 2013). If the spermatozoon that penetrates the blastodisc is defective due to abnormal DNA construction due to heat stress, fertilization will not take place (McDaniel *et al.*, 2004). Deposition of 40% of the albumen takes place in the isthmus. In a heat stressed hen, progesterone and testosterone which are responsible for albumen formation is in low concentration and this leads to production of eggs with poor albumen quality (De Andrade *et al.*, 1977; Dankoh, 1989; Rozenboim *et al.*, 2007).

Some albumen and the shell membranes are added in the ampulla. Heat stress affects the albumen deposition because of poor feed efficiency and decreased concentration of

reproductive hormones and the shell membranes are deposited together as one membrane (Lucas and Ostagno, 2013).

In the shell gland, the shell is added to the shell membranes. Calcium is needed in high quantity for this process (Odum *et al.*, 1986). In a heat stressed hen, Voluntary feed intake is reduced, and calcium ingestion is also reduced. Vitamin D is denatured and the little amount of calcium in the gut is poorly absorbed in to the blood. Oestrogen has a complex relationship with calcium metabolism and has been shown to increase serum calcium and in heat stress, oestrogen level is decreased which leads to a decrease in serum calcium concentration (Mahmoud *et al.*, 1996; Franco-Jimenez and Beck, 2005). Prolonged panting causes excessive loss of carbonate ions (Marder and Arad, 1989). Carbonate ions are required to react with calcium to form calcium carbonate for egg shell formation. The decrease in calcium and carbonate ions results in the production of thin egg shell during heat stress (De Andrade *et al.*, 1976; NRC, 1981; Deaton *et al.*, 1982; Hillman *et al.*, 1985; May *et al.*, 1986; Teether and Smith, 1986; Mahmoud *et al.*, 1996; Yahav *et al.*, 1997; Yahav, 1999). Melatonin secreted during heat stress causes a decrease in transient time of egg within the oviduct during egg formation and this leads to incomplete deposition of nutrients, shell membranes and shell in the egg produced (Zulkifli and Siegel, 1995).

EGG QUALITY: Heat stress causes reduction in the number of eggs produced, egg weight, Haugh unit, albumen quality, yolk index, shell thickness and shell quality because of decreased availability of nutrients, minerals, vitamins, water deficit (in chickens deprived of enough drinking water) and serum concentrations of reproductive hormones (Mueller, 1961; Carnon and Houston, 1965; El Boushy and Abada, 1970; Smith and Oliver, 1972; Kechick and Skyes, 1974; De Andrade *et al.*, 1976; De Andrade *et al.*, 1977;

Wolfenson *et al.*, 1979; Gadzama, 1986; Dankoh, 1989; Ubosi and Yusuf, 1989; Mills *et al.*, 1999; Muiruri and Harrison, 1991; El Boushy *et al.*, 1988; Ubosi, 1998; Borges *et al.*, 2003; Sahin and Kucuk, 2003; Rozenboim *et al.*, 2007; Ebeid *et al.*, 2012).

Laying pattern and clutches

When hens are heat stressed, their laying pattern becomes haphazard due to decrease in serum concentration of reproductive hormones and a delay in ovulation. When there is a delay in between subsequent ovulations, the number of eggs laid per clutch will be reduced (Lucas and Ostagno, 2013).

Fertilisation

Allen and Grigg (1957) reported that spermatozoa die before they reach the uterovaginal junction (where spermatozoa are stored) in the hen during heat stress and some spermatozoa that reach the uterovaginal junction die while in storage (Allen and Grigg, 1957). The spermatozoa that reach the ampullae cannot penetrate the blastodisc due to lack of acrosome cap or presence of defective acrosome cap (Joshi *et al.*, 1980). Some spermatozoa can penetrate the blastodisc but cannot fertilize it due to altered DNA construction during spermatocytogenesis (McDaniel *et al.*, 2004).

