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PHYSIOLOGICAL REACTIONS OF POULTRY TO HEAT STRESS AND METHODS TO REDUCE ITS EFFECTS ON POULTRY PRODUCTION

Narongsak Chaiyabutr*

Abstract

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PHYSIOLOGICAL REACTIONS OF POULTRY TO HEAT STRESS AND METHODS TO REDUCE ITS EFFECTS ON POULTRY PRODUCTION

Thermal environment is regarded as the prime factor governing the production because, both the feed consumption and heat production are related closely to the thermal environment. Therefore, heat stress is caused by a combination of environmental and animal factors that result in reduced performance as the animal attempts to cool itself. In this review, the physiological reactions of bodily functions during heat exposure in poultry are described. Thermal panting, the marked response of poultry to heat stress, is compensatory behavior to permit a high rate of evaporative cooling from the respiratory tract. Cardiorespiratory responses to thermal stress show different patterns among birds that are experienced or inexperienced with high ambient temperature. The effect of heat stress on changes of body fluids and renal function of poultry are reviewed. A number of methods to reduce heat stress on poultry production are presented along with various management considerations, including water management, dietary adjustments, increasing heat tolerance through the process of acclimation, designing mineral drinking water supplementation and designing rations for correcting the acid-base imbalance. The beneficial effects of supplemental anti-stress agents (e.g., ascorbic acid) in reducing heat stress are also noted.

Keywords : poultry, heat stress, bodily function

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*ผู้รับผิดชอบบทความ

บทคัดย่อ

ณรงค์ศักดิ์ ชัยบุตร*

ปฏิกิริยาทางสรีรวิทยาของไก่ต่อภาวะเครียดจากความร้อนและวิธีการลดผลจากความเครียดกับการเพิ่มผลผลิต

อุณหภูมิสภาพแวดล้อมถือเป็นปัจจัยหลักต่อการเพิ่มผลผลิตในไก่ การกินอาหารกับการเพิ่มความร้อนภายในร่างกายจะมีส่วนสัมพันธ์กับอุณหภูมิแวดล้อม การเกิดภาวะเครียดจากความร้อนจึงเป็นผลจากความร้อนทั้งที่อยู่รอบตัวสัตว์และเกิดจากภายในตัวสัตว์ทำให้ลดการเจริญเติบโตของไก่ในขณะที่สัตว์พยายามลดอุณหภูมิภายในร่างกายลง บทความนี้วิเคราะห์ปฏิกิริยาทางสรีรวิทยาของการทำงานของร่างกายขณะที่ไก่อยู่ในภาวะเครียดจากความร้อน การหายใจหอบของไก่เป็นพฤติกรรมแรกที่สัตว์แสดงออกที่จะระบายความร้อนจากร่างกายผ่านระบบทางเดินหายใจ การตอบสนองการทำงานของระบบหัวใจและหายใจในไก่ที่เคยมีความคุ้นเคยและไม่เคยมีความคุ้นเคยกับอุณหภูมิแวดล้อมที่สูงมาก่อน ผลของภาวะเครียดจากความร้อนต่อการเปลี่ยนแปลงของเหลวภายในร่างกายและการทำงานของไต มีการชี้ให้เห็นวิธีการที่จะลดความเครียดจากความร้อนในไก่ด้วยวิธีการต่างๆ รวมทั้งผลของการเสริมสารเคมี เช่น วิตามินซีในการลดภาวะเครียดจากความร้อน

คำสำคัญ : ไก่ ภาวะเครียดจากความร้อน การทำงานของร่างกาย

Introduction

Research development in poultry science has been shown by improving both breeds and quality of mixed feed for high productivity in poultry production. The techniques are not universally available throughout the world. The genetic potentiality of poultry would not be utilized fully. The main reason for this restriction is environment constraints. Therefore, an improvement of the production and its efficiency largely depends on the quality of the environmental management. Rational control of thermal environment requires at least two concepts of knowledge. One is an understanding of thermoregulation of poultry, and the other is an understanding of the environmental factors which affect poultry production, their characteristic mechanisms and the magnitude of the effects. High environmental temperatures or heat stress, however, have marked effects on the behavior, food and water consumption, blood composition, cardio-respiratory behavior, heat production and body temperature of poultry. When the animal is subjected to heat stress, regulatory mechanisms involve both specific action and

non-specific actions. The specific actions dealt with under homeostasis include heat loss and cardio-respiratory adjustments. Non-specific actions are dependent on integrative capacities of the nervous and endocrine systems. In the absence of responses, the bird would enter the stage of fatigue and die. All the available evidence indicates that poultry have lower heat tolerance than mammal. This brief review is, therefore, concerned with the effects of heat stress on bodily functions of poultry and methods to reduce its effect on poultry production.

