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Influence of high environmental temperature on egg production and shell quality: a review

A.O. OGUNTUNJI* and O.M. ALABI

Department of Animal Science and Fisheries Management, Bowen University, PMB 284, Iwo, Osun State, Nigeria
***Corresponding author: abelmendel@yahoo.co.in**

In spite of the large amounts of money invested in research, breeding, and the improvement of commercial egg-type strains, high environmental temperature (HET) has been identified as a major non-genetic constraint limiting expression of their full genetic potential. This environmental stressor has been implicated in adverse marked effects on egg production and eggshell quality of hens. Reports have conclusively indicated that poor performance (*i.e.* drop in egg production and poor eggshell quality) of layers reared in thermally-stressed environments could be adduced to a complex interplay of low feed intake, malfunctioning of the endocrine system, acid-base imbalance and poor physiological functioning of organs and mechanisms connected with the entire egg production process, via follicular recruitment and growth, ovulation, egg formation, shell formation, egg development, oviposition and oviposition interval.

Keywords: complex interplay; endocrine system; egg-type poultry; feed intake; ovulation; strain; stressor; thermally-stressed; climates

Introduction

Poultry production, like any other enterprise is not immune to day-to-day constraints. Notable amongst these, especially in developing nations located in hot tropical environments, are managerial ability, feed availability, competition with foreign poultry products, harsh climates, disease and poor government policies - among others. However, high ambient temperature (HAT) has been identified as one of the major stressors in poultry production in the tropical environment (Kadim *et al.*, 2008) and most especially in under-developed countries where poultry farmers cannot afford expensive modern artificial control of ambient temperature in poultry houses (Deeb and Cahaner, 2001).

Chickens suffer under HAT because their feather coverage hinders internal heat dissipation, leading to elevated body temperature (Yahav *et al.*, 1996). Oguntunji *et*

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al. (2008a) concluded that elevated environmental temperature poses a threat to the general well-being of fowls and an increase in body temperature outside the thermo-neutral range, without a prompt compensatory heat loss, as a result of prolonged exposure of birds to HAT, results in heat stress and its attendant effects. The term 'stress' is commonly used to describe the detrimental effects of a variety of situations on the health and performance of poultry (Rosales, 1994). Heat stress results from a negative balance between the net amount of energy flowing from the animal to its surrounding environment and the amount of heat produced by the animal (Ayo *et al.*, 2006). Ugurlu *et al.* (2001) asserted that heat stress in layers emanates from HETs and induces some variations in behaviour and performance of layers.

Synthesis of empirical reports in the last decade demonstrated that thermal stresses exert adverse effects on the reproductive efficiency of such heat-stressed female poultry by reducing egg production and eggshell quality of birds reared either in natural or controlled environments (Campbell *et al.*, 2003; Mashaly *et al.*, 2004; Rozenboim *et al.*, 2004; Chauhan and Roy, 2007; Franco-Jimenez *et al.*, 2007; Rozenboim *et al.*, 2007; Yakubu *et al.*, 2007; Oguntunji *et al.*, 2008a).

This paper is not an exhaustive review of all publications and mechanisms involved with the topic under review. Rather, it is the aim of the authors to discuss some of the underlying mechanisms mediating the characteristic poor reproductive (egg production and eggshell quality) performance of heat-stressed birds.

The effect of physiological disturbance on egg production in a hot environment

In spite of huge financial resources committed to the breeding and development of commercial laying strains; the rearing environment still plays a significant role in the expression of their full genetic potential (Oguntunji *et al.*, 2008b). As an important non-genetic and bio-climatic factor, environmental temperature influences optimum physiological functioning of egg-type poultry. Therefore, a suitable rearing environment is fundamental to the optimum metabolic, physiological and endocrinological activities connected with the entire egg production process (Oguntunji *et al.*, 2008b).

Reduced egg production in hot environments has been suggested to be a result of the fact that non-layers have a higher body temperature than layers (Hillerman and Wilson, 1955). There is an evidence that in an environment of 90°F (32.2°C) and higher, the temperature-regulating mechanism of the bird cannot adequately control body temperature within the normal range and it therefore rises (Heywang, 1938; Wilson, 1949). This rise produces a 'fever' (hyperthermia) which upsets the normal physiological processes sufficient to cause a decline or cessation of egg production (Huston *et al.*, 1957).

