EVALUATION OF SOME DIETARY AND ENVIRONMENTAL FACTORS INFLUENCING ENERGETIC EFFICIENCY IN GROWING BROILERS

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Submitted to the Faculty of the
Graduate College of the
Oklahoma State University
in partial fulfillment of
the requirements for
the Degree of
DOCTOR OF PHILOSOPHY
May, 2003

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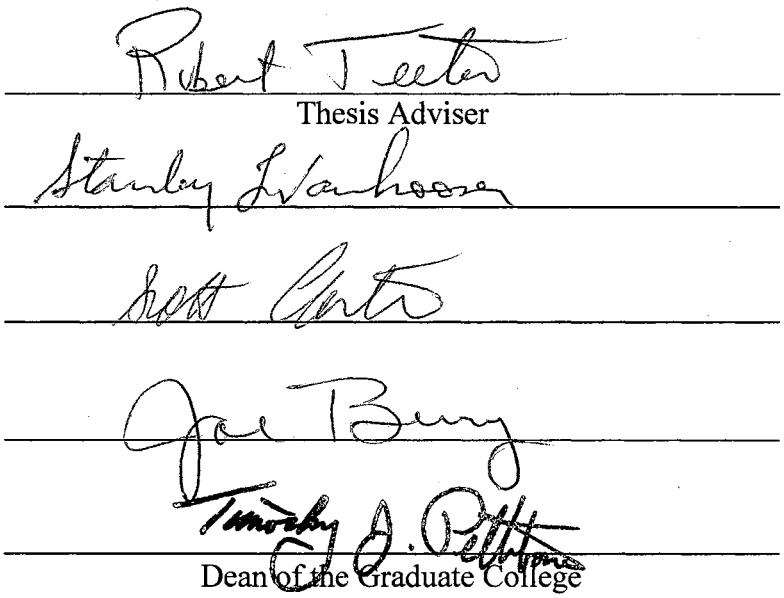
Thesis Approved:



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ACKNOWLEDGEMENTS

I would like to express my sincere gratitude to my major professor, Dr. R. G. Teeter, for his guidance and encouragement throughout my research and writing of this dissertation. I would also like to thank my committee members; Drs. S. L. VanHooser, Scott Carter, and Joe Berry for their valuable advices and suggestions.

Turkish Government, Professor Mustafa Sari, Oklahoma State University Department of Animal Science, ChemGen Inc., and Cobb-Vantress deserves a very special thank you for giving me the chance to pursue my Ph.D. here at Oklahoma State University, providing the facilities, equipment, and financial support.

Thanks are expressed to Drs. Ali Beker and Don Skinner Noble for their assistance and contribution on reviewing and correcting my dissertation chapters. Many thanks are also extended to my former (Aslam Qureshi, Sallee Dixson, Alejandro Corzo, Marsha Mooney, Jeff Swartzlander) and current (Leland McKinney, Diomede Buzingo, Sashidar Yadalam and Adnan Beker) fellow graduate students. Our former (Dr. Farzad Deyhim) and current (Larry Dixson) farm managers and Marcus Dixson also deserves a thank you. Without their help and support, this work simply would not exist.

Special thanks go to the faculty members, fellow graduate students, lab technicians (Fifi Melouk and Kathy Swenson), and secretaries (Lanelle Caldwell and Amanda Beyl) in Animal Science Department. I would like to extend my sincere thanks to Dr. Dave Buchanan for his generous hospitality and unlimited help on my data

analysis throughout my stay here at Oklahoma State University.

My love and gratitude go to my mother, Nuriye Daskiran, my sisters Meral Daskiran and Nural Yagmur, my brother Levent Daskiran, and my brother in law Mehmet Yagmur for the endless support and encouragement through the entire period of my study. Without their support, I could not have finished this study.

Finally, a very special note of thanks is extended to my wife, Yan Zhang, for her continuous support, encouragement, and the strength she gave me and for always being there whenever I needed her.

FORMAT OF THESIS

This thesis presented in the Journal of Applied Poultry Science and Poultry Science style and format allowing for independent chapters to be suitable for submission to scientific journals. Four papers have been prepared from research data collected at Oklahoma State University Oklahoma Agricultural Experiment Station to partially fulfill the requirements for the degree of Doctor of Philosophy. Each paper is complete in itself containing an abstract, introduction, materials and methods, results, discussion, and literature cited section.

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CHAPTER I

INTRODUCTION

Most poultry production in developed countries and an increasing percentage in developing countries occur in industrial systems. In recent years, the largest increases in demand for food of animal origin are for poultry and, as a result, intensive production is likely to increase (CAST, 1999).

Poultry meat production is constantly increasing all around the world due to high consumer demand. Between 1994 and 1999, poultry meat consumption rose by 11 million tones or by over 25% and over half the additional consumption during this time period took place in Asia where consumption has risen by almost 50% (Table 1). As poultry production becomes more and more commercialized in response to increased demand from consumers, satisfying increasing demand for feedstuffs to meet this demand becomes a major issue, especially for developing countries (Huang et al., 1995).

There are several problems the poultry industry is facing in developing countries today. First of all, in many developing countries, most part of the cereal grain production is consumed as human food and therefore availability of cheaper feed sources is a limiting factor for poultry industry (Figure 1). Various alternative feed sources for poultry production in Asia and the Pacific region has been well documented for energy sources (Ravindran and Blair, 1991), plant protein sources (Ravindran and Blair, 1992),

and animal protein sources (Ravindran and Blair, 1993). Especially by-products and underutilized feed sources such as guar gum, copra and palm kernel meal have the potential as alternative feed sources for poultry. However, these by-products usually contain antinutritional factors that restrict the utilization of these ingredients in high quantities in poultry diets and needs to be either processed (heat treatment, autoclaving, etc) or enzyme supplied. Heat stress is also an important problem for Asian poultry industry and therapeutic use is becoming a common practice to increase survivability and performance under chronic heat stress conditions in most Asian countries.

In today's economic climate, efficient feed conversation into broiler tissue is essential for successful poultry production. Over the years, broiler growth rate and feed conversion has been continuously improved through genetic selection and, today, genetics continue to reduce the time needed to grow a four pound broiler by one day per year (Table 2). Broiler energy need for maintenance, protein and fat accretion are dynamic and change continuously during the growth of the bird. Factors other than age, such as ambient temperature and genetic potential also have an impact on energy needs and body composition (Fisher and Wilson, 1974; Chambers et al., 1981; Emans, 1987). Therefore, we can say that geneticists have created a remarkably productive animal, but poultry nutritionists have done their part as well. Research on the nutritional requirements of poultry has resulted in the development of efficient and effective diets.

Efficient feed conversion into broiler tissue is essential for successful poultry production. Sophisticated knowledge of broiler's nutrient requirements, nutrient content of the feed ingredients, and genetic selection has helped us to achieve this goal, however, various dietary and environmental factors influence energetic efficiency in growing birds

and in order to increase the efficiency of tissue accretion and minimize the energy expenditures associated with the metabolism of intermediate and waste products, new methodologies are needed. The aim of this study is to evaluate and quantify the performance and energetic efficiency of broilers under different dietary and environmental stress conditions and to evaluate the effects of dietary enzyme and growth promoters on these birds performance and energetic efficiency. The outcome will enhance our knowledge on the mode of action of these products and may enable us to make proper suggestions to the producers to deal with such stress factors.

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Table 1. Global poultry meat consumption by region 1994 and 1999.

Regions	1994	1999	Volume change 1994-1999	Change 1994-1999
	(million ton)	(million ton)	(million ton)	(%)
Asia	11,215	16,805	5,590	50
North America	14,259	16,502	2,243	16
South America	4,196	5,515	1,319	31
European Union	6,829	7,754	925	14
Middle East	1,178	1,743	565	48
Africa	1,078	1,511	433	40
Eastern Europe	811	1,004	193	24
Oceania	489	586	97	20
Former Soviet Union	2,002	1,564	-438	-22
Total	42,057	52,984	10,927	26

Adapted from Dean (2002) and USDA FAS post reports (2001)

Table 2. Improvements in broiler production over time

Year	Average final body weight, lbs	Market age, weeks	Feed conversion, unit feed : unit gain	% Mortality
1920	2.0	16	5.0	18
1930	2.5	14	4.5	14
1940	3.0	12	4.0	10
1950	3.2	11	3.0	8
1960	3.4	10	2.5	7
1970	3.8	9	2.2	6
1980	4.0	8	2.1	5
1990	4.4	7	2.0	4
2000	5.1	7	2.0	5

Adapted from Lacy and Vest, 2000.

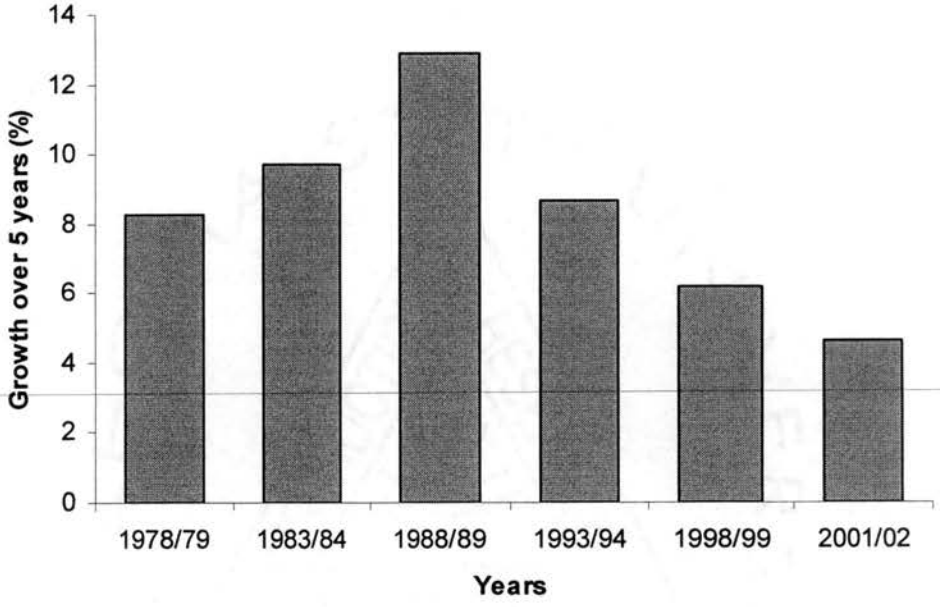

Figure 1. Five year rolling increase for wheat and coarse grains. (Adapted from Dean, 2002)

CHAPTER II

LITERATURE REVIEW

I. ANIMAL RELATED FACTORS

Bird Age

The growth rate of broiler increases from day 0 to 10, reaching a maximum of about 20% per day (Nitsan et al., 1991). The weekly proportionate increase in body weight declines as chicken ages (Marks and Pesti, 1984). Therefore, it would be expected that the relative amount of energy required for growth per unit metabolic size also declines. As the relative amount of energy required for growth declines, any excess energy consumed will result in increased rate of daily fat accretion. At early ages, birds must rapidly adapt to digestion of an exogenous diet in which nutrients are absorbed from the intestine. During this early growth stage, energy is supplied predominantly by carbohydrates in order to achieve their genetic potential. Some of the factors that may influence early growth rate include amount of yolk sack residue, quality and intake of feed and water, pancreatic and intestinal enzyme levels, gastrointestinal tract surface area, nutrient transporters, and overall nutrient digestibility. Also, the change in energy metabolism from lipids to carbohydrates must be considered.

During embryonic incubation, yolk lipid supplies the caloric needs of the bird and is delivered from the yolk sac via the blood stream (Freeman and Vince, 1974). The residual yolk is often very limited quantity after hatching and the energy available from yolk lipids is barely meets the energy requirement of the bird for 24 h (Phelps et al., 1987; Akiba and Murakami, 1995; Ding and Lilburn, 1996). Similarly, more than half of the protein contained in the yolk sack is transferred to the chick during the first two days of life, with virtually no protein remaining after four days (Nitsan et al., 1991).

There is also an indication that delaying placement of hatchlings may compromise immune status (Casteel et al., 1994). Early feeding promotes the maturation of immune system by affecting endogenous hormones or other immunomodulators, and by providing antigens for full differentiation of the primary immune cells. Maternal IgG from the residual yolk provides protection in the first weeks of life. Later, primary (bursa and thymus), and secondary (cecal tonsils, Harderian gland, Meckel's diverticulum, spleen and the lymphoid tissue of the intestinal and respiratory systems) immune organs becomes mature in the weeks following hatch (Dibner, 2001).

Rapid availability and consumption of feed and water is crucial to the development of organ systems and to long term survivability and performance (Fanguy et al., 1980; Pinchasow and Noy, 1993). Oral nutrition requires the presence of digestive enzymes to breakdown macromolecules to a size that can be absorbed. Digestive enzymes first appear during incubation (Moran, 1985) and their levels increase after hatch, however, their concentration per gram of feed intake does not change (Noy and Sklan, 1995). Physical factors in the gastrointestinal system such as length and surface area also plays an important role in limiting early growth in poultry, probably in a larger

extend than the enzyme availability (Nitsan et al., 1991). Research on lines selected for body weight indicates that the low body weight lines show lower relative intestine growth (Nitsan et al., 1991), shorter villi and lower enterocyte density (Uni et al., 1995) than do heavy weight or fast grower lines. Nir et al. (1978) suggests that surface area may limit growth even in older broilers that have been selected for rapid growth. These relationships may be age dependent and selection for heavy body weight may result in increased efficiency of absorption since relative total intestinal mucosa weight actually decreases with selection for higher body weight and feed efficiency.

Body Composition

Tissue composition (fat:lean) of the animal is the other factor that has an impact on the birds energetic efficiency. Teeter and Wiernusz (1994) found that lean birds have elevated oxygen need than the fat ones (3.12 vs 1.21 l/g/bird). Owens et al. (1994) reported that in case of cattle, the mass of fat increased quadratically with empty body weight whereas protein mass increase was linear. In case of broilers, Edwards et al. (1973) found that protein accretion was higher than fat. Therefore, one can imagine that energetic efficiency and metabolism of birds differing in fat:lean ratio may also be different. McLeod et al. (1987) compared the fasting heat production and maintenance energy requirement of lean and fat lines of broilers. The lean lines showed a higher fasting heat production and maintenance energy requirement than the fat lines (996 and 812 KJ/d vs 1058 and 887 KJ/d, respectively). The fat and lean lines had similar energy retention but differ in partitioning of retained energy into carcass fat and protein (37 vs

27 % of retained energy stored as carcass protein and 63 vs 73 % of retained energy stored as carcass fat in lean and fat lines, respectively).

Basal Metabolism

The basal metabolic rate is defined as the heat production of an animal at rest, awake, fasted and housed within a thermoneutral environment. It is the energy needed to sustain the life processes of an animal such as vital cellular activity, respiration and blood circulation. Energy needed for basal metabolism could be estimated as a function of body surface area. Under these conditions the rate of energy metabolism is a function of surface area. This relationship exists because heat loss is closely tied to body surface area (Brody, 1964). Surface area per unit body weight declines with increasing body weight and basal metabolism per unit weight similarly declines. Surface area however, is a difficult parameter to estimate and numerous attempts have been made to relate it to body weight (Brody, 1964). Kleiber (1945) reported that body surface area is exponentially related to body weight and hence the equation generally used to estimate basal metabolic energy needs is body weight in kilogram raised to the power 0.75. Brody (1964) showed that there is lack of direct linear relationship between maintenance energy needs and body weight where he observed a declining surface area/body weight ratio as bird weight increased. He postulated an exponential relationship between maintenance energy need and body weight with an exponent of 0.75 being applicable across species. His estimates, however, were derived from mature birds of different species that were fasted for 48 hours and were in negative energy balance. Applicability of this data to a given species at different stages of growth may be questionable. Metabolic weight for poultry is

commonly reported as body weight to the power 0.66, since this value was suggested by Brody (1964) to give a better estimate when comparing poultry within other species. If the correct power is chosen and body temperature and animal composition are constant, then heat production per unit metabolic weight should be relatively constant.

The basal state is seldom achieved with assurance in animals because of the varying time period required to achieve the post absorptive state and the physical, mental and emotional distress created by experimental conditions. In addition to this under basal conditions, heat energy is produced from various energy sources to offset heat loss and maintain constant body temperature. Misson (1974) found that laying hens required a 3 day exposure to the experimental situation before basal values could be achieved. He also demonstrated that the time required to reach the post-absorptive state was influenced by body weight. His data indicate that 24 hours are required for birds below 2.5 kg and 48 hours for those above this weight. The weekly proportionate increases in body weight declines as chicken ages (Marks and Pesti, 1984). Therefore, it would be expected that the relative amount of energy required for growth per unit metabolic size also declines. As the relative amount of energy required for growth declines, any excess energy consumed will result in increased rate of daily fat accretion. Meltzer (1983) proposed that broilers have two metabolic curves, the first with an exponent of 0.882 to the age of 23-26 days for both sexes and thereafter an exponent of 0.627 for the females and 0.483 for the males. Different investigators have reported that metabolic rate and hence the exponent used to convert bird body weight to metabolic weight is not constant, but is influenced by carcass composition, sex, ambient temperature, and strain (Emans, 1987;

Farrell, 1974; Farrell and Swan, 1977). Ledger and Sayers, (1977) also suggested that prior growth rate might have an impact on basal metabolic rate estimates.

A number of factors were reported to influence the maintenance needs of animals. These include nutritional balance of the diet, body weight, growth rate, strain, environmental temperature, etc. Fasting heat production has been used as an estimate of the animals fasting maintenance requirement by a number of workers. Birkelo et al. (1991) using respiration calorimetry estimated fasting heat production of beef cattle that were under different planes of nutrition in a thermoneutral environment. Animals on the high plane of nutrition (2.2 x maintenance) showed a 7 % increase in their fasting heat production compared to those on low (1.2 x maintenance) plane of nutrition. McLeod et al. (1987) compared the fasting heat production and maintenance energy requirement of lean and fat lines of broilers. The lean lines showed a higher fasting heat production and maintenance energy requirement than the fat lines (996 and 812 KJ/d vs 1058 and 887 KJ/d, respectively). The fat and lean lines had similar energy retention but differ in partitioning of retained energy into carcass fat and protein (37 vs 27 % of retained energy stored as carcass protein and 63 vs 73 % of retained energy stored as carcass fat in lean and fat lines, respectively). Mittelstaedt (1990) determined the maintenance feed required by broilers ranging in weight from 500 to 2500 g maintained on a diet of 2791 kcal ME_n/kg and 21.5 % crude protein (CP) at 24 C and observed that the maintenance feed requirement decreased quadratically as the weight of the birds increased. Emans (1987) reported that chicken maintenance requirement is directly proportional to body protein mass and no energy is required to maintain body lipid.

