HIGH ENVIRONMENTAL TEMPERATURE STRESS INDUCED ACID-BASE DISTURBANCE AND THE POTASSIUM REQUIREMENT OF BROILER CHICKENS

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PREFACE

At the present time, when the populations of the hotter tropical areas of the world are experiencing widespread hunger, anything that can be done to increase production of food items, is of value. I hope the work reported here will, in some small way, aid in the alleviation of the economic strain imposed on producers and consumers by environmental factors beyond their control. This work is not an end to itself, but rather creates more questions than it answers. However, I am pleased that it may set the stage for further investigations, and may in time, lead to answers which could prove to be valuable tools in the fight against hunger.

I wish to express my sincere gratitude and appreciation to all the people who have assisted me during the course of this study. My appreciation is extended to the members of my advisory committee, Drs. R. G. Teeter, chairman and thesis adviser, C. V. Maxwell, R. D. Geisert, J. W. Oltjen and S. Sangiah, for my extensive use of their time and advice. Recognition is extended to the members of the Poultry Research Farm crew for care of the experimental animals and the help they afforded me during my research trials. I am further grateful to have had the opportunity to interact with other graduate students, faculty, laboratory personnel and friends who have influenced, and contributed significantly to my professional and social development. These individuals include Dr. Bob Teeter, who

encouraged imaginative thought and reasoning as an adviser and friend. Dr. Joe Whiteman who aided considerably in my initial development as a scientist and who, along with Dr. Charlie Maxwell, was instrumental in my decision to attend graduate school.

Special thanks are due to my mother, brothers, and sister, who have provided moral support, even though time and distance separated us. I am indebted to my sons, Michael, Leighton, and Carey, for the many sacrifices made.

My greatest appreciation however, goes to my wife, Delores, for her endless patience, love, understanding and innumerable sacrifices during the course of my graduate program.

Finally, this dissertation is dedicated to the memory of my father Felix Smith and my mother-in-law Ruby Rhoden. Their lives touched mine deeply. I know they would have been pleased that their counselling was not in vain.

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CHAPTER I

INTRODUCTION

High environmental temperature-relative humidity stress constitutes a severe limitation for broiler producers. Heat stress has been shown to dramatically reduce the growth rate and feed intake of broiler chicks (Adams et al., 1962; Smith, 1983) as well as creating a substantial increase in mortality (Kubena et al., 1972; Reece et al., 1972). Nutritionally, attempts have been made to alleviate this problem via ration formulation to reduce dietary heat increment by increasing fat supplementation (Dale and Fuller, 1979), and improving the amino acid balance by lowering dietary protein level (Waldroup et al., 1976). The ability of the heat stressed chick to increase growth as a result of being force fed amounts equivalent to that consumed in a thermoneutral environment, was documented by Smith (1983) which is consistent with the hypothesis of Squibb (1959) that the decline in growth rate is at least in part, a direct result of feed intake. New technology is needed to enable maximum growth rate of heat stressed broilers to occur.

At high ambient temperatures, evaporative cooling through panting is an important avenue for heat loss (Curtis, 1981). As the temperature rises, the bird becomes more and more dependent upon dissipating its heat load by evaporation of water from the lungs (Freeman, 1966). If the humidity also rises under conditions of extremely high environmental temperature, then the fact that the air is highly saturated with water

causes a reduction in the ability of the lungs to promote evaporative cooling, thus the bird becomes subjected to severe physiological stress. However, hyperthermic panting under such conditions may precipitate respiratory alkalosis (Jukes, 1971; Kohne and Jones, 1975). Previous work conducted in our laboratory correlates blood alkalosis with reduced feed consumption, growth rate and increased mortality. Correcting the alkalotic state by the addition of hydrogen ion donors to feed or water enable birds to better cope with both chronic and acute heat stress, increasing feed consumption and growth rate, and decreasing mortality. During alkalosis in man, the hydrogen ion concentration within the renal tubular cells declines, (Harper et. al., 1977), reducing the competition between hydrogen and potassium ions for transport into the distal convoluted tubule and collecting duct thereby increasing urinary potassium excretion. Alkalosis tends to decrease the plasma potassium concentration in humans (Burnell et al., 1956), while urinary potassium concentration has increased markedly in dogs during blood alkalosis (Simmons and Avedon, 1959). If this mechanism is operative in poultry then potassium may explain the importance of acid-base balance in the heat stressed broiler.

The physiological role of potassium in the regulation of osmotic pressure, acid-base balance and tissue integrity has been well documented (Best and Taylor, 1961). The enzyme complex system of the Krebs cycle is activated by potassium ions (Holley and Carlson, 1955). Potassium is important in enhancing the synthesis of glycogen (Hue et. al., 1975) by promoting a faster inactivation of the enzyme phosphorylase a and activation of glycogen synthetase. Pyruvate kinase which functions in the transfer of a high energy phosphate bond from

phosphoenolpyruvate to ADP to form ATP during the glycolytic process also requires potassium for maximal activity (Harper et. al., 1977). In addition, the rate of amino acid utilization in protein anabolism is influenced by adequacy of cellular stores of potassium since high intracellular potassium concentration is necessary for protein biosynthesis by the ribosomes. Potassium is intricately related to muscle metabolism and function. It is involved in energy production by the muscle cells, is essential to the mechanism of contraction and affects muscle cell irritability. Potassium deficiency can lead directly to interferance with muscle cell nutrition and hence muscle weakness which may vary from mild to severe paralysis (Kochnel, 1984). Other clinical problem associated with hypokalemia are increased production of ammonia by the kidneys, decreased protein synthesis, negative nitrogen balance, decreased gastric acid production and growth retardation. Hypokalemia may be caused by redistribution of potassium into the cell due to factors that increase cellular uptake in addition to total body depletion due to renal, gastrointestinal or sweat losses (Brown, 1984). In humans with little sweat loss, of the total amount of potassium excreted, approximately 10% is excreted in the stool and 90% in the urine. Therefore the body balance of potassium generally depend on renal regulation of potassium excretion. Observations made in our laboratory indicate that the thermostressed alkalotic, bird in addition to poor growth, exhibits signs of muscular weakness and paralysis. Frequently, at the time of death, birds will show signs of extreme muscle tetany. This tends to lend credence to the hypothesis that the alkalotic bird is indeed suffering from hypokalemia.

If respiratory alkalosis precipitates urinary potassium losses in poultry, heat stress induced potassium deficiencies could result. Supplementing poultry rations and/or drinking water with potassium and/or hydrogen ion donors to replace the lost potassium or reverse the alkalotic condition, could increase growth rate and reduce mortality of heat stressed broilers. However birds exposed to high environmental temperatures reduce feed intake thereby making efficacy of feed administered potassium and/or hydrogen ions doubtful. Conversely, during heat stress, birds tend to consume large amounts of water making this avenue of supplementation preferred.

Early studies demonstrated the necessity for including potassium in the diet of chicks. Ben Dor (1941) concluded that at least 0.17% was needed to achieve maximum growth while Gillis (1948) stated that the requirement was 0.20 to 0.24% of the diet. Burns et al., (1953) reported that the requirement was between 0.23 and 0.40% of the diet depending on the growth rate of the chick. Leach et al., (1958) concluded that the potassium requirement of the chick increased if a high-protein, high-energy diet was fed. The National Research Council (1984) recommends that potassium be included in the diet of broiler chicks at 0.30% to 0.40%. If the hyperventilating bird excretes large amounts of potassium however, this recommended level may not be enough to meet the body's demand. The major portion of potassium in the diet is readily absorbed through the intestinal wall within about thirty minutes and under normal conditions is totally excreted within approximately four hours (Holley and Carlson, 1955). Under heat stress conditions, a faster rate of elimination could be expected. If the route of administration is via the drinking water, then compared with

administration in the diet, the requirement could concievably be different.

Under practical growing conditions, high environmental temperature stress is rarely constant. The normal pattern is for environmental temperature to be lowest in the early morning and to gradually climb until it peaks in the midafternoon. The body's requirement for potassium could be conceivably higher during the heat of the day when alkalosis from hyperventilation is more severe than during the cool mornings. If potassium is provided in increased amounts during the time when thermoneutral conditions prevail, hyperkalemia could possibly result, therefore time of potassium supplementation could be critical.

The study reported herein was conducted to assess the effects of administration of hydrogen ion and potassium ion donors on performance of heat stressed broiler chickens and to establish the potassium requirements of broiler chickens subjected to high environmental temperature stress. Specific objectives were to: (1) determine if a potassium X hydrogen ion interaction exists in the chronic heat stressed broiler, (2) establish the potassium requirement of 5 to 8 week-old broiler chicks subjected to chronic heat stress, (3) evaluate the effects of therapeutic levels of ammonium chloride and potassium chloride administered continuously or only during heat stress of a diurnal cycling temperature, and (4) evaluate the efficacy of therapeutic levels of ammonium chloride as a hydrogen ion donor and potassium chloride as a potassium donor administered in feed and water in enhancing survival of acutely heat-stressed broiler chicks.

Chapters are prepared as manuscripts in the style required by specific journals to facilitate publication of experimental results.

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CHAPTER II

REVIEW OF LITERATURE

Introduction

Current management practices in meat production are increasingly oriented towards the maximization of production and minimization of production costs through increased growth rate and efficiency of growth. Divergence from ideal climatic conditions that are ideal for growth frequently increase production costs. Heat stress has been shown to dramatically reduce broiler growth rate and feed efficiency (Adams et al., 1962; Smith, 1983) as well as create substantial increases in mortality (Kubena et al., 1972; Reece et al., 1972). Nutritionally, attempts have been made to alleviate this problem via ration formulation to reduce dietary heat increment through fat supplementation (Dale and Fuller, 1979), and improving amino acid balance (Waldroup et al.,1972). Nutrient consumption appears to limit growth rate as first proposed by Squibb et al. (1959) since the force feeding of amounts equivalent to that consumed in a thermoneutral environment increases carcass gain. (Smith, 1983). Heat stress induced growth rate reduction is, at least in part, the direct result of reduced feed intake.

At high ambient temperatures, evaporative cooling through panting is an important avenue for heat loss in the avian species (Curtis, 1981). As the temperature rises, the bird becomes increasingly dependent upon body heat dissipation through panting mediated water

evaporation from the lungs (Freeman, 1966). If the broiler is confronted with high relative humidity, a reduction in panting mediated evaporative cooling efficiency occurs. The likelihood of heat prostration increases dramatically when broilers are exposed to environmental conditions involving both high ambient temperature and relative humidity. Panting under such hyperthermic conditions may precipitate respiratory alkalosis (Kohne and Jones, 1975). Blood alkalosis in chickens have been correlated with reduced feed consumption, reduced growth rate (Teeter et al. 1985), and increased mortality (Smith and Teeter, 1985). Alkalosis tend to decrease the plasma potassium concentration in humans (Burnell et al., 1956), while urinary potassium concentration increased markedly in dogs (Simmons and Avedon, 1959). Huston (1978) observed lower blood potassium concentration in female chickens ranging in age from eight to eighty weeks when reared under heat stress conditions. Deetz and Ringrose (1976) observed a significant decline in plasma potassium level of laying hens exposed to heat stress. Potassium losses during heat stress could therefore partially explain the deleterious effects of acid-base imbalance.

The Role of the Respiratory System in Acid-Base Balance

Respiration, in the context of this discussion, refers to the interchange of gaseous oxygen and carbon dioxide between the body and its environment. Carbon dioxide is removed from the lungs at a rate proportional to alveolar ventilation (Robinson, 1975). Under normal resting conditions, the partial pressure of carbon dioxide (pCO_2) in the

blood is 40 mm Hg. Carbon dioxide in red blood cells react with water to form carbonic acid in the presence of the enzyme carbonic anhydrase (West, 1985). Carbonic acid rapidly and spontaneously dissociates into hydrogen and bicarbonate ions.

$$CO_2 + H_2O \rightleftharpoons H_2CO_3 \rightleftharpoons HCO_3^- + H^+$$

Normal blood equilibrium involves bicarbonate diffusion out of the erythrocyte into the plasma in exchange for chloride and hydrogen binding with hemoglobin. If the plasma bicarbonate concentration is normal, a respiratory control which keeps the partial pressure of carbon dioxide close to 40 mm Hg fixes the pH of the plasma at the same time. The kidneys and the lungs function together to regulate the blood pH while the buffer systems of the body provide immediate defense against rapid pH changes. During respiratory failure, the retention of carbon dioxide results in a lowering of plasma pH. The kidneys partially compensate for this disturbance by retaining bicarbonate. Conversely, in the event of hyperventilation, the resultant rise in plasma pH is partially alleviated by the excretion of excess bicarbonate through the kidney. The bicarbonate buffer system involves the weak carbonic acid which readily dissociates into bicarbonate plus a hydrogen ion.

The buffering power of any buffer system is greatest at a pH equal to its pKa. The Henderson-Hasselbalch equation gives the relationship :

$$pH = pKa + \log [Salt]/[Acid]$$

In body fluids, carbonic acid is in equilibrium with dissolved carbon dioxide and water. The Henderson-Hasselbalch equation for this reaction may therefore be written as follows:

$$pH = pKa + \log [HCO_3^-]/[CO_2]$$

This equation indicates that a change in any one of the components, bicarbonate or carbon dioxide, will result in altered pH. Respiratory regulation of pH is governed by the lungs regulating the partial pressure of carbon dioxide in the blood. Reactions to form bicarbonate will be reversed if the broiler chicken initiates panting to enhance evaporative cooling as carbon dioxide will also be eliminated forcing equilibrium in the reverse direction. Carbon dioxide is physiologically unique because of its high endogenous production and because its concentration is partially regulated by respiratory action (Astrup et al., 1960). The carbon dioxide tension can be increased by either increased production from peripheral tissues or decreased removal by ventilation. This will lead to a lower pH and acidosis. Carbon dioxide tension can be reduced by increasing the rate of alveolar ventilation i.e hyperventilation. This causes pH to increase and lead to respiratory alkalosis (Harper, 1979).

Classification of Respiratory Acid-Base Disturbance

Respiratory Acidosis. This is an increase in the partial pressure of carbon dioxide relative to bicarbonate which results in a lowering of blood pH. Respiratory acidosis may occur in any condition which favors hypoventilation. If the pCO_2 remains elevated, the body attempts to restore the pH to normal, using compensatory action by the kidneys to generate bicarbonate.

