HIGH AMBIENT TEMPERATURE EFFECTS ON BROILER THERMOBALANCE

By

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Bachelor of Science in Biology

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Scranton, Pennsylvania

1986

Submitted to the Faculty of the
Graduate College of the
Oklahoma State University
in partial fulfillment of
the requirements for
the Degree of
MASTER OF SCIENCE
July, 1991

Thesis
1991
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ACKNOWLEDGMENTS

I wish to extend my sincere gratitude to my major advisor, Dr. Robert Teeter, for his advice, guidance, expertise and friendship throughout my graduate study. Appreciation is also extended to Dr. David Weeks and Dr. Kenneth Bartels for their suggestions as members of my graduate committee.

A special thanks goes to members of the OSU Poultry Research Facility, Chris Harjo, Jim Cason, Afaf Melouk, Kevin McDonald, Gene Murray, and Leon Bloom, for the valuable knowledge and assistance that they provided.

To my colleague, Tsegaw Belay, I would like to extend a sincere thanks for his friendship, knowledge, assistance and encouragement through out my study. I could not have done it without him.

A very special note of thanks is extended to my very special friends, Lisanne Hewel (MLTT) and Luis Diaz, for their constant support and encouragement throughout my studies.

I dedicate this manuscript to my parents, Mr. and Mrs. Chester Wiernusz. This work could not have been completed without their love and encouragement.

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CHAPTER I

INTRODUCTION

High ambient temperature-relative humidity distress has been shown to perturb bird thermobalance and thereby reduce broiler body weight gain, feed efficiency, and survival. Regardless of environment, the birds total heat load may be attributed to a summation of heat gained or lost from the environment and metabolic heat production. Birds produce and must therefore dissipate considerable amounts of heat daily as their energetic efficiency is typically less than 15%. The problem is particularly severe as the ambient temperature rises since heat loss associated with nonevaporative cooling, the most efficient heat dissipation route, declines markedly.

During increased high ambient temperature exposure, the bird's heat load is increased due to the environmental heat gain and the energy cost associated with activation of metabolic processes required for heat dissipation (Meltzer, 1987). Heat dissipation is enhanced by postural adjustments to increase surface area (Baldwin, 1974), vasodilation of unfeathered extremities (Nolan et. al, 1978), by increasing water intake (Farrell and Swain, 1977), and elevating respiration rate from the basal 25 breathes per minute to

as much as 250 (Frankel et al, 1962). Respiration rate is of particular importance as water evaporation, in leu of nonevaporative loss, becomes a significant route for heat dissipation.

Total heat production is the sum of basal heat production (Bartels et al, 1973), heat production to maintain a constant body temperature when ambient temperature is low (van Kampen et al, 1979) or when ambient temperature is high (Meltzer, 1987), heat production due to intake and digestion of feed (Macleod and Shannon, 1978), heat production due to synthesis and production, and finally heat production due to muscular activity (Boshouwers and Nicaise, 1985). Birds reduce their heat production via consuming less feed which in turn reduces substrate availability (Teeter and Smith, 1987).

Though the specific routes of heat loss, as discussed, are well defined qualitatively little research has been conducted to quantitatively estimate the importance of each. Indeed, a comprehensive model has not been developed enabling their study much less their potential manipulation into therapeutic measures. A model is needed enabling the quantification of bird overall thermobalance during various environmental exposures. Once a model is available, methods manipulating the various processes may be conducted.

The objective of the studies described herein was first to develop a model enabling the quantification of bird thermobalance when housed in thermoneutral and heat distress

environments. Additionally, other objectives included the determination of acclimation to heat distress, nicarbazin toxicity during heat distress and feed intake effects on broiler thermobalance when housed in thermoneutral and heat distress environments. Data collected will be used to evaluate the birds inability to control its body temperature when exposed to high ambient temperature-relative humidity environments and determine if it may be attributed to the heat dissipation inefficiency, higher metabolic rate or a combination thereof. It is hoped that a thorough understanding of these processes will enable therapeutic developments that will benefit the poultry industry around the world.

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CHAPTER II

REVIEW OF LITERATURE

Introduction

All animal species, domesticated and wild, live within environments containing multiple factors capable of eliciting distress. Distress factors include disease agents such bacteria, viruses and protozoa, social stress created by other animals and man, malnutrition, various types of toxicities and the thermal environment among others. It is impossible to designate any one factor as the most critical, since the environment is indeed composed of all and the animals existence is dependent upon it successfully compensating for the continually changing pattern of stressors. The study of environmental distress must, by necessity, first occur on an individual stressor basis, so that their effects may be independently known and then examined in combination. It is this latter area that offers the greatest potential to alleviate problems associated with modern poultry and livestock production. This study will be devoted to expanding our knowledge base related to the thermal environment interaction with nutritional plane.

The thermal environment is determined by the combination of ambient temperature, relative humidity, wind,

precipitation, photoperiod, solar radiation intensity, and cloud cover. The ideal combination of thermal environment determinants, the combination that minimizes bird heat production at maintenance, is defined as the thermoneutral environment. Since birds are principally raised in confinement, critical factors are reduced to ambient temperature, relative humidity and wind velocity. Divergence of these factors from the ideal pattern results in animal distress accompanied by decreased growth rate, feed consumption and feed efficiency as well as increased mortality. A greater understanding of basic nutritional/physiological-thermal environment interactions must be understood such that managerial practices may be developed to reduce animal distress and thereby increase productivity.

BASAL METABOLISM: The basal metabolic rate or standard metabolic rate is defined as the heat production occurring by an animal at rest, awake, fasted and housed within its thermoneutral zone. Under these conditions the rate of energy metabolism is a function of surface area since heat loss is closely tied to this factor. Surface area per unit body weight declines with increasing body weight and basal metabolism per unit weight declines with increasing body weight. However, surface area is a difficult parameter to estimate and numerous attempts have been made to relate it to body weight (Brody, 1964). Typically body weight will be

raised to a power, most commonly .75, which is now regarded as the universal metabolic weight. Brody (1964) suggested weight$^{\cdot 73}$ be used as a reference base for basal-energy metabolism in mature animals of different species including a weight range from mice to elephants.

Brody reported that for mature birds of different species the exponent varies from .62 to .70. Metabolic weight for poultry is commonly reported as $W^{\cdot 66}$, since this value gives a better estimate when comparing poultry within species. If the correct power is chosen and body temperature and animal composition are constant then heat production per unit metabolic weight is relatively constant. Under basal conditions heat energy is produced from various energy sources to offset heat loss and maintain constant body temperature.

The basal state is seldom achieved with assurance in animals because of the varying time period required to achieve the postabsorptive state and the physical, mental and emotional distress created by the experimental conditions. Misson (1974) found that laying hens required a 3 day exposure to the experimental situation before basal values could be achieved and that the time required to reach the post-absorptive state was influenced by body weight requiring 24 hours for birds below 2.5 Kg and 48 hours for those above.

FEED ENERGY-METABOLISM RELATIONSHIPS: The total energy contained in feed is termed gross energy (GE). However only a portion of gross energy actually appears in animal products such as meat, eggs or milk. Considerable energy losses occur in the feces and heat through oxidative processes (Figure 1).

Feed energy absorbed in the gastrointestinal tract is termed digestible energy. The apparent digestible energy of the ration or individual nutrients such as carbohydrates, proteins and fats and may be estimated by subtracting fecal energy from gross energy. This value is termed apparent because the endogenous losses have not been accounted for. Endogenous losses may be estimated by subtracting gas and urinary energy loss from apparent digestible energy yielding apparent metabolizable energy.

Metabolizable energy (ME) is the energy provided for metabolism including tissue assimilation and oxidation. However, during ME utilization wasted heat is given off. Shortly after these molecules are absorbed into the gastrointestinal tract there is an increase in heat production referred to as the heat increment of the feed. Subtracting the wasted heat from ME provides an estimate of net energy, or the energy actually contained in tissues or utilized for work. The net energy is available to maintain body temperature, to provide energy for work and other activities, to be stored as adipose tissue and for growth and production.

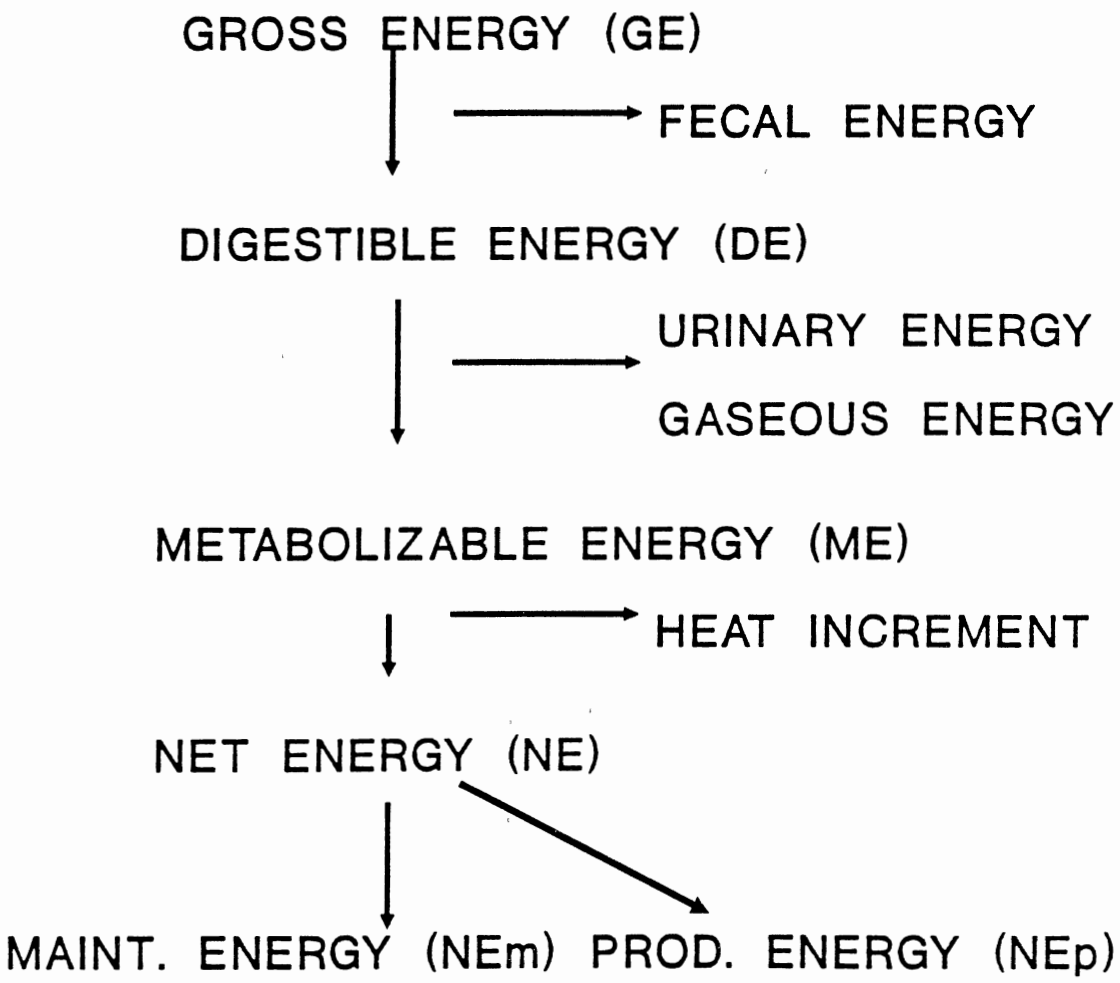

Figure 1. Energy utilization scheme

BODY TEMPERATURE: Birds and mammals are homeotherms and consequently maintain a relatively constant core body temperature. However, diurnal body temperature cycles have been detected and are influenced by age, sex, season, time of day, work, digestion, drinking of water and environmental temperature (Dukes, 1977). Overall animals have a temperature gradient declining from the body core to the peripheral tissues. The animals total heat load is dependent upon metabolism, the reactions by which chemical energy is transformed into heat, and the environmental temperature which the animal is subjected too. In order to maintain a constant body temperature the animal must balance its rate of heat dissipation against its rate of heat gain. Heat is added to the body by metabolism; in hot environments the body may gain heat by radiation, convection and/or conduction. However, with poultry the principle problem relates to reduced nonevaporative heat dissipation at high ambient temperatures.

The body temperature is determined by the balance between the amount of metabolic heat produced and the quantity of heat lost from the body to the environment. For the body temperature to remain constant the heat production must equal the heat dissipated. Sturkie (1986) proposed the following equation to describe the process:

$$HP = NHL + EHL$$

where

HP = metabolic heat production

NHL = nonevaporative heat loss

EHL = evaporative heat loss

Total heat production is the sum of basal heat production (Bartels et al, 1973), heat production to maintain a constant body temperature when ambient temperature is low (van Kampen et al, 1979) or heat loss is high (Meltzer, 1987), heat production due to intake and digestion of food (Macleod and Shannon, 1978), heat production due to synthesis and production, and finally heat production due to muscular activity (Boshouwers and Niciase, 1985). Birds reduce their heat production via consuming less feed which in turn reduces substrate availability (Teeter and Smith, 1987).

THERMOBALANCE: QUANTIFICATION

As discussed heat is produced by animals for a variety of purposes and its quantification is critical to many disciplines. Heat production may be estimated directly by measuring the heat lost from the body through radiation, convection, conduction and evaporation (direct calorimetry), by analysis of comparable carcasses at the start and end of a trial (comparative slaughter technique) and from the exchange of respiratory gases (indirect calorimetry). Both direct and indirect methods were originated by Lavoisier in 1777. Each method supplies essentially the same information however the difficulty and labor involved for each are quite

different. Each technique will be discussed and comparisons made as to the validity of each method.

BIOENERGETICS: Bioenergetics deals with the energy transformations in living things and is made possible by the Law of Conservation of Energy (energy can not be created or destroyed) and Hess's Law of Constant Heat Energy Summation (the heat released by a chain of reactions is independent of the chemical pathways and dependent only on the end products). These laws ensure that the heat produced from varies metabolic processes is the same as it would be if feed were allowed to combust to the same end-products.

The first law asserts that the total amount of energy in an isolated system remains constant. When the energy content of a system changes, the sum of all forms of energy given off by the system must be equal to the magnitude of the change. The first law is only concerned with initial and final energetic states of the system. The principle of conservation of matter also is taken into account in the first law because matter and energy are inseparable according to the theory of relativity. Matter and energy are different expressions of the same thing. In animal systems the energy equivalent of work, plus the maintenance energy of the animal, plus the heat increment of feed equals the energy generated from the oxidation of nutrients of the feed.

Hess's law of Constant Heat Summation states that all forms of energy are quantatively convertible to heat. This law states that in going from a particular set of reactions to a particular set of products, the change in enthalpy is the same whether the reaction takes place in one step or in a series of steps. Oxidation of substrates within an animal body is quite different from oxidation in a bomb calorimeter, however from a thermodynamic point of view these facts are incidental. According to Hess's law the physical and physiological heat values of all nutrients, with the exception of protein, are the same.

The end products of protein oxidation within the body and the bomb calorimeter are different because waste products of protein metabolism within the body are capable of further oxidation to produce carbon dioxide and water. In poultry this endproduct is mainly uric acid which accounts for 80% of metabolized nitrogen and secondly ammonia which accounts for 10% total nitrogen (Sturkie, 1986) and in mammals the major nitrogenous end product is urea.

DIRECT CALORIMETRY: Direct calorimetry is, in theory a simple technique but very difficult in practice. Measurements in heat loss must include not only sensible heat loss from radiation, convection and conduction, but also insensible heat loss or the latent heat of vaporization of water primarily from respiration and skin. Sensible heat

loss from an animal may be measured by two general types of calorimeters, adiabatic and gradient. Insensible heat loss may be estimated by determining the amount of water vapor added to the air by the animal.

In isothermal calorimeters the animal is confined in a well insulated chamber whose heat loss is zero. The first modern isothermal calorimeter was built by Rubner in 1894 for a dog. A major breakthrough leading to accurate and rapid isothermal measurements came from Benzinger and Kitzinger (1949) when they introduced a gradient layer calorimeter. Heat flows across a series of thermocouples uniformly interwoven throughout the calorimeter which records the total heat lost from the animal. There are usually two walls, the outer wall is electrically heated to the same temperature as the inner wall. Water circulating in a coil absorbs the heat from the inner wall and the volume and temperature of the water can be used to estimate the animal's sensible heat loss.

Heat-sink calorimeters are those in which heat lost through the surfaces is eliminated. Heat generated by the animal is removed from the chamber by a liquid cooled heat exchanger whose cooling power is regulated to equate the inlet and outlet air temperatures. The rate of heat removed by the heat exchanger is estimated from the flow rate and temperature increase of the coolant.

Other types of direct calorimeters include convection (Snellen et al., 1983) and differential calorimeters

(Deighton, 1939). The convection calorimeter relies on the measurement of the mean temperature rise of air leaving the animal compartment. Heat is prevented from dissipating from the walls by insulation or a water jacket. The method relies on very accurate comparisons between the temperatures of the inlet and outlet air. In the differential calorimeter there are two identical chambers, one containing the animal and the other equipped with an electrical heater whose output is adjusted to produce identical temperature increases in both chambers.

COMPARATIVE SLAUGHTER: Heat production can also be measured by the comparative slaughter technique. This technique can be quite accurate, but it is laborious, time consuming and forces one to assume initial animal composition. It estimates the actual amount of energy retained by the animal or conversely the measurement of all forms of energy loss. The variability that exists at 3 or 4 weeks of age, when the majority of comparative slaughter trials start, is great when you consider the differences in body composition.

In growing and fattening animals the measurement of energy retention has been proven useful in broilers, cattle and swine. In work done with broilers, the energy value of whole carcasses can be determined by bomb calorimeter after they have been dried and ground. Similar chicks are then fed for a specified period of time and the energy values of their carcasses is determined. Heat production is

calculated as the difference between the energy intake and the energy of body weight gain plus that of the excreta.

The comparative slaughter technique has several potential sources of error because it is difficult to obtain a truly representative sample of birds. Davidson and Mathieson (1965) suggested that the composition of birds killed at the beginning of a trial is probably representative of the remainder when very young birds are used. However, Fraps and Carlyle (1939) and Hanlan (1939) reported, variability exists within older birds especially due to their varying fat content. Errors may also be produced in the measurement of ME using either a total collection or marker and relating it to the entire experiment since Sibbald et al. (1960) and Burlacu et al. (1970b) indicated that the ME especially in young chicks may change with age and level of intake (Deighton and Hutchinson (1940).

INDIRECT CALORIMETRY: Indirect calorimetry is likewise a quantitative estimate of heat production using the gaseous reactants and end products of metabolism. Indirect calorimetry measures the heat generated by varies metabolic reactions by estimating respiratory gas exchange from materials consumed and produced during metabolism. Animal heat production is estimated by determining O_2 consumption and usually CO_2 production.

There are three general methods for gas collection which may be classified according to operating principle as closed-circuit, total collection and open-circuit. In a closed-circuit system the subject is placed within or breathes into a sealed container. The carbon dioxide, water vapor produced and the oxygen consumed are measured by metering in the gases required to replenish the system. In total collection systems all air expired is analyzed. In the open system the subject breaths from a constantly replenishing air supply and the air expired flows to an outlet for constant or periodic gas measurement. Air flow is measured at the inlet or outlet side.

Most respiratory exchange methods depend on the measurement of oxygen consumption, carbon dioxide production and possibly methane production and/or urinary nitrogen excretion. Methane is a by-product of microbial fermentation in the rumen (Kleiber, 1961) and is not considered when estimating poultry heat production. No correction is usually applied for nitrogen excretion since Romijn and Lokhorst (1961, 1966) indicated that the error resulting from this omission is about .2% and should not exceed 1.5% even at a high rate of protein catabolism in poultry. The preferred equation for determining heat production (HP) is (Brouwer, 1965):

HP = 16.18 (oxygen consumed) + 5.02 (carbon dioxide produced) which is modified for poultry. Where oxygen and

carbon dioxide are given in liters at standard, temperature and pressure.