Temperature regulation

Heat stress is a multifaceted adaptive response that occurs when an animal's capacity for heat dissipation is exceeded by the heat load acquired through excessive exposure to high environmental temperature. Poultry, like mammals, are homeothermic, which means that they have an external comfort zone (thermoneutral zone) of around 18°C to 36°C, while the internal environment (body temperature) of poultry remains relatively constant. However, the body temperature of domestic fowl varies

with size, breed and sex. An adult chicken has a narrow range of temperature in the thermoneutral zone (18°C-23.9°C) (Sturkie, 1965), while a broiler has a thermoneutral zone of 32°C-35°C. The upper critical temperature for broilers is between 36 and 37°C (Van Der Hel et al., 1991). The chicken is most comfortable when the ambient temperature is in the thermoneutral zone. Health, weight gain, productivity and feed efficiency are maximized and stress is minimized at these temperatures (Deaton et al., 1978). Body temperature represents the integrated response of an animal to its internal and external environment. Temperature, humidity and air velocity are the primary environmental factors which enter into the birds' ability to adapt to extensive changes in temperature. Stability of body temperature is an essential factor in production efficiency. Poultry maintain homeothermy at high environmental temperatures by developing a panting mechanism. It has been reported that broiler chicks start panting and begin showing compensatory behavior at an ambient temperature of 35.5°C and relative humidity of 50-70% (Van Der Hel et al., 1991). This will permit a high rate of evaporative cooling from the respiratory tract. Behavioral response to high environmental temperature is prevented by destruction of the hypothalamus. The entire panting responses to heat can be abolished by bilateral vagotomy in chickens (Siegel, 1968).

Heat gain and heat loss

Body heat gain results from three kinds of sources: chemical, mechanical and thermal sources. Chemical sources of body heat involve metabolism (e.g. digestion of feedstuffs). Mechanical sources are related to physical activity. Body heat gains derive from a thermal source if ambient (environment) temperature is greater than the body temperature. The animal has four principal ways to dissipate heat: conduction, convection, radiation and evaporation. Poultry have an ability to tolerate prolonged hyperthermia and higher body temperatures compared to mammals (Whittow, 1965). The skin of poultry is thin and elastic over most of the body. The dry scaly surface results

from desquamation of epithelial cells and the absence of sweat or sebaceous glands. During heat stress, therefore, evaporative heat loss from the outer surface is not extensive in poultry. Heat loss by conduction represents a small proportion of the total because the air is a poor thermal conductor. Heat loss from the deep tissue to the skin surface is largely affected by vascular connection. Such convective heat loss is directly related to the temperature differential and the surrounding air velocity. Heat loss through radiation is dependent on the size of the bird, i.e., the effective radiating surface. Radiation of body heat can only decrease the body temperature when the ambient temperature is lower than the birds' body temperature.

Non-evaporative and evaporative cooling

An absence of sweat glands in poultry is manifested in the behavior of their skin temperature. The skin temperature in areas covered by feathers remains close to the deep body temperature over a wide range of environmental temperatures, but the temperatures of the non-feather extremities are more variable. Since only small increases occur in the covered areas when the environmental temperature rises, heat loss from these areas is small. The degree of non-evaporative cooling is influenced by peripheral blood flow and the differential between bird body and ambient temperatures. In poultry, evaporative cooling occurs when moisture evaporates away from the respiratory tract. Evaporation is the primary means that poultry have to cool themselves when the environmental temperature equals or surpasses the body temperature. Panting occurs when the deep body temperature of poultry reaches 41-43°C (King and Farner, 1964). Panting aids cooling body temperature in two ways. First, it increases saliva secretion, which can increase evaporative cooling. Second, it cools by increasing evaporative cooling through the respiratory tract by increasing the frequency of breathing. The lungs of poultry are small relative to their body size and are not capable of the same elastic recoil as those of mammals. The structural arrangement within is unique in that all the

air passages are open at both ends, which permits free circulation of air in both directions. This allows repeated movement of inspired air from trachea to air sacs and its return. The largely nonvascular air sacs allow an extensive area of moist surface for evaporative cooling while not participating in gaseous exchange.

Cardio-respiratory responses

In addition to heat gained from the environment during heat exposure, body heat is also derived from metabolic heat including heat for maintenance plus increments for activities. In heart rate has been generally used as an index of heat production. The relationship between heat production and the heart rate has been demonstrated in farm animals for responses to stimuli provided both by the ambient temperature and the level of metabolic heat (Yamamoto, 1989). Heat stress can strain many body systems to the limits of regulatory ability.