In Figure 1, a rapid increase in the normal value of pCO_2 (40 mm Hg at point A) to 60 mm Hg (point B) such as could ocur in hypoventilation,

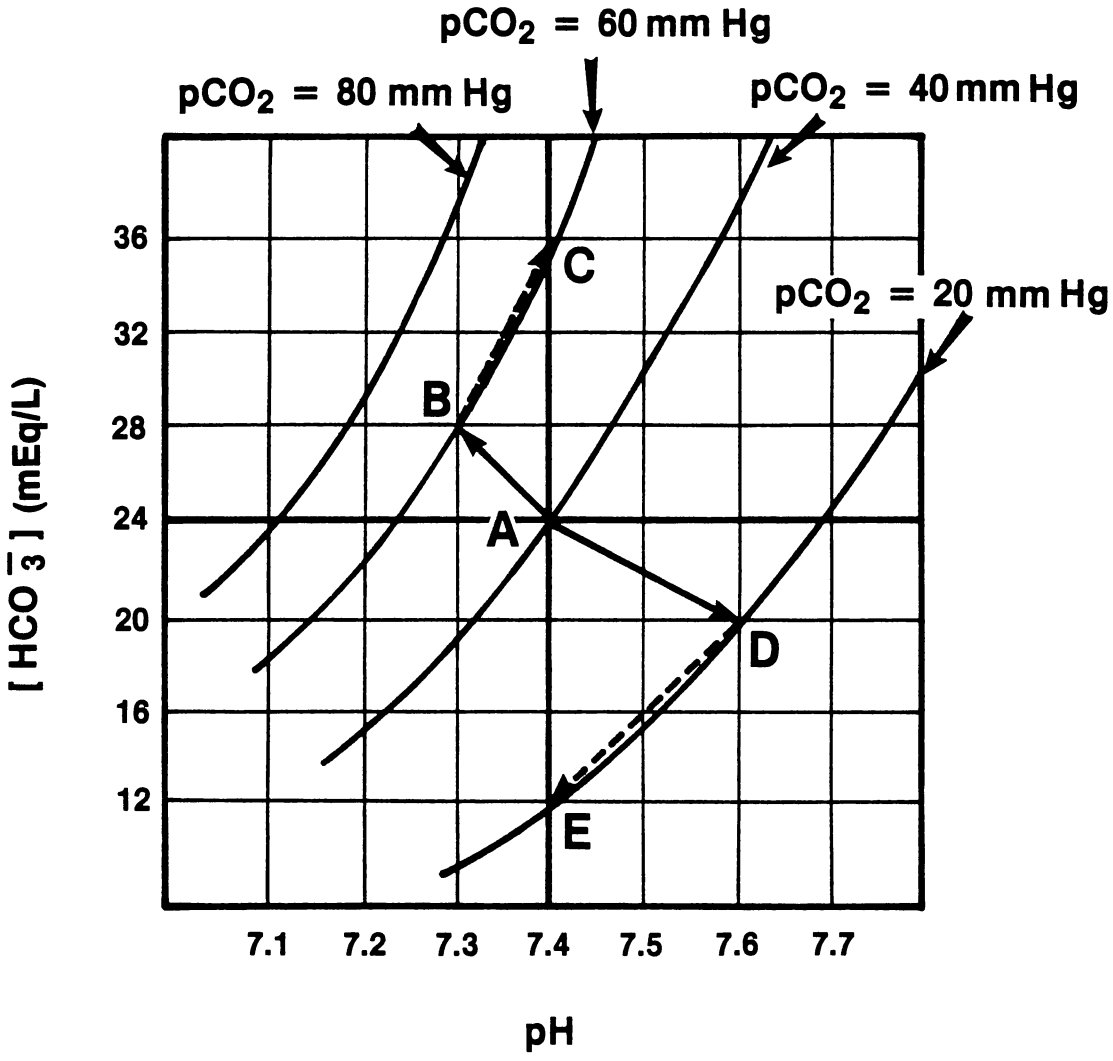

Figure 1. Relationships between HCO_3^-, pCO_2 and pH of blood under normal conditions (A), uncompensated respiratory acidosis (B), compensated respiratory acidosis (C), uncompensated respiratory alkalosis (D), and compensated respiratory alkalosis (E).

results in changes in pH and [HCO_3^-]. With the rise in pCO_2, in order for the Henderson-Hasselbalch equation to be satisfied, HCO_3^- increases to 28 mEq/L and pH falls to 7.3 from the normal 7.4. As hypoventilation persists, the kidneys compensate for the acidosis by retaining some bicarbonate, thereby increasing the plasma level to 36 mEq/L and thus permitting blood pH to return to normal (point C).

Respiratory Alkalosis. In respiratory alkalosis, a decrease in the partial pressure of carbon dioxide is the primary abnormality. This usually occurs without a significant change in the bicarbonate fraction and as a result the HCO_3^- ratio increases and pH is elevated. Hyperventilation is the usual cause of respiratory alkalosis. A compensatory response is initiated by the kidneys in that they fail to reabsorb all the filtered bicarbonate and also fail to generate new bicarbonate ions thus keeping the plasma bicarbonate concentration low.

In figure 1, if hyperventilation causes a fall in pCO_2 to 20 mm Hg, then pH becomes elevated to 7.6 while [HCO_3^-] falls to 20 mEq/L in order that the Henderson-Hasselbalch equation be satisfied (point D). The kidneys compensatory response in reabsorbing the filtered bicarbonate, failing to generate new bicarbonate ions and excreting the excess, results in a further lowering of the plasma bicarbonate concentration and return the plasma pH to 7.4 (point E).

Physiological Effects of Hyperventilation

Physiological responses of various species to high ambient temperature stress have been studied by several researchers. Randall and Hiesland (1939) indicated that panting rate is roughly proportional to body temperature within a narrow body temperature range (41.8-43 C).

As ambient temperature rises from 23.5 to 37 C, panting rate is maximized. As body temperature reaches an upper critical limit (45.9 C), the panting mechanism begins to fail and body temperature may increase dramatically. Hutchinson and Sykes (1953) observed heart rate in the fowl to be inversely related (r=-.99) to environmental temperature until a body temperature of 43.3 C is attained. Giebisch et al. (1954), indicated that acute respiratory alkalosis in dogs results in a compensatory reduction of buffer anions in the extracellular fluid, thereby preventing the development of lethal alkalosis. In acute respiratory alkalosis of the degree achieved in that experiment where $[H_2CO_3]$ fell to approximately 0.2 mM per liter, were no compensatory displacement of HCO_3^- to occur, a pH of 8.1 would result. The pH only rose to 7.7 however, indicating that extrarenal compensation had taken place. Calder and Schmidt-Nielson (1964) observed that pigeons exposed to high temperatures panted at rates more than twenty times the normal respiratory rate. In addition, pH increased, pCO_2 decreased and respiratory minute volume increased considerably. Linsley and Burger (1964) reported a rapid rise in the respiratory rate of hyperthermic cocks from 25 to over 250 breaths per minute. These birds also showed increased heart rate, decreased carbon dioxide tension and increased blood pH, indicating the existence of respiratory alkalosis. The cardiovascular response to hyperthermia in these birds was a fall in diastolic pressure followed by a fall in systolic pressure. These changes occured after heart rate was maximized. Higgins and Iampietro (1967), working with heat stressed dogs, observed respiratory rate to increase initially but then decline at the end of the 150-minute exposure time. In the same experiment,

blood pH and pO_2 increased while pCO_2 decreased. Frankel and Frascella (1968) measured body temperature and arterial blood concentrations of lactate and pyruvate as well as pH, pO_2 and pCO_2 of White Leghorn hens subjected to high environmental temperature. A decrease in pCO_2 and increases in all other components were observed. Calder and Schmidt-Nielson (1968) theorized that evaporative heat loss in heat stressed birds is determined by respiratory minute volume. They observed that panting and gular flutter in various species of flying birds resulted in hyperventilation of the gas exchange surfaces and respiratory alkalosis. Harrison and Biellier (1969) subjected White Leghorn hens to abrupt changes in environmental temperature and observed pulse rate, oxygen consumption and blood pressure to decrease when panting was initiated by increasing ambient temperature. Kohne and Jones (1975) exposed adult female turkeys to high ambient temperature and observed that the resulting polypnea induced profound respiratory alkalosis. The hyperthermia also caused significant decrease in plasma levels of sodium, total calcium, magnesium and phosphorus. They also reported a significant increase in plasma potassium level, but no change in plasma chloride levels.

More recently, El Hadi and Sykes (1981) concluded that alkalosis is a normal and inevitable response to heat stress. They also suggested that the fluctuations seen in pH measurement was the result of a conflict between the demands of thermoregulation which tended to increase respiration and the demands of pH homeostasis which tended to reduce it. At extremely high environmental temperatures, second-phase panting was also evident. This resulted in deeper respiration and greater alveolar ventilation.

Relationship Between Potassium and Acid-Base Balance

Renal and extrarenal potassium homeostasis are affected by changes in acid-base balance. Cooke et al. (1952) theorized that a shift of hydrogen ions into muscle cells in exchange for potassium is the basis for extracellular alkalosis observed in rats studied. Orloff et al. (1953) caused rats to become hypokalemic and alkalotic by feeding a diet deficient in potassium. They hypothesized that the alkalosis was confined to the extracellular compartment and was associated with intracellular acidosis brought about by a transfer of hydrogen ions into the cells in exchange for potassium. Kennedy et al. (1949) observed that urine of humans suffering from hypokalemic alkalosis contained a lower bicarbonate concentration than was expected. They concluded that this was likely due to an increase in the rate of hydrogen ion secretion as the bicarbonate content of the urine was dependent upon the interaction between filtered bicarbonate and secreted acid. An inverse relationship exist between the urinary secretion of hydrogen and potassium ions (Berliner et al., 1951; Orloff et al., 1952). Competition for renal secretion between the hydrogen and potassium ions has been suggested as a possible cause for this inverse relationship (Berliner et al., 1951).

The distal convoluted tubule and collecting duct absorb sodium with chloride and bicarbonate. Sodium is also absorbed via an additional transport system in which sodium absorption is coupled with the secretion of hydrogen or potassium ions. Potassium can replace hydrogen in the ion exchange between the tubular cell and the tubular lumen, thereby generating competition between these two ions. This is

apparently one of the mechanisms by which potassium is lost from the plasma during alkalosis since the deficiency of hydrogen ion creates a more favorable sodium-potassium exchange. It is also thought that the movement of the positively charged sodium out of the lumen favors the passive movement of potassium into the lumen. In addition, where there is active transport of hydrogen ions in exchange for sodium, a deficiency of hydrogen ions such as exist during alkalosis, results in the establishment of a chemical gradient down which potassium ions may diffuse into the tubular lumen for eventual excretion.

It has been demonstrated in humans (Bourdillon, 1937) and dogs (Berliner et al., 1950) that the administration of potassium chloride results in an elevation of plasma potassium concentration accompanied by a fall in plasma bicarbonate and pH. Loeb et al. (1932) observed that ingestion of potassium chloride resulted initially in alkaline urine which gradually became acidic and attributed this to rapid excretion of potassium ions followed more slowly by chloride ions. These changes were related to the differences in the rate of excretion of the potassium and chloride ions. Initially, 71% of the ingested potassium was excreted in the urine compared with only 51% of the chloride eliminated at that time. The observation has been made that the infusion of bicarbonate results in an increased renal excretion of potassium (Darrow, 1950). Roberts et al. (1953) demonstrated that the infusion of potassium chloride into normal dogs produced a decrease in plasma pH and bicarbonate concentration. These alterations were accompanied by a decrease in bicarbonate reabsorption and increased urine pH. These researchers also confirmed the work of Darrow (1950) that the infusion of bicarbonate produces a decrease in plasma

concentration and an increase in urinary excretion of potassium. The studies cited above indicate that infusion of potassium elevates intracellular pH while decreasing plasma pH and bicarbonate, thereby producing a more ideal internal environment compatible with optimal physiologic activity when alkalosis threatens to disrupt the system.

Early studies reported conflicting data on the effect of acid-base imbalance on the intracellular-extracellular potassium distribution. Darrow (1946) and Cooke et al. (1954) demonstrated that muscle potassium is low during metabolic alkalosis and normal to slightly higher during metabolic and respiratory acidosis. Conversly, Keating et al. (1953) suggested that metabolic acidosis results in a shifting of potassium from the cells to the extracellular space. Cotlove et al. (1951) confirmed the findings of Darrow (1946) and assumed that acidosis causes potassium to enter the cells and alkalosis causes potassium to leave the cells. These conflicting views may have been due to a failure to separate the effect of acid-base imbalance on the internal distribution of potassium from its effect on changes in total body potassium. This is possible since hypokalemia may be due to loss of total body potassium or to the shifting of potassium into the intracellular fluid. Scribner et al. (1955) studying the effects of altering pH on the internal equilibrium of potassium in dogs, found that respiratory alkalosis invariably caused a fall in serum potassium levels, while in acidosis, the ratio of extracellular potassium to intracellular potassium was elevated. These researchers hypothesized that the internal equilibrium of potassium is, in part, a function of pH, and that the intracellular-extracellular potassium ratio is increased by acidosis and decreased by alkalosis.

Inherent problems exist when attempts are made to quantify the relationship between extracellular pH change and change in serum potassium concentration. Metabolic and respiratory pH alterations may involve different intracellular buffering mechanisms such as hemoglobin, phosphate and bicarbonate buffer systems and thus may be quantitatively different. Changes in serum potassium concentration induced by changes in total body potassium also help to prevent clearcut interpretations since only approximately two percent of the total body potassium is found in the extracellular fluid. Burnell et al. (1956) consistently demonstrated in humans, that acidosis increases and alkalosis decreases serum potassium concentration independent of changes in total body potassium. They calculated that for every 0.1 unit change in extracellular pH, there was an average inverse change of 0.6 mEq per liter in serum potassium concentration. Simmons and Avedon (1959) working with dogs, indicated that for every unit change in pH, plasma potassium concentration changed inversely an average of 2.0 mEq per liter.

It is assumed that the same degree of acid-base disturbance whether of metabolic or respiratory origin, cause equal changes in plasma potassium concentration. This assumption formed the basis for the experiments of Scribner et al. (1955) who used inhalation of carbon dioxide to alter the pH of dogs and produced a shift of potassium out of the cells. Previous studies examining the relationship between potassium movement and acid-base balance focussed primarily on metabolic disturbances (Roberts et al., 1953; Keating et al., 1953). Orloff (1956) generalized that factors which can be assumed to decrease celullar pH diminish the excretion of potassium and that the converse

was true. Simmons and Avedon (1959) verified that changes in plasma potassium concentration are related to pH alone and are independent of changes in the partial pressure of carbon dioxide or bicarbonate concentration. These changes are therefore similar for comparable degrees of metabolic or respiratory disturbances.

Changes in plasma potassium concentration apparently reflect movement of potassium into or from certain tissues of the body as well as excretory losses. Simmons and Avedon (1959) suggested that the low plasma potassium values obtained during experimentally induced respiratory alkalosis was due to urinary losses. Brown and Mowlem (1960) demonstrated that potassium is lost from the heart immediately following high carbon dioxide breathing (in which dogs breathed air containing 30% carbon dioxide for four hours) after having gained potassium during the breathing exercise. Lade and Brown (1963) found that the gastrocnemius skeletal muscle of dogs lost potassium during hypercapnia while venous pH decreased. Skeletal muscle would therefore appear to be the most likely source of the gradually increasing plasma potassium concentration during acidosis. A possible explanation for the opposite movement of potassium in cardiac and skeletal muscle during respiratory acidosis suggested by Brown and Goott (1963) is that cardiac tissue is less well buffered than blood by a factor greater than the initial ratio of intracellular hydrogen ion concentration. When carbon dioxide tension is increased therefore, a greater rise in hydrogen ion concentration takes place inside the cell than outside and thus potassium would be expected to shift toward the poorer buffered compartment where the greater increase in hydrogen ion concentration occurred, in order to maintain equilibrium.

Factors determining the effect of changes in acid-base balance on plasma potassium concentration have been documented by Bia and DeFronzo (1981). They stated that metabolic disturbances cause a greater change in plasma potassium concentration than respiratory ones, and that acidosis elicits greater changes in plasma potassium concentration than alkalosis. Changes in plasma bicarbonate concentration, duration of acidosis or alkalosis and the extent of intracellular buffering have all been submitted as factors affecting potassium balance. In summary, the relationship between plasma potassium level and acid-base balance is complex and no single theory adequately explains it all. This subject needs to be evaluated at greater depths in poultry since inadequate data exist, and more studies need to be conducted to provide information on such areas as sites and cellular mechanisms by which plasma potassium levels is altered by changes in acid-base balance.

Physiological and Biochemical Functions of Potassium in the Body

Potassium is the principal cation of the intracellular fluids, but is also a very important constituent of extracellular fluid. Its involvement in vital life processes is reflected by its high intracellular concentration relative to that of other cations. Energy for the electrical potential of cells is derived from the differential gradient of potassium ions across the cell membrane, therefore changes in potassium homeostasis invariably affect health. Total body potassium when expressed as a percent of body weight, and its distribution within the body varies with age (Kravis and Kare, 1960) and sex (Cohn et al. 1980). Younger adult men exhibited higher total body potassium values

when compared with older individuals. The higher values for the younger males were attributed to the fact that these individuals had greater muscle mass. Similarly, females exhibited lower total body potassium values since their muscle mass were also lower. Potassium is absorbed mainly from the upper part of the small intestine, but some absorption take place in the ileum and large intestine (Conway and Hingerty, 1948). The major portion of potassium availaible in the diet is readily absorbed within about thirty minutes with less than 10% normally being eliminated in the feces (Brown, 1984). Documented below is a partial outline of the principal functions of potassium in the body.