The measurement of heat production by indirect calorimetry in poultry can be used to determine the energy of a feed that is available to the bird for maintenance and growth (Shannon and Brown, 1969; Burlacu et al., 1970a,b) or it can be used to estimate the energy required by the bird in a specific set of conditions. Factors like energy requirements for maintenance and production (Waring and Brown, 1965, 1967; Burlacu and Baltac, 1971), tissue synthesis (Shannon and Brown, 1970), effects of temperature (Romijn and Lokhorst, 1966; van Kampen, 1974; Farrell and Swain, 1977), deficiencies (Klieber, 1945), nutrient imbalances (Baldini, 1961), diseases (Sykes, 1970) and other conditions that may change the energy use of the bird.

RESPIRATORY QUOTIENT: The ratio of volume or moles of CO_2 produced to the volume or moles of O_2 consumed is known as the respiratory quotient (RQ). RQ's are only applicable when the biological substrates are completely oxidized to CO_2 and H_2O and the nitrogenous excretion products (Zuntz and Schumburg (1901). Typical RQ's (Table 1) for carbohydrate are 1.0; for mixed fats, .7; and for mixed protein .81. Each specific carbohydrate, fatty acid or protein may have distinctive RQ. The RQ's of fats with short-chain fatty acids is about .8 and long-chained fatty

Table 1. The heat equivalent of oxygen and of carbon dioxide under different circumstances (Blaxter, 1989)

Substrate oxidized	RQ	O_2 consumed (Kj/l)	CO_2 produced (Kj/l)
Lipid	.71	19.7	27.8
Protein	.81	19.2	23.8
Carbohydrate	1.00	21.2	21.2
Lipid synthesized	1.10	21.7	19.7

acids are typically around .7 (Church and Pond, 1988).

When indirect calorimetry is used to estimate RQ, CO_2 output and O_2 uptake of the lungs are measured, but in actuality the gases should be measured at the cellular level. The measurements only define correct energy data in a steady state condition when the gas exchange from the tissue is equal to that of the lungs. In short term experiments with low O_2 consumption and CO_2 production the possible changes in O_2 and CO_2 content of the body fluids may cause very large errors in the energy production data (Boshouwers and Nicaise, 1983).

Several investigators have reported RQ values lower than .7 in fasting fowl (Romijn and Lokhorst, 1961; Farrell, 1974; Morrison and Leeson, 1978). Boshouwers and Nicaise (1983) stated that RQ values lower than .7, discounting experimental error, must originate from a nonsteady state or from metabolic interconversions in which oxygen is used for the partial oxidation of the biological substrates. Bleibtreu (1901) reported RQ values greater than 1.0, he explained this high RQ by the synthesis of fat to carbohydrate which results in a partial liberation of O_2.

Considering the many deviations from biological RQ values, is it a useful measurement when using indirect calorimetry? Kleiber (1961) suggested that the RQ is inadequate as an index for the nature of intermediary metabolism, but he also indicated that the RQ provides a general indicator for the nature of metabolism and is useful

as a general indicator of the normality of an animal's metabolism. As an example, if we measured the basal metabolism of an animal and found an RQ of 1.0 instead of .7 we have reason to suspect that the animal was not fasted.

VARIATION IN HEAT PRODUCTION MEASUREMENTS

Poultry heat production has been measured extensively, but comparison of the results from various investigators is difficult due to variation in bird plane of nutrition, activity, sex, laying activity, environment, breed, age, feather insulation, seasonal effects, or because the experimental conditions vary or are insufficiently described. Placing heat production or basal metabolic rate on a per unit metabolic size basis aids in elimination of some sources of variation however, when comparisons are made caution should be used when relating energy expenditure between studies.

FEEDING: The ingestion of feed is accompanied by an increased rate of heat production. The amount of feed consumed depends on many factors including age, environmental temperature, day length, activity, stage of reproductive cycle, appearance and taste of feed and the availability of water (Sturkie, 1986). The heat increment of feed is a fraction of metabolizable energy and is formed by feed metabolism and to a smaller degree by digestion,

glandular secretion and movements of the gastrointestinal tract associated with feed consumption.

The amount of energy lost through the heat increment varies with a number of factors but especially with the plane of nutrition and the balance of nutrients in the ration. Heat production has been reported to be lower when poultry are restricted in their feed intake. MacLeod et al. (1979) indicated layers fed 80% of ad libitum intake had a 25%/$Kg^{\cdot 75}$ lower fed heat production then birds fed ad libitum.

There is a substantial variation in feed intake when birds are allowed to consume feed ad libitum. Feed intake should be controlled when studying metabolic parameters. Birds should be fasted, restricted in their feed intake or force fed. Teeter et al. (1984) has developed methodology and equipment for rapid force feeding poultry meals of a specific quantity. He reported similar weight gain, feed efficiency, dressing percentage, diet dry matter and starch digestibility between birds force fed and birds allowed to consume feed ad libitum.

ACTIVITY: Any movement which disturbs the feather cover of the bird will result in some increase in heat loss through the escape of warm air between the skin and feathers. Deighton and Hutchinson (1940) have reported that heat loss varies continuously except when the bird is completely motionless. De Shazer et al. (1970) reported a 20 to 40%

increase in sensible heat loss when standing compared with sitting. Activity resulting from feeding and general movement decreases during darkness. Romijn and Lokhorst (1964) observed a reduction in metabolic activity in early morning but increased at the onset of light, presumably as a result of increased muscle activity this may also be explained as part of the bird's circadian rhythm.

O'Neill et al. (1971) have indicated that total heat loss associated with activity may be divided into the muscular energy expenditure associated with work and a physical heat loss fraction associated with the breaking of the insulation layer. As the ambient temperature decreases, the physical heat loss fraction will presumably be more important, while the muscular energy fraction associated with feeding should be constant regardless of temperature (Balnave, 1974).

OTHER VARIABLES: Standard metabolic rate of White Leghorn layers is about 50% higher per unit body weight then cockerels (O'Neill and Jackson, 1974a). With most of the difference coming from laying because layers have a higher basal metabolic rate then nonlayers (Waring and Brown, 1967). Male broilers have a greater heat production than female broilers due to their increased growth rate and feed intake (Meltzer, 1983).

Breed differences may contribute to some of the variability. Experiments using similar conditions and techniques can be compared to show breed differences in mature fowl. O'Neill (1971) indicated that the White Leghorn had a starving heat production of 102.3 $Kcal \cdot kg \cdot^{-75} \cdot day^{-1}$ while Shannon and Brown (1969) reported a starving heat production of 68.7 $Kcal \cdot kg \cdot^{-75} \cdot day^{-1}$ of the light Sussex breed.

Total heat production increases as the bird matures because there is an enlargement of respiratory tissue (Yousef, 1985). In growing turkeys the specific heat production as measured per unit body weight decreases with age (Buffington et al, 1974).

O'Neill and Jackson (1974b) indicated that poorly feathered birds have a higher basal metabolic rate than do normally feathered birds because they must compensate for the additional heat lost due to poor insulation. As a result of increased heat loss reports have indicted that the basal metabolic rate under natural conditions is highest in the winter and lowest during the summer (Tasaki, 1969).

All variation can not be eliminated, many reports have indicated that metabolic rate is variable within the same strain or even in a single bird when measured at different times (Balnave, 1974). These reports should not be unexpected considering the multiple factors that affect metabolic rate.

THERMOBALANCE: PERCEPTION AND REGULATION

Deep body core temperature in birds and mammals is regulated at fixed and relatively stable levels. While peripheral cells can tolerate considerable fluctuations in temperature, deviation of core temperature is characteristic of a pathological condition or thermal distress, which could be fatal. Such an accurate system of body temperature regulation is made possible by a central function that integrates afferent information from core and peripheral thermoreceptors and the effector mechanism. The effector mechanism may result in large compensatory changes in heat production and exchange.

PERCEPTION: Simon (1981) has discussed the phylogeny of temperature regulation, and emphasizes the likelihood that birds and mammals have inherited the same basic network for temperature regulation. Homeothermic animals have two groups of temperature sensors: the peripheral receptors located just below the skin surface and the deep receptors, most of which are located within the central nervous system, i.e. in the hypothalamus, the medulla and the spinal cord. The peripheral thermoreceptors are divided into cold and warm receptors and contribute to temperature regulation. Afferent nerves from the cold receptors respond to a sudden fall of skin surface temperature by a transient burst of

high frequency impulses followed by a lower yet steady level of impulses. Similarly, afferent nerves from warm receptors respond to a sudden increase in temperature. In domestic birds peripheral cold receptors, which are more numerous than warm receptors, have been localized in the tongue, beak and bill (Dawson, 1975).

All animal species have temperature receptors located in the hypothalamus (Stanier et al., 1984). Heating of the anterior hypothalamus of a house sparrow results in decreased heat production whereas cooling this region has the opposite effect (Sturkie, 1986). Work conducted in the domestic fowl has indicated that thermal sensitivity is more important in the spinal cord then the hypothalamus (Avery and Richards, 1983), however Helfmann et al. (1981) has reported that the spinal cord thermosensitivity in the goose was low.

Regulation: Body temperature regulation (Figure 2), for heat production and loss occurs through both central and peripheral nerves. Neural mechanisms take part in input reception and analysis, decision making and effector activation. Endocrine mechanisms take part in neuroendocrine, which link the neural and endocrine mechanisms together, and endocrine-endocrine mechanisms as well as effector activation.

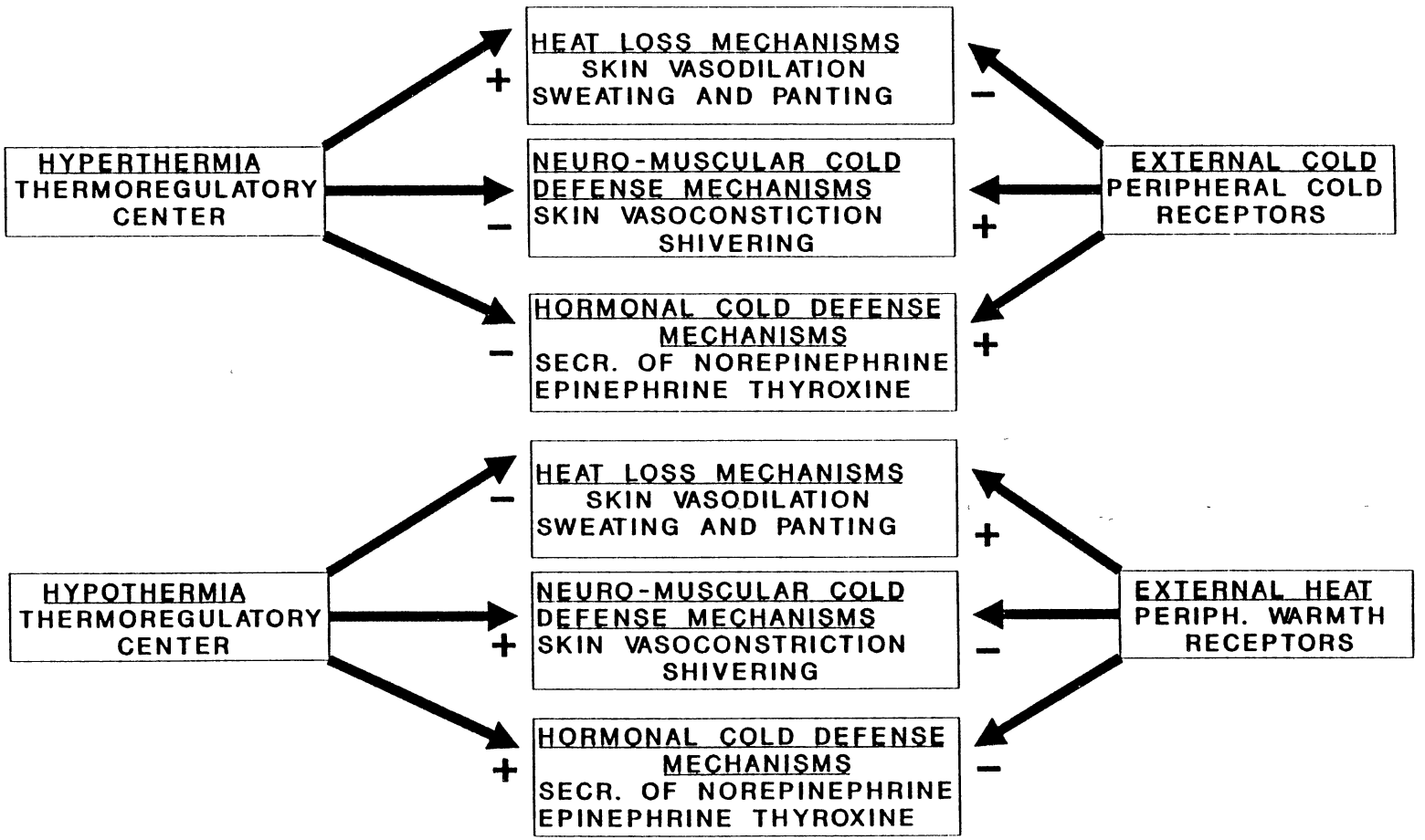

Figure 2. Body temperature regulatory mechanisms (Duke, 1977)

Thyroxine (T_4) is mediated by the release of thyroid stimulating hormone from the anterior pituitary. In birds and mammals the thyroid has the principle function of maintaining a normal heat production and is particularly responsive to environmental temperature. Thyroid secretion (microgams/100 grams of body tissue/day) has been observed to be .63 at 24°C, .34 at 35°C and .24 at 40°C (Heninger et al., 1960). Hoch (1971) observed thyroid size to be reduced in chicks exposed to high temperatures and deAndrade et al. (1977) found a reduced serum T_4 level in chicks during heat distress. During cold distress thyroid secretion rate has been reported to increase in pullets (Hendrich and Turner, 1964). Based on studies in which triiodothyronine (T_3) and T_4 were administered, it appears that T_3 serves an important function in the regulation of heat production and temperature regulation, especially when long-term adjustments to low and high ambient temperatures are considered (Yousef, 1985). Yousef suggested that T_3 is a more potent hormone than T_4 in stimulating metabolism. The stimulation of metabolism probably occurs through the enhancement of oxidative phosphorylation by thyroid hormones in the mitochondria (Sterling, 1977) and an increased activity of Na^+/K^+ ATPase which consumes ATP during its activity to transport Na^+ and K^+ across the cell membrane (Best and Taylor, 1985).

In all species of birds and mammals, the hypothalamus is the principle region in the central nervous system where

the afferent pathways from the temperature sensors act upon the efferent pathways. The efferent pathways activate autonomic and somatic nerves and endocrine glands to regulate body temperature. The anterior hypothalamus is principally involved in the control of responses to warm environments which include panting and/or sweating and increased peripheral blood flow and the posterior region is involved in control responses to cold such as shivering and other increases in heat production.

Epinephrine, norepinephrine and thyroxine play a major role in cold stimulated non-shivering thermogenesis and an increased secretion of these hormones occurs during cold stress. Intrahypothalamic injections of epinephrine, norepinephrine and 5-hydroxytryptamine reduced the body temperature in pigeons exposed to cold temperatures. This was due to the inhibition of shivering, a decrease heat production and peripheral vasodilation (Hissa and Rautenburg, 1975). It is therefore evident that both neural and endocrine mechanisms are involved in the temperature regulation of the domestic fowl.

It has been reported that injections of calcium and other cations cause an abrupt change in body temperature. Hasama (1930) found that excess sodium, potassium and barium injected directly into the hypothalamus of a cat resulted in hyperthermia while calcium produced a reduction in temperature. Denbow and Edens (1980) indicated broilers under thermoneutral conditions injected with sodium in the

cerebral spinal fluid had an increased rectal temperature and injection with calcium decreased rectal temperature. Maki et al. (1988) reported similar results in laying hens, however under temperature distress the action of calcium on the hypothalamus cells appears to be prevented. He speculated that a thermal stressor may have triggered an endogenous ion shift in the hypothalamus to prevent the effect of added exogenous cation. Similarly sodium did not effect body temperature at 20°C.

THERMOREGULATION: HEAT TRANSFER

To preserve homeothermy or homeokinesis animals must conserve heat in cold environments and dissipate it in hot environments (Kleiber, 1961). Heat produced within the body must be transported to the surface of the body for conductive and vascular convection dissipation or for evaporative loss to the mucosa lining of the upper respiratory tract. Transfer of heat from the animal's core to the environment depends on the temperature differential between its surface area and deep core temperature as well as its surface area where peripheral blood flow may be regulated (Nolan, 1978). Heat from the body surface flows to the environment because the surroundings are usually cooler than the body temperature. Compared with large birds small birds have a large surface area to volume ratio. As the ratio of surface area to volume increases efficiency of nonevaporative heat loss increases (Sturkie, 1986).

INTERNAL HEAT TRANSFER

CONDUCTION: Heat transfer by conduction involves the direct transfer of energy from molecule to molecule without the translocation of molecules. The rate of conduction depends on factors such as thickness of the tissue layer and tissue type either fat, skin or muscle. Thermal conductivity of fat, skin and muscle is 0.029, 0.337 and 0.500 watts·meters^{-1}·°C^{-1} respectively (Sturkie, p 230, 1986). Although fat has the lowest heat conductivity of all tissues, its role in birds is not as important as in mammals because birds do not have a substantial layer of fat beneath the skin. In birds fat is most variable among the major body tissues. It is localized in the abdominal cavity and serves as a energy reservoir. Heat transfer by conduction is important in cold environments when the blood flow to the skin is nominal.

Thermal conductivity is difficult to measure and to distinguish between avenues of heat transfer within the body so the heat transfer is defined as a single process and is defined as:

$$q = S\ ((Ti - Ts) \cdot r^{-1})$$ (Fourier, 1919)

where

q = rate of heat flow

S = surface area

Ti = body core temperature

Ts = mean skin temperature

r = specific insulation of the body

Thermal conductance (watts/meter^{2}·°C^{-1}) can be defined as S/r and is a measure of ease in which heat flows from the core to the skin surface. It takes into account the conductivity of the skin, muscle and fat, the thickness of these tissues, the shape of the body and the blood flow to the skin. The thermal conductance of tissues underlying feathered areas changes little with variation in temperature and it greater in smaller birds compared to large birds (Sturkie, 1986).

CONVECTION: Heat transfer by convection involves the actual movement of molecules of the air. Internal convection occurs as the blood acquires heat from the internal organs and transfers this heat to the skin. The skin temperature will increase and heat loss to the environment will also increase. Heat transfer between the core temperature and the skin is influenced by a processes referred to as countercurrent exchange system. The warm arterial blood from the internal organs enters the limb and releases its heat energy to the cool blood flowing through the veins from the distal parts of the limbs. In hot environments the venous flow from the distal parts of limbs occurs by the superficial limbs, hence the transfer of heat does not occur until the arterial blood has reached the skin.

HEAT LOSS

Thermophysiology involves the physiologic measurements that reveal the mechanisms of heat exchange between the organism and its environment (Slonim, 1974). Heat transferred through the tissues to the surface of the skin can be lost from the body by both nonevaporative and evaporative heat loss (Figure 3). Nonevaporative or sensible heat loss as it is sometimes called occurs in three main processes: radiation, conduction, and convection.