The regulatory-mechanism responsible for impaired reproductive efficiency of hyperthermic hens has been suggested to be linked to impaired ovarian function, as demonstrated by a significant reduction in ovarian weight and the number of large follicles observed at days 6 and 15 in heat-stressed (42°C) White Leghorn hens compared to those in thermo-neutral environments (24 to 26°C) (Rozenboim *et al.*, 2007). Marsh and Dawson (1989) and Rozenboim *et al.* (2007) suggested a reduction in ovarian blood supply because of peripheral vasodilation as a possible underlying mechanism responsible for the characteristic reproductive failure of heat-stressed hens. Insufficient blood supply to the ovary in contrast to the increased supply to the outer skin

might be one of the emergency physiological responses that alleviate endogenous thermal load via vasodilatation of the skin, shank, comb and wattle.

The ovary plays a critical and ubiquitous role in the reproductive activity of female poultry. Follicles (small white follicles) produced by the ovary are the major sources of oestrogen, producing over 80% of the total ovarian oestrogen (Senior and Furr, 1975; Armstrong, 1984; Nitta *et al.*, 1991). Oestrogen from ovarian follicles is also responsible for the growth of the reproductive tract (Campbell *et al.*, 2003), while progesterone, a major steroid hormone is secreted by the granulose cells of large ovarian hierarchical follicles (F_1-F_3) of laying hens (Huang *et al.*, 1979; Barh *et al.*, 1983; Porter *et al.*, 1991).

The attendant hypo-function of ovaries and low egg production in response to HATs might be mediated through inhibitory activity of heat stress on the hypothalamo-pituitary-gonadal axis. Interference with normal ovarian function will consequently obstruct or depress secretion and circulation of ovarian steroids (progesterone, oestradiol) and gonadotropins (luteinizing hormone, follicle stimulating hormone) thereby impairing regulation of physiological mechanisms fundamental to steroidogenesis, follicular recruitment and growth and ovulation thus resulting in poor egg production. Rozenboim *et al.* (2007) buttressed this assertion that heat stress induced a decline in plasma gonadal steroids (progesterone, testosterone, oestradiol) and follicular expression of mRNA for steroidogenic enzymes. Oguntunji *et al.* (2008a) also corroborated that the characteristic low egg production of commercial egg layers in the dry season, noted for HAT, in a derived savanna environment might be attributed to an upset in the physiologic mechanism of the laying hen as a result of heat-induced stress resulting in 'distortion' of hormonal equilibrium associated with ovarian activities and hence reduced egg production.

Effect of low feed intake and HAT on hormonal balance of heat-stressed hens

Another possible cause of low egg production in laying hens raised in hot environments is the complex interaction of HAT, low feed intake and hormonal imbalance. Egg production in poultry involves a complex physiological mechanism that is modulated by synergetic action of various reproductive hormones. Certain hormones such as progesterone (P_4), follicle stimulating hormone (FSH), luteinizing hormone (LH) and oestrogens have significant impact on egg production in female poultry. Nevertheless, the characteristic low dietary intake of heat-stressed hens exerts significant effects on growth, age at puberty, rate of lay and endocrine profile. Evidences abound that insufficient nutritional intake stemming from heat stress or through controlled feeding influences reproductive performance of poultry through its effect on reproduction endocrinology (Etches *et al.*, 1984; Renema *et al.*, 1999; Liu *et al.*, 2004; Mashaly *et al.*, 2004; Elijah and Adedapo, 2006; Onagbesan *et al.*, 2006; Yakubu *et al.*, 2007; Song *et al.*, 2009).