However, data on cardiovascular response, particularly concerning the distribution of blood flow during thermal stress, show variations between species. A study of the effects of heat stress on cardio-respiratory responses and behavior, in comparison between birds experienced and inexperienced with high ambient temperature, has been carried out (Zhou et al., 1996). Heat production has been shown to be higher in the inexperienced birds coinciding with a higher heart rate during exposure to high ambient temperature. These responses have been suggested to be due to changes in behavior during heat stress. The more frequent standing and lying behavior of the inexperienced birds during exposure to 36°C of ambient temperature has been observed. The respiratory rate of birds increased markedly in both experienced and inexperienced birds during heat stress (Table 1), but the respiratory rate was found to lower in the inexperienced birds.

Table 1. Changes in body temperatures, respiratory rate and heart rate of laying hens expose to high environmental temperature

| | Environmental temperature | | |
|--------------------------------|---------------------------|------|------|
| | 24°C | 32°C | 36°C |
| Abdominal temp. (°C) | 41.3 | 41.8 | 43.3 |
| Respiratory rate (breaths/min) | 20 | 112 | 257 |
| Heart rate (beats/min) | 259 | 252 | 287 |

Source : Zhou et al. (1996)

Alterations in body fluid compartments

It is known that changes in total body water content in animals under hot climate conditions are considered an adaptive reaction to ameliorate heat stress. This adaptive reaction has an important physiological consequence in terms of greatly increased water turnover, also related to water exchange and cardiovascular function. When poultry are exposed to high temperature, changes in blood volume and plasma volume are apparent as well as in their compositions (Zhou et al., 1999). Blood volume and plasma volume have been shown to increase while packed cell

volume slightly decreases when poultry are acutely exposed to high temperature for 4 h. Similar results have been obtained in large animal like buffalo during heat exposure (Chaiyabutr et al., 1987). During intravascular volume expansion in heat-stressed birds an increase in plasma volume (plasma water) could come from extravascular tissue space (i.e., the reduction of extracellular fluid volume) (Table 2). This adaptive mechanism by an increase in plasma water is useful in assisting the bird in heat tolerance due to the high specific heat of water that slow down the elevation in body temperature.

Table 2. Changes in blood volume, plasma volume, extracellular fluid volume and intracellular fluid volume of poultry exposed to high ambient temperature for 4 h

| | Environmental temperature | | |
|--|---------------------------|-------|-------|
| | 20°C | 28°C | 36°C |
| Packed cell volume (%) | 30.8 | -1.1* | -0.9* |
| Plasma volume (ml/100 g.BW) | 4.5 | +0.7 | +0.2 |
| Blood volume (ml/100 g.BW) | 6.4 | +1.0 | +0.4 |
| Extracellular fluid volume (ml/100 g.BW) | 22.7 | -2.3 | -3.5 |
| Intracellular fluid volume (ml/100 g.BW) | 51.2 | +1.4 | +4.7 |
| Total body water (ml/100 g.BW) | 73.8 | -0.9 | +1.8 |

Source : Zhou et al. (1999). *Difference (value at 28 or 36°C - value at 20°C)

Kidney function

It is known that control of the turnover of water and electrolytes is important in the survival of animals at high environmental temperature. The alimentary canal can provide the fluid for the body while the kidneys are responsible for retaining as much water as possible. There is little information on the kidney function of poultry in a hot environment, particularly from the point of view of a compensation for the loss of water and electrolytes. During acute heat exposure in poultry, variable changes in urinary electrolyte excretion have been reported. In the study of Belay and Teeter (1993), exposing chicks to acute heat stress (35°C) for 4 h has been shown to increase water loss by 64% compared with birds housed at 24°C. Water loss was mainly due to an increase in urine production and free water clearance independent of water consumption. An increment of total urinary K⁺, P⁻, Na⁺, Mg⁺⁺ and Ca⁺⁺ excretion has been reported in heat-stressed broilers. The excretion rout for this effect has been suggested to vary with the specific mineral and heat stress severity (Belay et al.,1992). In a different study, acute heat acclimated broilers exposed to 35°C of ambient temperature had significantly lower glomerular filtration rates (GFR), filtered loads of sodium and tubular sodium reabsorption rates than the control birds (Wideman et al., 1994). These changes in kidney function are hypothesized to minimize

urinary fluid and solute loss when heat-acclimated broilers consume large quantities of water to support evaporation cooling. Reductions in GFR, filtered quantities of water, and tubular sodium reabsorption rates may help heat-acclimated broilers to reduce the metabolic heat load associated with active (energy requiring) recovery of solute from the glomerular ultrafiltrate. However, more work is necessary to better understand how various factors could come into play in explaining this evidence.