II. FEED RELATED FACTORS

Diet Form and Composition

The manner in which feed is processed contributes to the partitioning of feed energy into carcass fat and protein. Studies with pelleting have been demonstrated to improve feed intake and to increase fatness (Fisher and Wilson, 1974; Leclercq et al., 1980). Pesti et al. (1983) observed a 23 % increase in abdominal fat upon feeding crumbled low density diet. Jenson (1982) indicated that mash feeding requires more consumption time and energy than pelleted feed.

Composition of the ration particularly calorie to protein ratio and energy density profoundly affects energetic efficiency and body composition of broilers. Increasing calorie to protein ratio in diets increased fat deposition while decreasing calorie to protein ratio decreased fat deposition (Bartov et al., 1974; Farrell, 1974).

Composition of the ration provided to the birds affects the body composition. Research indicates that increasing the energy density of the ration increases abdominal fat pad weight and increasing calorie to protein ratio in broiler diets increases fat deposition while decreasing the calorie to protein ratio has the opposite effect (Bartov et al., 1974). Today's poultry industry utilizes metabolizable energy (ME) as the reference standard for ration formulation. However, ME is not the final energy quanta available to the bird and 40-60 % of ME is lost as heat in growing chicks. Studies examining the impact of treatment on energetic efficiency would ideally report net energy. Net energy values include maintenance and growth with growth potentially partitioned into protein and fat. Yet maintenance energy can constitute a major part of the energy needs of the chicken.

Energy used for maintenance is used to maintain body functions with no energy gain reportedly accounts for approximately 70% of the adult chicken's total energy needs (Hurwitz et al., 1978). A major portion of the maintenance energy expenditure is basal metabolism, accounting for an estimated 85% of the maintenance energy needs (Brody, 1964).

To date, the metabolizable energy (ME_n) system has been accepted as the standard for ration formulation. However, the ME_n system by definition, does not quantitatively predict bird feed energy deposition. Any heat increment change alters ME_n utilization and thereby can affect the cellular energy:nutrient ratios. Alterations in the cellular energy:nutrient ratio may enhance fat deposition. For example, recent and ongoing studies directed at evaluating the ME_n system indicate that cellular energy supply does not necessarily reflect ME_n consumption. The greater heat increment from protein ME_n calories vs. those from starch and fat make low protein diets lipogenic. Oxygen required per unit protein synthesis is 380% greater than that for fat (Teeter and Wiernusz, 1994). Energetic efficiency of ME_n use for tissue gain depends upon numerous variables. Efficiency varies with substrate source, for lipogenesis being approximately 75, 84, and 61% for carbohydrates, fats and proteins, respectively (De Groote, 1969; Chudy and Schiemann, 1971; Hoffmann and Schiemann, 1971). The high availability of fat ME_n for tissue gain, however, requires that fat is used for lipogenesis (Bossard and Combs, 1961). Utilization of protein for tissue energy gain depends upon the biological value of the protein source and should not be constant (De Groote, 1973). Indeed, one could summarize that the bird's energetic efficiency for use of protein or any substrate is

the net result of partitioning consumed substrate energy into maintenance needs verses accretion of protein and fat.

Recommendations for dietary protein concentration for optimum rates of lean tissue accretion range from high (Kubena et al., 1972) to low levels complemented with specific amino acids (Waldroup et al., 1976). Whether the carcass leanness associated with feeding high protein diets is attributable to substrate limitations (amino acids), or due to greater heat production per kcal ME for dietary amino acids carbohydrate and fat is subject to debate. Research conducted at Oklahoma State University by Mittelstaedt (1990) examined the true metabolizable energy (TME) utilization of carbohydrate, protein and fat sources for energy, protein and fat gain. Despite similar TME consumption among the energy supplemented groups, carcass energy was impacted significantly. Total carcass energy gain was 17, 27, and 30% greater for the gelatin, starch, and corn oil groups, than for birds fed the basal diet. Estimated energy gain from the basal ration was similar among the energy supplemented groups due to nearly identical feed consumptions. However, total calories gained differed ($P < 0.05$) across experimental groups with the highest value of 436 kcal/bird observed for the corn oil group verses only 167 kcal/bird for the gelatin. As a result, energetic efficiency varied among the energy supplemented groups. Efficiency of ingredient TME usage for carcass energy deposition averaged 50.0, 39.1, and 19.9%, respectively for supplemental corn oil, starch and gelatin, respectively.

An additional consequence of low protein ME utilization efficiency is that the birds heat load is increased. Elevated heat load has little consequence when birds are housed at or below thermoneutral temperatures. However, if the bird's heat load is

elevated by high ambient temperature stress, without a concomitant increase in heat dissipation, elevated heat load can be devastating (Wiernusz and Teeter, 1993). Belay and Teeter (1992) fed birds various protein levels and calorie/protein ratios. Increasing dietary energy and (or) narrowing calorie-protein ratios by relaxing restrictions on amino acid balance (which necessitated increased dietary protein) significantly impacted bird carcass composition. Improving amino acid balance and lowering dietary crude protein concentration increased survival both in the thermoneutral environment (4.4%) and within the heat stressed environment (10.8%). Lowering crude protein (at adequate amino acid balance) for birds subjected to heat stress can prove beneficial.

Water intake is correlated with feed intake and thus any decrease in water consumption due to failure in the water supply or lack of watering space would results in decreased consumption of feed to a varying extent, depending on the age of the chickens and the degree of water restriction. Clean, fresh water is important to feed intake and conversion, but with contaminated water, the performance of chickens is almost always below average. It is believed that enclosed watering systems improve feed conversion as they protect water form carriers of bacterial contaminants such as dust, littler, feed, and fecal materials.

Litter is the first material that recently placed chicks might consume, before finding feed and water. Therefore it is important to ensure litter is high quality, free from foreign materials, and fresh. If quality litter is not available for the new flock, at least provide quality litter in the brooding area during the first week. Ingestion of contaminated litter can cause irritation of the gut linings of the young chick, resulting in poor nutrient absorption. Identifying the specific cause of decreased feed passage time may be difficult,

as many complex factors need to be considered. In some cases, several factors work together and limit the broiler's ability to digest and absorb feed, resulting in the passage of undigested nutrients. Investigation of the cause requires gross examination of the affected poultry, examination for presence of intestinal parasites, bacterial culture, virus isolation, histopathology, and toxicological testing. It is clear that management practices can play a major role in preventing this problem. Feed passage directly affects the most important broiler economic performance parameters, feed conversion and body weight. When feed passage is observed in broiler houses, evaluating basic management practices may be the most efficient manner to resolve the problem.

Dietary Fiber and Antinutritional Factors

The feed ingredients used in poultry diets are mostly plant origin and they are rich sources of carbohydrates. They are also sources of dietary fiber and various non-starch polysaccharides (NSP), which cannot be digested by endogenous enzymes. In addition, compared with pigs and rats, microbial degradation of dietary fiber in the caecum and colon of poultry appears to be low (Carre and Leclerco, 1985; Longstaff and McNab, 1987; Longstaff et al., 1988).

Dietary fiber affects the length and weight of the gastrointestinal tract (Savory and Gentle, 1976) and there is strong evidence that the differences in weight of visceral organs are highly related to differences in fasting heat production in animals mainly due to dietary fiber supplementation (Koong et al., 1985; Ferrell and Koong, 1986; Koong and Ferrell, 1990).

Non Starch Polysaccharides (NSP), or the presence of anti-nutritional factors (Annison and Choct, 1991; Bedford and Classen, 1993) are the main factors reducing nutrient bioavailability in poultry diets (Figure 1). Non starch polysaccharides are complex carbohydrates found in the structure of plant cell walls with higher molecular weight (Classen and Bedford, 1991; Annison and Choct, 1991). Among NSP, β-glucans are predominantly found in barley and oats, and arabinoxylans (pentosans) are found in wheat, rye, and triticale at higher rates (Classen and Bedford, 1991). In small extent galactose, mannose, uronic acid, and lignin are some of the other components found in poultry feedstuff (Classen and Bedford, 1991).

Beta-mannan (BM) is a NSP, which is found in various feedstuffs and protein concentrates (Table 1). Compared to cereal grains, plant protein concentrates have much more beta mannan in their structure. Studies indicate that the negative effects of BM supplemented to the broiler diets, include reduced feed intake, weight gain and feed:gain ratio (Ray et al., 1982; Furuse and Mabayo, 1996). Copra meal, a feedstuff containing 25-30% BM, was reported to have increased utilization with bacterial mannanase treatment (Teves et al., 1988). Therefore as the dietary proportion of plant proteins increases, one would expect that enzyme supplementation have the potential to improve the nutrient value.

Growth Promoters

Growth promoters such as acidifiers, antibiotics (Table 2), and probiotics have the potential to improve broiler performance under heat stress conditions by altering acid:base status, gastrointestinal tract pH and size and fasting heat production. Acid:base

value of the diet is also an important criteria to reduce the number of pathogen microbial growth in the intestine and maintain a healthy gut microflora. The reduction in bacterial count reduces the disease threat and results in reduced mortality. Also, reduced bacterial count results in decreased gut wall thickness and increased villi length, which will results in improved feed efficiency. Gastro Intestinal Tract (GIT) thickness has also been correlated Basal Metabolic Rate (BMR) and data collected within our laboratory has detected thinner GIT with Virginiamycin (VM) and a 6% reduction in BMR (Belay and Teeter, 1996). Such effect may explain VM effects to moderate HS consequences.

Exogenous enzymes have the potential to improve efficiency of converting feedstuff into broiler tissue. Enzyme supplementation has been widely reviewed (Annison and Choct, 1991; Campbell and Bedford, 1992; Marquardt et al., 1996) and investigated in poultry and results of these studies suggest that the full nutritional value of the many poultry feedstuff has not yet been achieved as improvements in weight gain, feed/gain ratio, and AME value of the diet have been noted (Brenes et al., 1993; Friesen et al., 1992; Annison et al., 1995). Additional benefits include reduced output of excreta including reduced phosphorus, water intake, digesta viscosity, and size of the gastrointestinal tract (Marquardt et al., 1996; Friesen et al., 1992).

A variety of organic and inorganic acidifiers have been evaluated for effects on health and performance of broilers with controversial findings. In some cases increase in broiler performance were noticed (Patten and Waldroup, 1988; Skinner et al., 1991) while in other cases either decrease in feed intake (Cave, 1982; Pritzl and Kienholz, 1973), decrease in nutrient utilization (Izat et al., 1990; Pinchasov and Jensen, 1989a; Furuse and Okumura, 1989) or no change in performance were reported (Maheswari and

Kadirvel, 1996). Organic acids such as lactic, acetic, propionic, and butyric acids, produced by facultative and obligate anaerobic microflora in the small intestine, decrease intestinal pH and are known to provide protection against pathogens. Reduced bacterial counts have been associated with intestinal tract thinning and a declining mucosal surface area (Gordon and Brucknerr-Cardoss, 1961), both of which are correlated with improved bird performance (Coates et al., 1963; Furuse and Yokota, 1984). Gastro-intestinal tract thickness has also been correlated to basal metabolic rate.

III. ENVIRONMENTAL FACTORS

Ambient Temperature

Elevated ambient temperature and relative humidity results in reduced poultry productivity and substantial economic lose. During cycling chronic heat stress (CCHS), productivity of birds is reduced by a complex array of metabolic and physiological responses aimed at maintaining homeostasis. Fast growing broilers are especially vulnerable to high ambient temperatures because of their increased metabolic rate associated with their higher growth rate. A continuous increase in their somatic growth rate and feather development as well as heat increment due to feeding and voluntary activity produces excess body heat.

Various factors affect bird's response to heat stress. Age of bird, size, genetic makeup and history of heat exposure are the main factors, which influence the bird's response to HS (Teeter and Belay, 1996). The chicken body temperature rises above normal when the environmental temperature rises to 30 ^{O}C or above (Romijn and

Lockhorst, 1966). Any variation in environmental temperature out of these limits will have negative impact on productivity. Donkoh (1989) reported that body temperature of the adult birds is normally in the range of 41 to 42 C and it is significantly increased when ambient temperature increased. Individual variation in body temperature was also found greater at higher temperature (Wilson, 1948). Upper and lower critical temperatures will vary depending on several factors, including degree of acclimatization, rate of production, air movement around the animal and relative humidity (Fuquay, 1981). In a more recent review, Teeter and Belay (1996) indicated that the comfort zones for poultry declines from 35 C at hatching to approximately 24 C at 4 weeks of age.

Chronic HS exposure results in not only heavy mortality but also reduced growth rate, egg production, fertility, and hatchability in poultry. Yahav et al. (1996) observed growth retardation after short-term exposure to HS during 1st wk of age, whereas marked increase in mortality was observed at 42-d-old birds when exposed to 35 C. Several studies demonstrated an increase in mortality in broilers exposed to prolonged periods of high temperatures, especially after 4 wk of age (McDougald and McQuistion, 1980; Thaxton and Pardue, 1984; Bottje and Harrison, 1985). Pardue et al. (1985) observed that CCHS leads to increase mortality and reduced growth rate in broilers. Heat stress response is to elevate the body temperature and inability to regulate body temperature (Keshavarz and McDougald, 1981). Heat stress also has a very crucial influence on the reduction of immune status of animals which, not only makes birds more susceptible to disease, but also increases mortality and reduces productivity due to diseases as well.

Poultry do not have the ability to sweat and must rely primarily upon panting (evaporative cooling) to remove surplus deep body heat under heat stress conditions. Birds classically increase respiration rate during heat stress to enhance evaporative cooling even though it results in CO_2 loss and respiratory alkalosis (Bottje and Harrison, 1985; Teeter et al., 1985). As therapeutic measures, drinking water carbonation (Bottje and Harrison, 1985) and acidifier inclusion (Teeter et al., 1985) have been reported to partially counter deleterious heat stress consequences.

Blood plays an important role in the diffusion of body heat. Chickens exposed to acute HS developed hypertension and an increase in cardiac output in the chicken. Donkoh (1989) stated that blood values of red cell counts, packed cell volume, hemoglobin concentration and plasma protein concentration were reduced of birds raised at 30 C and 35 C as compared to birds raised at 20 C. Exposure to heat resulted in an irreversible decrease in blood hematocrit (Yahav et al., 1996). Zhou et al. (1997) observed whole blood viscosity of broilers decrease significantly when they were exposed to a high ambient temperature. The decrease in whole blood viscosity may be advantageous in reducing peripheral resistance and the load on heat, and increasing tissue perfusion and circulatory distribution, including the blood supply to heat exchange surface. Thermal conductivity of skin increases linearly with rate of blood flow in skin (Ohara, 1981).

Heat production of animals for a specific age is minimal at environmental temperatures within the zone of thermoneutrality. Deviation from this zone is accompanied with increased energy expenditure in the form of heat production for the purpose of maintaining body temperature the result is a decrease of energetic efficiency.

Kubena et al. (1972) and Bray (1983) reported that at a moderate temperature, the correlation between temperature and total body fat content is positive.

Compensatory Growth

When growth is suppressed due to stress, it is possible for at least partial recovery if the stress is removed. As an example, high ambient temperature reduces performance of poultry and other livestock species. However, if the stress can be alleviated, potential for catch-up growth is possible.

The phenomenon of compensatory growth has long been recognized as having the potential to have profound effects on the rate of growth and body composition of most animals. Wilson and Osbourn (1960) reported that one of the first references on the subject was in 1908 where beef steers, which had been under nourished subsequently, recovered and reached normal mature weight and height. An animal whose growth has been slowed by nutritional deprivation may exhibit an enhanced rate of growth when realimented. If this exceeds the maximal rate of gain when adequate nutrition has been provided, the animal is said to have undergone compensatory or catch-up growth (McMurtry et al., 1988). Other trials have demonstrated compensatory growth in a number of species including: cattle (Horton and Holmes, 1978; Rompala et al., 1985; Hayden et al., 1993), swine (Robinson, 1964; Prince et al., 1983; Mersmann et al., 1987; Kyriazakis et al., 1991) and poultry (Wilson and Osbourn, 1960; Deaton et al., 1973; Moran, 1979; Plavnik and Hurwitz, 1991; Zubair and Leeson, 1994).

The mechanisms governing compensatory growth have been studied by a number of workers (Wilson and Osbourn, 1960; Winick and Nobel, 1966; Mosier, 1986; Pitts,

1986). Two theories have been proposed to explain how compensatory growth is regulated. First, compensatory growth mechanisms may involve a set-point or reference for body size appropriate for age and that the control resides in the central nervous system (Wilson and Osbourn, 1960; Mosier, 1986). The link between the compensatory growth control and GH release is regulated by photoperiod. Thus, after a period of undernutrition, the body tries to attain a size that is appropriate for age in the shortest possible time (Zubair and Leeson, 1994). According to Mosier (1986), the mechanism for sensing a deficit in body size and for stimulating compensatory growth acceleration remains unknown.

The second theory relates to so called "peripheral control" which suggests that tissues, per se, control body size through cell number or by the total content of DNA. As suggested by Pitts (1986), the number of DNA units is usually the principal determinant of mature size. In studies with adult rats, Pitts (1986) found that nutritional deprivation reduced the size but not the number of DNA units. Because the number of DNA units remained unchanged, it was felt that after nutritional stress, a certain memory mechanism took over to realiment the animal back to its appropriate size for age. If nutritional stress was imposed at too young an age, then the number of DNA units were changed, meaning that realimentation was not as successful, since the memory mechanism was unable to function properly. Winick and Nobel (1966), also working with rats, reported that a reduction in cell number seemed to result in permanent stunting, whereas a reduction in cell size resulted in recovery of normal stature after refeeding.

