◀Review▶

Nutritional Strategies to Maintain Efficiency and Production of Chickens under High Environmental Temperature

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Climate model projections indicate that the global surface temperature will probably rise a further 1.1 to 6.4°C during the twenty-first century, which is expected to have a significant impact on the efficiency, production, morbidity and mortality of domestic animals. Presently, high environmental temperature exposure is of major concern for the poultry industry especially in hot regions of the world because of the resulting poor performance, immuno-suppression and high mortality. There are different nutritional strategies for decreasing heat production to alleviate stress in high temperature-exposed chickens. The nutritional strategies are designed after considering factors such as the type of birds, age of birds, stage of production, duration of heat exposure, intensity of heat exposure and health of the birds. The nutritionists can base their strategy on less heat-production, increased nutrient intake, decreased energy wastage, and reduction in heat-induced oxidative stress and damage in birds to overcome the deleterious effects of high temperature on metabolism, physiology, feed efficiency, production performance and health. This can be accomplished by nutritional strategies to reduce heat stress by feeding good quality feed with high digestibility and nutrient density, adding fat as an energy source, balancing and provision of additional amino acids, and supplementing with vitamins, minerals and glucose. Recently, new concepts have been proposed for nutritional strategies that focus on the reactive oxygen species (ROS) production by the birds and decrease the oxidative stress and damage caused by exposure to high environmental temperature. None of these strategies are effective alone in terms of growth, feed efficiency, livability, product quality, stress tolerance or immune response, however, a combination of the nutritional strategies may help to alleviate the deleterious effects of heat stress and maintain the efficiency and production of chickens under high environmental temperature.

Key words: Heat stress, nutrition, oxidative damage, oxidative stress, UCP

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Introduction

During the last century, the global surface temperature increased $0.74 \pm 0.18^\circ\text{C}$, and climate model projections summarized in the IPCC report indicated that the global surface temperature will probably rise a further 1.1 to 6.4°C during the twenty-first century (IPCC, 2007). There is growing evidence that climate-health relationships pose increasing health risks for humans under future projections of climate change and the warming trend over recent decades has already contributed to increased morbidity and mortality in many regions of the world (Patz *et al.*, 2005). The same is true for birds and animals, where significant impact on efficiency, production, morbidity and mortality can be expected with the rise in global average temperature.

High temperature exposure is a major concern for the

poultry industry especially in hot regions of the world because of the resulting poor growth performance, immunosuppression and high mortality (Bottje and Harrison, 1985; Young, 1990; Mujahid *et al.*, 2005, 2009b). The continuous selection for fast growth has been associated with increased susceptibility of birds to high temperature (Geraert *et al.*, 1993; Cahaner *et al.*, 1995; Berong and Washburn, 1998). Exposure of chickens to high temperature causes significant changes in physiological responses (Harrison and Biellier, 1969; Altan *et al.*, 2003; Toyomizu *et al.*, 2005; Mujahid *et al.*, 2009b). Furthermore, thermal stress exerts deleterious effects on feed intake and body weight gain (Geraert *et al.*, 1996a) as well as on carcass yield, carcass protein, muscle calorie content and mortality rates (Smith, 1993; Tankson *et al.*, 2001). Exposure to high temperature induce elevation of rectal temperature, antioxidant enzyme activity, and formation of malondialdehyde (MDA) and protein carbonyls, as well as the dysfunction of mitochondrial respiration (Mujahid *et al.*, 2005, 2007; Tan *et al.*, 2010). High temperature decrease bursa of Fabricius, thymus, and spleen weights, and cause mild multifocal acute enteritis characterized by an in-

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creased presence of lymphocytes and plasmocytes within the jejunum's lamina propria. The stress-induced hypothalamic-pituitary-adrenal axis activation may be responsible for the negative effects observed on the chickens' performance, immune function, and intestinal mucosa on exposure to high temperature (Quinteiro-Filho *et al.*, 2010).