Excitability, Contractility and Conductivity in Nerves and Muscles

Cells of excitable tissue exhibit a resting membrane potential due to ionic diffusion and active transport of ions. The active transport of ions is carried out by the "Na^+-K^+ pump" which gets its energy from ATP through the action of a Na^+-K^+ sensitive Mg^{++}-dependent ATPase (Schuurmans and Bonting, 1981). This pump moves sodium out of and potassium into the cell with a ratio of three sodium ions to two potassium ions (Jorgenson, 1980). During hydrolysis of ATP, which takes place at the inner side of the membrane, the enzyme system transfers the three sodium ions per ATP molecule to the extracellular fluid in exchange for the two potassium ions, thus building up an electrochemical gradient. The imbalance created in electrical charge results in an "electrogenic pump". The membrane potential is related to ionic distribution across the cell membrane and to the ionic permeability of the membrane (Glynn and Karlish, 1975). This pump mechanism explains

why under normal steady-state conditions, the cytoplasm of most cells, contrary to the extracellular fluid, has a high (100-160 mM) potassium concentration but a relatively low (3-30 mM) sodium concentration. The accumulation of anions will disturb the patterns of anions that are usually measured in the plasma. It is a physical and chemical reality however, that in body fluids, anions and cations must be present in equal concentrations. When the sum of the measured anions do not fully counterbalance the sum of the cations, their difference in mEq/liter is termed the "anion gap" (Emmettt and Nairns, 1977). This simply means that anions other than chloride and bicarbonate must be present to counter the positive charges of sodium and potassium.

Since a concentration gradient for potassium is developed across the membrane, the resting potential of smooth muscle is primarily a K^+ potential. As long as potassium ions diffuse away from the anions, a resting membrane potential will be established. When the membrane potential force driving potassium ions into the cell is equal to the potassium concentration gradient force driving potassium out of the cell, the membrane potential is referred to as a potassium equilibrium potential. Potassium channels have been identified (Hodgkin, 1964; Huxley, 1964) along with sodium, chloride and calcium channels, through which the individual ions may move independently.

Potassium is intricately related to muscle metabolism and function. It influences the contractility of smooth, skeletal and cardiac muscles. The contractile proteins of muscle, actin and myosin, have specific requirements for potassium ions. The potassium concentration of the fluid bathing the heart is important since this ion initiates the

excitation process and affects conduction of impulses and as a result, a deficiency of potassium results in tetanic seizures.

Acid-Base Equilibrium

The distribution of potassium between the extracellular and intracellular fluids is determined by the relationship existing between itself and sodium, chloride and bicarbonate in the plasma. A decrease in the concentration of chloride ion in the plasma, as could occur in a chloride deficient diet, results in a compensatory rise in the bicarbonate level. When the bicarbonate ion concentration increases, there is a reciprocal shift of potassium out of the cells in exchange for sodium.

The function of potassium in maintaining acid-base balance is evident both in the parietal cells of the stomach (West, 1985) and in the tubular cells of the kidney (Androgue and Madias, 1981). In the stomach the production of hydrochloric acid in the lumen hinges on the active transport of hydrogen ions out of the cell in exchange for potassium ions. This exchange utilizes the K^{+}-sensitive, Mg^{++}-dependent ATPase and as such, potassium is playing a double role here.

In the kidney, maintenance of acid-base balance, to a large extent depends on the exchange of hydrogen ions for sodium ions (Pitts, 1958). Potassium is secreted by the distal tubular cells via this coupled mechanism. A normal competition exist between hydrogen ions and potassium ions for secretion (Pitts, 1958; Kokko, 1984). The presence or absence of potassium ions therefore will partially determine how many hydrogen ions are secreted and consequently, the acid-base status of the organism.

Regulation of Cellular Osmotic Balance

Water is retained in the body in fairly constant amount, but its distribution is continuously subject to change. The movement of water from one compartment of the body to another, is directed by osmotic forces which are the principal factors controlling the location and amount of fluid in various compartments (MacKnight and Leaf, 1977). One of the substances in solution in the fluids that has an effect on water retention is potassium. Since water diffuses freely across the cell membrane, its movement is determined by changes in the concentration of osmotically effective electrolytes (mainly sodium and potassium) on either side. Changes in extracellular electrolyte concentration are the most common basis for these water shifts. The function of potasssium in regulating water balance is linked to its exchange for sodium. Tubular reabsorption of sodium is accompanied by water reabsorpion, therefore, in effect, both water and sodium are exchanged for potassium (Pitts, 1958). This is under the influence of aldosterone.

Metabolic Functions

Potassium is the most abundant intracellular cation in man. It is involved in almost all of the body's vital processes and this is reflected in its high intracellular concentration. Approximately ninety percent of the total potassium in the body is found in cell protoplasm (Georgievskii et al., 1982). Potassium is found in the tissues of the body in varying amounts (Table 1), however, muscle tissues serve as the principal store, containing approximately eighty percent when compared with fat and bones (Table 2).

Table 1. Tissue potassium contents of chickens (Kravis and Kare, 1960)[1]

Age[2]	Brain	Skin	Muscle	Liver	Plasma
1	69.5	20.5	29.7	48.2	6.7
5	76.6	27.0	47.7	57.1	3.5
10	95.3	31.8	113.1	69.6	3.9
13	66.4	62.7	102.3	85.6	3.4
19	76.8	30.6	91.6	67.3	2.5
26	71.7	30.1	91.3	65.2	3.0
35	77.3	23.8	92.0	54.2	3.6
42	70.6	39.3	67.3	59.2	4.0
56	100.7	38.9	92.1	65.1	3.4

[1]Average results on tissue of four birds in each age group measured in mg/Kg wet weight.

[2]Days

Table 2. Potassium distribution in carcasses of pigs of different weights (Stant et al., 1969)

	Potassium Content			
	23 Kg	46 Kg	68 Kg	91 Kg
Muscles (%)	77.5	77.7	78.6	81.1
Fat (%)	2.3	5.2	7.7	7.1
Bones (%)	20.2	17.1	13.6	11.8
Total Potassium (g/Kg)	2.3	2.1	1.7	1.5

Carbohydrate Metabolism. Carbohydrate metabolism is influenced by the administration of potassium. The injection of potassium into rats have been reported to cause hyperglycemia and lowered muscle and liver glycogen (Fenn, 1940). Insulin is the only hormone which in physiological concentrations lowers the blood sugar level. Thompson and McQuarrie, (1934) observed that a high potassium diet in diabetic patients increases glycosuria. Absorption of glucose normally results in hyperglycemia which in turn is associated with enhanced insulin release from the pancreas. Insulin plays an important role in the extrarenal potassium homeostasis. Basal insulin secretion is an important factor in maintaining fasting potassium concentration within normal range. Small increments in plasma insulin (25-40 mU/ml) stimulate potassium uptake by muscle, liver, adipose and probably other tissues (Bia and DeFronzo, 1981). Plasma potassium in excess of 1.0-1.5 mEq/L, are capable of stimulating insulin release, and the resultant hyperinsulinemia is necessary to maintain normal potassium tolerance. Insulin can therefore be considered to play a permissive role in facilitating potassium uptake when the rise in plasma potassium concentration is small. Potassium is important in enhancing the synthesis of glycogen (Hue et al., 1975) by promoting a faster inactivation of the enzyme phosphorylase A and activation of glycogen synthetase. Several other enzymes that function in carbohydrate metabolism are activated by potassium. Adenosine triphosphatase which splits phosphates from energy-rich adenosine triphosphate (ATP) to release energy, is activated by potassium. ATP is important in the phosphorylation of hexoses during the glycolytic process. Other enzymes of the glycolytic pathway which are important in the transfer of energy

are also activated by potassium. These include hexokinase which catalyzes the conversion of glucose to glucose-6-phosphate, and pyruvate kinase which functions in the transfer of high-energy phosphate bond from phosphoenolpyruvate to ADP in the formation of ATP. Another enzyme fructokinase, which catalyzes the conversion of fructose to fructose-1-phosphate in the liver is activated by potassium. Potassium activation is also required by galactosidase which catalyzes the conversion of lactose to glucose and galactose, and by salivary amylase, an important enzyme in starch digestion.

Protein Metabolism. Potassium appears to be directly connected with the process of protein synthesis. If added to protein-deficient diets, it apparently promotes weight gain and feed utilization improvements in piglets (Georgieveskii et al., 1982). In addition, it has been shown that the rate of amino acid utilization in protein anabolism is influenced by cellular stores of potassium (Holley and Carlson, 1955). This is probably because high intracellular potassium concentration is necessary for protein biosynthesis by the ribosomes (Alexis et al., 1971). Robbins et al. (1982) indicated that dietary potassium level affect the muscle free amino acid pattern of chicks and hyothesized that under conditions of potassium deficiency, a ratio of muscle K^+ : Na^+ near one, rather than the absolute concentration of either, could be the cause of amino acid accumulation. Liebholtz et al. (1966) observed improved weight gain in pigs when potassium was added at levels of 0.4 or 0.8% of the diet and suggested that lysine utilization was improved. Froseth et al. (1982) theorized that supplemental potassium has a sparing effect on dietary lysine requirement of pigs. Arnauld and Lachance (1980) demonstrated tha potassium deficiency in rats resulted

in increased muscle lysine concentration, while Scott and Austic (1978) reported decreased blood concentrations of lysine in chicks as a response to potassium bicarbonate supplementation thereby suggesting more efficient lysine utilization.

Enzymes are specific proteins. Any effect of potassium on protein metabolism in general therefore applies to enzymes also. In addition, many enzymes have a specific requirement for potassium (Boyer et al., 1942; 1943) while in some cases, this ion may serve to influence enzyme activity. Added to the list of enzymes already mentioned under carbohydrate metabolism, are the following enzyme systems which are influenced or activated by potassium. Carbonic anhydrase, which functions in the production of carbonic acid from carbon dioxide and water; and cholinesterase, which inactivates muscle stimulation by the hydrolysis of acetylcholine to choline and acetic acid.

Water Metabolism. Water is such a common substance that it is not usually afforded close scrutiny in animal nutrition. The very important functions of temperature regulation, lubrication, hydrolysis and the fact that it functions as a transport medium and solvent for nutrient and waste products, dictates that proper recognition as a nutrient must be made. Rosenberg and Palafox (1956) suggested that potassium salts were the primary cause of diarrhea when cane molasses was included in chick's diet. This could probably be attributed to the fact that body water appears to be passively distributed in proportion to osmotic activity, and all or almost all of body potassium is osmotically active (Edelman et al., 1958). The osmotically active sodium and potassium ions in the body fluids are closely approximated by the exchangeable sodium (Na_e^+) and exchangeable potassium (K_e^+). Since sodium represents

the major electrolyte in plasma, the relationship between plasma sodium (P_{Na}), exchangeable potassium, and total body water (TBW) can be written as follows:

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

Kondo and Ross (1962a; 1962b) determined that the fecal moisture content of molasses-fed chicks was positively correlated with water intake which in turn was highly and positively correlated with potassium consumption. Potassium, when included in the diet of turkey poults at 1.7% was found to increase water intake by 36% independent of feed (Smith et al., 1973). The results of these studies highlight the importance of this electrolyte on water intake and hence water metabolism.

Effects of Potassium Deficiency

As has been indicated, the physiological and biochemical functions of potassium are quite numerous, and it has been shown that the nutrient plays a vital role in animal physiology. Under normal conditions the diets of both humans and farm animals are not likely to be deficient in potassium. This is indeed a fortunate situation since this nutrient must be supplied daily because of its rapid mobility. A decrease in the body's exchangeable potassium is usually due to excessive lossses of this ion and is easily corrected by potassium supplementation (Nagent de Deuxchaisnes et al., 1961).

Potassium deficiency can be the result of several causes occuring singly or in combination. Inadequate potassium in the diet, though improbable, will lead to inadequate intake and consequently, a deficient state. Reduced intake results in the catabolic release of potassium

from the tissues and may lead to a reduction in serum potassium concentration in humans (Ljunggren et al., 1957). The significance of serum potassium as a window for the potassium status of the body, is that serum potassium concentration is easily, accurately, and precisely quantified. It is the only clinically practical measure of potassium stores in the body. Low values, generally indicative of potassium deficiency, may be associated with a number of metabolic or structural abnormalities. More than 98% of the total body potassium (110mEq/L) is located intracellularly where it is difficult to quantify, whereas the normal serum potassium concentration (3.5-5.0 mEq/L in humans) is relatively easily measured. Significant hypokalemia results when physiologic, chemical or pathological conditions enhances chronic renal losses. Alkalosis, whether respiratory or metabolic, results in urinary potassium losses in dogs (Simmons and Avedon, 1959). The hypokalemia which is usually observed in conjunction with enteric problems such as emesis and gastric malfunctions is usually the result of renal potassium loss (Kaiser and Schwartz, 1966; Schwartz et al., 1968). Aldosterone secretion in response to sodium deficiency enhances potassium secretion into the distal tubule (Barger et al; 1958), while certain diuretic drugs and osmotic anions promote potassium losses (Mohr et al., 1979). The colon of the gastrointestinal tract, like the distal tubule of the kidney, secretes potassium in exchange for sodium under the influence of aldosterone. In various forms of diarrhea fecal potassium loss is increased though less dramatically than sodium (Fordtran and Inglefinger, 1968). Several factors contribute to potassium loss in chronic diarrhea regardless of cause i.e inflammation, infection, malabsorption or laxative abuse. First there is a brief initial loss of

potassium in large stool volumes (Fordtran and Dietchy, 1966) followed by a decrease in concentration. Additional potassium loss is brought about by a profuse shedding of the potassium-rich mucus lining if there is severe or sustained diarrhea.

The balance between potassium intake and excretion is reflected in the potassium content of the body. As was previously indicated, the energy for the electrical potential of the cells is derived from the differential gradient of potassium across the cell membrane. If potassium homeostasis is changed drastically, health in general will be adversely affected. The debilitating effects of potassium deficiency has long been recognized (Osborne and Mendel, 1918; Miller, 1923; Kornberg and Endicott, 1946; Carone and Cooke, 1953). The development of deficiency symptoms may depend on a number of factors affecting serum level of potassium (Holley and Carlson, 1955). The rate of production and duration of the deficiency, the ratio of extracellular to intracellular potassium concentration, and the individual sensitivity of the specific cells to the altered serum potassium concentration are factors influencing the manifestation of the clinical symptoms of potassium deficiency. Owing to the ubiquitous nature of potassium in the body and its major function in producing electrical potential, the various anatomical systems of the body as well as general nutrient metabolism are affected by its deficiency.

Nutrient Metabolism

The effect of severe potassium deficiency on the metabolism of glucose and glycogen in the intact animal was examined by Gardner et al. (1950). Rats on potassium deficient diet for 90-120 days exhibited

extremely low or absent liver and muscle glycogen, normal fasting blood and urine glucose concentration and elevated oral glucose tolerance. These researchers concluded that glycogenesis was blocked by the continued state of potassium deficiency, and that the inhibited tissue glycogenesis may potentiate the diabetic state. Impairment in glucose tolerance in potassium deficient persons have been demonstrated (Sagild et al., 1961). Conn (1965) reported a delay in serum insulin concentration response to glucose loading in patients with primary hyperaldosteronism. Spergel et al. (1967) stated that short term hypokalemia may lead to mild hyperglycemia and subtle impairment in insulin response to parenteral glucose, but not necessarily to marked glucose tolerance. This was consistent with the observations of Gorden (1973) on persons made hypokalemic with low potassium diets. He concluded that the primary effect of potassium deficiency on glucose tolerance was its inhibition of the early phase of glucose release.