Other reactions

Heat stress results from the combined effects of relative humidity and ambient temperature. The effects of heat stress include decreased voluntary feed intake, growth rate and feed efficiency and metabolizable energy intake (Van Kampen, 1981); reduction of egg production (Scott and Balname, 1988) including degradation of the egg shell quality in the hot season; increased breathing (panting); increased mortality and morbidity (Reece et al.,1972; Deaton et al., 1978). Blood electrolyte balance is altered during heat stress (Teeter et al., 1985). Blood potassium can be depressed in heat stressed poultry (Cheveille, 1979). Heat stress could also depress immune functions (Ferket and Qureshi, 1992).

Methods for reducing heat stress

Responses to high environmental temperature through several mechanisms of heat dissipation will affect the bodily functions of the animals. Physiological changes can then develop which involve many body systems. Understanding of the basis of physiological stress and insight into application of this knowledge during these situations would improve poultry performance. It is obvious that management of heat stress is a key to better poultry management in hot climates. Management factors can minimize the adverse affects of heat stress on poultry. Successful poultry management from the effect of heat stress should take into account the status of the flock, anticipating heat stress and responding by modifying the environment or modifying the diet.

Water management

It is well known that water is essential for most processes in living organisms. During heat stress, birds must rely on an increase evaporative cooling process to dissipate excess heat via respiration. Panting is the major route of heat loss, dissipating as much as 80% of heat production during heat stress (Van Kampen, 1981, Wiernusz and Teeter, 1993). Broilers will require 2 times more water

during periods of heat stress (24°C to 35°C) (Deyhim and Teeter, 1991). Managing poultry for maximum evaporative cooling potential per breath will center on water consumption, forcing poultry to sustain higher water intake levels than required to simply replace water lost due to evaporative cooling. Increasing water intake by addition of various salts to the drinking water has been performed. This will alter the poultry's osmotic balance, increase water consumption and affect water balance. Poultry in positive water balance would be better able to maintain body temperature homeostasis. The degree of evaporative heat dissipation would correlate with water consumption and balance (Belay and Teeter, 1993). During heat stress, water intake by drinking is a critical consideration which is markedly increased in an attempt to reduce the body core temperature. Providing adequate access to cool, clean water will help minimize heat stress. The interaction between salt addition to drinking water and drinking water temperature has been reported by Teeter (1994). It indicates that KCl drinking water fortification increased feed consumption and growth rate when the temperature of the consumed water was lower than the bird's body temperature (Table 3).

Table 3. Effects of water temperature and KCl on body temperature, water consumption and daily weight gain of heat-stressed broiler

| Water temperature (°C) | Body temperature (°C) | | Water temperature (%) | | Daily weight gain (Gm) | |
|---------------------------|-----------------------|---------|-----------------------|---------|------------------------|---------|
| | Control | 0.5%KCl | Control | 0.5%KCl | Control | 0.5%KCl |
| 13 | 42.8 | -0.1* | 100 | +29* | 55.4 | +4.8* |
| 31 | 43.1 | -0.2 | 99 | +29 | 50.3 | +6.2 |
| 42 | 43.3 | -0.2 | 100 | -7 | 47.0 | -4.5 |

Source : Teeter (1994). *Difference (0.5% KCl - control)

Dietary adjustments

Ideally, reduced feed intake is a behavioral response to heat stress in homeothermic animals. The total nutrient intake will decrease with the decrease of feed intake as a result of high environmental temperature. Metabolic rates increase with an increase in environmental temperature. Consequently, the utilization of total nutrients during catabolism will be increased. Thus, animals may suffer from a heat load during feed intake under high environmental temperature. Increasing feed consumption has been reported to elevate mortality during acute heat stress. Numerous dietary manipulations have been evaluated for efficacy to reduce heat stress consequences. Dietary adjustment by reduction of consumption would favor bird survivability. Several studies have shown the effects of feed consumption on bodily responses in heat-stressed poultry. In laying hens, heat production increased with increase in feed intake at an environmental temperature of 28(C while abdominal temperature varied little (Li et al., 1992). Abdominal temperature has been observed to

increase during feeding at environmental temperatures above 28°C, in contrast to the observation of Wierzmu and Teeter (1993) that the body temperature of broilers was nearly unchanged during feeding at 35°C although heat production increased. The level of feed intake has been shown not to affect the body temperature after feeding at environmental temperature of 24°C and 35°C (Teeter et al.,1987). Feed restriction or short-term feed withdrawal during heat stress has been reported to reduce mortality in poultry (Ouart et al., 1990). Results of this study are summarized in Table 4. Ouart et al. (1990) also suggested that the fasted, heat-stressed birds were less active during the period of increasing temperature. Feed withdrawal effects on broiler behavior may be an important part of the benefits of fasting. However, many dietary adjustments have been used with varying degrees of success. Adjustments to increase the dietary fat level during periods of heat stress may reduce the heat increment of the diet which will lower the body temperature (Fuller and Rendon, 1977).