RADIATION: Radiation heat is in the form of electromagnetic waves and the rate of heat exchange is proportional to the ambient temperature surface temperature differential. Radiation is supported by the internal steps comprising heat flow through circulatory convection and conduction from the body core, through the shell, to the skin surface and heat flow by conduction from the skin through the cover layer to the outer edge of cover. The net radiant heat exchange between the bird and environment has been estimated by the following equation:

$$R = \delta E_s E_r (T_s^4 - T_r^4) A \text{ (Sturkie, 1986)}$$

where

R = net radiant heat exchange (watts)

$\delta = 5.67 * 10^{-8}$ watts$\cdot m^{-2} \cdot {}^{\circ}K^{-4}$

(Stefan-Boltzmann constant)

E_s = emissivity of the surface of the bird

E_r = emissivity of the environmental surfaces

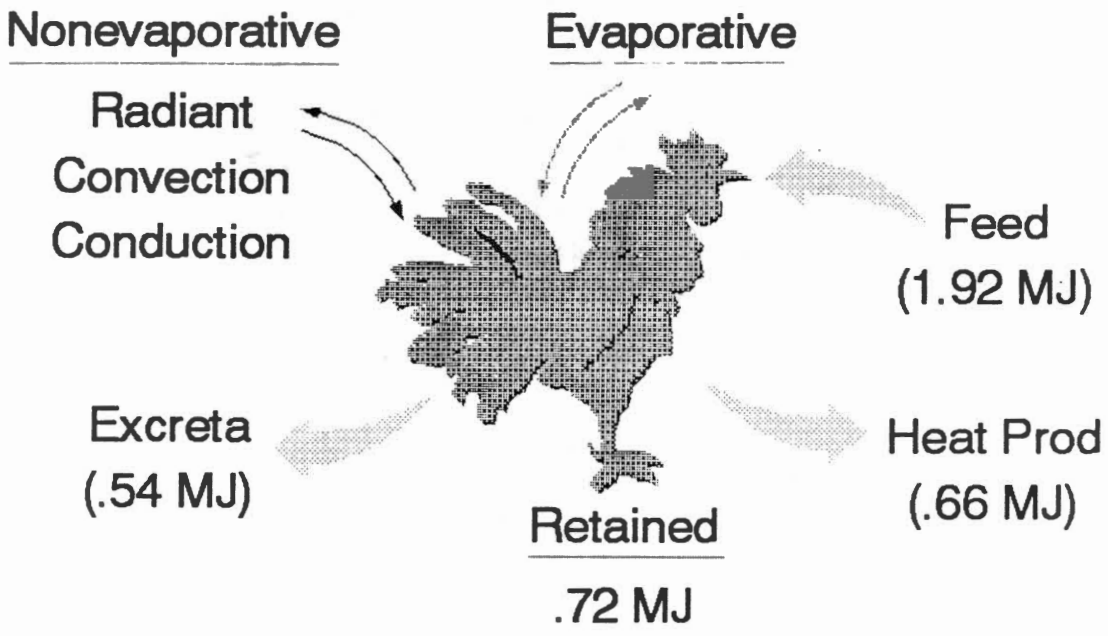

Figure 3. Principle energy exchanges between bird and environment

T_s = mean surface temperature of the bird (°K)

T_r = mean radiant temperature of the environment (°K)

A = effective radiating area (m^2)

The skin and feathers of a bird are very close to a perfect black body with respect to radiation of long wavelengths, therefore they radiate and absorb heat efficiently to the environment.

The skin and feathers absorb large quantities of wave radiation from the ground and sun. Intuitively speaking birds with dark plumage absorb more heat radiation from direct sunlight but measurements of heat transfer reveal a more complex situation because a bird that absorbs more radiant heat will usually lose more heat through convection. In wind speeds of excess of 3 m/sec black plumage actually absorbs less heat energy then white plumage (Walsberg et al., 1978). This is due in part because dark plumage absorbs the heat in the superficial layers were a greater convection takes place.

CONDUCTION: Conduction is the heat transfer through a medium without material movement or transfer. A warm molecule collides with a cool one and transfers some of its kinetic energy too that molecule. The conductive heat flow formula is represented as follows:

$$K = h_K (Ts - Ta)$$ (Sturkie, 1986)

where

K = conductive heat loss ($watts \cdot m^{-2}$)

h_k = conductive heat transfer coefficient

(watts·m^{-2}·°C^{-1})

T_s = mean surface temperature

T_a = ambient temperature

Thermal conductivity depends on the medium to which the heat is being lost be it air, water, or the ground. The knowledge of conductive heat flow is inadequate to formulate accurate conductive heat loss.

CONVECTION: Convection is the heat transported by streams of molecules from a warm place to a cool one. For convective heat flow the internal step includes heat flow by circulatory convection and conduction from the body core through the shell to the skin surface, heat flow by conduction from the skin to the outer edge of the cover and heat flow by conduction through the boundary layer to the outer edge.

There are two types of convection. Free convection occurs in still air when the air in contact with the skin warms then rises and is replaced by cooler less dense air. Forced convection occurs when winds and drafts move past the surface of the bird or when the animal moves. For forced convection it is known that the heat flux is directly proportional to the square root of air speed. Convection may be expressed as:

$$C = h_C (T_s - T_a)$$ (Sturkie, 1986)

where

C = convective heat loss (watts·m^{-2}

h_c = convective heat transfer coefficient (watts·m^{-2}·°C^{-1}

T_s = mean surface temperature

T_a = ambient temperature

Until a better understanding of the convective heat transfer coefficient is proven, measurements of convective heat flow will be difficult to analyze.

EVAPORATIVE HEAT LOSS: In poultry evaporation of moisture occurs from the skin surface and from the respiratory tract (Sturkie, 1986). When water is evaporated energy is lost at a rate of 586 cal·g water evaporated at 20°C. Evaporative water loss occurs at the skin due to passive diffusion of water vapor through the skin. The passive diffusion is not directly regulated by thermoregulation and may be estimated by:

$$CEHL = M\ A_{sur}\ (P_{skin} - P_a)$$ Ó (Yousef, 1985).

where

CEHL = rate of heat loss by water vapor through the skin

M = permeance coefficient of the skin to water vapor which is function of air velocity, air direction, geometry and nature of the skin surface

A_{sur} = surface area of the animal

P_{skin} = saturated partial pressure of water vapor at skin temperature

P_a = partial pressure of water vapor at the ambient

temperature

Ó = total heat of evaporation of water

Neither sweat glands nor sebaceous glands are present in the skin of birds. The amount of moisture that may be lost through the skin under heat distress is therefore limited. Nevertheless Bouverot (1974) indicated that in Peking ducks the cutaneous water loss accounted for 2/3 of the total evaporative heat loss at 20°C, 1/2 at 30°C and 1/4 at 35°C.

Evaporative heat loss occurs mainly from the upper respiratory tract. A significant proportion of the total heat loss is lost through respiratory evaporative heat loss when birds are panting. As the air passes over the wet surfaces of the respiratory tract, the air becomes saturated near the body temperature. Some of the heat is lost back to the upper respiratory tract and some of the water vapor is condensed as the bird exhales, however as long as the inspired air is not saturated and equal to the temperature of the body the expired air contains more heat than the inspired air. Respiratory evaporative heat loss may be estimated by:

$$REHL = rho * V (W_{ex} - W_a) Ó_{resp}$$ (Yousef, 1985).

where

REHL = rate of respiratory evaporative heat loss

p = mean density of air

V = ventilation rate of respiratory air

W_{ex} = humidity ratio of expired air

W_a = humidity ratio of inspired ambient air

$Ó_{resp}$ = latent heat of evaporation of water at mean temperature of the surface of respiratory tract

Thermal polypnea involves an increase in the respiratory minute volume which leads to an increase in respiratory evaporative heat loss. The increased volume is brought about by an increased respiration frequency while the tidal volume decreases. This respiratory pattern permits a maximum increase in respiratory minute volume and respiratory evaporative cooling, with a small amount of disturbance of the blood gases because the increased ventilation is limited to the respiratory dead space where gas exchange does not occur. However as the ambient temperature increases more emphasis is placed on the respiratory system to cool the body, the respiratory frequency reaches a maximal value an subsequently declines. This change in respiratory frequency is accompanied by an increase in tidal volume while the minute volume increases further, but at very high temperatures the respiratory minute volume declines. This pattern of breathing is characteristic of panting animals and it represents the breakdown of thermal polypnea.

Respiratory nonevaporative heat loss is a special process in which warming of inspired air takes place by convection, conduction in the upper respiratory tract. When air is inhaled it removes heat from the nasal passages and

when air is exhaled it is cooled and the nasal passage retains the heat. The temperature of the exhaled air depends on the surface area and width of the nasal passages, bird variation is significant in this respect. This counter exchange system is important in respiratory evaporative heat loss.

THERMOBALANCE: ENVIRONMENTAL INTERACTION

A thermoneutral environment or comfort zone is defined as one which the animal does not need to increase energy expenditure to either warm or cool the body. The critical temperature is defined as the point at which an animal must increase its heat production to prevent body temperature from falling or increase the rate of heat dissipation to prevent body temperature from rising.

Total productivity and production efficiency of all animals declines as the ambient temperature diverges from the thermoneutral zone. In broilers substantial declines in productivity occur as a result of alterations in feed consumption and feed utilization. At an environment below the thermoneutral zone, broiler maintenance requirement for energy is enhanced thereby forcing greater feed consumption to maintain normal levels of production. Feed efficiency declines in these cases due to the added energy requirement to maintain body temperature. At environmental temperatures above the thermoneutral zone maintenance requirements for

energy are also increased, but feed intake is reduced thereby lowering productivity and production efficiency.

Heat production and heat exchange vary with environmental temperature with the least amount of energy being produced and exchanged within the thermoneutral zone and referred to as the basal metabolic rate. Animals lose heat by conduction, convection and radiation from the body surface, and by evaporation of water from the body surface, lungs and oral surfaces. The rate of heat exchange depends on the difference between the body surface temperature and the environmental temperature. Surface temperature may be altered by constriction or dilation of blood vessels in the skin or extremities, thus increasing or decreasing surface temperature in relation to the environment. During ambient temperature distress birds will increase respiration rate, cardiac output and decrease blood pressure to enhance blood flow to the extremities and tissues of the respiratory system (Michael and Harrison, 1987). Other factors effecting heat loss include posture, sweating, panting and insulation of subcutaneous fat.

Environment plays a key role in heat production, but defining the thermoneutral zone in which the heat production is minimal has been shown to be variable. Barott and Pringle (1941) reported the thermoneutral zone for the hen to be between 22.8 and 27.7°C, while Randall (1943) suggests a range from 19 to 29°C. Van Kampen and Romijn (1970)

provided a line of best fit to the results of heat production and environmental temperature from -5 to 40°C:

$$HP = 0.2453\ Ta^2 - 18.90\ Ta + 803.3$$

where

HP = heat production $kJ \cdot Kg^{-.75}$

Ta = ambient temperature (°C)

This equation may be used to estimate heat production in widely differing temperatures.

The range of air temperatures over which birds are in thermoneutral zone (TN) varies from a few degrees °C (Figure 4) in younger birds to as much as 30°C in penguins (Le Maho, 1983). The heat production within the thermoneutral zone also depends on the level of feed intake prior to the measurement. The higher level of feed intake, the higher the heat production (Sturkie, 1986) and the heat increment will persist for a period of time before the basal metabolic rate is reached again.

In a thermoneutral environment, Brody (1964) indicated about 25% of the body heat is dissipated by moisture vaporization and about 75% of the heat is lost through sensible heat. Sensible heat may be measured by either direct or indirect calorimetry. Insensible heat must be estimated by determining the amount of water vapor lost by the animal. Farrell and Swain (1977) reported that EHL was 85% of HP at 35°C and declined to 30% at 2°C.

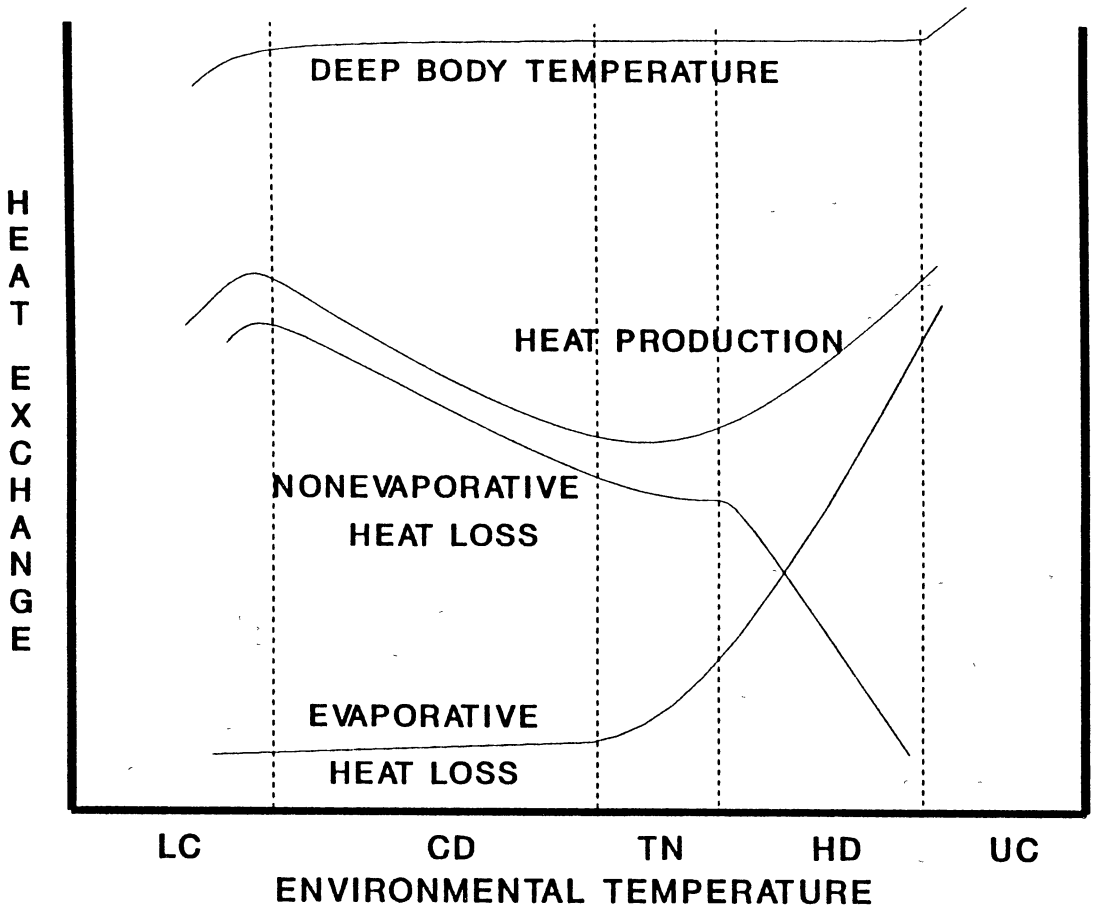

Figure 4. Environmental temperature effects on core body temperature, heat production, nonevaporative and evaporative heat loss

COLD DISTRESS: Homeotherms exposed to low temperatures increase their metabolic rates to maintain deep core temperature. When an animal is subjected to its lower critical temperature, the limit of heat production capacity is reached. If the environmental temperature becomes colder, the deep core body temperature begins to fall, metabolic heat production decreases and may lead to death by hypothermia.

Birds in cold environments must increase heat production in order to maintain homeostasis. Increased heat production may include biochemical calorigensis, heat increment due to feeding and muscular contraction from shivering or activity. Biochemical calorigensis refers to the basal metabolic rate and is a form of respiratory metabolism known as obligatory nonshivering thermogenesis. In birds the possible role of regulatory nonshivering thermogenesis like that of mammals, where enhancement of chemical calorigensis occurs in brown adipose tissue under sympathetic control, remains controversial. Heat production due to feeding may increase the basal metabolic rate as much as 20% and is referred to the heat increment of feed (Calder and King, 1974; Berman and Snapir, 1965). Shivering produces the largest sources of added heat production.

When the ambient temperature falls below the lower critical temperature, heat production increases due to shivering. The heat produced by shivering is derived

largely from the oxidation of fatty acids (Marsh and Dawson, 1982). The rate at which the heat production increases in response to exposure of low ambient temperatures depends on the insulation of the tissues and feathering. The greater the insulation the lower the rate of heat production. Smaller birds have less insulation then lager birds and as a result the rate of increase in heat production is greater.

Shivering occurs in a variety of muscle masses such as muscles of the leg, neck and in the pectoral muscle (Hillman et al., 1977). Shivering is evident at an early age and it coincides with the development of homeothermy (Odum, 1942). Randell (1943) and Freeman (1966) reported shivering was not pronounced or was absent in chicks 2 to 24 hours old, however in a more recent study electromyographic activity was found in day old chicks (Saarela, 1976).

COLD ACCLIMATION: Birds acclimated to cold temperatures have an altered shivering response. Arieli et al. (1979) reported that during cold exposure and with birds shivering, oxygen consumption is higher in cold acclimated birds than summer acclimated birds. No differences in basal metabolic rate were seen between the two groups of birds and it appears that, when exposed to cold distress, the difference comes from an increased capacity for oxidative metabolism. Cold exposure increases heart size resulting in an increase supply of blood to the muscles (Aulie, 1977). Davison (1973) suggested that gluconeogensis is activated during

cold exposure in neonatal fowl since a rapid increase in plasma free fatty acids and a drop in plasma triglycerides was observed shortly after cold exposure.

HEAT DISTRESS: As the environmental temperature rises within the thermoneutral zone temperature regulation is maintained by increased heat dissipation. At temperatures above the thermoneutral temperature the capacity of nonevaporative heat dissipation may be exceeded resulting in an increased body temperature and metabolic rate which may result to death in hyperthermia.

At ambient temperatures above the thermoneutral zone, the heat production increases as a result of an increase in body temperature. The increased temperature of tissues results in an acceleration of chemical reactions and consequently in an increased oxygen requirement and heat production, known as the van't Hoff-Arrhenius effect.

High ambient temperature-relative humidity distress has been shown to reduce broiler body weight gain, feed efficiency, and survival. The birds total heat load is due to a combination of environmental heat and metabolic heat production. Birds produce and therefore must dissipate considerable amounts of heat daily as their energetic efficiency is less that 15%. Heat distressed broilers must remove generated body heat in order to maintain body temperature at near normal levels. As the ambient temperature rises heat loss associated with nonevaporative

cooling declines markedly as the temperature differential between the bird and its environment is reduced.

During increased high ambient temperature exposure the bird's heat load is increased due to the environmental heat gain and the energy cost associated with activation of metabolic processes required for heat dissipation (Meltzer, 1987). Heat dissipation is enhanced by postural adjustments to increase surface area (Baldwin, 1974), vasodilation of unfeathered extremities (Nolan et. al, 1978), by increasing water intake (Farrell and Swain, 1977), by increasing urine volume (van Kampen, 1981) and elevating respiration rate from the basal 25 breathes per minute to as much as 250 (Frankel et al, 1962). Aureli (1980) estimated 4 $mg \cdot Kg^{-1} \cdot min^{-1} \cdot {}^{\circ}C^{-1}$ of evaporative water loss when broilers were subjected to ambient temperatures above 26°C. Respiration rate is of particular importance as a considerable amount of water is evaporated from the mucous membranes of the respiratory tract. During heat distress, blood flow to this region is doubled to provide water for evaporation (Freeman, 1984).

Barott and Pringle (1946) measured a 17% increase in bird heat production at ambient temperatures above the thermoneutral zone. They concluded that this increase resulted from the energetic cost of panting. However Romijn and Vreugdenhil (1969) did not observe an increase in heat production due to panting in fowl between 35 and 40°C even though the respiration rate increased from 30 to 150 breaths

per minute. The true cost of panting requires further study because an increase in the metabolic demand of muscles involved in panting may be offset by a decreased metabolic demand of other tissues (Weathers and Schoenbaechler, 1976).

Increased respiration rate during heat distress is critical to maintain constant body temperature. However, the increased respiration rate lowers the partial pressure of carbon dioxide in the lungs causing a lower concentration of bicarbonate in the blood resulting in a elevated blood pH. When chickens are exposed to heat distress, both partial pressure of carbon dioxide and bicarbonate in blood decreases thereby increasing blood pH (Bottje et al., 1985; Teeter et al., 1985).