Poor reproductive efficiency of under-fed hens may be connected with defective ovarian morphology and its malfunctioning compared to *ad libitum* fed counterparts. The deleterious effects of low feed intake on reproductive activities in broiler breeder hens was demonstrated with higher number of small follicle atresia, smaller ovarian weight, reduced number of medium white follicles, large white follicles, small yellow follicles and large yellow follicles (Renema *et al.*, 1999) which are the main sources of gonadal steroids modulating the egg production mechanism in female poultry. In a similar recent study, Song *et al.* (2009) reported cessation of egg production after the second day in fasted ducks. They also reported a regression of ovaries, significant

reduction in ovary weight, shrunken and regressed large yellow follicles even after three days of re-feeding whereas follicles were normal in control ducks.

The effect of feed-related stress on optimum hormonal functioning and egg production could be attributed to the fact that functional ovarian activity in the matured hen can be inhibited by low feed intake; the effect was ascribed to the failure of the pituitary gland either to produce or to release gonadotropins because the administration of these substances will cause follicular growth and ovulation (Campbell *et al.*, 2003). Feed restriction in egg-type hens is known to reduce LH concentrations (Tanabe *et al.*, 1981) which, in mammals, occurs through altered LH-releasing hormone secretion (Steiner, 1987; Cosgrove *et al.*, 1991). Recently, Song *et al.* (2009) reported that food deprivation of laying ducks significantly reduced plasma E_2 and depressed the expression of ovarian follicle-stimulating hormone receptor (FSHR) and luteinizing hormone receptor (LHR) mRNA which are two important reproductive hormone receptors. The decrease in E_2 levels has been implicated in termination of egg-laying and regression of the yellow follicles (Song *et al.*, 2009). Insufficient secretion or absence of E_2 in circulation may affect negative feedback on LH secretion and action. In similar vein, depression of ovarian FSHR and LHR mRNA was related to adverse effect of underfeeding on fasted ducks because FSHR mRNA might be involved in the differentiation and maturation of granulose cells in less mature follicles (Zhang *et al.*, 1997) and LHR mRNA might be related to the establishment of the follicles hierarchy and ovulation (Zhou *et al.*, 2003).

Furthermore, significant reduction in large yellow follicles, small white follicles and defective ovarian morphology, concomitant with low dietary intake, suggest a decline in secretion of reproductive hormones from those organs and denial of stimuli by the target organs to initiate and advance necessary physiological mechanisms controlling egg production. Follicles of various sizes (*i.e.* small yellow follicles, large white follicles and large yellow follicles) produced by the ovary are critical to endocrinological balance and robust reproductive activities of female poultry. The main sources of ovarian E_2 are small white follicles and ovarian stroma with numerous cortical follicles containing more than 50% of ovarian aromatase activity and producing more than 80% of ovarian oestrogen (Senior and Furr, 1975; Armstrong, 1984; Nitta *et al.*, 1991).

Since ovary and follicles are critical to gonadal hormone secretion and egg production it is instructive that manifestation of nutritional stress on ovarian morphology and functions in various forms will consequently impair secretion and lower circulating plasma levels of such hormones, resulting in sub-optimal and uncoordinated physiological mechanisms involved with follicular growth, egg development, oviposition and oviposition interval and hence low egg production of under-fed hens.

Furthermore, studies had revealed adverse relationship between HATs, plasma reproductive hormonal levels and potency of reproductive hormones regulating egg production mechanisms in female poultry. Attempts by egg-type poultry to offset the physiological stress induced by HATs is accompanied by alteration and disruption of hormonal equilibrium of laying hens thereby resulting in inefficient and impairment of the entire mechanism involved hence poor egg production of heat-stressed hens. The faulty mechanisms underlying the low laying rate of hyperthermic hens as a result of a 'distorted' endocrine profile of reproductive hormones is multifaceted in origin. One of the mechanisms might be linked to adrenal-ovarian axis involving corticosterone and prolactin (PrL). It has been suggested that there is an interaction between the adrenal glands and ovarian function in hen (Etches *et al.*, 1984). Rivier and Rivest (1991) summarized into three, the possible routes by which stress-related hormones exert their antagonistic effect on reproductive function at all levels of hypothalamus-pituitary-gonadal axis: the brain to inhibit GnRH secretion, the pituitary to interfere

with GnRH-induced LH release and gonads to alter the stimulatory effect of gonadotropins on sex steroid secretion.