Table 4. Effects of feed restriction on daily feed, daily water consumption and mortality of heat-stressed poultry

| | Daily feed/bird (%control) | Daily water consumption/bird (%control) | Mortality (%) |
|------------------------|-------------------------------|--|------------------|
| Unheated/fed (control) | 100 | 100 | 0 |
| Unheated/fasting | 87 | 90 | 0 |
| Heated/fed | 83 | 97 | 41 |
| Heated/fasting | 87 | 95 | 35 |

Source : Ouart et al. (1990).

Acclimation

Heat stress acclimation is the summation of all physiological responses initiated by the poultry to counter the high ambient temperature perturbation. Poultry could be acclimated by repeated, short, daily exposure to a hot, humid climate (e.g., 35-37 (C, 60% RH). The practical manifestation of this process is that poultry can survive a short period of acute heat exposure with a lower body temperature in an environmental temperature which

previously caused a marked rise in body temperature. Heat stress mortality has been shown to be less severe for acclimated than for unacclimated control poultry. Results of research on the acclimation of domestic fowl are summarized in Table 5. Exposing birds to high ambient temperature at 5 days of age has been shown to reduce mortality 36 days later when exposed to acute heat stress (Arjona et al., 1988). Broilers aged 46 days that were acclimated using a diurnal cycle of 24-35-24°C for 4 days

had significantly lower body temperature than control broilers during acute heat exposure to 41°C for 3.5 h (May et al., 1987). These authors observed that mortality was 60% and 10% for control and acclimated broilers when no feed was provided during the heat exposure. It is well known that feed consumption is positively associated with heat production, and it has been shown that feed intake plays a significant role in the acclimation process. The effect of feed intake on body temperature and water

consumption of acclimated broilers during heat exposure has been reported by Lott (1991). Acclimated and unacclimated birds given access to feed for 1 h before a heat exposure consumed 60 and 50 ml of water per broiler, respectively, during the heat exposure for 12 h. In broilers not receiving feed the water consumption was 58 and 30 ml per broiler, respectively. This study indicates that the increase in water consumption appears to be associated with the lower body temperature of the acclimated birds a compared with the unacclimated birds (Table 5).

Table 5. Water consumption, body temperature and mortality of acclimated and unacclimated poultry during feeding

| Group | Feeding treatment | Water consumption | | Body temp. | | Mortality (%) | Source |
|--------------------------------|---|-------------------|---------|------------|---------|---------------|-------------------------|
| | | (ml/broiler) | %change | (°C) | %change | | |
| Unacclimated (41 °C - 4 h) | - No feeding during heat exposure | Ad lib | | 45.61 | | 60 | May et al. (1987) |
| Acclimated (41 °C - 4 h) | - No feeding during heat exposure | Ad lib | - | 44.15 | -3.2 | 10 | |
| Unacclimated (34 °C - 12 h) | - Feeding 1h prior to heat exposure | 50 | | 44.1 | | - | Lott (1991) |
| | - No feeding 1 h prior to heat exposure | 30 | -40 | 44.7 | +1.4 | - | |
| Acclimated (34 °C - 12 h) | - Feeding 1h prior to heat exposure | 60 | | 44.2 | | - | |
| | - No feeding 1 h prior to heat exposure | 58 | -3 | 43.8 | -0.9 | - | |
| Unacclimated (35 °C - 6 h) | - Feeding intake (10%kg) during heat exposure | Ad lib | | 44.6 | | - | Teeter et al. (1992) |
| Acclimated (35 °C - 6 h) | - Feeding intake (10%kg) during heat exposure | Ad lib | - | 44.1 | -1.12 | - | |

Acclimated and unacclimated broilers given access to feed had similar body temperatures, but the acclimated broilers not receiving feed had a significantly lower body temperature than the unacclimated broilers. Body temperature increased linearly as the feeding level increased (Teeter et al., 1992). However, bird acclimation to heat stress has metabolic effects independent of feed

consumption. In comparison between acclimated and unacclimated birds, despite equalized feed and feeding patterns, birds acclimated to heat had a higher body temperature determined at normal ambient temperature and a lower body temperature when measured at 35°C ambient temperature. Teeter and co-workers (1992) suggested that an acclimated bird was capable of shifting

exothermic processes from times of heat stress to thermoneutral time periods. The birds' set point was elevated when exposed to cycling high ambient temperature. The heat dissipation mechanisms required greater than 12 h to restore normal body temperature. Heat acclimation of chickens will increase their heat resistance. In early exposed birds, it may promote birds' ability to cope with the subsequent heat load by altering thermoregulatory physiological responses (e.g., a higher panting rate) and behavioral patterns (e.g., lower standing-lying frequency and lying time) (Zhou et al., 1997). The reduction of hormone triiodothyronine and changes in bodily hemodynamics have also been suggested to be part of the mechanisms associated with improved thermorelance of acclimated poultry (Yahav and Hurwitz, 1996).

