# NUTRITIONAL AND WATER BALANCE CONSIDERATIONS FOR DEVELOPMENT OF BROILER HEAT DISTRESS THERAPEUTIC TECHNOLOGIES

By

TSEGAW BELAY

Bachelor of Science
Haile Sellasie I University
Addis Ababa, Ethiopia
1977

Master of Science
Addis Ababa University
Addis Ababa, Ethiopia
1981

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Thesis Approved:

Robert Teeter
Thesis Adviser

Charles V. Maxwell

Dan S. Buchanan

Howard E. Conklin

Norman N. Durham
Dean of the Graduate College

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# CHAPTER I

## INTRODUCTION

Both ambient temperature and relative humidity vary with ecological zone and season. In the tropics and during the summer in temperate zones elevated ambient temperature-relative humidity reduces poultry productivity creating substantial economic loss. During such distress productivity of domestic animals is determined by the successfulness of a complex array of physiological compensatory responses aimed at restoring and maintaining homeostasis. Bird failure to maintain homeostasis is responsible for the production decline.

Under high ambient temperature distress the bird's heat content increases. This occurs largely as the result of a reduced temperature gradient between the bird's body and environmental temperatures. Such a heat exchange disturbance imposes a distress to which the bird must respond to preserve homeostasis. Responses include reduced feed consumption (Squibb et al., 1959; Adams et al., 1962; Teeter et al., 1985; Howlider and Rose, 1989), increasing surface area by postural changes (Freeman, 1971), increasing respiration rate and hence water evaporation (Kleiber, 1961; Freeman, 1966; Jukes, 1971), increasing urine production

(Van Kampen, 1981) and metabolic alteration to reduce heat production (Van Kampen, 1977).

However, the physiological responses themselves are not without cost. Increased water loss through respiration and urine leads to body water pool disturbance (Van kampen, 1981). Hyperthermia induced panting alters arterial carbon dioxide and bicarbonate precipitating respiratory alkalosis (Arad and Marder, 1983; Teeter et al., 1985) which independently reduces feed consumption, growth rate and survivability (Teeter et al., 1985). Heat distress birds exhibit increased potassium excretion (Smith and Teeter, 1987a).

Therapeutic development aimed at offsetting heat distress consequences through electrolyte supplementation have been partially successful. Such therapies have multiple actions by improving acid base water and mineral balance simultaneous. Specific ion effects of electrolytes are frequently confounded with such responses making cause and effect evaluation a difficult task.

Numerous dietary manipulations also have been evaluated for efficacy to minimize heat distress consequences. Rations formulated to reduce dietary heat increment by fat addition (Dale and Fuller, 1979; Valencia et al., 1980) and reducing dietary protein (Waldroup et al., 1976) have been documented to improve broiler growth rate during heat distress. Such manipulations potentially reduce the birds heat load, waste heat and result in greater energy

availability for tissue synthesis. However, such manipulation can cause increase overall energy consumption thereby elevating the birds heat load. Definition of such responses is needed so that the risk benefit ratio may be evaluated.

Broilers susceptibility to pathogens is evaluated during high ambient temperature distress (Freeman, 1988). During heat distress or water deprivation birds increase output of adreno corticotrophic hormone leading to increased corticosteroid hormone levels. Corticosterone decreases the spleen, thymus and bursa of fabricius size thereby reducing circulating lymphocytes (Ben Nathan et al., 1976). During heat distress when bird body heat load is increased, antibiotic supplementation can be beneficial as a means to minimize challenges and resulting heat production.

The study reported herein was conducted to further refine the knowledge base regarding heat distress effects on broiler mineral balance and water balance, to evaluate ration caloric density and calorie protein ratio effects on growth rate and carcass composition, and to evaluate antibiotic efficacy for reducing heat distress consequences.

Chapters are prepared as manuscripts in the style required by Journal of Poultry Sciences to facilitate publication of experimental results.

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# CHAPTER II

# REVIEW OF LITERATURE

## Introduction

In birds and other homeotherms heat distress, characterized by elevated body temperature, occurs when heat production exceeds the bird's ability to dissipate heat. Heat distress occurs most frequently in the tropical and sub-tropical regions, but is also a seasonal problem in temperate climates. Husbandry strategy for minimizing the deleterious effects of heat distress is to reduce heat gain or increase heat loss by provision of suitable environmental modification. An alternative, or additional strategy is to exploit biological relationships for reducing bird heat production and or increasing heat dissipation extent and or efficiency. Improving bird thermobalance during heat distress through various biological manipulations is the subject of this review.

## Thermobalance

Bird thermobalance, is determined by summation of heat produced and lost. Avian species, like most mammals, are homeotherms and must consequently maintain deep body temperature relatively constant over a wide range of ambient

Add reference of Respiration How to do °C



ces (Meltzer, 1987). The deep body temperature of chickens is generally higher than mammals, being in of 41-42 vs 38 °C (Freeman , 1965). Under most conditions, there is a flow of heat from the bird vironment by radiation, convection, conduction and poration. The relative importance of these routes with environmental conditions (Freeman, 1971). omobalance has been quantified by Sturkie (1986)

$$H = E \pm R \pm C \pm K \pm S$$

lic heat production, E= evaporative heat loss, R= by radiation, C= heat loss by convection, K= heat conduction and S= body heat content. The amount of stored body heat is dependent upon both body temperature and its specific heat. The quantity of heat gain or loss is estimated as: S=delta T x body mass x 3.5

Where;

S= heat content change (kJ), delta T= body temperature change (°C), and 3.5= the mean specific heat of the body tissues (kJ/kg.°C).

Heat change measurements in the domestic fowl as influenced by environment are limited. However, body heat stored content is an important thermoregulatory mechanism, particularly during high ambient temperature, where a rise in body temperature creates a temperature difference between the bird and the environment so that heat is lost from the

body (Sturkie, 1986). Heat losses through radiation, convection and conduction may be lumped together as nonevaporative or sensible heat loss while heat loss through respiratory and cutaneous water evaporation may be referred to as evaporative or insensible heat loss. A discussion of these factors follows:

Heat production

Indirect calorimetry may be used to estimate bird heat production (HP). The methods depends on quantification of oxygen consumption and carbon dioxide production as described by Brouwer (1965) with out correction made for methane and urinary nitrogen.

HP = 16.18 x l oxygen communed + 5.02 x l carbon dioxide produced.

where;

HP = heat produced in kJ, oxygen consumed in liters, carbon dioxide produced in liters. In the equation no correction is usually applied for nitrogen excretion since Rominjn and Lockhorst (1961, 1966) indicated that the error resulting from this omission is about .2 % and should not exceed 1.5 % even at a higher rate of protein catabolism. Heat production measurement by indirect calorimetry has been used to determine feed energy availability for growth and maintenance (Shannon and Brown, 1969; Burlacu et al., 1970a,b) as well as estimate the energy requirement under specific conditions. Bird energy use for maintenance and

production is affected by environmental temperature (Van kampen, 1974; Farrell and Swain, 1977), nutrient deficiencies (Klieber, 1945), diseases (Sykes, 1970) and the dietary ratio of protein to energy (Davidson et al., 1968). Under normal conditions, animals have maximum energy utilization efficiency for growth when heat losses associated with maintaining normal physiological process are minimal. In this text the effect of ambient temperature on forms of heat loss the bird undergo for regulating body temperature will be reviewed. Due to the variation in metabolic rate between large and small birds, heat production values are expressed per unit metabolic body weight (Kg $BW^{\cdot 75}$) (Luiting, 1990) to minimize variability and make values largely independent of body weight. The relationship between ambient and body temperature with heat production is displayed in figure. 1. The temperature range over which heat production is minimal, defined as the thermoneutral (TN) zone. The TN zone is bounded by the upper and lower critical temperatures. Generally, the TN zone is inversely related to bird age (Meltzer, 1983). Heat production is linearly related to caloric intake (Luiting, 1990) and feed consumption (Van Kampen, 1977; Wiernusz et al., 1991). Per unit body weight male broilers have a 24 % higher HP than females (Meltzer, 1983).

Bird heat production is linearly related to the deep body temperature (Van Kampen and Romijn, 1970; Farrell and Swain, 1977) extending over a range of -5 to 40 °C (Romijn

and Vreugdenhil, 1969). The inverse relationship of heat production to ambient temperature (Van Kampen, 1974, 1981a ; Williamson et al., 1985; Chwalibog et al., 1985) may be attributed to reduced feed consumption at higher temperature. Chwalibog and Eggum, (1989) reported that broilers exposed to gradually increasing high ambient temperature had a decreasing oxygen consumption and carbon dioxide production, suggesting that reduced heat production during heat distress could be due to decreased gas exchange. In contrast, during low ambient temperature exposure heat production is elevated so that body temperature may be maintained (figure.1).

Modulating Bird HP:

Thyroid hormones are involved in modulating bird HP and hence body temperature (Yousef, 1985). Kittok et al. (1982) observed increased sensible heat loss in adult chickens following administration of triiodothyronine ($T_3$) or thyroxine ($T_4$). Oxygen consumption of chickens between 1-8 weeks of age is correlated with the circulating concentration of $T_3$ but not $T_4$ (Bobek et al., 1977; Klandorf et al., 1981), suggesting that $T_3$ is the metabolically active thyroid hormone. Secretion of $T_3$ is reported to be dependent on ambient temperature (Freeman, 1983). During cold exposure $T_3$ secretion and HP is increased (Kuhn and Nouwen, 1978). In contrast at higher ambient temperatures basal heat production is overshadowed by elevated HP with

increased respiration rate (Robertshaw, 1981). However, basal heat production declines with $T_3$ secretion (Williamson et al., 1985; Mitchell et al., 1986). Modulating $T_3$ secretion is a homeostatic mechanism used to reduce heat distress consequences. Indeed, feeding $T_3$ or $T_4$ to 6-8 week old broilers exposed to heat distress reduced survival time while thiouracil, a blocker of T3 secretion, enhanced bird survival time when exposed to similar heat distress (Fox, 1980; May, 1982). Blood $T_3$ and $T_4$ concentration is influenced by nutrient intake (klandrof et al., 1981), raising the possibility that such responses observed in heat distressed birds are related to feed consumption.

Plasma corticostrone is elevated during acute cold distress and is accompanied by elevated heat production (Freeman, 1982), although similar response was not reported by Davison et al., (1980). Acute heat exposure also increases serum corticostrone in chickens (Ben Nathan et al., 1976; Siegel and Gould, 1982; Mitchell et al., 1986; Deyhim et al., 1990). Corticostrone has been documented to protect against the lethal effects of high ambient temperature (Sammelwitz, 1967). Apparently the effect is mediated through reducing the circulating levels of $T_3$ and hence HP. However, according to Edens (1978) elevated corticostrone during acute heat exposure is short lived, since progressive heat exposure is accompanied by lower adrenal corticosteriod concentration, hypoglucosemia and, alkalosis along with elevated release of catecholamines.

Nonevaporative heat loss

Body heat loss by means of radiation, convection and conduction is referred as nonevaporative heat loss (Yousef, 1985). Nonevaporative heat loss (NHL) may be estimated as (Sturkie, 1986):

$$NHL = H - EC \pm S$$

where;

H= heat production, EC= evaporative heat loss, S= heat body content.

All poultry classes utilize NHL as the major means of heat dissipation when housed below and within the thermoneutral ambient temperature environments (Arieli et al., 1980; Van kampen, 1981b). According to Romijn and Lokhorst (1966), nonevaporative heat loss represents about 75% of total heat loss at the thermoneutral environment heat loss from the head appendages has been estimated to range from 9.3 to 25.6% of total heat production at environmental temperatures from -5 upto 40°C (Van Kampen, 1974). As the ambient temperature falls below the lower critical temperature heat loss can exceed production and the bird must increase its metabolic rate to prevent a body temperature fall (Freeman, 1971).

Decreasing water consumption and hence urinary output during cold distress conserves body heat (Van Kampen, 1981a). At the same time physical thermoregulatory mechanisms like covering the legs by sitting and fluffing

out feathers to increase insulatory protection will be used to minimize the metabolic effort during cold. The amount of feathering is an important determinant of heat production at thermoneutral environmental temperature (O'Neill et al., 1971). At a temperature of 20°C a fully feathered bird produces heat at half the rate of a naked bird, making feathered birds more efficient in feed utilization. Arterial vasoconstriction reduces heat loss from the extremities and hence reduced countercurrent heat exchange (Van kampen 1981b). According to Van Kampen (1981b), such cardiovascular modifications during cold may reduce the sensible heat loss from the head and legs by 15 to 20%. If these responses are inadequate heat production via shivering increases largely (Freeman, 1988).

As ambient temperature increases the temperature gradient between the bird and the environment declines with the result that nonevaporative heat is reduced and a reversal of the responses observed during cold distress will occurs. Further thermal insulatory effectiveness of the feathers is enhanced by posturally increasing effective surface area (Freeman, 1971). Vasodialation during high ambient temperature increase nonevaporative heat loss by reducing peripheral resistance to blood flow and concomitantly increasing resistance to the viscera, thereby shunting blood and hence heat to peripheral tissues (Bottje and Harrison, 1984). Increased blood flow to the comb and wattles is an important mechanism to dissipate core body

heat (Michael and Harrison, 1987). A second, though less important avenue of nonevaporative cooling is increased urine production when water loss is compensated with consumed water having a lower temperature than the bird. However, sensible heat dissipation efficiency declines as ambient temperature rises due to the reduced differential between the bird and ambient temperature (Van Kampen, 1974; Wiernusz, et al., 1991). Consequently, the bird resorts increasingly to dissipating excess heat by evaporative of water loss.

Evaporative heat loss

Evaporative heat dissipation is estimated by coupling grams water evaporation (respiratory and cutaneous water) with the latent heat of vaporization (2.365 KJ/g $H_2O$). Evaporation of water is one of the routes by which the fowl can control its body temperature. Due to its molecular structure and bonding, water has a high latent heat of vaporization. For every gram of water that evaporates approximately 2.4 KJ of heat are lost. Evaporative heat loss takes place through the body surface and the respiratory tract. Since the fowl has no sweat glands, loss in this manner overwhelmingly occurs via the moist surface layer of the respiratory tract to the inspired air which is 'saturated' with water vapor at body temperature (Kerstens, 1964). Evaporative rate is proportional to respiratory rate.

Heat lost through evaporation at lower temperature represents only a fraction, but increases dramatically through 26-35° C where it may contribute as much as 80 % of total heat loss from the body (Kerstens 1964; van Kampen, 1981; Wiernusz et al., 1991). Arieli et al., (1980) estimated 4 mg/Kg.min.°C of evaporative water loss from heat distressed birds housed at ambient temperature of above 26°C, which this represents 8 fold larger than at 2-26°C temperature range. Bird dependency upon evaporative heat loss increases as the ambient temperature exceeds the upper critical temperature. In contrast below the lower critical temperature heat loss through evaporation of water from the skin also takes place but quantitatively represents a small proportion (Van Kampen, 1971; Richards, 1976). As is illustrated in figure. 1, cutaneous water ($E_C$) loss increases in absolute terms as ambient temperature increases but represents only 40% once panting is initiated (Van kampen, 1971) and falls to 15% of total heat loss when the bird is actively panting (Van kampen, 1974).

During high ambient temperature birds increase respiration rate (Michael and Harrison, 1987), as a result of which considerable quantity of water is evaporated from the mucous membranes lining of the upper respiratory tract and to provide the water loss for evaporation, blood flow through the carotid artery, which serves the upper respiratory tract is enhanced (Freeman, 1984). Panting is thus the main route for dissipating heat during high ambient

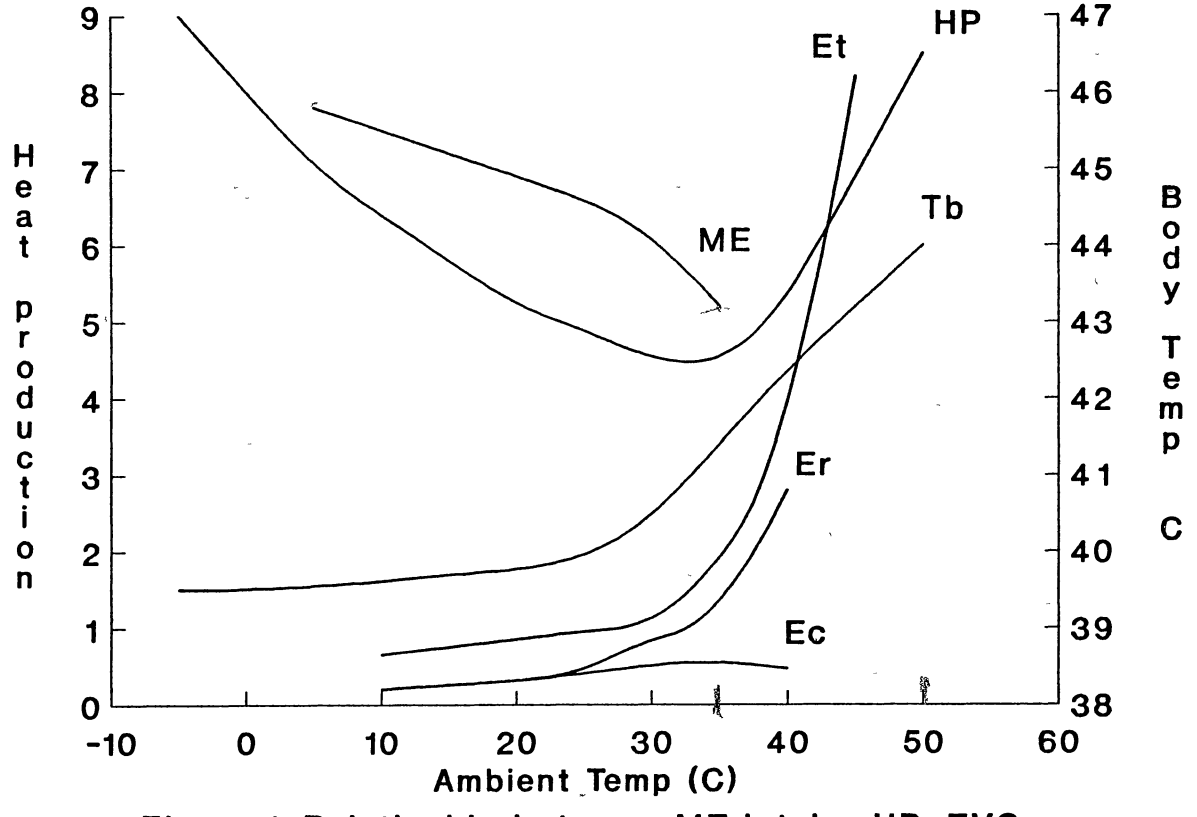


Figure 1. Relatioship between ME intake, HP, EVC (Et, Er, and Ec, Total Respiratory and cutaneous heat loss).

temperature distress. Indeed, heat distressed birds dissipate over 80% of heat production via evaporative cooling (Van Kampen, 1974; Wiernusz, et al., 1991). Respiratory water ($E_r$) loss in this way is minimal until ambient temperature surpasses the thermoneutral zone. The efficiency of evaporative heat loss is influenced by the relative humidity of the surrounding air. Romijn and Lokhorst (1966), reported that at ambient temperature of 34°C and relative humidity of 40 % an adult hen dissipate over 80 % of total heat by evaporative means but this was reduced to only 39 % on increasing the relative humidity to 90% and the bird becomes hyperthermic.

Respiratory evaporative heat loss has linear correlation with respiration rate, this in turn is associated with perturbed acid base balance. Heat distressed broilers have elevated blood pH and reduced $HCO_3^-$ and $PCO_2$ (Marder, et al., 1974; Arad and Marder, 1983; Bottje and Harrison , 1985; Teeter et al., 1985 ; Branton et al., 1986). Respiratory alkalosis is associated with a myriad of physiological changes intimately associated with the electrolytes. Indeed respiratory alkalosis has been reported to increase potassium excretion by over 600 % in broilers (Smith and Teeter, 1987b) and 45.4 % in layers (Deez and Ringers 1976) thereby reducing plasma sodium and potassium (Edens, 1977; Deyhim and Teeter, 1990). As intercellular potassium and $HCO_3^-$ is replaced by hydrogen ion precipitating an intercellular acidosis (Gray, 1989).

Since optimal enzymatic activity for protein synthesis (Stryer, 1981) and nutrient transport (Mongin, 1981) occur within a narrow pH range deviation caused due to hyperventilation may thereby reduce bird productivity.

## Water Metabolism and Balance

Water is the largest single constituent of the body and represents upto 70 % of the adult body weight. Approximately 70 % of the bird's body water is found in the intercellular fluid (ICF) while the remaining 30 % is in the exteracellular fluid (ECF), partitioned between the interstitial space (75 %) and the plasma (25 %)(Freeman, 1971). Water balance and metabolism is related to the maintenance of a dynamic equilibrium within and between these compartments. Under conditions of *ad libitum* water intake, the metabolism and balance of body water is attained. It is necessary to consider the various routes by which water enters and leaves the body of the fowl as variables in these activities affect the different methods by which water balance is achieved.

## Water intake

Drinking water intake of birds on an absolute basis increases with age although its consumption per unit body weight decreases with age (Leeson et al., 1976). Voluntary water intake of birds represents 74% of the total daily intake and the remaining 26 % comes from nutrient metabolism

and consumed feed (NRC, 1981). Drinking behavior is associated with hypothalamic control centers in the brain (Wagner, 1964). Since water intake is correlated with feed intake, factors affecting feed consumption indirectly affect water intake (Zeigler, et al., 1971). High levels of dietary constituents such as molasses (Ross, 1960) and salt (Herrick, 1971; Teeter, 1988) are known to stimulate water consumption.

Ambient temperature has long been recognized to affect water consumption of laying hens (Van Kampen, 1981) and broilers (Leeson et al., 1976). As ambient temperature increases from 21 to 37 $^{\circ}$ C, water consumption increases by 250 % (NRC, 1981). Budgell (1970) proposed three hypotheses to describe the relationship between high ambient temperature and increased water consumption, including 1. dryness of the orophoryngeal receptors, 2. systemic dehydration and 3. hypothalamic alteration such as increased antiduretic hormone secretion associated with elevated ambient temperature and dehyderation. At cold temperature, water intake in turkeys (Parker et al., 1972), laying hens (Van Kampen, 1981) and broilers (Leeson et al., 1976) was reduced. However, hens reduce urine output during cold (Van Kampen, 1981) to compensate for the reduced intake.

Drinking Water temperature

Miller and Sunde (1975) suggested that layers are more

heat distress tolerant when they consume cool drinking water. Similarly Teeter et al. (1987) reported that heat distressed broilers offered drinking water maintained at 15.6° C had greater growth rate compared to those offered water at higher temperature. They suggested that, cold water may serve as heat sink enabling the bird to lower body heat load and increase its feed consumption. Salt addition to drinking water maintained at the bird's body temperature reduced growth rate. Fox (1951) hypothesized that, infusing a volume of cool water in the crop, which is in close proximity to the main arteries supplying the head region, could have a cooling effect on blood going to the brain, and possibly venous blood returning from the wattles and comb. Such a cooling effect could prevent what is believed to be the major cause of death from heat prostration, namely paralysis of the brain respiratory centers.

Bianca (1964) reported differences in respiratory rate of oxen housed at 40° C when offered water at 14 or 40° C. Drinking cold water immediately decreased respiration rate from 130 to 40 respirations/min while animals consuming hot water increased their rate from 130 to 180/min. Hydrated birds exposed to high ambient temperature (44° C) had lower metabolic heat production and higher cooling efficiency (evaporative heat loss/ total heat production) than the dehydrated birds (Arad, 1983). Providing cold water to heat distressed broilers to minimize hyperventilation, thereby preventing respiratory alkalosis, and/or increasing water

consumption through drinking water salt supplementation becomes an important management criteria that require consideration.