A potassium requirement for protein synthesis has been demonstrated in the cell-free system of the mammalian liver (Sachs, 1957) and in E. coli (Lubin, 1964). Rinehart et al. (1968) investigating the effects of dietary potassium deficiency on protein synthesis in young chicks, observed that potassium deficient chicks incorporated significantly less L-leucine-1-^{14}C into skeletal muscle proteins than non-deficient controls. These researchers also noted that there was an increase in the incorporation of labelled leucine into plasma protein in the potassium deicient birds. They concluded that although plasma potassium decreases in the deficient chicks, synthesis of plasma protein in the liver may occur at near normal rate since liver potassium resists depletion. This is in contrast to the faster depletion rate for

skeletal and heart muscle (Rinehart et al., 1969). Furthermore, as a result of higher specific activity in the deficient birds due to reduced synthesis of muscle proteins, increased amounts of radioactivity would be observed in the plasma proteins without an increase in protein synthesis. The effects of inadequate dietary potassium supply on in vivo and cell-free aspects of protein synthesis in skeletal muscle of rats was examined by Alexis et al. (1971). Their results indicated that potassium deficient rats consumed less feed, gained less weight and had a lower potassium concentration than the controls. In vivo incorporation of ^{3}H-leucine into muscle protein was seventy percent of the value for the controls. Ribosomal protein synthesis was reduced by feeding a potassium deficient diet. Experiments performed by Muntwyler et al. (1953) on potassium and/or protein deficient rats to elucidate the relationship between potassium and protein metabolism indicated that the poor growth resulting from potassium deficiency is at least, in part, related to protein metabolism. The nitrogen intake of the control and experimental animals were similar, therefore it would appear that nitrogen anabolism is impaired in potassium deficiency resulting in reduced growth. Inadequate protein in a low potassium diet caused rats to grow slowly but did not result in potassium deficiency. Apparently potassium from the tissue replaced the extracellular potassium lost in the urine. When a protein supplemented low potassium diet was fed to protein-starved animals, growth was enhanced but potassium deficiency developed.

Cardiovascular

The effects of potassium deficiency on the cardiovascular system has been well described by a number of authors. Schrader et al. (1937) noted that massive erosions involving both the endocardium and the myocardium were found in both ventricles of the heart. They reported complete destruction of the cardiac musculature and replacement with scar tissue along with engorgement of the cardiac vessels with death occuring within 23 days, on the average. Follis et al. (1942) observed microscopic changes in the heart tissue of rats after eight days on a potassium deficient diet with gross lesions appearing after twenty-one days. Changes included loss of striations of individual muscle fibers, increased proliferation of connective tissue and general necrosis of muscle fibers. Kornberg and Endicott (1946) noted that in addition to these changes, there was leucocytic invasion of the necrotic areas. French (1952) produced myocardial lesions in less than twelve days in protein-depleted, potassium-deficient rats. He observed that within the area of the lesions there was a primary degeneration of muscle substance which led to necrosis and disintegration of myocardial fibers, and that the muscle debris was removed by phagocytes. Secondary to the fiber degeneration, there was interstitial edema in the connective tissue. This edema probably occur because it appears that cardiac muscle is susceptible to injury from an ionic imbalance. Edema is usually caused by expanded interstitial cell volume and increased sodium, because sodium is the major extracellular fluid solute. A deficiency of potassium therefore could cause an accumulation of sodium followed by water.

Molnar et al. (1962) found great variation in the state of contraction of adjacent myocytes of potassium deficient rats to be the earliest abnormality to develop in addition to increased membrane permeability induced by the electrolyte imbalance. Oxygen consumption in potassium deficient muscle fails to change relative to controls which indicate an inefficient conversion of energy into work. Harrison et al. (1970) found depressed mitochondrial ATPase activity associated with the swelling of mitochondria in potassium deficient rats. This suggests that normal energy production may be disrupted in the mitochondria. Disturbance in heart rhythm is one consequence of increased excitability of heart muscle as a result of potssium depletion in humans (Potts et al., 1977). Decreasing the extracellular potassium concentration in the bath surrounding isolated cardiac muscle immediately produced substantial increase in contractile tension. Major changes in the cellular membrane potential are associated with this increase. These changes include an increase or decrease in the resting membrane potential, increase in the amplitude of the action potential, decrease in the duration of the plateau, and an increase in the total duration of the action potential. The foregoing review of existing literature describing myocardial lesions that appear during potassium deficiency, indicate that without exception, the essential feature common in all observations is the presence of necrosis in the cardiac muscle.

Renal

The effects of potassium deficiency on renal histology and functions have been extensively reviewed. Relman and Schwartz (1958) summarized previously reported observations on structural changes as

ranging from an enlargement of the kidneys to changes in tubular structure. The changes in renal mass begin to occur within a few days and the microscopic changes in tubules within one week. The most prominent functional abnormality is a defect in the concentrating power of the kidneys resulting in their inability to concentrate urine. Holliday et al. (1960) found that persistent potassium deficiency in humans resulted in a progressive decline in urinary concentrating ability. This was possibly the result of the irreversible scarring of the kidney. They also indicated an additional element of architectural disorganization and scarring of renal tissue in addition to the tubular lesions. Abbrecht (1969), indicated that the decrease in renal concentrating ability correlated well with plasma potassium concentration.

In a further examination of potassium deficient dogs Tate et al. (1978) observed as much as fifty percent increase in the weight of kidneys when compared with control. This change in the gross aspects was accompanied by microscopic changes in the epithelial cells lining the collecting tubules. These cells exhibited vacuolation, hypertrophy and hyperplasia. Another renal complication of acute potassium deficiency is development of interstitial nephritis. Impairment in renal blood flow and changes in renal ionic composition due to increases or decreases in excretion rates are all characteristics of potassium deficiency.

Secretion of potassium into the distal convoluted tubule of the kidney begins with the active transport of these ions from the interstitial space where concentration is low, into the tubular cell where concentration is high. This involves a pump. A chemical gradient

is set up between the tubular cell and the lumen where potassium concentration is as low as in the interstitial space. Potassium then moves into the lumen by diffusing down its concentration gradient. An electrical gradient is generated by the movement of positively charged sodium ions out of the lumen. This electrical gradient also favors the movement of potassium into the lumen. In the distal tubule hydrogen ions are actively secreted. The greater the number of hydrogen ions that are pumped into the lumen in exchange for absorbed sodium, the greater the reduction in the electrical gradient, thereby limiting the movement of potassium into the lumen. When alkalosis is present, less hydrogen ions are available for secretion, thus there is a tendency for more potassium to be secreted into the lumen thereby contributing to hypokalemia. The rate of sodium reabsorption into the cells of the distal tubule can be directly influenced by the plasma potassium concentration because increased potassium concentration can directly stimulate aldosterone release from the adrenal gland independent of the renin-angiotensin-aldosterone system. Aldosterone permits increased tubular sodium reabsorption and thus increased potassium secretion.

Gastrointestinal Tract

The effects of potassium deficiency on the body is partially manifested in changes in intestinal motility and volume of gastric secretions. Webster et al (1950) working with potassium deficient rats and dogs, observed a progressive decrease in the tone and movements of the whole digestive tract of the rats, but little change in the dogs. They also noted a reduction in the strength and rate of rhythmic contractions in the dogs. In these same studies, potassium deficiency

produced an increase in the volume of gastric secretions. Perdue and Phillips (1952) observed that potassium deficiency in rats lowered the propulsive motility of the small intestine by about fifty percent. Streeten and Vaughn-Williams (1952) theorized that the depression of intestinal propulsion which gave rise to complete intestinal paralysis in severe deficiencies, was a result of potassium ions moving away from the cells into the plasma. The composition of gastric juice of normal and potassium deficient rats was studied by Carone and Cooke (1953). They found that the pH of the gastric juice of potassium deficient but non-alkalotic rats was higher than that of the controls. They also noted a corresponding decrease in potassium concentration and increase in sodium concentration in the potassium deficient rats.

Skeletal Muscles

Since the energy for the electrical potential of cells is derived from the differential gradient of potassium ions across the cell membrane, a deficiency of this element is easily reflected in skeletal muscle weakness. Potassium depleted dogs demonstrated a loss of skeletal muscle integrity (Knochel and Schlein, 1972). Muscle blood flow and potassium release was subnormal in these dogs and resulted in rhabdomyolysis when these animals were exercised. In contrast, Lowensohn et al. (1978) saw no evidence of skeletal muscle damage in potassium depleted dogs. However, the muscle potassium contents of the dogs in this study, decreased an average of twenty-four percent from the controls compared with a depletion of forty-eight percent achieved by Knochel and Schlein (1972). Tate et al. (1978) recognized a slight swelling of the skeletal muscle fibers of dogs as the first effects of

potassium deficiency on the skeletal muscle. These workers observed necrotic lesions in muscle fibers, which became more severe as deficiency persisted. Since potassium is an important regulator of glycogen synthesis in the cell (Hue et al., 1975) and glycogen is the major fuel utilized in muscle work, the glycogen content of skeletal muscle decreases in the potassium deficient animal and is a possible cause of cellular injury during exercise.

General Symptoms of Potassium Deficiency

In cases where animals are made potassium deficient, usually as a result of dietary or disease-causing factors, deficiency symptoms may appear within a relatively short period of time due to lack of storage and the high mobility of this mineral. Georgievskii et al. (1982) cited impaired growth, anorexia, weight loss, bristling hair, ataxia and atony of the intestines as some signs of potassium deficiency likely to be seen in most farm animals. Gillis (1948) observed symptoms of potassium deficiency in chickens to include retarded growth, weakness and loss of use of the legs. Death, when it occured in that study was preceded or accompanied by tetanic seizures in which the muscles were unable to relax. Severe potassium deficiencies in lactating cows have resulted in decreased feed intake, decreased milk production, pica, loss of hair glossiness and decreased pliability of hides. Polydipsea and polyurea have also been observed in potassium deficient rats.

Effects of Heat Stress on Potassium Homeostasis

Excretion of potassium in excess of intake could be catabolic, consequent to thermal stress. Knochel et al. (1972) observed that

sweating and urinary excretion were the major causes of potassium losses in humans subjected to exhaustive physical exercise during summer. Gordon and Andrews (1966) observed a seven percent loss (176 mEq) in the initial serum potassium content of the body when human volunteers were subjected to heat stress. At the initial stage of this study, the change from a diet that was optimal in its potassium content to one that was low in potassium resulted in negative potassium balance with a loss of 91 mEq due to diet change. These researchers observed however, that a steady state was soon reestablished.

Potassium is a potent vasodilator. It is released from the contracting fibers of skeletal muscles and its rising concentration is thought to dilate arterioles (Kjellmer, 1965). It is logical therefore that a higher plasma potassium concentration would be required in order to fascilitate heat loss in hotter climatic conditions. Knochel, (1974) observed that forty-six percent of heat stroke paitents whose serum potassium concentration were measured, were hypokalemic. An increase in serum potassium has been noted in cats (Brewer, 1940), dogs (Golberg et al., 1952) and rats (Frankel, 1959) with hyperthermia. Golberg et al. (1952) further demonstrated that serum potassium levels of dogs made hyperthermic by external heat application rose progressively to critically high levels, enough to constitute the major cause of death. Acidosis, which was present in the latter stages of these experiments (pH <7.1) caused the increase in plasma potassium level. When metabolic acidosis was induced by ammonium chloride administration, plasma potassium values rose by 11-13% over controls. Kozlowski and Saltin (1964), observing humans in a 38 C environment, reported a rise in serum potassium attributed to a combination of heat and exercise, but saw no

increase in potassium upon exposure to heat alone. Coburn et al. (1966) observing humans in a greater degree of heat stress (46 C), noted an increase in serum potassium with hyperthermia. The rise in serum potassium content was not evident in potassium deficient persons and was thought to be a result of intracellular potassium depletion.

Movement of potassium from intracellular to extracellular spaces evidently occurs during short term exposure to heat stress. If the stress conditions persist however, urinary losses (Loeb et al., 1932; Darrow, 1950; Roberts et al., 1953) could result in total body potassium depletion and redistribution to intracellular spaces. Respiratory alkalosis, similar in degree and duration to that reported with heat exposure, is associated with an initial abrupt increase in serum potassium followed by a persistent reduction in serum potassium concentration and intracellular gain (Burnell et al., 1956; Hall and Reeser, 1962). Several researchers have attempted to quantify the blood potassium concentration of chickens reared under thermoneutral conditions and when exposed to high temperature stress (Table 3). The results of these experiments indicate that the chicken's plasma potassium values range from approximately 3 mEq/L to 5 mEq/L and that for the most part, during heat stress these values decline substantially. In broiler chickens, heat stress induced hyperventilation results in respiratory alkalosis (Teeter et al., 1985). If the bird's physiologic response to heat stress is similar to other species, then potassium losses under such conditions could partially explain the debilitating effects of heat stress.

Table 3. Blood potassium concentration of chickens at thermoneutral conditions and when exposed to heat stress

		Potassium Concentration (mEq/L)		
		Thermoneutral	Heat stress	
Rinehart et al.	(1969)	5.2		(Plasma)
Deetz and Ringrose	(1976)	3.8	3.0	(Plasma)
Vo et al.	(1978)	4.19	4.11	(Plasma)
Huston	(1978)	27.3	23.7	(Whole blood)
Robbins et al.	(1982)	5.2		(Plasma)

Potassium Requirements of the Chicken

Potassium plays a vital role in animal physiology. This knowledge has spurred several researchers to make attempts to quantify the requirements of various species for this element. Apparently, the earliest documented work on the potassium requirements of chickens is that of Ben Dor (1941) who demonstrated the necessity for including potassium in the diet of young chicks. The results of his work placed the potassium requirement at 0.17% of the diet in order to achieve maximum growth, and 0.13% to prevent mortality. Gillis (1948) concluded that the dietary potassium requirements of chickens ranges from 0.20 to 0.24%. He indicated that the higher level of potassium was required if the phosphorus content of the diet was marginal, thereby suggesting an interaction between the two elements. Gillis further indicated that the level of potassium had an effect on bone calcification. Burns et al. (1953) theorized that the minimum potassium requirement of chickens varied with the rate of growth. They indicated that the dietary requirement ranged from 0.23 to 0.40%, with the higher level being necessary during the fast growth rate period of the first four weeks of life. Leach et al. (1958) reported that maximum potassium requirement of the chick was affected by the protein and energy content of the diet. They demonstrated an interrelationship between dietary protein level and potassium requirement whereby increasing the protein content of the diet resulted in a corresponding increase in the potassium requirement for growth and survival. These investigators also indicated that a high-energy diet increased potassium requirement. The chick's potassium requirement was also observed to increase with increasing age. More

recently, Robbins et al. (1982) reported maximization of chick gain between 0.22 and 0.24% dietary potassium. Presently, the National Research Council (1984) recommends that potassium be included in the diet of broilers at a level of 0.4% from hatching to three weeks. The recommended level is reduced to 0.3% between six and eight weeks of age. As presented, these recommendations make no reference to environmental conditions such as ambient temperature. Deetz and Ringrose (1976) suggested that 0.6% potassium in the diets of hens is a suitable level to meet total requirements for potassium when these birds are subjected to heat stress.

If respiratory alkalosis indeed precipitates urinary potassium losses in poultry, then the potassium requirement of poultry exposed to heat stress must be re-evaluated.

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CHAPTER III

HIGH AMBIENT TEMPERATURE STRESS EFFECTS ON ACID-BASE BALANCE AND POTASSIUM REQUIREMENT OF BROILER CHICKS

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Abstract

Four experiments were conducted to evaluate the effect of heat stress on acid-base status and the potassium (K) requirement of broilers. The corn-soybean meal-fish meal basal ration (.73% K) contained more K than required for chicks reared at 24 C and 55% relative humidity. However, chicks reared under continuous thermostress (35 C, 70% relative humidity) exhibited panting phase blood alkalosis (pH of 7.46) and potassium deficiency symptoms. Supplementing drinking water with .2% NH_4Cl reduced panting phase blood pH to normal values and increased live weight gain (23%) and feed efficiency (7.7%). Supplementing drinking water with .15% KCl also increased ($P<.05$) live weight gain (46%) and feed efficiency (15.4%) but did not affect ($P<.46$) blood pH. A significant ($P<.01$) interaction existed between NH_4Cl and KCl for body weight gain. Potassium chloride alone or with NH_4Cl can

alleviate some consequences of heat stress, but supplementing drinking water with a high level of NH_4Cl (.5%) decreased ($P<.05$) blood pH to acidotic levels and reduced body weight gain. Potassium chloride supplementation exacerbated NH_4Cl toxicity. Potassium carbonate and KCl were found to be equally efficacious indicating that the response can be attributed to K^+ and not Cl^-. Beneficial effects of NH_4Cl therapy for heat stressed broilers may act through K conservation.
(Key words: heat stress, respiratory alkalosis, ammonium chloride, potassium chloride, potassium carbonate).