Birds under heat distress must deal with the conflict between the hyperthermic response to increase tidal volume for greater evaporative heat loss and the need for minimizing hypercapnic alkalosis by reducing tidal volume. When the body temperature reaches about 44°C respiration rate reaches a peak of 150 to 260 breathes per minute (Frankel et al., 1962; Kassim and Sykes, 1982), tidal volume decreases as respiration rate increases, but this is not enough to prevent minute volume from increasing. This pattern of respiratory events is termed phase I panting which is followed by phase II if hyperthermia proceeds further. Phase II panting is characterized by a slow deep breathes which increases the tidal volume. Phase II panting results in a acute respiratory alkalosis (Yousef, 1985)

HEAT ACCLIMATION: Bird acclimation to heat distress may be defined as physiological adaptations made to maintain homeostasis during high ambient temperature. The process has been studied by Hutchinson and Sykes (1953); Reece et al. (1972); Farrell and Swain (1977); Bohren et al. (1982); May et al. (1987). Bird acclimation to heat distress reduces the mortality loss associated with high ambient temperature distress (Reece et al., 1972; Bohren et al., 1982). Davis et al. (1972) indicated that the fasting heat production is reduced in heat-acclimated birds and specified that the response may be due to a decrease in thyroid activity (Shafie et al., 1979). Long term adaptation to heat does not appear to be related to increased respiratory evaporative heat loss efficiency (Weiss et al., 1963). In contrast DeShazer et al. (1970) reported that the sensible heat loss was enhanced by an increased blood flow to the unfeathered extremities when birds became acclimated to heat distress.

APPLICATION AND BENEFIT

Though the specific routes of heat loss, as discussed, are well defined qualitatively little research has been conducted to quantitatively estimate the importance of each. Indeed, a comprehensive model has not been developed enabling their study much less their potential manipulation into therapeutic measures. A model is needed which enables

the quantification of thermobalance by the heat distressed bird. Once a model is available, methods manipulating the various processes may be evaluated.

The purpose of this study is to develop a model enabling the quantification of thermal balance in the heat distressed broiler. Additionally, the effects of feed intake on the heat distressed bird will be examined. Data collected will be used to evaluate if the birds inability to control its body temperature at high ambient temperature-relative humidity is due to the bird's inefficiency of heat dissipation or higher metabolic rate.

Numerous studies have been conducted estimating bird heat production, evaporative heat loss and nonevaporative heat loss but few have ever put the concept of thermobalance into one system. The system designed at the Oklahoma State University Poultry Research Center has been designed to give rapid analysis of the various physiological processes involved in poultry heat balance during thermoneutral and heat distress environments.

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CHAPTER III

FEEDING EFFECTS ON BROILER THERMOBALANCE DURING THERMONEUTRAL AND HIGH AMBIENT TEMPERATURES

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ABSTRACT

To estimate feed consumption and environment (env) effects on broiler thermobalance (TB). Thermobalance was defined as: heat production (HP) = evaporative heat loss (EHL) ± nonevaporative heat loss (NHL) ± change in bird heat content (HC). Birds were precision fed 0, 35, 70 and 105% of metabolic body weight (mwt) per day during thermoneutral (TN; 24 C) and HD (32-35 C) conditions. Bird HP, averaged over a 12 hr period, increased ($P<.01$) linearly with feeding level within both envs. Averaged over env HP ranged from 22.2 to 24.6 kJ/hr·mwt^{-1} for 0 and 105% feeding levels, respectively. High ambient temperature exposure increased HP over all feeding levels. Route of heat dissipation varied with env. Within the TN, EHL and NHL accounted for

4.6 and 20.1 kJ/hr·mwt^{-1} while in HD, 8.2 and 13.3 kJ/hr·mwt^{-1} were noted respectively. These data indicate that feeding level and HD effects on HP are additive. When environmental conditions limit nonevaporative heat dissipation, feed consumption exacerbates the HD state.

(Key Words: broiler, feeding, heat distress, thermobalance

INTRODUCTION

Avian homeostatic processes must function to maintain bird homeostasis during heat distress (HD) or body temperature rises (May et al., 1987) and growth and survivability declines. As ambient temperatures increase, the birds ability to dissipate heat as nonevaporative heat loss declines since the differential between bird body temperature and the environment is reduced. Consequently during high ambient temperature exposure the compensatory responses, such as increased respiration rate (Frankel et al., 1962), adjustments to increase surface area (Baldwin, 1974), vasodilation of extremities (Nolan et al., 1978) and increased water intake (Farrell and Swain, 1977), must be elicited to maintain homeostasis. Such compensatory responses have been reported to increase the bird's heat load.

Before significant progress can be made to eliminate the deleterious consequences of HD, rate limiting factors

impacting thermobalance (TB) must be identified. Bird TB may be estimated as: heat production (HP) = evaporative heat loss ± nonevaporative heat loss (NHL) ± change in bird heat content (HC) (Sturkie, 1986). Presumably, minimizing bird HC changes would be associated with optimal bird TB during high ambient temperature exposure.

Nonevaporative heat loss is likely the most energetically efficient means to dissipate heat. Birds manipulate NHL by increasing vasodilation of extremities (Nolan et al., 1978; Bottje et al., 1983) and shunting blood to and from the gastrointestinal tract. However, as discussed, the extent of NHL declines as ambient temperature rises (van Kampen, 1974; Wiernusz, et al., 1991) and the bird must consequently reduce HP or increase other heat dissipating mechanisms to minimize HC change.

EHL through the latent heat of vaporization for H_2O is increased by enhancing respiration rate. Cutaneous moisture loss from birds represents only 15% of total EHL during heat distress (van Kampen, 1974). Indeed EHL may become the major heat dissipation route during heat distress (van Kampen, 1981) as such birds may dissipate over 80% of heat production via this method provided the relative humidity is not excessive (van Kampen, 1974).

The effects of high ambient temperature on HD have been mixed. Van Kampen (1974) reported that bird heat production is inversely related to ambient temperature. In contrast, van Kampen (1981) and Chwalibog and Eggum (1989) reported

that HP increases when birds are exposed to HD. Such contradictions may be related to the duration of HD and the bird's feed consumption pattern. Feed consumption has long been known to increase bird heat production (Macleod et al., 1979) and to decline with HD exposure (Squibb et al., 1959). Therefore, it is expected that studies conducted when feed intake is uncontrolled will give varying results.

The importance of broiler feed consumption on TB was documented by McCormick et al. (1979), who reported that fasting broilers for 24, 48 or 72 hours prior to HD exposure increased bird survival time. Similarly, Teeter et al. (1987) reported that removing feed from chicks for as little as 6 hours prior to HD initiation increased survivability and that bird survival increased with fasting duration. Smith (1983) suggested that bird feed consumption alterations are in response to an existing stress which does not allow sufficient time for feed to clear the bird's digestive tract and reduce substrate availability.

Quantitative measurements describing these relationships must be determined, therefore the objective of this study was to quantify feeding effects on broiler thermobalance criteria in thermoneutral (24°C) and heat distressed (32-35°C) environments.

Materials and Methods

The Cobb x Cobb male broilers used in the following experiment were fed a 23% crude protein corn-soybean based

ration and reared in floor pens on rice hull through 28 days posthatching. On the first day of the 5th week 24 birds were transferred to individual 47 x 26 cm wire floored cages housed within an environmentally controlled room. Ambient temperature was maintained at 24 ± 1°C and treatment groups were switched to a 21% crude protein grower ration (Table 1). During week 5 posthatching, 12 birds were selected for similar body weight, aseptically prepared and implanted with a radiotelemetry temperature transmitter (Mini-Mitter telemetry system, Sunriver, Oregon 97707) in the abdominal cavity following the procedure described in the Dataquest III User's Manual of Mini-Mitter telemetry (Data Sciences, Inc., St. Paul, MN 55113, Document no. 10000-05, 1988). Anesthetic induction and maintenance was achieved by a ketamine HCl intramuscular injection (40 mg/kg of body weight) and halothane in oxygen via Bain nonre breathing system, respectively. Following 2 days for surgical recovery, birds were placed in 12 individual respiration chambers (51 x 34 x 41 cm) and allowed a 3 day adjustment period to chambers as recommended by Misson (1974).

Experiment 1

Birds were fasted 12 hours prior to experimental initiation and precision fed (Teeter et al., 1984) the grower mash at 0, 35, 70 and 105% metabolic body weight (mwt) per day. Treatment groups were maintained at an

Table 1 Diet composition of grower ration[1]

Ingredients	Percent
Ground corn	61.12
Soybean meal (48.5% CP)	31.06
Fat	3.80
Dicalcium phosphate (22% Ca; 18.5% P)	1.70
Limestone (38% Ca)	1.30
salt	.40
Vitamin mix[2]	.30
DL-Methionine, 99%	.22
Trace mineral mix[3]	.10
Total	100.00

[1]Calculated analysis:

ME Kcal/Kg	3128.40
Crude protein (%)	20.60
Calcium (%)	1.00
Phosphorus (% av)	.44

[2]Mix contained Vit A, 3,968,280 I.U.; Vit D_3, 1,102,300 I.U; Vit E, 13,228 I.U.; Vit B_{12}, 7.9 mg; Riboflavin, 2,646 mg; Niacin, 17,637 mg; d-Pantothenic Acid, 4,409 mg; Choline, 200,178 mg; Menadione, 728 mg; Folic Acid, 441 mg; Pyridoxine, 1,587 mg; Thiamin, 794 mg; d-Biotin, 44 mg per Kg.

[3]Mix contained Manganese, 12.0%; Zinc, 10.0%; Iron, 7.5%; Copper, 1.0%; Iodine, .25%; Calcium 13.5%.

ambient temperature of 24±.5°C for 12 hr. Water was available for ad libitum consumption and consumption was monitored throughout the experiment. Bird TB response variables monitored included HP, EHL, core body temperature, dHC, NHL, respiration rate and respiration efficiency as described below.

Respiratory Chambers

The 12 (51 x 34 x 41 cm) respiratory chambers were constructed of clear 63.5 mm acrylic plexiglas and fitted with a Hart watering cup. Water supplying the Hart cup for each chamber was contained in a 1000 ml graduate cylinder. Water disappearance from the cylinder was computed 3 x daily such that water consumption could be estimated. The chamber floor (51 x 34 cm) was constructed of wire mesh suspended 9 cm above a 51 x 34 cm excreta collection pan containing 4 cm of mineral oil so that voided excreta moisture would be 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, for air mixing ensuring 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 (Gardner-Denver, Quincy, IL. 62305) and dried by a compressed air dryer (Hankison, Cannonsberg, PA. 15317). Incoming air for each chamber moved through independent 64 mm diameter tubing such that the air temperature within each tube reached room temperature prior to entry 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. A pressure regulator released flow at a constant pressure from the air compressor and microvalves were used to direct the desired flow rate through each chamber. All flow rates were monitored by an electronic mass flow meter (Omega Engineering, Stanford, CT 06907). Air flow fluctuated less than $\pm$ 1% throughout the experiment.

Relative Humidity, O_2 and CO_2 Analysis

Air moisture, O_2 and CO_2 concentrations of bird breathing air were determined by an Omnidata International (Logan, UT. 84321) relative humidity probe (accuracy $\pm$ 1%) and Ametek (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/mwt·hour^{-1}) 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 Lokhorst 1961, 1966).

Evaporative Heat Loss (EHL)

Bird water evaporative heat losses (respiratory and cutaneous) were estimated by coupling water evaporative losses with latent heat of vaporization for water. To estimate water evaporative loss relative humidity (RH) was converted into g H_2O/L^3 gained as the air passed through the respiratory chambers. To accomplish this, air at saturation for various ambient temperatures (Handbook of Chemistry and Physics, 1987) was regressed against ambient temperature (SAS, 1982) and the resulting equation:

$_{sat}H_2O = 8.694 - .218391 \times T + .03145 \times T^2$

where

$_{sat}H_2O$ = saturation of water

T = air temperature

The equation was used to estimate water content of air exiting the chamber as follows:

$$H_2O = {}_{sat}H_2O \times (RH/100) \times .001$$

Bird H_2O evaporative losses were then estimated as:

$$H_2O_{prod} = (flow_t \times H_2O_t) - (flow_{ref} \times H_2O_{ref}) \times 60$$

where

H_2O_{prod} = water production (g/min)

t = test chamber

ref = reference chamber

The total evaporative heat loss (respiratory water and cutaneous) was calculated by multiplying the estimated evaporative water loss by the latent heat of vaporization (Sturkie, 1986).

$$EHL = H_2O_{prod} \times 2.365$$

where

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

Bird Heat Content (HC)

Body temperature observations were made utilizing the radio telemetry system, described previously, with data recorded once every 1.5 minutes for each bird was used to estimate body heat content change ($kJ \cdot hr^{-1}$) as:

dT (C) X M (Kg) x SH (Sturkie, 1986).

where

dT = body temperature change

M = bird mass

SH = specific heat

The mean specific heat of body tissues was estimated in our laboratory to be 3.17 kJ/Kg·°C^{-1} which differs from 3.5 kJ/Kg·°C^{-1} estimated by Sturkie (1986). The differences may be attributed to age, species and body composition.

Nonevaporative Heat Loss (NHL)

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

Respiration Rate (RR) and Efficiency (RE)

During a respiration cycle (inhale and exhale) chamber pressure declines followed by an increase in pressure. 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) estimated respiration rate 8 times per hour per bird.

Evaporative heat loss is determined by the combined moisture loss from respiratory and cutaneous sources. A small amount of energy is likely utilized to manipulate NHL in contrast to panting, when considerable amount of energy is utilized. Since cutaneous moisture loss from the bird represents only 15% of total EHL during heat distress (van Kampen, 1974), an estimate of respiration efficiency

($J \cdot breath^{-1}$) was obtained by relating EHL with RR as: EHL/breath.

Data Acquisition

The chamber environment and all data measurements were controlled and monitored by a Workhorse Data Acquisition and Control System (Omega Engineering, Stanford, CT 06907). Gas concentration (RH, O_2, CO_2) quantifications, flow rate, respiration rate and ambient temperature were made and recorded once on each of the 12 compartments every 12 minutes.

Statistical Analysis

Variables within the distressed environments were judged for deviation from thermoneutral homeostatic levels to identify potentially rate limiting factors related to bird thermobalance. Response variables, which included: HP, NHL, EHL, dT, RR and RE were regressed against time utilizing SAS (SAS, 1982) such that time dependent polynomial equations could be used to describe the data. Comparisons were made by integrating experiment time intervals to provide quantitative values. All integrated values as well as BT and water consumption values were analyzed by analysis of variance using Proc GLM (Steel and Torrie, 1960).

Experiment 2

The second experiment was conducted to quantify broiler thermobalance criteria during HD. The same birds used in experiment 1 were maintained in the respiratory chambers at an ambient temperature of 24±.5°C. Feed was supplied for ad libitum consumption for 5 consecutive days to allow birds to adjust from the previous force feeding levels. Upon completion of the adjustment period, birds were fasted 12 hr and ambient temperature was increased 3°C/hr over a 2½ hr to reach the target of 32°C. Birds were reallocated to treatment groups and precision fed as described in experiment 1. Following precision feeding ambient temperature was increased 1.5°C/hr until a target temperature of 35±.5°C was obtained. Bird response variables monitored were as described in experiment 1 with the exception of respiration rate and respiration efficiency.

Results and Discussion

Exposing broilers to heat distress resulted in a increased ($P<.01$) body temperature and NHL (Table 2). The reduced NHL, presumably due to the reduced bird-environment temperature differential, is similar to previous reports (Roller and Dale, 1963; Olson et al., 1974). The lowered NHL forces the bird to enhance other heat dissipating mechanisms such as EHL via elevating respiration rate. Frankel et al (1962) reported that birds increased

Table 2 Effects of feeding level and ambient temperature on broiler heat production, nonevaporative heat loss, evaporative heat loss, change in heat content, body temperature, water consumption, respiration rate and respiration efficiency

	Thermoneutral				Heat Distress			
Feeding level[1]	0 %	35 %	70 %	105 %	0 %	35 %	70 %	105 %
HP[2] (kJ/mwt·hr^{-1})	20.3^{f}	21.3^{de}	22.3^{cd}	24.5^{b}	20.7^{ef}	22.9^{c}	25.0^{ab}	26.0^{a}
NHL[3](kJ/mwt·hr^{-1})	15.8^{c}	17.3^{b}	18.0^{b}	19.6^{a}	12.9^{e}	13.5^{e}	14.7^{d}	15.9^{c}
EHL[4](kJ/mwt·hr^{-1})	4.5^{cd}	4.1^{d}	4.4^{cd}	4.8^{c}	7.6^{b}	9.2^{a}	9.8^{a}	9.8^{a}
dHC[5](kJ/mwt·hr^{-1})	-.2^{b}	-.2^{b}	-.1^{b}	.1^{ab}	.5^{a}	.3^{a}	.7^{a}	.8^{a}
BT[6] (C)	40.2^{cd}	40.0^{d}	40.5^{c}	40.6^{c}	41.4^{b}	41.5^{b}	42.2^{a}	41.6^{b}
MWATER[7](ml/mwt)	25^{c}	26^{c}	35^{c}	67^{b}	85^{b}	120^{a}	119^{a}	134.^{a}
RPM[8]	33	35	33	32				
RESEFF[9](J/breath)	2.6	2.1	2.3	3.0				

$^{a-f}$Means within a row differ with unlike superscripts differ (P<.05)
[1]precision fed % of metabolic body weight
[2]HP=heat production
[3]NHL=nonevaporative heat loss
[4]EHL=evaporative heat loss
[5]dHC=change in heat content
[6]BT=body temperature at the end of each period
[7]MWATER=voluntary water consumption + water contained in feed when precision fed
[8]RPM=respiration per minute
[9]RESEFF=respiration efficiency

respiration rate from 25 to as much as 250 breaths/min during HD. No respiration rates were recorded during the HD phase due to a faulty respiration monitor. However, in this study EHL increased ($P<.01$) by 104% (Table 2) during HD similar to data reported by van Kampen (1974, 1981) and Wiernusz (1991).

Heat production increased ($P<.05$) linearly with feeding level in both environments. Averaged over feeding level HP was elevated ($P<.05$) during HD (Table 2). Increased HP during HD has been observed (van Kampen, 1974; Wiernusz et al., 1991) and suggested by Barott and Pringle (1946) to be related to the energetic cost of panting. However, other reports have indicated that HP of HD broilers is reduced (van Kampen, 1981; Chwalibog and Eggum, 1989). Such contradictory studies may be related to variation in feed consumption as Macleod et al. (1979) reported that restricted feeding lowered HP. Indeed in this study, fasted birds had a similar ($P>.1$) HP in both environments. Regressing HP on feeding level within the two environments yielded a .04 kJ/% feed$\cdot$mwt^{-1} for thermoneutral in contrast to .05 kJ/% feed$\cdot$mwt^{-1} for birds exposed to HD. The greater HP response with feeding in broilers exposed to HD suggests that reduced substate availability increases the bird's heat load during HD. Due to the additional heat production during HD, body temperature and heat content were increased ($P<.01$). Water consumption increased ($P<.05$) with feeding level. This may be partially attributed to the force

feeding however, this effect was not observed during the thermoneutral phase of the experiment. Water intake is correlated with feed intake (Zeigler et al. 1971) and is especially significant at high ambient temperatures when water consumption may be elevated 2 ½ times (NRC, 1981).

Within the thermoneutral exposure period, HP and NHL were linearly ($P<.05$) related with feeding level. Fasted birds had a lower ($P<.05$) HP, body temperature and NHL compared to the other treatment groups (Table 2) with the 105%/mwt feeding level being the highest ($P<.05$) in HP and NHL. Evaporative heat loss, respiration rate and respiration efficiency was similar ($P>.1$) for all treatments. These data indicate, when environmental conditions permit, that the preferred heat dissipation route is NHL.

Similar to the thermoneutral environment, HP and NHL were linearly ($P<.05$) related with increasing feeding level during HD (Table 2). It is apparent from the data that birds continued to favor NHL during HD, since fed birds had an increased NHL. This suggests that NHL may be an important factor to manipulate during HD.

Fasted birds continued to have a lower ($P<.05$) HP and EHL compared to fed birds. No differences ($P>.1$) were observed in body temperature or heat content.

In summary, two experiments were conducted to quantify feeding effects on broiler thermobalance in thermoneutral (24°C) and heat distressed (32-35°C) environments.. HP and

NHL were linearly related to feed intake in both phases of the experiment. HP increased for birds fed 35, 70 and 105%/mwt feeding levels. No differences were observed between environments in birds fasted 12 hr prior to HD initiation, suggesting that fasting is beneficial during HD by lowering HP. These results indicate that feeding level and HD effects on HP are additive. When environmental conditions limit nonevaporative heat dissipation feed consumption exacerbates the HD state.