Metabolic water

Metabolic water is produced from fat, carbohydrate and protein oxidation at the rate of 1.18, .6 and .5 gram of water/g of nutrient, respectively (Kerstens, 1964). Total metabolic water can be estimated from caloric intake since approximately 0.135 gram of water is produced per oxidized Kilocalorie (Kerstens, 1964). Metabolic water enters the body pool and represents about 15 % of total water intake. Because of the higher metabolic rate compared to humans the amount of metabolic water produced by birds is more as expressed in relation to body size (Mullkey and Huston, 1967). However, the contribution of metabolic water to the overall body water pool expressed as a percentage decreases as the ambient temperature increases since water consumption also increase (Van Kampen, 1981). Van Kampen (1981) modified a formula derived for mammals by van Es (1969) to calculate metabolic water production in layers as follows:

$$H_2O = 0.181 \times 1\ O_2 + 0.49 \times 1\ CO_2 + 0.232\ N,$$

where;

$H_2O$ is metabolic water produced (g), $O_2$ is oxygen consumption, $CO_2$ is carbon dioxide production and N is urinary nitrogen (g). Van kampen suggested that ignoring N

in the formula would result in less than 2 % metabolic water production underestimation.

Water loss

Water excretion in the feces and urine varies with water intake. Broilers produce excreta containing 60-70 % moisture (Kerstens, 1964), while layers excreta contains 80 % moisture (Anderson and Hill, 1968). Under thermoneutral environments laying hens produce 29 to 35 ml urine per kg body weight of urine daily (Dicker and Haslam, 1972; Isshiki, 1985). Urine production of laying hens increased 200 % during heat distress (Van Kampen, 1981a).

During high ambient temperature distress, due to elevated respiration rate, water loss through respiratory evaporation contributes a substantial quantity. Table 1 shows the significance of evaporative water loss in broiler chicken at various ages housed at thermoneutral environment and indicates an approximate loss equivalent to drinking water intake.

Water balance

Under normal physiological conditions for the adult birds, water intake and output are controlled to maintain body water pools. A positive water balance is found in the growing bird commensurate with tissue deposition (Table 1). Lopez et al., (1973) recorded water retaintion values of 57 and 76 % of body weight for 7 year old hen and 5 month-old

Table 1. Water metabolism in different age group of broiler chickens (from Leeson et al., 1976).

| Age (wks) | Environment temp. (°C) | Relative humidity (%) | Drinking water intake/d (g) | Water intake in feed (g) | Metabolic body water/d (g) | Water retained by growth/d (g) | Water excreted in urine & feces/d (g) | Evapor. water loss/d (g) |
|---|---|---|---|---|---|---|---|---|
| 1 | 31 | 70 | 11.4 | 1.5 | 3.1 | 4.6 | 4.1 | 7.3 |
| 2 | 25 | 70 | 11.8 | 2.4 | 5.0 | 6.7 | 6.3 | 6.2 |
| 3 | 25 | 70 | 21.4 | 3.6 | 7.4 | 10.2 | 9.6 | 12.6 |
| 4 | 23 | 70 | 48.8 | 4.8 | 9.3 | 15.0 | 12.8 | 35.1 |
| 5 | 22 | 70 | 72.3 | 6.1 | 12.7 | 17.1 | 16.1 | 58.0 |
| 6 | 20 | 75 | 90.2 | 7.4 | 17.7 | 15.9 | 19.4 | 80.0 |
| 7 | 20 | 79 | 109.3 | 8.3 | 22.4 | 12.8 | 21.8 | 105.3 |
| 8 | 20 | 67 | 114.7 | 9.0 | 21.3 | 20.4 | 23.9 | 100.8 |
| 9 | 20 | 67 | 127.6 | 9.8 | 25.9 | 16.1 | 25.7 | 121.5 |

pullets, respectively. This effect may be related to increased fat deposition with age, since body fat and body water are negatively correlated (Chwalibog and Eggum, 1989) or due to increased proportions of other tissues of low osmotic activity such as collagen and bone (Cunningham and Morrison, 1975) in older bird.

Water balances is achieved by the maintenance of an equilibria between the two water compartments of the body, i.e. intercellular (IC) and exteracellular (EC). The primary determinant of the distribution of water in these compartments is the number of osmotically active solute consisting principally of sodium and potassium (Best and Taylor, 1990). Sodium is the major EC osmotically active cation with potassium being the predominant IC ion. Osmotically active sodium and potassium in the body fluids is closely approximated by the exchangeable sodium ($Na+_e$) and potassium ($K+_e$) pool, respectively. The major exchangeable ions $K^+$ and $Na^+$ in the body fluids represents over 70 % and 90 % of the total body content respectively. In contrast to what are found in the bone. However, sodium being quantitatively the major electrolyte in plasma, a relationship between plasma sodium ($P_{Na}$), exchangeable potassium and total body water (TBW) is given by Edelman et al.(1958) as follows:

$$TBW = (Na_e^+ + K_e^+)/P_{Na}$$

Plasma osmolarity changes with the sodium concentration and has been used to approximate the plasma volume (Best and

Taylor, 1990). In conditions where ECF osmolarity is higher than the ICF or vise versa, there will be a net shift of water between the two compartments (Tosteson and Hoffman, 1960). Barragry (1974) stated that the principal cause of a water or fluid shift between the extra and intra-cellular fluid is due to a change in composition of the former. Although a simple loss of extracellular fluid does not markedly affect the intercellular fluid as its ionic composition remains relatively unchanged despite its volume is reduced. Water movement across the cell membrane is initiated by changes in the osmotically active electrolyte concentration of the intra-and extra-cellular fluids, such that the initial equilibrium with respect to osmolarity is achieved. Such changes in osmolarity may be due to loss of either water or electrolytes. Indeed hemodilution evidenced during heat distress chickens (Vo et al., 1978) parallels elevated potassium excretion (Smith and Teeter, 1987) and decreased plasma sodium and potassium ( Deetz and Ringrose, 1976; Edens, 1977; Deyhim and Teeter, 1990).

Secretion of arginine vasotocin (AVT) hormone depends on plasma electrolyte concentration (Bottje et al., 1990). During heat distress birds have decreased plasma electrolyte, circulating plasma AVT will be insufficient to enhance water reabsorption by the renal collecting tubules and water will be lost in the urine. The depletion of carcass sodium and potassium (Sharma and Gangwar, 1987) and reported higher carcass dry matter of heat distressed

broilers (Chwalibog and Eggum, 1989) is likely associated with the dehydration occurring as the result of increased water output and lower water retention.

Effect of Heat Distress on Mineral Balance

Broilers exposed to 40 C ambient temperature distress were found to have reduced skeletal growth as evidenced by shank to toe length shortening compared to thermoneutral controls (Williamson et al., 1985). Heat distress affects phosphorous utilization and subsequently influences leg disorders (Orban and Roland, 1990). Similarily, problems related to leg weakness in broilers during the summer months have been reported (Reece et al., 1971). Higher incidence of leg problems in heat distress broilers was due to reduced bone weight and strength (Siegel et. al., 1973; Ernst et al., 1984) similar in turkeys (Magruder and Nelson, 1967). According to Seigel et al. (1974), heat distress reduces breaking strength and mineralization of long bones in broilers. In addition, heat stressed chicks exhibited signs of muscular weakeness, paralysis and at time of death, extreme muscle tetany (Gillis, 1948). Suggesting that heat distress could predispose chicks to hypokalmia.

Broilers exposed to a 32$^{\circ}$C ambient temperature for 42 days had lower calcium (Ca), copper (Cu), iron (Fe), potassium (K), magnesium (Mg), manganese (Mn), sodium (Na), phosphorus (P), and zinc (Zn) retention (Husseny and Creger, 1981). The reduced retention efficiency was accompanied by

a lower carcass concentration of each elements. Sonaiya, (1989) reported that heat distressed broilers have lower total ash compared to their counter parts in thermoneutral environment, similar to Sharma and Gangwar (1987) who reported lower sodium and potassium content in breast and thigh muscles of heat distressed broilers. However, specific ambient temperature effects on blood mineral concentration have been mixed.

Mineral balance greatly depends on the bird's physiological condition. During high ambient temperature-relative humidity distress birds develop respiratory alkalosis (Teeter et al., 1985) reducing competition between hydrogen and potassium for transport into the distal convoluted tubules. As such excretion favors potassium since the hydrogen ion concentration is reduced (Harper, et al., 1977) thereby depleting intracellular potassium (Best and Taylor, 1990). Coupling increased excretion with a reduced feed consumption at higher ambient temperature distress could precipitate mineral deficiencies.

Kohne and Jones (1975) exposed turkeys to acute hyperthermia and observed that birds to develop a profound alkalosis and an increased plasma potassium concentration. In contrast Huston (1978) subjected female chickens up to 30$^{\circ}$C and observed an inverse relationship between ambient temperature and blood potassium concentration as was recently reported by Deyhim, et al. (1990). Simmons and Avedon (1959) suggested that intercellular potassium could

buffer extracellular potassium concentrations during acid-base perturbation and mask the excretion effect. Potassium in the plasma increases during the development of heat prostration (Eden, 1975). This increase is highly suggestive of cell damage with a resultant leakage of the potassium into the general circulation. Such a mechanism may lead to a drain of both intercellular and extracellular potassium during heat distress and account for the variability associated with plasma potassium measurements during heat distress. In addition when plasma potassium levels are increased by leakage from muscle cells, nerves and/or kidney failure nerve transmission and normal cardiovascular function are disrupted. Indeed cardiovascular system failure has been suggested to cause heat prostration (Eden, 1977).

Heat distress induced perturbation of bird mineral balance has been associated with decreased plasma sodium, calcium, magnesium, and inorganic phosphorus concentration in turkeys (kohne and Jone (1975). More specifically ionic calcium pool is inversely related to the blood PH. During respiratory alkalosis elevation in blood pH reduces the ionized blood calcium pool by increasing the concentration of complexed calcium thereby restricting the calcium availabile for egg formation (Mueller, 1959; Odom et al., 1986). The authors suggested that compensation for the blood alkalosis elevates blood organic acids, pyruvate and lactate which will bind the calcium and make it unavailable.

Reduced plasma inorganic phosphate and calcium are also known to occur in broilers during heat distress (McCormick and Garlich, 1982).

Heat distress reduced calcium, phosphorus and magnesium retention in hens to the extent resulting negative balance (Kamar et al, 1987). According to them the negative balance for these elements is associated with increased excretion of calcium and magnesium via the excreta while that of phosphorous was mainly through the urine. Similarily, Wolfenson et al., (1987) reported reduced potassium, phosphorous, and calcium absorption in turkeys and suggested that heat distressed causes decreased intestinal absorption of these elements.

Plasma inorganic phosphate in broilers is reduced immediately upon exposure to the high temperature environment and it was noted that there is an apparent relationship between plasma inorganic phosphate and plasma sodium (Edens, 1976b). According to him a decrease in plasma sodium caused a respective decrease in phosphate and suggested that during heat distress sodium is excreted via the kidney and phosphate is carried along passively.

Plasma sodium is also reduced upon exposure of broilers to heat distress (Edens, 1976b; Deyhim, et al. 1990 ). Since sodium is involved in bird thermoregulation, causing an increase in hypothalamic body temperature setpoint, the loss may be an attempt to decrease body temperature (Edens,

1976b). However, the loss of plasma sodium may be indicative of sodium loss from other body tissues as well.

Increased plasma alkaline phosphatase, a significant portion of which originates from the intestine and some from bone forming cells, is associated with increased osteoblastic activity (Bourine, 1972). Decline of this enzyme in the plasma consequently leads to a general reduction in the anabolic rate of bone (Bell, 1971). An explanation for the reduced mineralization and breaking strength of the long bones from birds reared at high ambient temperature may be attributable to lower alkaline phosphatase, which is depressed during heat distress in broilers (Seigel et *al*. 1974). Bide and Dorward (1970) reported that plasma alkaline phosphatase was reduced by 50 % during 48 hours of starvation in broiler chicks, thus during heat distress when feed consumption is reduced there is likely an impaired mineral metabolism due to reduced intestinal activity.

Fisher and Boorman (1986) recommended adjustments for mineral requirement of poultry according to the prevailing ambient temperature by increasing 1.5 % of the requirement for each $^{\circ}$ C above 16$^{\circ}$ C. Broilers subjected to 35$^{\circ}$C pair fed to mimic thermoneutral controls, excrete 27.3% more potassium (Smith and Teeter, 1987b) similar to observations made by Deetz and Ringrose (1976), for hens housed at 37.7$^{\circ}$C. Compensating evaluated potassium excretion in heat distress was evaluated by Smith and Teeter (1987b).

According to their study increasing dietary potassium above the thermoneutral requirement for heat distressed broilers increases gain, suggesting the need for evaluation of mineral requirements in heat distressed broilers.

## Separate Urine and Feces Collection in Metabolism Studies

Colostomized birds have been widely used in experimentations involving water and electrolyte balance (Van Kampen, 1981a; Lee and Campbell, 1983; Isshiki, 1985), nitrogen and amino acid metabolism (Gruhn and Henning, 1980; Okumura et al., 1981).

In the domestic fowl the cloaca forms a common duct for the digestive, urinary and reproductive system. Studies with any of these systems will be interfered with excretory products of the other two. In particular, nutrition studies often require the collection of uncontaminated feces from the digestive system and or urine from the urinary system. Therefore, metabolism studies frequently necessitate separate collection of urine and feces. Non surgical methods for separate collection of urine and feces have involved the use of funnels designed to fit into the interior cloaca (Pitts, 1938) or cannulation of the ureters (Hester, 1940). These have been found useful for short term experiments and may have undesirable side effects such as increased urine flow.

Surgical modifications and/or use of mechanical devices to separate urine and fecal excreta has been recommended

(Paulson, 1969). Non surgical techniques such as plugging the rectum and attaching catheters in or around the urethral orifices have been suggested for excrement separation (Davis, 1927, Caulson and Huges 1930). Fussell (1969) reported, that the use of such non-surgical techniques for extended periods of time are unsatisfactory due to the high degree of urethral irritation.

A surgical techniques for exteriorizing the urethral orifices for short term experiments in growing birds has been described (Hester et al. 1940; Newberne et al. 1957; Ainsworth, 1965 ). However, Richardson et al. (1959) and Fussell (1969) reported that surgically exteriorized ureters were frequently associated with suture breakdown, irritation to the bird, and excreta contamination.

Isolation of the rectum from the cloaca and its diversion through the abdominal wall has been described (Rothchild, 1947) though details for post operative conditions and subsequent bird performance were not discussed. Fussell (1960), described a long-term metabolism study in which one year old layers weighing over 1.5 Kg were used for separate collection of urine and feces. Fussel later (1969) discussed how breed, species, sex and age differences might affect operational suitability and recommended that birds weighing less than 0.75 kg not be used. Several studies utilizing adult or nearly mature birds includes Ariyshi and Morimoto (1957), Okumura (1976) and Isshiki (1985).

Polin et al., (1967) utilized 4 week old white leghorn cockerels, but reported colostomy blockage and body weight loss. Post operative problems also include narrowing of the colostomy site and constipation (Ariyoshi and Morimoto, 1957; Richardson, etal., 1960; Colvin, et al., 1966; Okumra, 1976). Richardson et al (1959) and Fussel (1969) suggested that placing a glass cannulas within the colostomy stoma will dilate the strictured bowel . Okumura (1976) used a polyvinyl chloride (PVC) cannula inserted into the stoma on the tenth postoperative day. While Gunnsey and Johns (1986) recommended utilizing a PVC rod. In general reported poultry surgical modification lack procedural details, have been criticized as to bird usability for research and restricted to short term assays with only adult birds.

Feeds and Feeding During Heat Distress

Research reports are documenting ambient temperature effects on calorie requirements of broilers reared at different ambient temperature. Caloric requirements increase in bird housed below the thermoneutral zone as they consume more feed to maintain body temperature. As temperature decreases feed efficiency is reduced, due to nutrient diverting away from gain toward maintenance (Cerniglia et al., 1983). In contrast as the ambient temperature increases approaching the bird body temperature both heat loss and energy requirement declines forcing the bird to decrease nutrients intake, to minimize excess body

heat (Fuller and Mora, 1973; Cerniglia et al., 1983). Deficiencies usually manifested by depressed growth and production. It is in fact recognized that depression of growth rate observed in heat distressed broilers is partially related to reduced feed intake (Squib et al., 1959; Cowan and Michie, 1978).

Conventional approaches regarding ration formulation during heat distress has been to increase nutrient density. However, such an approach seems erroneous since the natural defence mechanism of heat distressed birds is to reduce feed intake to lower its metabolic heat production. Indeed MacLeod et al. (1979) and Wiernusz et al. (1991) demonstrated that heat production of birds exposed to heat distress is elevated with increased feed consumption. Feeding high calorie rations to increase nutrient intake may therefore increase oxygen consumption and metabolic heat production arising both from environment and feed. Indeed, force feeding heat distressed broilers to levels observed by thermoneutral broilers increases mortality (Smith and Teeter, 1987a).

Fat Effect

Few hours after feeding bird's heat production will increase above the level represented by basal metabolism. Such an increase in heat production due to digestion and metabolizing feed is known as heat increment of the diet (MacDonald et al., 1988). Unless the bird is at ambient temperatures below the thermoneutral zone heat increments

from feed have no value that the energy it is lost to the ambiance. However, feed heat increment is deleterious during heat distress since it contributes additional heat to the body. Attempts to reduce the effects of heat distress on broilers by reducing the dietary heat increment have been suggested by adding fat. Performance and feed conversion of broilers was improved by increasing caloric density primarily through fat supplementation (Dale and Fuller, 1979; Valencia et al., 1980). Heat production measured by energy balance was lower in broilers fed diets with a higher ratio of fat to non fat calorie (Dale and Fuller, 1979). The decreased heat increment of a fat supplemented diet resulted in a greater percentage of the diet being available for tissue synthesis in animals maintained at or above thermoneutality (Coffey et al., 1982).

The addition of fat to the diet does not insure positive benefits under extreme heat distress environment as the reduced heat may override the reduced heat increment effect (Dale and Fuller, 1980). According to Sykes and Salih (1986) even under conditions of mild heat distress an increased energy intake with fat addition can lead to loss of heat tolerance in birds since rectal temperature was observed to rise. Therefore, during heat distress feeding high caloric density diets potentially increases the bird heat load and mortality.

Protein Effects

Reducing dietary protein while maintaining essential

amino acids lowers ration heat increment (Waldroup et al., 1976; Baghel and Pradhan, 1989). Sinurat and Balnave, (1985 and 1986) demonstrated that feed intake and feed efficiency of broilers in heat distress improves with increased caloric density and reduced amino acid to calorie ratio. The use of high energy rations with lower protein to form high calorie/protein ratio rations since it reduces heat increment, improves performance of heat distressed broilers. The study of Mcnaughton and Reece (1984) with heat distressed broilers indicate that high protein diet is advantageous for reducing carcass fat but can be deleterious due to higher heat increment.

Nutrient Balance

Fisher and Wilson (1974) reported that the increase in weight gain with increasing caloric density was less pronounced at high temperature than at low. The decrease in feed intake with increasing ration caloric density was less at high temperature. This could be regarded as consistent with the need for concentrated rations at high temperature (Harris et al., 1974). Later Reece and McNaughton (1982) and Abdelkarim et al. (1985) confirmed that the response to increase dietary energy was less pronounced at high temperature. Sinurat and Balnave (1985) suggested that although the response was less pronounced at high temperature it could be exploited during hot weather by increasing dietary energy and at the same time permitting a slight amino acid deficiency so as to stimulate feed intake.

However, nutritionally unbalanced diets will have a larger heat increment (Deaton, 1983). Therefore, it was suggested that higher caloric density responses in heat distressed broilers can only be achieved when essential amino acids such as lysine are provided in adequate levels to minimize deficiency (Mcnaughton and Reece, 1984; Baghel and Pradhan, 1989).

The process of prehension, digestion and metabolizing feed generates heat. The mere presence of feed in the digestive tract during periods of heat distress influences the rate of change in body temperature. When turkeys exposed to a temperature rise from 21 to 49° C, those with feed in the tract started panting and spreading their wings sooner than those not given feed and had shorter survival time (Kohne et al., 1973). Van kampen (1977) indicated that the thermic response to feeding at thermoneutral environments is maximum only during the first 2 hours after feeding, while at 35°C this thermogenic response was still high during 4-5 hours after feeding. He concluded that effects due to feed can be minimized if the thermogenic effect of feed is controlled not to coincide with heat distress temperature occurring during a day.

McCormick et al. (1979), noted that fasting chicks for up to 72 hours resulted in progressively increased survival time when exposed to heat distress. However, such fasting times are unpractical. Teeter et al., (1987a) observed that fasting broilers 6 hours prior to or during heat distress

reduces heat distress mortality. Since a relationship between feed intake and thyroid hormone activity has been noted (May, 1978; Harvey and Klandorf, 1983). The fasting techniques (Teeter et al. 1987a) may be related to plasma $T_3$ and fasting also result metabolic acidosis that may offset the respiratory alkalosis (Leeson, 1986).

Minerals and electrolyte Effect

Potassium chloride, NaCl and $NaHCO_3$ drinking water supplementation has been reported to increase gain (Reece et al., 1972; Riley et al., 1976; Teeter et al., 1985) and survivability (Branton, et al., 1986; Teeter et al., 1987b) of heat distressed broilers. The effects are correlated with increased water consumption and lower body temperature rise during heat distress (Teeter et al., 1987b). However, the effect is not mediated solely by water consumption as specific ion effects appear likely. Potassium chloride has consistently given a slightly greater benefit than NaCl. Indeed, Deyhim and Teeter (1990) reported that KCl, but not NaCl reduced plasma corticosterone of heat distressed birds. In the same study KCl lowered bird mortality from 12 % to 3 % while NaCl was intermediate at 7 %. Numerous modes of action, for electrolyte efficacy during heat distress are possible. Since respiratory alkalosis predisposes negative potassium balance, KCL water supplementation might reduce potassium deficiency.

Deetz and Ringrose (1976) suggested that in order to maintain normal plasma potassium, the minimal requirement

for laying hens should be increased from .4 % of the diet at 25.6°C to .6 % or greater at 37.7°C. Teeter and Smith (1986), observed broiler drinking water supplementation with potassium chloride supplying .05 to .15 g $K^+$/l to increase survivability, live weight gain and feed efficiency during heat distress despite the ration exceeding NRC (1984) $K^+$ recommendations by 150 %. Potassium enhances cellular uptake of neutral amino acids and glucose (Church and Pond, 1988). The potassium deficiency postulated to occur during high ambient temperature distress (Houstin, 1978; Smith and Teeter, 1987b) would be expected to result in reduced protein synthesis and thereby growth. Therefore, there may be benefits gained by increasing electrolyte supplementation to counter consequences of heat distress.

Phosphorus in the diet may be critical factor in heat resistance. Phosphate in blood and other body tissues is extremely responsive to dietary content since phosphate is lost rapidly from the body of chicks during heat distress (McCormick et al., 1980). Marginally phosphorus deficient broiler chicks had shorter survival time during acute heat distress than those received adequate phosphorus, though body weight was not affected (McCormick and Garlich, 1982). According to them marginally deficient and inadequate groups died rapidly, since blood phosphate was low and was further decreased by the heat distress. Lack of body phosphate probably may reduce the ATP production required for increased metabolic activity occurring during heat distress.

Additionally, the lack of adequate phosphate probably reduced glucose utilization by body tissues, and the heat load of the deficient chicks probably increased more rapidly because they could not get rid of it as fast as the adequate chicks. Charles and Duke (1981) also reported that high ambient temperature increases phosphorus requirement of laying hens, that increased phosphorus intake resulted in increased production rate.