The pattern of compensatory growth is influenced by the age and maturity of the animal, the severity of prior undernutrition, duration of undernutrition, as well as sex of

the animal and the type of realimentation diet used. During a period of undernutrition animals may be fed at, above or below maintenance energy requirements. Usually the level of restriction imposed is calculated to meet the maintenance energy requirement (Zubair and Leeson, 1994). Plavnik and Hurwitz (1985) restricted broiler chickens to about 40 kcal ME/bird/day, or approximately 35% of the normal ad libitum intake for 2 weeks (7-21 days of age). In spite of this severe feed restriction, broilers gained approximately 4 g/day and so the maintenance energy need was likely overestimated. Upon refeeding, however, growth of the restricted birds exceeded controls and at 7-8 weeks of age the birds had almost completely compensated for the previous loss in body weight. Contrary to these results, other researchers (Calvert et al., 1987; Robinson et al., 1992; Pinchasov and Jensen, 1989b) were unable to demonstrate complete recovery of broilers subjected to similar levels of feed restriction. Wilson and Osbourn (1960) state that undernutrition in the earlier stages of growth is more detrimental to an animal than is restriction at a later stage. Consequently, the age at which an animal is subjected to undernutrition may be as important as the severity of undernutrition. With broilers (depending on the sex of the bird) it is generally recommended that feed restriction start at approximately 6 days of age which usually allows for full recovery of body weight (McMurtry et al., 1988; Zubair and Leeson, 1994). Males and females may respond differently to compensatory growth. For example, male broilers have been shown to have a greater ability to exhibit compensatory growth than do females (McMurtry et al., 1988; Plavnik and Hurwitz, 1991). This is likely due to the higher innate rate of growth of male broilers and their lower deposition of body fat (Fisher, 1984). Similarly, Plavnik and Hurwitz (1985) demonstrated that male, but not female, broilers were able to exhibit

complete compensatory growth when subjected to similar conditions. However, Kyriazakis et al. (1991) found no significant difference in growth rate between either male or female pigs upon realimentation after feeding a low protein diet.

Housing and Space Requirements

Population density has been long known to negatively impact broiler performance (Cravener et al., 1992; Dafwang et al., 1987; Proudfoot et al., 1979; Weaver et al., 1973) and elevated stocking density exacerbates heat stress consequences (Cravener et al., 1992; Pesti and Howarth, 1983; Siegel, 1960).

IV. GENETICS

Genetic variation has long been searched on broiler body composition (lean:fat) and energetic efficiency in fast and slow growing broiler lines. Nir and Lin (1982) conducted an in vitro test and found that chicken of heavy lines show more lipogenic activity than leaner lines. Research on lines selected for body weight indicates that the low body weight lines show lower relative intestine growth (Nitsan et al., 1991), shorter villi and lower enterocyte density (Uni et al., 1995) than do heavy weight or fast grower lines. Nir et al. (1978) suggested that surface area may limit growth even in older broilers that have been selected for rapid growth. These relationships may be age dependent and selection for heavy body weight may result in increased efficiency of absorption since relative total intestinal mucosa weight actually decreases with selection for higher body weight and feed efficiency.

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Table 1. Beta mannan in common feedstuffs.

Feedstuffs	Beta-mannan content (%, as dry wt.)[1]
Palm kernel meal	30-35
Copra meal	25-30
Sesame meal	2.8-3.5
Soy hull	10-12
Soybean meal (48%)	1.30
Soybean meal (44%)	1.50-1.70
Rapeseed meal	0.49
Lupin seed meal	0.42
Peanut meal	0.51
Sunflower seed meal (33%)	0.57
Cotton seed meal	0.36
Rice bran	0.32
Wheat	0.10
Wheat bran	0.07
Corn	0.09
Sorghum	0.09
Barley	0.49
Oats	0.30
Rye	0.69

[1]Based on ChemGen's analysis

Table 2. Modes of action of in-feed antibacterial nutritional additives

Physiological and Nutritional		**Metabolic**	
Gut food transit time	-	Ammonia – toxic amine production	-
Gut wall diameter, length, weight	-	Alpha-toxin production	-
Gut absorptive capacity	+	Mitochondrial fatty acid oxidation	-
Feed Intake	- 0 +	Bacterial cell wall –DNA synthesis	-
Mucosal cell turnover	-	Bacterial protein synthesis	-
Stress	-	Faecal fat excretion	-
Energy retention	+	Liver protein synthesis	+
Vitamin – trace mineral absorption	+	Gut alkaline phosphatase	+
Fatty acid, glucose, calcium absorp.	+	Gut urease	-
Plasma nutrients	+		

Adapted from Rosen, 1995

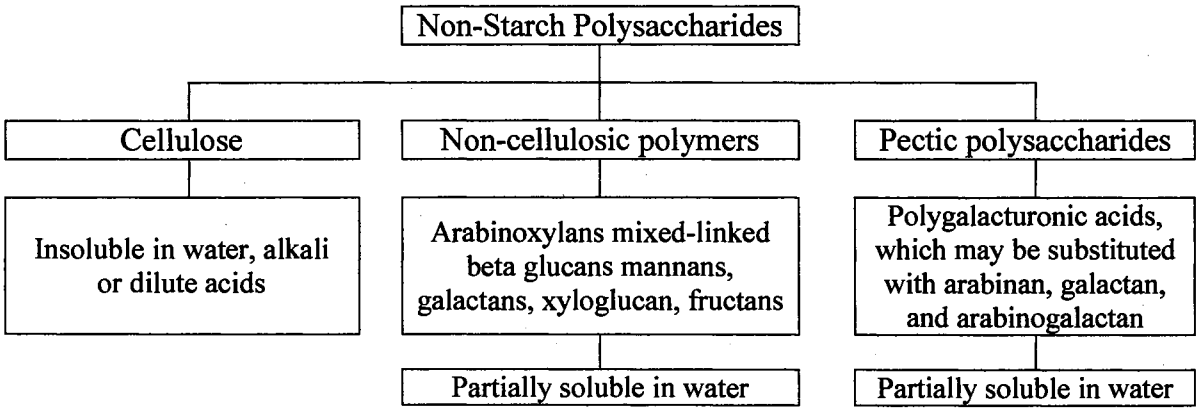

Figure 1. Classification of non-starch polysaccharides (Adapted from Cocht, 2002)

CHAPTER III

EFFECT OF DIETARY ACIDIFICATION ON MORTALITY RATES, GENERAL PERFORMANCE, CARCASS CHARACTERISTICS, AND SERUM CHEMISTRY OF BROILERS EXPOSED TO CYCLING HIGH AMBIENT TEMPERATURE STRESS[1]

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Primary audience: Nutritionists, Physiologists, Plant managers, Flock supervisors

Running title: Heat stress acidifier efficacy

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SUMMARY

A two-phase (starter and grower) experiment was conducted to evaluate efficacy of a commercial phosphoric and citric acid based dietary acidifier (Lucta'cid®) on broiler performance, survivability, serum chemistry, and carcass characteristics during cycling high ambient temperature stress. Starter phase acidifier supplementation (0 and 0.2% of diet) had no impact ($P > 0.1$) on broiler performance. The grower phase examined the two starter phase acidifier exposure histories along with five acidifier inclusion levels (0, 0.05, 0.10, 0.20, 0.30%) under cycling high ambient temperatures. A significant starter phase acidifier history x grower phase acidifier inclusion level interaction was noted on final BW, BW gain, and mortality. Mortality for birds lacking starter phase acidifier exposure declined quadratically ($P < 0.05$) with acidifier level whereas no such response was noted for chicks consuming acidifier during the starter period. Broiler performance and survivability data, combined to estimate productivity per bird placed, indicated a continued mortality driven acidifier history x inclusion level interaction ($P < 0.10$) on hot carcass weight, chilled carcass weight, breast yield, and leg quarter weight. Birds lacking starter phase acidifier and consuming acidifier during grower phase exhibited increased BW and edible meat yield per bird placed. In conclusion, data suggest that grower acidifier supplementation has the potential to reduce economic losses attributable to heat stress when acidifier is absent in the starter period.

Key words: Dietary acidifier, cycling chronic heat stress, broiler, mortality, performance

DESCRIPTION OF PROBLEM

High ambient temperature in conjunction with high relative humidity can result in significant economic loss due to reduced growth rate, feed conversion, and survivability. Birds classically increase respiration rate during heat stress to enhance evaporative cooling even though it results in CO_2 loss and respiratory alkalosis [1, 2]. As therapeutic measures, drinking water carbonation [1] and acidifier inclusion [2] have been reported to partially counter deleterious heat stress consequences. Other studies have demonstrated that dietary supplementation with anions such as Cl and SO_4 depress blood pH [3, 4, 5, 6, 7] and, as such, may moderate heat stress if acidemia can be avoided.

Acidifiers presumably have the potential to improve broiler performance under heat stress conditions by altering acid:base balance. A variety of organic and inorganic acidifiers have been evaluated for their effects on health and performance of broilers with controversial findings [8, 9, 10, 11, 12, 13, 14, 15]. Organic acids such as lactic, acetic, propionic, and butyric acids, produced by facultative and obligate anaerobic microflora in the small intestine, decrease intestinal pH and are known to provide protection against pathogens [16, 17, 18]. Reduced bacterial counts have been associated with intestinal tract thinning and a declining mucosal surface area [19], both of which are correlated with improved bird performance [20, 21, 22, 23]. Gastro-intestinal tract thickness has also been correlated to basal metabolic rate. Data from our laboratory has associated thinner gastro-intestinal tract with virginiamycin supplementation and a 6% reduction in basal metabolic rate. Such effects may explain virginiamycin effects to moderate heat stress consequences reported by Belay and Teeter [24]. Based on these findings, some may speculate that organic acidifiers may moderate heat stress consequences by reducing

gastro-intestinal tract bacterial count, mass, and therefore basal metabolic rate. The study reported herein was conducted to evaluate efficacy of a commercial dietary acidifier for alleviating heat stress consequences on growth and carcass variables, mortality and metabolic effects on serum chemistry.

MATERIALS AND METHODS

A two-phase experiment was conducted to evaluate the effects of a dietary acidifier on growth, feed efficiency, survivability, serum chemistry, and carcass characteristics of broilers reared under cycling temperature stress. Birds had ad libitum access to feed (except as noted) and water throughout the study with lighting under 24 hour fluorescent light. Birds were checked twice daily for mortality with dead birds weighed and sent to a qualified disease laboratory for necropsy to identify the cause of death.

Starter Phase

Starter phase was conducted to examine acidifier inclusion effects on chicks reared in floor pens from hatching through 18 days of age. One thousand four hundred forty (1440) day-old Cobb x Cobb male broilers were obtained from a commercial hatchery, wing banded, allocated into groups of 36 chicks, group weighed, and randomly assigned to 40 floor pens. Starter period treatments included two dietary acidifier levels (0 and 0.2%) in a common diet (Table 1). Chicks were reared according to the breeder recommendations. Each pen contained two hanging tube feeders and eight nipple drinkers. At 18 d, individual BWs and feeder weights were recorded for weight gain and feed conversion calculations.

Grower Phase

At 19 d of age, (following an overnight fast) 960 birds were transferred to a large scale thermostatically controlled environmental chamber complex that has been previously described [25]. Chicks were allocated to compartments such that each compartment contained 3 chicks originating from both starter acidifier levels. Grower phase treatments were formed by acidifier inclusion (0, 0.05, 0.10, 0.20, 0.30%) to the grower ration (Table 1). Each treatment consisted of 32 replicates of six chicks each and that were blocked according to compartment position within the complex. Each 61 x 92 cm compartment contained two feeders, two cup drinkers, and continuous fluorescent lighting. Relative humidity was maintained at a constant 70% ± 5%. Ambient temperature was initially held at 24 C for days 1-2, and then the high temperature was gradually increased (2.5 C/d) to provide a daytime high of 35 C and daily low temperature of 24 C. Cycling ambient temperature consisted of 12 h of 24 C, 6 h of 24-35 C and 6 h of 35-24 C for the cycling temperature stress.

Upon completion of the 22 d grower period, individual bird BW, and compartment feed weights were recorded such that weight gain, feed consumption, and feed:gain ratio could be determined for each compartment. Two birds from each compartment were randomly selected for serum chemistry [26] and carcass yield measurement. Blood sampling was performed in a small room held at room temperature (24 C) before they were transferred for carcass processing. Data were analyzed using ordinary least squares [27]. Starter phase model included acidifier level and block as main effects. Grower phase model included starter phase acidifier level (that is, previous acidifier history), grower phase acidifier level, battery level, and block as main effects

and appropriate interactions. Mean separation was accomplished using Least Significant Difference [28]. Significance implies $P < 0.05$.

RESULTS AND DISCUSSIONS

Acidifier effects on BW, feed conversion, and survivability in the starter phase were lacking ($P > 0.1$, Table 2). In general, the outcome was in agreement with previous studies using organic acids such as malic acid [15] and fumaric acid [10, 14]. However, results of the grower phase indicated grower phase acidifier level x starter phase acidifier history interactions for 40 d BW, 19-40 d weight gain, and mortality (Table 3). As a result, the combined starter phase acidifier history and grower phase acidifier effects upon total bird productivity must be considered.

Starter phase dietary acidifier exposure appeared to determine broiler response to the grower phase acidifier levels (Table 3). As the interaction between the starter phase acidifier history and grower phase acidifier level for live BW and weight gain is likely related to mortality, it will be discussed first. Birds consuming acidifier during the starter phase largely failed to respond to dietary acidifier inclusion during the grower phase whereas an apparent dose dependant mortality reduction occurred for birds lacking starter phase acidifier exposure. Birds not consuming acidifier during starter phase realized a quadratic reduction in mortality ($P < 0.05$) with increasing dietary acidifier inclusion in grower phase. All grower phase acidifier treatment groups, not consuming acidifier during starter phase, exhibited less mortality ($P < 0.05$) than their non acidifier controls. The mortality data is particularly striking as each of the 32 treatment replicates contained equal numbers of starter phase acidifier free and acidifier treated birds.

Previous studies have demonstrated that acidifier use during heat stress has beneficial effects on bird survivability [1, 2]. Generally, such treatments have been considered most effective in drinking water since feed consumption declines during the heat stress episode. High cycling temperature and relative humidity stress results in elevated respiration rate, respiratory alkalosis [1, 2, 25] and profound reduction in the balance of several minerals [29]. Heat stress-induced perturbation of bird mineral balance has been associated with decreased serum Na, Ca, Mg, and inorganic P concentration in broilers [25, 30, 31, 32]. Such results are presumably caused by alteration in acid-base balance. In this study, however, acidifier inclusion in the diet failed to impact serum chemistry values with the exception of phosphorus (Table 4). Birds from 0.1% dietary acidifier treatment exhibited lower serum P values ($P < 0.05$) than rest of the grower phase acidifier consuming treatments, however, no difference was observed between the acidifier free control group and acidifier consuming groups.

A starter phase acidifier history x grower phase acidifier inclusion interaction ($P < 0.05$) was observed for both 40 d BW and 19-40 d weight gain (Table 3). Regardless of starter phase acidifier history, control birds exhibited numerically higher BW and weight gain than their acidifier-consuming counterparts during the cycling ambient temperature in grower phase. Elevated mortality for control birds decreased their stocking density at a greater rate than acidifier-consuming birds. Population density has been long known to negatively impact broiler performance [33, 34, 35, 36] and elevated stocking density exacerbates heat stress consequences [33, 37, 38]. This may explain the acidifier history x grower phase acidifier interaction between control and acidifier-consuming birds during cycling chronic heat stress.

Due to the experimental design employed, it was not possible to examine the interaction between starter phase acidifier history and grower phase acidifier level for feed consumption or conversion. Grower phase feed conversion averaged 2.22 and was within an acceptable range for stressed birds (feed conversion was not adjusted for mortality). Yet, feed intake is an important factor affecting broiler survivability during heat stress as heat production increases with feed catabolism. Mortality and performance data (Table 3) indicate that control birds had slightly higher live and dead BW as compared to their acidifier treated counterparts, suggesting higher feed consumption for the controls. Studies with such dietary organic acids as caprylic and lauric [9], propionic [12] and acetic [12, 39] have reported reduced feed intake. This, however, may not be the case under heat stress condition as observed in this reported study.

Grower phase (40 d) hematocrit readings and serum calcium, potassium, magnesium, sodium, chloride, and potassium concentrations are presented in Table 4. At 40 d of age, blood samples were taken from two randomly selected birds representing both starter phase acidifier history. Lack of treatment differences on 40 d serum chemistry may be attributed to the brief time birds were held at 24 C. This short time (2 h) at 24 C may have allowed birds to return to a normal state. No acidifier or acidifier history effects were observed for blood hematocrit and serum calcium, magnesium, sodium, chloride, and potassium. Additionally, no dietary acidifier effects were observed on serum P between acidifier free control group and acidifier consuming groups ($P > 0.05$).

The 40 d hot carcass weight, dressing percentage, chilled carcass weight, breast yield, percent breast yield:chilled carcass weight ratio, leg quarter weight, percent leg

quarter weight:chilled carcass weight ratio, and abdominal fat pad weight data are presented in Tables 5 and 6. No acidifier or acidifier history effects were observed for 40 d dressing percentage. Significant acidifier x acidifier history effects were noted for chilled carcass weight, breast yield, and leg quarter weight. In general, 40 d BW (Table 3) and dressing percentages (Table 5) were the principle factors driving carcass traits and were similar in most cases. Control birds consuming acidifier during starter phase and 0.2% acidifier treatment with no acidifier supplementation during starter phase had higher carcass traits whereas 0.2% acidifier treatment consuming acidifier during starter phase and 0.1% acidifier treatment with no starter phase acidifier supplementation had lower carcass traits than rest of the treatments ($P < 0.05$). Birds consuming acidifier during starter phase had lower percent leg quarter weight:chilled carcass weight ratio ($P < 0.05$) and tend to have higher percent breast yield:chilled carcass weight ratio ($P = 0.0790$). When the data are adjusted using 40 d BW as covariant, no dietary impact on hot carcass weight, dressing percentage, and chilled carcass weight observed ($P > 0.1$), suggesting that BW is the principle driving factor for the yield data. Leg quarter weight was lower for birds consuming dietary acidifier in starter phase compared to starter control birds ($P < 0.05$). Similar trends were observed on percent leg quarter weight:chilled carcass weight ratio and percent breast yield:chilled carcass weight ratio. Neither acidifier nor acidifier history impacted dressing percentage or abdominal fat pad in both analyses ($P > 0.1$).