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CHAPTER IV

ACCLIMATION EFFECTS ON FASTED BROILER THERMOBALANCE DURING HIGH AMBIENT TEMPERATURE DISTRESS

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ABSTRACT

One experiment was conducted, to quantify effects of broiler acclimation to heat distress (HD) on bird thermobalance in birds fasted and exposed to subsequent HD exposure. Thermobalance was defined as bird: heat production (HP) = evaporative heat loss (EHL) ± nonevaporative heat loss (NHL) ± change in bird heat content (dHC). Respiration rate (RR) and water consumption were monitored for correlation with thermobalance criteria. On day 1, 6 birds were exposed to a 4 hr HD (32-35°C) acclimation period while 6 controls were housed at 24°C. On day 2 both acclimated birds and unacclimated controls were fasted 12 hr and exposed to a 5 hr HD period. Upon HD exposure, acclimated birds exhibited lower ($P<.01$) HP (22.8 vs 25.6 $kJ/mwt \cdot hr^{-1}$), EHL (7.5 vs 8.8 $kJ/mwt \cdot hr^{-1}$), and RR

(129 vs 160 breaths/minute) compared with unacclimated controls. Though EHL and RR were lower in acclimated birds Joules of heat energy dissipated per breath was 8% higher and the kJ heat dissipated as a percent of HP was 3% higher. The reduced HP and the tendency for greater heat dissipation efficiency is reflected in the reduced (P=.08) core body temperature (41.8 vs 42.4°C) for birds acclimated to HD. (Key Words: broiler, heat distress, thermobalance, acclimation)

Introduction

Broiler acclimation to HD may be defined as physiological adaptations made to maintain homeostasis during high ambient temperature. The process has been studied by Hutchinson and Sykes (1953), Reece et al. (1972), Farrell and Swain (1977), Bohren et al. (1982) and May et al. (1987). Broiler acclimation to HD reduces the mortality loss associated with high ambient temperature distress (Reece et al., 1972; Bohren et al., 1982). Birds acclimated to high temperatures have lower body temperature (Hutchinson and Sykes, 1953; May et al., 1987) than their unacclimated counterparts. The mechanisms involved in the acclimation processes have not been defined though Sykes and Fataftah (1986) indicated acclimated birds have a reduced heat production.

Avian homeostatic processes must function to maintain bird homeostasis during heat distress (HD) or body

temperature rises (May et al., 1987) and growth and survivability declines. As ambient temperatures increase the birds ability to dissipate heat as nonevaporative heat loss declines since the differential between bird body temperature and the environment is reduced. Consequently during high ambient temperature exposure the compensatory responses, such as increased respiration rate (Frankel et al., 1962), adjustments to increase surface area (Baldwin, 1974), vasodilation of extremities (Nolan et al., 1978) and increased water intake (Farrell and Swain, 1977), must be elicited to maintain homeostasis. Such compensatory responses have been reported to increase the bird's heat load.

Before significant progress can be made to eliminate the deleterious consequences of HD, rate limiting factors impacting thermobalance (TB) must be identified. Bird TB may be estimated as: heat production (HP) = evaporative heat loss $\pm$ nonevaporative heat loss (NHL) $\pm$ change in bird heat content (dHC) (Sturkie, 1986). Presumably, minimizing bird HC changes would be associated with optimal bird TB during high ambient temperature exposure.

Nonevaporative heat loss is likely the most energetically efficient means to dissipate heat. Birds manipulate NHL by increasing vasodilation of extremities (Nolan et al., 1978; Bottje et al., 1983) and shunting blood to and from the gastrointestinal tract. However, as discussed the extent of NHL declines as ambient temperature

rises (van Kampen, 1974; Wiernusz, et al., 1991) and the bird must consequently reduce HP or increase other heat dissipating mechanisms to minimize HC change.

EHL through the latent heat of vaporization for H_2O is increased by enhancing respiration rate. Cutaneous moisture loss from birds represents only 15% of total EHL during heat distress (van Kampen, 1974). Indeed EHL may become the major heat dissipation route during heat distress (van Kampen, 1981) as such birds may dissipate over 80% of heat production via this method provided the relative humidity is not excessive (van Kampen, 1974).

The effects of high ambient temperature on HD have been mixed. Van Kampen (1974) reported that bird heat production is inversely related to ambient temperature. In contrast van Kampen (1981) and Chwalibog and Eggum (1989) reported that heat production increases when birds are exposed to HD. Such contradictions may be related to the duration of HD and the bird's feed consumption pattern. Feed consumption has long been known to increase bird heat production (Macleod et al. 1979) and to decline with HD exposure (Squibb et al., 1959). Therefore, studies conducted when feed intake is uncontrolled will be expected to give varying results.

Work conducted by Teeter and Smith (1988) to evaluate relationships between feed intake, water consumption and acclimation during HD, suggest that feed intake and water consumption play significant roles in the acclimation process. Their data further suggest that feed consumption

had the potential to mask the acclimation response. Though broilers normally decrease feed consumption during HD it appears that the birds are reacting to existing stress which does not allow sufficient time for feed to clear the bird's digestive tract and reduce substrate availability (Smith, 1983). Credence is added to this concept as fasting broilers prior to HD initiation has been observed to increase survival ability (McCormick et al., 1979; Teeter at al., 1987). Wiernusz et al. (1991) reported that increasing feeding from 0 to 105% bird metabolic body weight/day increased HP linearly in thermoneutral and HD environments. Therefore, it is evident that feed intake must be controlled to evaluate the acclimation effect independent of feeding level.

The objective of this study was to quantify acclimation effects on bird thermobalance in fasted broilers exposed to high ambient temperature-relative humidity distress.

Materials and Methods

The Cobb x Cobb male broilers used in the following experiment were fed a 23% crude protein corn-soybean based ration and reared in floor pens on rice hull through 21 days posthatching. On the first day of the 4th week 50 birds were transferred to individual 47 x 26 cm wire floored cages housed within an environmentally controlled room. Ambient temperature was maintained at 24±1°C and birds were switched to a 21% crude protein grower ration (Table 1). During week

Table 1 Diet composition of grower ration[1]

Ingredients	Percent
Ground corn	61.12
Soybean meal (48.5% CP)	31.06
Fat	3.80
Dicalcium phosphate (22% Ca; 18.5% P)	1.70
Limestone (38% Ca)	1.30
salt	.40
Vitamin mix[2]	.30
DL-Methionine, 99%	.22
Trace mineral mix[3]	.10
Total	100.00

[1]Calculated analysis:

ME Kcal/Kg	3128.40
Crude protein (%)	20.60
Calcium (%)	1.00
Phosphorus (% av)	.44

[2]Mix contained Vit A, 3,968,280 I.U.; Vit D_3, 1,102,300 I.U; Vit E, 13,228 I.U.; Vit B_{12}, 7.9 mg; Riboflavin, 2,646 mg; Niacin, 17,637 mg; d-Pantothenic Acid, 4,409 mg; Choline, 200,178 mg; Menadione, 728 mg; Folic Acid, 441 mg; Pyridoxine, 1,587 mg; Thiamin, 794 mg; d-Biotin, 44 mg per Kg.

[3]Mix contained Manganese, 12.0%; Zinc, 10.0%; Iron, 7.5%; Copper, 1.0%; Iodine, .25%; Calcium 13.5%.

5 posthatching, 12 birds were selected for similar body weight, aseptically prepared and implanted with a radiotelemetry temperature transmitter (Mini-Mitter telemetry system, Sunriver, Oregon 97707) in the abdominal cavity following the procedure described in the Dataquest III User's Manual of Mini-Mitter telemetry (Data Sciences, Inc., St. Paul, MN 55113, Document no. 10000-05, 1988). Anesthetic induction and maintenance was achieved by a ketamine HCl intramuscular injection (40 mg/kg of body weight) and halothane in oxygen via Bain nonre breathing system, respectively. Following 2 days for surgical recovery birds were placed in 12 individual respiration chambers (51 x 34 x 41 cm) and allowed a 3 day adjustment period to chambers as recommended by Misson (1974).

To equalize feeding effects between acclimated and nonacclimated groups, 2 days prior to the experiment birds were force fed (Teeter et al., 1984) the grower mash at 15% of metabolic body weight 3 x daily and fasted 12 hr prior to experiment initiation. On day 1 birds were divided into two groups, the first group comprised of 6 broilers was exposed to a 4 hr HD as illustrated in Figure 1, with ambient temperature cycling to mimic a summer environment. Immediately following the heating phase room temperature was reduced to 24°C and the second group (unacclimated controls) placed in the remaining 6 individual respiratory chambers. On day 2 both groups were exposed to the same HD exposure of day 1 (Figure 1). Water was supplied for ad libitum

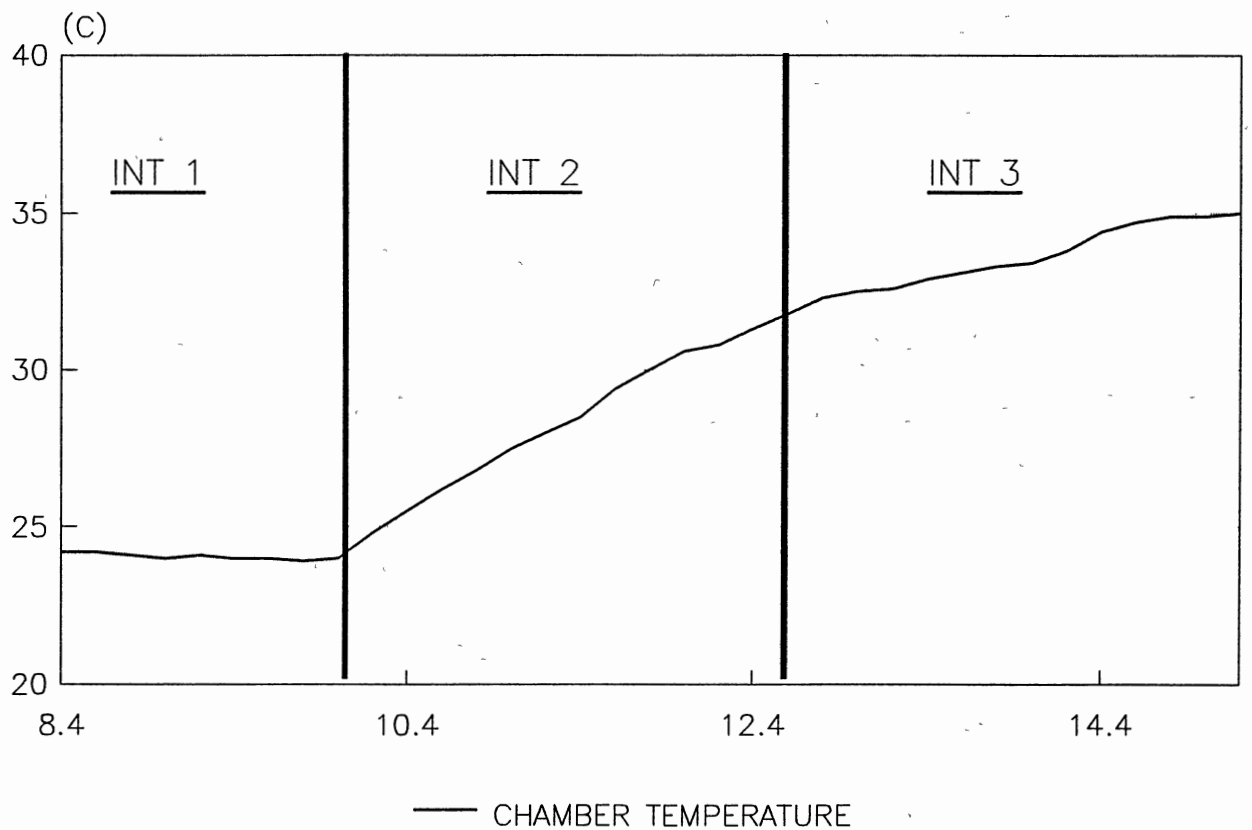

Figure 1. Chamber temperature profile

consumption throughout the experiment and monitored every 8 hr for consumption. Bird response variables monitored on day 2, included HP, EHL, core body temperature, dHC, NHL, respiration rate, respiration efficiency as described below.

Respiratory Chambers

The 12 (51 x 34 x 41 cm) respiratory chambers were constructed of clear 63.5 mm acrylic plexiglas and fitted with a Hart watering cup. Water supplying the Hart cup for each chamber was contained in a 1000 ml graduate cylinder. Water disappearance from the cylinder was computed 3 x daily such that water consumption could be estimated. The chamber floor (51 x 34 cm) was constructed of wire mesh suspended 9 cm above a 51 x 34 cm excreta collection pan containing 4 cm of mineral oil so that voided excreta moisture would be 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, for air mixing ensuring 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 (Gardner-Denver, Quincy, IL. 62305) and dried by a compressed air dryer (Hankison, Cannonsberg, PA.

15317). Incoming air for each chamber moved through independent 64 mm diameter tubing such that the air temperature within each tube reached room temperature prior to entry 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. A pressure regulator released flow at a constant pressure from the air compressor and microvalves were used to direct the desired flow rate through each chamber. All flow rates were monitored by an electronic mass flow meter (Omega Engineering, Stanford, CT 06907). Air flow fluctuated less than ± 1% throughout the experiment.

Relative Humidity, O_2 and CO_2 Analysis

Air moisture, O_2 and CO_2 concentrations of bird breathing air were determined by an Omnidata International (Logan, UT. 84321) relative humidity probe (accuracy ± 1%) and Ametek (Pittsburgh, PA. 15238) O_2 (accuracy ± .2%) and CO_2 (accuracy ± .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 ($kJ/mwt \cdot hour^{-1}$) 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 Lokhorst 1961, 1966).

Evaporative Heat Loss (EHL)

Bird water evaporative heat losses (respiratory and cutaneous) were estimated by coupling water evaporative losses with latent heat of vaporization for water. To estimate water evaporative loss relative humidity (RH) was converted into g H_2O/L^3 gained as the air passed through the respiratory chambers. To accomplish this, air at saturation for various ambient temperatures (Handbook of Chemistry and Physics, 1987) was regressed against ambient temperature (SAS, 1982) and the resulting equation:

$$_{sat}H_2O = 8.694 - .218391 \times T + .03145 \times T^2$$

where

$_{sat}H_2O$ = saturation of water

T = air temperature

The equation was used to estimate water content of air exiting the chamber as follows:

$$H_2O = {}_{sat}H_2O \times (RH/100) \times .001$$

Bird H_2O evaporative losses were then estimated as:

$H_2O_{prod} = (flow_t \times H_2O_t) - (flow_{ref} \times H_2O_{ref}) \times 60$

where

H_2O_{prod} = water production (g/min)

t = test chamber

ref = reference chamber

The total evaporative heat loss (respiratory water and cutaneous) was calculated by multiplying the estimated evaporative water loss by the latent heat of vaporization (Sturkie, 1986).

$EHL = H_2O_{prod} \times 2.365$

where

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

Bird Heat Content (HC)

Body temperature observations were made utilizing the radio telemetry system, described previously, with data recorded once every 1.5 minutes for each bird was used to estimate body heat content change ($Kj \cdot hr^{-1}$) as:

dT (C) X M (Kg) x SH (Sturkie, 1986).

where

dT = body temperature change

M = bird mass

SH = specific heat

The mean specific heat of body tissues was estimated in our laboratory to be 3.17 $kJ/Kg \cdot °C^{-1}$ which differs from 3.5 $kJ/Kg \cdot °C^{-1}$ estimated by Sturkie (1986). The differences may be attributed to age, species and body composition.

Nonevaporative Heat Loss (NHL)

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

Respiration Rate (RR) and Efficiency (RE)

During a respiration cycle (inhale and exhale) chamber pressure declines followed by an increase in pressure. 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) estimated respiration rate 8 times per hour per bird.

Evaporative heat loss is determined by the combined moisture loss from respiratory and cutaneous sources. A small amount of energy is likely utilized to manipulate NHL in contrast to panting, when considerable amount of energy is utilized. Since cutaneous moisture loss from the bird represents only 15% of total EHL during heat distress (van Kampen, 1974), an estimate of respiration efficiency ($J \cdot breath^{-1}$) was obtained by relating EHL with RR as: EHL/breath.

Data Acquisition

The chamber environment and all data measurements were controlled and monitored by a Workhorse Data Acquisition and Control System (Omega Engineering, Stanford, CT 06907). Gas concentration (RH, O_2, CO_2) quantifications, flow rate, respiration rate and ambient temperature were made and recorded once on each of the 12 compartments every 12 minutes.

Statistical Analysis

Variables within the distressed environments were judged for deviation from thermoneutral homeostatic levels to identify potentially rate limiting factors related to bird thermobalance. Response variables, which included: HP, NHL, EHL, dT, RR and RE were regressed against time utilizing SAS (SAS, 1982) such that time dependent polynomial equations could be used to describe the data. Comparisons between environments were made by integrating specific time intervals (day and distress periods within day) to provide quantitative values for the intervals shown in figure 1. All integrated values as well as body temperature and water consumption values were analyzed by analysis of variance using Proc GLM (Steel and Torrie, 1960).

Results and Discussion

Data resulting from the experiment are displayed in table 2. NHL declined as ambient temperature increased, presumably due to the reduced bird-environmental temperature differential, similar to previous reports (Roller and Dale, 1963; Olson et al., 1974). The decline in NHL forces the bird to initiate other heat dissipation mechanisms such as EHL. Evaporative heat loss may be increased by elevating respiratory and/or cutaneous water loss. In this study respiration rate (RR) increased ($P<.01$) from 33 to 160 breaths/min. Such processes increase bird energy expenditure. Indeed, HP increased with RR (Table 2) which is similar to results of van Kampen (1974). Barott and Pringle (1946) suggested that the increase in heat production for birds exposed to heat distress was due to the energetic cost of panting. However, other reports have indicated that broilers exposed to heat distress have a lower heat production (van Kampen, 1981; Chwalibog and Eggum, 1989) which may be related to reduced feed consumption. Macleod et al. (1979) reported that restricted feeding lowered heat production and Wiernusz et al. (1991) indicated that broiler heat production increased linearly with increased feed consumption during both thermoneutral and heat distress exposures. Evaporative heat loss and core body temperature also increased ($P<.05$) at elevated ambient temperatures similar to the results of van Kampen (1974).

Table 2 Effects of ambient temperature and acclimation on fasted broiler heat production, nonevaporative and evaporative heat loss, change in heat content, body temperature, respiration rate and respiration efficiency.

	Nonacclimated			Acclimated		
Ta[1]	24°C	25-30°C	31-35°C	24°C	25-30°C	31-35°C
HP[2] (kJ/mwt·hr^{-1})	23.0^{b}	22.8^{b}	25.6^{a}	19.1^{c}	20.1^{c}	22.8^{b}
NHL[3](kJ/mwt·hr^{-1})	18.6^{a}	16.8^{b}	14.6^{cd}	15.3^{c}	15.1^{c}	13.8^{d}
EHL[4](kJ/mwt·hr^{-1})	4.4^{d}	5.4^{c}	8.8^{a}	3.8^{e}	4.5^{d}	7.5^{b}
dHC[5](kJ/mwt·hr^{-1})	0^{b}	.5^{b}	2.2^{a}	0^{b}	.6^{b}	1.6^{a}
BT[6] (C)	40.8^{bc}	41.3^{b}	42.4^{a}	40.5^{c}	41.0^{bc}	41.8^{ab}
RPM[7]	33^{e}	98^{c}	160^{a}	29^{e}	60^{d}	129^{b}
RESEFF[8](J/breath)	2.2^{a}	.9^{b}	.9^{b}	2.1^{a}	1.3^{b}	1.0^{b}

$^{a-e}$Means within a row differ with unlike superscripts differ (P<.05)
[1]Ta=ambient temperature
[2]HP=heat production
[3]NVC=nonevaporative heat loss
[4]EVC=evaporative heat loss
[5]dHC=change in heat content
[6]BT=body temperature at the end of each period
[7]RPM=respiration per minute
[8]RESEFF=respiration efficiency

Within the thermoneutral temperature birds exhibited a lower ($P<.05$) heat production, nonevaporative and evaporative heat loss compared to the nonacclimated group (Table 2) in agreement with data reported by Sykes and Salih (1986).

