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Effect of vitamin E in heat-stressed poultry

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Heat stress is associated with compromised performance and productivity in poultry due to declines in feed intake, nutrient utilisation, growth rate, egg production and quality, feed efficiency and immunity. Additionally, heat stress is characterised by reduced antioxidant status in birds, resulting in increased oxidative stress. Heat stress is also linked with increased economic losses due to mortality of birds. Vitamin E is a major chain-breaking antioxidant in biological systems. An optimum response with supplementation of vitamin E in feed has been found to improve feed intake, body weight gain, feed efficiency, egg production and quality, nutrient digestibility, immune response and antioxidant status in poultry birds. This work compiles past and present information about the role of vitamin E in heat-stressed poultry.

Keywords: heat stress; productivity; vitamin E; poultry

Introduction

Poultry production has been reported to be the fastest growing livestock industry, particularly in tropical and sub-tropical regions of the world (Daghir *et al.*, 2009; Ajakaiye *et al.*, 2010). Heat stress is the major problem encountered in these regions, adversely affecting feed intake, body weight, growth and carcass characteristics (Sahin and Kucuk, 2001a; 2001b). Heat stress is also the principal cause of mortality and economic losses in tropical environments. St-Pierre *et al.* (2003) stated that total annual economic losses to the livestock industry due to heat stress were between $1.69 and $ 2.36 billion. Of these losses, $128 to $165 million was in the poultry sector.

The term 'heat stress' has many connotations which vary from region to region in the world. In broad terms, if a bird is acclimatised to low temperatures, temperature stress

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appears when the ambient temperature begins to rise above 25°C. However, in many countries of the world, temperatures below 32°C are not oppressive and much higher temperatures must therefore be considered as heat stress (Balnave, 2004). As a general term, stress is used to describe the sum of non-specific responses or defence mechanisms of the body when confronted with abnormal or extreme demands (Sahin *et al.*, 2009). Animals have known zones of thermal comfort that vary by species, physiological status and the environmental conditions of air, temperature and solar radiation (NRC, 1981). For birds, the thermoneutral (TN) zone ranges between 18 and 22°C. Within this zone, animals are expected to be comfortable and experience no problems in physical temperature regulation (NRC, 1981; Ensminger *et al.*, 1990; Sahin *et al.*, 2009). Animals are considered to be heat stressed, when the temperature of the animal's body exceeds the upper critical level of the TN zone.

Birds usually have the ability to adapt themselves to their new environment. However, this requires redistribution of body reserves of energy and proteins at the cost of decreased growth and reproductive efficiency (Puron *et al.*, 1994). During the initial phase of heat stress, postganglionic neurons and the medullary tissue of the adrenal glands release catecholamine. Catecholamine prepares the birds for 'fight and flight', triggering the rapid release of glucose. Finally, if the bird does not recover from the stress and the adaptability of the bird is not adequate, a third or exhaustive phase leads to fatigue of the homeostatic mechanisms and subsequently death of the bird (Puron *et al.*, 1994).

High ambient temperatures have a detrimental effect on growth in broilers (Smith, 1974; Yalcin *et al.*, 2001; Sahin *et al.*, 2009). Reductions in egg weight and egg quality in laying hens, and depressed growth in rapidly growing broilers are very common symptoms of heat stress (Emery *et al.*, 1984; Sandercock *et al.*, 2001; Wolfenson *et al.*, 2001; Sahin *et al.*, 2006). Several studies have shown that high ambient temperatures may adversely influence the immune system of poultry, decrease mineral (*e.g.*, Fe. Zn and Cr) and vitamin (*e.g.*, vitamin C, A, and E) levels in the blood and increase their excretion from the body (McDowell, 1989; Donker *et al.*, 1990; Siegel, 1995; Klasing, 1998; Sahin *et al.*, 2009).

Postulated strategies to alleviate heat stress

Several methods are currently available to alleviate the detrimental effects of heat stress. Environmental strategies include keeping birds in open-sided cages, increasing ventilation, intermittent light schedules, early heating conditioning, early feed restriction, and lowering stocking density (NRC, 1981; Siegel, 1995; Lin *et al.*, 2006). Selection of genetically heat tolerant strains has also been advocated (Lin *et al.*, 2006). Decreasing protein concentration with concomitant improvements in amino acid balance has also been recommended to alleviate the negative effects of heat stress (Sahin *et al.*, 2009). Gous and Morris (2005) reviewed six broad categories of nutritional approaches to alleviate heat stress. Four involved potential changes to the heat increment of the food and two considered changes in acid-base balance and the use of additives. These authors concluded that the latter two approaches had the most potential for alleviating the effects of heat stress. Supplementation with electrolytes in cold drinking water favourably improves weight gain in broilers and eggs shell quality in laying hens under heat stress (Ait-Boulahsen *et al.*, 1995; Balnave and Muheereza, 1997; Hayat *et al.*, 1999; Lin *et al.*, 2006). The use of other nutrients such as pro-biotics, trace elements and vitamins have been proven beneficial in alleviating the adverse effects of heat stress (Lin *et al.*, 2006; Sahin *et al.*, 2009). The objective of this article is to review