Ambient Temperature Distress and Immunity

Both acute and chronic environmental extremes lead to altered bird thermobalance. High body temperature has been reported to increase bird susceptibility to salmonella infection (Thaxton et al., 1974) and fowl cholera (Simensen et al., 1980). Hutt and Crawford (1960) reported that bird body temperature is inversely related to chick susceptibility to Salmonella infection. Lower ambient temperature has been demonstrated to increase fowl resistance to aflatoxin (Wyatt et al., 1976; Manning and Waytt, 1984).

The increased susceptibility of heat distressed birds to infection is likely related to reduced immune competence. Corticosteroids have been implicated in the modification of immunological function. Corticostrone effects include reductions in lymphatic tissue mass (thymus, spleen, bursa of Fabricius) and depression in the circulating lymphocytes (Ben Nathan et al., 1976; Williamson et al., 1985). During

heat exposure bird serum corticostrone is increased (Ben Nathan et al., 1976; Siegel and Gould, 1982; Mitchell et al., 1986; Deyhim et al., 1990). Therefore, a relationship may exist between ambient temperature distress and corticostroids.

The bursa Fabricius is a primary lymphatic tissue devoted to B-cell proliferation and differentiation. B-cells are precursors of antibody producing plasma cells (Glick, 1957). The thymus is involved in T-cell production. T-cells are important in cell mediated immunity and as effector cells for B-cell proliferation and function. These effects are mediated through lymphokine (interleukins) production (Talmadge and Claman, 1964).

Adrenal involvement in the suppression of cell mediated immune response in heat stress fowls has been documented (Regnier and Kelley, 1981). However, the inability of steroids to alter all cell mediated immune responses suggest that either all T-cell populations are not influenced in the same manner by corticosteroids (Blecha et al., 1982) or that other substances, such catecholamines, which are also stimulated by high ambient temperature distress in chickens (Edens and Siegel, 1975) might suppress corticotropin-releasing ability in hypothalamic tissue (Jones and Hillhouse, 1977).

Literatures on the effects of ambient temperature distress on the immune response have not been consistent. Studies with cold distress in chickens indicate that reduced

temperature or metabolical alterations associated with lowered body temperature may also reduce immune responses (Blecha and Kelley, 1981). Brown and Nestor, (1973) reported that cold distressed turkeys exposed to *Mycoplasma meleagridis* had a lower antibody production response to the antigen. Similarly, cell mediated immune responses are also suppressed in chickens exposed to cold distress (Blecha and Kelley, 1981). According to Sinha et al. (1957) resistance to *Pasterurella multocida* and Newcastle disease was increased in chickens exposed to cold, while chick susceptibility to salmonella infection was increased during exposure (Thaxton et al., 1974).

Heat distress induced immunosuppression, although usually evident in cell mediated immunity, is not always observed when humoral antibodies, such as those represented by agglutinin reactions are measured (Morgan et al., 1976; Subba Rao and Glick, 1977). Regnier et al. (1980), were not able to detect differences in antibody production between acute heat distress and thermoneutral housed chickens. Gross and Siegel, (1980), suggested that the immune response to high temperature distress is influenced by the type of bird, those selected for high antibody response showing a reduction response while those selected for a low response showing an enhanced one.

Acute exposure to cold distress fails to alter blood corticostrone concentration in the neonatal fowl but markedly increases corticostrone in the older birds.

Therefore, age differences between birds used in immunological response studies can attribute for controversies reported in literatures (Freeman, 1982). The mechanism of action of corticostrone on the immune response have been reported. Studies with cell culture showed that corticostroids are bound to specific protein receptors in the cytoplasm of lymphatic cells, and the complex pass into the nucleus of these cells to alter enzymatic activity and nucleic acid metabolism (Thompson and Lippman, 1974; Sullivan and Wira, 1979). Consequently, glucose uptake and protein synthesis are suppressed as well as cell proliferation factor, a lymphokine produced by T-cell in response to antigen stimulation, is reduced (Gilis et al., 1979).

Studies with birds exposed to heat distress or injected with Adrenocortical trophic hormone indicate increased endogenous corticostrone bound in lymphocytes (Gould and Siegel, 1981; Siegel and Gould, 1982). However, pre heat distress treatment with metyrapone, a blocker of corticostrone synthesis, modifies the distress effect and demonstrates that the immunosuppression is, at least in part, mediated by adrenal corticosteroids (Thaxton and Siegel, 1973; Siegel and Latimer, 1974). For the most part immunological effect of corticostrone is reported to be on the T-cell population and reduced circulating antibody concentrations (Thaxton and siegel, 1970; Pardue and Thaxton, 1984).

Nutrition and Immune Response

It is well known that the nutritional status of an animal has a significant effect on host defence mechanisms. Moderate restriction of intake of a balanced diet for 10 days prior to immunization had little effect on the haemagglutinin response to sheep erythrocytes in birds at 25°C. In contrast a similar feed restriction increased haemagglutinin responses in birds maintained at ambient temperatures of 15 or 35 C or in those in environments fluctuating from 10 to 20°C or from 30 to 40°C (Henken et al., 1983). However, restricting feed intake at temperatures below 10°C or subjecting birds to sudden 10°C changes in temperature depressed haemagglutinin titters (Henken et al., 1982). These results suggest that there is an interaction of immunological response with metabolic activity of the bird. Severe energy alone, or in combination with lysine and sulphur amino acids deficiencies during the first five weeks posthatch, have been reported to depress primary haemagglutination responses to sheep erythrocytes (Glick et al., 1981) and delayed hypersensitivity reactions to human gamma globulin in chicks (Glick et al., 1983). They also reported that immune parameters were returned to normal when birds were fed nutritionally adequate diet. Interaction between environmental temperature and immunological response of animals is mediated through feed consumption. Voluntary

decrease in feed intake due to deficiency have been documented to depress the immune response (Pimentel et al., 1991).

Homeotermic animals subjected to ambient temperature below their thermoneutral, respond by increasing heat production to maintain body temperature. Thyroid regulator hormones, adenylate cyclase activity and thus cyclic AMP increase with feed consumption to increase metabolic rate. As a result protein synthesis and specifically cyclic AMP increases the synthesis of antibody protein. Increased antibody responses in animals under moderately low ambient temperature has been reported (Subba Rao and Glik, 1977) despite increase in adrenal cortical activity. However, in animals under heat distressed nutrient deficiencies as a result of decreased feed intake possibly contribute to the depressed immune responses.

Specific nutrients have been documented to alter immunological status in poultry. Bhargava et al. (1970) reported that hemagglutination inhibition titer to Newcastle disease virus were higher in chicks fed diets deficient in methionine than in those fed diets adequate in methionine. Similar results were reported by Tsiagbe et al. (1987) that .063, 125, and .25 % extra supplementation of methionine above the requirement for broiler chicks dose dependently increased total antibody response to sheep red blood cell and immunogloblin (IgG). Amino acid imbalanced diets created by methionine supplementation was reported to

depressed immune response in rats (Kenny et al., 1970). Therefore, contradictions between reported results on methionine supplementation may be related to amino acid imbalance. However, extra methionine is important for the synthesis of the IgG and required for thymus derived T-cell function (Tsiagbe et al., 1987). This study indicate that the requirement for maximum growth appears to be below the requirement for maximum antibody response, thus growth criteria for establishing the nutrient requirement may be inadequate for the overall well-being of the chick.

The widely accepted roles of vitamin E and selenium are related to antioxidant properties. Vitamin E participates in nucleic acid metabolism as enzymes cofactor in arachidonic acid metabolism (Franchini et al., 1988). The conversion of arachidonic acid to thromboxanes and prostaglandin is inhibited by the vitamin through blocking enzymes, lipoxygenase and cicoxygenase into hydroperoxides and endoperoxides, intermediate metabolites of prostacylins, thromboxane and prostaglandin (Tengerdy and Brown, 1977).

Prostaglandins are reported to have an effect in reducing the multiplication and functional activity of cells involved in the immune response (Tengerdy and Brown, 1977) and the macrophage functions (Gebremichael et al., 1984). lymphocyte proliferation of chickens fed fat rich in polyunsaturated fatty acid (omega 3 fatty acids), precursors of prostaglandin was reduced (Kevin et al., 1991). These evidences suggest that effects of Vitamin E is carried out

through inhibition of the output of prostaglandins (E-type) thereby activating humeral immunity and phagocytosis (Likoff et al., 1981; Panganamala and Cornwell, 1982). Vitamin E stimulates the multiplication of T-lymphocytes and macrophages thereby the general immune response by increasing interferon which are important for regulating the production of antibodies and the function of macrophages (Tanaka et al., 1979; Geberemichael et al., 1984). Franchini et al., (1990) reported that the production of alpha and beta interferon was increased in broilers by feeding Vitamin E above the requirement (325ppm). Therefore, at times when there is a deficiency of Vitamin E in chickens impairment in normal humoral immune responses to sheep red blood cells (SRBC) is observed (Marsha et al., 1981).

Selenium has been shown to play a major role in the development and maintenance of the bird's immune response. Similar to Vitamin E this trace element has antioxidant effects during macrophage-lymphocyte interactions (Brune and Spitznagel,1973) which appear to be oxidative in nature (Weiss et al., 1983). Dietary selenium supplementation, above the generally accepted levels, increased the number of blood leucocytes during infection with coccidia and thereby improved the immune status (Colgano et al., 1984). Selenium being a component of glutathione peroxidase enzyme, an intercellular scavenger of hydroperoxides. Deficiency of this element has been reported to reduce the activity and

synthesis of the enzyme glutathione peroxidase (Xia et al., 1989). The mode of action of selenium in enhancing the immune response is mediated through inhibiting the enzymes of arachidonic acid metabolism likely similar to vitamin E (Schoene et al., 1986).

Deficiencies of both vitamin E and Se in chicks depressed bursa weight, reduced the overall number of lymphocytes found in the primary lymphoid organs and spleen, and resulted in destructive histological changes within these tissues (Marsha et al., 1982). Increased body weight and reduced mortality from Eimeria tenella oocyte infestation was observed when both vitamin E and Se were supplemented beyond requirements (Colnago et al., 1984).

Zinc deprivation has been shown to cause involution of the thymus to 50 % of normal in rats (Dowd et al., 1986) and involution of spleen to 61 % of normal in mice (Leucke et al., 1978). Depression of immune function occurs in mice and rats with only a moderate depression in body weight (Leucke et al., 1978; Beach et al., 1980). Zinc-deficient mice have decreased antibody response (De-Pasquale-Jardiev and Fraker, 1984) and impaired cellular immune response via selective effects on helper thymus derived lymphocyte (Dowd et al., 1986). The immune response of chicks and hens to zinc deficiency has been reported by Burns (1983). According to him, chicks maintained on a zinc deficient diet from hatching to 14 days of age had reduced antibody response to bovine serum albumin. Other reports regarding

zinc deficiency in chickens and the immune function showed that breeding hens fed a marginally zinc deficient diet, exhibited lower antibody responses in progeny (Stahl et al., 1989a). However, in chickens raised in a controlled environment, antibody titers to sheep red blood cell were unaffected by feeding various levels of zinc (Stahl et al., 1989b).

low plasma ascorbic acid levels have been associated with reduced immunological function in fowls (Pardue and Thaxton, 1984). Ascorbic acid is required for interferon production (Siegel, 1974) and for the transformation of lymphocytes to plasma cells (Siegel and Morton, 1977). During heat distress corticosteroids appear to cause a depletion of plasma ascorbic acid and dietary supplementation with the vitamin corrects this depletion (Pardue and Thaxton, 1984). According to them the addition of 1 gm/kg ascorbic acid in the diet of heat distressed broilers reduces weight loss and mortality. However, no improvement in weight gain and survivability was observed in other studies when the same level of supplementation was made for heat distressed broilers (Freeman et al., 1983; Stilborn et al., 1988).

Antibiotic Supplements

Growth rate improvements through antibiotic supplementation in broilers have been documented. Body weight gain and feed efficiency of broilers fed bambermycin

(Kling et al., 1977), zinc bactcitracin (Wicker et al., 1977) and virginiamycin (Miles et al., 1984a) was higher compared to unsupplemented groups. Survivability of broilers was improved with tetracycline and oxytetracycline treatment (Peterson et al., 1991). Similar observations were also reported for bambermycin (Kling et al., 1977) and zinc bactcitracin (Wicker et al., 1977) supplementation. In particular, virginiamycin have been consistently found to improve broiler growth rate and feed efficiency (March et al., 1978; Miles et al., 1984b; Harms et al., 1986; WoodWard et al., 1988) as well as carcass yield (Leeson, 1984; WoodWard et al., 1988).

The increased carcass yield with virginiamycin has been attributed to reduced intestinal tract weight relative to body weight (Henry et al., 1986; Izat et al., 1989; Salmon and Stevens, 1990). Improved performance of broilers fed diets containing virginiamycin is associated with increased feed consumption (Leeson 1984; Buresh et al., 1985a) and nutrient absorption efficiency (Nelson et al., 1963; March, et al., 1981). Enhanced utilization of sulfur amino acids (Miles and Harms, 1983; Miles, et al., 1984a), phosphorus (Buresh et al., 1985b) and manganese (Henry et al., 1986) has been reported for birds consuming Virginiamycin. In general, the beneficial activities of antibiotics was recognized in environments with poor sanitation, other wise under germ free conditions improvements are minimal (Coats et al., 1963; Freeman et al. 1975; Klasing and Benson,

1990). Various modes of action for antibiotics efficacy have been proposed.

The reduction in intestinal weight of chicks fed antibiotics was first reported by Gordon (1952) who also observed that chicks reared in a germ-free environment had reduced intestinal weights. Further studies by others confirmed such observations (Coates et al., 1955; Jukes et al., 1956; Hill et al., 1957; Istifanus et al., 1985). Hill et al. (1957) reported that the decrease in intestinal weight occurs before increased body weight is noted. Gordon and Bruckner-Kardoss (1959) found that in germ-free chickens supplemented with antibiotics the amount of lamina propria, lymphoid tissue, and numbers of free reticulo-endothelial cells in the ileal mucosa are reduced concurrently with the reduction in intestinal weight. King (1974) reported virginiamycin reduces the net mass of the small intestine and thickness of the mucosa membrane. Similarly Stutz et al. (1983) also reported that feeding zinc bacitracin to chicks significantly reduced the weight, length, and percent moisture of the small intestine. According to them the reduction was more pronounced in the jejunum and ileum than in any other part of the intestine. On the basis of these observations, and that of Solca et al. (1980), it is likely that antibiotics stimulate growth of animals by eliminating undesirable microorganisms producing toxins or metabolic products that irritate and inflamate the intestinal wall.

Such model may also account for the increased nutrient absorption.

The role of the bursa of Fabricius as an integral component of the avian immune system has been recognized and described (Glick et al., 1956; Glick, 1968, 1979). In studies with bursectomized chicks, Glick et al. (1956) were able to show that removal of the bursa resulted in an impairment of the immune response to a variety of antigens. The addition of antibiotics to the diets of the chemically bursectomized chicks reduced mortality and improved body weight gain but did not improve their immunocompetence. According to Istifanus et al. (1985) growth response to antibiotics is highest in broilers during the first 3 weeks of the chick's life, when rate of growth of the bursa is most rapid, and immunocompetence is highest. Though chicks fed antibiotics tend to have heavier bursea, effects on the spleen and antibody response to sheep red blood cells were not affected by antibiotic supplementation, suggesting that effect of dietary antibiotics is not mediated through enhanced immunocompetence.

It has been postulated that the feeding of antibiotics does cause immunosuppression (Stevens, 1953; Glick, 1968; Tarnawski and Batko, 1973). They suggested that the effect of antibiotics on the immune reaction is attributed to properties such as elimination of antigen, influence on macrophages, phagocytosis and indirectly by inhibition of antibody synthesis. Tu (1980a, b, c) inoculated mice and

pigs that had been subjected to different doses of penicillin before or at inoculation time with live and inactivated swine erysipelas vaccine. The author reported that the antibiotics caused immunosuppression in mice, but results with pigs were inconsistent. The immunosuppression to swine erysipelas vaccine by antibiotics was dose related and was affected by the time differential between antibiotic administration and vaccine inoculation and by whether the vaccine was live or inactivated. Recently, Klasing and Barbara (1990) observed that birds challenged with poor sanitation have increased immune system is activation, plasma interleukin secretion from macrophages and lymphocytes and increased associated with increased glucose oxidation, muscle protein degradation, increased body temperature and decreased feed intake and decreased growth. They suggested that decreasing the degree of activity of the immune system can be achieved by antibiotics that aid in decreasing the bacteria-host interactions. According to this model if immunosuppressive is accompanied by reduced heat production, then during heat distress when bird body heat load is increased antibiotics could be beneficial to minimize the additional heat load from enhanced immune response.

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# CHAPTER III

## SURGICAL PREPARATION OF BROILER CHICKS FOR SEPARATE COLLECTION OF FECES AND URINE.

Belay, T, C. J. Wiernusz, R. G. Teeter.
Deparment of Animal Sciences
K. E. Bartels
Vet. Teaching Hospital.
Oklahoma State University, Stillwater, OK. 74078

### Abstract

A surgical procedure for the separation of avian urinary and fecal excrement applicable to broiler chicks at least 3 1/2 weeks of age is described. The procedure involves excising the rectum caudal to the ileocecocolic junction about 2.5 cms cranial to the cloaca. A small circular hole 1.5 cm in diameter is made through the left ventrolateral abdominal wall and the transected proximal end of the rectum is sutured directly to the opening. Cannulae of 7 mm and 9 mm inner diameter were inserted into the stoma at 7 and 12 days postoperatively, respectively. These cannula remained in place until they were replaced with a larger unit (12 mm diameter cannula) 15 days postoperatively. Of 68 chicks undergoing the surgical procedure, 49 (72%) were suitable for experimental use in

long term studies. Surgically modified broilers had a lower ($P<.05$) body weight and gain compared to their respective pair fed non-operated counter part 10 days postoperatively. Daily urine production of the colostomized broilers averaged $114\pm5$ ml under a thermoneutral environment (23-25°C). Surgically modified colostomized birds have been maintained for up to 3 months without complications.

(*Key words*: surgical procedure, feces, urine, broiler, colostomy).

## Introduction

Metabolism studies frequently necessitate separate analysis of urine and feces. However, since poultry void mixed urine and feces it is necessary to utilize surgical modifications and/or mechanical devices to separate poultry excreta (Paulson, 1969). Reports ( Davis, 1927, Caulson and Huges 1930) have described non-surgical techniques of plugging the rectum and attaching catheters in or around the ureterial orifices for excrement separation. Fussell (1969) reported that the use of such non-surgical techniques for extended periods of time were unsatisfactory due to the high degree of ureteral irritation. Surgical techniques for exteriorizing the ureteral orifices for short term experiments in growing birds has also been described (Hester et al. 1940; Newberne et al. 1957; Ainsworth, 1965 ). However, Richardson et al. (1959) and Fussell (1969) reported that surgically exteriorized ureters were

frequently associated with suture breakdown, irritation to the bird, and excreta contamination.

Isolation of the rectum from the cloaca and its diversion to a point on the abdominal wall was described by Rothchild (1947) though details for postoperative conditions and subsequent bird performance were not discussed. Fussell (1960) described a long-term metabolic study in which year old layers weighing over 1.5 kg were used for separate collection of urine and feces. Fussel later (1969) discussed how breed, species, sex and age difference might affect operational suitability. He recommended that birds weighing less than 0.75 kg not be used. Several studies utilizing adult or nearly mature birds includs Ariyshi and Morimoto (1957), Okumura (1976) and Isshiki (1985). Polin et al. (1967) utilized 4 week old white leghorn cockerels but reported colostomy blockage and loss of body weight problems. Postoperative problems include narrowing of the colostomy site and obstruction (Ariyoshi and Morimoto, 1957, Richardson, et al., 1960, Colvin, et al., 1966; Okumra, 1976). To avoid these problems, placing glass cannulae within the colostomy stoma was suggested to dilate the constricted bowel (Richardson et al. 1959, Fussel 1969). Okumura (1976) used a polyvinyl chloride (PVC) cannula inserted into the stoma on the tenth postoperative day, while Gunnsey and Johns (1986) recommended utilizing a PVC rod.

In general, previous reports concerning this surgical modification in poultry lack procedural details, have been criticized as to their suitability for research, and have been used primarily for short term assays in adult birds. The objective of this project was to simplify the description of surgical procedure and evaluate the suitability of young broilers for use in nutrient metabolism studies.

## Materials and Methods

BIRD AND PREOPERATIVE PROCEDURES: Acre X Vantress broiler chicks were raised on rice hull litter to 3 weeks posthatching, and transferred to individual (30 X 40 cm) cages. Cages were housed in an enviromentaly controlled room maintained at 24°C and 55 % relative humidity under continuous lighting. Birds were fed broiler growers ration (Table 1) with daily consumption recorded, and provided drinking water fortified with vitamins, electrolytes[1], and oxytetracycline Hcl soluble powder Terramycin[2] at a concentration of 1.5 gms or 2.0 gms per liter of drinking water.

Surgical Procedure: Surgery was performed on 68 chicks averaging 629 gram in weight at 3 1/2 weeks of age. Anesthetic induction and maintenance was achieved by a

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1 Vitamins and electrolyte soluble, Salsbury Laboratories, Inc.

2 Oxytetracycline Hcl Terramycin soluble power, Pfizer.

ketamine HCl[3] intramuscular injection (40 mg/kg of body weight) and halothane[4] in oxygen via a Bain none rebreathing system, respectively. Following anesthetic induction, the bird was secured to the surgery table by gently retracting the right leg caudally and the left leg cranially. Feathers were removed from a 30 square cm area on the left lateral side cranial to the cloaca and the skin was aseptically prepared for surgery utilizing chlorhexidine surgical scrub[5]. The left wing was carefully abducted and retracted caudally away from the surgical site.

A 1.5 cm circular skin incision was made 2.5 cm cranial to the cloaca on the left ventrolateral abdomen. This incision was extended to the deeper tissues including the abdominal musculature, air sac, and peritoneum. The distal colon was gently directed to the surgical site by inserting a blunted sterile glass rod 5 mm in diameter through the cloacal orifice.

The distal colon was exteriorized and gentle traction applied with a hemostatic forcep. This segment of distal colon was isolated by passing a hemostatic forcep through the mesocolon avoiding any major blood vessels supplying the colon. The colon was cross-clamped with a forcep approximately 1 cm from the proximal cloaca. The colon was then completely transected proximally to the forcep and the proximal segment to be colostomized was withdrawn from the

3 Ketaset, Fort Doge Lab. Inc., Fort Doge, Iowa 50501.
4 Halothane, UPS, Fort Doge Lab., Inc. Fort Doge, Iowa 50501.

surgical site and isolated with a sterile saline-soaked gauze sponge. The distal colonic segment was double ligated with 3-0 absorbable surgical suture[6] and returned to the abdominal cavity.

The isolated proximal colonic segment was temporarily stabilized outside the abdomen with stay sutures of 3-0 absorbable suture material[6] to prevent it from retracting and contaminating the peritoneal cavity. The abdominal cavity was carefully lavaged with sterile physiologic saline and all hemorrhage was carefully controlled with ligation.

The exteriorized edges of the transected proximal colonic segment were sutured to the adjacent abdominal musculature and skin with a minimum of six full-thickness simple interupted sutures of absorbable suture material[6] spaced equidistantly around the circumference of the colonic stoma.