Hyperthermia can induce the metabolic changes that are involved in the induction of oxidative stress, and heat stress is responsible for stimulating reactive oxygen species (ROS) production. Mujahid *et al.* (2005) provided for the first time the direct evidence of mitochondrial superoxide generation in skeletal muscle of acute heat-stressed birds using both electron spin resonance (ESR) spectroscopy, with 5,5-dimethyl-1-pyrroline N-oxide as a spin trap agent, and lucigenin-derived chemiluminescence (LDCL). In chickens the liver is more susceptible to oxidative stress than heart during acute heat exposure (Lin *et al.*, 2006). Heat stress causes oxidative damage to mitochondrial proteins and lipids in skeletal muscle of chickens, and also causes higher serum MDA levels (Mujahid *et al.*, 2007c) that depends on duration of exposure (Pamok *et al.*, 2009). Heat stress-induced increased superoxide production in the skeletal muscle mitochondria cause oxidative stress and inhibit growth of the chickens (Mujahid *et al.*, 2005). Under heat stress conditions, down-regulation of avian uncoupling protein (avUCP) and mRNA expression are accompanied by increased mitochondrial superoxide production (Mujahid *et al.*, 2006), and these effects occur in a time-dependent manner (Mujahid *et al.*, 2007b).

As ambient temperatures increase within the thermoneutral range, birds initially utilize sensible heat loss mechanisms to control body temperature with little or no loss in growth or production. However, under moderate or severe heat stress, birds minimize heat production since the major route of heat loss, evaporation of water from the respiratory tract (panting), requires considerable energy expenditure. Birds respond by reducing their metabolizable energy (ME) and feed intake to reduce thermogenesis. Although ME intake has been shown to decline at an increasing rate with increasing ambient temperature, it does so more rapidly than the corresponding decline in metabolic heat production. Therefore, less energy would be available for production processes, as ambient temperature increases.

Many factors influence the response of chickens to changes in environmental temperature. The intensity of environmental temperature, humidity, radiant heat, wind velocity, duration of exposure and previous acclimatization of the birds influence the response of chickens to high temperature exposure. Presently, birds are increasingly being subjected to environmental temperatures that are above their comfort zone. Additionally, as a result of genetic selection, and advancement in husbandry, veterinary health and nutritional practices, birds are growing faster and producing more than ever before, and are thus heavier and more productive than they were previously at

any given age with marked change in their metabolic activities.

Birds in general, perform well within a relatively wide temperature range, 10–27°C (Milligan and Winn, 1964; de Albuquerque *et al.*, 1978; Mardsen and Morris, 1987). The highest growth rate of broiler chickens occurs in the range of 10–22°C, while maximum feed efficiency is at 27°C (Kampen, 1984). The ideal optimum temperature range is different for growth and feed efficiency, e.g., feed efficiency in laying hens is reduced below 21°C, while egg production and growth rate are reduced below 10°C. Exposing chickens to high temperatures significantly decreases the feed intake, although high environmental temperature has a significant and direct impact on performance and feed efficiency unrelated to feed intake. For example, exposure of laying hens to 21 and 38°C temperature results in 40–50% reduction in egg production, and the reduction in egg weight at 38°C is only due to reduced feed intake while the reductions in shell thickness and shell strength are mainly due to high temperature (Smith and Oliver, 1972). Similarly, only 63% reduction in broiler chicken growth on exposure to heat stress is due to reduced feed intake (Dale and Fuller, 1979). In addition to the effect of high temperature on feed intake, heat stress-exposure results in increased mitochondrial superoxide production, reduced ATPase activity, and oxidative damage to body proteins and fats, resulting in cellular metabolic changes and growth reduction (Mujahid *et al.*, 2005, 2007a-c; Feng *et al.*, 2008).