Introduction

High ambient temperature-relative humidity stress drastically reduces growth rate of broilers. The heat-stressed broiler pants for evaporative cooling to lose heat (Jukes, 1971). Though the physiological effects of hyperthermic panting have been studied, the impact of panting on acid-base balance and growth rate is not well understood. Parker and Boone (1971) observed that blood pH in the male turkey decreased when exposed to thermal stress. Kohne and Jones (1975a) reported that acute hyperthermia produced alkalosis but chronic hyperthermia had no effect (Kohne and Jones, 1975b). Darre et al. (1980) observed that pH increased curvilinearly as Leghorn hens were exposed to an increasing ambient temperature. Siegel et al. (1974) and Vo and Boone (1979) found that blood pH for broilers reared under continuous 35 C and thermoneutral conditions were similar but Bottje et al. (1983) reported that blood pH was elevated ($P<.05$) at the higher temperature. Teeter et al. (1985) observed that chronically thermostressed chicks exhibit a respiratory cycle with panting and

nonpanting phases. Blood pH of panting birds ($P<.05$) was elevated while that of nonpanting chicks ($P>.10$) was not. Correcting this respiratory alkalosis by addition of NH_4Cl to the diet increased ($P<.05$) live weight gain by 25%. Deeper understanding of the biological perturbations of panting is essential to modify the deleterious effect of environmental heat stress.

Urinary potassium excretion increases during alkalosis of mammalian species (Harper et al., 1977). The decline in hydrogen ion concentration within the renal tubular cells causes secretion of potassium to increase because of competition between hydrogen and potassium ions for reabsorption in the distal tubule. This mechanism for increased potassium excretion has not been verified in poultry though plasma potassium has been measured. Kohne and Jones (1975a) exposed turkeys to acute hyperthermia and observed that birds developed a profound alkalosis and an increased plasma potassium concentration. In contrast, Huston (1978) subjected female chickens to 8, 19, and 30C and observed an inverse relationship between ambient temperature and blood potassium concentration. Hence, current data do not allow one to verify or refute the distal tubule competition theory. Plasma K values can be altered by factors in addition to excretion if changes occur in water evaporation from lungs or in feed and water consumption. In addition, Simmons and Avedon (1959) suggested that intracellular K stores could buffer extracellular concentrations which could mask excretion effects.

The objective of the following study was to determine if the benefit of NH_4Cl for heat stressed broiler chicks is related to the broilers potassium needs.

Materials and Methods

Arbor Acre x Vantress chicks were raised on rice hull litter and fed a corn soybean meal starter diet during the first 3 weeks posthatching. On the first day of the 4th week, following an overnight fast, chicks were sexed, weighed and randomly allotted to treatment. Each treatment contained 10 replicates with 6 chicks (3 male, 3 female) per replicate. Birds were housed in wire floored grower batteries containing 61 x 82 cm compartments within a thermostatically and humidistatically controlled environmental chamber under continuous tungsten filament lighting. Week 4 was utilized to adapt all birds to the grower ration (Table 1) and their environment. Conditions for optimal growth were obtained with the environmental chamber set at 24 C and 55% relative humidity. For thermostress, the chamber temperature was increased by 2 C/day to 35 C at 70% relative humidity. Feed and water were continuously available. Body weight gains and feed consumption were determined upon completion of each test period. Supplemental KCl, K_2CO_3 and NH_4Cl were added to the drinking water. Previous observations in our laboratory indicated that under heat stress adding .3% NH_4Cl to the drinking water of broilers increased live weight gain. Where blood pH data are reported, blood was obtained via wing venipuncture and pH was determined immediately utilizing a Corning blood pH/gas analyzer. All data were subjected to analysis of variance using the General Linear Model of the statistical analysis system (Barr et al., 1976). Duncan's multiple range test was used to determine differences ($P<.05$) between means when a significant F statistic was indicated by the analysis of variance (Steel and Torrie, 1960). Correlation coefficients were estimated as specified by Steel and Torrie (1960).

Table 1. Composition of the Grower Ration Used in Experiments 1 through 4.

Ingredient	Numerical Name[1]	%
Ground Corn	4-02-931	57
Soybean Meal (44%)	5-04-604	22
Fish Meal (Menhaden)	5-02-009	9
Tallow		5
Ground Alfalfa	1-00-023	2.7
Dicalcium Phosphate	6-01-080	2.5
Calcium Carbonate	6-01-069	.9
Vitamin Mix[2]	--	.4
Salt	6-14-013	.3
Trace Mineral Mix[3]	--	.1
D-L-Methionine	--	.1

[1]Atlas of Nutritional Data on United States and Canadian Feeds.

[2]Mix contained Vit. A, 3,527,360 I.U.; Vit D_3, 1,322,760 I.U.; Vit E, 11,905 I.U.; Vit B_{12}, 3.5 mg; Riboflavin 2,205 mg; Niacin 6614 mg; d-Panthothenic Acid, 7055 mg; Choline, 176,368 mg; Menadione, 291 mg; Folic Acid, 441 mg; Pyridoxine, 882 mg; Thiamine, 882 mg; d-Biotin, 44 mg per Kg.

[3]Mix contained Manganese, 12.0%; Zinc, 8.0%; Iron, 6.0%; Copper, 1.0%; Iodine, .1%; Calcium, 18%.

Experiment 1.

The basal ration was tested for K adequacy in this 2 week study at thermoneutral conditions. Broilers received 0, .05, .1 or .15% supplemental K. Body weight gain and feed efficiency were monitored.

Experiment 2.

This experiment evaluated the interaction between NH_4Cl and KCl supplementation. Treatments were arranged in a 4 x 4 factorial. Ammonium chloride levels in drinking water were 0, .3, .4, and .5% while KCl levels were 0, .05, .1 and .15%. Body weight gain, feed effficiency and blood pH were measured under heat stress conditions over a 3 week period.

Experiment 3.

The effect of K supplement anion composition on body weight gain, feed efficiency and blood pH were evaluated over a 3 week period. Broilers were supplemented with 0 or .2% K from KCl or K_2CO_3 under heat stress conditions.

Experiment 4.

This experiment was conducted to determine the NH_4Cl by KCl interaction at lower NH_4Cl levels under heat stress over a 2 week period. Ammonium chloride levels were 0, .1, .2 and .3% and KCl level were 0, .05, .1 and .15%. Body weight gain, feed efficiency and blood pH were measured.

Results and Discussion

In the first experiment, the test diet (Table 1) in all experiments was evaluated for potassium adequacy in broilers reared in a thermoneutral environment. Supplementing the basal diet (.73% K) with .05, .10 and .15% additional K (Table 2) did not improve ($P<.10$) either body weight gain or feed efficiency. A K response was not expected as the basal diet contained .73% K, considerably above the .3% suggested to be the dietary K requirement (National Research Council, 1984).

In the second experiment, the effects of supplementing drinking water with KCl and NH_4Cl and on growth rate, blood pH and feed efficiency of chicks housed within the chronically heat stressed environment were evaluated. Broilers consuming the test ration and untreated tap water had a mean panting pH of 7.45 (Table 3). Hurwitz (1973), correlating live weight gain with blood pH, indicated that growth rate was maximum when blood pH was 7.28 and that growth rate declined markedly when pH values either exceeded 7.30 or fell below 7.20. Similarly, Teeter et al., (1985) indicated that when panting blood pH values exceeded 7.35 with environmental heat stress, growth rate and feed consumption by broilers was reduced. In this experiment supplementing the drinking water of heat stressed chicks with .3% NH_4Cl alone reduced blood pH to 7.33 and increased ($P<.10$) live weight gain by 7.2%. Supplementing drinking water with .4 and .5% NH_4Cl reduced panting phase pH to 7.25 and 7.17 and decreased body weight gain by 3.1 and 22.3%, respectively. These levels probable caused acidosis during the non-panting respiration phase. When panting phase blood pH values falls below 7.28, nonpanting phase acidosis and decreased body weight gain of chicks exposed to heat stress have been observed previously.

Table 2. Broiler Weight Gain and Feed Efficiency for Experiment 1.

Basal K (%)	.73	.73	.73	.73
Supplemental K (%)	0	.05	.10	.15
Total K (%)	.73	.78	.83	.88
Gain (g)	1157.5	1121.7	1200.6	1077.9
Gain/Feed	.37	.37	.38	.35

Table 3. Broiler Weight Gain, Feed Efficiency and Blood pH Values for Experiment 2.

%				
K	NH_4Cl	Gain (g)	Gain/Feed	Blood pH
0	0	376.9^{efg}	$.29^{a}$	7.45^{a}
0	.3	403.0^{cde}	$.31^{a}$	7.33^{bc}
0	.4	387.6^{def}	$.27^{a}$	7.25^{cde}
0	.5	352.3^{fg}	$.29^{a}$	7.17^{efg}
.05	0	389.2^{def}	$.30^{a}$	7.39^{ab}
.05	.3	452.7^{ab}	$.32^{a}$	7.27^{cd}
.05	.4	399.7^{cde}	$.29^{a}$	7.23^{de}
.05	.5	344.3^{b}	$.27^{a}$	7.14^{fgh}
.1	0	416.5^{bcd}	$.30^{a}$	7.41^{ab}
.1	.3	456.6^{ab}	$.29^{a}$	7.27^{cd}
.1	.4	431.7^{bc}	$.32^{a}$	7.19^{def}
.1	.5	303.8^{h}	$.25^{a}$	7.16^{fg}
.15	0	444.6^{b}	$.31^{a}$	7.40^{ab}
.15	.3	484.7^{a}	$.31^{a}$	7.17^{efg}
.15	.4	358.5^{fg}	$.27^{a}$	7.09^{gh}
.15	.5	263.6^{i}	$.18^{b}$	7.07^{h}

^{abcdefghi}Means within a column with unlike superscripts differ ($P<.05$).

Use of ammonium chloride to combat heat stress appears effective, but caution must be exercised to avoid precipitating acidosis during the nonpanting phase. Based on conditions in this experiment, the optimal level for NH_4Cl supplementation of water was .3% or less.

Adding .05, .1, and .15% K as KCl to broiler drinking water also linearly increased ($P<.01$) live weight gain. However, KCl supplementation failed to alter ($P>.10$) blood pH. This result suggests that blood pH was important only when K intake was insufficient to maximize growth rate. However, an interaction ($P<.01$) existed between KCl and NH_4Cl supplementation for gain and blood pH. Increasing drinking water KCl level exacerbated the reduction in gain with .4 and .5% NH_4Cl indicating that K^+ and H^+ ions are related in metabolism. However, Cl^- contributions to growth cannot be ruled out in this experiment.

Relative effects of chloride and CO_3^- were examined in the third experiment by providing an equal amount of K from KCl and K_2CO_3 for heat stressed chicks. Added KCl and K_2CO_3 increased ($P<.05$) weight gain of the thermostressed chicks (Table 4). Lack of a difference between KCl and K_2CO_3 indicated that the beneficial effect was due to K.

The KCl x NH_4Cl interaction was further evaluated at lower NH_4Cl supplementation levels in the fourth experiment. Ammonium chloride levels evaluated included .1, .2 and .3% (Table 5). As in the second experiment, NH_4Cl addition to drinking water enhanced live weight gain and depressed blood pH of the heat stressed chicks. However, the NH_4Cl effect on live weight gain was maximum at the .2% supplementation level. As in experiment 2, KCl enhanced live weight gain and the KCl x NH_4Cl interaction was significant ($P<.01$).

Table 4. Broiler Weight Gain and Feed Efficiency Values for Experiment 3.

Ration	Gain (g)	Gain/Feed
Basal	445.5[b]	.27
+ .15% K as KCl	561.8[a]	.31
+ .15% K as K_2CO_3	552.2[a]	.30

[ab]Means within column with unlike superscripts differ ($P<.05$).

Table 5. Broiler Weight Gain, Feed Efficiency and Blood pH Values for Experiment 4.

% K	% NH_4Cl	Gain (g)	Gain/Feed	Blood pH
0	0	190.3^{e}	.26	7.47^{a}
0	.1	217.3^{de}	.27	7.37^{bcd}
0	.2	233.5^{cd}	.28	7.35^{bcde}
0	.3	221.4^{de}	.27	7.25^{f}
.05	0	220.8^{de}	.29	7.40^{abc}
.05	.1	212.0^{de}	.29	7.37^{bcd}
.05	.2	246.8^{abcd}	.29	7.35^{bcde}
.05	.3	241.9^{bcd}	.29	7.32^{cdef}
.1	0	228.1^{d}	.30	7.41^{abc}
.1	.1	236.1^{cd}	.30	7.42^{ab}
.1	.2	274.3^{ab}	.31	7.36^{bcde}
.1	.3	223.7^{de}	.29	7.27^{f}
.15	0	279.3^{a}	.30	7.42^{ab}
.15	.1	266.7^{abc}	.29	7.38^{bc}
.15	.2	275.1^{ab}	.30	7.36^{bcde}
.15	.3	236.6^{cd}	.29	7.28^{def}

^{abcdef}Means within a column with unlike superscripts differ ($P<.05$).

Data reported herein substantiate the previous suggestion that acid-base balance of chronically heat stressed broilers is altered which in turn reduces growth rate. Results also establish an interaction between K^+ and H^+. Based on a mean water intake of heat-stressed birds, it appears that the daily K intake needed by heat stressed broilers is 4 to 6 times the .3% specified by the NRC though under thermoneutral conditions, .73% K in the diet was sufficient to maximize growth rate and feed efficiency. NH_4Cl supplementation consistently reduced blood pH, but an elevated blood pH had no adverse effect on gain or feed efficiency when a high level of K was provided.

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CHAPTER IV

POTASSIUM EXCRETION AND REQUIREMENT OF THE 5 TO 8 WEEK-OLD BROILER EXPOSED TO CONSTANT HEAT OR CYCLING HIGH TEMPERATURE STRESS

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Abstract

Four experiments were conducted to determine the effects of ambient temperature-relative humidity stress upon the potassium (K) excretion and potassium requirement of broilers exposed to chronic heat and cycling temperature stress. Potassium excretion, corrected for K intake and body weight, increased by 633% as ambient temperature was increased from a constant 24 C to a constant 35 C. Feed (.73% K) and drinking water (0% K) were supplemented with KCl so that the K requirement could be estimated. In a previous study at thermoneutral temperature, no benefit to K above .73% was observed while in this study a dietary level of 1.5 to 2% K appears necessary to maximize gain under conditions of high ambient temperature stress. This equals about 1.8 to 2.3 g K per chick daily or about .24 to .3% of the drinking water.

(Key words: potassium, potassium excretion, heat stress).

Introduction

Maximal growth rate of the 3 to 7 week old broiler chick occurs at approximately 20 C (McNaughton and Reece, 1982). As ambient temperature exceeds this temperature feed intake and growth rate decline. Squibb et al. (1959) suggested that the reduced growth rate of heat stressed broilers is the result of reduced feed consumption. Many studies have been conducted to identify the most limiting nutrients under heat stress conditions. Fat supplementation of broiler diets reduce the heat increment and can increase feed intake and growth rate. Similar effects are apparent in cooler environments, however (Dale and Fuller, 1979). Protein supplements have generally failed to enhance growth rate though Waldroup et al. (1976) enhanced growth rate by improving the amino acid balance and lowering protein level. Other than manipulating heat increment, little progress has been made in identifying dietary inadequacies. We have observed that broilers exposed to cycling (diurnal) temperature stress eat most of their feed during the cooler portion of the day. Hence, short term nutrient deficiencies could develop during the hotter parts of the day. Such deficiencies could easily be corrected by water supplementation since consumption of water increases with ambient temperature.

Acid-base balance of heat stressed broilers has recently been reported to limit growth rate (Bottje and Harrison, 1985; Teeter et al., 1985a). Teeter and Smith (1985b) observed an interaction between hydrogen ion donors and potassium upon growth rate of broilers housed at 35 C. Growth rate was increased ($P<.05$) by supplementation with either NH_4Cl or KCl. Heat stress precipitates respiratory alkalosis which

reduces competition between hydrogen and potassium for urinary excretion and could thereby increase urinary loss of potassium.