Studies directed to unveil relationship between corticosterone, elevated ambient temperature and egg production revealed higher plasma concentration of corticosterone at HATs (Siegel and Gould, 1982; Pardue *et al.*, 1985; Donoghue *et al.*, 1989) with a corresponding drop in lay. In addition, exogenous introduction of corticosterone (30ug/ hour) in laying hens was observed to induce cessation of egg production besides severe ovarian regression, significant regression in total ovarian stroma weight, higher number of atretic follicles and decreased number of large yolk-filled follicles in contrast to the control birds (Etches *et al.*, 1984).

Similarly, elevated plasma concentrations of PRL in response to heat stress (El Halawani *et al.*, 1984: Rozenboim *et al.*, 2004) and seasonal photoperiodic stimulation (Shi *et al.*, 2007; Huang *et al.*, 2008) have been linked with poor reproductive performance in turkey and geese respectively via its antagonistic activity against hormones modulating egg production. Furthermore, the antagonistic relationship between PrL and reproductive activity was further elucidated by blocking of PrL secretion with bromocriptin (so inducing a decrease in prolactin concentrations) which enhanced higher egg production, less pause days, elevated serum LH (with regular interval and duration of LH surges), estradiol and P_4 concentrations required for egg formation and oviposition (Reddy *et al.*, 2007).

The inhibitory effect of heat stress on hypothalamo-pituitary-gonadal hormones secretion and regression of the reproductive system might be due to the combined effect of direct inhibition of GnRH secretion by PrL and corticosterone and lack of sensitivity at the site of action of the target organs. Reduced reproductive activities have been shown to be mediated by high levels of PrL which directly inhibit hypothalamic secretion of GnRH, which in turn reduces pituitary secretion of LH and leads to regression of gonads (Sharp 1989; Curlewis,1992; El Halawani and Rozenboim 1993). In addition, low plasma concentrations of LH, P_4 and oestradiol were observed alongside elevated cortiocosterone level in laying hens subjected to heat stress (Donoghue *et al.*, 1989) or infused with corticosterone (Etches *et al.*, 1984).

Hypothalamic hypo-function has been suggested as a possible mediator of the reproductive failure during thermal stress (Donoghue *et al.*, 1989). These authors hypothesised that there is a possibility that the avian brain may be adversely affected by elevated levels of circulating corticosterone during thermal stress, resulting in reduced hypothalamic function, and hence reduced LH releasing ability of the hypothalamic and circulating levels of LH in laying hens. Part of the inhibitory effects of PrL on reproductive activity is also mediated at pituitary level. Scanes *et al.* (1977) observed that plasma concentrations of PrL were depressed immediately before and during the pre-ovulatory LH peak and suggested that the decline might facilitate the secretion of ovarian steroids known to have positive feedback action on LH release. It is worth emphasis that LH plays significant role in regulation of physiological mechanism involved with ovulation of matured follicles. Attendant low concentration of this vital reproductive hormone as a result of higher concentrations of PrL and corticosterone is indicative of suppressed reproductive activity of this pituitary hormone. During the ovulatory cycle, ovulation is induced by a pre-ovulatory release of LH accompanied by a pre-ovulatory release of P_4 from the largest follicle (Huang *et al.*, 1979; Barh *et al.*, 1983; Porter *et al.*, 1991). F_1 is the major source of P_4 that feeds back for the ovulation, inducing LH, the delay in maturation of follicles will result in extended intervals among LH surges, decreased ovulation rates and egg production. (Onagbesan *et al.*, 2006).

Furthermore, the influential role of LH on egg production was further elucidated by Liu *et al.* (2002; 2004) that the intervals between the LH surges are longer in broiler breeder

and turkey hens with low egg production rates. In similar vein, the depressed laying rate observed during the non-breeding season was found to parallel low LH secretion and circulation in geese (Shi *et al.*, 2007; Huang *et al.*, 2008). Poor laying rate in connection with low LH surges has been linked with delayed follicle maturation and ovulation, inducing P_4 (Onagbesan *et al.*, 2006). Donoghue *et al.* (1989) supported the findings that reproductive decline in heat-stressed hens is mediated by reduced LH releasing ability of hypothalamus.