Mineral drinking water supplementation

Exposure to high ambient temperature and high relative humidity caused an altered respiratory rate and other physiological alterations. Non-sweating animals normally exhibit panting and respiratory alkalosis during heat stress. The alkalosis induced by heat stress has been shown to relate to negative electrolyte balance for K^+ as well as Na^+ in both avian and mammalian species (Belay et al., 1992; Chaiyabutr et al., 1997). In poultry, heat stress depresses plasma potassium concentration (Huston, 1978) enhances urinary potassium excretion and reduces body potassium retention (Smith and Teeter, 1987). It is known that potassium is the most abundant intracellular cation and potassium is known to be required for the maintenance of normal metabolic processes in many species of animals, including poultry. Such an occurrence of potassium depletion during heat stress may cause animal discomfort and potentiate alterations in certain physiological functions including metabolic processes, nerve conduction, excitation-contraction in muscle cells, and regulation of cell volume. In many studies published to date on the dietary adjustments to counteract these changes, various managements have been used in poultry

but with mixed results (Table 6). Deyhim and Teeter (1991) reported that 0.5% KCl and 0.39% NaCl drinking water supplementation reduce the severity of heat stress in broilers exposed to a high ambient temperature of 35°C. Heat-stressed birds consuming 0.5% KCl drinking water had higher survivability by approximately 10% as compared with heat-stressed birds consuming normal water. Increased survivability during heat stress has been shown to correlate with water consumption. During heat stress, KCl increased water consumption by 68% and NaCl by 35% over controls, with bird survivability paralleling water consumption. During acute heat stress, the urine production of broilers has been shown to increase independently of water consumption, whereas the degree of evaporative heat loss is correlated positively with water consumption. Manipulation of water intake via salt supplementation of drinking water would offer the potential to increase the tolerance of broilers to acute heat stress. Belay and Teeter (1993) showed that supplementing the drinking water of broilers with 0.75% KCl increased water consumption by 91%, evaporation heat loss by 20% and apparent respiration efficiency 27%. Body weight gain and water consumption have been shown to be influenced by electrolytes with NaCl supplements in drinking water. Birds on the NaCl treatment consumed up to 37% more water than unsupplemented birds and gained 10.5% more weight than those receiving no water additive. However, there was no effect of electrolytes on carcass characteristics (Smith, 1994). The study of Ait-Boulaheh and co-workers (1995) on acute heat-stressed 7 week-old chickens indicated that heat tolerance of chickens as evaluated by body temperature and prostration time was improved by KCl supplementation of the drinking water. The use of KCl at 0.6% gave the most beneficial electrolyte solution for reducing the heat-induced elevation in body temperature and increasing prostration time. Using KCl at 0.6% also produced favorable changes in blood acid-base balance, water consumption and water elimination.

Table 6. Physiology responses to KCl supplementation in heat-stressed poultry

| Feeding treatment | Water consumption (% control) | Body temp. (°C) | Respiration (breaths/min) | Weight gain (% control) | Survivability (%) | Plasma | | | Source |
|---------------------|----------------------------------|--------------------|------------------------------|----------------------------|----------------------|--------|-----|-----|------------|
| | | | | | | Na | K | Cl | |
| HS+H ₂ O | 100 | 42.9 | - | - | 88 | - | - | - | Deyhim & |
| HS+KCl (0.5%) | 168 | 42.9 | - | - | 97 | - | - | - | Teeter |
| HS+NaCl (0.39%) | 135 | 42.9 | - | - | 93 | - | - | - | (1991) |
| Control (TN) | 100 | - | 34 | - | - | 150 | 5 | 114 | Belay & |
| HS+H ₂ O | 178 | - | 64 | - | - | 136 | 3.9 | 121 | Teeter |
| HS+0.75%KCl | 191 | - | 69 | - | - | - | - | - | (1993) |
| HS+H ₂ O | 100 | 42.6 | - | 100 | - | - | - | - | Smith |
| HS+0.48%KCl | 116 | 42.5 | - | 101 | - | - | - | - | (1994) |
| HS+0.376%NaCl | 122 | 42.5 | - | 104 | - | - | - | - | |
| HS+H ₂ O | 100 | 44.2 | - | 100 | - | 142.1 | 5.6 | 106 | Ait-Boulah |
| HS+0.3%KCl | 107 | 44.3 | - | 107 | - | 142.2 | 5.8 | 107 | Sen |
| HS+0.6%KCl | 117 | 43.8 | - | 107 | - | 141.3 | 6.2 | 107 | (1995) |
| HS+0.9%KCl | 132 | 43.6 | - | 92 | - | 142.0 | 7.0 | 110 | |