After the surgical procedure was completed, the surface of the operated area and surrounding skin were lightly coated with white petrolatum jelly to prevent dermal irritation from feces. The stoma was not disturbed for three postoperative days except for cleaning with sterile saline-soaked gauze sponges to remove feces. Thereafter, the surgical site was gently cleaned with warm water and to remove feces and foreign matter. The stoma was closely

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5.Novalsan Surgical Scrub-FT, Doge, Iowa 50501.
6 Vicryl, Polygalactin 910, Ethican Inc., Sommerville, N.J.
7 Tygon tube R-3603
8 Silk-Ethican, Inc., Sommerville, N.O.

monitored during the entire postoperative period. Postoperatively, irregardless of daily feed consumption, each of the operated chicks were force fed (Teeter and Smith, 1984) 35 gms of feed daily for 4 days. Intact chicks originating from the same flock were used as a reference point to monitor surgical effects.

CANNULATION, FECES AND URINE COLLECTION

Dried blood and serum surrounding the operative site was removed each day for 4 days after surgery. On the seventh postoperative day, a rubber[7] cannula was inserted through the stoma and fixed to the skin and underlying tissue with 5 to 6 interrupted suture[8] (2-0 non-absorbable) in a simple interrupted pattern. Birds were anesthesized and maintained with halothane. This initial cannula was removed and replaced with a medium (9 mm inner diameter and 20 mm length) and larger (12 mm diameter and 2.5 cm long) cannulae on postoperative day 12 and 15, respectively .

Fecal and urine collection were initiated on the nineteen postoperative day as follows: A plastic sleeve of 3.5 cm in length for suspending fecal collection bags was made with 5 cm diameter carbon ring attached to one of its end. This was placed on its carbon ring free end centering the stoma sutured to the skin. Similarily a plastic sleeve of 1.5 cm length with 3.7 cm diameter carbon ring was also made and attached to the skin surrounding the cloacal with non-absorbable suture for suspending urine collection bags.

Feces and urine were collected in suspended plastic bags secured with a rubber band to the plastic sleeves on the respective sites.

## Results and Discussion

Total recovery time from the surgical procedure was measured by determining the time required for the birds to initiate feed and water consumption. Recovery time averaged 60±10 minutes per bird. In this particular investigation, 49 out of the 68 birds were suitable for experimental use as judged by feed consumption, freedom of blockage, and no clinical signs of illness on the nineteen postoperative day. Eight birds were rejected due to weight loss attributable to blockage at the surgical site. Bird mortality (11) was attributed to retraction of the exteriorized colon into the peritoneal cavity with a resulting peritonitis.

After surgery birds were housed in individual chambers as described, while feed and water were available ad libtum. Feed consumption during the posoperative period was recorded for operated birds while only body weight was monitered for controls.

Birds used for the operation had a similar initial body weight compared to unoperated controls. Body weight and gain at 10 days of postoperation was lower ($P<.05$) for the operated compared to unoperated controls (Table1). However, 20 days postoperative body weight and gain of operated birds was similar to non operated control broilers . Daily weight

gain for operated broilers was 33 gms and 29.9 gms at 10 and 20 days of postoperative, respectively. This gain was comperable to non operated broilers which had 36 gms and 31.8 gms at the same age. The operated broilers had a lower ($P<.05$) mean body weight compared to controlls at 38 days of age. However, body weight of operated and controlls was similar ($P>.2$) at 48 days postoperative.

The criteria for inclusion of operated birds in the study were absence of blockage of the colostomy stoma, no clinical sign of illness, and normal feed consumption. It must be mentioned that intensive postoperative care was necessary for these birds to ensure success of the surgical procedure. Reported urine production estimates of adult chickens from short period collection ranged from 86 to 1000 ml/day (Davis, 1927, Coulson and Hughes, 1930, Hester et. al., 1940, Dicker and Haslam, 1966). In this study using the colostomized broilers at 54 days of age, daily urine production was estimated to be 114 ml/day during a 36 hours collection period from 22 broilers kept at thermoneutral environment. Daily urine volume output was increased to 220 ml/day when the birds were exposed to an elevated constant ambient temperature of 32-35$^{\circ}$C. This result were similar to those reported by Ariyoshe and Morimoto (1956).

Surgically modified birds survived up to 3 months without complications and thereafter, mortality was due to blockage of the stoma by an over growth of tissue around the site and dried fecal material in the enlarged colon.

Accurate apposition of the colon to the skin and abdominal musculature needs to be emphasized. Hasty closure that results in dehiscence can be disastrous and life-threatening, and can ultimately lead to stenosis of the stoma if the bird survives.

Table 1. Initial, 10 and 20 postoperative day body weight and gain of operated and non operated broilers.

| Days Postoperation | Non-operated | Operated |
|---|---|---|
| Initial Wt. | 619 | 637 |
| 10 Wt. (g) | 979[a] | 907[b] |
| 20 Wt. (g) | 1255 | 1235 |
| 10 gain (g) | 360[a] | 330[b] |
| 20 gain (g) | 636 | 598 |

[a,b] Means within each row with no common superscripts are significantly different ($P<.05$).

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# CHAPTER IV

# MINERAL BALANCE AND URINARY AND FECAL EXCRETION PROFILE OF BROILERS HOUSED IN THERMONEUTRAL AND HEAT DISTRESSED ENVIRONMENTS

Belay, T., C.J. Wiernusz, K. E. Bartels and R.G. Teeter

Animal Science Department, Oklahoma State University

Stillwater, OK 74078

## Abstract

Two experiments were conducted utilizing 4 to 6 week posthatching Vantress X Arbor Acre male broilers to evaluate heat distress effects on mineral balance partitioned into urinary and fecal loss. In the first study 8 colostomized and 14 intact birds were allotted to two environmental chambers maintained at either a thermoneutral (TN, 24°C) or a cycling temperature heat distress (HD, 24-35°C) in a switch-back experimental design. Birds were precision fed 2% of body weight 3X daily to equalize consumption prior to and over each 48 hr experimental period. Feces and urine for colostomized and total excrement for intact birds were collected and analyzed for mineral content. Averaging over

surgical classification HD decreased (P<.05) mineral balance for $K^{+}$, $P^{-3}$, $S^{-2}$, $Mg^{+2}$, $Cu^{+}$, $Mo^{+6}$, $Se^{-2}$, and $Zn^{+2}$. Despite little HD effect on urine production minerals disproportionately excreted in urine included $K^{+}$, $Mg^{+2}$, $P^{-3}$, and $S^{-2}$ while $Cu^{+}$, $Se^{-2}$ and $Mg^{+2}$ were lost primarily in feces. In the second study forty two colostomized birds were used to expand the urinary excretion data with the HD period held at 35°C for 36 hr. Heat distress increased (P<.05) urine output from 52.3 to 109.9 ml/12 hr and urinary $K^{+}$, $P^{-3}$, $S^{-2}$, $Na^{+}$, $Mg^{+2}$, $Ca^{+2}$, $Mn^{+2}$ excretion. These data provide evidence that HD adversely impacts bird mineral balance and further that the excretion route varies with the specific mineral and possibly HD severity.
(*key words*: mineral balance, heat distress, urine, broilers)

## Introduction

Numerous consequences of high ambient temperature -relative humidity distress on poultry production have been documented. Among the effects, heat distress reduces bone weight and strength while increasing the incidence of leg problems in broilers (Siegel *et. al.*, 1973, Ernst *et al.*, 1984, Smith and Teeter, 1987) and turkeys (Magruder and Nelson, 1967). Such effects are likely the result of impaired mineral metabolism. Kamar *et al.*, (1987) observed calcium, phosphorus and magnesium retention to be reduced (P<.05) in heat distressed hens while Wolfenson et al., (1987) reported reduced $K^{+}$, $P^{-3}$, and $Ca^{+2}$ absorption in

turkeys. Broilers subjected to a 32°C environment days had lower calcium (Ca), copper (Cu), iron (Fe) potassium (K), magnesium (Mg), manganese (Mn), sodi ..., phosphorus (P), and zinc (Zn) retention (Husseny and Creger, 1981). The reduced retention was accompanied by a lower carcass concentration of each elements and is in agreement with Sonaiya, (1989) and Sharma and Gangwar (1987).

Specific ambient temperature effects on blood $K^+$ have been mixed as Kohne and Jones (1975) observed blood levels to increase during heat distress while Huston (1978) and Deyhim, et al. (1990) reported such blood levels to decline. Simmons and Avedon (1959) suggested that intercellular $K^+$ could buffer extracellular $K^+$ concentrations during acid base perturbations and on this basis was proposed (Smith and Teeter, 1987b) to delay heat distress consequences. Respiratory alkalosis occurring during heat distress reduces renal tubule $H^+$ ion concentration thereby increasing urinary $K^+$ excretion. However, the effects on plasma $K^+$ concentration are buffered by intercellular $K^+$ exchange for extracellular $H^+$ AS occurs during metabolic alkalosis (Harper, *et al.*, 1977; Best and Taylor, 1985). Such a mechanism leads to an eventual drain of both intercellular and extracellular $K^+$ during heat distress and accounts for the variability associated with plasma $K^+$ during heat distress. Indeed, interpretation of in vivo mineral fluid content can lead to varying interpretation. Nonetheless, heat distress effects on mineral loss are dramatic. Heat

distress induced perturbations of bird mineral balance have been associated with decreased plasma $Na^{+}$, $Ca^{+2}$, $Mg^{+2}$, and inorganic phosphorus concentration in turkeys (kohne and Jone (1975) and reduced inorganic phosphate and calcium in broilers (McCormick and Garlich, 1982).

Broilers subjected to 35°C pair fed to mimic thermoneutral controls, excrete 27.3% more $K^{+}$ (Smith and Teeter, 1987a) similar to observations made by Deetz and Ringrose (1976), for hens housed at 37.7°C. Deetz and Ringrose suggested that in order to maintain normal plasma potassium, the minimal requirement should be increased from .4% of the diet at 25.6°C to .6% or greater at 37.7°C. Smith and Teeter (1986), observed broiler drinking water supplementation with potassium chloride supplying .05 to .15 g $K^{+}$/l to increase survivability, live weight gain and feed efficiency during heat distress despite the ration exceeding NRC (1984) $K^{+}$ recommendations by 150%. Therefore, there can be little doubt that delineating environmental effects on bird mineral metabolism is fundamental to developing therapeutic measures to counter consequences of heat distress. The objective of the studies reported herein was to evaluate heat distress effects on broiler mineral balance and further to partition such effects into urinary and fecal excretion components.

## Materials and methods

*Experiment 1.* The first experiment was conducted to

evaluate heat distress effects on urinary and fecal mineral excretion. Twenty eight commercial Vantress X Arbor Acre broiler chicks were selected for the study with fourteen remaining intact and fourteen being colostomized (Belay, *et al.*, 1990) at 4 weeks posthatching. On day twenty, 8 colostomized and fourteen intact birds were allocated at random to individual 61 X 82 cm broiler grower battery compartments housed within two environmental chambers (24°C) in a switch back experimental design. Colostomized birds were selected from the initial fourteen undergoing surgery on the basis of normal feed intake, absence of colostomy stoma blockage and freedom from illness. Following a 2 day adaptation period to chamber facilities, with feed and water available for *ad libtum* consumption, the birds were fasted overnight and weighed. During the 4 day preliminary and subsequent 2 days collection intervals chicks were precision fed at 2% of body weight 3 X daily (Teeter, *et al.*, 1984). Ambient temperature of the chamber designated for cycling temperature heat distress was increased 2.8°C per day to 35°C such that birds were provided 11 hr daily in excess of 32°C. The process was repeated for period 2 with the exception that birds exposed to heat distress during period 1 were housed at thermoneutral and the thermoneutral birds exposed to heat distress. Experimental periods were separated by 4 days housing at thermoneutral with feed and water available for *ad libtum* consumption. Feces and urine or total excrement from the colostomized and intact

broilers, respectively were collected at 12 hour intervals, quantified by gravimetric analysis and composited. Feed and fecal samples were prepared for mineral analysis according to Trudeau and Freier (1967) while the method of DeRuig (1986) was used for urine. All samples were analyzed by an Inductively Coupled Argon Plasma Emission Spectrophotometer (ICAP) for $Ca^{+2}$, $K^{+}$, $Cu^{+}$, $Fe^{+2}$, $Mg^{+2}$, $Mn^{+2}$, $Mo^{+6}$, $P^{-3}$, $S^{-2}$, $Se^{-2}$, $Zn^{+2}$, and $Na^{+}$. Daily fecal, urinary and mixed excreta mineral loss were calculated as milligram per day per Kilogram body weight ($mg.d^{-1}.BW^{-1}$) and overall balances as total intake less the total excretion per Kg body weight for each mineral.

*Experiment 2*. To examine heat distress effects on urinary mineral excretion in greater detail forty birds were colostomized and handled as described in experiment 1 with the exception that 6 week old birds were used, with one 2% body weight feeding, excreta collection phases consisted of 3 consecutive twelve hour intervals with ambient temperature held at 35°C, only urine samples were collected and urine chloride ($Cl^{-}$) excretion was also determined. Urine samples were analyzed for $Cl^{-}$ immediately following collection via Chloridometer (Model 4425000, Haake Buchler Instru. Inc.).

Upon completion of both experiments period, ambient temperature, surgical history and appropriate interaction effects were evaluated using the general linear model procedure of the statistical analysis system (SAS, 1985). When a significant F statistic was conducted means were

separated using least significant differences (Steel and Torrie, 1960).

## Results and Discussion

Mineral excreta analysis for experiment 1 are displayed in Table 2. Surgical history and period interaction with ambient temperature were not significant (P>.1) and data were pooled for total mineral balance evaluation. As a result, all birds were used in the computation of total mineral balance with colostomized birds used to partition mineral loss into urinary and fecal components. Urine output over the 4 day collection period was not influenced by ambient temperatures with 65 and 60 ml being excreted for heat distress and thermoneutral birds, respectively. Urine production has been reported to increase with heat distress (Deetz and Ringers, 1976; Van Kampen, 1981; Deyhim, et al., 1990). Reasons for the lack of a HD effect on urinary production in this experiment are unknown. Nonetheless, heat distress reduced (P<.05) mineral retention for $K^{+}$, $P^{-3}$, $S^{-2}$, $Na^{+}$, $Zn^{+2}$, $Se^{-2}$, $Mo^{+6}$, $Mn^{+2}$, $Mg^{+2}$, and $Cu^{+}$. The lower mineral retention was reflected by increased (P<.05) urinary excretion for $Mg^{+2}$, $K^{+}$, $P^{-3}$, and $S^{-2}$ and higher (P<.05) fecal excretion for $Mg^{+2}$, $Cu^{+}$, and $Se^{-2}$. Manganese and $Na^{+}$ loss tended (P<.1) to be disproportionately associated with urine and fecal excretion, respectively. Zinc and $Mo^{+6}$, though exhibiting reduced retention, had a balanced excretory route. Calcium and $Fe^{+2}$ retention tended to be

lower ($P<.1$) for heat distress. Results of the first experiment are in agreement with Husseiny and Creger (1981) in that broilers subjected to heat distress had a significantly lower ($P<.05$) $Ca^{+2}$, $Cu^{+}$, $Fe^{+2}$, $K^{+}$, $Mg^{+2}$, $Mn^{+2}$, $Na^{+}$, $P^{-3}$, and $Zn^{+2}$ retention.

In the second experiment, conducted under chronic rather than cycling ambient temperature distress, heat distress increased ($P<.05$) urine output from an average of 52.3 ml/bird to 109.9 ml/bird for each of the 12 hr collection intervals. Similar to the first study, period X ambient temperature interaction effects were not judged significant and the data were subsequently pooled. Heat distressed birds had increased ($P<.05$) excretion for urinary $K^{+}$, $P^{-3}$, $S^{-2}$, $Na^{+}$, $Mg^{+2}$, $Ca^{+2}$, $Mn^{+2}$ and $Se^{-2}$. While $Zn^{+2}$, $Mo^{+6}$, $Fe^{+2}$ and $Cu^{+}$ were not affected (Table 3). The second experiment, with its geater bird number and more sever heat distress added $Na^{+}$, $Ca^{+2}$, $Mn^{+2}$ and $Se^{-2}$ to the list of minerals with increased urinary excretion during heat distress. Similar to the first study urinary loss of $Zn^{+2}$, $Mo^{+6}$, $Fe^{+2}$ and $Cu^{+}$ was not increased by heat distress again suggesting that the decreased retention associated with heat distress, observed in the first experiment is by fecal excretion.

In contrast, to the minerals discussed above urine chloride excretion (Meq/kg body weight.12 $hr^{-1}$) decreased ($P<.05$) from .53 for birds housed at thermoneutral to just .32 during heat distress. During heat distress respiratory

alkalosis is reflected in lower plasma carbon dioxide, bicarbonate and elevated pH (Teeter, *et al.*, 1985, Branton, *et al.*, 1986), while Khone and Jones (1975) reported similar blood gas changes along with an elevated plasma chloride in heat distressed turkey. Partial compensation for the acid-base imbalance occurs through exchange of plasma bicarbonate for chloride ions (Mongin, 1981). As such one would also expect that plasma $Cl^-$ in heat distress broilers would be elevated.

The data reported herein adds credence to reported observations regarding heat distress effects on mineral balance and further that the route of mineral excretion varies with the specific mineral and possibility heat distress severity. Considering this report and other published works, the mineral requirements of heat distressed broilers requires further investigation.

Table 1 . Composition of diet[1]

| Ingredient | % |
|---|---|
| Ground corn | 56.8 |
| Soybean meal | 36.0 |
| Fat | 3.0 |
| Dicalcium phosphate | 2.35 |
| Calcium carbonate | .90 |
| Salt | .50 |
| Vitamin mix[2] | .25 |
| Trace mineral[3] | .10 |
| DL-Methionine | .10 |
| Total | 100.00 |

[1]Analyzed mineral composition: Ca % (1.25), K % (1.08), P % (1.02), S % (3.6), Na % (.21), Mg % (.19), Cu ppm (11.13), Fe ppm (481.4), Mn ppm (360.4), Mo ppm (4.94), Se ppm (32.35), ZN ppm (148.25).

[2]Mix Supplied per kilogram of diet: 14,109 I.U. Vitamin A, 5291 I.U. vitamin $D_3$, 47.62 I.U.vitamin E, .014 mg vitamin $B_{12}$, 8.82 mg Riboflavin, 26.5 mg Niacin, 28.2 mg d-pantothenic Acid, 705.5 mg Choline, 1.16 mg Menadione, 1.176 mg Folic Acid, 3.52 mg Pyridoxine, 3.52 mg Thiamine, .176 mg d-Biotin.

[3]Mix supplied per kilogram of diet: 160 mg Ca, 100 mg Zn, 120 mg Mn, 75 mg Fe, 10 mg Cu, 2.5 mg Iodine.

Table 2. Urinary, fecal excretion and mineral balance of broilers housed at TN and HD environments (Experiment 1)

| | Parameters | | | | | |
|---|---|---|---|---|---|---|
| | Fecal Excretion[1] | | Urinary Excretion[1] | | Mineral Balance[1] | |
| Mineral | TN[2] | HD[3] | TN | HD | TN | HD |
| $Ca^{+2}$ | 337 | 377 | 7.69 | 7.67 | 268 | 163 |
| $P^{-3}$ | 214 | 239 | 46[b] | 93[a] | 271[a] | 59[b] |
| $K^{+}$ | 213 | 206 | 57[b] | 286[a] | 299[a] | 57[b] |
| $Na^{+}$ | 78 | 75 | 20 | 34 | 10.3[a] | -52.8[b] |
| $Mg^{+2}$ | 60.2[b] | 77.7[a] | 9.4[b] | 17.4[a] | 43.5[a] | 19.3[b] |
| $S^{-2}$ | 53 | 68 | 73[b] | 140[a] | 1976[a] | 1751[b] |
| $Mn^{+2}$ | 5.0 | 5.4 | .02 | .02 | 15.3[a] | 12.9[b] |
| $Se^{-2}$ | .26[b] | .35[a] | .28 | .26 | 1.4[a] | 1.1[b] |
| $Fe^{-2}$ | 17 | 16.1 | .13 | .13 | 9.5 | 5.4 |
| $Zn^{+2}$ | 4.7 | 4.8 | .58 | 1.1 | 3.1[a] | 1.7[b] |
| $Mo^{+6}$ | .12 | .14 | .06 | .11 | .04[a] | -.05[b] |
| $Cu^{+}$ | .67[b] | .87[a] | .023 | .023 | -.04[a] | -.26[b] |

[a,b]Means within a row and major column heading with unlike superscript differ (P<.05).
[1]Expressed as mg/kg body weight.day$^{-1}$.
[2]TN=thermoneutral, HD=heat distress.

Table 3. Urinary mineral excretion of broilers housed at TN and HD environments (Experiment 2)

| | Urinary mineral excretion[1] | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Temp[2] | $Ca^{+2}$ | $K^{+}$ | $P^{-3}$ | $S^{-2}$ | $Na^{+}$ | $Mg^{+2}$ | $Mn^{+2}$ | $Se^{-2}$ | $Fe^{-2}$ | $Zn^{+2}$ | $Mo^{+6}$ | $Cu^{+}$ |
| TN | $1.8^{b}$ | $26.0^{b}$ | $37.9^{b}$ | $20.5^{b}$ | $6.4^{b}$ | $4.3^{b}$ | $.01^{b}$ | $.06^{b}$ | .02 | .02 | .17 | .03 |
| HD | $4.0^{a}$ | $54.2^{a}$ | $60.4^{a}$ | $26.0^{a}$ | $11.0^{a}$ | $6.5^{a}$ | $.02^{a}$ | $.16^{a}$ | .03 | .03 | .17 | .03 |

$^{a,b}$Values within a column with different superscript differ ($P<.05$).

[1]Expressed as mg/Kg body weight.12 $hr^{-1}$.

[2]Temp=temperature, TN=thermoneutral, HD=heat distress

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# CHAPTER V

## BROILER THERMOBALANCE AS INFLUENCED BY ENVIRONMENT AND WATER CONSUMPTION

Belay, T., C. J. Wiernusz and R. G. Teeter
Department of Animal Science, Oklahoma State University
Stillwater,74078.

## Abstract

Overall bird thermobalance is determined by the summation of heat production (HP) with nonevaporative (NVC) and evaporative (EVC) heat loss. Broilers housed in thermoneutral (TN, 24°C), environments have little trouble maintaining normal body temperature (BT) while during cycling temperature heat distress (HD, 24-35°C) heat loss is insufficient and body temperature (BT) rises. To evaluate relationships between water consumption and bird thermobalance 10 colostomized, Cobb x Cobb broilers were placed in individual respiratory chambers at 49 days posthatching and force fed 3% of body weight (BWT) 2 x daily. Voluntary Water consumption (WC), retention (WR) and excretion (WE) increased ($P<.05$) by 76.4, 59.7 and 144% respectively for HD broilers. Osmolar and free water clearance increased ($P<.05$) for HD broilers with urine excretion increasing hypoosmotically. Evaporative cooling

(r=.73), respiration efficiency (r=.73), urine production (r=.72) and free water clearance (r=.74) were all significantly (P<.05) correlated with water consumption. Plasma $Na^+$ and $K^+$ were decreased (P<.05) while $Cl^-$ was increased (P<.05) by heat distress. In a second study , 24 birds were used to evaluate the influence of HD on water excretion at fixed feed (3.5%/bwt) and water consumption (8.5%/bwt). Similar to the first study, water loss was increased (P<.05) by 63% in HD broilers despite equalized consumption. In the third experiment, 12 intact broiler were offered drinking water supplemented with 0, and 0.75 % Kcl and were placed in an individual respiratory chamber at HD environment. In the third experiment, as in the first, both evaporative cooling and respiration efficiency were increased (P<.05) by increasing water consumption. These studies illustrate mechanistically the relationship between bird water consumption and thermobalance providing a basis for therapeutic development.

KEY WORDS: heat distress, water balance, urine, evaporative cooling, clearance rate.