As ambient temperature increased, birds previously acclimated to heat distress maintained a lower ($P<.05$) HP. As a result, the amount of heat they were required to dissipate for homeostasis was less and was reflected in lower NHL, EHL, core body temperature, and respiration rate (Table 2) similar to Sykes and Fataftah (1986). Since EHL is expressed per unit metabolic body weight (mwt) the animals surface area should be equalized. If cutaneous losses are small then EHL is principally by respiratory means and the data reflect a respiration efficiency. Sturkie (1986) reported that the amount of water evaporated through the skin is limited, consequently the error associated with this measurement may be low. In this study respiratory efficiency expressed as $EHL \cdot mwt^{-1}$ dissipated per breath was similar ($P>.1$) for intervals 2 and 3, grouping these intervals revealed that acclimated birds had a greater ($P<.05$) respiration efficiency being 6.6 vs. 5.6 J/breath for the nonacclimated birds. Overall water consumption expressed as $ml \cdot mwt^{-1}$ was significantly higher ($P<.01$) for acclimated birds (31 vs 16 $ml \cdot mwt^{-1}$) compared to the nonacclimated group similar to data reported by Lott (1991).

In summary, bird acclimation to HD plays a major role in the ability of broilers to maintain homeostasis during successive HD exposures. This study must be qualified by the restriction that fasted birds were used. The controversy surrounding the HP response of birds to acclimation must clarified. Further studies are needed to determine if indeed HP fluctuates during HD and if it is influenced by feeding to the point that acclimation responses may be masked.

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CHAPTER V

ACCLIMATION EFFECTS ON PRECISION FED BROILER THERMOBALANCE DURING HIGH AMBIENT TEMPERATURE DISTRESS

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ABSTRACT

One experiment was conducted to quantify effects of broiler acclimation to heat distress (HD) on thermobalance in birds precision fed 45% of metabolic body weight (mwt) daily. Days were partitioned into 3 periods and each period was divided into 3 intervals. Two intervals comprised of increasing ambient temperature distress and 2 intervals were used to estimate bird responses when housed at 24°C. Thermobalance was defined as bird: heat production (HP) = evaporative heat loss (EHL) ± nonevaporative heat loss (NHL) ± change in bird heat content (dHC). Respiration rate (RR) and water consumption were monitored for correlation with thermobalance criteria. On day 1, 6 birds were exposed to a 4 hr HD (32-35°C) acclimation period while 6 controls were

housed at 24°C. On day 2 all birds, acclimated and nonacclimated, were exposed to a temperature cycle composed of a 2 hr adjustment period at 24°C and a temperature rise of 1.8°C/hr to 35°C. On day 3 the birds were exposed to HD cycle only. Acclimated birds exposed to HD on day 2 exhibited a higher ($P<.05$) HP (29.1 vs 28.2 $kJ/mwt \cdot hr^{-1}$) and EHL (10.5 vs 9.6 $kJ/mwt \cdot hr^{-1}$) than their nonacclimated counterparts. Heat production and EHL was elevated ($P<.05$) in birds exposed to 3 consecutive HD compared to birds exposed twice. Acclimated birds maintained a higher ($P<.05$) HP throughout the subsequent thermoneutral period. No correlations were observed for RR and water consumption with thermobalance data. This study suggests that bird feeding level markedly influences the HD broilers acclimation response and in fact has the capacity to mask it.
(Key Words: broiler, heat distress, thermobalance, acclimation)

Introduction

Broiler acclimation to HD may be defined as physiological adaptations made to maintain homeostasis during high ambient temperature. The process has been studied by Hutchinson and Sykes (1953), Reece et al. (1972), Farrell and Swain (1977), Bohren et al. (1982) and May et al. (1987). Broiler acclimation to HD reduces the mortality loss associated with high ambient temperature distress (Reece et al., 1972; Bohren et al., 1982). Birds acclimated

to high temperatures have lower body temperature (Hutchinson and Sykes, 1953; May et al., 1987) than their unacclimated counterparts. The mechanisms involved in the acclimation processes have not been defined though Sykes and Fataftah (1986) indicated acclimated birds have a reduced heat production.

Avian homeostatic processes must function to maintain bird homeostasis during heat distress (HD) or body temperature rises (May et al., 1987) and growth and survivability declines. As ambient temperatures increase, the birds ability to dissipate heat as nonevaporative heat loss declines since the differential between bird body temperature and the environment is reduced. Consequently, during high ambient temperature exposure, the compensatory responses, such as increased respiration rate (Frankel et al., 1962), adjustments to increase surface area (Baldwin, 1974), vasodilation of extremities (Nolan et al., 1978) and increased water intake (Farrell and Swain, 1977) must be elicited to maintain homeostasis. Such compensatory responses have been reported to increase the bird's heat load.

Before significant progress can be made to eliminate the deleterious consequences of HD, rate limiting factors impacting thermobalance (TB) must be identified. Bird TB may be estimated as: heat production (HP) = evaporative heat loss $\pm$ nonevaporative heat loss (NHL) $\pm$ change in bird heat content (dHC) (Sturkie, 1986). Presumably, minimizing

bird HC changes would be associated with optimal bird TB during high ambient temperature exposure.

Nonevaporative heat loss is likely the most energetically efficient means to dissipate heat. Birds manipulate NHL by increasing vasodilation of extremities (Nolan et al., 1978; Bottje et al., 1983) and shunting blood to and from the gastrointestinal tract. However, as discussed the extent of NHL declines as ambient temperature rises (van Kampen, 1974; Wiernusz, et al., 1991) and the bird must consequently reduce HP or increase other heat dissipating mechanisms to minimize HC change.

EHL through the latent heat of vaporization for H_2O is increased by enhancing respiration rate. Cutaneous moisture loss from birds represents only 15% of total EHL during heat distress (van Kampen, 1974). Indeed EHL may become the major heat dissipation route during heat distress (van Kampen, 1981) as such birds may dissipate over 80% of heat production via this method provided the relative humidity is not excessive (van Kampen, 1974).

The effects of high ambient temperature on HD have been mixed. Van Kampen (1974) reported that bird heat production is inversely related to ambient temperature. In contrast van Kampen (1981) and Chwalibog and Eggum (1989) reported that heat production increases when birds are exposed to HD. Such contradictions may be related to the duration of HD and the bird's feed consumption pattern. Feed consumption has long been known to increase bird heat production (Macleod et

al., 1979) and to decline with HD exposure (Squibb et al., 1959). Therefore, studies conducted when feed intake is uncontrolled may be expected to give varying results.

Work conducted by Teeter and Smith (1988) to evaluate relationships between feed intake, water consumption and acclimation during HD, suggest that feed intake and water consumption play significant roles in the acclimation process. Their data further suggest that feed consumption had the potential to mask the acclimation response. Though broilers normally decrease feed consumption during HD it appears that the birds are reacting to existing stress which does not allow sufficient time for feed to clear the bird's digestive tract and reduce substrate availability (Smith, 1983). Credence is added to this concept as fasting broilers prior to HD initiation has been observed to increase survival ability (McCormick et al., 1979; Teeter at al., 1987). Wiernusz et al., (1991) reported that increasing feeding from 0 to 105% of bird metabolic body weight/day increased HP linearly in thermoneutral and HD environments. Therefore, it is evident that feed intake must be controlled to evaluate the acclimation effect independent of feeding level.

The objective of this study was to quantify acclimation effects on bird thermobalance in fed broilers exposed to high ambient temperature-relative humidity distress.

Materials and Methods

The Cobb x Cobb male broilers used in the following experiment were fed a 23% crude protein corn-soybean based ration and reared in floor pens on rice hull through 21 days posthatching. On the first day of the 4th week 50 birds were transferred to individual 47 x 26 cm wire floored cages housed within an environmentally controlled room. Ambient temperature was maintained at 24±1°C and birds were switched to a 21% crude protein grower ration (Table 1). During week 5 posthatching, 12 birds were selected for similar body weight, aseptically prepared and implanted with a radiotelemetry temperature transmitter (Mini-Mitter telemetry system, Sunriver, Oregon 97707) in the abdominal cavity following the procedure described in the Dataquest III User's Manual of Mini-Mitter telemetry (Data Sciences, Inc., St. Paul, MN 55113, Document no. 10000-05, 1988). Anesthetic induction and maintenance was achieved by a ketamine HCl intramuscular injection (40 mg/kg of body weight) and halothane in oxygen via Bain nonre breathing system, respectively. Following 2 days for surgical recovery birds were placed in 12 individual respiration chambers (51 x 34 x 41 cm) and allowed a 3 day adjustment period to chambers as recommended by Misson (1974).

To equalize feeding effects between birds, birds were precision fed (Teeter et al., 1984) the grower mash at 45% metabolic body weight (mwt) per day 2 days prior to HD continuing throughout the experimental period.

Table 1 Diet composition of grower ration[1]

Ingredients	Percent
Ground corn	61.12
Soybean meal (48.5% CP)	31.06
Fat	3.80
Dicalcium phosphate (22% Ca; 18.5% P)	1.70
Limestone (38% Ca)	1.30
salt	.40
Vitamin mix[2]	.30
DL-Methionine, 99%	.22
Trace mineral mix[3]	.10
Total	100.00

[1]Calculated analysis:

ME Kcal/Kg	3128.40
Crude protein (%)	20.60
Calcium (%)	1.00
Phosphorus (% av)	.44

[2]Mix contained Vit A, 3,968,280 I.U.; Vit D_3, 1,102,300 I.U; Vit E, 13,228 I.U.; Vit B_{12}, 7.9 mg; Riboflavin, 2,646 mg; Niacin, 17,637 mg; d-Pantothenic Acid, 4,409 mg; Choline, 200,178mg; Menadione, 728mg; Folic Acid, 441; Pyridoxine, 1,587; Thiamin, 794; d-Biotin, 44 mg per Kg.

[3]Mix contained Manganese, 12.0%; Zinc, 10.0%; Iron, 7.5%; Copper, 1.0%; Iodine, .25%; Calcium 13.5%.

On day 1 birds were divided into two groups, the first group comprised of 6 broilers was exposed to a 4 hr HD as illustrated in Figure 1, with ambient temperature cycling to mimic a summer environment. Immediately following the heating period, room temperature was reduced to 24°C and the second group (nonacclimated) placed in the remaining 6 individual respiratory chambers for 15 hours. On day 2 both groups were exposed to the same ambient temperature regime as day 1 (Figure 1). Day 3 commenced after the heating period. Days were partitioned into 3 periods (2 periods of thermoneutral and 1 period HD) and each 7 hr period was divided into 3 intervals. Bird responses were examined during the HD period and the thermoneutral period prior to HD each day.

Water was supplied for ad libitum consumption throughout the experiment and monitored every 8 hr for consumption. Bird response variables monitored included HP, EHL, core body temperature, dHC, NHL, respiration rate, respiration efficiency as described below.

Respiratory Chambers

The 12 (51 x 34 x 41 cm) respiratory chambers were constructed of clear 63.5 mm acrylic plexiglas and fitted with a Hart watering cup. Water supplying the Hart cup for each chamber was contained in a 1000 ml graduate cylinder. Water disappearance from the cylinder was computed 3 x daily such that water consumption could be estimated. The chamber

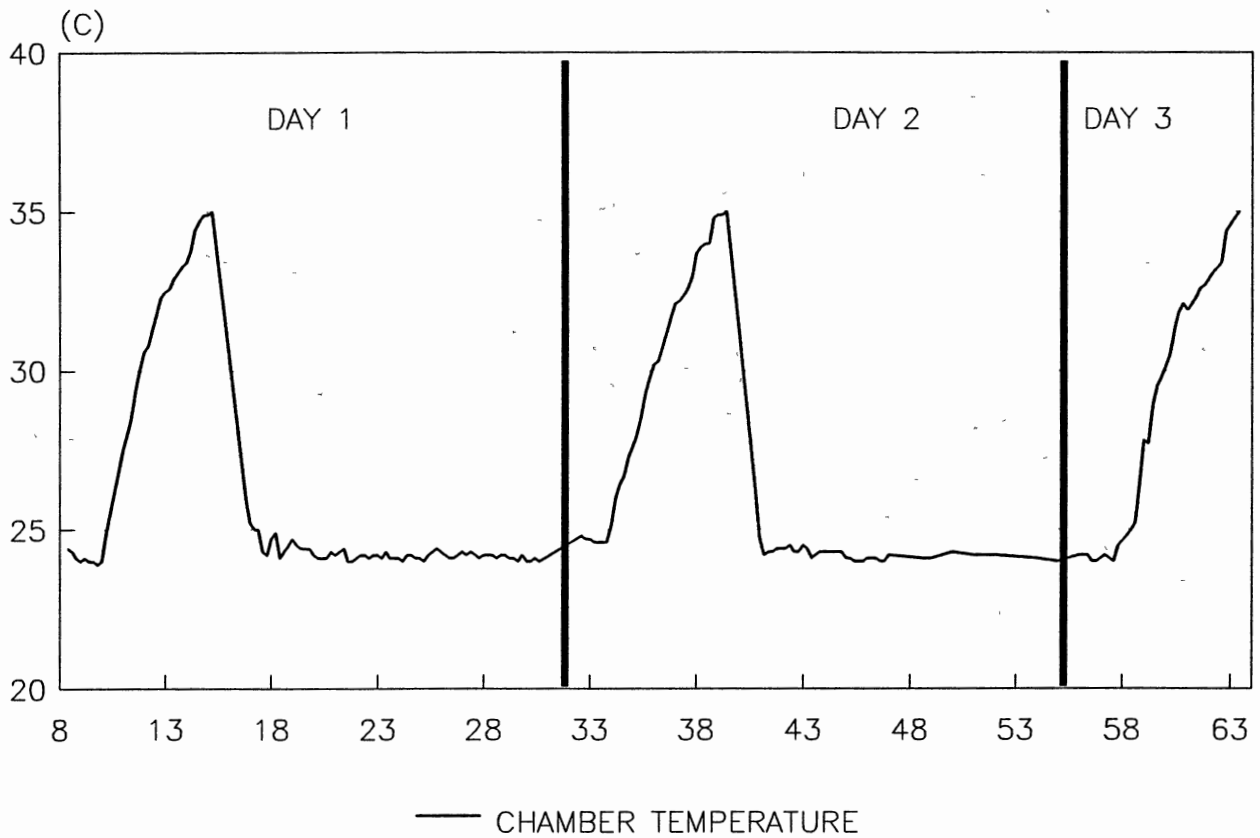

Figure 1. Chamber ambient temperature profile

floor (51 x 34 cm) was constructed of wire mesh suspended 9 cm above a 51 x 34 cm excreta collection pan containing 4 cm of mineral oil so that voided excreta moisture would be 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, for air mixing ensuring 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 (Gardner-Denver, Quincy, IL. 62305) and dried by a compressed air dryer (Hankison, Cannonsberg, PA. 15317). Incoming air for each chamber moved through independent 64 mm diameter tubing such that the air temperature within each tube reached room temperature prior to entry 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. A pressure regulator released flow at a constant pressure from the air compressor and microvalves were used to direct the desired flow rate through each chamber. All flow rates were monitored by an electronic mass flow meter (Omega Engineering, Stanford, CT 06907). Air flow fluctuated less than $\pm$ 1% throughout the experiment.

Relative Humidity, O_2 and CO_2 Analysis

Air moisture, O_2 and CO_2 concentrations of bird breathing air were determined by an Omnidata International (Logan, UT. 84321) relative humidity probe (accuracy ± 1%) and Ametek (Pittsburgh, PA. 15238) O_2 (accuracy ± .2%) and CO_2 (accuracy ± .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/mwt·hour^{-1}) 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 Lokhorst 1961, 1966).

Evaporative Heat Loss (EHL)

Bird water evaporative heat losses (respiratory and cutaneous) were estimated by coupling water evaporative losses with latent heat of vaporization for water. To

estimate water evaporative loss relative humidity (RH) was converted into g H_2O/L^3 gained as the air passed through the respiratory chambers. To accomplish this, air at saturation for various ambient temperatures (Handbook of Chemistry and Physics, 1987) was regressed against ambient temperature (SAS, 1982) and the resulting equation:

$$_{sat}H_2O = 8.694 - .218391 \times T + .03145 \times T^2$$

where

$_{sat}H_2O$ = saturation of water

T = air temperature

The equation was used to estimate water content of air exiting the chamber as follows:

$$H_2O = {}_{sat}H_2O \times (RH/100) \times .001$$

Bird H_2O evaporative losses were then estimated as:

$$H_2O_{prod} = (flow_t \times H_2O_t) - (flow_{ref} \times H_2O_{ref}) \times 60$$

where

H_2O_{prod} = water production (g/min)

t = test chamber

ref = reference chamber

The total evaporative heat loss (respiratory water and cutaneous) was calculated by multiplying the estimated evaporative water loss by the latent heat of vaporization (Sturkie, 1986).

$$EHL = H_2O_{prod} \times 2.365$$

where

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

Bird Heat Content (HC)

Body temperature observations were made utilizing the radiotelemetry system, described previously, with data recorded once every 1.5 minutes for each bird was used to estimate body heat content change ($kJ \cdot hr^{-1}$) as:

dT (C) X M (Kg) x SH (Sturkie, 1986).

where

dT = body temperature change

M = bird mass

SH = specific heat

The mean specific heat of body tissues was estimated in our laboratory to be 3.17 $kJ/Kg \cdot °C^{-1}$ which differs from 3.5 $kJ/Kg \cdot °C^{-1}$ estimated by Sturkie (1986). The differences may be attributed to age, species and body composition.

Nonevaporative Heat Loss (NHL)

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

Respiration Rate (RR) and Efficiency (RE)

During a respiration cycle (inhale and exhale) chamber pressure declines followed by an increase in pressure. 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) estimated respiration rate 8 times per hour per bird.

Evaporative heat loss is determined by the combined moisture loss from respiratory and cutaneous sources. A small amount of energy is likely utilized to manipulate NHL in contrast to panting, when considerable amount of energy is utilized. Since cutaneous moisture loss from the bird represents only 15% of total EHL during heat distress (van Kampen, 1974), an estimate of respiration efficiency ($J \cdot breath^{-1}$) obtained by relating EHL with RR as: EHL/breath.

Data Acquisition

The chamber environment and all data measurements were controlled and monitored by a Workhorse Data Acquisition and Control System (Omega Engineering, Stanford, CT 06907). Gas concentration (RH, O_2, CO_2) quantifications, flow rate, respiration rate and ambient temperature were made and recorded once on each of the 12 compartments every 12 minutes.

Statistical Analysis

Variables within the distressed environments were judged for deviation from thermoneutral homeostatic levels to identify potentially rate limiting factors related to

bird thermobalance. Response variables, which included: HP, NHL, EHL, dT, RR and RE were regressed against time utilizing SAS (SAS, 1982) such that time dependent polynomial equations could be used to describe the data. Comparisons between environments were made by integrating specific time intervals (day and distress periods within day) to provide quantitative values for the intervals shown in figure 1. All integrated values as well as BT and water consumption values were analyzed by analysis of variance using Proc GLM (Steel and Torrie, 1960).