Total carcass component yields for each experimental unit were estimated by combining performance as survivability data (Table 3) and carcass data (Table 5) as:

Part Yield = [(# of live birds x mean body weight)] x [(mean part yield / (mean hot carcass weight x 100 / dressing percentage))]

representing the effects of both of starter phase acidifier history and grower phase acidifier levels on carcass traits. Total parts yield favored dietary acidifier supplementation. The impact appears driven by mortality reduction as the body weight covariance failed to separate mass yield estimate. Nonetheless, birds lacking acidifier in their diet during starter period exhibited quadratic increases in total part output reflecting the trend on their survivability under heat stress conditions. Control and starter phase acidifier-consuming birds had similar ($P > 0.1$) edible meat yield.

All animals constantly adjust their bodily states according to changes occurring in their environment in an apparent effort to maintain metabolic homeostasis. If the stress factor is removed, or the resulting consequences appropriately handled via adaptive mechanisms [40], the animal survives and may exhibit some level of productivity. Studies have demonstrated that chronic metabolic acidosis mediated by NH_4Cl supplementation via drinking water in rats [41, 42] and rabbits [43] increases the kidney's capacity for H^+ secretion and HCO_3^- absorption. This may also explain the better survivability of birds lacking acidifier supplementation during starter phase. Presumably, early acidifier exposure through the diet mediated an adaptation to the dietary acidifier by improving the kidney's capacity for H^+ secretion and HCO_3 retention, which subsequently reduced the therapeutic activity of acidifier when needed to offset the heat stress mediated respiratory alkalosis later in life. These data suggest that acidifier supplementation to the grower phase birds has the potential to reduce economic losses

attributable to heat stress if high ambient temperature is anticipated by the broiler producer.

CONCLUSIONS AND APPLICATIONS

1. Compared to control birds, birds lacking starter phase acidifier exposure responded to dietary grower phase acidifier inclusion with higher survivability and similar carcass yield data during grower phase chronic cycling heat stress exposure.
2. Total carcass yield data favored dietary acidifier supplementation, mainly due to increased survivability.
3. Dietary acidifier supplementation should be considered when a heat stress episode is anticipated by the broiler producer as it has the potential to reduce economic losses attributable to heat stress. However, the response is appeared to be tempered by acidifier exposure when chicks lack the heat stress condition.

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44. Technical and financial help provided by Lucta SA, 08170 Montornés del Vallès, Barcelona, Spain is acknowledged.

Table 1. Composition of starter and grower basal diets

Ingredients and composition	Starter diet, %	Grower diet, %
Corn, ground	59.00	62.51
Soybean meal	32.80	29.08
Vegetable fat	2.82	3.90
Fish meal, menhaden	2.00	1.00
Dicalcium phosphate	1.21	1.00
Calcium carbonate	1.42	1.78
Salt	0.38	0.30
DL-Methionine	0.21	0.23
L-Lysine	0.047	0.085
Vitamin premix[A]	0.05	0.05
Mineral premix[B]	0.05	0.05
Amprolium®	0.025	0.025
Choline chloride	0.05	0.04
Selenium premix, ppm	10	19
Calculated analysis		
ME, kcal/kg	3080	3175
CP	22.65	20.60
Methionine	0.59	0.57
Lysine	1.32	1.20
Calcium	0.96	1.00
Total phosphorus	0.75	0.59

[A]Supplied per kilogram of diet: vitamin A, 38,500 IU; vitamin D_3, 11,000 IU; vitamin E, 55 IU; vitamin B_{12}, 0.066 mg; riboflavin, 33 mg; niacin, 165 mg; d-pantothenic acid, 55 mg; menadione, 11 mg; folic acid, 3.3 mg; pyridoxine, 13.75 mg; thiamin, 6.66 mg; d-biotin, 0.28 mg.
[B]Supplied per kilogram of diet: manganese, 120 mg; zinc, 100 mg, copper, 10 mg, iodine, 2.5 mg; calcium, 135 mg.

Table 2. Effects of dietary acidifier supplementation on d 19 BW, feed:gain ratio, and mortality

	Variables		
Acidifier level, %	d 19 BW, g	FCR[A], d 0-19	Mortality, %
0	663.6	1.418	1.250
0.2	667.1	1.419	1.111
SEM	3.110	0.008	0.013
Source of variation		probability[B]	
Acidifier	NS	NS	NS

[A]Feed conversion ratio expressed as g feed:g gain
[B]NS = Not significant (P > 0.1)

Table 3. Effects of dietary acidifier supplementation and starter phase acidifier history on grower phase initial (19 d) and final (40 d) BW, 19-40 d BW gain, grower phase mortality and feed conversion ratio (g feed:g gain)

Treatments		Variables						
					Mortality			
Acidifier history[A]	Acidifier, %	d 19 BW, g	d 40 BW, g	d 19-40 BW gain, g	%	n	dead BW	FCR[BC], d 19-40
0	0	676.0	1828^{ab}	1154^{a}	18.90^{a}	18	1431	
0	0.05	680.6	1797^{abc}	1116^{a}	9.37^{bcde}	9	1131	
0	0.1	681.9	1741^{c}	1056^{b}	7.29^{cde}	7	1151	
0	0.2	684.0	1804^{ab}	1120^{a}	6.25^{de}	6	1111	
0	0.3	681.4	1821^{ab}	1139^{a}	5.21^{e}	5	1318	NT
1	0	678.5	1834^{a}	1155^{a}	14.42^{abcd}	14	1336	
1	0.05	681.3	1805^{ab}	1123^{a}	9.37^{bcde}	9	1182	
1	0.1	682.9	1833^{a}	1150^{a}	16.67^{ab}	16	1244	
1	0.2	675.5	1773^{bc}	1100^{ab}	12.50^{abcde}	12	1277	
1	0.3	685.5	1825^{ab}	1140^{a}	15.62^{abc}	15	1316	
SEM		5.63	20.4	19.5	3.16			
Acidifier								
0		677.2	1831	1154	16.67	32	1389	2.323
0.05		681.0	1801	1120	9.37	18	1156	2.171
0.1		682.4	1787	1103	11.98	23	1216	2.195
0.2		679.8	1788	1110	9.37	18	1222	2.228
0.3		683.5	1823	1140	10.42	20	1316	2.165
Acidifier history								
0		680.8	1798	1117	9.41	45	1272	NT
1		680.7	1814	1133	13.72	66	1277	
Source of variation		probability[D]						
Acidifier		NS	NS	0.0709	NS			0.4121
Acidifier history		NS	NS	NS	0.0313	-	-	NT
Acidifier x Acidifier history		NS	0.0377	0.0408	0.0216			

[abcde]Means within columns with different superscripts differ significantly ($P < 0.1$)

[A]Acidifier history "0" denotes birds were not provided acidifier during starter phase whereas acidifier history "1" denotes they were provided 0.2 % acidifier in starter diet

[B]FCR = Feed conversion ratio (g feed:g gain)

[C]NT = Experimental design precludes statistical testing

[D]NS = Not significant ($P > 0.1$)

Table 4. Effects of dietary acidifier supplementation and acidifier history on serum hematocrit, calcium, potassium, magnesium, sodium, chlorine, and potassium

Treatments		Variables						
Acidifier history[A]	Acidifier, %	Hematocrit, %	Ca, mg/dl	P, mg/dl	Mg, meq/l	Na, mmol/l	Cl, mmol/l	K, mmol/l
0	0	28.72	9.35	7.19	2.02	149.8	104.4	6.46
0	0.05	28.60	9.57	7.60	2.03	149.8	103.6	6.49
0	0.1	28.91	9.05	7.97	2.00	150.3	104.2	6.61
0	0.2	29.05	9.55	7.71	2.00	149.3	103.5	6.79
0	0.3	29.80	9.19	7.58	2.01	149.9	104.8	6.63
1	0	29.24	9.46	7.63	2.02	150.4	105.2	6.54
1	0.05	28.87	9.29	7.40	1.97	149.8	104.9	6.37
1	0.1	28.87	9.83	7.24	2.05	149.4	104.0	6.29
1	0.2	27.83	9.42	7.51	2.04	150.0	104.0	6.75
1	0.3	29.25	9.39	7.48	2.05	149.3	102.6	6.65
SEM		0.65	0.22	0.17	0.03	0.50	0.86	0.17
Acidifier								
0		28.75	9.40	7.41[ab]	2.02	150.1	104.8	6.50
0.05		28.73	9.43	7.50[a]	2.00	149.8	104.3	6.51
0.1		28.89	9.44	7.10[b]	2.03	149.9	104.1	6.45
0.2		28.44	9.49	7.61[a]	2.02	149.6	103.8	6.77
0.3		29.52	9.29	7.53[a]	2.03	149.6	103.7	6.65
Acidifier history								
0		28.92	9.34	7.41	2.01	149.8	104.1	6.63
1		28.81	9.48	7.45	2.03	149.8	104.1	6.52
Source of variation		probability[B]						
Acidifier		NS	NS	0.0301	NS	NS	NS	NS
Acidifier history		NS	NS	NS	NS	NS	NS	NS
Acidifier x Acidifier history		NS	NS	NS	NS	NS	NS	NS

[ab]Means within columns with different superscripts differ significantly ($P < 0.05$)

[A]Acidifier history "0" denotes birds were not provided acidifier during starter phase whereas acidifier history "1" denotes they were provided 0.2 % acidifier in starter diet

[B]NS = Not significant ($P > 0.1$)

Table 5. Effects of dietary acidifier supplementation and acidifier history on 40 d hot carcass weight, dressing percentage, chilled carcass weight, breast yield, percent breast yield:chilled carcass weight, leg quarter weight, percent leg quarter weight:chilled carcass weight, and abdominal fat pad weight

Treatments		Hot carcass	Dressing	Chilled carcass	Breast		Leg quarter		Abdominal fat
Acidifier history[A]	Acidifier, %	weight, g	percentage, %	weight, g	yield, g	% chill	weight, g	% chill	pad weight, g
0	0	1265^{abc}	73.5	1311^{abc}	267^{bcd}	20.48	442^{abc}	33.73	14.6
0	0.05	1249^{bc}	72.9	1297^{bc}	266^{cd}	20.64	437^{abc}	33.65	16.3
0	0.1	1229^{c}	73.2	1277^{c}	262^{d}	20.52	425^{bc}	33.32	14.5
0	0.2	1310^{a}	73.6	1359^{a}	286^{a}	21.20	450^{a}	33.17	16.0
0	0.3	1279^{abc}	73.5	1327^{abc}	278^{abcd}	20.82	438^{abc}	33.05	16.4
1	0	1302^{ab}	73.8	1353^{ab}	285^{a}	21.15	444^{ab}	32.88	17.2
1	0.05	1278^{abc}	73.4	1324^{abc}	280^{abc}	21.29	443^{ab}	33.58	14.8
1	0.1	1291^{ab}	73.5	1343^{ab}	279^{abcd}	20.96	446^{a}	33.15	15.2
1	0.2	1251^{bc}	73.2	1298^{bc}	265^{cd}	20.48	423^{c}	32.59	14.1
1	0.3	1281^{abc}	73.3	1325^{abc}	283^{ab}	21.42	433^{abc}	32.70	16.0
SEM		20.1	0.26	20.4	6.3	0.301	7.1	0.273	1.01
Acidifier									
0		1284	73.7	1332	276	20.82	443	33.30^{ab}	16.0
0.05		1264	73.2	1311	273	20.97	440	33.61^{a}	15.5
0.1		1261	73.3	1310	270	20.74	435	33.24^{ab}	14.9
0.2		1281	73.4	1329	276	20.84	436	32.88^{b}	15.1
0.3		1280	73.4	1326	281	21.12	436	32.87^{b}	16.2
Acidifier history									
0		1267	73.3	1314	272	20.73	438	33.39^{a}	15.6
1		1281	73.4	1329	278	21.06	438	32.98^{b}	15.5
Source of variation					probability[B]				
Acidifier		NS	NS	NS	NS	NS	NS	0.0382	NS
Acidifier history		NS	NS	NS	0.0924	0.0790	NS	0.0208	NS
Acidifier x Acidifier history		0.0421	NS	0.0269	0.0100	NS	0.0173	NS	NS

[abcd]Means within columns with different superscripts differ significantly ($P < 0.05$)

[A]Acidifier history "0" denotes birds were not provided acidifier during starter phase whereas acidifier history "1" denotes they were provided 0.2 % acidifier in starter diet

[B]NS = Not significant ($P > 0.05$)

Table 6. Effects of dietary acidifier supplementation and acidifier history on 40 d hot carcass weight, dressing percentage, chilled carcass weight, breast yield, percent breast yield:chilled carcass weight, leg quarter weight, percent leg quarter weight:chilled carcass weight, and abdominal fat pad weight (d 40 BW is used as covariant)

Treatments		Hot carcass	Dressing	Chilled carcass	Breast		Leg quarter		Abdominal fat
Acidifier history[A]	Acidifier, %	weight, g	percentage, %	weight, g	yield, g	% chill	weight, g	% chill	pad weight, g
0	0	1275	73.5	1321	268	20.48	445	33.71	14.8
0	0.05	1266	73.0	1314	267	20.64	442	33.62	16.6
0	0.1	1273	73.4	1321	272	20.52	439	33.24	15.1
0	0.2	1274	73.5	1323	278	21.20	439	33.24	15.5
0	0.3	1275	73.5	1323	274	20.82	437	33.06	16.3
1	0	1279	73.7	1330	277	21.15	437	32.92	17.0
1	0.05	1272	73.4	1318	277	21.29	442	33.59	14.7
1	0.1	1272	73.4	1324	273	20.96	439	33.19	14.9
1	0.2	1272	73.3	1320	269	20.48	429	32.55	14.4
1	0.3	1270	73.2	1314	277	21.42	430	32.72	15.8
SEM		4.3	0.25	4.6	4.1	0.297	7.1	0.272	0.98
Acidifier									
0		1277	73.6	1325	273	20.82	441	33.32[ab]	15.9
0.05		1269	73.2	1316	272	20.97	442	33.60[a]	15.6
0.1		1273	73.4	1322	272	20.74	439	33.22[ab]	15.0
0.2		1273	73.4	1322	274	20.84	434	32.90[b]	15.0
0.3		1273	73.4	1319	276	21.12	433	32.89[b]	16.1
Acidifier history									
0		1273	73.4	1320	272	20.73	440[a]	33.37[a]	15.7
1		1273	73.4	1321	275	21.06	435[b]	32.99[b]	15.4
Source of variation					probability[B]				
Acidifier		NS	NS	NS	NS	NS	0.0848	0.0486	NS
Acidifier history		NS	NS	NS	NS	0.0790	0.0361	0.0282	NS
Acidifier x Acidifier history		NS	NS	NS	NS	NS	NS	NS	NS

[ab]Means within columns with different superscripts differ significantly ($P < 0.05$)

[A]Acidifier history "0" denotes birds were not provided acidifier during starter phase whereas acidifier history "1" denotes they were provided 0.2 % acidifier in starter diet

[B]NS = Not significant ($P > 0.1$)

Table 7. Effects of dietary acidifier and acidifier history on edible meat yield parts (hot carcass weight, chilled carcass weight, leg quarter weight, breast yield, and abdominal fat pad weight) of each experimental unit (numbers reflects the estimated total yield of live birds from each compartment)

Treatments		Hot carcass	Chilled carcass	Leg quarter	Breast	Abdominal fat
Acidifier history[A]	Acidifier, %	weight, g	weight, g	weight, g	yield, g	pad weight, g
0	0	3236	3354	1131	686.7^{c}	37.24^{f}
0	0.05	3544	3680	1237	759.2^{abc}	46.12^{abc}
0	0.1	3573	3712	1236	761.8^{abc}	42.83^{bcd}
0	0.2	3737	3879	1287	818.7^{a}	46.20^{abc}
0	0.3	3808	3951	1306	818.8^{a}	48.20^{a}
1	0	3512	3649	1199	769.9^{abc}	47.41^{ab}
1	0.05	3606	3735	1254	795.2^{ab}	41.92^{cdef}
1	0.1	3452	3589	1190	762.6^{abc}	40.62^{def}
1	0.2	3393	3523	1149	719.1^{bc}	37.34^{ef}
1	0.3	3389	3506	1147	750.5^{abc}	42.09^{cde}
Acidifier						
0		3374	3502	1165	728.4	42.32
0.05		3575	3708	1246	777.2	44.02
0.1		3513	3651	1213	762.2	41.72
0.2		3565	3701	1218	768.9	41.77
0.3		3599	3729	1227	784.7	45.14
Acidifier history						
0		3579	3715	1240	769.1	44.11
1		3471	3601	1187	759.5	41.88
Source of variation		probability[B]				
Acidifier		NS	NS	NS	NS	NS
Acidifier history		NS	NS	0.0934	NS	0.0414
Acidifier x Acidifier history		0.0890	0.0807	0.0944	0.0206	0.0001

[abcdef]Means within columns with different superscripts differ significantly ($P < 0.05$)

[A]Acidifier history "0" denotes birds were not provided acidifier during starter phase whereas acidifier history "1" denotes they were provided 0.2 % acidifier in starter diet

[B]NS = Not significant ($P > 0.1$)

CHAPTER IV

Comparative Effects of Betaine, Electrolytes, and Ambient Temperature on General Performance, Compensatory Gain, Basal Metabolic Rate and Body Composition of Male Broilers[1]

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Section: Metabolism and Nutrition

Running head: Heat stress, betaine, electrolytes, and compensatory gain

Abbreviation list: HS: Heat stress; CCHS; Cycling chronic heat stress; BMR: Basal metabolic rate; TN: Thermoneutral; FCR: Feed conversion ratio

Full-Length Paper _____X_____ Research Note: _____

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ABSTRACT Two experiments were conducted to evaluate the effects of electrolytes and betaine on overall performance, survivability, basal metabolic rate and body composition of male broilers exposed to cycling chronic heat stress (CCHS). Experiment 1 was conducted to evaluate the effects of betaine and electrolytes on BW, feed conversion ratio (FCR), and survivability of male broilers raised under cycling chronic heat stress conditions from 19 to 47 d of age. Electrolytes did not impact final BW whereas birds provided betaine via drinking water had a tendency ($P = 0.0642$) to improve the d-47 BW. A significant betaine effect was observed on broiler survivability during this trial ($P < 0.05$). Experiment 2 was conducted to evaluate the effects of betaine and electrolytes on the BW, BW gain, basal metabolic rate (BMR), and body composition as well as to evaluate their effects on broiler catch-up or compensatory growth. Birds were transferred from cycling chronic heat stress environment to thermoneutral environment at 33 d of age and their subsequent performance, BMR, and body composition data were compared with thermoneutral and heat stress birds. Heat stressed birds had significantly higher body fat and lower lean tissue as compared to their TN counterparts at d 33, 37, and 41. Environment also had significant impact on bird's BMR heat production at d 33, 37, and 41. Basal metabolic rate heat production was lower for the heat stressed birds with the exception of d 46. When the birds were removed from heat stress environment at d 33, they had higher overall body weight gain and BMR ($P < 0.05$), particularly during first week.