## Introduction

Bird heat production is inversely related to ambient temperature (Van Kampen, 1981a). During low ambient temperature exposure, heat production is elevated so that enhanced sensible heat loss is compensated for and body temperature maintained. However, heat production data for

birds exposed to heat distress are inconsistent. Higher heat production for adult poultry during heat distress have been reported by Van Kampen, (1974), while Chawlibog and Eggum, (1989) and Chawlibog, (1990) reported an inverse relationship between heat production and ambient temperature. Despite the bird's elevated heat load, Robertshaw (1981) suggested that heat production would be elevated due to cooling mechanisms activation. Wiernusz et al. (1991) observed that heat production during heat distress parallels feed consumption and suggested that variability associated with its measurements may related to variation in feed consumptions.

All poultry classes depend more on nonevaporative cooling for heat dissipation when housed in low and thermoneutral temperature environments (Romijn and lokhorst, 1966; Arieli et al., 1980; Van kampen, 1981b). Birds manipulate nonevaporative cooling primarily by reducing peripheral resistance to blood flow and concomitantly increasing resistance to the viscera, thereby shunting blood and hence heat to peripheral tissues (Bottje and Harrison, 1984). During heat distress increased blood flow to the comb and wattles have been reported as an important mechanism to dissipate core body heat (Michael and Harrison, 1987). A second, though less important, avenue of nonevaporative cooling is increased urine production when the water loss is compensated with consumed water (Van kampen, 1981a). Water consumption increases during high

ambient temperature (NRC, 1981). Whether due to increased water consumption, or other physiological effects, water diuresis have been observed in layers during summer (Okumura et al., 1977).

Nonevaporative heat dissipation efficiency declines as ambient temperature rises due to the reduced differential between the bird and ambient temperature (Van Kampen, 1974; Wiernusz et al., 1991). Consequently, above the critical ambient temperature birds start panting in order to dissipate excess heat by increasing evaporation of water from the respiratory tract (Van kampen, 1981a). Such body cooling mechanisms increase respiratory rate and minute volume, while decreasing respiratory amplitude and tidal volume (Arad, 1983).

The increased respiration rate of during heat distress broilers precipitate respiratory alkalosis as evidenced by increased pH and decreased blood $Co_2$ and bicarbonate levels (Teeter et al., 1985; Bottje and Harrison, 1985). Plasma electrolyte concentrations are also altered during heat distress (Kohne and Jones, 1975; Edens, 1976; Deyhim et al., 1990 ). Increased potassium excretion has been related to respiratory alkalosis in broilers (Smith and Teeter, 1987a) and layers (Deetz and Ringrose, 1976).

Various chemical compounds have been administered to poultry classes in an effort to modulate heat distress physiological effects. Drinking water $NH_4CL$ and $NaHCO_3$ supplementation (Teeter et al., 1985; Branton et al., 1986)

and salt (Teeter et al., 1987) have been reported to improve heat distressed broiler growth rate. Bottje and Harrison (1985) reported that $NaHCO_3$ supplementation in drinking water reduced heat distress mortality. However, Teeter (1988) indicated that other parameters also play a critical role in the broiler's physiological response to heat distress. Branton, et al., (1986), reported regression coefficients of .03 and -.72 for blood pH and water consumption, respectively suggesting that water consumption being major than blood pH as a factor for survival of heat distressed broilers.

Teeter and Smith (1986) reported that heat distressed broilers drinking water supplementation with levels of .05, .10, or .15% KCl increased weight gain linearly. They suggested that KCl was without effect on blood pH. In a similar study, Smith and Teeter (1987b) reported administering .2 % KCl via drinking water for heat distressed broilers also increased gain response over controls receiving tap water. Broilers receiving salt supplemented drinking water consumed more water and exhibited greater weight gain, suggesting that the gain response may have been partially due to increased water consumption acting as a heat sink as was correlated to a decline in rectal temperature. Fox (1951) demonstrated that when laying hens were exposed to a temperature of 42° C, a longer survival time associated with the persistency with which a bird continued to drink water. Increased carcass

water uptake during chilling for heat distressed broilers (Izat et al., 1989) coupled with higher carcass dry matter (Chwalibog and Eggum, 1989; Chwalibog, 1990) and elevated urine excretion (Van Kampen, 1981a) suggests that significant alterations in water balance occurred. However, studies in water balance and the effect of consumption water on bird theromobalance are limited.

The objective of the three experiments reported herein were to evaluate the effects of high ambient temperature distress on broiler water consumption, urine and fecal water excretion, plasma osmolality, blood electrolyte concentration, free water and osmolal clearance and thermobalance including HP, NEC, EVC, RPM and respiration efficiency.

## Materials and Methods

### Experiment 1

The first study was conducted to establish the effects of ambient temperature on broiler water consumption and thermobalance. The Cobb x Cobb male broilers used in the study were fed a 23% crude protein corn-soybean based ration and reared in rice hull covered floor pens through 21 days posthatching. On day 22 birds were transferred to individual 47 x 26 cm wire floored cages housed within an environmentally controlled room. Upon completion of the $4^{th}$ week birds were colostomized Belay et al. (1990) and fed a grower ration (Table 1). Following a 2 1/2 weeks recovery

period, 10 broilers with similar body weight and free of surgical and health complications were selected and implanted with a radiotelemetry temperature transmitter (Mini-Mitter telemetry system, Sunriver, Oregon 97707) in the abdominal cavity as described by Misson (1974). On day 2 birds were placed in individual respiration chambers (51 x 34 x 41 cm) for a 2 day chamber adaptation as recommended by Misson (1974). At 49 days posthatching following a 10 hr fast birds were precision fed (Teeter et al., 1984) the grower mash at 3 % of body weight 2 x daily. On day 50 birds were exposed to a 12 hr of thermoneutral ambient temperature (TN, 25° C) period. Following the TN phase birds were exposed to a gradually (1.7C/hr) increasing ambient temperature till chamber temperature reached 35° C for 6 hr. Water was offered for ad libitum consumption throughout the experiment. Separate total feces and urine excrements were collected in a plastic bag and 1 ml blood was collected from each bird at the start and end of TN and HD phases. Bird response variables monitored included water consumption, urine production, fecal water, plasma electrolyte concentrations ($Na^+$, $K^+$ and $Cl^-$) and both plasma and urinary osmolality.

Total urine and fecal were gravimetrically determined for each bird and fecal samples oven dried for dry matter analysis (Van kampen, 1981). Plasma samples were analyzed for sodium and potassium concentration by flame photometry. Plasma osmolality was determined on 8 ul samples using

Vapour Pressure Osmometer (5100B, Wescor Inc.) while chloride was measured by Chloride titerater (Model 4425000, Haake Buchler Instru. Inc.). Osmolal clearance was calculated as urine volume X the urine to plasma osmolality ratio and free water clearance as the difference between urine volume and osmolal clearance (Best and Taylor, 1990).

Variables within the distressed environment were evaluated for deviation from the thermoneutral subdivided into two water consumption levels in an effort to identify the relationship of drinking water to bird thermobalance. In addition, $O_2$ consumption, $CO_2$ production, core body temperature, respiration rate, evaporative cooling and nonevaporative cooling were monitored for each bird as follows:

Respiratory Chambers

The 12 (51 x 34 x 41 cm) respiratory chambers were constructed of clear 63.5 mm acrylic plexiglas and fitted with Hart watering cup attached to a 1000 ml graduated cylinder (water reservoir). Water disappearance from the cylinder was computed such that water consumption could be monitored. A wire mesh floor (51 x 34 cm) was suspended 9 cm above a 51 x 34 cm excreta collection pan containing 4 cm of mineral oil such that excreta moisture was isolated from the chamber environment. Each compartment was fitted with a 3 cm fan (Radio Shack cooling fan cat. # 273-244), located at the top center of each chamber, to mix air and thereby

insure gas sampling uniformity. Temperature probes (model ES-060 Omnidata International, Logan, UT. 84321) were used to monitor chamber temperature throughout the experimental period.

Bird Breathing Air supply

Air presented to the birds for respiration processes was compressed and dried by a compressed air dryer (Hankison, Cannonsberg, PA. 15317). Incoming air for each chamber moved through independent 64 mm tubing within the environmental chamber such that it reached chamber ambient temperature prior to entery into the chamber. The respiratory chambers sustained approximately 8.5 mm Hg positive pressure created by the 5 liter per minute flow rate through each chamber. All flow rates were monitored by an electronic mass flow meter (Omega Engineering, Stanford, CT 06907) and air flow fluctuated less than $\pm$1% throughout the experiment.

Relative humidity O2 and Co2 Analysis

Air moisture, $O_2$ and $CO_2$ concentration of bird breathing air were determined by an Omnidata International (Logan, UT. 84321 ) relative humidity probe (accuracy $\pm$1%) and Ametec (Pittsburgh, PA. 15238) $O_2$ (accuracy$\pm$.2%) and $CO_2$ (accuracy$\pm$.03%) analyzers respectively. Oxygen consumption ($O_2$ cons) and carbon dioxide production ($CO_2$ prod) of each bird were calculated by computing the difference between gas

concentration of the reference and test chambers and multiplying by the chamber flow rate

$O_2$ cons = (flow * ($O_2$/100))c - (flow * ($O_2$/100))b

$CO_2$ prod = (flow * ($CO_2$/100))b - (flow * ($CO_2$/100))c

Heat Production (HP)

The equation of Brouwer (1965) was used to estimate heat production (kJ/hour) from liters oxygen consumed and liters carbon dioxide produced:

HP = 16.18 $O_2$ consumed + 5.02 $CO_2$ produced.

No correction was utilized for nitrogen excretion as the error created by its omission is about .2 % (Romijn and Lockhorst 1961, 1966).

Metabolic Water (MW)

The equation of Van Es (1969) was used to estimate metabolic water production (g/hr) from liters oxygen consumed and liters carbon dioxide produced:

MW = 0.181 $O_2$ consumed + .490 $CO_2$ produced.

No correction was utilized for nitrogen excretion as the error created by its omission is about 1 to 2% (Van kampen, 1981a).

Evaporative Cooling (EHL)

Bird Water evaporative heat losses (respiratory + cutaneous) were estimated by coupling evaporative losses with the latent heat of vaporization (Van Kampen, 1981b). To estimate water evaporative loss RH was converted into g

$h2o/L^3$ breathing gained as the air passes through the respiratory chambers. To accomplish this g $h2o/l^3$ air at saturation for various ambient temperature (Weast, 1987) was regressed against ambient temperature and the resulting equation:

$$satH_2O = 8.694 - .218391 \times T + .03145 \times T^2$$

where

$satH_2O$ = saturation of water

used to convert RH into water concentration for the test and reference chambers. Bird water production per liter was then estimated as:

$$H_2O = satH_2O \times (rh/100) \times .001$$

$$H_2Oprod = (flow \times H_2O) - (flowref \times H_2Oref) \times 60$$

where

$H_2Oprod$ = water production (g/min)

$$EHL = H_2Oprod \times 2.365$$

where

EHL = evaporative water loss (kJ/min)

2.365 = latent heat of vaporization (kJ/g $H_2O$)

Bird Heat Content (HC)

Body temperature observations made utilizing the radio telemetry system described previously with data recorded once every 1.5 minutes for each bird. Body heat content change was estimated by utilizing body temperature change (dT), bird mass (kg) and specific heat (SH) as: dT X kg x

SH. The mean specific heat of body tissue estimate of 3.17 $kJ/kg \cdot C^{-1}$ was used (Wiernusz, 1991)

Nonevaporative Cooling (NHL)

Nonevaporative heat loss ($kJ/MBW \cdot hr^{-1}$) was estimated by the following equation: HP - EC $\pm$ HC (Sturkie, 1986). Estimating nonevaporative heat loss permitted the calculation of total bird thermobalance. Errors associated with this type of calculation include the fact that nonevaporative heat loss is calculated as a difference and not estimated directly.

Respiration Rate (RR)

During a respiration cycle (inhale and exhale) chamber pressure varies. Bird respiration rate may therefore be computed by determining the number of chamber pressure cycles over a timed interval. A Columbus Instruments respiration monitor (Columbus, Ohio 43204) used to estimates respiration rate 8 times/ hr.

Respiration Efficiency (RE)

Since cutaneous moisture loss from birds represents only 15 % of total evaporative heat loss during heat distress (Van kampen, 1974), an estimate of respiration efficiency (joules/Breath) was obtained by relating EC with RR as: EC/RR.

The chamber environment was controlled and all data measurements recorded by Workhorse Data Acquisition and

Control System (Omega Engineering, Stanford, CT 06907). Gas concentration quantifications ($O_2$, $Co_2$, RH) and ambient temperature were made and recorded once on each of the 10 compartment every 12 minutes.

## Statistical Analysis

Response variables, which included: Heat production, metabolic water production, nonevaporative cooling, evaporative cooling, heat content, respiration rate and respiration efficiency were regressed against time in each environment utilized the General Linear Model of the Statistical Analysis System (SAS, 1985) such that time dependent polynomial equations could be used to describe the data. Comparison between environments for all variables were made from integrated values by analysis of variance (Steel and Torrie, 1960).

## Experiment 2

The second experiment was conducted utilizing twenty four Cobb x Cobb male intact broilers to evaluate the effect of ambient temperature on fecal water excretion. Birds were fed a 23% crude protein corn-soybean based ration and handled as described in the first experiment. On day 45 posthatching 24 broilers with similar body weight were selected and divided into two groups of 12 birds. Each group was randomly allocated to individual cages housed within two environmentally controlled TN (24°C) or HD (32° C) rooms. The experiment was initiated after each bird in

each of the environmental rooms were force fed (Teeter et al., 1984) a grower ration (Table 1) 3.5 % and drenched water 8.5 % of body weight, respectively. During the 4 hrs experimental period, total excrement from each bird was collected for determination of fecal water excretion. Fecal water was determined by subtracting total oven dried fecal sample from total wet fecal weight.

Experiment 3

The third experiment was conducted to examine water consumption effects on thermobalance of heat distressed broilers in greater detail. Twelve birds were used in a protocol similar to experiment 1 with the exception that 6 week old intact birds were used and 2 days prior experiment initiation bird drinking water was supplemented with 0 % or 0.75 % potassium chloride. Water was offered for ad libitum consumption during the 8 hrs experimental period.
Bird response variables monitored included: water consumption, HP, EVC, RPM and respiration efficiency. Variables were regressed against time utilized the General Linear Model of the Statistical Analysis System (SAS, 1985) such that time dependent polynomial equations could be used to describe the data. Comparison between Kcl treatment for all variables were made from integrated values by analysis of variance (Steel and Torrie, 1960).

## Results and Discussion

Experiment 1

Water consumption (table 2) of heat distressed broilers was increased ($P<.05$) similar to other studies (Vo et al., 1978; NRC, 1981; Van kampen, 1981a; Teeter, 1987; Lott, 1991; Whiting et al. 1991)). Since bird feed intake was controlled by force feeding, water contribution from feed was the same between the environments assuming no environmental effect on ration digestibility and metabolizability. The contribution of metabolic water decreased ($P<.05$) by 31.6 % with heat distress in agreement with work reported by Van kampen (1981a). The decline in metabolic water would be expected as HP estimate by indirect calorimetry decline during HD (Chwalibog and Eggum, 1989). This has been postulated to be related to bird acclimation to HD. Nonetheless, bird metabolic water contribution to total water consumption is less than 15%.

In order to evaluate water consumption relationship in broilers were divided into low (consumption 1 ) and high (consumption 2), such that the higher consumption in the TN environment was similar to the lower consumption in HD. Therefore, the following results of measured parameters are reported in reference to consumption 1 and consumption 2 in the respective environment.

Effects of ambient temperature and water consumption levels on urine volume, urine and plasma osmolality, free water and osmolal clearance, and fecal water excretion for

experiment 1 are displayed in table 3. Heat distressed birds had higher ($P<.05$) urine production, free water and osmolal c and decreased ($P<.05$) urine osmolality. Plasma osmolality ($P>.28$) and fecal water content were not affected ($P>.2$) by environmental temperature nor water consumption level. Urine production was increased ($P<.05$) for the highest water consumption level in the TN but not in HD. Results for effect of heat distress on urinary excretion are similar to Van Kampen (1981) who used colostomized birds and laying hens (Okumura et al., 1977) This was possibly due to increased urine production as reported in this study. Similarly, urine osmolality was reduced ($P<.05$) with increased water consumption in the TN but not HD environment. Water consumption level did not affect ($P>.2$) free water or osmolal clearance in either environment. However, osmolal clearance was increased ($P<.05$) by HD but was not affected by water consumption level. Correlation between water consumption and urine production and free water clearance were significant ($P<.05$) at $r=.72$ and $r=.74$, respectively.

Urinary osmolality was reduced ($P<.05$) by heat distress. Inverse relationships between urine osmolality and urine flow rates has been demonstrated (Wideman and Gregg, 1987) similar to results reported herein. The effect of heat distress on increasing hypotonic urine production could be due to increased water consumption observed in heat distress. However, it has been shown that urine flow rate

is reduced with lower renal arterial perfusion and pressure (Wideman and Gregg, 1987). In extreme cases of dehydration, blood flow to the kidney nephrons has been reported to stop causing urine formation to cease (Nishimura, 1985). During heat distress bird blood pressure is reduced and blood is shanted to the periphery to transport body heat (Micheal and Harrison, 1987). Such cardiovascular changes coupled with increased water consumption during HD might result in increased renal arterial perfusion and as a result a greater glomerular filtration rate and urine flow.

Glomerular filtration rate (GFR) estimated by the ratio of plasma osmolality to urine osmolality in this study was 1.5 and 2.3 for TN and HD, respectively. The value of GFR in the TN of this study agrees with Dicker and Haslam (1966). The positive and higher ($P<.05$) free water clearance observed in the HD broilers of this study suggests a failure of kidney tubules to reabsorb water. Studies on the effect of ambient temperature on free water and osmolal clearance are limited making it difficult to contrast data reported herein with others. Increased osmolal clearance and reduced water reabsorption observed in HD broilers might also contribute to increase electrolyte wash out. In this study plasma osmolality was not affected ($P>.28$) by ambient temperature nor by water consumption levels. Reported studies are inconsistent on effect of heat distress on hematocrit. Similar to this study Deyhim et al. (1990) were not able to observe differences in hematocrit of broilers

housed in TN and HD, while decreased hematocrit value for HD birds was reported by Kubena et al. (1972). Possible reasons for inconsistency on the effect of ambient temperature on hematocrit values could arise from differences in ambient temperature range and pattern employed during the distress period and bird water availability during experimentation.

Relationship between ambient temperature and water consumption level on broiler thermobalance are shown in Table 4. Heat distress reduced ($p<.05$) broiler heat production and nonevaporative cooling by 24 and 51 %, respectively. Evaporative cooling increased ($p<.05$) by 298% due to heat distress but failed to completely compensate for the nonevaportative cooling decline, consequently bird heat content increased ($P<.05$) during heat distress. The results for heat production, evaporative and nonevaporative cooling reported in this study are in agreement with others (Van Kampen, 1981a; Chwalibog and Eggum, 1989; Chwalibog, 1990). Respiration frequency was elevated ($P<.05$) by heat distress. Higher water consumption was associated with increased ($P<.05$) respiration frequency and HP in the TN while higher water consumption was associated with reduced ($P<.05$) respiration frequency and higher ($P<.05$) efficiency in the heat distressed environment. Though, it was not statistical different bird body heat content in the heat distress environment was lowered by 9.1% with increased water consumption.

During heat distress elevated water consumption was significantly (P<.05) correlated (r=.74) with higher (P<.05) evaporative cooling (7.1 %). Despite the reduced respiration rate in the HD birds consuming more water, respiration efficiency as measured by joules of heat dissipated per breath was significantly (P<.05) correlated (r=.73) (41.7 %) with water consumption. Such cooling effect of drinking water in heat distress birds was also reported by Arad (1983).

Plasma electrolyte concentrations for broilers used in experiment 1 are shown in table 5. Plasma sodium and potassium were reduced (P<.05) by heat distress, while chloride increased (P<.05). Water consumption levels did not impact (P>.2) plasma electrolyte in either environment. The effect of ambient temperature on plasma electrolyte concentration, while plasma osmolality is not affected is unclear. Nonetheless, the result reported in this experiment for plasma sodium and potassium are in agreement with Huston (1978) and Deyhim, et al. (1990), but not with Kohne and Jones (1975) who observed blood potassium levels to increase during heat distress. Edens (1976) observed increased broiler sodium excretion and lowered plasma sodium in heat distressed broiler as reported in this study. Effects of respiratory alkalosis compensatory mechanisms have been implicated to increase potassium excretion in poultry during heat distress (Deetz and Ringrose, 1976; Smith and Teeter, 1987a). Therefore, the reduced plasma

potassium observed in this study possibly is due to increased urinary excretion.

The increased plasma chloride in the heat distressed broilers agrees with the result of Kohne and Jones (1975) who observed similar responses in heat distressed turkeys. Respiratory alkalosis, occurring during heat distress (Teeter et al., 1985; Bottje and Harrison, 1985) will reduce plasma $Co_2$ and $HCo_3^-$. Therefore, the elevated plasma chloride observed in this experiment and reduced urinary chloride excretion in broilers during heat distressed (Belay and Teeter, 1991) is possibly a kidney function to regulate acid-base imbalance and to compensate for reduced plasma bicarbonate. To derive concert evidence on the role of kidney in acid-base regulation during heat distress need further studies.

Experiment 2

The result of the experiment, conducted to ramify the effect of ambient temperature on fecal water excretion is shown on figure 1. In this experiment heat distress increased ($P<.05$) excreta water excretion despite equalized feed and water consumption. Since ambient temperature effect on excreta water in experiment 1 were of urinary origin, the effects observed here are also likely attributable to urinary origin.

Experiment 3

The results of experiment 3, conducted to further elaborate the result on the relationship between consumption water and thermobalance observed in experiment 1 are shown in table 6. Drinking water supplementation with potassium chloride increased ($P<.05$) water consumption compared to birds consuming tap water. This response is to results previously reported (Teeter and Smith, 1986; Smith and Teeter, 1987b). Other salts have also been reported to increase water consumption and heat distress broilers survivability (Bottje and Harrison, 1985; Branton, et al., 1986; Teeter et al., 1987).

Heat production was not affected by potassium chloride supplementation as was not expected since birds were force fed 43 % of metabolic body weight. In contrast evaporative cooling was increased ($P<.05$) by 19 % in potassium chloride supplemented birds. Similar to data reported in experiment 1, respiration efficiency was correlated with water consumption. Enhancing water consumption of HD broilers has the potential to act as a heat sink (Teeter et al., 1987) and based on the results these experiments are also has the potential to increase evaporative cooling and respiratory efficiency.

In summary, the results of these experiment indicate that manipulating broiler water availability during heat distress has the potential to improve bird thermobalance. The results of Fox (1951) that persistency of drinking water of a laying hen exposed to ambient temperature of 42° C, was

association with longer survival time. The results of experiments reported herein indicate drinking water increases respiration efficiency, helping the bird to cope with the temperature distress. Water beneficial effects to improve HD bird survivability could also be related to reduced respiration rate which would alleviate respiratory alkalosis. The elevated plasma chloride is likely a kidney compensatory response for prevailing respiratory alkalosis.

Table 1. Composition of ration used for the experiments

| Ingredients | Percent |
|---|---|
| Ground corn | 61.12 |
| Soybean meal (48.5%) | 31.06 |
| Fat | 3.8 |
| Dicalcium phosphate (22% ca; 18.5% P) | 1.70 |
| Limestone (38% Ca) | 1.30 |
| salt | .40 |
| Vitamin mix[1] | .30 |
| DL-Methionine, 99% | .22 |
| Trace mineral mix[2] | .10 |
| Total | 100.00 |
| Calculated analysis: | |
| ME Kcal/Kg | 3128.4 |
| Crude protein (%) | 20.60 |
| Calcium (%) | 1.00 |
| Phosphorus (% av) | .44 |

[1] Mix contained per kilogram: 3,690,280 I.U vitamin A, 1,102,300 I.U vitamin $D_3$, 13,228 I.U vitamin E, 7.9 mg vitamin $B_{12}$, 2646 mg Riboflavin, 17,637 mg Niacin, 4,409 mg d-panthotenic Acid, 200,178 mg choline, 728 mg Menadione, 440 mg Folic Acid, 1,584 mg Pyridoxine, 792 mg Thiamine, 44 mg d-Biotin.