Early studies demonstrated need for potassium in the diet of chicks. Ben Dor (1941) concluded that at least .17% K was needed to achieve maximum growth, while Gillis (1948) stated that the K requirement was from .2 to .24% of the diet. Burns et al. (1953) reported that the requirement was from .23 to .4% of the diet depending on the growth rate of the chick. Leach et al. (1958) concluded that the potassium requirement of the chick increased when a high-protein, high-energy diet was fed. Robbins et al. (1982) reported maximum gain between .21 and .24%. Presently, the National Research Council (1984) recommends that potassium be included in the diet at a level of .3%. As presented, this requirement makes no reference to environmental conditions such as ambient temperature. Deetz and Ringrose (1976) suggested that .6% dietary potassium is a suitable level to meet the total requirement for K by hens subjected to heat stress. If respiratory alkalosis precipitates urinary potassium loss, the potassium needs of poultry must be re-evaluated.

The objective of the following study was to determine if heat stress increases potassium loss and to establish the potassium requirement of the 5 - 8 week old broiler chick exposed to constant heat or cycling ambient temperature stress.

Materials and Methods

Unless otherwise indicated, Arbor Acre x Vantress chicks were raised in floor pens on rice hull litter and fed a corn-soybean meal based diet until they reached 4 weeks of age. On the first day of the

4th week, following an overnight fast, birds were sexed, weighed and randomly allotted to treatments such that each treatment contained 10 or 20 replicates of eight chicks (4 males and 4 females) per replicate. Birds were housed in wire-floored grower batteries containing 61 x 82 cm compartments within a temperature and humidity controlled chamber under continuous tungsten filament lighting. Week 4 was utilized to adapt birds to chamber surroundings, diet (Table 1) and to increase ambient temperature at the rate of 2 C per day until the appropriate temperature setting was reached. Feed and water were continuously available. All data were subjected to analysis of variance using the General Linear Model of the Statistical Analysis System (Barr et al., 1976) and Duncan's Multiple Range test (Duncan, 1955) was used to separate treatment means when a significant treatment difference was detected by analysis of variance (Steele and Torrie, 1960).

Experiment 1.

Fifty 7-week old birds were used to study the effects of elevated temperature on potassium excretion. Birds were randomly allotted to individual 45 x 30 x 45 cm wire cages within the environmental chamber and allowed a seven day adaptation period with environmental conditions maintained at 24 C and 50% RH. Feed consumption was monitored during this adaptation period. Starting on day 8, half of the birds in the chamber were fasted for 48 hours to clear the gastrointestinal tract of digesta, weighed, and force fed a meal equivalent to one third the average daily ad libitum consumption per unit body weight. Total excreta was collected for the next 48 hours. The remaining birds were treated similarly with the exception that ambient temperature and

relative humidity were maintained at 36 C and 60%, respectively. Feed and fecal samples were analyzed for potassium using a Perkin-Elmer 4000 Atomic Absorption Spectrophotometer.

Experiment 2.

This experiment was conducted to estimate the K requirement of broilers exposed to chronic heat stress. Environmental temperature and relative humidity were maintained constant at 35 C and 70% for the 21 day trial. Seven treatments were examined in which added KCl supplied dietary potassium from 0 to 1.2% in .2% increments. All birds were allowed to consume feed and water ad libitum. Feed consumption, weight gain and gain/feed were calculated at the conclusion of the 21 day trial.

Experiments 3 and 4.

To test if the potassium requirement under cyclic temperature conditions would differ from that under constant high temperature stress, two experiments were conducted. In experiment 3, four treatments were evaluated. Each treatment consisted of 20 replicates. Diets contained 0, 1.0, 1.5 or 2% supplemental potassium as KCl in the feed. Environmental temperature was cycled over each 24 hour period from a low of 26 C (8 hours/day) to a high of 36.7 C. Relative humidity was held constant at 70%. The experimental period was 23 days and feed consumption, body weight gain and gain/feed were calculated.

In experiment 4, eight treatments were evaluated, each consisting of 20 replicates. Treatment 1 was the designated heat stress control with no supplemental potassium. Treatments 2 through 7 had potassium in

the water ranging from .06 to .36% in .06% increments. Birds on these treatments were subjected to cyclic environmental temperature and constant humidity as in experiment 3. One additional treatment (treatment 8) was evaluated in which birds were kept under thermoneutral conditions (24 C, 40% RH) with no supplemental dietary potassium. The trial lasted 22 days and feed consumption, water consumption, body weight gain and gain/feed were calculated.

Results and Discussion

Due to restricting intake to 33% of ad lib intake, feed K intakes were similar per unit of body weight for birds at each temperature (Table 2). Birds at 35 C excreted 27.3% more ($P<.05$) K than those at 24 C. Net potassium loss by heat stressed birds was 633% greater ($P<.05$) compared to their thermoneutral counterparts. These results confirm that birds do excrete more potassium during heat stress. Since birds void feces and urine together and the two were not separated, the route for K excretion is not certain though it seems reasonable to assume that the increase is in kidney clearance of K under heat stress similar to that in other species.

In the second experiment, the effects of supplementing broilers maintained at a constant temperature of 35 C with KCl was examined. The basal ration (Table 1) contained .73% K which was previously determined to be adequate in K under thermoneutral conditions (Teeter and Smith, 1985) and considerably above the National Research Council (1984) recommendation of .3% dietary K or .23 g daily K consumption for maximum growth of this age broiler. Results of experiment 2 (Table 3) indicate that weight gain was increased by the addition of K. Birds receiving

Table 1. Composition of the Grower Ration Used in Experiments 1 through 4.

Ingredient	Numerical Name[1]	%
Ground Corn	4-02-931	57
Soybean Meal (44%)	5-04-604	22
Fish Meal (Menhaden)	5-02-009	9
Tallow		5
Ground Alfalfa	1-00-023	2.7
Dicalcium Phosphate	6-01-080	2.5
Calcium Carbonate	6-01-069	.9
Vitamin Mix[2]	--	.4
Salt	6-14-013	.3
Trace Mineral Mix[3]	--	.1
D-L-Methionine	--	.1

[1]Atlas of Nutritional Data on United States and Canadian Feeds.

[2]Mix contained Vit. A, 3,527,360 I.U.; Vit D_3, 1,322,760 I.U.; Vit E, 11,905 I.U.; Vit B_{12}, 3.5 mg; Riboflavin 2,205 mg; Niacin 6614 mg; d-Panthothenic Acid, 7055 mg; Choline, 176,368 mg; Menadione, 291 mg; Folic Acid, 441 mg; Pyridoxine, 882 mg; Thiamine, 882 mg; d-Biotin, 44 mg per Kg.

[3]Mix contained Manganese, 12.0%; Zinc, 8.0%; Iron, 6.0%; Copper, 1.0%; Iodine, .1%; Calcium, 18%.

Table 2. Change in Potassium Status of Birds at Two Temperatures (Experiment 1)[1]

	Temperature		Probability Level
	24 C	35 C	
DM Intake (g/100 g body weight)	2.18	2.19	>.1
Fecal DM output (g/100 g body weight)	.78	.72	>.1
DM retention (%)	64.2	66.8	>.1
Potassium intake (mg/100 g body weight)	21	21	>.1
Potassium output (mg/100 g body weight)	22[b]	28[a]	<.005
Potassium concentration in excreta (%)	2.81[b]	3.98[a]	<.001
Potassium retention (mg/100 g body weight)	-.95[a]	-7.0[b]	<.005

[ab]Values in rows with unlike superscripts differ (P<.05)

[1]Means for 25 birds at each temperature.

Table 3. Gain, Feed Consumption and Gain/Feed of birds supplemented with K^+ in feed (Expt. 2)[1].

K Concentration (%)					
Added	Total Diet	Gain (g/day)	Feed (g/day)	Gain/Feed	K Cons. (g/day)
0	.73	21.10^{b}	83.05^{ab}	$.26^{c}$	$.60^{d}$
.2	.93	24.38^{ab}	76.38^{b}	$.32^{ab}$	$.71^{d}$
.4	1.13	24.57^{ab}	88.00^{a}	$.29^{bc}$	$.99^{c}$
.6	1.33	24.90^{ab}	78.28^{ab}	$.31^{ab}$	1.04^{bc}
.8	1.53	25.62^{a}	76.57^{b}	$.32^{ab}$	1.17^{b}
1.0	1.73	25.81^{a}	82.90^{ab}	$.31^{ab}$	1.43^{a}
1.2	1.93	27.81^{a}	79.23^{ab}	$.35^{a}$	1.53^{a}

^{abcd}Means in columns with unlike superscripts differ ($P<.05$).

[1]Constant temperature of 35 C.

more than .8% supplemental K (or over 1.17 g supplemental K) gained more ($P<.05$) weight than unsupplemented birds. Averaged across K levels, the gain/feed of supplemented birds was greater ($P<.05$) than for birds receiving no additional K despite an increase in intake at .4% K and a drop in feed intake at higher K levels.

In production, cycling temperatures are more common than a constant high temperature. The third experiment was therefore designed to test if K supplementation would alter weight gain of birds under cycling temperature stress. Since response to supplemental K had not reached a plateau at 1.2% K in experiment 2, the levels of supplemental K tested were increased to a high of 2% (Table 4) in this experiment. Birds consuming 1.5% supplemental dietary K (2.34 g supplemental K per day) gained more weight ($P<.05$) than their unsupplemented counterparts. A level of 2% supplemental K did not increase gains above those at 1.5% supplemental K.

Feed intake generally has decreased under heat stress while water consumption increases. Hence, addition of K to drinking water may automatically increase K intake under heat stress conditions. The fourth experiment was conducted to check the benefit of K addition to drinking water. Water supplementation with .24% or above K increased gain ($P<.05$) (Table 5). Potassium toxicity was not evident even at .36%. Water intake was increased with heat stress and was not decreased by addition of KCl to the drinking water. Compared with birds at thermoneutral conditions, birds exposed to heat stress had 31% slower gains. Addition of .36% supplemental K to drinking water decreased this difference to 22%. Deleterious effects of heat stress on growth and feed efficiency can be partially alleviated by K supplementation.

Table 4. Gain, feed consumption and gain/feed of birds supplemented with K^+ in the feed during cycling temperature[1] (Expt. 3).

K Concentration (%)					
Added	Total Diet	Gain (g/day)	Feed (g/day)	Gain/Feed	K Cons. (g/day)
0	.73	41.35[b]	118.13	.36	.73[d]
1.0	1.73	41.43[b]	121.89	.35	1.80[c]
1.5	2.23	44.17[a]	117.26	.38	2.34[b]
2.0	2.73	42.96[ab]	122.74	.37	2.89[a]

[abcd]Values within a column with unlike superscripts differ ($P<.05$).

[1]26 - 35 C.

Table 5. Gain, feed consumption and gain/feed of birds supplemented with K^+ in the water during cycling temperature[1] (Expt. 4).

K Concentration Added (%)	Gain (g/day)	Feed Consumption (g/day)	Water Consumption (ml/day)	Gain/Feed	K Consumption (g/day)	Dietary Equivalent (%)
0	36 68^{d}	127^{ab}	400^{cd}	.29^{f}	.93^{f}	73
06	37 64^{d}	124^{b}	399^{cd}	.31^{e}	1.18^{e}	.95
12	38 59^{cd}	119^{c}	376^{d}	.32de	1.32^{d}	1 11
18	38 36^{d}	115^{cd}	471^{ab}	33^{cd}	1.78^{c}	1.55
24	40.45^{bc}	116^{cd}	418^{bcd}	35^{bc}	1.81^{c}	1.56
30	41.36^{b}	114^{d}	456^{abc}	.36^{b}	2.28^{b}	2.00
36	40 73^{bc}	118^{c}	490^{a}	.35^{bc}	2.67^{a}	2.26
0[2]	53 41^{a}	130^{a}	257^{e}	.41^{a}	.95^{f}	.73

^{abcdef}Values within a column with unlike superscripts differ ($P<.05$)

[1] 26 C - 35 C

[2] Thermoneutral (constant at 24 C)

The effects of potassium deficiency previously reported (Gillis, 1948; Knochel, 1984) are quite variable and detrimental to broiler production. Increased potassium excretion under heat stress is presumably a major factor in predisposing these birds to hypokalemia. The results of these experiments indicate that the level of dietary potassium currently considered adequate for maximum growth and efficiency of birds under thermoneutral conditions must be adjusted upward to maximize growth and efficiency under heat stress conditions. Under the heat stress conditions of these experiments, a dietary level of 1.5 to 2% total K or 1.8 to 2.3 g daily is needed to maximize gain. This intake could be obtained by adding .24 to .3% K from KCl to the drinking water. Water supplementation may be preferred since birds are more likely to drink water than consume feed under heat stress and addition can be instantaneous as weather changes. This avoids the need to formulate and await delivery of a fresh batch of feed for changed weather conditions.

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CHAPTER V

CONSTANT VS. INTERMITTENT SUPPLEMENTATION OF AMMONIUM CHLORIDE AND POTASSIUM CHLORIDE DURING CYCLING TEMPERATURE STRESS IN BROILERS

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Abstract

A 21 day experiment utilizing 560 broiler chicks was conducted to evaluate the effect of constant vs. intermittent administration of potassium chloride and ammonium chloride during cycling high ambient temperature stress. Constant administration of ammonium chloride during cycling temperature resulted in weight gain depression while intermittent supplementation had no effect. Potassium chloride supplementation enhanced gain response irrespective of method of administration. Water consumption is increased by KCl and NH_4Cl supplementation and is affected by time of administration.
(Key words: potassium chloride, ammonium chloride, cycling temperature, constant vs. intermittent).

Introduction

At high ambient temperatures, evaporative cooling through panting is an important avenue for heat loss (Curtis, 1981). As the temperature rises, birds become more and more dependent upon dissipating their body heat by evaporation of water from the lungs (Freeman, 1976), and as a result, the rate of hyperthermic panting is increased. Such conditions precipitate respiratory alkalosis (Jukes, 1971; Kohne and Jones, 1975). Teeter et al. (1985), correlated blood alkalosis with reduced feed consumption, growth rate and increased mortality. That study also indicated correcting the alkalosis by the addition of hydrogen ion donors enabled birds to better cope with the debilitating effects of high temperature stress.

During alkalosis in man, the hydrogen ion concentration within the renal tubular cells declines (Harper et al., 1977), reducing the competition between hydrogen and potassium ions for transport into the distal convoluted tubule and collecting duct and increasing urinary potassium excretion. Alkalosis tends to decrease the plasma potassium concentration in humans (Burnell et al., 1956), while urinary potassium concentration was increased markedly in dogs during blood alkalosis (Simmons and Avedon, 1959). Teeter and Smith (1985b, submitted) found that a significant interaction existed between potassium and hydrogen ion supplementation to birds exposed to chronic high temperature stress. This observation tended to confirm the suggestion that potassium and hydrogen ions are indeed related through metabolism. Observations made by Smith and Teeter (1985a, submitted) indicated that total fecal plus urinary potassium excretion from heat stressed broilers was 27.3% greater than from their thermoneutral counterparts force fed similar

amounts of the same ration. The mechanism whereby humans and dogs increased potassium excretion during alkalosis is therefore probably operative in poultry.

The potassium requirement of broilers exposed to chronic stress was determined to be at least .2% (Smith and Teeter, 1985a submitted) when administered in the drinking water. However, under practical growing conditions, high environmental temperature stress is rarely constant. The normal pattern is for environmental temperatures to fluctuate from a high in mid-afternoon to a low in early morning. The body's requirement for potassium could conceivably be higher during the heat of the day when alkalosis from hyperventilation is severe. If potassium is provided in increased amounts during the time when thermoneutral conditions prevail, hyperkalemia could possibly result, therefore the time of potassium suplementation could be critical. Teeter et al. (1985a) indicated that supplementation of the diet of non-panting heat stressed broilers with 1% NH_4Cl resulted in acidosis.