The metabolic and nutritional statuses affect the tolerance of chickens exposed to high ambient temperature and there is an interrelationship between nutritional status and resistance to acute heat stress (McCormick *et al.*, 1979; Garlich and McCormick, 1981). Recent nutritional strategies are aimed at decreasing heat production and thus alleviating the heat stress in chickens on exposure to high environmental temperature. The nutritional strategies are designed after considering factors such as type of birds, stage of production, age of birds, duration of heat exposure, intensity of heat exposure and health of the birds. Nutritionists can base their strategy on less heat-production, increased nutrient intake, decreased energy wastage, and reduction in heat-induced oxidative stress and damage in birds to overcome the deleterious effects of high temperatures on metabolism, physiology, feed efficiency and production performance. The adverse effects of high temperature on production performance may be alleviated by the following dietary modifications:

Feed Density

Although the ME requirement decreases with increasing temperature above 21°C, mainly due to a reduction in the energy requirement for maintenance, the requirement for production is not influenced by environmental temperature (Daghir, 1995). High energy feed has commonly been used for chickens in warm regions probably to overcome the negative effects of low feed intake and to reduce

the heat increment. Under severe heat stress, the metabolizable energy (ME) requirement increases due to the bird's need to dissipate body heat by respiratory heat loss. The additions of fat, lysine and methionine have been shown to improve the performance in hot weather (Mickleberry *et al.*, 1966; McNaughton and Reece, 1984; Jiang *et al.*, 2007). The higher fat content of the diet contributes to reduction of the heat production, since fat is metabolized with greater efficiency and has a lower heat increment than either proteins or carbohydrates. High environmental temperature increases the food passage time (Wilson *et al.*, 1980) while fat has been shown to decrease the rate of food passage in gastrointestinal tract (Mateos *et al.*, 1982), thus increasing the nutrient utilization. Therefore, the addition of fat to the diet also appears to increase the energy value of the other feed constituents (Mateos and Sell, 1981).

Alterations in the dietary ME concentration show a limited influence on the feed and nutrient intake and egg mass output of hens in early lay kept at 10–24, 6–16 or 25–35°C temperatures. Even the highest intakes of ME and protein achieved at high temperatures failed to increase the egg mass output to the values attained on any diet at low temperatures (Scott and Balnave, 1988). Increases in energy and calcium intake helped partially to maintain normal egg production, egg weight, and prevent egg shell deformation on the exposure of laying hens to high temperature (Tanor *et al.*, 1984).

However, when chickens are reared in a warm environment, the body weight response to increased dietary energy level occurs only when adequate amino acid levels are supplied (McNaughton and Reece 1984). Increasing dietary ME at particular amino acid: ME ratios significantly improves the growth and feed utilization of chickens kept at 18–26 and 25–35°C ambient temperatures during the finishing period. The optimum amino acid: ME ratio varies with the dietary ME concentration in a hot, but not in a moderate environment. Relatively greater increases in feed intake and growth rate occur in a hot environment when dietary ME increases and the amino acid: ME ratio decreases. Increasing the dietary protein at particular ME concentrations had little or no effect on the feed intake and growth rate of birds kept at high temperatures. The rectal temperatures of birds in a hot environment increase with age and, towards the end of the finishing period, when higher energy diets are fed (Sinurat and Balnave, 1985).

Feed Form and Feeding Time

Offering pelleted feed to broilers can result in a 67% reduction in the energy required for eating. Whereas the ME of the feed is the same whether pelleted or not, the energy sparing effect of pellets is about 6% as a result of the reduced activity (McKinney and Teeter, 2003). Because the physical nature of the pellets allows the birds to consume their feed with less wasted energy, the quality and durability of the pellets are particularly important. A change of 10% in fines may result in a change of 0.01 in

feed conversion ratio. At high temperatures there should be an advantage in providing broilers with high quality pellets, with the minimum amount of fines, thereby reducing the proportion of heat expended in acquiring food (see the review Gous and Morris, 2005). Highest weight gain of broiler chickens can be achieved with good pellet quality. To achieve similar performance with poor pellet quality, higher levels of balanced protein is needed. Additionally, optimum dietary amino acid levels for growing chickens may vary among different physical feed forms (Lemme *et al.*, 2006).

Heat production by chickens can be reduced by withholding feed prior to, and during, a limited period of high temperature stress. Survival is increased if feed is withdrawn at least four to six hours before the period of heat stress (Smith and Teeter, 1988; Francis *et al.*, 1991; Boulahsen *et al.*, 1993; Hiramoto *et al.*, 1995). Withdrawing feed during day, and replacing it at night once the temperature has declined, would appear to be a sensible approach to deal with uncomfortably high temperatures. The mechanism responsible for this beneficial effect is that the heat increment associated with feeding is reduced during the hottest part of the day. To get beneficial effect of this practice, withdrawal of the feed must take place few hours before the high temperature is experienced.