HS = Heat stress environment; TN = Thermal neutral environment.

The body response to KCl- drinking water is probably due to the fact that an elevation of plasma potassium has been shown to cause vasodilation (Haddy, 1977). The vasodilating property of K⁺, coupled with the increased the water intake, would lead to a higher fluid retention in the peripheral vascular volume during heat stress.

Acid-base balance

Panting by poultry allows them to increase their evaporative heat loss during heat stress. This increased respiratory rate causes changes in the gas exchange and acid-base balance within the poultry which is due to the loss of carbon dioxide from the respiratory system. The effect of these reactions is a high level of blood pH. There is also a relative loss of bicarbonate ions and hydrogen ions which creates a situation of respiratory alkalosis. During heat exposure, a decrease in plasma potassium and increase in plasma chloride concentration has been reported. These variations in electrolytes, which alter

acid-base balance, also influence the metabolism of amino acid including glutamic acid. Potassium reduction causes increase intracellular accumulation of basic amino acid as well as glutamic acid have been proposed to be an important role in metabolic regulation of acid-base balance (Austic and Calvert, 1981). However, many methods were designed to look at the effects of correcting this acid-base imbalance. Dietary manipulations have been carried out which may enable the bird to cope more completely with acute or chronic heat stress. Ammonium chloride (NR₄Cl) treatment of blood alkalosis and supplementing birds with bicarbonate (HCO₃⁻) for the reduction of blood HCO₃⁻ concentration have been utilized during heat stress (Table 7).

Table 7. Effects of NH₄Cl and NaHCO₃ supplementation on heat-stressed poultry

| Treatment | Feed | Water | Blood pH | Mortality (%) | Sources |
|---|----------------------------|----------------------------|----------|---------------|--------------|
| | consumption (% of control) | consumption (% of control) | | | |
| HS+H ₂ O (control) | 100.0 | 100.0 | 7.82 | 16 | Branton |
| HS+0.63% NH ₄ Cl | 86.5 | 99.2 | 7.50 | 11 | Et al., 1986 |
| HS+3.10% NH ₄ Cl | 73.8 | 19.7 | 7.60 | 49 | |
| HS+0.32% NaHCO ₃ | 95.9 | 105.0 | 7.84 | 24 | |
| HS+0.63% NaHCO ₃ | 93.2 | 123.5 | 7.85 | 8 | |
| HS+H ₂ O on litter (control) | - | 100 | - | 35 | Deaton, |
| HS+1.26% NaHCO ₃ on litter | - | 184 | - | 6 | 1987 |
| HS+H ₂ O on wire (control) | - | 96 | - | 85 | |
| HS+1.26% NaHCO ₃ on wire | - | 176 | - | 50 | |

The study of Branton and co-workers (1986) on broilers which were 42 to 45 days of age exposed to high ambient temperature (40.6°C, 33% RH) for 4 h showed that blood pH of birds increased during the heat exposure period. Blood pH of birds was substantially lowered by consumption of NH₄Cl, while consumption of NaHCO₃ did not affect blood pH. The treatment of a 3.1% level of NH₄Cl during heat stress has been shown to markedly reduce water consumption and increase mortality proportionately. The results of these experiments indicate that increased water consumption is associated with reduced mortality and decreased water consumption, associated with increased mortality. However, the study of Deaton (1987) showed that the addition of NaHCO₃ to the water increased water consumption markedly. Mortality was not reduced by this increased water consumption if the poultry were not on a litter floor. It has been suggested that the birds are able to burrow into the litter and dissipate their body heat through conductive mechanisms. This may indicate that acid-base balance becomes rate limiting for both growth and survival when water consumption is equalized.