Furthermore, reproductive quiescence, associated with activity of PrL in laying hens, has been shown to inhibit oestrogen production at the ovarian level and gonadotropin stimulated ovulation, thus regulating egg production (Reddy *et al.*, 2007). Similar low plasma oestradiol concentrations have been reported alongside elevated plasma corticosterone levels (Donoghue *et al.*, 1989). Reduced circulating oestradiol, as a result of increased serum corticosterone and PrL levels, could be adduced to the reduced number of small white follicles - the main source of oestradiol. This is indicative of impairment of all egg production mechanisms mediated directly or synergetically by oestrogen and also reduction in P_4 and LH secretions and actions. It has been shown that declined plasma levels of E_2 might result in termination of egg-laying and the regression of yellow follicles (Song *et al.*, 2009) needed for P_4 secretion. For ovulation to occur, a follicle must be sufficiently mature to produce enough P_4 which acts as feed-forward control to induce LH secretion and to stimulate the release of LH in the open period (Onagbesan *et al.*, 2006). It could be deduced that reduced number of SWF associated with elevated corticosterone and PrL prevents follicular recruitment and maturation which normally precede entry into the hierarchy of large yolky follicles via low plasma E_2, thus ovulation.

It is apparent that interplay of insufficient nutritional intake via low feed consumption and HATs alters the endocrine profile and impairs endocrinological activities involved with avian egg formation and production. This is indicative that any deviation from the optimum hormonal balance of laying hens in response to low feed intake and HAT exerts an adverse effect on reproductive mechanisms, resulting in malfunctioning of organs and mechanisms concerned with egg production. Donoghue *et al.* (1989) and Novero *et al.* (1991) reinforced this assertion that heat stress disruption of egg production of laying hen could be attributed to synergetic effect of low feed intake and hormones responsible for ovulation and reduced responsiveness of granulose cells to luitenizing hormone.

Feed intake and eggshell quality in hot environments

Eggshell formation is a complex phenomenon and is influenced by heredity, nutrition, hormone, environment, pathology and management. Producing good quality eggshell is feasible only when hens are raised in ideal conditions and all necessary factors are present and functioning in harmony (Dhawale, 2008).

The characteristic poor eggshell quality of fowls subjected to thermal stress can be partially accounted for by low feed intake. The ability of hens to produce good quality shell depends largely on the availability of calcium (Ca) from the ingested food and skeletal reserves (Farmer *et al.*, 1983). Washburn (1982) corroborated that Ca is the most important dietary factor affecting shell quality. The significance of calcium requirements in layers can be determined by the fact that the eggshell of hens contains 94.4% calcium carbonate (Romanoff and Romanoff, 1949). Reports from various studies indicate that egg shell qualities are compromised during HATs as a result of a decrease in feed intake. Elijah and Adedapo (2006) attributed small egg size, low egg production and incidence of cracks to the inability of hens to take adequate feed necessary to manufacture egg shell

at HAT. Reduced feed intake has also been reported to limit blood Ca availability for egg shell formation (Robert, 2004), and that decrease in shell strength and thickness of thermally-stressed hens could be attributed to reduction in free ionized Ca in the blood (Odom *et al.*, 1986). Furthermore, low dietary intake of Ca due to heat stress induces bone resorption and hyperphosphatemia. Hyperphosphatemia (increased phosphorus in the blood) inhibits the formation of $CaCO_3$ in the uterine glands of layers due to diversion of more blood to the skin for cooling, thereby reducing blood flow to the uterus and Ca supply to the uterus (Chauhan and Roy, 2007), resulting in eggs with poor shell quality. Since preponderance of Ca availability for egg shell is through feed intake, it is highly suggestive that any factor (such as HAT) that influences low feed intake will simultaneously effect reduction in dietary Ca intake, mobilization and availability, and contribute directly to poor shell quality.

Influence of acid-base imbalance on egg shell quality in hot environment

Furthermore, poultry birds employ various strategies such as behavioural, hormonal, physiological, and biochemical adjustments to maintain homeostasis at HATs. Among behavioural adjustments observed in hyperthermic birds are flapping of the wings and increased panting. Panting helps in cooling body temperature at HATs through evaporative cooling but simultaneously contributes to the alteration of acid-base balance. Acid-base balance is an important factor in formation of eggs with good shell quality.