Amino Acids

Heat stress markedly affects the amino acids requirement of broiler chickens for optimum production. Exposure of broiler chickens to heat stress increases the endogenous amino acids in ileal flow (Soleimani *et al.*, 2010). It has been shown that increasing the amino acid levels in the diet of chickens reared under high temperature condition improves their production performance (Corzo *et al.*, 2003; Jiang *et al.*, 2007). A well-balanced amino acid supply should minimize the energy loss from excreting surplus nitrogen and might therefore help the chicken to cope with heat stress. At a high ambient temperature, there is a decrease in protein synthesis (Geraert *et al.*, 1996a), probably due to reduced plasma amino acid concentration and lower energy supply (Temim *et al.*, 2000), as observed in chicken muscle tissue. During heat stress, circulating levels of thyroid hormones are decreased (Geraert *et al.*, 1996b; Tao *et al.*, 2006; Lin *et al.*, 2008), presumably because thyroid hormone increases metabolic rate, and thus metabolic heat production. This heat stress induced decreases in plasma thyroid concentration and increases in plasma corticosterone levels known to reduce protein deposition through alterations in protein turnover in birds and other species (Yunianto *et al.*, 1997). An interaction exists between the amino acid balance and the environmental temperature. Exposure to high environmental temperature has been shown to influence amino acid digestibility and significantly decrease the uptake of certain amino acids from the intestine (Wallis and Balnave, 1984; Balnave and Olivia, 1991; Brake *et al.*, 1998). Additionally, high ambient temperatures affect the ideal amino acid balance for broilers (Brake *et al.*, 1998; Chamrusspollert *et*

al., 2004) and increasing the dietary Arg: Lys ratio improves broiler performance at high temperature (Brake *et al.*, 1998). An interaction also exists between the dietary NaCl and the Arg: Lys ratio, and the dietary NaCl can affect the apparent ileal digestibility of Arg and Lys at certain Arg: Lys ratios (Brake *et al.*, 1998; Balnave and Brake, 2001; Chen *et al.*, 2005). In particular, Brake *et al.*, (1998) observed that at a low dietary NaCl concentration (1.2 g/kg), the feed conversion ratio and body weight gain of heat-stressed chickens were significantly improved with increasing Arg: Lys ratios, but at a higher dietary NaCl concentration (2.4 g/kg), no such response occurred.

Vitamins

Vitamin A and E in combination (15,000 IU retinol and 250 mg dl- α -tocopheryl-acetate/kg diet) reduced the MDA concentration (an indicator of lipid peroxidation) by more than half in the serum and liver of heat stress-exposed chickens, but showed less effect when fed alone (Sahin *et al.*, 2002). The immune response of chickens can be improved by dietary vitamin E supplementation under heat stress as both primary and secondary antibody responses are significantly increased by dietary vitamin E when birds are exposed to high environmental temperature (Niu *et al.*, 2009). Ascorbic acid (vitamin C) has been shown to improve chicken performance at high ambient temperature. Birds experience a less severe stress response after exposure to high temperatures when they are provided dietary ascorbic acid (Mahmoud *et al.*, 2004). Optimum responses in growth, feed efficiency and/or livability in chickens under heat stress were reported to occur with supplements of about 250 mg ascorbic acid/kg diet (Kutlu and Forbes, 1993). Ascorbic acid and chromium have similar effects when fed at high temperatures, and a combination of ascorbic acid (250 mg/kg diet) and chromium (400 μ g Cr/kg of diet) may offer a potential protective management practice in preventing heat-stress related depression in the performance of chickens (Sahin *et al.*, 2003).

Studies have shown that the livability and production of heat-stressed hens can be improved by supplementing their diet with ascorbic acid or vitamin E (Njoku and Nwazota, 1989; Cheng *et al.*, 1990; Bollengier-Lee *et al.*, 1999; Puthongsiriporn *et al.*, 2001). Supplemental vitamin C at 200 mg/kg diet increases eggshell weight, eggshell thickness, and shell breaking strength. A combination of vitamin C (200 mg/kg) and vitamin E (125 IU/kg) is beneficial in eliciting higher antioxidant status in hens exposed to tropical summer conditions (Panda *et al.*, 2008).