In view of the interaction that exists between potassium and hydrogen ions, and the observation that acidosis prevails in non-panting hydrogen ion supplemented birds, the following study was done to examine the effects of constant versus intermittent supplementation of hydrogen and potassium ions on the growth of broiler chickens subjected to cycling environmental temperature stress.

Materials and Methods

A 21-day experiment in which five hundered and sixty Arbor Acre x Vantress broiler chicks that had been raised on rice hull litter and fed a corn-soybean meal starter diet (Table 1) during the first three weeks post-hatching, was conducted. On the first day of the fourth week,

Table 1. Composition of the Grower Ration

Ingredient	Numerical Name[1]	%
Ground Corn	4-02-931	57
Soybean Meal (44%)	5-04-604	22
Fish Meal (Menhaden)	5-02-009	9
Tallow		5
Ground Alfalfa	1-00-023	2.7
Dicalcium Phosphate	6-01-080	2.5
Calcium Carbonate	6-01-069	.9
Vitamin Mix[2]	--	.4
Salt	6-14-013	.3
Trace Mineral Mix[3]	--	.1
D-L-Methionine	--	.1

[1]Atlas of Nutritional Data on United States and Canadian Feeds.

[2]Mix contained Vit. A, 3,527,360 I.U.; Vit D_3, 1,322,760 I.U.; Vit E, 11,905 I.U.; Vit B_{12}, 3.5 mg; Riboflavin 2,205 mg; Niacin 6614 mg; d-Panthothenic Acid, 7055 mg; Choline, 176,368 mg; Menadione, 291 mg; Folic Acid, 441 mg; Pyridoxine, 882 mg; Thiamine, 882 mg; d-Biotin, 44 mg per Kg.

[3]Mix contained Manganese, 12.0%; Zinc, 8.0%; Iron, 6.0%; Copper, 1.0%; Iodine, .1%; Calcium, 18%.

following an overnight fast, chicks were sexed, weighed and randomly allocated to treatments such that each treatment contained 10 replicates of eight birds (4 males, 4 females) per replicate. Chicks were housed in wire floored grower batteries which were comprised of 61 x 82 cm compartments, within a thermostatically and humidistatically controlled environmental chamber under continuous tungsten filament lighting. Week 4 was utilized to adapt birds to chamber surroundings. Feed and water were provided ad libitum.

Starting with the first day of the fifth week treatments evaluated (Table 2) were .2% K (as KCl) and .2% NH_4Cl supplied continuously or intermittently in the drinking water, with one treatment devoid of supplementation being maintained as control. The level of potassium and ammonium chloride used had been found to be optimal in alleviating weight gain depression noted in chickens subjected to chronic heat stress (Teeter and Smith, 1985b submitted).

Temperature in the environmental chamber was allowed to cycle between a low of 26 C and a high of 35 C over a 24 hour period by using a combination of several thermostats and a timer. In an attempt to simulate a normal summer day, the temperature was held constant at 26 C for 10 hours (12:30 a.m. to 10:30 a.m.) and then gradually allowed to rise and peak when it reached 35 C (2:30 p.m.) where it was maintained for 2 hours, then gradually declined to the 26 C low. The drinking water of birds on each treatment was provided by thermostatically controlled electric pumps. Water was pumped from one of two containers assigned to each treatment, with each container having either plain tap water, .2% K (as KCl), .2% NH_4Cl, or a combination of both, depending on the treatment designation. Pumps in each container would switch on or

Table 2. Experimental Treatments

Trt.	K (%)	NH_4Cl (%)	Supplementation Method: Constant	Supplementation Method: Intermittent[1]
1	0	0	X	
2	.2	0	X	
3	.2	0		X
4	0	.2	X	
5	0	.2		X
6	.2	.2	X	
7	.2	.2		X

[1]Administered between 31 and 35 C only.

off when the temperature reached 31 C on the upward or downward movement. This arrangement ensured that birds on each treatment had a continuous supply of water and that the potassium and hydrogen ion supplementation was either continuous or intermittent according to the assigned treatment. Throughout the 21 day experimental period, the system was checked twice daily to ensure proper functioning. Feed and water consumption were continually monitored. At the end of the experiment, birds were weighed and body weight gain, feed consumption, water consumption and gain/feed ratio were calculated. All data were subjected to analysis of variance using the General Linear Model of the Statistical Analysis System (Barr et al., 1976) and Duncan's Multiple Range test (Duncan, 1955) used to separate treatment means when a significant test statistic was indicated by the analysis of variance (Snedecor and Cochran, 1967).

Results and Discussion

Males exposed to cycling temperature stress gained more ($P<.05$) than females irrespective of treatment (Table 3). K^+ containing treatments tended to enhance gain regardless of supplementation method. Constant administration of NH_4Cl as the sole additive tended to depress weight gain when compared with unsupplemented birds. This treatment also resulted in an 11% growth depression ($P<.05$) compared with K supplementation. It appears therefore that constant administration of this level of NH_4Cl during cycling temperature stress is somewhat toxic to the birds. Intermittent supplementation of NH_4Cl only, did not affect body weight gain. When K was combined with NH_4Cl, the deleterious effect of constant NH_4Cl administration was removed. Gain

Table 3. Body weight gain.

Supplement		Constant Supp.		Intermittent Supp.	
K (%)	NH_4Cl	Male	Female	Male	Female
0	0	973^{abc}	850^{d}	--	--
.2	0	1042^{a}	902^{bcde}	1063^{a}	853^{de}
0	.2	933^{bcd}	832^{e}	986^{ab}	878^{cde}
.2	.2	1017^{ab}	850^{de}	1013^{ab}	865^{cde}

^{abcde} Means with unlike superscripts differ ($P<.05$).

response to the combination of K and NH_4Cl was not influenced by method of supplementation.

Intermittent administration of K and NH_4Cl to birds resulted in an overall enhancement ($P<.05$) of feed intake (Table 4). This increase in feed intake combined with the lack of gain response decreased ($P<.05$) the gain/feed of these birds. Constant administration of NH_4Cl resulted in feed intake depression ($P<.05$). The decrease observed in gain and feed intake when NH_4Cl was given constantly indicates that the birds were probably acidotic (Teeter et al., 1985; Smith and Teeter, 1985).

Regardless of supplementation method, birds consumed more water when exposed to high temperatures (31-35 C). This could be attributed to the higher temperatures and the longer exposure time. Salt supplementation resulted in an increase ($P<.05$) in water consumption irrespective of temperature or method of administration. Water consumption was higher ($P<.05$) when K and NH_4Cl administration was constant. Birds receiving NH_4Cl consumed less ($P<.05$) water then those receiving either KCl alone or combined with NH_4Cl.

Results of this experiment indicate that time of NH_4Cl supplementation is important during cycling high temperature stress. Supplementing birds with NH_4Cl without considering temperature will result in weight gain depression possibly as a result of birds becoming acidotic. Time of KCl supplementation is unimportant to weight gain and need only be dictated by cost effectiveness.

Table 4. Feed consumption and gain/feed of birds supplemented with NH_4Cl and KCl during cycling temperature stress.

Supplement		Constant		Intermittent	
K(%)	NH_4Cl(%)	Feed (g)	gain/feed	Feed (g)	gain/feed
0	0	2626^{b}	$.36^{a}$	--	--
.2	0	2604^{b}	$.38^{a}$	2965^{a}	$.33^{b}$
0	.2	2302^{c}	$.38^{a}$	2964^{a}	$.33^{b}$
.2	.2	2376^{c}	$.39^{a}$	2982^{a}	$.32^{b}$

^{abc}Values in columns across supplementation methods with unlike superscripts differ ($P<.05$).

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CHAPTER VI

AMMONIUM CHLORIDE AND POTASSIUM CHLORIDE EFFECTS ON BROILER ABILITY TO SURVIVE ACUTE HEAT STRESS

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Abstract

Five experiments were conducted to evaluate the importance of acid-base balance and the effects of NH_4Cl and KCl on the 6 - 8 week old chicks' ability to survive acute heat stress. Blood pH in broiler chickens increased ($P<.01$) from 7.33 (24 C, 60 % relative humidity) to 7.56 upon exposure to acute stress (40 C, 70% relative humidity). Supplementing feed and water with 3 and .5% NH_4Cl respectively held blood pH of stressed broilers below 7.35 and dramatically increased ($P<.05$) survival. Potassium addition (0, .2, .4, .6, .8%) to broiler drinking water resulted in a dose dependent increase ($P<.05$) in survival. Data reported herein indicate that broiler acid-base and potassium balance dramatically influence survival of chicks experiencing acute heat stress.

(Key words: heat stress, acid-base balance, ammonium chloride, potassium chloride, blood pH).

Introduction

The deleterious effects of high ambient temperature-relative humidity environments upon broiler growth rate (Adams et al., 1962; Smith et al., 1983) and survival (Kubena et al., 1972; Reece et al., 1972) are well documented. During the summer of 1980 severe heat stress conditions resulted in broiler mortality losses approaching 20 x 10^6 and retail price elevations of 12 to 20% creating problems for producers and consumers alike.

Though hyperthermic panting is an important means of heat loss (Curtis, 1981) it precipitates respiratory alkalosis (Kohne and Jones, 1975). Correcting heat stress induced alkalosis by addition of NH_4Cl (Teeter et al., 1985a) to broiler diets or CO_2 to drinking water (Bottje and Harrison, 1985), without precipitating non-panting phase acidosis increased ($P<.05$) weight gain by 25%. Teeter and Smith (1985b, submitted) observed a significant interaction between NH_4Cl and KCl supplemention on weight gain suggesting that K as well as acid-base balance limits growth rate of heat stressed broilers. During alkalosis in man, hydrogen ion concentration within the renal tubular cell declines (Harper et al., 1977), reducing competition between hydrogen and potassium ions for transport into the distal convoluted tubules and increases urinary potassium excretion. Huston (1978) reported an inverse relationship between ambient temperature and blood potassium concentrations. Smith and Teeter (1985, submitted) measured K balance of acutely thermostressed chicks and observed a 633% greater K loss in broilers housed at 39 C versus 24 C and a 400% increase in the K requirement for maximal growth rate. Potassium is intricately related

to sarcomere metabolism and function with deficiency leading to muscle weakness and paralysis (Kochnel, 1984). Thermostressed chicks exhibit signs of muscular weakness, paralysis (Gillis, 1948) and frequently at the time of death extreme muscle tetany. These data and observations suggest that broiler heat stress induced prostration may be related to alkalosis and ensuing hypokalemia.

The objective of the following study was to determine if supplementing poultry rations and drinking water with hydrogen and/or potassium ion donors enhances survival of acutely thermostressed chicks.

Materials and Methods

Arbor Acre x Vantress chicks were raised on rice hull litter and fed a corn-soybean meal starter diet during the first five weeks posthatching. On the first day of the sixth week, birds were sexed, weighed and randomly allotted to treatments within a thermostatically and humidistatically controlled environmental chamber under continuous tungsten filament lighting. Unless otherwise indicated birds were housed in wire floored grower batteries (61 x 82 cm compartments). Week six was used to adapt chicks to test rations (Table 1) and chamber surroundings. Feed and water were available on a continuous basis. Where blood data are reported, blood was obtained via wing venipuncture and pH immediately determined utilizing a Corning blood pH/gas analyzer. Analysis of variance was performed on all data using the General Linear Model of the Statistical Analysis System (Barr et al., 1976) and Duncan's Multiple range tests (Duncan, 1955) used to separate treatment means when a significant F statistic was indicated by the analysis of

Table 1. Composition of the Grower Ration.

Ingredient	Numerical Name[1]	%
Ground Corn	4-02-931	57
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Fish Meal (Menhaden)	5-02-009	9
Tallow		5
Ground Alfalfa	1-00-023	2.7
Dicalcium Phosphate	6-01-080	2.5
Calcium Carbonate	6-01-069	.9
Vitamin Mix[2]	--	.4
Salt	6-14-013	.3
Trace Mineral Mix[3]	--	.1
D-L-Methionine	--	.1

[1]Atlas of Nutritional Data on United States and Canadian Feeds.

[2]Mix contained Vit. A, 3,527,360 I.U.; Vit D_3, 1,322,760 I.U.; Vit E, 11,905 I.U.; Vit B_{12}, 3.5 mg; Riboflavin 2,205 mg; Niacin 6614 mg; d-Panthothenic Acid, 7055 mg; Choline, 176,368 mg; Menadione, 291 mg; Folic Acid, 441 mg; Pyridoxine, 882 mg; Thiamine, 882 mg; d-Biotin, 44 mg per Kg.

[3]Mix contained Manganese, 12.0%; Zinc, 8.0%; Iron, 6.0%; Copper, 1.0%; Iodine, .1%; Calcium, 18%.

variance. Correlations were determined as described by Steele and Torrie (1960).

Experiment 1.

This experiment was conducted to evaluate time course changes occurring in blood pH and survival of acutely heat stressed broiler chicks. Ninety six 6 week old male chicks with a mean initial weight of 1030 g were placed in individual 30.5 x 38.1 cm wire cages housed in the environmental chamber. Birds were maintained at 24 C and 55% relative humidity for one week prior to experiment initiation. At experiment initiation all birds were fasted for 6 hours and force fed 1 meal (3% of body weight) containing 0, 1 or 3% NH_4Cl. Immediately following force feeding, environmental conditions were changed to 40 C and 70% relative humidity over a 2.5 h period. Birds were monitored for relative respiration rate by chamber observers. Blood samples were taken from 2 birds per treatment every 10 m and pH determined. Broiler survival was monitored continuously by two observers.

Experiment 2.

This experiment was conducted to evaluate the effects of ammonium chloride and sodium bicarbonate drinking water supplementation on survival of chicks exposed to ideal (24 C, 50% relative humidity) and acutely thermostressed (41 C, 75% relative humidity) environments. Treatments evaluated were .5% ammonium chloride and .75% sodium bicarbonate. Treatments were replicated 6 times with 6 birds per replicate. Parameters monitored included blood pH and bird survival (%).

Experiment 3.

The relationship between heat stress enduced prostration and potassium (K) supplementation of broiler drinking water was examined in this experiment. Birds were randomly allotted to four treatments (0, .2, .4 and .6% K) adminstered 48 hr prior to stress initiation. Ambient temperature was varied from 26 C to 32 C during the adaptation period. Ambient temperature during the test period increased from a low of 26 C to a peak of 38.3 C over 5 hr where it was maintained for 2 hours and then declined over 5 hr. Relative humidity was maintained at 52%. Potassium levels 0 and .6% were replicated 16 times while the .2 and .4% K levels contained 14 replicates of 5 birds each.

Experiment 4.

In an effort to verify the results of experiment 3, a fourth experiment was conducted in which the cycling high temperature stress was monitored over 3 days. Temperature and humidity peaked at 40 C and 54% respectively on each of the three days, under otherwise similar treatment and environmental conditions as described for experiment 3. Water and K consumption were monitored.

Experiment 5.

This experiment was conducted to evaluate potassium drinking water supplementations of 0, .4, .6 and .8%. Ambient temperature was allowed to cycle to 41 C. Potassium levels 0 and .4% were replicated 8 times while the .6 and .8% supplementation levels had 7 replicates each. All replicates contained eight birds per replicate.

Results and Discussion

Exposing broiler chicks to simulated acute summer stress in the first experiment provides data illustrating (Figure 1) the time course of changes occurring in blood pH and survival. During this simulation study both respiration rate as noted subjectively by chamber observers and blood pH increased with stress initiation. Survival across treatment groups fell to 50% as blood pH approached 7.47. Addition of 3% NH_4Cl to the force fed meal delayed pH changes and prolonged survival. At experiment completion pH was inversely correlated with survival (R^2=.97) suggesting that a relationship exists between broiler ability to survive inclement summer stress and acid-base balance.

Exposing broilers to acute thermostress in the second experiment (Table 2) reduced survival from 100% in the thermoneutral environment to just 67%. This vividly demonstrates problems encountered by the poultry industry during periods of summer heat stress. In this study supplementing poultry drinking water with .5% NH_4Cl reduced ($P<.05$) panting phase blood pH of thermostressed birds from 7.44 to 7.24 and increased ($P<.05$) survival to 97%. Addition of sodium bicarbonate to poultry drinking water however increased blood pH to 7.52 and decreased survival to just 56%. Once again pH was inversely correlated (R^2=.98) with broiler survival.