[2] Mix supplied per kilogram of diet: 140 gm Ca, 100 gm Zn, 120 gm Mn, 75 gm Fe, 10 gm Cu, 2.5 gm Iodine.

Table 2 Effect of ambient temperature on broiler water availability and consumption levels (g) (Experiment.1)

| | Environment | |
|---|---|---|
| Source | Thermoneutral | Heat Distress |
| Drinking | 122.8[b] | 219.4[a] |
| Feed | 48.2 | 51.1 |
| Metabolic | 10.4[a] | 7.9[b] |
| Mean total | 181.4[b] | 278.4[a] |
| Consumption 1 | 134.1[c] | 222.4[b] |
| Consumption 2 | 228.7[b] | 334.4[a] |

[abc]Means within a row with unlike superscripts differ (P<.05)

Table 3 Effect of ambient temperature distress and water consumption on urine output, fecal water, osmolar clearance and osmolality (Expriment 1)

| Parameters | Thermoneutral | | Heat Distress | |
|---|---|---|---|---|
| | Cons 1[1] | Cons 2[2] | Cons 1[3] | Cons 2[4] |
| Urv[5] (ml/12 hr-1) | 51[c] | 75[b] | 150[a] | 158[a] |
| Ur. Osm[6] (mosm/kg) | 240[a] | 134[b] | 153[b] | 119[b] |
| Pl. Osm[7] (mosm/kg) | 310 | 309 | 307 | 307 |
| $C_{H2O}$[8] (ml/12 hr-1) | 13[b] | 44[b] | 76[a] | 95[a] |
| Osm Clr[9] (ml/12 hr-1) | 38[b] | 31[b] | 74[a] | 63[a] |
| FW[10] (g/kg bwt/12 hr) | 32 | 29 | 22 | 19 |

[a-c]Means within a parameter and row with unlike superscripts differ (P<.05)

[1] Cons 1=Water consumption (134.1 g)
[2] Cons 2=Water consumption (228.7 g)
[3] Cons 3=Water consumption (222.4 g)
[4] Cons 4=Water consumption (334.4 g)
[5] Urv=Urine volume
[6] Ur. Osm=Urinary osmolality
[7] Pl=Plasma osmolality
[8] $C_{H2O}$=Free water clearance
[9] Osm Clr=Osmolal clearance
[10] FW=Fecal water gram per kg body weight

Table 4 Effect of ambient temperature on thermobalance and respiration efficiency in broilers subdivided by water consumption (Experiment 1)

| Parameters | Thermoneutral | | Heat distress | |
|---|---|---|---|---|
| | Cons[1] 1 | Cons[2] 2 | Cons[3] 1 | Cons[4] 2 |
| HP[5] (kJ/mwt·hr$^{-1}$) | 28.9$^{b}$ | 30.6$^{a}$ | 22.2$^{c}$ | 23.0$^{c}$ |
| NVC[6] (kJ/mwt·hr$^{-1}$) | 25.7$^{b}$ | 27.5$^{a}$ | 13.4$^{c}$ | 13.5$^{c}$ |
| EVC[7] (kJ/mwt·hr$^{-1}$) | 2.8$^{c}$ | 3.1$^{c}$ | 8.5$^{b}$ | 9.1$^{a}$ |
| dHC[8] (kJ/mwt·hr$^{-1}$) | -.03$^{b}$ | -.05$^{b}$ | .36$^{a}$ | .33$^{a}$ |
| RPM[9] | 30$^{d}$ | 42$^{c}$ | 73$^{a}$ | 54$^{b}$ |
| RESEFF[10] (J/Breath) | 1.6$^{c}$ | 1.2$^{c}$ | 1.9$^{b}$ | 2.8$^{a}$ |

$^{a-e}$Means within a parameter and row with unlike superscripts differ ($P<.05$)

[1] Cons 1=Water consumption (134.1 g)
[2] Cons 2=Water consumption (228.7 g)
[3] Cons 3=Water consumption (222.4 g)
[4] Cons 4=Water consumption (334.4 g)
[5] HP=heat production
[6] NVC=nonevaporative cooling
[7] EVC=evaporative cooling
[8] dHC=change in body heat content
[9] RPM=respiration rate/minute
[10] RESEFF=respiration efficiency (joules/Breath)

Table 5 Effect of ambient temperature and water consumption levels on plasma electrolyte concentration (meq/l) (Experiment. 1)

| | Thermoneutral | | Heat distress | |
|---|---|---|---|---|
| Electrolyte | Cons[1] 1 | Cons[2] 2 | Cons[3] 1 | Cons[4] 2 |
| $Na^+$ | 150.1[a] | 149.6[a] | 136.4[b] | 136.3[b] |
| $K^+$ | 5.1[a] | 4.9[a] | 4.0[b] | 3.9[b] |
| $Cl^-$ | 114.1[b] | 113.3[b] | 120.8[a] | 120.4[a] |

[ab]Means within a an electrolyte and row with unlike superscripts differ (P<.05)

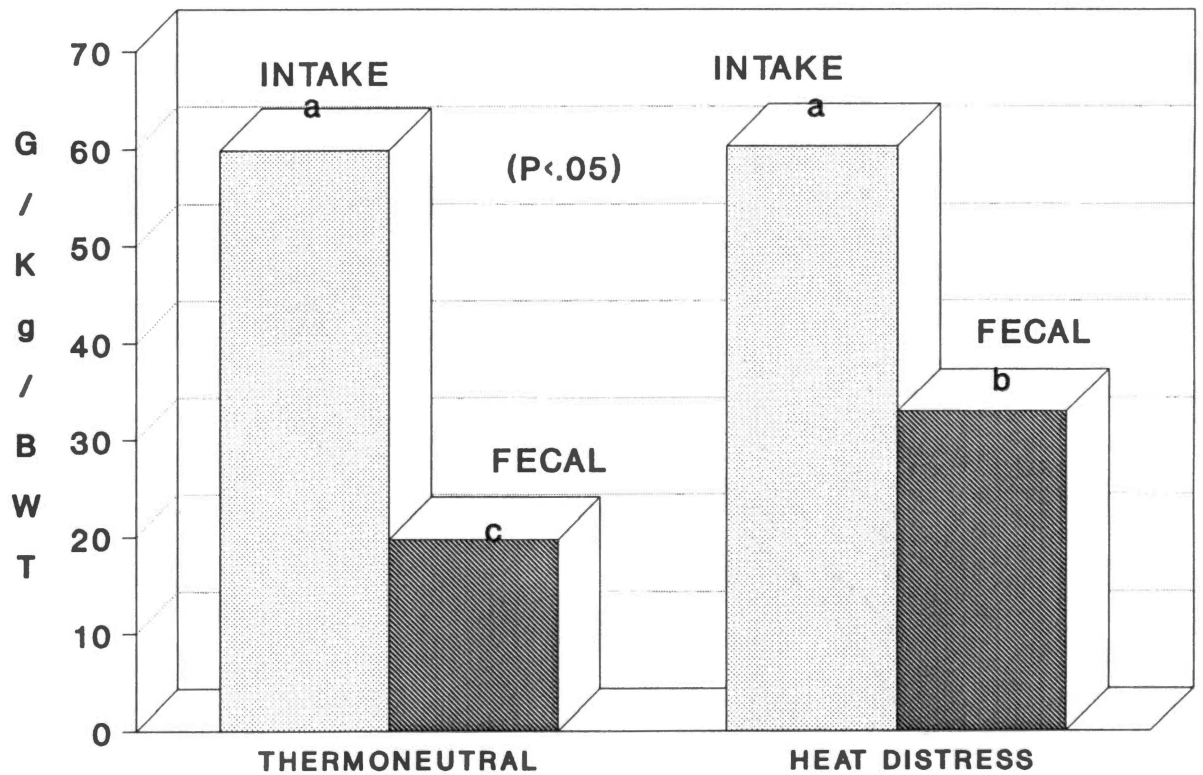


Figure.1 Ambient temperature effect on fecal water loss of intact broilers at equalized water intake

Table 6. Effects of water Kcl supplementation on thermobalance and respiration Efficiency of heat distress broilers (Experiment 3)

| Parameters | KCl (%) | |
|---|---|---|
| | 0 | .75 |
| Water cons[1] | 127[b] | 239[a] |
| HP[2] | 25.6 | 25.6 |
| RPM[2] | 74 | 69 |
| EVC[2] | 8.4[b] | 10.0[a] |
| RESEFF[2] | 1.9[b] | 2.4[a] |

[ab] Means within a parameter and a row with unlike superscripts differ (P<.05)

1 Water cons= Water consumption (ml/8 hr)
2 Look table 4 for abbreviations

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CHAPTER VI

# CALORIC DENSITY AND CALORIE-PROTEIN RATIO EFFECTS ON THE 4-7 WEEK POSTHATCHING BROILER GROWTH RATE, FEED CONVERSION, CARCASS FAT CONTENT AND SURVIVABILITY WHEN REARED IN THERMONEUTRAL AND HEAT DISTRESS ENVIRONMENTS.

Belay, T. and R.G. Teeter
Animal Science Department, Oklahoma state University
Stillwater, OK 74078

## Abstract

Two experiments were conducted utilizing 1,872 male Cobb x Cobb broilers to evaluate caloric density and calorie-protein ratio effects on broiler growth rate, feed conversion, carcass fat and survivability when reared in thermoneutral (TN, 24° C ) and cycling temperature (25-35° C) heat distress (HD) environments. In the first study energy (2880, 3200 Kcal ME/kg) and calorie-protein ratio (140, 155) were evaluated in a 2 x 2 factorial treatment arrangement. Increasing energy level overall increased (P<.05) energy consumption and within HD reduced (P<.05) feed consumption. Increasing energy level numerically improved growth rate within the TN (2.5 %, P= .18) and HD (4.1 %, P= .13) environments. Calorie-protein ratio had

little effect on TN growth rate while the lower ratio was associated with increased feed consumption and growth rate in HD. Both heat distress and ration energy level increased percent carcass fat (CF) by 3.7 % (P<.07) and 12.9 % (P<.05), respectively. Mortality was increased (P<.05) by 13.7 % due to HD. Within HD mortality was reduced (P<.05) at 8.3% for the low energy high calorie-protein ratio ration compared to high energy and low calorie/protein ratio ration. In experiment 2, a 3 x 3 factorial combination of energy levels (2826, 3200, 3574) and calorie-protein ratios (140, 160, 180) were evaluated. Similar to the first study, gain linearly increased (P<.05) within TN and HD with increasing energy while increasing calorie-protein ratio decreased (P<.05) gain in both TN and HD environment. Mortality was increased by HD (10.2 %, P<.01) and within HD by increasing caloric density (P<.05). Lowering crude protein and/ or increasing calorie-protein ratio reduced (P<.05) mortality in HD at the lowest and highest energy levels. Carcass fat was increased (P<.05) in HD and by increasing ration metabolizable energy in both environments. These experiments, demonstrate that, increasing caloric density improves growth rate in TN and HD environments, that lower calorie-protein ratios are also associated with increased broiler growth rate in TN environment, but during HD elevated (P<.05) mortality.

KEY WORDS: broiler, Heat distress, calorie protein ratio,

## Introduction

It is well documented that both feed intake and growth rate decline during high ambient temperature-relative humidity distress (squibb et al., 1959; Cowan and Michie, 1978). However, it has been demonstrated that the heat distressed broiler retains some potential for enhanced growth rate as force feeding heat distress birds to levels mimicking thermoneutral controls elevated growth rate (Smith and Teeter, 1987). The authors also indicated, however, that the beneficial effects are not without cost as mortality was increased.

A number of dietary manipulations have been proposed to enhance growth rate of heat distressed birds. Kubena et al. (1972a) recommended increased ration protein and amino acid fortification levels to counter reduced feed consumption. Adams et al. (1962) reported that increasing ration caloric density by substituting fat for corn and increasing protein levels were beneficial in overcoming high ambient temperature distress effects on gain. The authors further indicated that increasing ration caloric density improved gain independent of environmental temperature similar to Dale and Fuller (1980). Specific environmental effects can not be ruled out as several studies suggested that the gain response is less pronounced at higher ambient temperatures (Fisher and Wilson, 1974; Abdelkarim et al., 1985; Sinurat and Balnave, 1985 and 1986).

In sharp contrast to Kubena's et al. (1972) recommendation Waldroup et al. (1976) recommended amino acid levels to improve growth rate of heat distressed broilers. The basic premise here was to reduce ration heat increment.

The degree of fatness of broilers has been reported to be influenced by several factors, of which the most widely investigated is the dietary calorie to protein ratio (Summers and leeson, 1979; Jones and Wiseman, 1985; Bartov, 1987). Reported data are consistent that increasing dietary calorie/protein ratio increase broiler fat pad weight and carcass fat. Rations formulated to contain high dietary crude protein are advantageous for reducing carcass fat in heat distressed broilers.

Broiler body fat content increases independent of diet by high ambient temperature distress (Swain and Farrel, 1975) with higher breast fat and lower meat to fat ratio (Sonaiya et al., 1990). Howlider and Rose, (1987) review numerous published papers and findings that total and abdominal fat increased by 0.8% and 1.6% respectively, with each degree rise in ambient temperature.
Sonaiya et al. (1990) suggested carcass fat is reduced by reducing ration caloric density and increasing protein. However, the benefits were overridden by the effects of high ration heat increments (Macnaughton and Reece, 1984). Though, the effects of varying ration caloric density and /or calorie/protein ratio on broiler growth rate, and carcass composition are exceedingly studied in thermoneutral

environment, information is scarce regarding such effects during high ambient temperature distress.

Therefore, the objective of the study reported herein was to evaluate caloric density and calorie protein ratio effects on broiler feed and energy consumption growth rate, feed conversion, mortality and carcass composition in birds housed in thermoneutral and cycling high ambient temperature distressed environments.

## Materials and Methods

Two experiments were conducted utilizing 1,872 male Cobb x Cobb broilers. In both experiments, day old chicks were fed a corn soybean meal based starter ration containing, 23 % crude protein, and raised on rice hull litter to 26 days of age. On day 26 birds were transferred to wire-floored grower battery compartments (61 X 82 cm) housed within two environmental chambers, chamber facility have been described elsewhere. The chambers were thermostatically and humidistatically controlled such that the thermoneutral (TN) designated chamber was maintained at 24 C and the heat distress (HD) chamber cycled between 24 and 35 C. Ambient temperature was increased 2 C daily to achieve the desired range and provided 6 hr in excess of 32.2° C. Relative humidity in both chambers was 55±5%. On day 28, following 2 day adaptation period, birds were fasted overnight, weighed, and the environmental phase was initiated. Feed and water were available for ad libitum

consumption throughout the experimental period. Mortality and weight of birds succumb during the experiment were recorded daily.

On day 49 posthatching birds and feed were weighed. Following the 21 day environmental period bird from each replication were slaughtered so that dressing percentage, fat pad weight and carcass specific gravity could be determined. Birds were randomly selected and processed as follows: Birds were weighed, hung on a rail, electrically stunned, bled for 15 min. following severing of jugular and carotid veins, passed through a scalding vat, plucking machine, hand eviscerated, and Carcass were weighed. In both experiments, dressing percentage was calculated as dressed carcass weight without the necks and giblets divided by live weight. Fat pad was hand removed from the fat surrounding the bursa of Fabricius, cloaca, and adjacent abdominal muscles. Fat pad weight was calculated as a percentage of dressed carcass weight. Carcasses were weighed in air and water for computation of specific gravity (Teeter and Smith, 1985). Carcass fat content was estimated from carcass specific gravity using the predictive equation of Teeter, (1991) by the relation: % carcass fat= 336.97008-(310.19727*specific gravity).

Experiment 1

The first experiment was conducted to evaluate the effect of caloric density and calorie/protein ratio in TN

and HD housed broilers. Treatments rations used in this experiment (Table 1) consisted of a factorial arrangement of 2 ambient temperatures X 2 caloric density (2880, 3200 Kcal ME/kg) X 2 calorie protein ratios (140 and 155). Experimental rations were formulated by altering ration Soybean meal, fat and starch contents. The study contained 16 and 8 replicates of 6 chicks each in the HD and TN environments, respectively.

Experiment 2

The second experiment was conducted to expand the caloric density and calorie/protein range. Treatments (Table 2) consisted of a factorial arrangement of 2 ambient temperatures X 3 caloric density (2826, 3200, 3574) X 3 calorie protein ratios (140, 160, 180). Ration amino acid content was controlled to exceed NRC (1984) recommendation with the 180 calorie/protein ratio by supplementing a crystalline amino acid and for the 140 C/P ratio by varying the quantity of soy bean meal and cellulose (a filler). The study was consisted of 16 and 8 replicates of 6 chicks each in the HD and TN environments, respectively.

The general linear model procedure of the Statistical Analysis system (SAS, 1985) was used to analyze the data in both experiments. Treatment means were separated by using Duncan's (1955) multiple range test.

## Results and Discussion

Results of experiment 1 are shown in tables 3, 4 and 5. Averaged over the experimental rations body weight gain, feed and energy consumption, gain/feed ratio and survivability were reduced (P<.05) during heat distress by 27.3 %, 21.5%, 22.1%, 18.0% and 11.8%, respectively. The responses are in agreement with other reported studies (Cowan and Michie, 1978; Teeter et al., 1987; Smith and Teeter, 1987).

In the heat distress environment, feed consumption of broilers was reduced (P<.05) by 5.7 and 5.2 % for the highest 3200 Kcal $ME_n$/Kg caloric density and calorie-protein ratio ration, respectively. Caloric consumption and adjusted gain/feed ratio were increased (P<.05) only for the increased rations caloric density. Within a calorie protein ratio, survivability in the heat distressed environment was reduced (P<.05) by the higher caloric density ration. Increased energy consumption effect on survivability of heat distressed broilers reported in this study were similar to previous studies (Kubena et al., 1972b; Sykes and Salih, 1986).

Caloric density and calorie protein ratio in the first study did not affect gain, feed consumption, gain/feed, survivability and adjusted gain/feed in the thermoneutral environment. However, energy consumption increased (P<.05) with increasing caloric density of the ration similar to the heat distress.

Considering the carcass parameter (Table 5), at 49 days posthatching heat distress increased (P<.05) dressing percentage and carcass fat (P<.07), but had no effect on fat pad percentage. Increased broiler carcass fat during heat distress has been reported by several workers (Janky et al., 1983; Howlider and Rose, 1987; Sonaiya et al., 1990). Significant (P<.05) interaction between environmental temperature X caloric density and environmental temperature X calorie protein ratio was observed for fat pad and dressing percentage. Increasing caloric density increased (P<.01) percentage fat pad within the thermoneutral environment, but not within the heat distressed environment. Higher calorie protein ratio increased (P<.09) dressing percentage in the heat distress. Increased ration caloric density tended to increase (P<.08) carcass fat in the TN and HD (P<.1) environments.

Results of the second experiment are shown in tables 6, 7, 8, 9, 10 and 11. Similar to the first experiment, heat distress reduced (P<.05) gain, feed and energy consumption, gain/feed ratio, survivability and gain/feed ratio adjusted for mortality. The results for main effects and interactions is displayed in table 11. The 3 way Ambient temperature X ration caloric density X calorie protein ratio interaction was not significant for the parameters shown in tables 6 and 7. However, the 2 way ambient temperature X caloric density interaction was significant (P<.05) for

gain, feed consumption, calorie consumption, gain/feed ratio and survivability.

In the heat distressed environment (Table 9 and 10) increasing caloric density increased ($P<.05$) weight gain linearly ($P<.001$). Feed consumption was not affected by ration caloric density levels, therefore, energy consumption increased linearly ($P<.01$). However, increased energy consumption was associated with reduced survivability hence gain/feed ratio not adjusted for mortality in the heat distressed environment. The results reported herein for caloric density effect during heat distress agrees with other published works (Abdelkarim et al., 1985; Sinurat and Balnave, 1985 and 1986).

Feed and energy consumption in the heat distressed environment was increased quadratically ($P<.05$) with increasing calorie/protein ratio. The ambient temperature X caloric protein ratio interaction was significant ($P<.05$) only for feed and energy consumption. However, heat distressed broilers fed 180 calorie/protein ratio rations had lower ($P<.05$) body weight gain compared to those received the 160 ratio. Bird survivability decreased ($P<.05$) as calorie/protein ratio declined, presumably as a result of increased ration heat increment. This observation is in agreement with Macnaughton and Reece (1984).

In the thermoneutral housed broilers weight gain, feed and energy consumptions were increased linearly ($P<.05$) with increasing caloric density. Gain/feed ratio was highest

(P<.05) at only the medium caloric density rations compared to same caloric density within the TN, due to a lower feed consumption. Increasing ration calorie/protein ratio at a constant caloric density tended to decreased (P<.08) weight gain, but did not affect other parameters. Donaldson (1985) reported that increasing calorie-protein ratio above 139 reduces growth similar to the trend observed in this study. The tendency for increased gain observed with reducing calorie-protein ratio in this study supports the hypothesis that protein increases growth rate and is in agreement with the study of Bartov (1987) and Jackson et al. (1982).

Results of carcass analysis for experiment 2 are shown in Table 8. Heat distress increased (P<.05) carcass fat, but the effect on percentage fat pad was minimal (P<.09). A significant (P<.05) interaction between environmental temperature X caloric density X calorie protein ratio was observed on dressing percentage. In heat distress environment dressing percentage was not affected by caloric density levels, but percent fat pad and carcass fat were increased (P<.05) with increasing caloric density. In the thermoneutral housed broilers a linear increase (P<.05) in dressing, fat pad and percent carcass fat were observed with increasing caloric density. Increasing calorie/protein ratio similarly increased (P<.05) both percent fat pad and carcass fat. Results reported in this study for the carcass analysis in the thermoneutral environment are similar with

others (Griffiths et al., 1977; Summers and leeson, 1979; Jones and Wiseman, 1985; Donaldoson, 1985; Bartov, 1987)

These studies indicated that weight gain of both heat distressed and thermoneutral housed broilers is increased by increasing ration caloric density from 2880 to 3200 Kcal/Kg $ME_n$ of ration. In experiment 2 weight gain was significantly ($P<.01$) correlated ($r=.18$) with ration caloric density. The increase in body weight gain with increasing caloric density was related to the increase in energy consumption. Correlation ($r=.64$) of gain with caloric intake was significant ($P<.05$). Suggesting that even the growth rate depression in heat distress can be partly minimized by increasing ration caloric density. However, other than the adverse effect on carcass fat, increasing ration caloric density was associated with a linear increased ($P<.05$) in mortality of heat distressed broilers. Significant ($P<.05$) negative correlation ($r=-.25$) was observed between caloric density and broiler survivability. At a constant caloric density, mortality of heat distressed broilers was elevated by decreasing the calorie/protein ratio. Evidences support that dietary fat decreases heat increment of diet (Dale and Fuller, 1979; 1980). Despite dietary fat differences, in the high caloric density 3574 Kcal $ME_n$ category (Table 2) mortality was not compromised. Suggesting that at higher caloric density the advantage of dietary fat to reduce ration heat increment is minimal. Feeding ration with higher calorie/protein ratio similar to

the high caloric density ration resulted higher carcass fat, suggesting that there was an energy surplus for fat deposition. Nonetheless, with lower calorie/protien ratio rations, since energy was possibly wasted for the elimination of excess nitrogen from the body it would have affected the feed utilization to result in reduced carcass fat. In heat distressed broilers the lower calorie/protien ratio resulted in higher mortality without affecting the feed intake. This indicates that increasing ration crude protein in order to increase protein intake could increase ration heat increment and thereby potentially increase bird heat load and mortality. Therefore, during high ambient temperature distress the practice of feeding high protein and caloric density rations may worsen the heat distress state.