Hatchability

Keirs (1982) and McDaniel *et al.* (2004) reported a decline in the number of fertilised eggs that hatch during heat stress. Incubating eggs were candled by McDaniel *et al.* (2004) and they observed that some eggs were not properly fertilised while some eggs revealed embryo mortality. Embryo's frequently become overheated during incubation in hot periods even when the incubator is operating correctly within a narrow range of temperature set points (Meijerhof and van Beek, 1993; Hulet and Meijerhof, 2001). Thus, high environmental

temperature causes decreased hatchability and chick quality.

MANAGEMENT OF HEAT STRESS

The environmental heat in the semi-arid region of Nigeria during the hot periods between March and May is unavoidable. However, taking steps to prevent heat stress in our poultry flock can help keep chickens comfortable and reproductively active during the hot weather. The management of heat stress in chickens is approached through conscious measures taken in poultry housing, feeding, water provision, administration of anti-stress agents, proper stocking density and selection of chickens with traits of superior heat tolerance.

Housing

Location

The poultry house should be located in an area that has a lot of trees and shade. The presence of trees will provide shade during hot weather and thus, reduce the environmental temperature around the poultry house. The house should not be located in a busy and noisy area because chickens need to be kept as calm as possible during heat stress.

Roofing

The materials for roofing should have a bright colour to reflect hot rays of the sun. The roof should be pitched and oriented in such a way that sun rays do not fall directly on it. The roofing material should be able to serve as an insulator to reduce heat transfer by radiation from the roof into the house. Ubosi (1981) observed that putting 'zana' mat as an insulator in the ceiling reduced the effects of heat stress.

Ventilation

The house should be well ventilated so that the hot and moist air from among birds inside the building can move out freely and fresh air flow in (Ubosi, 1981). Large

windows should be constructed on opposite sides of the building, but they should not be facing the direction of sun rise and sun set, so that sun rays do not fall directly on the birds through the large windows.

Walls and floor

The walls of the house should be built using materials that are poor heat conductors such as cement blocks, mud bricks and timber. The use of these materials will bring about a decrease in room temperature during high environmental temperatures. The floor should be made of materials such as sand, straw and sawdust that can insulate the chickens from floor heat. Kwari and Ubosi (1991) observed that construction of shallow water pools on the floor for chickens to step into reduced the effects of heat stress because the chickens stopped panting immediately they stepped into the water.

Stocking density

The stocking density of chickens during the hot weather should be reduced. Chickens spread out their wings to dissipate heat and a lot of space is needed to be able to do that. Radiant heat transfer is greater with high stocking rates and humid hot air gets trapped between them. When chickens are stocked in high populations, their body temperatures increase (Lowe and Heywang, 1964; Tower *et al.*, 1967). Egg production decrease when chickens are densely populated (Moore *et al.*, 1965; Owings *et al.*, 1967; Wilson *et al.*, 1967; Grover *et al.*, 1972; Davami *et al.*, 1985; Engster *et al.*, 1985).

Breed of chicken

The breeds of chickens to be kept in hot climates need to be considered to reduce mortality associated with heat stress due to heat intolerance. Chickens with the genes for heat tolerance should be stocked for hot periods because genetic variation in response to heat stress has been shown to exist among breeds (Fox, 1951, 1980;

Ahmed 1976, Nayak *et al.*, 2015). Chickens with the gene for expressing heat shock protein in large quantities should also be sought for. Exposure of chickens to heat induces heat shock protein (Hsp 70) production and on re-exposure to heat, the Hsp 70 released will be increased if the second exposure involves higher temperature for a longer duration (Arjona *et al.*, 1988, 1990, Lindquist and Craig, 1998). Cheng *et al.* (1990) reported that the naked neck chicken had less effects of heat stress when compared to the full feathered chicken. Feathers serve as insulators for chickens. The reduced feathering of chickens is essential for combating the effects of heat stress (Yahav *et al.*, 1998). Avigdor Cahaner from Israel's Hebrew University has cross-bred a small bare skinned bird with a regular broiler chicken to produce a naked chicken. The naked chicken does not suffer much heat stress because it cools off faster (Yahav, 1998).