Ascorbic acid supplementation

It is known that acute heat stress hugely increases the release of adrenal medullary hormone and catecholamine from the sympathetic nervous system in both poultry and mammals. These compounds cause rapid release from body reserves of glucose, primarily by glycogenolysis. This alarm reaction produces available energy to elude a stressor. The sudden onset of very hot ambient temperature creates such a situation. In acute heat stress the poultry has not had sufficient time to adapt to the hot ambient temperature which may be followed by a resistance stage. If the adrenal cortex is depleted (by exhaustion) before the stressor is removed, then death will occur. All species of poultry are usually capable of ascorbic acid synthesis and dietary supplementation with ascorbic acid is not necessary when the animals are managed properly (Pardue and Thaxton, 1986). It has been reported that the decrease in adrenal ascorbic acid concentration coincided with adrenocortical insufficiency during heat stress. The proposed mechanism for this effect is through inhibition of the activity of the hydroxylase enzymes (21-hydroxylase and 11-beta hydroxylase) by ascorbic acid in the steroid (corticosterone) biosynthetic pathway. This caused reduced synthesis of corticosterone and possibly some increase in androgen synthesis.

The net effect would be to prevent adrenal cortical depletion. The continuing production of corticosterone still occurs during heat stress. Ascorbic acid supplementation has been shown to reduce heat stress related mortality in poultry (Table 8). Studies by Pardue et al. (1985) indicated that mortality was influenced by ascorbic acid supplementation. Mortality was reduced by 7% during heat stress after feeding a diet containing 1000 mg/kg (ppm), whereas non-supplemented birds showed 22% mortality during exposure to high ambient temperature (43 °C and 40% RH) (Pardue et al., 1985). These authors suggested that ascorbic acid can alter endogenous synthesis and

secretion of adrenal steroids. Supplemented birds had a mean plasma corticosteroid level of 8.8 ng/ml compared with 18.1 ng/ml in heated chicks. However, the mortality of heat stressed birds has been shown to be partly dependent on the level of ascorbic acid supplemented in the diet (Gross, 1988). When ascorbic acid was included at 0-330 mg/kg in the diet one day before heat exposure, the mortality rate reduced by 40% to 0%. An increased ascorbic acid level greater than 330 mg/kg diet has been reported to increase the mortality. There is still a need for more studies describing the optimized level of ascorbic acid in diet in the heat stressed poultry.

Table 8. Effects of ascorbic acid supplementation on heat-stressed poultry

| Treatment | Body temp. (°C) | Plasma corticosteroid (ng/ml) | Mortality (%) | Sources |
|------------------------|--------------------|----------------------------------|------------------|-------------------------|
| Unheated | 41.4 | 3.2 | 2 | Pardue et al. (1985) |
| Unheated + 1000 ppm AA | 41.5 | 4.9 | 2 | |
| Heated | 44.5 | 18.1 | 22 | |
| Heated + 1000 ppm AA | 44.7 | 8.8 | 7 | Gross (1988) |
| Heated | - | - | 40 | |
| Heated + 220 ppm AA | - | - | 12 | |
| Heated + 330 ppm AA | - | - | 0 | |
| Heated + 660 ppm AA | - | - | 31 | |
| Heated + 1100 ppm AA | - | - | 15 | |

Poultry housing facilities

It is known that good quality house leads to healthy birds and productive housing. An importance of adequate poultry housing is the place to begin an effective heat stress management. Quality characteristic relate to bird comfort include temperature, humidity and air ventilation. Many systems are recommended to use to reduce heat stress in poultry (e.g., fans in house to facilitate air movement, evaporative cooling system, water spraying, sprinkling, dripping and proper light etc.). However, there is no such item as a perfect poultry house (Vest, 1997). Reducing heat stress need to first understand how birds cope with high temperatures before they can

effectively control their house environment. A thorough understanding of the physiological and anatomical basis of bird for attempting the management alterations is necessary. The appropriate housing facilities relating to biological approach can be used to reduce internal body temperature of birds. The anatomy of chickens should be examined, structures are found on their head especially in turkeys that help alleviate heat stress. Featherless area, e.g., comb, waddles, some areas of shank, breast and legs are effectively used for heat dissipation to the exterior body surface. It is necessary to have a knowledge, how much increase in the humidity corresponds to a unit temperature increase, how much temperature drops in case of wind is

given, how much change in the temperature is given turn off the light and so on.

Conclusion

Heat stress is caused by a combination of environmental and animal factors that result in reduced performance (growth rate, feed efficiency, survivability) as the animal attempts to cool itself. Management for increased survivability during heat stress should elevate heat dissipation and/or reduce heat production. Methods and consideration for selection of management for high heat tolerance in poultry still remain to be found. Therapeutic approaches should seek to impact bird thermobalance in a manner consistent with stress severity. Basic knowledge concerning the normal functioning of temperature regulation in poultry must be gained before its mode of action in heat stress can be fully understood and the full potential of production exploited.

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