Birds rely primarily on respiratory evaporative cooling to maintain thermal homeostasis when exposed to HATs (Calder and Schmidt-Neilsen, 1996). Attempts by thermally-stressed birds to maintain homeostasis resulted in increased ventilation (evaporative cooling) which produces an acid-base disturbance (respiratory alkalosis). Respiratory alkalosis is a state due to excess loss of CO_2 from the body (Borges *et al.*, 2004). Metabolic alkalosis is a disturbance in which acid-base status of the body shifts toward the alkaline side because of changes in the fixed (non-volatile) acid and bases (Dorland, 1965). This is characterised by the alteration of plasma electrolytes and blood gases wielding influence on formation of eggs with good shell quality.

Certain macrominerals, such as sodium, potassium and chloride, are crucial in acid-base homeostasis (Olanrewaju *et al.,* 2006). In a recent study, Mujahid *et al.* (2009) reported significantly ($p<0.05$) lower plasma concentrations of potassium (after 6 and 12 hours exposure to heat stress) and sodium (after 6 hours exposure to heat stress) but marginal decrease in plasma calcium level after 6, 12 and 18 hours of subjecting broilers to heat stress (34°C). They also observed marked ($p<0.05$) reduction in blood pCO_2 and HCO^-_3 after 6, 12 and 18 hours of thermal stress in contrast to their counterparts raised in control temperature (25°C). In a related study, Franco-Jimenez *et al.* (2007) studied the relationship between blood HCO^-_3 with shell quality. Their results revealed the depressing effect of HAT on blood carbonate and shell quality and inverse relationship between HCO^-_3 and shell quality in the egg-strains compared.

Among the suggested possible underlying mechanisms put forward that may be responsible for modulating poor shell formation in thermally-stressed hens, one is that HATs may reduce the activity of carbonic anhydrase enzyme which results in the formation of bicarbonate which contributes carbonate to the egg shell (Balnave *et al.*, 1989) hence formation of eggs with poor shell quality by hyperthermic hens. Furthermore, insufficient circulating Ca level has been linked with production of eggs with poor shell quality in heat-stressed hens. Poor shell formation as a result of low

plasma Ca at HATs could be linked to reduced blood flow to the ovary (Rozenboim *et al.*, 2007) and other internal organs such as uterus of chicken hens under thermal stress. It is apparent that insufficient blood flow will deny ovary the needed blood Ca, oxygen and nutrients for optimum physiological functioning of this vital organ with attendant effects on shell formation and quality. In addition, it has been reported that the eggshell-forming enzyme present in uterus is less active as a result of heat stress (Chauhan and Roy, 2007). Smith (2001) stated that any decline in shell thickness, which occurs above the panting threshold, is almost certainly due to the reduced ability of blood to carry Ca, a phenomenon associated with alkalosis resulting from panting. Mahmoud *et al.* (1996) suggested that alteration in acid-base balance, the status of Ca, and diminished ability of duodenal cells to transport Ca could be critical factors in the detrimental effects of heat stress on egg production, egg shell characteristics and skeletal integrity often documented in laying hens. Campbell *et al.* (2003) substantiated these reports further, reporting that chickens cool themselves by panting and this in turn changes the chemical composition of their blood. Carbonate is lost during panting and, as a result, less Ca is available for deposition in egg shells of heat-stressed hens.

Conclusions

It is evident in this review that HAT is a major non-genetic factor contributing to low rates of lay and poor eggshell quality of chicken hens reared in hot environments. Total control of this problem is difficult and the costs involved are not affordable by poultry farmers in warm and hot climates which are mostly affected. It is therefore necessary to evolve low-cost measures that will minimise the negative aspects of HAT to the minimum via dietary adjustments, husbandry, management practices and or application of genetic approach by incorporation of heat resistant genes such as naked neck (Na), Frizzle (F) and scaleless (sc) which have been demonstrated to enhance thermoregulatory ability of chicken in hot environments.

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