(*Key words*: broiler, heat stress, compensatory gain, betaine, electrolytes, survivability)

INTRODUCTION

Elevated ambient temperature and relative humidity results in reduced poultry productivity and substantial economic lose. During cycling chronic heat stress (CCHS), productivity of birds is reduced by a complex array of metabolic and physiological responses aimed at maintaining homeostasis. Fast growing broilers are especially vulnerable to high ambient temperatures because of their increased metabolic rate associated with their higher growth rate. A continuous increase in their somatic growth rate and feather development as well as heat increment due to feeding and voluntary activity produces excess body heat.

Various factors affect bird's response to heat stress. Age of bird, size, genetic makeup and history of heat exposure are the main factors, which influence the bird's response to HS (Teeter and Belay, 1996). The chicken body temperature rises above normal when the environmental temperature rises to 30 C or above (Romijn and Lockhorst,1966). Any variation in environmental temperature out of these limits will have negative impact on productivity. Donkoh (1989) reported that body temperature of the adult birds is normally in the range of 41 to 42 C and it is significantly increased when ambient temperature increased. Individual variation in body temperature was also found greater at higher temperature (Wilson, 1948). Upper and lower critical temperatures will vary depending on several factors, including degree of acclimatization, rate of production, air movement around the animal and relative humidity (Fuquay, 1981). In a more recent study, Teeter et al. (1996) have demonstrated that the comfort zones for poultry declines from 35 C at hatching to approximately 24 C at 4 weeks of age.

Chronic HS exposure results in not only heavy mortality but also reduced growth rate, egg production, fertility, and hatchability in poultry. Yahav et al. (1996) observed growth retardation after short-term exposure to HS during 1st wk of age, whereas marked increase in mortality was observed at 42-d-old birds when exposed to 35 C. Several studies demonstrated an increase in mortality in broilers exposed to prolonged periods of high temperatures, especially after 4 wk of age (McDougald and McQuistion, 1980; Thaxton and Pardue, 1984; Bottje and Harisson ,1985). Pardue et al. (1985) observed that CCHS leads to increase mortality and reduced growth rate in broilers. Heat stress response is to elevate the body temperature and inability to regulate body temperature (Keshavarz and McDougald, 1981). Heat stress also has a very crucial influence on the reduction of immune status of animals which, not only makes birds more susceptible to disease, but also increases mortality and reduces productivity due to diseases as well.

Poultry do not have the ability to sweat and must rely primarily upon panting (evaporative cooling) to remove surplus deep body heat under heat stress conditions. Birds classically increase respiration rate during heat stress to enhance evaporative cooling even though it results in CO_2 loss and respiratory alkalosis (Bottje and Harrison, 1985; Teeter et al., 1985). As therapeutic measures, drinking water carbonation (Bottje and Harrison, 1985) and acidifier inclusion (Teeter et al., 1985) have been reported to partially counter deleterious heat stress consequences.

Blood plays an important role in the diffusion of body heat. Chickens exposed to acute HS developed hypertension and an increase in cardiac output, hypothermia likewise depress blood pressure in the chicken. Donkoh (1989) stated that blood values

of red cell counts, packed cell volume, hemoglobin concentration and plasma protein concentration were reduced of birds raised at 30 C and 35 C as compared to birds raised at 20 C. Exposure to heat resulted in an irreversible decrease in blood hematocrit (Yahav et al., 1996). Zhou et al. (1997) observed whole blood viscosity of broilers decrease significantly when they were exposed to a high ambient temperature. The decrease in whole blood viscosity may be advantageous in reducing peripheral resistance and the load on heat, and increasing tissue perfusion and circulatory distribution, including the blood supply to heat exchange surface. Thermal conductivity of skin increases linearly with rate of blood flow in skin (Ohara ,1981).

Therapeutic application to lessen the consequences of HS through electrolyte supplementation has been observed with partial success. These therapeutic applications have multiple actions by improving acid base balance, water balance, and mineral balance simultaneously. Studies shows that $NaHCO_3$, $KHCO_3$, $CaCl_2$, and NH_4Cl helps to correct the acid-base imbalance of the blood, by counteracting the pH rises of respiratory alkalosis. Teeter et al. (1985), observed that birds may show positive responses to the use of dietary supplements of $NaHCO_3$ during HS. Heat exposed birds may exhibit a reduction in the levels of plasma CO_2 and HCO_3 with or without panting (Belnave and Gorman, 1993). Teeter et al. (1985) noted the loss of HCO_3 ions during heat exposure may affect the blood pH and induce in the bird nutritional requirement for bicarbonate. Bonsembianate et al. (1990) reported that supplementing feed of 7-d-old turkeys with 5 g $NaHCO_3$/kg in the diet resulted in an improvement in growth when temperatures ranged from 26 to 30 C and relative humidity ranged from 75 to 90%. Working with heat stressed finishing broilers (30 C), Belnave and Oliva (1991) reported

that diets supplemented with 16.8 g $NaHCO_3$/kg and drinking water supplemented with 5.6 g $NAHCO_3$/l produced a significant improvement in bird production response. Betaine is a metabolite of choline that donates methyl groups to homocystein to form methionine, and also to the folate pool. Betaine is formed from choline and that growth responses obtained from betaine are due to its ability to provide methyl group (Kidd et al., 1997). Birds maintain the intracellular concentration of water that is crucial for homeostasis by osmoregulation. Osmoregulation is the ability of a cell to maintain its structure and function by regulation movement of water in and out of the cell (Kidd et al., 1997). The osmoprotective properties of betaine are well conserved in many forms of life, including bacteria (Chambers and Kunin, 1987), and animals (Law and Burg, 1991). Betaine is the most important osmoprotective compound in bacteria (Imhoff and Rodriguz-Valera, 1984). Betaine is known as to serve as an osmoprotectant in bacteria by replacing intracellular K^+ and restoring the osmotic turgor without accumulation of K^+ when the environmental salinity increased (Sutherland et al., 1986). Beneficial osmoprotective properties may be due to the dipolar zwitterin characteristics of betaine and its high solubility in water (Chambers and Kunin, 1985). The unique chemical properties of betaine play a key role in providing osmoprotective properties in microorganisms and these attributes have a parallel in more complex organism (Bagnasco et al., 1986). Saunderson and MacKinley (1990) evaluated growth and hepatic enzymes in male broiler chicks as influenced by dietary supplementation with combinations of methionine, betaine and choline. Betaine inclusion improved the growth of chicks fed a semi- purified diet (McGinnis et al., 1991). Finkelstein et al. (1983), evaluated that supplemental betaine and choline at dietary levels of 0.2% on hepatic betaine-

homocystine methyltransferase activity increased as dietary levels of betaine and choline increased.

Thermal conditioning at an early age has bee reposted to result in reduced weight gain during the 1st week of life, followed by an accelerated growth which leads to a higher body weight than that of non-conditioned chickens at marketing age (Yahav et al., 1997). Accelerated growth in broiler chickens has also been observed after food restriction at an early age (Plavnik and Hurwitz, 1985, 1988, and 1989; Deaton, 1995; Zhong et al., 1995). During feed restriction requirement of maintenance energy declined, potentially because of reduction in heat production as observed in mammals (Forsum et al., 1981) but it returned to normal upon resumption of normal feeding (Plavink and Hurwitz, 1985). Feed restriction has been found to result in changes in hormones (McMurtry et al., 1992), metabolic status (Rosebrough et al., 1986; Zhong et al., 1995) and digestive enzymes activities (Palo et al., 1995a). Al-Harthi and MacLeod (1996) demonstrated that apparent metabolize able energy intake was 25% lower ($P<0.05$) in the 30 C and pair –fed birds than in the 20 C control group. Ad libitum fed birds at 20 C gained more weight than the 30 C and pair fed groups. Heat production was greater in the 20 C control group while the 20 C pair fed birds had a higher heat production than birds on the same food intake kept at 30 C. Feed restriction can be vital in helping an animal to survive in an heat stress environment. Teeter et al (1992) suggested that when birds were precision fed graded amounts of feed (5 %, to 10 % body weight) they were observed to have a lower body temperature. Pierce (1980) mentioned withdrawing the feed in the morning so birds were fasted during the hottest part of the day. This practice has been relatively successful and is used routinely by many companies when there is a possibility

that heat stress-related mortality might occur. Teeter et al.(1987) noted reduced mortality when chickens in wire-cage batteries were fasted prior to heat stress in an environmental chamber.

The objective of the experiment described here was to evaluate the therapeutic effects of betaine and electrolytes on bird performance under CCHS conditions and to quantify the effects of therapeutics and two environmental conditions (CCHS and thermoneutral) on bird fasted heat production and body composition. The latter phase included a third environment in which birds were reared under CCHS conditions until 33 d of age and transferred into thermoneutral environment to quantify the compensatory or catch-up growth.

MATERIALS AND METHODS

General

Two experiments were conducted to evaluate the effects of electrolytes and betaine on overall performance, survivability, basal metabolic rate and body composition of and heat exposed male broilers. Experiment 1 conducted to evaluate the effects of betaine and electrolytes on overall performance and survivability of male broilers raised under cycling chronic heat stress. Experiment 2 was conducted to evaluate the effects of betaine and electrolytes on the overall performance, basal metabolic rate, and body composition as well as to evaluate their effects on broiler catch-up or compensatory growth when they were transferred from cycling chronic heat stress environment to a thermoneutral environment. A control group was also maintained in a thermoneutral environment so that effects of therapeutics on catch-up growth could be quantified and compared to their counterparts raised under thermoneutral conditions.

Starter phase

Flocks of four hundred and eighty (Experiment 1) and five hundred and sixty (Experiment 2) Cobb x Cobb male commercial strain broilers were reared on floor pens from day of hatching to 18 d of age. Experimental flocks were reared on four floor pens in a thermostatically controlled environment. Chicks were pooled randomly and were placed on concrete floor covered with wood shavings as litter. Birds were reared under optimum growth conditions recommended by breeders. Starter feed was provided in open tray feeders for fist four days and then after hanging feeder were used simultaneously for feeding. Corn-soy based starter mash diet (Table 1) was offered ad libitum until 18 days of age. Automatic nipple drinkers were placed prior to arrival of chicks and water was available ad libitum until the brooding period. At 14 days of age, all birds were wing banded. Feed consumption and daily mortality was recorded on prescribed data capture farm. At the age of 17 days all birds were fasted overnight. At d 18, all birds were weighted and birds weighing not less than 490 g were collected and transferred to battery house.

Grower phase

At 18 d of age, (following an overnight fast) birds were transferred to a large scale thermostatically controlled environmental chamber complex that has been previously described (Belay and Teeter, 1993). Battery house had 30 batteries each containing eight compartmens 61x 92 cm in size. Twenty of the total 30 batteries were located in heat stress section whereas ten batteries were located in thermoneutral section. In heat stress section, batteries were positioned in north (side A) to south (side B) orientation. Chicks were allocated to compartments such that each compartment contained six chicks in

CCHS side A, and seven chicks in CCHS side B and half of the previously labeled TN compartments. At 18 d of age, all birds were individually weighted and birds weighing not less than 490 g were used in grower phase. Each 61 x 92 cm compartment contained two feeders, two cup drinkers, and continuous fluorescent lighting. Relative humidity was maintained at a constant 70% ± 5%. Ambient temperature was initially held at 24 C for days 1-2, and then the high temperature was gradually increased (2.5 C/d) to provide a daytime high of 35 C and daily low temperature of 24 C. Cycling ambient temperature consisted of 12 h of 24 C, 6 h of 24-35 C and 6 h of 35-24 C for the cycling temperature stress.

Drinkers of each compartment were attached to a water barrel, pumping motor were fixed for continuous water flow to drinkers. Under continuous fluorescent filament lighting, these chicks were provided different treatments, through their drinking water and feed. Birds had free access to water and feed throughout study (excluding birds selected for fasted heat production and body composition analysis).

At the age of 33 days, 280 birds moved from HS to TN chambers. All birds were allotted same treatment as in heat stress birds. Transferred birds were used to measure compensatory gain compared to TN birds. Body weight of compensatory birds was recorded at the age of 40 and 48 days. At the age of 36 and 46 days two birds from each compartments were used as for body temperature.

At the age of 31 d and 35 d, 6 birds per treatment from HS (30 birds) and TN (30) at the age of 39 and 44 d, 4 birds per treatment of HS (20 birds) TN (20 birds) and compensatory gain (CG) birds (20 birds) were randomly pooled out for basal metabolic rate in respiratory chambers. These birds were weighted and fasted on floor in battery

house for 36 hours under originating environmental conditions. Plain water was provided in plastic drinkers. After 36 hours these birds were again weighted and shifted to metabolic chambers described elsewhere (Belay and Teeter, 1993). Birds were placed in as one bird per chamber and distributed evenly in all three rooms. Birds were fasted and were kept at room temperature (24 C) for 6 hours. During six hours stay in respiratory chambers, room temperatures, O_2 consumption and CO_2 production were recorded through automatic computerized data acquisition system. At the end of six hours, birds were weighted again, euthanized, and kept in deep freezer for scanning for body composition.

Electrolytes were mixed sufficient for 66- liter water (Table 2). Every morning treated water were measured and treatment was added if birds consumed at least 66 liter of water (left over water 40 liter) .Two levels of betaine were also mixed on the whenever required bases and were added in to the drinking water. Every third day left over water was removed and replaced with fresh water.

Birds were sacrificed at the end of heat BMR recording and were scanned with Hologic XRay Densitometer for bone mineral density, whole body fat, and lean analysis.

Upon completion of above three experiments, effects of ambient temperature and appropriate effects of treatments were evaluated using general linear model procedure of the statistical analysis system. When a significant statistic was conducted means were separated using least square means.

Data Analysis

The data were analyzed using ordinary least squares (SAS, 1991). The model included environment (thermoneutral, CCHS, and combination of both for compensatory

gain birds) and therapeutic treatments (control, betaine, and electrolytes) as main effects. Interaction between main effects was included in the model. Mean separation was accomplished using Least Significant Difference (Steel and Torrie, 1960). Carbon dioxide production and O_2 consumption were regressed against elapsed time with equations for each individual chamber being integrated to compute CO_2 production and O_2 consumption for the time period.

RESULTS AND DISCUSSIONS

Results for d 47 individual BW, d 19-47 survivability, feed consumption, and feed conversion ratio (FCR) in Experiment 1 are presented in Table 3. No betaine x electrolytes interaction was observed for the variables mentioned. Electrolytes did not impact final BW whereas birds provided betaine via drinking water had a tendency (P = 0.0642) to improve the d-47 BW. Both electrolytes and betaine either numerically or significantly improved broiler survivability during d 19-47 CCHS. A significant betaine effect was observed on broiler survivability during this trial ($P < 0.05$). Birds provided only betaine via drinking water showed the highest survivability rate whereas control birds with no electrolyte or betaine supplementation showed the lowest survivability rate. Betaine supplementation also improved the electrolyte effect on broiler survivability. The improvement in broiler survivability with electrolyte and betaine treatment was higher than the improvement in broiler survivability with electrolytes only. However, it was still lower than the betaine only treatment. Both betaine and electrolytes failed to impact both feed consumption and feed conversion ratio ($P > 0.1$).

Effect of environment, betaine and electrolytes on broiler fasted BW, % body fat, % body protein, bone mineral density, and fasted heat production of 33-, 37-, 41-, and 46-

d-old birds in Experiment 2 are presented in Tables 4, 5, 6, and 7. In this part of the study, either 30 birds from two environment history (days 33 and 37 from CCHS or TN environments) or 20 birds from three environment history (days 41 and 46 from CCHS, TN, and their combination for compensatory gain birds) were randomly selected and used in the study. Because environment x treatment interaction was detected on fasted broiler BW at d 33 and environment effect was detected on d 46 BW, variables are expressed as per unit BW. No treatment x environment interaction was noted on broiler heat production, % lean and fat tissue, and bone mineral density although there was a tendency at d 46 on % lean and fat tissue analysis. Neither electrolyte nor betaine supplementation had an impact on broiler % lean, % fat, and bone mineral density throughout the study. However, environment had a significant impact on broiler body composition at d 33, d 37, and d 41 as expected. Heat stressed birds had significantly higher body fat and lower lean tissue as compared to their TN counterparts at d 33, 37, and 41. The environment effect disappeared as the birds aged d 46. Birds transferred from HS environment to TN environment for compensatory gain assessment also showed similar trends with HS birds. Parallel to the literature, birds originating from TN environment had higher lean tissue and lower fat retention. Increased carcass fat during CCHS was in agreement with other studies (Howlider and Rose, 1987; Sonaiya et al., 1990; Belay and Teeter, 1992) and may be an indicator of metabolic adjustments to reduce heat production as heat production is positively correlated with lean tissue accretion (Teeter and Wiernusz, 1994). Sonaiya (1988) showed a significant increase in polyunsaturated to saturated fatty acid ratio in the abdominal of heat-exposed chicken. Calorimetric studies conducted with broilers so far have shown no conclusive evidence of

the role of metabolic heat production during and after early feed restriction on body composition and growth rat (Jones and Farrell, 1992).