Requirements for thiamine have been shown to be significantly increased for chicks raised at 32.5°C, as compared with chickens raised at 21°C (Mills *et al.*, 1947).

Minerals

Dietary modifications in mineral concentration offer a practical way to alleviate the effect of high environmental temperature on chicken performance. Mineral supplementation may reduce the consequences of heat stress in

birds and have beneficial effects on production performance and meat quality. Increased mineral excretion is one of the major consequences of heat stress. Retention rates of phosphorus, potassium, sodium, magnesium, sulfur, manganese, copper, and zinc are lower in chickens raised at high cycling ambient temperatures (24–35°C) compared with those housed at 24°C (Belay and Teeter, 1996).

Egg weight and eggshell strength decline at high environmental temperature. Also, the lower concentrations of plasma calcium and inorganic phosphate in hens exposed to 30°C compared to 18°C (Usayran and Balnave, 1995) may provide some evidence of an increased requirement for these minerals in heat-stressed hens.

Stress causes secretion of epinephrine and corticosteroids and results in Mg loss (Seelig, 1980, 1981). Mg-aspartate supplementation increases the body weight of chickens during heat stress (Donoghue *et al.*, 1990). Zinc supplementation results in an improved live weight gain, feed efficiency, and carcass traits, as well as a decrease in serum MDA concentrations in chickens reared at high temperature (Kucuk *et al.*, 2003).

When chickens are exposed to heat stress (34°C), plasma sodium values reduces after 6 h while no such change occurs after 12 or 18 h of exposure. Potassium levels are lower by 6 or 12 h of heat stress and no differences in blood calcium levels are observed between the control and heat-stressed chickens (Mujahid *et al.*, 2009b). Exposure to different durations of heat stress results in significant decreases in the levels of blood HCO_3^- and pCO_2 and significant concomitant increase in blood pH which is dependent on duration of heat stress exposure (Toyomizu *et al.*, 2005, Mujahid *et al.*, 2009b).

Panting during heat stress to dissipate body heat may result in an increased loss of carbon dioxide and a consequent depletion of blood bicarbonate (Gorman and Balnave, 1994) which can induce respiratory alkalosis and this may be exacerbated by high RH that makes respiratory heat loss less efficient. Blood alkalosis limits growth rate of chickens reared under high environmental temperature, however, this induced respiratory alkalosis can be partially alleviated by dietary modifications. Therefore, it is theoretically possible that heat stress may induce a metabolic requirement for the bicarbonate ion (Teeter *et al.*, 1985).

One of the means for alleviating the problem of respiratory alkalosis associated with panting has been to supplement the diet with sodium bicarbonate. This procedure has the additional merit of improving the shell quality of eggs from heat-stressed hens (Balnave and Muheereza, 1997). However, it has been reported that the dietary bicarbonate should be consumed during the period of egg shell formation (Balnave and Muheereza, 1997, 1998).

Enhanced body weight gains can be achieved among chickens kept at high temperatures with the addition of either sodium bicarbonate or ammonium chloride, the latter (at 10 g/kg of diet) resulting in a 25 percent increase in growth rate over the controls (Teeter *et al.*, 1985).

Sodium salts reduce the alkalotic pH and enhance the blood sodium content, which ultimately improves the blood electrolyte balance and overall performance of heat-stressed chickens. Supplementing diet with sodium salt improves the live performance of heat-stressed chickens and NaHCO_3 gives better production performance than other sodium supplements (Ahmad *et al.*, 2006).