Results and Discussion

Exposing birds to HD on days 1, 2 and 3 yielded classical responses (Table 2). Within a day, averaged over acclimation level NHL declined ($P<.05$) as ambient temperature increased presumably due to the reduced bird-environment temperature differential. This response is similar to other published reports (Roller and Dale, 1963; Olson et al., 1974). The decline in NHL forces the bird to initiate other heat dissipation mechanisms such as EHL. Evaporative heat loss may be increased by elevating respiratory and/or cutaneous water loss. In this study, averaged over environment, RR increased ($P<.01$) from 31 to 96 (Table 3). Such processes were suggested by Barott and Pringle (1946) to increase heat production for HD birds due to the energetic cost of panting. Indeed, HP increased ($P<.05$) with HD exposure (27.1 vs 28.2 $kJ/mwt \cdot hr^{-1}$) similar

Table 2 Acclimation and ambient temperature effects on broiler heat production, nonevaporative and evaporative heat loss and change in heat content

Previous HD exposure	HP[1]		NHL[2]		EHL[3]		dHC[4]	
	TN[5]	HD[6]	TN	HD	TN	HD	TN	HD
0	27.1^{d}	28.2^{c}	22.4^{a}	16.7^{b}	5.8^{d}	9.6^{c}	.1	.4
1	28.5^{c}	29.2^{b}	21.4^{a}	16.3^{b}	6.0^{d}	10.5^{b}	0	.5
2	28.4^{c}	30.3^{a}	22.6^{a}	15.7^{b}	5.7^{d}	11.3^{a}	.1	.6

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

[1]HP=heat production ($kJ/mwt \cdot hr^{-1}$)

[2]NHL=nonevaporative heat loss ($kJ/mwt \cdot hr^{-1}$)

[3]EHL=evaporative heat loss ($kJ/mwt \cdot hr^{-1}$)

[4]dHC=change in heat content ($kJ/mwt \cdot hr^{-1}$)

[5]TN=thermoneutral (24°C)

[6]HD=heat distress (32-35°C)

Table 3 Acclimation and ambient temperature effects on broiler body temperature, respiration rate, respiration efficiency and water consumption

Previous HD exposure	BT[1]		RPM[2]		RESEFF[3]		MWATER[4]	
	TN[5]	HD[6]	TN	HD	TN	HD	TN	HD
0	40.5^{b}	42.2^{a}	32^{b}	92^{a}	3.1^{d}	1.8^{c}	80^{b}	133^{a}
1	40.8^{b}	42.0^{a}	31^{b}	98^{a}	3.2^{d}	1.8^{b}	95^{b}	142^{a}
2	40.7^{b}	42.2^{a}	30^{b}	98^{a}	3.1^{d}	1.9^{a}	92^{b}	128^{a}

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

[1]BT=body temperature (C)

[2]RPM=respiration rate (breath/minute)

[3]RESEFF=respiration efficiency (J/breath)

[4]MWATER=voluntary water consumption + water contained in feed when precision fed ($ml \cdot mwt^{-1}$)

[5]TN=thermoneutral (24°C)

[6]HD=heat distress (32-35°C)

to results reported by van Kampen (1974) (Table 2). In contrast, numerous reports have indicated that broilers exposed to heat distress have a lower heat production (van Kampen, 1981; Chwalibog and Eggum, 1989) which may be related to reduced feed consumption. Macleod et al. (1979) reported that restricted feeding lowered heat production and Wiernusz et al. (1991) indicated that broiler heat production increased linearly with increased feed consumption during both thermoneutral and heat distress exposures. Evaporative heat loss (Table 2) and core body temperature (Table 3) also increased ($P<.05$) at elevated ambient temperatures similar to the results of van Kampen (1974).

Since EHL is expressed per unit metabolic body weight (mwt) the animals surface area should be equalized. If cutaneous losses are small then EHL is principally by respiratory means and the data reflect a respiration efficiency. Sturkie (1986) reported that the amount of water evaporated through the skin is limited, consequently the error associated with this measurement may be low. In this study respiratory efficiency expressed as $EHL \cdot mwt^{-1}$ dissipated per breath was decreased ($P<.05$) during HD from a thermoneutral value of 3.1 to 1.8 J/breath (Table 3). Overall water consumption expressed as $ml \cdot mwt^{-1}$ was significantly higher ($P<.05$) during HD (134 vs 89 $ml \cdot mwt^{-1}$) similar to data reported by Lott (1991).

Statistical analysis for day effects independent of age and acclimation interaction was made possible by the experimental design. A significant difference was detected between days 1 and 2. However, no difference was detected between days 2 and 3. The data for day 1 was therefore dropped from the study with the exception that the HD history was maintained for the bird. Since no difference averaged over bird group was detected for the HD exposure for each group, day 2 vs day 3. Days 2 and 3 were considered similar (P>.1). Therefore, 6 birds were used to judge exposure 1, 12 for exposure 2 and 6 for exposure 3 effects on TB.

Birds exposed to HD for three consecutive days had a higher HP and EHL compared to birds exposed to HD for the first time (Table 2). No differences (P>.1) were observed in NHL, dHC, BT, RPM, respiration efficiency, water consumption (Tables 2 and 3) and no correlations were observed for RR and water consumption with thermobalance data.

In summary, previous reports have shown bird acclimation to HD plays a major role in the ability of broilers to maintain homeostasis during HD (Hutchinson and Sykes, 1953; Reece et al., 1972; Farrell and Swain, 1977; Bohren et al., 1982; May et al., 1987; Wiernusz et al., 1991). However in this study the HD acclimation response was markedly influenced by the bird feeding level and in fact had the capacity to mask it. This suggests that rate

of passage and/or nutrient utilization is decreased during heat distress. In this study, heat distress appeared to cause a carry over effect of nutrient utilization into the next day's heat distress period. This carry over effect may be caused by adding the combination of consumed feed and feed already contained in the gut exacerbating the heat distressed state. Thus, it may be speculated that even though broilers normally decrease feed intake during heat distress, a delay in feed utilization will shift its use to the next day and broilers may not elicit the benefit of the reduced intake.

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CHAPTER VI

NICARBAZIN EFFECTS ON BROILER THERMOBALANCE DURING HIGH AMBIENT TEMPERATURE DISTRESS

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ABSTRACT

Two experiments were conducted to estimate nicarbazin (125 ppm) effects during (HD) on broiler thermobalance. Thermobalance was defined as bird: heat production (HP) = evaporative heat loss (EHL) ± nonevaporative heat loss (NHL) ± change in bird heat content (dHC). In the first study, birds nonacclimated to HD were precision fed at 45% of metabolic body weight (mwt) per day. Water was provided for ad libitum consumption. Upon exposure to a 4 hr HD period of increasing severity (24-35°C), nicarbazin increased HP ($P<.01$) (39.9 and 37.7 kJ/mwt·hr^{-1}) in comparison to controls not fed nicarbazin. EHL and NHL were similar ($P>.1$) at 12.2 vs 11.8 and 24.4 vs 22.4 kJ/mwt·hr^{-1} for birds consuming ration with and without nicarbazin

respectively. Nicarbazin fed birds had a decreased (P<.05) respiration rate (RR) and an increased (P=.08) body temperature (BT). Joules of EHL dissipated per breath was increased (P<.05) with nicarbazin supplementation from 2.3 to 1.8 J/breath for birds not fed nicarbazin. In second study, using birds previously acclimated to HD, no differences (P>.1) in HP, EHL, NHL, RR or BT were noted with nicarbazin supplementation. The data indicate that nicarbazin toxicity in birds not acclimated to HD is related to increased HP. However, no such effects were noted in birds acclimated to HD suggesting that bird acclimation to HD may play a major role in HD induced nicarbazin toxicity.

Key words: broiler, nicarbazin, heat distress, thermobalance, acclimation

INTRODUCTION

Nicarbazin, a commercially available coccidostat, is well documented to elevate mortality during high ambient temperature-relative humidity heat distress (HD) though the mechanism is not defined. Nicarbazin effects on HD induced mortality were well documented by Sammelwitz (1965) who observed mortality to increase dose dependently from just 5 to 500 ppm dietary inclusion levels. Nicarbazin usage during HD has been observed to decrease live weight gain, and feed efficiency when birds are subjected to high ambient temperature (Keshavarz and McDougald, 1981).

Farny (1965) suggested that nicarbazin toxic effects may be due to increase metabolic rate and hence body temperature. Indeed, metabolic rate is one component of bird survivability during HD. However a more complete model, proposed by Sturkie (1986), involves defining bird thermobalance as: heat production (HP) = evaporative heat loss (EHL) ± nonevaporative heat loss (NHL) ± change in bird heat content (dHC) A discussion of these components follows.

As ambient temperature rises heat loss associated with NHL declines markedly (Olson et al., 1974) as the differential between bird body temperature and the environment declines. As NHL falls, while HP is constant or elevated, other heat dissipation mechanisms such as respiration rate and EHL must increase. If these compensatory responses are insufficient to counter reduced NHL dissipation efficiency body temperature will rise (May et al., 1987) and bird survivability compromised. Several authors (Farny, 1965; Beers et al., 1989) have suggested that nicarbazin fed birds have decreased respiration rate (RR) and an increased body temperature during HD. Beers et al. (1989) observed that broilers fed nicarbazin had an elevated heart rate and indicated that it was due to the increased body temperature. Studies are needed to further define nicarbazin effects on bird thermobalance during HD to enhance nicarbazin's usage.

The objectives of this study was to evaluate nicarbazin fed broilers physiological responses to high ambient

temperature-relative humidity distress in acclimated and nonacclimated broilers.

Materials and Methods

Experiment 1

The Cobb x Cobb male broilers used in the following experiment were fed a 23% crude protein corn-soybean based ration and reared in floor pens on rice hull through 21 days posthatching. On the first day of the 4th week 24 birds were transferred to individual 47 x 26 cm wire floored cages housed within an environmentally controlled room. Ambient temperature was maintained at 24 $\pm$ 1°C and treatment groups were allowed to consume a 21% crude protein grower ration (Table 1) containing either 0 or 125 ppm nicarbazin. During week 6 posthatching, 8 birds were selected for similar body weight, aseptically prepared and implanted with a radiotelemetry temperature transmitter (Mini-Mitter telemetry system, Sunriver, Oregon 97707) in the abdominal cavity following the procedure described in the Dataquest III User's Manual of Mini-Mitter telemetry (Data Sciences, Inc., St. Paul, MN 55113, Document no. 10000-05, 1988). Anesthetic induction and maintenance was achieved by a ketamine HCl intramuscular injection (40 mg/kg of body weight) and halothane in oxygen via Bain nonre breathing system, respectively. Following 2 days for surgical recovery, birds were placed in 12 individual respiration

Table 1 Diet composition of grower ration used in experiments 1 and 2[1]

Ingredients	Percent
Ground corn	61.12
Soybean meal (48.5% CP)	31.06
Fat	3.80
Dicalcium phosphate (22% Ca; 18.5% P)	1.70
Limestone (38% Ca)	1.30
salt	.40
Vitamin mix[2]	.30
DL-Methionine, 99%	.22
Trace mineral mix[3]	.10
Total	100.00

[1]Calculated analysis:

ME Kcal/Kg	3128.40
Crude protein (%)	20.60
Calcium (%)	1.00
Phosphorus (% av)	.44

[2]Mix contained Vit A, 3,968,280 I.U.; Vit D_3, 1,102,300 I.U; Vit E, 13,228 I.U.; Vit B_{12}, 7.9 mg; Riboflavin, 2,646 mg; Niacin, 17,637 mg; d-Pantothenic Acid, 4,409 mg; Choline, 200,178 mg; Menadione, 728 mg; Folic Acid, 441 mg; Pyridoxine, 1,587 mg; Thiamin, 794 mg; d-Biotin, 44 mg per Kg.

[3]Mix contained Manganese, 12.0%; Zinc, 10.0%; Iron, 7.5%; Copper, 1.0%; Iodine, .25%; Calcium 13.5%.

chambers (51 x 34 x 41 cm) and allowed a 3 day adjustment period to chambers as recommended by Misson (1974).

Two days prior to experiment initiation, birds were precision fed (Teeter et al., 1984) the grower mash, containing 0 or 125 ppm nicarbazin, at 45% metabolic body weight (mwt) per day to equalize feeding effects between the control and nicarbazin fed groups. Equalized feed consumption was necessary to evaluate nicarbazin responses independent of feed specific dynamic action. Treatment groups were exposed to a 4 hr HD as illustrated in figure 1. Water was supplied for ad libitum consumption throughout the experiment. Bird TB response variables monitored included HP, EHL, core body temperature, dHC, NHL, respiration rate and respiration efficiency as described below.

Respiratory Chambers

The 12 (51 x 34 x 41 cm) respiratory chambers were constructed of clear 63.5 mm acrylic plexiglas and fitted with a Hart watering cup. Water supplying the Hart cup for each chamber was contained in a 1000 ml graduate cylinder. Water disappearance from the cylinder was computed 3 x daily such that water consumption could be estimated. The chamber floor (51 x 34 cm) was constructed of wire mesh suspended 9 cm above a 51 x 34 cm excreta collection pan containing 4 cm of mineral oil so that voided excreta moisture would be isolated from the chamber environment. Each compartment was fitted with a 3 cm fan (Radio Shack cooling fan cat. # 273-

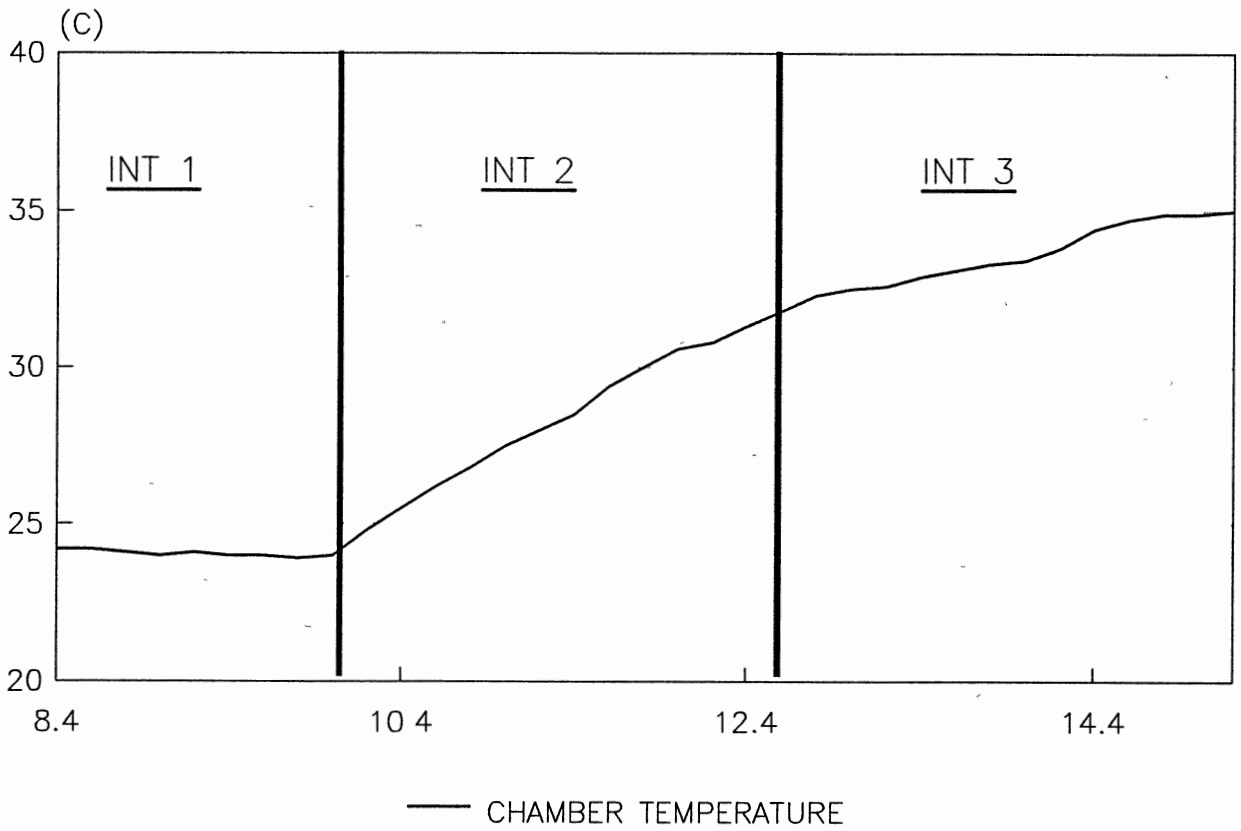

Figure 1. Chamber temperature profile

244), located at the top center of each chamber, for air mixing ensuring 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 (Gardner-Denver, Quincy, IL. 62305) and dried by a compressed air dryer (Hankison, Cannonsberg, PA. 15317). Incoming air for each chamber moved through independent 64 mm diameter tubing such that the air temperature within each tube reached room temperature prior to entry 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. A pressure regulator released flow at a constant pressure from the air compressor and microvalves were used to direct the desired flow rate through each chamber. All flow rates were monitored by an electronic mass flow meter (Omega Engineering, Stanford, CT 06907). Air flow fluctuated less than $\pm$ 1% throughout the experiment.

Relative Humidity, O_2 and CO_2 Analysis

Air moisture, O_2 and CO_2 concentrations of bird breathing air were determined by an Omnidata International (Logan, UT. 84321) relative humidity probe (accuracy $\pm$ 1%)

and Ametek (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/mwt·hour^{-1}) 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 Lokhorst 1961, 1966).

Evaporative Heat Loss (EHL)

Bird water evaporative heat losses (respiratory and cutaneous) were estimated by coupling water evaporative losses with latent heat of vaporization for water. To estimate water evaporative loss relative humidity (RH) was converted into g H_2O/L^3 gained as the air passed through the respiratory chambers. To accomplish this, air at saturation for various ambient temperatures (Handbook of Chemistry and

Physics, 1987) was regressed against ambient temperature (SAS, 1982) and the resulting equation:

$$_{sat}H_2O = 8.694 - .218391 \times T + .03145 \times T^2$$

where

$_{sat}H_2O$ = saturation of water

T = air temperature

The equation was used to estimate water content of air exiting the chamber as follows:

$$H_2O = {}_{sat}H_2O \times (RH/100) \times .001$$

Bird H_2O evaporative losses were then estimated as:

$$H_2O_{prod} = (flow_t \times H_2O_t) - (flow_{ref} \times H_2O_{ref}) \times 60$$

where

H_2O_{prod} = water production (g/min)

t = test chamber

ref = reference chamber

The total evaporative heat loss (respiratory water and cutaneous) was calculated by multiplying the estimated evaporative water loss by the latent heat of vaporization (Sturkie, 1986).

$$EHL = H_2O_{prod} \times 2.365$$

where

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

Bird Heat Content (HC)

Body temperature observations were made utilizing

the radio telemetry system, described previously, with data recorded once every 1.5 minutes for each bird was used to estimate body heat content change ($kJ \cdot hr^{-1}$) as:

dT (C) X M (Kg) x SH (Sturkie, 1986).

where

dT = body temperature change

M = bird mass

SH = specific heat

The mean specific heat of body tissues was estimated in our laboratory to be 3.17 $kJ/Kg \cdot °C^{-1}$ which differs from 3.5 $kJ/Kg \cdot °C^{-1}$ estimated by Sturkie (1986). The differences may be attributed to age, species and body composition.

Nonevaporative Heat Loss (NHL)

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

Respiration Rate (RR) and Efficiency (RE)

During a respiration cycle (inhale and exhale) chamber pressure declines followed by an increase in pressure. 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) estimated respiration rate 8 times per hour per bird.

Evaporative heat loss is determined by the combined moisture loss from respiratory and cutaneous sources. A small amount of energy is likely utilized to manipulate NHL in contrast to panting, when considerable amount of energy is utilized. Since cutaneous moisture loss from the bird represents only 15% of total EHL during heat distress (van Kampen, 1974), an estimate of respiration efficiency ($J \cdot breath^{-1}$) was obtained by relating EHL with RR as: EHL/breath.

Data Acquisition

The chamber environment and all data measurements were controlled and monitored by a Workhorse Data Acquisition and Control System (Omega Engineering, Stanford, CT 06907). Gas concentration (RH, O_2, CO_2) quantifications, flow rate, respiration rate and ambient temperature were made and recorded once on each of the 12 compartments every 12 minutes.

Statistical Analysis

Variables within the distressed environments were judged for deviation from thermoneutral homeostatic levels to identify potentially rate limiting factors related to bird thermobalance. Response variables, which included: HP, NHL, EHL, dT, RR and RE were regressed against time

utilizing SAS (SAS, 1982) such that time dependent polynomial equations could be used to describe the data. Comparisons were made by integrating experiment time intervals to provide quantitative values. All integrated values as well as BT and water consumption values were analyzed by analysis of variance using Proc GLM (Steel and Torrie, 1960).