These studies indicate that, during high ambient temperature when the bird is faced with the need to dissipate its heat load can be increased by ration formulation.

Table 1 Composition of experimental rations (%) (Experiment 1)

| | ME in feed (kcal/kg)[1] | | | |
|---|---|---|---|---|
| | 2880 Kcal/Kg | | 3200 Kcal/Kg | |
| | c/p ratio | | c/p ratio | |
| Ingredient | 140 | 155 | 140 | 155 |
| | (%) | | | |
| Ground Corn | 60.36 | 69.90 | 43.71 | 51.94 |
| Soybean meal(48.5%) | 34.64 | 28.20 | 43.16 | 36.30 |
| Fat | 0.90 | —— | 9.11 | 7.65 |
| Dicalphosphate | 1.60 | 1.68 | 1.51 | 1.58 |
| calcium carbonate | 1.25 | 1.25 | 1.24 | 1.25 |
| Salt | 0.41 | 0.41 | 0.41 | 0.41 |
| Vit. Mix[2] | 0.25 | 0.25 | 0.25 | 0.25 |
| D-L-Methionine | 0.21 | 0.21 | 0.21 | 0.21 |
| Trace Mineral[3] | 0.10 | 0.10 | 0.10 | 0.10 |
| Lysine | —— | 0.17 | —— | ——. |
| Filler | 0.30 | 0.70 | 0.30 | 0.30 |
| Starch | —— | 0.25 | —— | —— . |

[1]Calculated using values from National Research Council (1984)

[2]Mix contained per kilogram: 3,690,280 I.U vitamin A, 1,102,300 I.U vitamin $D_3$, 13,228 I.U vitamin E, 7.9 mg vitamin $B_{12}$, 2646 mg Riboflavin, 17,637 mg Niacin, 4,409 mg d-panthotenic Acid, 200,178 mg choline, 728 mg Menadione, 440 mg Folic Acid, 1,584 mg Pyridoxine, 792 mg Thiamine, 44 mg d-Biotin.

[3]Mix supplied per kilogram of diet: 140 gm Ca, 100 gm Zn, 120 gm Mn, 75 gm Fe, 10 gm Cu, 2.5 gm Iodine.

Table 2 Composition of experimental ration (%) (Experiment 2)

| | $ME_n$ in feed (kcal/kg)[1] | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|
| | 2828 | | | 3200 | | | 3574 | | |
| | c/p ratio | | | c/p ratio | | | c/p ratio | | |
| Ingredient | 140 | 160 | 180 | 140 | 160 | 180 | 140 | 160 | 180 |
| | (%) | | | | | | | | |
| Gr.Corn | 62.58 | 66.61 | 69.81 | 52.97 | 61.00 | 67.34 | 45.54 | 43.42 | 50.52 |
| Soy(48.5%) | 29.56 | 24.01 | 19.63 | 36.75 | 29.85 | 24.38 | 34.41 | 37.82 | 31.73 |
| Fat | ——— | ——— | ——— | 6.23 | 5.03 | 4.08 | 15.59 | 14.23 | 13.16 |
| Dical | 1.54 | 1.59 | 1.63 | 1.77 | 1.82 | 1.86 | 2.03 | 2.09 | 2.13 |
| $CaCO_3$ | 1.09 | 1.11 | 1.13 | 1.19 | 1.23 | 1.26 | 1.28 | 1.30 | 1.34 |
| Salt | 0.38 | 0.40 | 0.38 | 0.41 | 0.40 | 0.41 | 0.45 | 0.45 | 0.45 |
| Vit. Mix[2] | 0.36 | 0.36 | 0.36 | 0.36 | 0.36 | 0.36 | 0.36 | 0.36 | 0.36 |
| D-L-Meth. | 0.22 | 0.22 | 0.22 | 0.22 | 0.22 | 0.22 | 0.22 | 0.22 | 0.22 |
| Trace Min[3]. | 0.10 | 0.10 | 0.10 | 0.10 | 0.10 | 0.10 | 0.11 | 0.11 | 0.10 |
| Filler | 4.19 | 5.60 | 6.76 | ——— | ——— | ——— | ——— | ——— | ———. |

[1]Calculated using values from National Research Council (1984)

[2]Mix contained per kilogram: 3,690,280 I.U vitamin A, 1,102,300 I.U vitamin $D_3$, 13,228 I.U vitamin E, 7.9 mg vitamin $B_{12}$, 2646 mg Riboflavin, 17,637 mg Niacin, 4,409 mg d-panthotenic Acid, 200,178 mg choline, 728 mg Menadione, 440 mg Folic Acid, 1,584 mg Pyridoxine, 792 mg Thiamine, 44 mg d-Biotin.

[3]Mix supplied per kilogram of diet: 140 gm Ca, 100 gm Zn, 120 gm Mn, 75 gm Fe, 10 gm Cu, 2.5 gm Iodine.

Table 3 Caloric density and protein ratio effects on gain and feed and energy Consumption of broilers housed in theromoneutral (TN) and heat distress (HD) environments (Experiment 1).

| Calorie | Ratio | Gain (g) TN | Gain (g) HD | Feed (g) TN | Feed (g) HD | Energy (kcal $ME_n$/b) TN | Energy (kcal $ME_n$/b) HD |
|---|---|---|---|---|---|---|---|
| 2880 | 140 | 1480[a] | 917[b] | 2960[a] | 2170[b] | 8.5[b] | 6.1[cd] |
| 2880 | 155 | 1455[a] | 899[b] | 2981[a] | 2053[b] | 8.6[b] | 5.9[d] |
| 3200 | 140 | 1484[a] | 959[b] | 2838[a] | 2046[b] | 9.1[a] | 6.5[c] |
| 3200 | 155 | 1525[a] | 931[b] | 2920[a] | 1901[c] | 9.3[a] | 6.1[cd] |
| Means | | 1486[a] | 927[b] | 2925[a] | 2030[b] | 8.9[a] | 6.2[b] |

[a-d] means within a major heading with unlike superscripts differ (P<.05)

Table 4 Caloric density and protein ratio effects on broiler gain/feed, survivabilty (Surv) and adjusted gain/feed housed in thermoneutral (TN) heat distress (HD) (Experiment 1).

| | | Gain/Feed | | Surv (%) | | Adj[1]. Gain/Feed | |
|---|---|---|---|---|---|---|---|
| Calorie | Ratio | TN | HD | TN | HD | TN | HD |
| 2880 | 140 | .50[a] | .36[b] | 100[a] | 83.2[b] | .50[a] | .40[b] |
| 2880 | 155 | .49[a] | .40[b] | 100[a] | 91.7[ab] | .49[a] | .42[b] |
| 3200 | 140 | .51[a] | .41[b] | 97.5[a] | 87.5[ab] | .51[a] | .44[b] |
| 3200 | 155 | .52[a] | .39[b] | 100[a] | 80.2[b] | .52[a] | .44[b] |
| Means | | .51[a] | .39[b] | 99.4[a] | 85.7[b] | .51[a] | .42[b] |

[a-d] means within a major heading with unlike superscripts differ (P<.05)
[1]Gain/feed ratio calculated by adjusting for dead birds during experimental period.

Table 5 Effect of caloric density and protein ratio on dressing percentage, fat pad and carcass fat (Experiment 1).

| | | Dressing (%) | | Fat Pad (%) | | CF (%) | |
|---|---|---|---|---|---|---|---|
| Calorie | Ratio | TN | HD | TN | HD | TN | HD |
| 2880 | 140 | $68.4^{ab}$ | $68.0^{ab}$ | $1.70^{ab}$ | $1.80^{ab}$ | $12.2^{bc}$ | $12.7^{abc}$ |
| 2880 | 155 | $66.8^{b}$ | $69.5^{a}$ | $1.52^{b}$ | $1.60^{ab}$ | $12.1^{c}$ | $12.6^{abc}$ |
| 3200 | 140 | $67.6^{ab}$ | $68.6^{ab}$ | $2.07^{a}$ | $1.63^{ab}$ | $13.3^{ab}$ | $13.0^{abc}$ |
| 3200 | 155 | $67.7^{ab}$ | $69.7^{a}$ | $1.90^{ab}$ | $1.90^{ab}$ | $12.3^{bc}$ | $13.5^{a}$ |
| Means | | $67.6^{b}$ | $68.9^{a}$ | 1.79 | 1.72 | $12.5^{*}$ | $12.9^{*}$ |

$^{ab}$ means within a major heading with unlike superscripts differ ($P<.05$)
$^{*}$differ ($P<.07$)

Table 6 Caloric density and protein ratio effects on gain, feed and energy consumption (Experiment 2).

| | | Gain (g) | | Feed (g) | | Energy (kcal $ME_n$/b) | |
|---|---|---|---|---|---|---|---|
| Calorie | Ratio | TN | HD | TN | HD | TN | HD |
| 2826 | 140 | $1194^{cd}$ | $952^{gh}$ | $2652^{bc}$ | $2163^{ef}$ | $7.5^{cde}$ | $6.1^{g}$ |
| 2826 | 160 | $1173^{de}$ | $994^{fgh}$ | $2671^{b}$ | $2393^{cde}$ | $7.5^{cde}$ | $6.8^{efg}$ |
| 2826 | 180 | $1085^{ef}$ | $898^{h}$ | $2628^{bcd}$ | $2294^{ef}$ | $7.4^{cde}$ | $6.4^{fg}$ |
| 3200 | 140 | $1360^{a}$ | $995^{fgh}$ | $2703^{b}$ | $2081^{f}$ | $8.6^{b}$ | $6.6^{efg}$ |
| 3200 | 160 | $1250^{bcd}$ | $1036^{fg}$ | $2598^{bcd}$ | $2366^{de}$ | $8.3^{bc}$ | $7.5^{cde}$ |
| 3200 | 180 | $1273^{abcd}$ | $964^{gh}$ | $2592^{bcd}$ | $2259^{ef}$ | $8.3^{bc}$ | $7.2^{def}$ |
| 3574 | 140 | $1295^{abc}$ | $993^{fgh}$ | $3033^{a}$ | $2242^{ef}$ | $10.8^{a}$ | $8.0^{def}$ |
| 3574 | 160 | $1313^{ab}$ | $1015^{fg}$ | $2821^{ab}$ | $2306^{ef}$ | $10.0^{a}$ | $8.2^{bc}$ |
| 3574 | 180 | $1297^{abc}$ | $983^{fgh}$ | $3020^{a}$ | $2231^{ef}$ | $10.8^{a}$ | $7.9^{bcd}$ |
| Means | | $1249^{a}$ | $981^{b}$ | $2746^{a}$ | $2259^{b}$ | $8.8^{a}$ | $7.2^{b}$ |

a-h Means within a major heading with unlike superscripts differ ($P<.05$)

Table 7 Caloric density and protein ratio effects on gain/feed, survivabilty (Surv) and adjusted gain/feed and (Expriment 2).

| | | Gain/Feed | | Surv (%) | | Adj. Gain/Feed | |
|---|---|---|---|---|---|---|---|
| Calorie | Ratio | TN | HD | TN | HD | TN | HD |
| 2826 | 140 | $.43^{bc}$ | $.39^{cdefg}$ | $95.8^{ab}$ | $87.5^{abcd}$ | $.44^{b}$ | $.40^{bcd}$ |
| 2826 | 160 | $.43^{bc}$ | $.40^{cdef}$ | $97.9^{a}$ | $95.8^{ab}$ | $.44^{b}$ | $.40^{bcd}$ |
| 2826 | 180 | $.41^{cde}$ | $.37^{defg}$ | $100{\cdot}0^{a}$ | $93.8^{ab}$ | $.41^{bcd}$ | $.38^{d}$ |
| 3200 | 140 | $.47^{ab}$ | $.36^{efg}$ | $93.8^{ab}$ | $76.0^{d}$ | $.48^{a}$ | $.40^{bcd}$ |
| 3200 | 160 | $.48^{ab}$ | $.39^{cdef}$ | $97.9^{a}$ | $90.6^{abc}$ | $.48^{a}$ | $.41^{bcd}$ |
| 3200 | 180 | $.48^{a}$ | $.40^{cdef}$ | $97.9^{a}$ | $92.7^{ab}$ | $.48^{a}$ | $.41^{bcd}$ |
| 3574 | 140 | $.41^{cde}$ | $.34^{g}$ | $95.8^{ab}$ | $77.1^{d}$ | $.42^{bcd}$ | $.38^{d}$ |
| 3574 | 160 | $.41^{cd}$ | $.36^{defg}$ | $89.6^{abc}$ | $83.3^{bcd}$ | $.43^{bc}$ | $.39^{cd}$ |
| 3574 | 180 | $.43^{bc}$ | $.35^{fg}$ | $100.0^{a}$ | $80.2^{cd}$ | $.43^{bc}$ | $.38^{d}$ |
| Means | | $.44^{a}$ | $.37^{b}$ | $96.5^{a}$ | $86.3^{b}$ | $.44^{a}$ | $.40^{b}$ |

a-g Means within a major heading with unlike superscripts differ (P<.05)

Table 8 Effect of caloric density and protein ratio on dressing Percentage, fat pad and carcass fat (CF) (Expriment 2)

| | | Dressing | | Fat Pad | | CF (%) | |
|---|---|---|---|---|---|---|---|
| Calorie | Ratio | TN | HD | TN | HD | TN | HD |
| 2826 | 140 | $70.7^{cd}$ | $71.5^{abcd}$ | $1.03^{f}$ | $1.46^{def}$ | $10.9^{f}$ | $13.3^{cde}$ |
| 2826 | 160 | $71.4^{abcd}$ | $71.3^{abcd}$ | $1.62^{bcde}$ | $1.39^{ef}$ | $12.7^{e}$ | $12.6^{e}$ |
| 2826 | 180 | $69.8^{d}$ | $71.7^{abc}$ | $1.63^{bcde}$ | $1.57^{cde}$ | $12.8^{e}$ | $13.0^{de}$ |
| 3200 | 140 | $71.2^{abcd}$ | $71.3^{abcd}$ | $1.81^{abcde}$ | $2.07^{ab}$ | $12.5^{e}$ | $13.8^{bcde}$ |
| 3200 | 160 | $71.7^{abc}$ | $72.7^{a}$ | $1.78^{abcde}$ | $1.88^{abcde}$ | $13.2^{de}$ | $13.7^{bcde}$ |
| 3200 | 180 | $71.6^{abcd}$ | $71.6^{abcd}$ | $1.99^{abc}$ | $2.25^{a}$ | $13.5^{bcde}$ | $13.5^{bcde}$ |
| 3574 | 140 | $70.8^{cd}$ | $71.3^{abcd}$ | $2.11^{ab}$ | $2.11^{ab}$ | $13.8^{bcde}$ | $14.5^{abcd}$ |
| 3574 | 160 | $71.1^{abcd}$ | $70.8^{bcd}$ | $1.89^{abcd}$ | $2.23^{a}$ | $13.8^{bcde}$ | $15.3^{a}$ |
| 3574 | 180 | $72.7^{ab}$ | $72.4^{abc}$ | $2.25^{a}$ | $2.21^{a}$ | $14.9^{ab}$ | $14.7^{abc}$ |
| Means | | 71.2 | 71.6 | $1.8^{*}$ | $1.9^{*}$ | $13.1^{b}$ | $13.8^{a}$ |

$^{a-f}$ Means within a major heading with unlike superscripts differ ($P<.05$)
$^{*}$ differ ($P<.09$)

Table 9 Caloric density and calorie protein ratio effects on gain, gain/feed, dressing percent and Carcass fat (Experiment 2).

| Calorie | Ratio | Gain (g) | | Gain/Feed | | Energy (kcal $ME_n$/b) | |
|---|---|---|---|---|---|---|---|
| | | TN | HD | TN | HD | TN | HD |
| | 140 | $1283^{a}$ | $980^{bc}$ | $.44^{a}$ | $.36^{c}$ | $9.0^{a}$ | $6.9^{c}$ |
| | 160 | $1245^{ab}$ | $1015^{b}$ | $.44^{a}$ | $.39^{b}$ | $8.6^{a}$ | $7.5^{b}$ |
| | 180 | $1218^{b}$ | $948^{c}$ | $.44^{a}$ | $.37^{bc}$ | $8.8^{a}$ | $7.2^{bc}$ |
| 2826 | | $1151^{b}$ | $948^{d}$ | $.42^{b}$ | $.38^{c}$ | $7.5^{d}$ | $6.4^{e}$ |
| 3200 | | $1294^{a}$ | $998^{c}$ | $.48^{a}$ | $.38^{c}$ | $8.4^{b}$ | $7.1^{d}$ |
| 3574 | | $1302^{a}$ | $997^{cd}$ | $.42^{b}$ | $.35^{d}$ | $10.6^{a}$ | $8.0^{b}$ |

a-e Means within a calorie and a ratio and major heading with unlike superscripts differ ($P<.05$)

Table 10 Caloric density and calorie protein ratio effects on feed, consumption, survivabilty (Surv) and carcass fat (Experiment 2).

| Calorie | Ratio | Feed (g) | | Surv (%) | | CF (%) | |
|---|---|---|---|---|---|---|---|
| | | TN | HD | TN | HD | TN | HD |
| | 140 | $2796^{a}$ | $2161^{c}$ | $95.1^{ab}$ | $80.2^{c}$ | $12.4^{d}$ | $13.8^{a}$ |
| | 160 | $2696^{a}$ | $2355^{b}$ | $95.1^{ab}$ | $89.9^{b}$ | $13.2^{c}$ | $13.9^{a}$ |
| | 180 | $2747^{a}$ | $2261^{bc}$ | $99.3^{a}$ | $88.9^{b}$ | $13.8^{ac}$ | $13.7^{ac}$ |
| 2826 | | $2650^{b}$ | $2283^{c}$ | $97.9^{a}$ | $92.4^{a}$ | $12.2^{d}$ | $12.9^{c}$ |
| 3200 | | $2631^{b}$ | $2235^{c}$ | $96.5^{a}$ | $86.5^{b}$ | $13.1^{c}$ | $13.7^{bc}$ |
| 3574 | | $2958^{a}$ | $2260^{c}$ | $95.1^{a}$ | $80.2^{c}$ | $114.2^{b}$ | $14.8^{a}$ |

abc Means within a calorie and a ratio and major heading with unlike superscripts differ ($P<.05$)

Table 11 Analysis of variance for gain, G/F, feed consumption, energy consumption, Survivability, dressing, fat pad and carcass fat percentage (Experiment 2)

| S. O. V[1] | df | Gain | G/F[2] | Feed[3] | Encon[4] | Surv[5] | DP[6] | FP[7] | CF[8] |
|---|---|---|---|---|---|---|---|---|---|
| Main effects | | | | | | | | | |
| Temp (T)[9] | 1 | ** | * | ** | ** | ** | NS | * | ** |
| Calorie (C) | 2 | ** | * | * | ** | ** | NS | ** | ** |
| Linear | 1 | ** | * | ** | ** | ** | | | |
| quad | 1 | ** | ** | * | * | NS | | | |
| Ratio (R) | 2 | * | NS | NS | NS | ** | NS | * | * |
| linear | 1 | ** | ** | ** | ** | ** | | | |
| quad | 1 | ** | ** | ** | ** | NS | | | |
| Interactions | | | | | | | | | |
| T x C | 2 | * | * | ** | ** | NS | NS | NS | NS |
| T X R | 2 | NS | NS | ** | * | NS | NS | NS | * |
| T X C X R | 8 | NS | NS | NS | NS | NS | * | NS | NS |

* P<.05
** P<.01
NS= not significant
[1] Source of variance
[2] gain/feed ratioTemperature
[3] feed consumption
[4] energy consumption
[5] survivabilty
[6] dressing %
[7] fat pad %
[8] carcass fat %
[9] ambient temperature

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CHAPTER VII

# VIRGINIAMYCIN EFFECTS ON BROILER GROWTH RATE, FEED EFFICIENCY, CARCASS YIELD AND SURVIVABILITY WHEN REARED IN HEAT DISTRESSED AND THERMONEUTRAL ENVIRONMENTS.

T. Belay, C. Sundeep and R.G. Teeter
Department of animal Science, Oklahoma State University, Stiilwater, OK 74078

## Abstract

An experiment utilizing 1296 Cobb X Cobb male broilers housed in thermoneutral and heat distressed environments was conducted to evaluate virginiamycin (VM) effects on broiler weight gain (G), feed consumption (FC), gain/feed ratio (G/F), survivability (SURV), dressing percentage (DP), carcass fat (CF) estimated by specific gravity (SG) and carcass water uptake during chilling ($CH_2O$). Virginiamycin levels evaluated included 0, 15 and 20 ppm. Birds received VM and were reared under recommended brooding environments to 25 days posthatching at which time they were transferred to two environmental chambers providing thermoneutral (TN, 24$^{o}$C) and cycling temperature heat distress (HD, 24-35 $^{o}$C). Heat distress controls exhibited significantly reduced ($P<.05$) G (2.4%), G/F (8.8%), SURV (4.7%), DP and increased

(P<.05) carcass fat. Within the TN environment, 15 and 20ppm VM supplementation impacted, compared to TN controls, G (+1.3%, +2.2%), G/F (+2.0%, +6.1%), FC (+1.6%, -.65%) and SURV (+1.5%, +2.1%), but did not impact (P>.26) carcass parameters. Within the heat distressed environment, 15 and 20 ppm VM supplementation, compared to HD controls, impacted G (+3.1%, +1.7%), G/F (+7.5%, +10%), FC (+1.6%, -.64%) and SURV (+3.1%, +6.2%), but did not affect carcass parameters. Virginiamycin effects on bird mortality during heat distress were indeed marked at the 20 ppm level presumably due to reduced immune challenge and heat production. The environmental effect on FC, DP, CF and $CH_2O$ was significant (P<.05), but the parameters were unaffected by VM. This study indicates that economic advantages of using VM during heat distress are marked.

*(Key words:* Heat distress, Virginiamycin, growth, broilers)

## Introduction

Virginiamycin (VM) is an effective antibiotic against gram positive microorganisms (De Somers and van Dijck, 1955) and has been well documented to improve broiler growth rate and feed efficiency (Woodward et al., 1988; Harms et al., 1986; Miles et al.,1984b; March et al., 1978 ) as well as carcass yield (woodWard et al., 1988; Leeson, 1984 ). Improved performance of broilers fed diets containing VM is associated with increased feed consumption (Buresh et al., 1985a; Leeson 1984) and nutrient absorption efficiency

(Nelson et al., 1963; March, et al., 1981). Enhanced utilization of sulfur amino acids (Miles, et al., 1984a; Miles and Harms, 1983), phosphorus (Buresh et al., 1985b) and manganese (Henry et al.,1986) has been reported for birds consuming Virginiamycin. The improved nutrient absorption may be related to reduced intestinal mass and mucosal membrane thickness (King , 1974; Solca et al., 1980). Indeed, the increased carcass yield has been attributed to reduced intestinal tract weight relative to body weight (Izat et al., 1989; Salmon and Stevens, 1990; Henry et al., 1986). The beneficial antibiotic effects on growth rate have been attributed to gastrointestinal flora as positive responses are not noted under germ free conditions (Coates et al., 1963; Freeman et al., 1975).