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studies dealing with the effect of vitamin E supplementation to heat-stressed poultry with respect to its role in metabolism, performance and productivity, antioxidant status and immune-modulating effects. The postulated mechanisms of action of vitamin E in preventing and alleviating heat stress are also discussed.

Vitamin E

Vitamin E is used to denote two classes of compounds: tocopherols and tocotrienols. After ingestion, Vitamin E is hydrolysed and is absorbed through the intestinal epithelium in its un-esterified form. It is readily incorporated into the cellular membrane, where it promotes the integrity of the cell membrane. According to NRC (1994), the recommended dose of vitamin E for normal performance of poultry ranges from 5 to 25 IU/kg of feed. However, at higher doses, vitamin E has been found to improve the performance of poultry. Stress is the major modifier of the action of vitamin E. Nutritional imbalances, heat and cold, overcrowding, noise and transportation may predispose birds to infectious diseases by lowering defence mechanisms. A stress-induced infection may cause diarrhoea and decrease feed utilisation, thus further leading to increase requirements for this vitamin.

There are many reasons for supplementing vitamin E in the diet. Vitamin E is included in animal feed to improve performance, strengthen immunological status and increase the vitamin E content of animal meat (McDowell, 1989). In this respect vitamin E is used in poultry feed on the basis that vitamin E synthesis is impaired during heat stress (Naziroglu *et al.*, 2000; Sahin and Kucuk, 2001a; 2001b). It was suggested that heat stress increases lipid peroxidation in poultry (Naziroglu *et al.*, 2000), although it has been reported that vitamin E protects the liver from lipid peroxidation and cell membrane damage (Whitehead *et al.*, 1998; Sahin and Kucuk, 2001a; 2001b). Vitamin E is a major chain-breaking antioxidant and an important lipid component of biological membranes (Halliwell and Gutteridge, 1989; Sahin and Kucuk, 2001a; 2001b). It is mainly found in the hydrocarbon part of the membrane lipid bi-layer towards the membrane interface and in close proximity to oxidase enzymes, which initiate the production of free radicals (McDowell, 1989; Sahin and Kucuk, 2001a; 2001b). Vitamin E therefore protects cells and tissues from oxidative damage induced by free radicals (Sahin and Kucuk, 2001a; 2001b).

Nutrient digestibility and physical growth

Heat stress not only reduces feed intake but also impairs the digestibility of the diet (Wallis and Balnave, 1984; Bonnet *et al.*, 1997; Sahin and Kucuk, 2003; Mashaly *et al.*, 2004; Attia *et al.*, 2009). Wallis and Balnave (1984) found that the digestibility of amino acids decreased in broilers exposed to high environmental temperatures. Zuprizal *et al.* (1993) demonstrated that the true digestibility of protein and amino acids decreased as the temperature increased from 21°C to 32°C. Bonnet *et al.* (1997) reported that exposure to heat stress led to a decreased digestibility of protein, fat and starch. According to Hai *et al.* (2000) the expelling of digesta from the crop or small intestine was suppressed by high temperatures (32°C) and the activities of three enzymes (trypsin, chymotrypsin and amylase) were decreased at this temperature. Hai *et al.* (2000) also reported that the amount of chyme in the whole digestive tract was decreased by the cold environment (5° C) and increased by hot temperatures (32°C).