Experiment 2

The second experiment was conducted to examine HD induced nicarbazin toxicity in birds acclimated to HD. With the exception, of the birds being reared in floor pens on rice hull through 35 days posthatching and being acclimated to Oklahoma summer conditions in which the house temperature reached 33°C, this study was conducted as described above.

Results and Discussion

Experiment 1

Exposing nonacclimated birds to heat distress yielded classical responses. Exposing nonacclimated broilers to heat distress, resulted in an increased ($P<.01$) body temperature and a decreased respiratory efficiency (Table 2). NHL decreased ($P<.05$) as ambient temperature increased presumably due to the reduced bird-environment temperature differential, which is similar to previous reports (Roller and Dale, 1963; Olson et al., 1974) (Table 2). The inefficiency of NHL forces the bird to initiate other heat

Table 2 Effects of ambient temperature and nicarbazin supplementation on nonacclimated broiler heat production, nonevaporative heat loss, evaporative heat loss, change in heat content, body temperature, respiration rate and respiration efficiency.

	Control			Nicarbazin		
Ta[1]	24 C	25-30 C	31-35 C	24 C	25-30 C	31-35 C
HP[2] (kJ/mwt·hr^{-1})	32.0^{d}	35.3^{c}	37.7^{b}	36.8^{bc}	38.5^{ab}	39.9^{a}
NHL[3] (kJ/mwt·hr^{-1})	26.8^{a}	25.1^{ab}	21.4^{b}	29.1^{a}	27.2^{ab}	22.7^{b}
EHL[4] (kJ/mwt·hr^{-1})	6.1^{c}	8.2^{b}	11.8^{a}	6.4^{c}	8.9^{b}	12.2^{a}
dHC[5] (kJ/mwt·hr^{-1})	1.2	1.6	2.4	1.4	2.4	3.0
BT[6] (C)	41.2^{c}	42.0^{b}	43.1^{a}	41.1^{c}	42.3^{b}	43.7^{a}
RPM[7]	24^{d}	55^{c}	108^{a}	30^{d}	62^{c}	87^{b}
RESEFF[8] (J/breath)	4.2^{a}	2.5^{b}	1.8^{b}	3.6^{a}	2.4^{b}	2.3^{b}

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

[1]Ta=ambient temperature

[2]HP=heat production

[3]NHL=nonevaporative heat loss

[4]EHL=evaporative heat loss

[5]dHC=change in heat content

[6]BT=body temperature at the end of each interval

[7]RPM=respiration per minute

[8]RESEFF=respiration efficiency

dissipating mechanisms such as increasing respiration rate and EHL. In this study respiration rate for the control birds increased ($P<.01$) from 24 to 108 breath/min increasing ($P<.01$) EHL by 34% (Table 2).

Heat production increased ($P<.05$) as ambient temperature increased (Table 2) which is similar to results of van Kampen (1974) and Wiernusz et al. (1991). Barott and Pringle (1946) suggested that the increased heat production for birds exposed to heat distress could be attributed to the energetic cost of panting. However, other reports have indicated that HP of broilers exposed to heat distress is reduced (van Kampen, 1981; Chwalibog and Eggum, 1989). Such contradictory studies may be related to decreased feed intake. Macleod et al. (1979) reported that restricted feeding lowered HP. Wiernusz et al. (1991) observed HP increased ($P<.01$) linearly as the force feeding level increased from 0 to 105% mwt. The response was consistent in both thermoneutral and HD environments, being additive with HD.

Within the thermoneutral exposure period, control birds had a lower ($P<.05$) heat production compared to the nicarbazin fed group (Table 2). This response is similar to results reported by Farny (1965) in which nicarbazin fed birds were observed to have a higher metabolic rate at temperatures of 23-24°C.

As ambient temperature increased, the control birds maintained a lower ($P<.05$) heat production similar to Farny

(1965). Farny (1965) indicated that nicarbazin fed birds had a higher skin temperature in unfeathered chest area which was suggested to reflect an increase in NHL. This would be expected with increased HP as NHL is the preferential means of heat dissipation (Wiernusz et al., 1991). In the study reported herein nicarbazin fed birds had higher heat production while NHL and EHL were similar in both groups. Farny (1965) reported that the nicarbazin fed birds had a lower EHL presumably due to the reduced respiration rate (RR). In this study, RR was reduced ($P<.05$) 87 vs 108 breaths/min and core body temperature was higher ($P=.08$) with nicarbazin feeding (Table 2) similar to other reports (Farny, 1965; Beers et al., 1989). Beers et al. (1989) suggested that nicarbazin fed birds either entered into phase II panting sooner or are unable to maintain phase I panting.

Since EHL is expressed per unit metabolic body weight (mwt) the surface area between animals should be equalized. Sturkie (1986) reported that the amount of water evaporated through the skin is limited, if cutaneous losses are small then EHL is principally via respiratory means. As such EHL and RR data may be related to estimate a respiratory efficiency. In this study respiratory efficiency expressed as EHL ($J/mwt \cdot hr^{-1}$)/ breath decreased ($P<.01$) for both groups of birds as the ambient temperature increased due to the increased respiration rate (Table 2). Water consumption expressed as ml/mwt was significantly higher ($P<.01$) for

control birds (100 vs 71 g water/mwt) compared to the nicarbazin fed group. In previous studies in our laboratory Belay et al., (1991) reported that water consumption was positively correlated with respiration efficiency (r=.72). In contrast to this study no correlation (P>.1) was observed.

Experiment 2

In contrast to the first experiment, the second study utilized birds that had previously been exposed to HD and were therefore acclimated. Nonevaporative heat loss decreased (P<.01) while EHL and respiration rate increased (P<.01) as ambient temperature was elevated (Table 3) similar to experiment 1. However, in contrast to experiment 1, heat production did not increase with increased ambient temperature (Table 3) and it appears that birds acclimated to heat distress do not increase heat production. Sykes and Fataftah (1986) reported similar results in laying hens acclimated over a 15 day intermittent heat exposure.

No differences were observed in heat production, NHL, EHL, dHC, RPM, respiration efficiency and core body temperature between the control birds and the nicarbazin fed group (Table 3). This suggests that acclimation to HD plays a role in birds fed nicarbazin such that birds appear to have greater nicarbazin tolerance during high ambient temperature relative humidity distress.

Table 3 Effects of ambient temperature, nicarbazin supplementation and acclimation on broiler heat production, nonevaporative heat loss, evaporative heat loss, change in heat content, body temperature, respiration rate and respiration efficiency.

	Control			Nicarbazin		
Ta[1]	24 C	25-30 C	31-35 C	24 C	25-30 C	31-35 C
HP[2] (kJ/mwt·hr^{-1})	30.1	32.3	30.6	30.3	30.4	29.6
NHL[3] (kJ/mwt·hr^{-1})	23.4^{a}	23.2^{a}	18.7^{b}	23.8^{a}	21.9^{a}	18.4^{b}
EHL[4] (kJ/mwt·hr^{-1})	6.1^{c}	8.1^{b}	10.3^{a}	6.2^{c}	7.4^{b}	9.5^{a}
dHC[5] (kJ/mwt·hr^{-1})	.7^{bc}	1.1^{b}	1.7^{a}	.3^{c}	1.0^{b}	1.7^{a}
BT[6] (C)	41.3^{c}	41.9^{b}	42.7^{a}	41.4^{c}	41.9^{b}	42.7^{a}
RPM[7]	38^{c}	76^{b}	125^{a}	33^{c}	63^{b}	116^{a}
RESEFF[8] (J/RPM)	2.7^{ab}	1.8^{c}	1.4^{c}	3.1^{a}	2.0^{bc}	1.4^{c}

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

[1]Ta=ambient temperature

[2]HP=heat production

[3]NHL=nonevaporative heat loss

[4]EHL=evaporative heat loss

[5]dHC=change in heat content

[6]BT=body temperature at the end of each interval

[7]RPM=respiration per minute

[8]RESEFF=respiration efficiency

In summary, experiments were conducted to determine the effects of nicarbazin in nonacclimated (Experiment 1) and acclimated broilers (Experiment 2) during HD. In experiment 1, nicarbazin fed birds had a lower respiration rate and despite a higher HP, NHL and EHL were similar to the control group suggesting that feeding nicarbazin during HD results increased broiler heat production. Broilers acclimated to heat distress appear to have a greater nicarbazin tolerance.

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CHAPTER VII

MAXIBAN EFFECTS ON HEAT DISTRESSED BROILER GROWTH RATE AND FEED EFFICIENCY

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ABSTRACT

One study was conducted utilizing 192 male broilers, at 4-7 weeks posthatching, to evaluate a narasin/nicarbazin drug combination (Maxiban^{TM}) for effects on bird growth rate, feed efficiency and survivability during cycling ambient temperature (24-35°C) distress. Maxiban did not impact ($P>.1$) live weight gain, but reduced ($P<.05$) gain/feed ratio, (unadjusted for mortality) from .29 for controls to .21 as bird survivability declined ($P<.05$) from 87.5 to 69.8%. Adjusting feed efficiency by adding the gain of birds dying of heat prostration to that for birds surviving the experiment, resulted in feed efficiency for Maxiban supplemented birds being similar ($P=.48$) to birds not consuming Maxiban. Under the conditions of this study, Maxiban increased mortality of male broilers exposed to

cycling ambient temperature of 24-35°C during 4 to 7 weeks posthatching.

(Key Words: Maxiban, nicarbazin, narasin, heat distress, broilers)

INTRODUCTION

In spite of nicarbazins long term usage McDougald *et al*. (1986) reported it to have superior control over 99 field isolates of coccidia when compared to the more recently introduced anticoccidials, monensin, salinomycin and amprolium. However, nicarbazin usage is frequently tempered during heat distress (HD) as it has been observed to decrease live weight gain, feed efficiency and survivability when birds are subjected to high ambient temperature (Keshavarz and McDougald, 1982). Nicarbazin effects on HD induced mortality were well documented by Sammelwitz (1965) who observed mortality to increase dose dependently from just 5 to 500 ppm dietary inclusion levels. The precise mode of action for nicarbazin mortality effects has not yet been defined though Beers *et al*. (1989) observed HD broilers consuming nicarbazin to have elevated body temperature and heart rate. Darre and Harrison (1987) reported that HD broilers exhibit lower heart rate and postulated that it is a means of lowering heat production. Whether nicarbazin toxicity is related to bird heart rate and/or heat production during HD is not known.

Shuttle programs have been advocated as a means of combating decreased anticoccidial drug efficiency with prolonged usage (McDougald *et al.*, 1986). Anthony suggested (ConAgra Poultry Company, El Dorado, AR., personal communication) that nicarbazin may be used effectively in these programs during heat distress to 10 days posthatching, as young chicks are less susceptible to heat distress. Bird acclimation to HD may also play a role as such birds appear to have greater nicarbazin tolerance (Garcia, MSD Agvet, Rahway, NJ., personal communication).

Maxiban, a drug combination consisting of narasin and nicarbazin at 50 ppm each was introduced as a anticoccidial drug in 1989. The narasin/nicarbazin combination has been suggested as a means to reduce HD induced mortality (Long *et al.*, 1988) as male and female chicks subjected to a 42°C, consuming the mixture to 42 days posthatching, had a higher survivability than birds fed nicarbazin alone (125 ppm). Long suggested that the drug combination ameliorated heat distress nicarbazin effects. Male birds are frequently fed to 49 days posthatching or longer today and potentially have greater susceptibility to HD as surface area (for heat dissipation) per unit body weight is reduced. The following study was conducted to evaluate Maxiban effects on growth rate, feed efficiency and survivability of male broilers reared to 49 days posthatching during simulated summer heat distress.

MATERIALS AND METHODS

Vantress x Arbor Acre chicks were raised on rice hull litter and allowed to consume a 23% crude protein, nicarbazin free corn-soybean meal based ration through 21 days posthatching. During the first day of the 4th week 192 birds were randomly allotted to wire floored grower battery compartments (32 x 30.5 x 38.1 cm) housed within a thermostatically (24± 1°C) and humidistatically (55± 5%) controlled environmental chamber. Lighting, within the environmental chamber, was continuous and of tungsten filament origin. Treatments consisted of 16 replicates of 6 birds each arranged in blocks such that chamber position effects could be included in the analysis of variance. All birds were allowed to consume a corn-soy grower ration (Smith and Teeter, 1987) containing either 0 or 50 ppm narasin plus 50 ppm nicarbazin (Maxiban) and water *ad libitum* throughout the 21 day trial. Maximum daily ambient temperature increased by 3.3° C per day, during the first 3 days of the study, such that ambient temperature cycled between 24 and 35° C for the remaining 18 days. The temperature cycle provided 6 hours daily in excess of 32° C and 12 hours of constant 24°C with the remaining 6 hours increasing or decreasing at 1.3°C/hour to complete the cycle. Relative humidity was maintained at 55 ± 5%. Live weight gain, feed consumption and number of birds completing the study were tallied upon completion of the study. Feed efficiency unadjusted for mortality was determined as total

bird live weight gain/total feed consumed for each compartment. Feed efficiency adjusted for mortality was estimated by adding the weight gain of birds dying of heat prostration to the weight gain of the birds surviving the experiment for each replicate.

An analysis of variance was performed using the general linear model procedure of the Statistical Analysis System (SAS, 1982). Duncan's multiple range test was used to separate treatment means.

RESULTS AND DISCUSSION

Supplementing the ration of HD broilers with Maxiban had no significant impact ($P>.1$) upon weight gain. However, Maxiban supplementation reduced ($P<.01$) bird survivability from a mean of 87.5 to just 69.8%. Maxiban lowered ($P<.05$) feed efficiency, unadjusted for mortality, from a mean of .29 to just .21. Adjusting for mortality elevated feed efficiency of both treatments, but markedly so for Maxiban such that control and Maxiban treated bird feed efficiencies were similar ($P=.48$). Under the conditions of this study, Maxiban increased the mortality of male broilers exposed to a cycling ambient temperature of 24-35°C during 4 to 7 weeks posthatching. These data are consistent with previous studies demonstrating nicarbazin toxicity when broilers are exposed to heat distress (Sammelwitz, 1965 Keshavarz and McDougald, 1982; Beers *et al.*, 1989).

TABLE 1 Effects of 50 ppm narasin and 50 ppm nicarbazin on weight gain, feed efficiency and survivability

Treatment	Weight gain (g)	Gain feed	Adj. Feed[1] efficiency	Survivability (%)
Control	900	.29*	.33	87.5**
Narasin/nicarb	870	.21	.30	69.8
Pooled standard error	22.72	.02	.01	.04

Means within a column differ *, P<.05; **, P<.01.

[1]estimated as (weight of birds surviving the experiment + weight of those dying of heat prostration) / feed consumed.

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CHAPTER VIII

SUMMARY AND CONCLUSIONS

The interaction between high ambient temperature-relative humidity distress and reduced broiler performance has long been recognized. The problem of reduced growth rate, feed efficiency and survival is compromised due to the combination of environmental heat and metabolic heat production. Though the specific routes of heat production and dissipation are well defined qualitatively little research has been conducted to quantitatively estimate the importance of each. Indeed, a comprehensive model has not been developed enabling their study much less their potential manipulation into therapeutic measures. Once a model is available, methods manipulating the various processes may be conducted.

In this study, research was conducted to (1) develop a model enabling the quantification of thermobalance in the heat distressed broiler (2) establish the effects of feed intake on broiler thermobalance during thermoneutral and high ambient temperatures (3) evaluate the potential benefit in broilers acclimated to heat distress and the feeding consequences that may be involved (4) evaluate the effects

of the commonly used anticoccidial drug, nicarbazin during broiler heat prostration and (5) evaluate Maxiban, a new anticoccidial drug combination consisting of nicarbazin and narasin, effects on broiler performance.

In each study conducted, exposing birds to heat distress yielded classical responses. Nonevaporative heat loss declined as ambient temperature increased due to the reduced bird-environment temperature differential. The decline in nonevaporative heat loss forces the bird to initiate other heat dissipation mechanisms such as an increase in respiration rate. In these studies, respiration rate and heat production increased suggesting heat distressed broilers have an increased metabolic rate. Evaporative heat loss and core body temperature also increased at elevated ambient temperatures.

In the first study, one experiment was conducted to establish the effects of feed intake on broiler thermobalance during thermoneutral and heat distressed environments. Heat production and nonevaporative heat loss were positively correlated with feed intake in both environments. Thermobalance was improved in broilers fasted prior to heat distress, by reducing heat production. Results from this study indicated that feeding level and high ambient temperature effects on heat production are additive and when environmental conditions limit nonevaporative heat dissipation, and that feed consumption exacerbates the broilers heat distressed state. The

nutritionist should consider fasting as a viable tool in improving broiler performance during high ambient temperatures.

The second study was conducted to evaluate the potential benefit of broilers acclimated to heat distress. Acclimated birds maintained a lower heat production throughout the high ambient temperature distress period compared to unacclimated controls. As a result, the amount of heat the acclimated broilers needed to dissipate was reduced and reflected in a lower nonevaporative heat loss, evaporative heat loss, core body temperature, and respiration rate. In this study, respiratory efficiency expressed as evaporative heat loss per breath revealed that acclimated birds had a greater respiration efficiency which was correlated with their increased water consumption. The results of this study indicate bird acclimation to heat distress plays a major role in the ability of broilers to maintain homeostasis during heat distress exposures. However, this study must be qualified by the restriction that fasted birds were used. The controversy surrounding the heat production response of birds to heat distress acclimation must clarified. If indeed heat production fluctuates during heat distress and is influenced by feeding to the point that acclimation responses do not lower heat production then perhaps the quantity of feed consumed has the potential to negate or cover up the acclimation

response. Such a possibility was suggested in the third study.

The data from experiment three suggests that bird feeding level markedly influences the heat distressed broiler's acclimation response. Birds previously subjected to heat distress on concurrent days and fed at levels similar to those consumed during heat distress had an elevated heat production during heat distress compared with birds not subjected to heat distress on the previous day. This suggests that rate of passage and/or nutrient utilization is decreased during heat distress. In this study, heat distress appeared to cause a carry over effect of nutrient utilization into the next day's heat distress period. This carry over effect may be caused by adding the combination of consumed feed and feed already contained in the gut exacerbating the heat distressed state. Thus, it may be speculated that even though broilers normally decrease feed intake during heat distress, a delay in feed utilization will shift its use to the next day and broilers may not elicit the benefit of the reduced intake.

The two experiments in the fourth study were conducted to evaluate nicarbazin effects on the physiological responses to high ambient temperature-relative humidity distress in acclimated and nonacclimated broilers. In the first experiment, nonacclimated birds fed nicarbazin had a lower respiration rate and despite a higher heat production nonevaporative and evaporative heat loss were similar with

and without nicarbazin consumption. These results indicate that feeding nicarbazin during heat distress reduces bird performance by increasing heat production. Nicarbazin toxicity during heat distress was not evident in acclimated broilers. This suggests that acclimation appears to benefit birds consuming nicarbazin during heat distress by increasing their nicarbazin tolerance.

The final study was conducted to evaluate Maxiban, a anticoccidial drug combination consisting of nicarbazin and narasin, effects on growth rate, feed efficiency and survivability of male broilers reared to 49 days posthatching during simulated summer heat distress. Supplementing the ration of heat distressed broilers with Maxiban had no impact upon body weight gain. However, Maxiban supplementation reduced bird survivability and lowered feed efficiency when unadjusted for mortality. Adjusting for mortality elevated feed efficiency of both control and Maxiban fed broilers, but markedly so for Maxiban such that control and Maxiban treated bird feed efficiencies were similar. This study indicated that the Maxiban, nicarbazin/narasin combination, does not eliminate the heat distressed induced toxicity encountered with nicarbazin use in commercial male broilers reared to 49 days posthatching.

All animal species, domesticated and wild, live within

environments containing multiple factors which cause distress. The list of distress factors is infinite and it is not possible to designate any one factor as the most critical, as the total environment is a combination of all and the animals existence is dependent upon its success in adapting to the continually changing patterns of stressors. The study of environmental stressors must, by necessity, occur on an individual basis, so their effects may be independently known and then examined in combination. It is this latter area that offers the greatest potential to solve problems associated with modern poultry and livestock production.

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