High ambient temperature-relative humidity distress decreases broiler live weight gain, survivability, feed intake, and feed efficiency while increasing carcass fat content (Teeter et al., 1985b, Howlider and Rose, 1987, Teeter et al., 1987; Smith and Teeter, 1987; Howlider and Rose, 1989). Heat distressed birds are also reported to have lower blood agglutinin titers against heat-killed *Salmonella pullorum* antigen and suppressed cell-mediated immunity ( Siegel and Latimer, 1984; Regnier and kelly, 1981). Immunological responses mediated via leukocytic cytokines have been reported to increase bird heat production (Klasing and Barbara, 1990). Whether the depressed immunological response exhibited by heat

distressed broilers is due specifically to reduced immunological capability or on an adaptive response to reduce heat production is not known. However, the beneficial effects of antibiotic use during heat distress could potentially be two fold via the reduced immune challenge decreasing bird heat production and the incidence of subclinical disease. The objective of the study reported herein is to evaluate Virginiamycin efficacy in broilers housed in thermoneutral and cycling temperature heat distress environments and exposed to two immune challenge levels via poultry litter incorporation into the basal ration.

## Materials and Methods

A total of 1296 Day old Cobb x Cobb male chicks were divided into 6 pens at hatching and raised on rice hull litter to 25 days of age. Experimental rations consisted of corn soybean meal based starter rations to 25 days posthatching and grower rations thereafter supplemented with 0, 15 and 20 ppm Virginiamycin (Table 1). The grower ration was supplemented with 1% finely ground polyethylene glycol (PEG) or rice hull based poultry litter to provide 2 nonspecific immune challenge levels. The PEG corrected for the ration space occupied by the poultry litter and would not be expected to constitute an immune challenge. Therefore, the PEG and litter simulated clean and dirty environments.

On day 25 posthatching feed consumption was tallied, birds were group weighed and transferred to wire-floored (61 X 82 cm) grower batteries housed within two environmental chambers. The chambers were thermostatically and humidistatically controlled such that the thermoneutral (TN) designated chamber was maintained at 24 C and the heat distress (HD) chamber cycled between 24 and 35$^{o}$C. Relative humidity in both chambers was maintained at 55$\pm$5%. Therefore, treatment groups consisted of a factorial arrangement of 2 ambient temperatures X 3 VM fortification levels X 2 immune challenge levels. Replicates contained 24 and 12 replicates of 6 chicks each in the HD and TN environments, respectively. Following a 4 day chamber adaptation period (25-29 days) birds were fasted overnight, weighed, and the environmental phase initiated. Feed and water were available for ad libitum consumption throughout the experimental period and mortality and dead bird's weight was recorded daily.

Following the 21 day environmental period body weight gain, feed consumption, dressing percentage, carcass specific gravity (Teeter and Smith, 1985a) and percent carcass water uptake were determined. For dressing percentage, carcass specific gravity and water uptake analysis broilers, one from each of HD and 2 from each TN replicate, were randomly selected and processed as follows: Birds were weighed, hung on a rail, electrically stunned, bled by severing jugular and carotid veins, passed through a

scalding vat and plucking machine and hand eviscerated. Carcasses were weighed in air and water for computation of specific gravity (Barton et al, 1981). Carcass water uptake was determined by comparing weights prior to and following chilling in an ice-water bath for 2 hours.

Data collected were subjected to analysis of variance using the General Linear Model of the statistical analysis system (SAS, 1985) and differences were determined using Duncan's (1955) multiple range comparision.

## Results and discussion

Chick weights and feed consumption values through 25 days posthatching are displayed in table 2. Though birds were reared in a relatively clean brooding environment, they experienced low ambient temperature distress due to prevailing ambient temperature and Virginiamycin supplementation of 15 and 20 ppm improved G/F (10.53%, 12.28%) Survivability (4.2%, 1.7%). No differences were detected in body weight attributable to VM supplementation.

Main effects of VM, environment and litter were each significant ($P<.05$) at 49 days posthatching following the environmental phase of the study. The interaction between dietary litter and Virginiamycin was not significant ($P>.10$). Therefore, results of performance (Table 3) and carcass parameters (Table 4) are discussed separately for litter and VM combinations with environmental temperature.

Heat distress significantly (P<.05) reduced gain (16.6%), feed consumption (9.1%), gain/feed ratio (18.3%) and broiler survivability (9.2%) similar to previous published works (Teeter et al. 1987; Smith and Teeter 1987; Teeter et al. 1985; Bray 1983; Cowan and Michie 1978). Averaged over VM supplementation, the addition of 1% poultry litter to the basal diet failed to impact production parameters of birds housed in the TN environment (Table 6). However, birds fed the poultry litter supplemented ration and exposed to HD exhibited markedly reduced (P<.01) survivability. The negative synergism between litter consumption and heat distress is presumably due to the additive effect of the bird immunological response and high ambient temperature on the bird's total heat load. Klasing and Barbara (1990) reported increased heat production for birds reared in dirty vs clean environments.

In the TN environment the 15 and 20 ppm VM supplementation level, averaged over litter fortification numerically improved gain (1.27%, 2.02%), gain/feed ratio (2.04%, 6.12%) and SURV (1.5%, 2.1%). Though the effects were not statistically significant several responses just missed significance overall are similar to other published works demonstrating efficacy (Miles et al., 1984b; Leeson et al., 1984; Harms et al., 1986; Stutz and Lawton ,1984). Creating a more homogeneous data set by adding in the weight gain of birds dying during the experiment to adjust for mortality resulted in a higher (P<.05) gain/feed ratio at 20

ppm VM supplementation. Indeed, a dose dependent survivability benefit was detected with VM in the TN environment. The VM mortality response is complimentary to results of Miles et al., (1984b) and Proudfoot et al., (1990). Averaging over the 15 and 20 ppm VM level yielded improved gain, gain/feed ratio and survivability (Table 5).

Birds reared in the cycling temperature heat distress environment supplemented with 15 and 20 ppm VM exhibited, compared to HD controls, improved gain (3.1%, 1.7%), gain/feed ratio (7.5%, 10.0%) and SURV (3.1%, 6.2%), respectively. The gain/feed ratio and mortality improved ($P<.05$) in a dose dependent manner with VM supplementation levels. However, optimal gain was observed at 15 ppm, though it was not different to the 20 ppm supplementation level. The VM survival response averaged 4.7% in HD and 1.8% in TN demonstrating that VM effects during heat distress are particularly marked. The VM efficacy during heat distress is occurring at a time when the birds immunological responses are suppressed (Regnier and Kelley, 1981, Siegel and Latimer,1984). As such antibiotic supplementation might be expected to have improved efficacy. Whether the effect is due to a reduced immune challenge and heat production, lowered incidence of disease or a combination of thereof is unknown.

Considering the carcass parameter, heat distress increased ($P<.05$) dressing percentage, carcass water uptake during chilling and carcass fat. Increased broiler carcass

fat during heat distress has been implicated by several workers (Howlider and Rose, 1987; Janky et al., 1983; Farrell et al., 1981; Kubena et al., 1974). The effect of heat distress on carcass water uptake are likely due to the effect of heat distress on water balance and intracellular dehydration (Belay et al., 1991). Published reports of Virginiamycin effects on carcass parameters have been inconsistent as Leeson (1984) reported increased carcass weight and yield, while Izat et al. (1989) reported no effect of VM on carcass weight or dressing percentage. In this study, Carcass parameters were not affected (P>.10) by VM level within either environmental temperatures (Table 4). Lack of a VM effect on carcass water uptake is similar to the data reported by Izat et al. (1989) and Woodward et al. (1988).

Broiler growth and carcass composition responses to VM have at times been mixed. Veltmann and Weideman (1987) failed to observe growth rate improvements while Miles et al. (1984a) reported improved growth rate and feed efficiency with Hyline strains pullets fed VM. Positive responses with Cobb broiler chicks have been consistently reported (Mile et al., 1984a; Harms et al., 1986; Henry et al., 1986; Miles et al., 1984b). Likewise, the effects of VM supplementation on carcass characteristics of commercial broilers of similar genetic makeup reported by Leeson (1984) were contrary results reported by Izat et al. (1989). Comparative differences are difficult to explain, however,

as demonstrated both environment and management have the potential to profoundly impact VM efficacy.

Table 1. Composition of the starter and finisher diet used for the experiment

| Ingredients | Percent | |
|---|---|---|
| | Starter | Finisher |
| Ground corn | 55.20 | 54.29 |
| Soybean meal,48.5% | 37.10 | 34.37 |
| Fat | 3.44 | 6.29 |
| Dicalcium phosphate (22% ca; 18.5% P) | 1.98 | 1.68 |
| Limestone (38% Ca) | 1.22 | 1.28 |
| salt | 0.40 | .40 |
| Vitamin mix[1] | 0.36 | .36 |
| DL-Methionine, 99% | 0.21 | .22 |
| Trace mineral mix[2] | 0.10 | .10 |
| polyethylene glycol or Poultry Litter | _____ | 1.00 |
| Total | 100.00 | 100.00 |
| Calculated Analysis: | | |
| ME Kcal/Kg | 3050 | 3190 |
| Crude protein (%) | 23.00 | 21.61 |
| Calcium (%) | 1.10 | 1.00 |
| Phosphorus (% av) | .50 | .44 |

[1]Mix supplied per kilogram of diet: 14,109 I.U vitamin A, 5291 I.U vitamin $D_3$, 47.62 I.U vitamin E, .014 mg vitamin $B_{12}$, 8.82 mg Riboflavin, 26.5 mg Niacin, 28.2 mg d-panthotenic Acid, 705.5 mg choline, 1.16 mg Menadione, 1.176 mg Folic Acid, 3.52 mg Pyridoxine, 3.52 mg Thiamine, .176 mg d-Biotin.

[2]Mix supplied per kilogram of diet: 160 mg Ca, 100 mg Zn, 120 mg Mn, 75 mg Fe, 10 mg Cu, 2.5 mg Iodine.

Table 2 Body weight, G/F and mortality of broilers fed two levels of Virginiamycin (0-25 days of age)

| VM[1] | Body Wt(g) | G/F | Survivability (%) |
|---|---|---|---|
| 0 | 763.2 | .57[b] | 89.8[b] |
| 15 | 762.6 | .63[a]b | 94.0[a] |
| 20 | 756.5 | .64[a] | 91.5[ab] |

[a,b] Means within a column under major heading with different superscript differe (P<.05).
[1] Virginiamycin parts per million

Table 3 Gain, feed consumption, G/F, and mortality of broilers fed two levels of Virginiamycin in TN and HD environments.

| | Gain (g) | | Feed consumed(g) | | Gain/Feed (kg per kg) | | Survivability (%) | |
|---|---|---|---|---|---|---|---|---|
| VM1 (ppm) | TN[2] | HD[3] | TN | HD | TN | HD | TN | HD |
| 0 | 1336[a] | 1141[c] | 2642[a] | 2439[b] | .49[a] | .40[c] | 97.4[a] | 86.3[c] |
| 15 | 1353[a] | 1176[b] | 2684[a] | 2429[b] | .50[a] | .43[b] | 98.9[a] | 89.4[bc] |
| 20 | 1363[a] | 1160[bc] | 2625[a] | 2418[b] | .52[a] | .44[b] | 99.5[a] | 92.5[b] |

[a-c] Means with different superscript within a row and a column under a major heading differ (P<.05).
1Represents virginiamycin
2TN= thermoneutral
3HD= heat distress

Table 4. Carcass parameters of broilers fed two levels of virginiamycin in TN and HD environments

| VM[1] (ppm) | Dressing Percentage | | Carcass Fat | | Water Uptake (%) | |
|---|---|---|---|---|---|---|
| | TN[2] | HD[3] | TN | HD | TN | HD |
| 0 | 71.49[b] | 72.69[a] | 12.81[a] | 13.43[a] | 5.20[b] | 6.10[a] |
| 15 | 71.07[b] | 72.24[a] | 12.81[a] | 13.43[a] | 4.77[b] | 5.66[b] |
| 20 | 71.37[b] | 72.58[a] | 13.43[a] | 13.43[a] | 4.87[b] | 5.02[b] |
| Mean | 71.31[b] | 72.50[a] | 13.12[b] | 13.43[a] | 4.95[b] | 5.59[a] |

[ab]Means within a major heading and within arow and a column with different superscript differ (P<.05)
[1]Represents virginiamycin
[2]TN= thermoneutral
[3]HD= heat distress

Table 5. Virginiamycin effects on broiler gain, G/F and mortality (25-49 days) averaged over environment

| VM | Gain(g) | G/F | Survivability (%) | Feed Con(g) | ADj.G/F[1] |
|---|---|---|---|---|---|
| 0 | 1238[b] | .45[b] | 91.8[b] | 2540 | .47[b] |
| 15 | 1264[a] | .47[ab] | 94.2[ab] | 2556 | .48[ab] |
| 20 | 1261[ab] | .48[a] | 96.0[a] | 2523 | .49[a] |

[ab] Means with different superscript within a major heading and a column differ (P<.05)

[1] Adjusted by adding weight gain of birds dying during the trial to total gain

Table 6 Litter effect on gain, feed consumption and mortality of broilers averaged over TN and HD environments.

| | Gain (g) | | Feed consumed(g) | | Survivability (%) | |
|---|---|---|---|---|---|---|
| lit(%) | TN[2] | HD[3] | TN | HD | TN | HD |
| 0 | 1353[a] | 1163[b] | 2632[a] | 2436[b] | 99.5[c] | 92.4[b] |
| 1 | 1348[a] | 1159[b] | 2659[a] | 2443[b] | 97.2[c] | 87.7[a] |

[a b,c] Means with different superscript within a row and a column under major heading differe (P<.05)
[2]TN= thermoneutral
[3]HD= heat distressed

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# CHAPTER VIII

## SUMMARY AND CONCLUSIONS

The deleterious effects of high ambient temperature on broiler growth rate and feed efficiency has long been recognized. The heat distress effect on bird physiology is complex and influences an array of function.

The study reported herein was conducted to: (1) develop a suitable surgical method for long term separate collection of urine and feces applicable in young broilers to study the impact of high ambient temperature distress on water and mineral balances, (2) determine the influence of acute heat distress upon Na, K, P, S, Se, Mg, Mn, Fe, Cu, Mo, and Zn balance and urinary excretion, (3) to determine the effect of heat distress on water balance, urine production, plasma electrolyte, Free water clearance, osmolal clearance, urinary and plasma osmolality and to understand the mechanism of drinking water effect on broiler thermobalance, (4) to evaluate the effect of rations varying in calorie/ protein ratio on growth and carcass composition of broilers housed in thermoneutral and heat distress, (5) to evaluate the efficacy of therapeutic level of virginiamycin on growth rate, survivability and carcass

composition of broilers housed in thermoneutral and heat distressed environments.

In the first experiment, a surgical procedure enabling separate collection of urine and feces in young broilers is described. As we observed the colostomy technique to be a successful procedure for determining mineral and water balance. The procedure described was used to prepare the model birds for the experiments dealing with mineral and water balance in this dissertation projects. The colostomized broilers were able to maintain their body weight and, in addition, were healthy comparable to intact broilers for up to 3 months.

The two experiments in the second study were conducted to evaluate the effect of heat distress on mineral balance and urinary mineral excretion. The study has revealed that heat distress broilers have negative mineral balance. In the first experiment, the effect of heat distress was minimal on urine production however, urinary excretion for $K^{+}$, $Mg^{+2}$, $P^{-}$, and $S^{-2}$ were elevated by heat distress while $Cu^{+}$, $Se^{-2}$ and $Mg^{+2}$ were lost primarily in feces. In the second experiment, heat distress increased urine output and urinary $K^{+}$, $P^{-3}$, $S^{-2}$, $Na^{+}$, $Mg^{+2}$, $Ca^{+2}$, $Mn^{+2}$ excretion, while urinary $Cl^{-}$ excretion and concentration was reduced. These studies provide evidence that heat distress adversely impacts bird mineral balance and further that the excretion route varies with the specific mineral and possibly heat distress severity.

In the third study three experiments were conducted to evaluate heat distress effects on urine output, free water, and osmolal clearance and urinary and plasma osmolality. In this experiment, relationships between water consumption, broiler thermobalance and effect of heat distress on broiler water balance and plasma electrolyte concentration were studied. Heat distress did not affect fecal water excretion, but increased water consumption, urine production, free water clearance and osmolal clearance. These responses were highly correlated with water consumption levels. Inverse relationships between urine osmolality and urine flow rates were also demonstrated. In this study plasma osmolality was not affected by heat distress nor water consumption levels, but plasma $K^+$ and $Na^+$ were reduced while $Cl^-$ was increased by heat distress. Heat distress reduced broiler metabolic water and heat production and nonevaporative cooling. Evaporative cooling was increased due to heat distress, but failed to compensate for the nonevaportative decline. Consequently the bird's heat content increased with heat distress. Respiration frequency increased with water consumption in the thermoneutral while higher water consumption decreased respiration rate of heat distressed broilers. Increased water availability, in this study, was associated with increased heat production in the thermoneutral environment, but not in the heat distressed birds. The findings of this study indicate that, during heat distress, the elevated water consumption is positively

correlated with higher evaporative cooling (r=.73) and respiration efficiency (r=.73). This study is the only one of its kind to demonstrate the mode of action of drinking water effect on heat distressed broiler's thermobalance. However, whether the increased urine production in heat distressed broilers is a means of increasing nonevaporative body heat dissipation and/or hyperventilation induced alkalosis is not known.

In the second experiment, where intact birds were used and water consumption equalized, heat distress increased fecal water excretion. Since high ambient temperature distress did affect fecal water content of colostomized broilers in experiment one, the increased fecal water in the heat distressed intact broilers is attributable to urinary origin. The results of the third experiment demonstrates that drinking water potassium chloride supplementation for heat distressed broilers increased water consumption and similar to the first experiment increased evaporative cooling and respiration efficiency without affecting heat production. This also suggests that cooling efficiency and thereby survivability of heat distressed broilers can be manipulated by increasing water consumption via electrolyte therapy.

In the fourth study two experiments were conducted to evaluate the effects of caloric density and calorie protein ratio on broiler growth rate, gain/feed ratio, survivability and carcass parameters. In the first experiment, averaged

over the experimental rations and the two environments, body weight gain, feed and energy consumption, gain/feed ratio and survivability were reduced by heat distress . In the heat distress environment, feed consumption of broilers was reduced by the high caloric density and calorie-protein ratio rations. Survivability in the heat distressed environment was reduced by increased caloric consumption. Within the thermoneutral environment caloric density and calorie protein ratio did not affect gain, feed consumption, gain/feed, survivability nor adjusted gain/feed . However, energy consumption was increased with increasing caloric density of the ration similar to the heat distress. Carcass parameters revealed that heat distress increased dressing and carcass fat percentages. Higher ration caloric density also increased percentage fat pad within the thermoneutral environment.

Results of the second experiment, for the fourth study similar to the first experiment, indicated that heat distress reduced gain, feed and energy consumption, gain/feed ratio, survivability and gain/feed ratio adjusted for mortality. In both the heat distressed and thermoneutral environments a linear increase in weight gain was observed with increasing caloric density. Caloric consumption was increased without a decline in feed consumption in both environments. The increased energy consumption and reduced calorie/protein ratio was associated with reduced survivability in the heat distressed

environment. In the thermoneutral environment, increasing ration calorie/protein ratio at a constant caloric density tended to decrease weight gain, but did not affect other parameters.

Carcass analysis results for the second experiment showed that heat distress increased carcass fat. In heat distressed birds percent fat pad and carcass fat increased with increasing caloric density. In the thermoneutral housed broilers a linear increase in dressing, fat pad and carcass fat percentages were observed with increasing caloric density. Similarly, increasing calorie/protein ratio increased both percent fat pad and carcass fat, suggesting optimum calorie/protein ratio need to be maintained for lean tissue synthesis.

Studies on the effect of dietary manipulations indicated that weight gain of both heat distress and thermoneutral housed broilers can be increased by increasing ration caloric density. However, other than the adverse effect on carcass fat, higher caloric density rations studied in these experiments increased mortality of heat distressed broilers. Growth potential of broilers can be increased by increasing calorie-nutrient density, but during heat distress heat prostration occurs due to the combination of dietary heat increment and reduced nonevaporative heat dissipation efficiency.

In the last study the efficacy of virginiamycin supplementation was evaluated on body weight gain and

gain/feed ratio during the starter phase. In addition, the respective virginiamycin supplemented broilers were housed in a thermoneutral and heat distressed environments during the grower phase, and were fed rations with and without 1 % poultry litter as a stressor to study the effect of virginiamycin on growth and carcass parameters. During the starter phase differences were not detected in body weight attributable to virginiamycin supplementation. However, overall virginiamycin supplementation improved gain/feed ratio and survivability compared to unsupplemented control. The result of the finishing period of this experiment like the others reported indicated that heat distress reduced gain, feed consumption, gain/feed ratio and survivability. Interaction between dietary litter and virginiamycin was significant. However, only in birds fed the poultry litter supplemented ration and exposed to heat distress exhibited markedly reduced survivability, suggesting an additive effect of bird immunological response and high ambient temperature on the bird's total heat load. Virginiamycin supplementation did not affect growth parameters in the thermoneutral environment, but in heat distressed gain, gain/feed ratio and mortality were improved by the supplementation compared to control. The differential effect of the antibiotic supplementation only in heat distress would possibly be due to the protection from disease and suppression of the immune response thereby

reducing the heat load. However, this may be a mere speculative.

In the last study, carcass parameters were not affected by virginiamycin level within either environmental temperatures, though carcass water uptake was higher for heat distressed broilers. The data reported on the effect of heat distress on water balance is indeed agrees with this last experiment.

The overview of the studies in this dissertation verify the existence of interaction between ambient temperature and mineral balance. Evaluation of ambient temperature effects on true mineral absorption and tissue mineralization need to be a focus of future works to fully understand the therapeutic means. Mineral elements like zinc and selenium may be important in the immunological response of birds to heat distress. Before establishing the effect of high ambient temperature on the immune response the adequate intake and positive balance for mineral elements need consideration. Chloride, being an anion, its conservation by heat distressed birds could possibly be used to replace the bicarbonate loss and maintain cellular electrical neutrality or exteracellular pH. Thus the observed phenomenon could be a kidney mechanism to compensate for the respiratory alkaosis. However, the role of the kidney in acid base balance of heat distressed birds requires further investigations.

In conclusion, the potential exists to significantly impact bird thermobalance and hence productivity during high ambient temperature-relative humidity distress. Methods to manipulate bird thermobalance are of physiological and dietary origin.

VITA

TSEGAW BELAY

Candidate for the degree of

Doctor of Philosophy

Thesis: NUTRITIONAL AND WATER BALANCE CONSIDERATIONS FOR DEVELOPMENT OF BROILER HEAT DISTRESS THERAPEUTIC TECHNOLOGIES

Major Field: Animal Nutrition

Biographical:

Personal Data: Born in Dabat Aweraga, the province of Gondar, Ethiopia, in April 27, 1953, the son of Belay and Alemaz. Married to Muluemebet on December 29, 1977.

Education: Graduated from Haile Sellasie I High School Nekempti, Ethiopia, in June 1972; received Bachelor of Science in Agriculture Degree from Hale Sellasie I University, Ethiopia, in June, 1977; received Master of Science Degree from Addis Ababa University, Ethiopia, in June, 1981; received Diploma in International Poultry Husbandry and Animal feed preparation from Barneveld College The Netherlands, in May, 1982; completed requirements for the Doctor of Philosophy Degree at Oklahoma State University in July, 1991.

Professional Experience: Graduate Assistant, Addis Ababa University, Ethiopia, July, 1977 to July, 1978; Assistant Lecturer, Addis Ababa Universuty, Ethiopia, July, 1978 to December, 1980; Lecturer, Alemaya College of Agriculture, Ethiopia, December, 1980 to January, 1986; Lecturer and Dean of Students Alemaya University of Agriculture, Ethiopia, February, 1986 to June, 1987.

Professional Organizations: Poultry Science Association; The Netherlands Poultry International.