Experiments 3-5 (Table 3) evaluated the impact of broiler drinking water KCl supplementation on survival of heat stressed broilers. Environmental conditions were progressively worsened in the three studies. Supplementing drinking water with KCl resulted in a dose dependent increase in chick survival in each of the 3 experiments. The daily K intake of growing broilers is indeed influenced by ambient

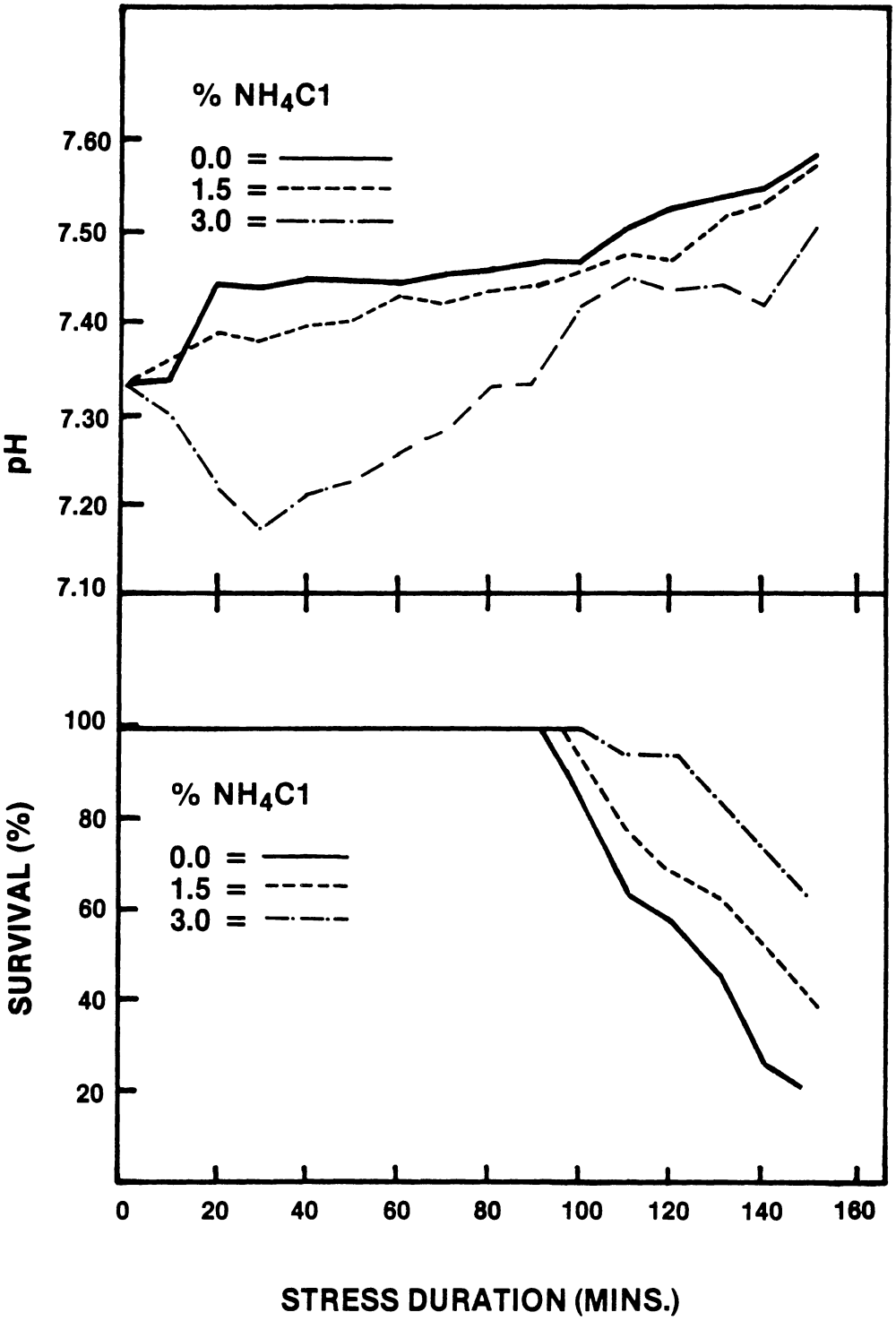

Figure 1. Time course of changes in blood pH and survival(%) in broilers receiving 3 supplemental levels of NH_4Cl in a 40 C and 70% relative humidity environment.

Table 2. Percent survival and blood pH of thermonuetral and acutely thermostressed broilers (Experiment 2).

Treatment	Thermoneutral		Thermostressed	
	Survival(%)	Blood pH	Survival(%)	Blood pH
Untreated water	100	7.29^{a}	62.7^{b}	7.443^{a}
Basal + .5% NH_4Cl	100	7.18^{b}	96.6^{a}	7.241^{b}
Basal + .75% $NaHCO_3$	100	7.30^{a}	55.5^{b}	7.517^{a}

^{ab}Values within a column with unlike superscripts differ ($P<.05$).

Table 3. Survival rate and potassium intake of birds exposed to acute heat stress (Experiments 3, 4 and 5).

	Experiment 3	Experiment 4		Experiment 5
Supplemental K (%)	Survival (%)	K intake (g/bird/day)	Survival (%)	Survival (%)
0	77.3[b]	.43	40.5[b]	15.6[b]
.2	74.3[b]	1.55	44.6[b]	--
.4	90.0[a]	3.41	50.0[b]	26.6[b]
.6	96.2[a]	4.83	77.3[a]	68.4[a]
.8	--	--	--	72.9[a]

[ab]Values within a column with unlike superscripts differ ($P<.05$).

temperature-relative humidity stress as it has been estimated to be .23 g by the National Research Council for good growing conditions, 1.8 - 2.3 g under cycling temperature growth limiting heat stressed environments (Smith and Teeter, 1985) and over 4 g within the life threatening environments reported in this manuscript.

Data reported herein establish a relationship between broiler ability to survive acute heat stress, blood pH and potassium intake. Further, these data demonstrate that survival of acutely heat stressed chicks may be dramatically increased through water supplementation practices.

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CHAPTER VII

SUMMARY AND CONCLUSIONS

The interaction that exists between environmental temperature and production performance of broiler chickens has long been recognized. The problem of reduced growth and decreased efficiency of production during heat stress has been examined by physiologists and nutritionists alike, but only partial solutions have emerged from these efforts. It is becoming increasingly apparent that broiler producers need to alter nutrient inputs in response to environmental change if productivity is to be optimized. The establishment of nutrient requirements for broiler chickens, like most other species, have been carried out in a thermal environment where conditions did not account for the possible climatic extremes. The fact that in practice, environmental conditions are less than ideal, causes animal performance to be below their genetic potential. The relevancy of such stated requirements under less than optimum environmental conditions in which stressful situations are dominant can be questioned.

The likelihood of heat prostration increases dramatically when broiler chickens are exposed to environmental conditions involving both high ambient temperature and high relative humidity. In addition, physiological and behavioral adjustments by the birds, arising from these external stressors affect nutrient intake and its metabolism

within the body, the level of productivity, and the efficiency of feed utilization.

In this study, research was conducted to (1) determine if a potassium X hydrogen ion interaction exists in the chronic heat stressed broiler chicken, (2) establish the potassium requirement of 5 to 7 week old broiler chicks subjected to heat stress, (3) evaluate the effects of therapeutic levels of ammonium chloride and potassium chloride administered continuously or only during heat stress of a diurnal cycling temperature, and (4) evaluate the efficacy of therapeutic levels of ammonium chloride as a hydrogen ion donor and potassium chloride as a potassium donor administered in feed and in the water in enhancing survival of acutely heat stressed broiler chicks.

In the first study, four experiments were conducted to evaluate the effects of heat stress on acid base status and the potassium requirement of broilers. Althouh the basal ration contained more potassium than is required for chicks reared under thermoneutral conditions, heat stressed birds exhibited panting phase blood alkalosis and potassium deficiency symptoms. Supplementing the drinking water with ammonium chloride reduced panting phase blood alkalosis and returned blood pH to normal values, increased live weight gain, and increased feed efficiency. The supplementing of drinking water with potassium also increased live weight gain and feed efficiency, but did not affect blood pH. The suggestion that acid-base balance of chronically heat stressed broilers is altered was substantiated. The results also established that the alteration in acid-base balance precipitated a reduction in growth rate, and that an interaction exists between potassium and hydrogen ion for

growth. It is also possible that the beneficial effects of added hydrogen ions is through potassium conservation.

The four experiments in the second study were conducted to determine effects of ambient temperature-relative humidity stressd on the potassium requirement of broiler chickens exposed to chronic, acute, and cycling temperature stress. Potassium excretion corrected for potassium intake, increased as the environment was changed from a thermoneutral to a thermostressed one. The increased potassium excretion under heat stress is presumeably a major factor in predisposing these birds to hypokalemia. The results of this study indicate that the dietary level of potassium currently considered adequate for maximum growth and efficiency of growth under thermoneutral conditions needs to be adjusted upward to maximize growth and efficiency under heat stress conditions. In addition, water supplementation may be the preferred route of administration since birds are more likely to drink water than consume feed under heat stress, and additions can be instantaneous as climate changes.

The effect of constant versus intermittent administration of potassium chloride and ammonium chloride during cycling high ambient temperature stress was evaluated in the third study. Constant administration of ammonium chloride during cycling temperature resulted in weight gain depression while intermittent supplementation had no effect. Potassium chloride supplementation enhanced gain, regardless of supplementation method. Salt supplementation resulted in increased water consumption irrespective of temperature or supplementation method.

The importance of acid-base balance and the effects of ammonium chloride and potassium chloride on the broiler chicken's ability to

survive acute heat stress was evaluated in the fourth study. Broiler chicken blood pH increased upon exposure to heat stress. Supplementing feed or water with ammonium chloride held blood pH at normal levels and dramatically increased survival.

In the growth studies cited above, exposure to heat stress reduced reduced feed consumption by 30 to 50 percent, and increased water consumption upwards of 100 percent. The addition of high levels of potassium to the drinking water of acutely heat stressed birds in one study substantially reduced feed intake, while at the same time dramatically increasing survival. A subsequent study to define the effects of feed intake on survival indicated that fasting broiler chickens prior to heat stress initiation, allowed body temperature to be maintained at normal levels and increased survival of acutely heat stressed birds, while at the same time water consumption was reduced. The results of all these studies leads one to speculate on the ramifications of these physiological and behavioral changes.

It is possible that nutrient requirements are influenced by environmental temperature changes in two ways. Firstly, the excretion of potassiuim and possibly other electrolytes, is increased under heat stress conditions and secondly, the reduced feed intake accounts for less nutrient intake. Apparently, the decline in feed intake is a result of a shift in energy metabolism and the subsequent physiological consequences.

Heat stressed poultry, like other homeotherms, have to remove the body heat it generates in order to keep its body temperature normal. As ambient temperature rises, the bird has greater difficulty in removing the body heat it generates, and absorbs from the environment, in order

to keep its body temperature from increasing. Convection and radiation losses are minimized because of the hinderance provided by feathers. Latent heat of vaporization of water therefore assumes a very important role in the removal of body heat which results from maintenance energy. The growing broiler chicken generates heat of cellular metabolism for growth. In a heat stressed environment, that heat must also be removed. When attempting to keep the body temperature constant, the bird encounters some difficulty since ambient temperature keeps rising and approaching body temperature. In order to compensate for this, the bird increases its breathing rate to a panting frequency. This mechanism however, also generates more heat from the associated movements. The continued rise in body temperature induces increased cellular metabolism and therefore more heat is produced. If the air temperature is so high that that the bird cannot lose heat to the environment, then something has to fail. The bird probably becomes exhausted in its attempts to remove heat, while the cells may become poisoned from the non-removal by the blood of the by-products of cellular metabolism. The decrease of the blood flow may be a result of heart problems. Thus it is conceivable that heat induced mortality results from failure of the kidneys, lungs, and/or heart as a direct consequence of an override of the hypothalamic set point and brain controlled mechanisms.

During the panting process, blood gases become altered as a result of the rapid elimination of carbon dioxide. This is simply a side result of the bird attempting to remove body heat faster by way of evaporative cooling through respiration. Blood alkalosis ensues and as a consequence, cations and anions are shifted in the blood with the excessive excretion of potassium via the kidneys. With the overall

disruption of the physiology, and the inability to lose heat, the appetite becomes suppressed since eating will generate more heat. The bird is then placed in a situation where it is losing valuable electrolytes while at the same time failing to replace them. Among the multitude of physiological processes that go awry when broiler chickens are subjected to heat stress is the excretion of large quantities of potassium. Potassium is intricately connected with the process of protein synthesis, in addition to being part of a large number of enzyme systems. The weight gain decline experienced by heat stressed broilers is possibly due, in part, to a deficiency of potassium brought about by the excessive urinary excretion of this essential mineral element. This could explain the weight gain response exhibited by potassium supplemented birds under heat stress conditions, if the composition of the gain is protein accretion. Birds subjected to heat stress may be partially dehydrated if the water lost by panting is not replaced. Addition of substances such as potassium salts to the drinking water may cause birds to increase their water intake. Water serves as a heat sink, and in addition to keeping the birds cool, may simply rehydrate the tissues thereby enhancing weight gain.

The environmental nutritionist should consider fasting the birds during the hotter portions of the day and shifting the feeding times to the cooler hours. Admittedly this will work well with layers, however the impact of restricting broiler feed needs to be considered. One could increase the nutrient content of the diet such that nutrient intake remains at the same level as in a thermoneutral environment. Although it is easy to increase the energy content of the diet, a proportional increase in all other nutrients would be needed. When

nutrient intakes are altered, during adverse environmental conditions, depression of production should be alleviated by correspondingly adjusting the dietary levels of the nutrients to compensate for altered daily consumption. From the studies reported in this research effort, it is apparent that one should attempt to maintain the pH of the blood at normal levels during heat stress. This could be achieved by including non-traditional dietary additives which are more acidic. Full success from this practice can only be achieved if feed consumption is adequate. One area explored in this research which offers new and exciting possibilities, is the supplying of nutrients in the water. Increasing the acidity of the water by the addition of mild acids will work. However, since acids are by nature corrosive, the long term effect on housing and equipment must be taken into consideration. Since alkalosis results in increased potassium excretion and reduced intake of other nutrients, the supplying of these nutrients in the water could alleviate the effects of alkalosis rather than the alkalotic condition itself. This would eliminate the need to use the corrosive acids in the watering systems while at the same time achieving the overall objective of the bird receiving aadequate nutrients. One problem presents itself however, that is, the addition of nutrient sources to the drinking water cause a shift in the osmotic pressure. This itself could cause a decline in water consumption, which will then affect estimates of concentrations of nutrients required to ensure adequate intake. The problem of loose fecal droppings would also need to be addressed.

One fact that is clear from the many observations and measurements made while conducting the research presented in this dissertation, is that once the heat stressed bird starts to pant, the usual nutritional

considerations need to be modified. Overall indications are that nutrient intake alone will not solve completely the problems associated with the adverse effects of heat stress. One area that offers exciting possibilities and in which research efforts should be directed, is the identification of chemicals that will either lower body temperature per se, or lower the metabolism of the bird without adversely affecting growth. Basic research to establish accurate and reliable patterns of voluntary feed and water intake in different environments is needed so that nutrient adjustments can more readily be incorporated in management systems. Maintenance energy requirement needs to be known for various degrees of thermal stress. Nutrition-environment models need to be developed so that hot weather requirements can be evaluated. This should take into account any adaptive or compensatory mechanisms. An understanding of the mechanisms and ramifications of acclimatization to environment would enhance the development of nutrition and management strategies to deal with changing environments.

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VITA

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Candidate for the Degree of

Doctor of Philosophy

Thesis: HIGH ENVIRONMENTAL TEMPERATURE STRESS INDUCED ACID-BASE DISTURBANCE AND THE POTASSIUM REQUIREMENT OF BROILER CHICKENS

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