Glucose

Exposure of chickens to elevated environmental temperature markedly reduce food intake and the associated lower growth rate is accompanied by increased plasma glucose levels (McCormick *et al.*, 1979) and increased *in vivo* uptake of galactose and methionine when measured on a tissue dry weight basis (Mitchell and Carlisle, 1992). This apparently enhanced absorption capacity was confirmed by *in vitro* studies in which enterocytes from chronically heat-adapted birds showed a 50% increase in galactose accumulation ratio compared with cells from control chickens (Mitchell *et al.*, 1995). Heat stress increased microvillous length in chickens, indicating that the surface brush-border membrane is increased even in a metabolic situation characterized by a general reduction in protein synthesis (Geraert *et al.*, 1996a). This effect on apical surface, together with increased activity of sodium-dependent glucose transporter 1, enhances the capacity to absorb glucose and can, therefore, be interpreted as physiological adaptations of the chicken jejunum to guarantee energy supply (Garriga *et al.*, 2006). Thus, supplemental glucose intake by chickens on exposure to high temperature alleviates the influence of heat stress and prolongs the survival time. When chickens are exposed to high temperature, rectal temperature enhances quickly in birds given tap water, while slow increases are found in birds offered glucose water with subsequently higher plasma glucose levels (Iwasaki *et al.*, 1998). Oral administration of glucose prevents decrease in feed intake and growth rate, normalizes physiological and immunological responses, and alleviates the influence of heat stress on whole blood viscosity and plasma osmolality in heat stress-exposed chickens (Zhou *et al.*, 1998; Takahashi and Akiba, 2002).

Dietary Fatty Acids

Fatty acid oxidation is increased to meet the energy requirements of birds exposed to heat stress (McKee *et al.*, 1997) as heat stress increases the plasma fatty acids (Mujahid *et al.*, 2007b), triglycerides, cholesterol (Sahin *et al.*, 2006) and enzymes involved in the transport and oxidation of fatty acids (Mujahid *et al.*, 2007b). Hyperthermia promotes the oxidation of fatty acids as respiratory quotient of heat-stressed birds is decreased (McKee *et al.*, 1997). However, cells of the heart, kidney and liver of heat-stressed chickens exhibit an abnormally high accumulation of lipid droplets in the cytoplasm and massive fatty degeneration (Aengwanich and Simarak, 2004) and the fatty acid composition of adipose tissue is altered by heat stress (Geraert, 1998). The alterations in fatty acid

mobilization, imbalance partitioning and defects in fatty acid oxidation are detrimental to overall bioenergetics of birds and are associated with pathology of heat stress. Dietary manipulation of fat content to ameliorate the effect of heat stress in chickens is presumably based on the concept that lipid sources have lower heat increments than the carbohydrates that they replace, while little attention is given to the fatty acid composition of the fat source.

There is evidence that dietary supplementation with long chain fatty acids such as palmitic, linoleic and oleic acid can ameliorate the deleterious effects of high temperature in poultry (Njoku and Nwazota, 1989; Balnave, 1998; Mujahid *et al.*, 2009a). Recently, the mechanism of heat stress that exerts its adverse effects on mitochondrial bioenergetics has been elucidated and strongly suggests that up-regulation of avian (av) uncoupling protein (UCP) expression using specific fatty acids may prevent the cascades of events that lead to energy wastage, decreased production and tissue damage during heat stress.

It is well-known that ROS production can be decreased by mild uncoupling of mitochondrial respiration (Brand *et al.*, 2004; Skulachev, 1998). UCPs are specialized members of the mitochondrial transporter family that allow passive proton transport through the mitochondrial inner membrane. Recent data support the idea that avUCP is involved in the control of ROS generation (Abe *et al.*, 2006). The mitochondrial oxidative phosphorylation and role of avUCP as an additional re-entry port for proton has been shown in Figure 1 (Mujahid *et al.*, 2007a) which acts as a type of "pressure-relief valve" to prevent excessive accumulation of protons in the mitochondrial intermembrane. There is functional *in vitro* evidence that avUCP regulates mitochondrial reactive oxygen species (ROS) production in situations of enhanced metabolic activity (Rey *et al.*, 2010). This fits with the hypothesis that mild uncoupling caused by the UCPs decrease ROS production and it can be assumed that avUCP, expressed appropriately, may play a role in the alleviation of mitochondrial ROS production and thus acting as an antioxidant under conditions of heat stress.