Analysis of BMR heat production of these birds yielded similar results with body composition data. Environment had significant impact on bird's BMR heat production at d 33, 37, and 41. Similar to body composition data, no environment effect was observed on bird HP at d 46 suggesting that birds reached their genetically set body weight limits and their metabolic rate was similar to their thermoneutral counterparts. Basal metabolic rate heat production was lower for the heat stressed birds with the exception of d 46 indicating that there is a correlation between body composition and BMR. This supports the theory that increased carcass fat during CCHS is an indicator of metabolic adjustments to reduce heat production as heat production is positively correlated with lean tissue accretion (Teeter and Wiernusz, 1994). During the phase of forced feed restriction or by exposing to heat stress, bird's BMR heat production, in other words energy requirements for maintenance, is declined may be because of reduced heat production and it is returned to normal upon resumption of normal feeding.

Current study indicated compensatory gain in birds pre exposed to heat stress (37 C) for 15 days from 18 to 33 days of age and then reared in thermoneutral environment (24 C) for 15 days. Results of compensatory birds were compared with control (thermoneutral) birds. Most of studies conducted on compensatory gain were based on feed restriction or feeding low energy or protein diet. Current study on compensatory gain was conducted by reducing feed intake through exposure in high ambient temperature. Compensatory gain birds performance was superior to thermoneutral birds particularly within first week of stress removal ($P < 0.01$). Weight gain was similar

during week 2. This result could be affected by the removal of 20 compensatory gain birds for BMR and body composition evaluation. Overall 33-47 d body weight gain data was also in favor of compensatory gain birds ($P < 0.05$). In general, these results were in agreement with other studies (Yahav and Hurtwitz, 1996; Yahav et al., 1997) of accelerated growth with the early age thermal conditioning of birds. Accelerated growth was also observed after food restriction (Plavnik and Hurwitz, 1985, 1988, 1989). Yahav and Plavnik (1998) exposed birds to HS at the age of 5 days and observed compensatory growth until the age of 42 d. Current results are similar to their observations regarding the accelerated growth, observed at the age of 41 d and 47 d in CG birds compared with TN birds.

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Table 1. Experimental rations fed during starter (0-18 d) and grower (19-44 d) periods

Ingredient	Starter diet, %	Grower diet, %
Corn	70.802	60.306
Soybean meal	21.305	30.800
Fat, vegetable	3.602	2.257
Calcium carbonate	1.742	1.179
Dicalcium phos	1.153	1.200
Pro-Pak[1]	0.654	3.584
Salt	0.230	0.300
DL-Methionine	0.198	0.143
L-Lysine	0.092	0.000
Vitamin premix[2]	0.050	0.050
Mineral premix[3]	0.050	0.050
Lasolosid	0.050	0.050
Choline chloride	0.040	0.050
Copper sulfate	0.030	0.030
Selenium premix	0.002	0.001
Calculated analysis		
M.E, kcal/kg	22	17.00
C.P, %	3073	3225

[1]Pro-pak® is a marine and animal protein product that contains 60% CP and is a registered trade mark of H.J Baker & Bro., Inc.

[2]Supplied per kilogram of diet: vitamin A, 38,500 IU; vitamin D_3, 11,000 IU; vitamin E, 55 IU; vitamin B_{12}, .066 mg; riboflavin, 33 mg; niacin, 165 mg; d-pantothenic acid, 55 mg; menadione, 11 mg; folic acid, 3.3 mg; pyridoxine, 13.75 mg; thiamin, 6.66 mg; d-biotin, 0.28 mg.

[3]Supplied per kilogram of diet: manganese, 120 mg; zinc, 100 mg, copper, 10 mg, iodine, 2.5 mg; calcium, 135 mg

Table 2. Electrolytes composition in Experiment 1 and Experiment 2

Ingredient	Electrolytes, g/46 l water
Potassium Bicarbonate	64.68
Potassium Chloride	13.86
Potassium Sulfate	7.81
Sodium Bicarbonate	36.03
Magnesium	34.62
Manganese	2.37
Zinc	1.38
Copper	0.315
Selenium	0.00036

Table 3. Effect of betaine and electrolytes on broiler d 47 BW, d 19-47 feed conversion ratio, and survivability in Experiment 1

Treatments		Variables			
Betaine	Electrolytes	d 47 BW	d 19-47 survivability	d 19-47 feed consumption	d 19-47 feed conversion ratio
		(g)	(%)	(g)	(g:g)
0	0	2240±18.0	81.5±2.28	3704±66.1	2.184±0.039
0	1	2227±24.3	90.6±3.48	3633±100.7	2.185±0.059
1	0	2261±24.8	94.1±3.24	3578±93.7	2.120±0.055
1	1	2291±24.1	92.7±3.12	3699±90.3	2.144±0.053
Betaine					
0		2234±15.1	86.1±2.07[b]	3669±60.2	2.185±0.035
1		2276±17.3	93.4±2.25[a]	3639±65.1	2.132±0.038
Electrolytes					
0		2250±15.3	87.8±1.98	3641±57.4	2.152±0.034
1		2259±17.1	91.7±2.33	3667±67.6	2.165±0.040
Source of variation		probabilities			
Betaine		0.0642	0.0196	0.7375	0.3172
Electrolytes		0.6964	0.2103	0.7739	0.8137
Betaine x Electrolytes		0.3540	0.0918	0.2877	0.8217

[a-b] Means in a column with common superscript do not differ significantly ($P < 0.05$)

Table 4. Effect of environment, betaine and electrolytes on broiler fasted BW, % body fat, % body protein, bone mineral density, and fasted heat production of 33-d-old birds in Experiment 2

		Variables				
Environment	Treatment	Fasted BW (g)	Heat production (kcal/kg per day)	Body protein (%)	Body fat (%)	BMD (g/cm^2)
Heat Stress	Control	1393	87.3	83.34	15.02	0.151
	Betaine	1218	103.2	85.49	12.90	0.149
	Electrolyte	1326	103.4	85.59	13.03	0.145
Thermoneutral	Control	1293	107.3	87.07	11.35	0.149
	Betaine	1345	113.9	86.06	12.48	0.150
	Electrolyte	1327	111.7	86.68	11.70	0.152
SEM		39.03	5.56	1.00	0.97	0.0039
Environment						
Heat Stress		1313	97.9[b]	84.80[b]	13.64[a]	0.148
Thermoneutral		1322	111.0[a]	86.60[a]	11.84[b]	0.150
Treatment						
Control		1344	97.3	85.20	13.18	0.150
Betaine		1282	108.6	85.77	12.69	0.149
Electrolyte		1327	107.5	86.13	12.36	0.148
Source of variation		probabilities				
Environment		0.7756	0.0068	0.0340	0.0283	0.4999
Treatment		0.2588	0.1516	0.6883	0.7324	0.9078
Environment x Treatment		0.0244	0.6157	0.3278	0.3034	0.5499

[a-b] Means in a column with common superscript do not differ significantly ($P < 0.05$)

Table 5. Effect of environment, betaine and electrolytes on broiler fasted BW, % body fat, % body protein, bone mineral density, and fasted heat production of 37-d-old birds in Experiment 2

		Variables				
Environment	Treatment	Fasted BW (g)	Heat production (kcal/kg per day)	Body protein (%)	Body fat (%)	BMD (g/cm^2)
Heat Stress	Control	1548	76.6	81.84	16.58	0.156
	Betaine	1508	90.0	83.95	14.40	0.155
	Electrolyte	1571	98.2	83.69	14.78	0.148
Thermoneutral	Control	1577	88.0	86.36	12.10	0.152
	Betaine	1551	105.0	86.46	11.91	0.153
	Electrolyte	1589	100.7	86.41	12.02	0.158
SEM		39.20	4.23	0.76	0.75	0.0037
Environment						
Heat Stress		1542	88.3^{b}	83.16^{b}	15.25^{a}	0.153
Thermoneutral		1572	97.9^{a}	86.41^{a}	12.01^{b}	0.154
Treatment						
Control		1562	82.3^{b}	84.08	14.34	0.154
Betaine		1529	97.5^{a}	85.21	13.15	0.154
Electrolyte		1580	99.5^{a}	85.05	13.40	0.153
Source of variation		probabilities				
Environment		0.3709	0.0086	0.0001	0.0001	0.6539
Treatment		0.3627	0.0012	0.3869	0.3405	0.8737
Environment x Treatment		0.9392	0.2480	0.4490	0.4555	0.1099

[a-b] Means in a column with common superscript do not differ significantly ($P < 0.05$)

Table 6. Effect of environment, betaine and electrolytes on broiler fasted BW, % body fat, % body protein, bone mineral density, and fasted heat production of 41-d-old birds in Experiment 2

		Variables				
Environment	Treatment	Fasted BW (g)	Heat production (kcal/kg per day)	Body protein (%)	Body fat (%)	BMD (g/cm^2)
Compensatory	Control	1793	101.9	84.51	13.95	0.162
	Betaine	1824	85.4	83.33	15.06	0.158
	Electrolyte	1793	86.6	82.94	15.54	0.160
Heat Stress	Control	1834	76.7	82.19	16.30	0.163
	Betaine	1843	80.6	85.04	13.39	0.155
	Electrolyte	1724	84.6	83.03	15.36	0.155
Thermoneutral	Control	1815	94.7	86.71	11.70	0.159
	Betaine	1878	92.9	87.06	11.34	0.161
	Electrolyte	1821	90.5	86.58	11.86	0.157
SEM		59.7	4.9	1.33	1.26	0.0031
Environment						
Compensatory		1804	91.3[a]	83.60[b]	14.85[a]	0.160
Heat Stress		1800	80.7[b]	83.42[b]	15.02[a]	0.158
Thermoneutral		1838	92.7[a]	86.79[a]	11.63[b]	0.159
Treatment						
Control		1814	91.1	84.47	13.98	0.161
Betaine		1848	86.3	85.15	13.26	0.158
Electrolyte		1780	87.2	84.19	14.26	0.158
Source of variation		probabilities				
Environment		0.6985	0.0086	0.0034	0.0027	0.7736
Treatment		0.2848	0.5335	0.5674	0.5326	0.5362
Environment x Treatment		0.8553	0.2352	0.7038	0.6967	0.7335

[a-b] Means in a column with common superscript do not differ significantly ($P < 0.05$)

Table 7. Effect of environment, betaine and electrolytes on broiler fasted BW, % body fat, % body protein, bone mineral density, and fasted heat production of 46-d-old birds in Experiment 2

		Variables				
Environment	Treatment	Fasted BW (g)	Heat production (kcal/kg/day)	Body protein (%)	Body fat (%)	BMD (g/cm^2)
Compensatory	Control	2202	67.7	86.24	12.28	0.167
	Betaine	2248	66.8	84.46	14.01	0.167
	Electrolyte	2312	67.4	84.06	14.49	0.165
Heat Stress	Control	2124	66.1	84.55	13.93	0.166
	Betaine	1933	76.3	85.79	12.73	0.156
	Electrolyte	2046	75.2	85.96	12.48	0.170
Thermoneutral	Control	2397	63.1	85.43	13.02	0.182
	Betaine	2181	70.8	85.00	13.51	0.162
	Electrolyte	2137	71.5	88.33	10.14	0.171
SEM		73.7	4.3	1.01	1.00	0.0043
Environment						
Compensatory		2254a	67.3	84.92	13.59	0.166
Heat Stress		2034b	72.5	85.43	13.04	0.164
Thermoneutral		2238a	68.5	86.25	12.22	0.172
Treatment						
Control		2241	65.6	85.41	13.07	0.172a
Betaine		2121	71.3	85.08	13.42	0.162b
Electrolyte		2165	71.4	86.12	12.37	0.169a
Source of variation		probabilities				
Environment		0.0011	0.3317	0.2682	0.2470	0.0841
Treatment		0.1793	0.2619	0.3802	0.3650	0.0172
Environment x Treatment		0.1756	0.7731	0.0741	0.0664	0.1336

[a-b] Means in a column with common superscript do not differ significantly ($P < 0.05$)

Table 8. Effect of environment, betaine and electrolytes on broiler body weight and body weight gain during compensatory gain period in Experiment 2

		Variables					
Environment	Treatment	d 33 BW (g)	d 33-40 BW gain (g)	d 40 BW (g)	d 40-47 BW gain (g)	d 47 BW (g)	d 33-47 BW gain (g)
Compensatory	Control	1570.5	496.3	2066.9	379.6	2400.0	832.9
	Betaine	1548.3	505.6	2053.8	385.7	2409.1	870.7
	Electrolyte	1565.8	519.2	2084.9	400.4	2490.1	911.7
Thermoneutral	Control	1699.5	438.7	2138.3	454.0	2553.2	879.2
	Betaine	1668.9	456.9	2118.3	345.2	2442.7	811.1
	Electrolyte	1645.7	399.0	2044.7	383.0	2435.6	792.4
SEM		29.19	20.30	32.64	26.71	48.82	38.03
Environment							
Compensatory		1561.5b	507.0a	2068.5	388.6	2433.1	871.7a
Thermoneutral		1671.4a	431.6b	2100.4	394.1	2477.2	827.5b
Treatment							
Control		1635.0	467.5	2102.6	416.8	2476.6	856.0
Betaine		1608.6	481.3	2086.1	365.5	2425.9	840.9
Electrolyte		1605.7	459.1	2064.8	391.7	2462.8	852.0
Source of variation		probabilities					
Environment		0.0001	0.0001	0.3660	0.7279	0.5703	0.0413
Treatment		0.6175	0.4824	0.5363	0.2125	0.5542	0.9157
Environment x Treatment		0.6439	0.1152	0.1306	0.1340	0.1403	0.1314

[a-b] Means in a column with common superscript do not differ significantly ($P < 0.05$)

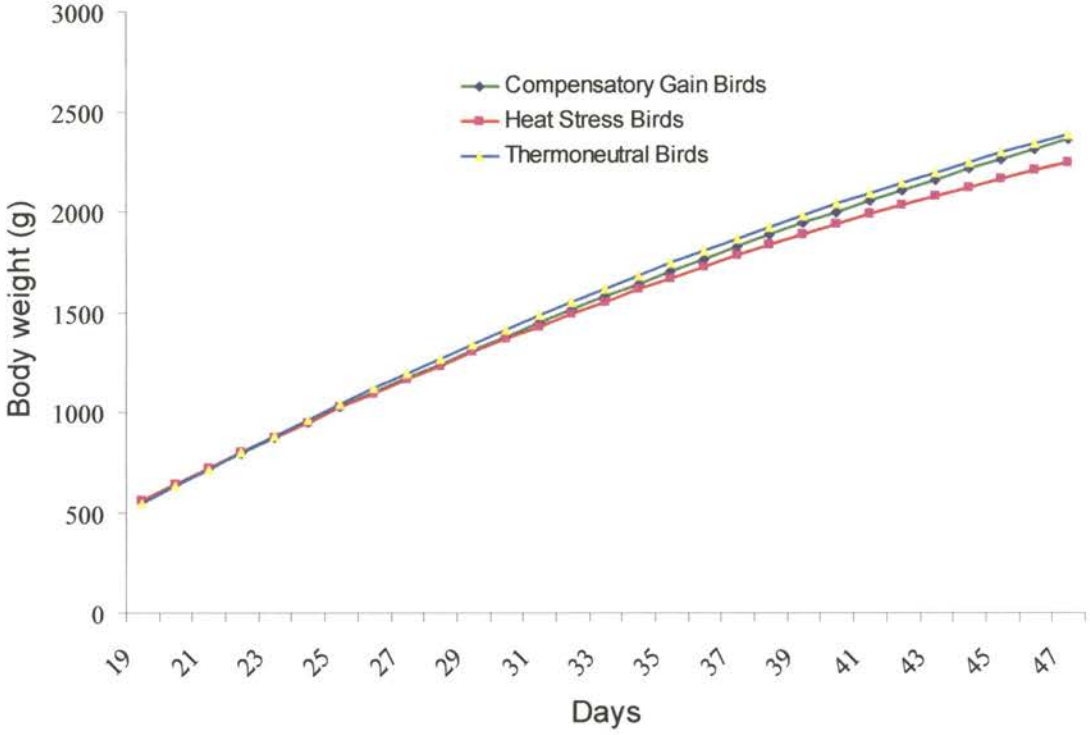

Figure 1. Effects of temperature on broiler body weight in grower phase

CHAPTER V

An Evaluation of endo-β-D-mannanase (Hemicell®) Effects on Broiler Performance and Energy Utilization in Diets Varying in β-mannan Content[1]

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Section: Metabolism and Nutrition

Running head: Evaluation of endo-β-D-mannanase

Abbreviation key: NSP = non-starch polysaccharides; BM = beta mannan; GIT = gastrointestinal tract; NE = net energy

Full-Length Paper _____X_____ Research Note: _____

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ABSTRACT Two experiments were conducted to evaluate the effects of a commercial endo-β-D-mannanase (Hemicell®) on some serum parameters, ME, net energy (NE), and overall performance of broilers fed diets varying in β-mannan (BM) level (Experiment 1), and to evaluate its inclusion level effect on the same variables of broilers fed a diet high in BM (Experiment 2). As a semi-purified BM source, guar gum was used to alter the BM level in diets. In Experiment 1, guar gum was added at 0, 0.5, 1, and 2% in a corn-soy based starter diet with (0.5%) and without endo-β-D-mannanase supplementation in a 4x2 factorial design. Enzyme supplementation improved ($P < 0.01$) feed efficiency at control and each guar gum inclusion level, whereas 2% guar gum supplementation reduced ($P < 0.01$) BW and increased ($P < 0.01$) d 14 feed:gain ratio. Enzyme supplementation also increased dietary ME and NE. In Experiment 2, endo-β-D-mannanase was added at 0, 0.5, 1, and 1.5% in a corn-soy based starter diet containing 1% guar gum. Increasing endo-β-D-mannanase supplementation did not affect ($P > 0.10$) final body weight, but improved 14 d feed:gain ratio at all inclusion levels. Similar to the first experiment, ME improved ($P < 0.05$) with increasing enzyme inclusion. Dietary endo-β-D-mannanase inclusion either numerically or significantly reduced water:feed ratio and reduced total dry fecal output ($P < 0.001$). In conclusion, these results suggest that endo-β-D-mannanase (Hemicell®) supplementation can improve the utilization of nutrients from diets containing BM.