Environmental temperature is the most important variable affecting feed intake and

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weight gain in birds. Many studies have demonstrated that growth rates and feed intakes decreased when environmental temperature increased (Hurwitz *et al.*, 1983; Hai *et al.*, 2000; Ciftci *et al.*, 2005). Niu *et al.* (2009) suggested that for every 10°C increase in ambient temperature above 20°C, there is a 17% reduction in feed intake. Hurwitz *et al.* (1980) reported that appetite decreased as a primary response to high temperatures. At high temperatures, thyroid activity is arrested, which in turn affects metabolic rate, oxygen consumption, feed intake and growth rate (Mckee *et al.*, 1997). Decreased body weight is attributed to reductions in feed intake as a result of high ambient temperature. During heat stress, birds divert metabolisable energy from production to performance in the maintenance of homeothermy (Mckee *et al.*, 1997). Heat-exposed birds decrease their feed consumption to reduce their thermogenic effect associated with nutrient absorption, assimilation and utilisation. In addition, reduced blood flow to the gastrointestinal tract (GIT) during heat-induced peripheral vasodilatation may impair nutrient absorption (Mckee *et al.*, 1997). Decreased feed intakes and reduced blood flow to the GIT may exacerbate metabolic energy input. Subsequently these factors lead to depressed performance characteristics in poultry exposed to heat stress (Mckee *et al.*, 1997).

Sahin and Kucuk (2001a; 2001b) found that the digestibility of dry matter (DM), organic matter (OM), crude protein (CP) and ether extract (EE) was higher in vitamin E (250 mg/kg feed) supplemented Japanese quails. Sahin *et al.* (2002) reported that doses of 250 or 500 mg/kg increased the digestibility of DM, OM, CP and EE in Japanese quails reared under conditions of heat stress (34°C). Sahin and Kucuk (2001a; 2001b) reported that 250 mg of vitamin E per kg of feed resulted in higher feed intakes, body weight change and feed efficiency compared to 125 mg/kg of vitamin E in Japanese quails. In the same experiment, the interaction between vitamin E and selenium also resulted in higher feed utilisation, higher body weight and better feed efficiency suggesting the synergistic effect of this vitamin with selenium. Sahin *et al.* (2006) found that laying hens reared at 34°C consumed less feed and had better body weight, feed efficiency and carcass quality when fed 250 mg/kg of vitamin E. Sahin *et al.* (2002) reported that with 250 or 500 mg/kg additional vitamin E, body weight, feed intake and feed efficiency improved in Japanese quails reared under conditions of heat stress (34°C). Ciftci *et al.* (2005) found that a combination of vitamin E (125 mg/kg) and vitamin C (200 mg/kg) improved feed efficiency and body weight gain although feed intakes did not differ compared to the negative control.

Egg production and egg quality

It is well known that egg production decreases when ambient temperature increases above the TN zone. Mashaly *et al.* (2004) concluded that egg production is inversely related to high temperature. Whitehead *et al.* (1998) and Kirunda *et al.* (2001) reported that egg production in White Leghorns decreased when they were exposed to high environmental temperatures. This decrease in egg production has been ascribed to decreased feed consumption, reducing the availability of essential nutrients for egg production (Mashaly *et al.*, 2004). Exposure to high heat levels decreased plasma protein concentration and plasma calcium (Ca), both of which are required for egg formation (Mashaly *et al.*, 2004). Additionally heat stress increases the conversion of norepinephrine to epinephrine, which induces the degradation of ovarian follicles (Ciftci *et al.*, 2005). High ambient temperatures also impair the synthesis and release of vitellogenine which is necessary for yolk formation (Ciftci *et al.*, 2005). Yardibi and Türkay (2008) stated that vitamin E increases egg productivity by preventing liver

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damage, which is important for egg yolk protein synthesis. Kirunda *et al.* (2001) found in laying hens that supplementation of vitamin E (60 IU/kg) improved feed intake when birds were exposed to conditions of heat stress (34°C).

It has been shown that the negative effects of heat stress can be neutralised via dietary vitamin E supplementation (Puthpongsiriporn *et al.*, 2001). Sahin *et al.* (2002) reported that supplementation of vitamin E at the level of 250 or 500 mg/kg of feed increased egg production, egg weight, egg specific gravity, egg shell thickness and Haugh unit in Japanese quails reared under conditions of heat stress (34°C). Bollengier-Lee *et al.* (1998) reported that supplementation of 125 and 500 mg/kg of vitamin E increased egg production by an average of 7% and 4% respectively in hens heat stressed for four weeks compared to birds given 10 mg/kg of vitamin E. Ciftci *et al.* (2005) reported that vitamin E given at the rate of 200 mg/kg improved egg production, egg weight and egg yolk in White Leghorn birds reared under chronic heat stress. Yardibi and Türkay (2008) reported that Haugh unit, egg shell thickness, egg weight and egg specific gravity improved after supplementation of vitamin E (50 mg/kg) in heat stressed layers (35°C). Puthpongsiriporn *et al.* (2001) reported that egg mass, egg yolk and Haugh unit were greater with supplementation of vitamin E (65 IU) during heat stress. Asli *et al.* (2007) showed that 200 mg of vitamin E in the basal diet improved yolk percentage in laying hens exposed to heat stress (33°C). Kirunda *et al.* (2001) found in laying hens that supplementation of vitamin E (60 IU/kg) improved egg production, yolk and albumin solids, foam stability and angel cake volume during heat exposure.