Nutrition

Feed

The feed given to chickens during the hot weather should have increased calories because chickens eat less, and feed efficiency is reduced. Chickens should not be fed in the mid-morning and afternoon so that metabolic heat production associated with digestion does not add to the heat stress which is worst in the afternoon and so chickens should be fed in the late afternoon and throughout the night. The effect of heat stress in laying hens during the day is mostly on egg shell quality (which is more adverse) but the night heat stress caused a decrease in egg production (Wolfenson *et al.*, 1979). Ubosi and Nwaka (1998) reported that albumen index, specific gravity and shell thickness of egg improved significantly when heat stressed chickens were fed at night.

Feed supplementation

The effect of heat stress is reduced when chickens are given supplements such as vitamins in their feed (Ubosi and Chickweren, 1988). Vitamin A reduces the effects of heat stress but when vitamin A is given in excess it reduces the immune status of the chickens (Friedman *et al.*, 1991). Many researchers reported that ascorbic acid supplementation has beneficial effect on egg production, egg shell strength, shell thickness and interior egg quality in heat stressed hens (El Boushy and Abada, 1970; Dohr and Balloun, 1976; El Boushy *et al.*, 1988; McDowell, 1989; Orban *et al.*, 1993; Ubosi and Gandu 1995; Bains, 1996). Administration of ascorbic acid in feed of heat stressed cocks improves semen quality (McDaniel *et al.*, 2004). Ascorbic acid reduces the concentration of corticosterone in plasma when administered at 150 ppm but at 300 ppm, ascorbic acid did not have any effect on plasma concentration of corticosterone (McKee and Harrison, 1995). At 500 ppm and 1000 ppm, ascorbic acid proved to be detrimental to fertility in heat stressed cocks (Mc Daniel *et al.*, 2004). The ability of ascorbic acid to reduce the effects of heat stress on reproduction is dose limiting (Orban *et al.*, 1993). Vitamin E supplementation reduced the effects of heat stress in laying hens (Whitehead *et al.*, 1998; Bollengier-Lee *et al.*, 1998; Puthonsiriporn *et al.*, 2001). Vitamin E improved egg production, vitelline membrane strength, yolk and albumen quality (Kirunda *et al.*, 2001). Bollengier-Lee *et al.* (1998) reported that 50 mg vitamin E/kg increased egg production by 7% in heat stressed hens compared to hens given 10 mg vitamin E/kg. Administration of tetramycin Q increased feed efficiency in heat stressed chickens (Ubosi and Azubogu, 1989). Bottje and Harrison (1985) reported that addition of 1% sodium bicarbonate and 1% ammonium chloride to feed reduced blood pH in heat stressed chickens. Administration of dietary acetyl salicylic

acid in feed to heat stressed cocks did not have any effect in reducing infertility (McDaniel and Parker, 2004).

Water supplementation

Cool drinking water should be continuously provided for chickens. The use of Vitonic® (vitamins A, 3,000,000 I.U.; B₁₂, 1,000mg; C, 3500mg; pantothenic acid, 1,100mg; choline chloride, 30,000mg; riboflavin, 500mg; niacin, 2,000mg; K₃, 400mg; pyridoxine, 600mg; folic acid, 60mg and thiamine, 400mg/kg) anti-stress at 2.5g/L of drinking water improved egg weight, shell weight, shell thickness, specific gravity, albumen index and Haugh unit of eggs (Ubosi and Geidam, 1991-1992). Administration of carbonated water to heat stressed Broilers does not reduce blood pH

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levels (Bottje and Harrison, 1985). Electrolyte solutions (sodium, chloride, potassium and bicarbonate) in drinking water will replenish the electrolytes and correct the acid/base balance (Balnave *et al.*, 1989; Koelkebeek *et al.*, 1992, 1993; Smith, 1994; Balnave and Muheereza, 1997).

CONCLUSION

Environmental heat stress causes physiological changes in chickens which affect reproduction resulting to decreases in semen quality, number and quality of eggs produced and chicks hatched. Good management practices such as provision of proper feeding regimen, adequate drinking water, anti-stress supplements and well-ventilated houses in shaded areas will reduce the effects of heat stress.

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