(*Key words*: broiler, guar gum, enzyme supplementation, metabolizable energy, net energy)

INTRODUCTION

The identification and alleviation of factors inhibiting nutrient utilization are necessary for successful poultry production. Among potential factors reducing nutrient bioavailability are the non-starch polysaccharides (NSP). Non-starch polysaccharides are complex high molecular weight carbohydrates found in the structure of plant cell walls (Classen and Bedford, 1991; Annison and Choct, 1991; Bedford and Classen, 1993; Chocht, 2002). The NSP include various fiber types such as lignin, β-glucans, arabinoxylans (pentosans), uronic acid, galactose, and mannose in poultry feedstuffs (Aman and Graham, 1990). The β-glucans are predominantly found in barley and oats, whereas the arabinoxylans are found in wheat, rye, and triticale at higher rates (Classen and Bedford, 1991). Both fiber types, however, occur at some lower concentrations in most feedstuff. Certain protein concentrates, especially palm kernel meal, copra meal, and guar meal are among the feedstuffs rich in glucomannans and galactomannans (Carré, 2002).

Among NSP, mannans occur in the forms of glucomannans, galactomannans, glucogalactomannans, and glucuronomannans in plant cell walls (Aman and Graham, 1990). In BM, repeating D-mannose units with β-1,4 bonds and D-galactose units are attached in a 2:3 ratio (Ward and Fodge, 1996; Carré, 2002). Several studies demonstrated the negative effects of dietary BM found in palm kernel meal, copra meal, and guar gum and meal (Ray et al., 1982; Teves et al., 1988; Furuse and Mabayo, 1996).

Enzyme supplementation of poultry rations have been widely reviewed and well documented to improve efficiency of converting feedstuffs into broiler tissue (Annison and Choct, 1991; Campbell and Bedford, 1992; Bedford and Morgan, 1996; Marquardt et

al., 1996; Chocht, 2001). Reported benefits include improved weight gain, feed:gain ratio, and ME as well as reduced excreta and P output, water intake, digesta viscosity, and gastrointestinal tract (GIT) size (Friesen et al., 1992; Brenes et al., 1993; Annison et al., 1995; Marquardt et al., 1996). Copra and guar meals, feedstuffs rich in BM, were also reported to have increased utilization with bacterial mannanase treatment (Verma and McNab, 1982; Patel and McGinnis, 1985; Teves et al., 1988).

Guar (*Cyamopsis tetragonoloba*) is an annual legume and widely grown in countries such as India and Pakistan (Patel and McGinnis, 1985). Its endosperm is a rich source of a galactomannan polysaccharide, guar gum (Vohra and Kratzer, 1964a; Couch et al., 1967). Guar gum has long been known to depress growth when fed to chicks (Vohra and Kratzer, 1964b; Ray et al., 1982). Studies with guar gum, classified as a soluble dietary fiber have demonstrated that increased digesta viscosity in GIT is associated with a delay in gastric emptying (Holt et al., 1979) and reduced nutrient utilization (Jenkins et al., 1978; Taylor, 1979; Blackburn and Johnson, 1981). Jorgensen et al. (1996) observed a significant increase in the length and weight of the gastrointestinal tract of broiler chickens fed rations containing high levels of NSP. Addition of soluble NSP to broiler diets significantly elevated fermentation in small intestine and depolymerization of the NSP almost completely overcame this problem (Choct et al., 1996).

Chemical structure of BM in soy and in guar gum are almost identical (Whistler and Saarnio, 1957) which provides a potential method to elevate BM in practical diets. This study sought to investigate the efficacy of endo-β-D-mannanase supplementation

level in broiler diets varying in BM level as guar gum on broiler growth rate, feed:gain ratio, water:feed ratio, ME_n, NE, and some serum metabolites.

MATERIALS AND METHODS

General procedures

Two experiments were conducted to evaluate efficacy and inclusion level effects of endo-β-D-mannanase (Hemicell®[3]) in broiler diets varying in beta-mannan content. Because of its high BM content, guar gum was used as a semi-purified BM source. In both experiments, day-old Cobb x Cobb male chicks were obtained from a commercial hatchery, wing banded, individually weighed, and randomly assigned to metabolic chambers. Metabolic chamber facilities and procedures utilized for bird rearing and indirect calorimetry measurements have been documented elsewhere (Belay and Teeter, 1993). Briefly, the system utilizes the differential concentrations of incoming and outgoing O_2 and CO_2 to calculate bird O_2 consumption and CO_2 production. The equation developed by Brouwer (1965) was subsequently used to calculate bird heat production from CO_2 production and O_2 consumption measurements. In all cases, chicks were reared according to the breeder recommendations (Cobb x Cobb) with ad libitum access to feed (Table 1) and water. The chambers were monitored four times daily for mortality and general conditions. Chick live weighs, feed consumption, and water consumption measurements were recorded on days 7 and 14. Both trials were terminated on day 14 with chicks being individually weighed and blood samples collected via vein puncture (from two birds per chamber selected at random in Experiment 1; and four such birds per chamber in Experiment 2). Samples were tested for hematocrit by microcentrifugation.

Serum concentrations of inorganic P (No. 44031), Ca (No. 44033), glucose (No. 47382), and triglycerides (No. 44120) were determined.[4] Serum variables were measured using Cobas Mira[5] wet chemistry analyzer.

Excreta from chamber-housed birds was quantitatively collected and stored in plastic bags at -20 C until analysis. Samples were dried at 55 C, ground to a fine powder, and analyzed for C and N,[6] and gross energy via a bomb calorimeter.[7] Apparent metabolizable energy was corrected to zero N balance (Titus et al., 1959).

Experiment 1. This experiment was conducted to examine the effects of 4 dietary inclusion levels of guar gum each with and without enzyme supplementation on broiler performance, water consumption, serum Ca, P, glucose and triglycerides, dietary ME and NE, and total fecal output. The study utilized 144 day-old chicks, which were randomly assigned to 8 dietary treatments in 4x2 factorial treatment arrangement in a randomized block design. Each metabolic chamber had 4 birds throughout the study. Four guar gum levels (0, 0.5, 1, and 2 %) and 2 enzyme levels (0, and 0.05% Hemicell®) were used. Due to limitations in metabolic chamber number (36 total), treatments with 0 and 1% guar gum inclusion levels had 5 replications while treatments with 0.5 and 2% guar gum inclusion levels had 4 replications. Diet samples were analyzed for beta mannanase activity. Enzyme activities for diets containing 0, 0.5, 1, and 2% guar gum supplemented with enzyme were 152.7, 127.7, 145.9, 141.5 respectively.

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[3]Hemicell® is a registered trademark of ChemGen Corp., Gaithersburg, MD 20877. dried *B. lentus* fermentation solubles with 158 million units/kg minimum enzyme activity. Recommended usage rate is 0.05% of feed.
[4] Hoffman-LaRoche, Nutley, NJ 07042
[5] Roche Diagnostics Systems Inc., Montclair, NJ 07042-5199
[6] LECO CN-2000
[7] Parr Co., Moline, Illinois

Experiment 2. This experiment was conducted to examine the effects of 4 dietary enzyme supplementation levels on broiler performance, water consumption, serum Ca, P, glucose and triglycerides, dietary ME and NE, and total fecal output. This study utilized 216 day-old chicks randomly assigned to 4 dietary enzyme levels (0, 0.05, 0.1, and 0.15%) included in a starter ration (Table 1) containing 1% guar gum in a completely randomized block design. Each treatment was replicated with 6 metabolic chambers containing 9 birds each. Diet samples were analyzed for beta mannanase activity. Determined enzyme activities (million units per ton) for 0, 0.05, 0.1, and 0.15% enzyme supplementation levels were 1.4, 145.9, 225.8, and 349.8, respectively.

Data Analysis

The data were analyzed using ordinary least squares (SAS, 1991). The model included guar gum and Hemicell® as main effects. Interaction between main effects was included in the model. Mean separation was accomplished using Least Significant Difference (Steel and Torrie, 1960). Carbon dioxide production and O_2 consumption were regressed against elapsed time with equations for each individual chamber being integrated to compute CO_2 production and O_2 consumption for the time period.

RESULTS

Experiment 1.

Experiment 1 results for BW, water:feed ratios, and feed:gain ratios for wk 1, wk 2, and overall are presented in Table 2. Significant Hemicell® x guar gum interaction was detected for wk 1 and wk 2 BW and overall water:feed and feed:gain ratios. Feed efficiency and BW were severely reduced by the dietary inclusion level of 2% guar gum as early as completion of wk 1. During wk 1, addition of dietary Hemicell® either

significantly (0, 0.5, and 2% guar gum levels) or numerically (1% guar gum level) improved feed efficiency at each guar gum level, including negative control. Week 2 BW was restored to control values for all treatments but the 2% guar gum by Hemicell® addition. Among the enzyme free treatments, 0, and 0.5% guar gum inclusion levels had similar BW at 2 wk of age whereas they had lower wk 2 BW than control and higher wk 2 BW than 2% guar gum levels ($P < 0.05$). Guar gum level of 2% depressed wk 2 BW to a greater extent than all other Hemicell® free treatments as compared with the guar gum free control. Further, the wk 2 Hemicell® fortification improved ($P < 0.05$) the feed efficiency at every guar gum inclusion level, including the negative control. Feed efficiency was similar for the guar gum free control and the 1% guar gum with Hemicell® treatments. Only 2% guar gum inclusion with Hemicell® had a feed efficiency poorer ($P < 0.05$) than the control. Data reported for the full two wk interval was similar to overall two wk results. Guar gum inclusion, in general, increased water:feed ratio.

Effects of guar gum and enzyme supplementation on ME_n, NE, and total N and dry fecal output data in Experiment 1 are presented in Table 3. Similar to performance data, there was a dramatic decrease in ME_n with 2% guar gum levels. With the exception of control group, enzyme supplementation increased ($P < 0.05$) ME_n of the diet at each guar gum level ($P < 0.0001$). Dietary NE values had similar trend with ME_n. Increasing guar gum level in the diet increased total excreted N and total dry fecal output with the highest increase at 2% level whereas enzyme supplementation had the opposite effect particularly at higher guar gum levels.

Results for hematocrit, serum glucose, triglycerides, calcium, and phosphorus in Experiment 1 are displayed in Table 6. In our study, neither guar gum, nor enzyme

supplementation had an impact on hematocrit, serum glucose, triglyceride, Ca, and P levels. However, parallel to the literature, as the guar gum level increased in diets (0.5, 1, and 2%), birds had higher hematocrit, and lower serum glucose, triglyceride, Ca, and P levels as compared with birds on no guar gum diets (0%) in Experiment 1.

Experiment 2.

Experiment 2 results for live weight, water:feed ratio, and water:feed ratio for wk 1, wk 2, and overall are presented in Table 4. Increasing dietary beta mannanase supplementation did not have an impact on final BW of these broiler chicks, however, a significant improvement in feed utilization was noticed with increasing enzyme level from 0 to 0.15% of the diet. Overall Hemicell® effect on feed efficiency was quadratic and its efficiency gradually declined as enzyme level increased. Dietary enzyme supplementation either numerically or significantly reduced water:feed ratio and least water consumption and water:feed ratio were recorded with highest enzyme concentration.

Effects of guar gum and enzyme supplementation on ME_n, NE, and total nitrogen and dry fecal output data in Experiment 2 are presented in Table 5. Regardless of its level, enzyme supplementation significantly improved ME_n as compared with negative control group. As seen in Experiment 1, dietary beta mannanase supplementation reduced both total nitrogen excreted and dry fecal output. The decrease in both total nitrogen excreted and dry fecal output was significant between control and enzyme supplemented groups.

Results for hematocrit, serum glucose, triglycerides, calcium, and phosphorus in Experiment 2 are displayed in Table 6. Enzyme supplementation numerically reduced

hematocrit and increased serum glucose, triglycerides, Ca, and P at all inclusion levels as compared with negative control, indicating better absorption and consequently utilization of nutrients listed.

DISCUSSIONS

Guar gum has long been known to depress growth when fed to chicks due to its high NSP portion called guaran (Vohra and Kratzer, 1964b; Ray et al., 1982). Guaran has β-D-mannose backbone with α-(1-6) linked galactose residues, and hydrolysis of guaran yields D-galactose and D-mannose in the ratio of approximately 1:2 (Chandrasekaran, 1997, Jumel et al., 1996). The adverse effects of this ingredient on bird performance are well documented and numerous enzyme supplementations and feed processing techniques have been applied to either guar gum or guar meal in an effort to improve its utilization in broiler rations (Ray et al., 1982; Verma and McNab, 1982; Patel and McGinnis, 1985; Takahashi et al., 1994, Ellis et al., 1995; Furuse and Mabayo, 1996). In the current study, parallel to the literature cited, feed efficiency and BW were severely reduced by the dietary inclusion level of 2% guar gum. These results were in agreement with results from Vohra and Kratzer (1964a) and Ray et al. (1982). Vohra and Kratzer (1964a) reported a 20 to 25% reduction in BW with 2% inclusion of guar gum in chicken diet. Ray et al. (1982) tested the effectiveness of a guar degrading enzyme fraction of a commercial hemicellulase in chicks fed 2% guar gum and found that the enzyme was effective in eliminating the detrimental effects of guar gum on 14-d-old female broiler performance. In our study, BW was restored to control values for all guar gum inclusion levels but the 2% guar gum by Hemicell® addition in Experiment 1. In Experiment 2, a significant improvement in feed utilization was noticed with increasing enzyme level

from 0 to 0.15% when guar gum inclusion level was set 1% in all diets. Other studies with guar gum or guar meal yielded similar results. Verma and McNab (1982) observed reduced body weight gain, feed consumption, and gain:feed ratio when broiler chickens were fed diets with guar meal substitution, and enzyme supplementation improved growth on these birds compared to control birds. Vohra and Kratzer (1965) also suggested that if guar meal is to be used in chicken diets, diets should be supplemented with enzymes capable of breaking down gums and hemicellulose.

Guar gum is a high molecular galactomannan, which cannot be digested in the small intestine and consumption of indigestible non-starch polysaccharides are well known to have an impact on gastrointestinal tract viscosity and digestion and absorption of nutrients (Ikegami et al, 1990; Salih et al., 1991; Almirall et al., 1995; Annison et al., 1995). In Experiment 1, the amount of water consumption per unit feed consumption was increased with increasing guar gum inclusion levels whereas beta mannanase supplementation tended to reduce water consumption per unit feed consumed in Experiment 2. This may be partly explained by the increased gastrointestinal tract viscosity with increasing dietary guar gum levels and the effect of the enzyme on guar gum. The absence of enzyme effect on water:feed ratio in Experiment 1 was presumably due to fewer number of birds in each metabolic chamber and higher variation. Non-starch polysaccharides are shown to increase intestinal viscosity (Almirall et al., 1995; Annison et al., 1995), decrease the rate of digestion and absorption (Ikegami et al, 1990; Almirall et al, 1995), and reduce performance (Salih et al., 1991). Studies with rats and chicks showed that partially hydrolyzed guar gum had less detrimental effects compared to intact guar gum. (Takahashi et al., 1994; Furuse and Mobayo, 1996). Viscous

polysaccharides also cause physiological and morphological changes to the digestive system in various species (Brown et al., 1979; Cassidy et al., 1981; Jacobs, 1983; Morgan et al., 1985; Jorgensen et al., 1996). Jorgensen et al. (1996) observed a significant increase in the length and weight of the gastrointestinal tract of broiler chickens fed rations containing high levels of NSP. They also noted that ME intake was depressed whereas excretion of organic acids (which accounted for up to 2% of ME intake) increased with increasing dietary NSP. Ikegami et al. (1990) concluded that rats having prolonged consumption of highly viscous polysaccharides exhibited enlargement of digestive organs, increased secretion of digestive enzymes, and depressed apparent ileal protein digestibility. This may be the result of inhibition of protein breakdown, reduction in amino acid absorption, or increased endogenous protein secretion and loss of intestinal cells. Addition of guar gum into a fiber free diet led to a 19.1% increase in mucosal cell mass, resulting from the increase in the length of the small intestine rather than in villus length in rats (Jacobs, 1983). The same study also demonstrated that the higher rate of epithelial cell migration in the pectin and guar-fed groups shortened their estimated villus cell transit times to 36.4 ± 0.7 and 37.0 ± 1.4 h, respectively, when compared with 41.1 ± 1.0 h in the control. Because GIT size has been reported to contribute to basal metabolic rate (BMR), any reduction in GIT size and viscosity could reduce bird heat production and improve net energy of diet.

The viscous nature of guar gum also contributes to reduced absorption and utilization of nutrients. As the guar gum level increased in diets from 0.5% to 2%, birds had higher hematocrit, and lower serum glucose, triglyceride, Ca, and P levels compared to control birds (0% guar gum) in Experiment 1. Enzyme supplementation, in general,

reduced hematocrit and increased serum glucose, triglycerides, Ca, and P at all inclusion levels compared to the negative control, indicating better absorption and consequently utilization of nutrients listed. Studies indicated that increased viscosity of the gastrointestinal contents slows the gastric emptying, impairs the mixing of substrate with digestive enzymes, and reduces the rate of contact of nutrients with the absorptive epithelium (Read, 1986). Guar gum forms viscous solutions in the gastrointestinal system and has been long known to delay the absorption of other nutrients such as glucose (Blackburn and Johnson, 1981) and fat (Higham and Read, 1992). Guar gum also decreases the absorption and utilization of protein (Poksay and Schneeman, 1983). Galactomannan is also found in soybean hulls in small quantities (Whistler and Saarrino, 1957) and in an attempt to improve the utilization of soybean meal in swine diets, Pettey et al. (1999) used dietary beta mannanase in nursery diets. They observed a significant increase in gain:feed ratio and improvement on average daily gain at latter phase of the nursery period with beta mannanase supplementation. Radcliffe et al. (1999) examined the effect of beta mannanase on apparent total tract digestibilities of energy, Ca, P, DM, and apparent ileal digestibilities of amino acids, DM, Ca, and P in pigs fed a low and high protein corn-soybean meal diet. Enzyme supplementation significantly increased apparent ileal digestibility of DM and apparent total tract digestibility of energy.