Antioxidant effects

ROS induce oxidative damage to macromolecules, cells and tissues that consequently leads to increased morbidity and mortality rates and subsequently substantial economical losses (St-Pierre *et al.*, 2003). Environmental stress has been associated with elevated lipid peroxidation in serum and the liver and decreased concentrations of antioxidant vitamins in poultry (Sahin and Kucuk, 2003; Gursu *et al.*, 2004; Sahin *et al.*, 2005). Stress leads to the generation of free radicals which can damage cell membranes by inducing lipid peroxidation of polyunsaturated fatty acids in the cell membrane, destroying membrane integrity. Vitamin E scavenges ROS by breaking chain propagation and thus terminates oxidants at an early stage (McDowell, 1989). According to Sahin *et al.* (2006) liver malondialdehyde (MDA), an indicator of lipid peroxidation, significantly decreased in response of 250 mg/kg dietary vitamin E in Japanese quails reared at 36°C. In chickens, Morrissey *et al.* (1997) reported that dietary supplementation of vitamin E increased tissue α-tocopherol and decreased MDA concentration. Likewise, Sahin *et al.* (2002) found that broilers supplemented with dietary vitamin E had a significant reduction in MDA in serum and tissue. Puthpongsiriporn *et al.* (2001) reported that levels of MDA continued to rise in hens exposed to heat stress (32°C) and that laying hens fed rations containing the highest dose of vitamin E (65 IU/kg) had the lowest plasma MDA values. Yardibi and Türkay (2008) reported that vitamin E at the level of 50 mg/kg of feed significantly improved plasma MDA, erythrocytes MDA, erythrocytes GSH-Px, erythrocytes CAT and SOD enzymes in laying hens exposed to heat stress (35°C). Rashidi *et al.* (2010) also reported improved levels of MDA when vitamin E was supplemented at a level of 100 IU in feed of broiler chicks exposed to heat stress (32°C).

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Immunity

Numerous studies have demonstrated the effect of heat stress on immune parameters in chickens. Kadymov and Aleskerov (1988) concluded that high temperatures inhibit the synthesis of T and B lymphocytes and suppress the phagocytic activity of blood leukocytes. Mashaly *et al.* (2004) reported a decline in white blood cells (WBC) after exposure to heat stress and decreased heterophil to lymphocyte (H/L) ratios in White Leghorn hens. The H/L ratio has been used as a reliable indicator of stress in birds (Gross and Siegel, 1983; Mashaly *et al.*, 2004). Mashaly *et al.* (2004) reported that humoral immunity of birds from heat stress is depressed. Zulkifli *et al.* (2000) showed that heat stress caused a reduction in antibody synthesis. This reduction has been ascribed to an increase in inflammatory cytokines under stress, which stimulates the hypothalamic production of corticotropin releasing factor (Sapolsky *et al.*, 1987; Mashaly *et al.*, 2004). Furthermore, heat stress has been known to depress T-helper cytokines, which are important for antibody production (Lebman and Coffman, 1988; Mashaly *et al.*, 2004).

The antioxidant properties of vitamin E have been considered to have a role in the development of immune responses in chickens. Vitamin E has been reported to protect cells involved in immune responses such as lymphocytes, macrophages and plasma cells against oxidative damage and to enhance the function and proliferation of these cells (Puthpongsiriporn *et al.*, 2001). Niu *et al.* (2009) found that broilers receiving 200 mg/kg of vitamin E had significantly higher macrophage activities and more IgM and IgG antibodies under heat stress (38°C) than those receiving no supplementation. Rashidi *et al.* (2010) reported significantly increased T lymphocytes, phagocytosis, SRBC antibody and weight of spleen and bursa but reduced H/L ratios, when vitamin E (100 IU) was added to the feed of broiler chicks exposed to conditions of heat stress (32°C). Asli *et al.* (2007) reported that 200 mg of vitamin E in the basal diet improved antibody titre against SRBC in laying hens exposed to heat stress (33°C).

Conclusions

In conclusion, heat stress causes significant reductions in performance, productivity, nutrient digestibility, immune status and antioxidant profiles. These implications result in increased disease incidence and economic losses in poultry operations. Because of its significant role in poultry production and performance, vitamin E is one of the most important nutrients to consider when formulating poultry feed for animals exposed to heat stress.

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