Heat stress increased mitochondrial ROS production and MDA levels, and decreased amount of avUCP in skeletal muscle mitochondria (Mujahid *et al.*, 2006, 2007c, 2009a). Therefore, up-regulation of avUCP could attenuate oxidative damage caused by heat stress. Birds fed an olive oil-supplemented diet, as a source of oleic acid (oleic acid constitute 70–80% of olive oil, Tripoli *et al.*, 2005), showed better production performance compared with control diet-fed birds, whether under thermoneutral conditions or when exposed to heat stress (Mujahid *et al.*, 2009a). On exposure to heat stress, the control diet-fed birds showed marked body weight loss, whereas in oleic acid-fed birds, the negative effects of heat stress on growth were reduced. Feeding birds an oleic acid supplemented diet increased the expression of avUCP in skeletal muscle mitochondria and decreased ROS production and oxidative damage. Studies on mitochondrial function showed

Fig. 1. Possible role of uncoupling protein (UCP) and A nucleotide translocator (ANT) in control of superoxide production in mitochondria. A mitochondrial proton gradient generated by complexes of respiratory chain across inner mitochondrial membrane is used by 2 mechanisms. First, it is used by adenosine triphosphate (ATP) synthase to phosphorylate adenosine diphosphate (ADP) and produce ATP (A). This oxidative phosphorylation is the main consumer of the proton gradient. Second, another mechanism consuming the proton gradient is proton leak (B). When the proton gradient is dissociated by UCP or ANT, the re-entry of protons into the matrix is not coupled to ATP synthesis. In this way, UCP and ANT cause mitochondrial proton leak, resulting in reduction of the proton gradient. During the process of oxidative phosphorylation some electrons leak out from the electron transport chain (ETC) and combine with molecular oxygen to generate superoxide (C). This superoxide production is very sensitive to the proton gradient. Mild uncoupling that reduces the proton gradient can result in decreased superoxide production at the expense of a small loss of energy.

FADH₂=reduced flavin A dinucleotide; FAD⁺=flavin A dinucleotide; NADH=reduced nicotinamide A dinucleotide; NAD⁺=nicotinamide A dinucleotide (Mujahid *et al.*, 2007a)

that heat stress increased membrane potential in state 4, which was reversed by feeding birds an oleic acid supplemented diet, although no differences in basal proton leak were observed between control and heat-stressed groups. Results demonstrated that under heat stress, mitochondrial ROS production and oleic acid-induced reduction of ROS production may occur due to changes in respiratory chain activity as well as avUCP expression in skeletal muscle mitochondria (Mujahid *et al.*, 2009a). The specificity of UCPs for certain types of fatty acids may explain why the practice of feeding high-fat diets to poultry exposed to heat stress has been successful in some instances and has failed in others (Cronje, 2011).

Oilseed crops such as soybean, rapeseed (canola), peanut, sunflower and cottonseed have now been bred or engineered to produce oil high in oleic acid (Liu *et al.*, 2002). Recent advances in the patho-physiology of heat

stress strongly suggest that the strategic use of new oil seed varieties may ameliorate the adverse effects of heat stress in poultry (Cronje, 2011).

Conclusions

High environmental temperature exposure is of major concern for poultry industry especially in the hot region of the world. Different nutritional strategies have been used to alleviate stress in high temperature-exposed chickens. The nutritional strategies are designed after considering the factors such as type of birds, age of birds, stage of production, duration and intensity of heat exposure, and health of the birds. The nutritionists can base their strategy on less heat-production, increased nutrient intake, decreased energy wastage, and reduction in heat-induced oxidative stress and damage in birds to overcome the deleterious effects of high temperatures on metabolism,

physiology, feed efficiency, production performance and health. This can be accomplished by nutritional strategies to reduce heat stress by feeding good quality feed with high digestibility and nutrient density, adding fat as an energy source, balancing and provision of additional amino acids, and supplementing with vitamins, minerals and glucose. Recently, there are new concepts for nutritional strategies to focus on the ROS production by the chickens and to decrease the oxidative stress and damage on exposure to high environmental temperature by feeding specific fatty acids. None of these strategies are effective alone in terms of growth, feed efficiency, livability or meat quality, therefore, a combination of the nutritional strategies may help to alleviate the deleterious effects of heat stress and maintain the efficiency and production of chickens under high environmental temperature.

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