Current research is examining different approaches to reduce the amount of N and P excreted by poultry. In Experiment 2, dietary beta mannanase supplementation dramatically reduced N and dry fecal output. In addition, increased serum Ca and P concentrations are indicators of better utilization of Ca and P.

In conclusion, this study demonstrated that 2 % guar gum has impairable effects on 2-wk-old broiler performance although enzyme supplementation can improve it partially. However, as low as 0.05 % enzyme supplementation was effective in eliminating the negative effects associated with guar gum supplemented up to 1 % into broiler diets. Improvement in BW and better feed efficiency at each dietary guar gum level, including guar gum free corn-soy based diet suggests that enzyme supplementation potentially increases profitability of broiler operations as well as it enables poultry producers to use alternative cheaper feedstuffs in diets at tolerable levels by birds. Improvement in feed efficiency with increasing dietary beta mannanase supplementation with diets high in BM also indicates that enzyme supplementation is a necessary step to reduce the cost of operation when ration is high in NSP.

ACKNOWLEDGEMENTS

Appreciation is expressed to ChemGen Corp., Gaithersburg, MD for their financial and technical support of this study.

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Table 1. Composition of starter basal diet

Ingredients and composition	Starter diet, %
Ground corn	60.04
Soybean meal	29.00
Pro-pak©[1]	5.05
Vegetable fat	3.15
Dicalcium phosphate	1.20
Calcium carbonate	0.81
Salt	0.38
Methionine	0.13
Vitamin premix[2]	0.05
Mineral mix[3]	0.05
Choline chloride	0.05
Lasalosid	0.05
Copper sulphate	0.03
Selenium premix	0.0015
Calculated analysis	
ME, kcal/kg	3150
CP	22.67
Methionine	0.54
Lysine	1.25
Calcium	0.95
Total phosphorus	0.72

[1]Pro-pak® is a marine and animal protein product that contains 60% CP and is a registered trade mark of H.J Baker & Bro., Inc.
[2]Supplied per kilogram of diet: vitamin A, 38,500 IU; vitamin D_3, 11,000 IU; vitamin E, 55 IU; vitamin B_{12}, .066 mg; riboflavin, 33 mg; niacin, 165 mg; d-pantothenic acid, 55 mg; menadione, 11 mg; folic acid, 3.3 mg; pyridoxine, 13.75 mg; thiamin, 6.66 mg; d-biotin, 0.28 mg.
[3]Supplied per kilogram of diet: manganese, 120 mg; zinc, 100 mg, copper, 10 mg, iodine, 2.5 mg; calcium, 135 mg.

Table 2. Effects of guar gum and endo-β-D-mannanase (Hemicell®) supplementation on the growth, performance, and water:feed ratio of broiler chicks during Experiment 1

Treatments		Variables[1]							
		Wk 1			Wk 2			Overall	
Guar gum	Endo-β-D-mannanase	BW	Water:Feed	Feed :Gain	BW	Water:Feed	Feed:Gain	Water:Feed	Feed:Gain
(%)	(%)	(g)	(g:g)	(g:g)	(g)	(g:g)	(g:g)	(g:g)	(g:g)
0	0	182.6^{a}	2.588^{d}	1.032^{cde}	394.8^{a}	2.374	1.282^{d}	2.446^{c}	1.182^{d}
0	0.05	174.5^{bc}	2.759^{c}	1.006^{f}	390.2^{a}	2.319	1.239^{e}	2.453^{c}	1.149^{e}
0.5	0	170.2^{c}	2.904^{b}	1.041^{c}	366.2^{bc}	2.481	1.294^{d}	2.617^{ab}	1.193^{d}
0.5	0.05	179.5^{ab}	2.606^{d}	1.002^{f}	396.2^{a}	2.287	1.247^{e}	2.398^{c}	1.150^{e}
1	0	169.9^{c}	3.068^{a}	1.037^{cde}	376.2^{b}	2.519	1.319^{c}	2.700^{a}	1.211^{c}
1	0.05	171.7^{c}	2.993^{ab}	1.028^{de}	390.0^{a}	2.437	1.293^{d}	2.615^{ab}	1.193^{d}
2	0	151.7^{d}	3.035^{ab}	1.173^{a}	335.7^{d}	2.372	1.565^{a}	2.579^{b}	1.417^{a}
2	0.05	155.9^{d}	3.050^{a}	1.146^{b}	354.0^{c}	2.398	1.448^{b}	2.602^{ab}	1.337^{b}
SEM		1.77	0.051	0.004	4.43	0.037	0.006	0.036	0.004
Enzyme, %									
0		168.6	2.899	1.071	368.2	2.437^{a}	1.365	2.585	1.251
0.05		170.4	2.852	1.045	382.6	2.361^{b}	1.307	2.517	1.208
Guar Gum, %									
0		178.6	2.673	1.019	392.5	2.347^{a}	1.261	2.449	1.656
0.5		174.9	2.755	1.021	381.2	2.385^{a}	1.271	2.507	1.172
1		170.8	3.031	1.032	383.1	2.478^{b}	1.306	2.657	1.202
2		153.8	3.043	1.160	344.8	2.386^{a}	1.506	2.591	1.377
Source of Variation		probabilities							
Enzyme		0.5973	0.4949	0.0001	0.0002	0.0060	0.0001	0.0182	0.0001
Guar Gum		0.0001	0.0001	0.0001	0.0001	0.0025	0.0001	0.0001	0.0001
Enzyme x Guar Gum		0.0002	0.0002	0.0202	0.0014	0.0612	0.0001	0.0064	0.0001

[a-d] Means in a column with no common superscript differ significantly ($P < 0.05$)

[1]Initial weight for enzyme levels (0 and 0.05%) and guar gum levels (0, 0.5, 1, and 2%) were 40.8, 41.0, 41.3, 40.7, 40.8 and 40.8 gr. respectively

Table 3. Effects of guar gum and endo-β-D-mannanase (Hemicell®) supplementation on ME_n, NE, and total N and fecal output during Experiment 1

Treatments		Variables			
Guar Gum	Endo-β-D-mannanase	MEn	NE	Total N excreted	Dry fecal output
(%)	(%)	(kcal/kg)	(kcal/kg)	(g)	(g)
0	0	3177[a]	1403	15.36	323.7
0	0.05	3144[ab]	1377	15.20	315.3
0.5	0	3107[b]	1372	15.63	320.5
0.5	0.05	3166[ab]	1448	16.34	325.8
1	0	3018[c]	1304	18.23	359.0
1	0.05	3106[b]	1373	16.70	342.4
2	0	2678[e]	1116	26.39	506.8
2	0.05	2827[d]	1269	24.05	442.2
SEM		22.36	35.28	1.04	19.88
Enzyme, %					
0		2995	1298[b]	18.90	377.5
0.05		3061	1366[a]	18.07	356.5
Guar Gum %					
0		3160	1390[a]	15.28[c]	319.5[b]
0.5		3136	1410[a]	15.99[bc]	323.2[b]
1		3062	1338[a]	17.46[b]	350.7[b]
2		2752	1193[b]	25.22[a]	474.5[a]
Source of Variation		probabilities			
Enzyme		0.0030	0.0421	0.3397	0.2462
Guar Gum		0.0001	0.0001	0.0001	0.0001
Enzyme x Guar Gum		0.0089	0.1731	0.5685	0.4944

[a-d] Means in a column with common superscript do not differ significantly ($P < 0.05$)

Table 4. Effects of guar gum and endo-β-D-mannanase (Hemicell®) supplementation on the growth, performance and water/feed ratio of broiler chicks during Experiment 2

Treatment		Variables							
		Wk 1			Wk 2			Overall	
Guar gum	Endo-β-D-mannanase	BW	Water:Feed	Feed:Gain	BW	Water:Feed	Feed:Gain	Water:Feed	Feed:Gain
(%)	(%)	(g)	(g:g)	(g:g)	(g)	(g:g)	(g:g)	(g:g)	(g:g)
1	0	171.3^{bc}	3.046^{a}	1.074^{a}	346.5	2.753^{a}	1.533^{a}	2.853^{a}	1.336^{a}
1	0.05	171.5^{b}	2.987^{ab}	1.049^{c}	346.9	2.632^{c}	1.495^{b}	2.752^{b}	1.304^{b}
1	0.1	176.0^{a}	3.045^{a}	1.036^{d}	348.1	2.699^{b}	1.493^{b}	2.821^{a}	1.291^{c}
1	0.15	168.8^{c}	2.947^{b}	1.056^{b}	345.5	2.523^{d}	1.454^{c}	2.664^{c}	1.286^{c}
SEM		0.86	0.025	0.0014	1.43	0.017	0.004	0.018	0.002
Source of Variation		probabilities							
Enzyme		0.0001	0.0187	0.0001	NS	0.0001	0.0001	0.0001	0.0001

[a-c] Means in a column with common superscript do not differ significantly ($P < 0.05$)

* Mean initial weight for enzyme levels (0, 0.05, 0.1, and 0.15%) were 40.9, 41.1, 41.1, and 40.3 respectively

Table 5. Effects of guar gum and endo-β-D-mannanase (Hemicell®) supplementation on ME_n, NE, and total N and fecal output during Experiment 1

Treatment		Variables			
Guar Gum (%)	Endo-β-D-mannanase (%)	MEn (kcal/kg)	NE (kcal/kg)	Total N Excreted (g)	Dry Fecal Output (g)
1	0	2971[b]	1247	44.11[a]	855.6[a]
1	0.05	3057[a]	1299	35.99[b]	754.3[b]
1	0.1	3067[a]	1255	35.03[b]	726.3[b]
1	0.15	3061[a]	1263	33.32[b]	703.5[b]
SEM		12.00	24.75	1.34	22.17
Source of Variation		probabilities			
Enzyme		0.0005	0.5182	0.0011	0.0041

[a-g] Means in a column with common superscript do not differ significantly ($P < 0.05$)

Table 6. Effects of guar gum and endo-β-D-mannanase (Hemicell®) supplementation on serum hematocrit, glucose, triglyceride, Ca, and P of broiler chicks during Experiment 1 and Experiment 2

Treatment		Variables				
Guar Gum	Endo-β-D-mannanase	Hematocrit	Glucose	Triglycerides	Ca	P
(%)	(%)	(%)	(mg/dl)	(mg/dl)	(mg/dl)	(mg/dl)
Experiment 1						
0	0	28.21	155.5	62.25	5.255	5.395
0	0.05	28.44	172.9	71.85	5.557	6.289
0.5	0	29.10	150.7	64.00	5.570	5.674
0.5	0.05	27.84	165.6	63.50	5.070	5.439
1	0	29.07	161.0	72.05	5.219	5.251
1	0.05	28.68	152.7	52.95	4.772	4.615
2	0	28.42	149.2	53.87	5.105	5.894
2	0.05	28.97	151.1	58.87	4.939	4.886
SEM		0.590	16.24	6.49	0.301	0.361
Enzyme, %						
0		28.70	154.1	63.04	5.287	5.553
0.05		28.49	160.7	61.79	5.084	5.307
Guar gum, %						
0		28.33	164.2	67.05	5.406	5.842
0.5		28.47	158.2	63.75	5.320	5.556
1		28.87	156.9	62.50	4.995	4.933
2		28.70	150.4	56.37	5.022	5.390
Source of Variation		probabilities				
Enzyme		NS	NS	NS	NS	NS
Guar Gum		NS	NS	NS	NS	0.0928
Enzyme x Guar Gum		NS	NS	NS	NS	0.0693
Experiment 2						
1	0	30.83	152.8	40.72	5.203	6.085
1	0.05	30.31	167.0	47.38	5.918	7.029
1	0.1	30.55	149.3	46.61	5.575	6.536
1	0.15	30.41	166.1	44.92	5.704	6.614
SEM		0.376	7.15	2.918	0.217	0.249
Source of Variation		probabilities				
Enzyme		NS	NS	NS	NS	0.0748

[a-d] Means in a column with common superscript do not differ significantly ($P < 0.05$)

CHAPTER VI

An Evaluation of Endo-β-mannanase (Hemicell®) and Caloric Density on Broiler Performance and Partitioning of Energy Utilization into That Used for Maintenance in Broilers Aged to 7 Weeks of Age[1]

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Running head: Evaluation of endo-β-D-mannanase

Abbreviation key: NSP = non-starch polysaccharides; BM = beta mannan; GIT = gastrointestinal tract; NE = net energy

Full-Length Paper _____X_____ Research Note: _____

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ABSTRACT An experiment was conducted to evaluate the effects of dietary energy and a commercial endo-β-D-mannanase (Hemicell®) on broiler performance, carcass characteristics, gastrointestinal tract (GIT) weight and size, feed and energy requirement for body weight homeostasis, and heat production. Starter, grower, and finisher high and low energy diets were formulated accordingly so that similar calorie:protein and calorie:nutrient ratios were achieved. In floor pen part of the study, dietary enzyme supplementation did not affect starter, grower, or finisher BW whereas high energy diet improved BW continuously ($P < 0.01$). Significant enzyme x energy interaction was observed on starter, grower, and finisher broiler BW gain. Feed conversation efficiency increased with high dietary energy during starter phase ($P < 0.05$), and was better during grower and finisher phases. As a result, overall feed efficiency was also significantly better for birds consuming high energy diets. High energy diet improved breast yield and leg quarter weight. High dietary energy significantly increased GIT weight at all growth phases. In addition, increasing dietary energy increased small intestine weight ($P < 0.05$ for all phases) and percent small intestine weight : GIT weight ratio ($P = 0.0592$ for grower and $P = 0.0522$ for finisher phase). Hemicell supplementation significantly reduced feed requirement expressed as % BW at d 35 and 49 whereas energy did not have an impact. Dietary enzyme or energy supplementation did not affect basal metabolic rate or fed heat production at equalized feed consumption ($P > 0.1$).

(*Key words*: broiler, beta mannan, enzyme supplementation, metabolizable energy, maintenance energy requirement)

INTRODUCTION

In today's economic climate efficient feed conversation into broiler tissue is essential for successful poultry production. The sophisticated knowledge of the broiler's nutrient requirements and the nutrient content of the feed ingredients has helped us to progress these goals in the poultry industry. Major improvements in poultry nutrition in the future are likely to come from improvements of the nutrient utilization from feedstuffs since some feedstuffs are under or not utilized due to poor nutrient availability.

Among potential factors reducing nutrient bioavailability are the non-starch polysaccharides (NSP). Non-starch polysaccharides are complex high molecular weight carbohydrates found in the structure of plant cell walls (Classen and Bedford, 1991; Annison and Choct, 1991; Bedford and Classen, 1993; Chocht, 2002).

One polysaccharide reducing utilization of nutrients from feedstuffs is beta-mannan (BM). Mannans occur in the forms of glucomannans, galactomannans, glucogalactomannans, and glucuronomannans in plant cell walls (Aman and Graham, 1990). In BM, repeating D-mannose units with β-1,4 bonds and D-galactose units are attached in a 2:3 ratio (Carré, 2002). Several studies demonstrated the negative effects of dietary BM found in palm kernel meal, copra meal, and guar gum and meal (Ray et al., 1982; Teves et al., 1988; Furuse and Mabayo, 1996).

Compared to cereal grains, plant protein concentrates have much more beta-mannan in their structure. Therefore as the dietary proportion of plant proteins increases, one would expect that enzyme supplementation have the potential to improve the nutrient value. As one of the most commonly used poultry feedstuffs all around the world, soybean meal also has been found to contain small quantities of galactomannan polysaccharides

(Whistler and Saarrino, 1957). Analysis showed that, depending on its quality, soybean meal may contain 1.3 to 1.7% beta-mannan on DM basis (unpublished lab analysis by ChemGen).

Enzyme supplementation of poultry rations have been widely reviewed and well documented to improve efficiency of converting feedstuffs into broiler tissue (Annison and Choct, 1991; Campbell and Bedford, 1992; Bedford and Morgan, 1996; Marquardt et al., 1996; Chocht, 2001). Reported benefits include improved weight gain, feed:gain ratio, and ME as well as reduced excreta and P output, water intake, digesta viscosity, and gastrointestinal tract (GIT) size (Friesen et al., 1992; Brenes et al., 1993; Annison et al., 1995; Marquardt et al., 1996).Studies indicate that the negative effects of the beta galacto-mannan, supplemented to the broiler diets, include reduced weight gain and feed/gain ratio (Ray et al. 1982). Also, work conducted by Teves et al. (1988) established that bacterial mannase treatment increases the nutritional value of copra meal in broiler diets. Similarly, recent unpublished data provided by ChemGen demonstrate that, broilers on beta mannase (Hemicell®) supplemented diets have better performance both at low and high energy levels of diets.

Composition of the ration provided to the birds affects the body composition. Research indicates that increasing the energy density of the ration increases abdominal fat pad weight and increasing calorie to protein ratio in broiler diets increases fat deposition whereas decreasing the calorie to protein ratio has the opposite effect (Bartov et al., 1974).

Today's poultry industry utilizes metabolizable energy (ME) as the reference standard for ration formulation. However, ME is not the final energy quanta available to

the bird and 40-60 % of ME is lost as heat in growing chicks. Studies examining the impact of treatment on energetic efficiency would ideally report net energy. Maintenance energy can constitute a major part of the energy needs of the chicken. Maintenance energy requirement, which is used to maintain body functions with no energy gain, reportidly accounts for approximately 70% of the adult chicken's total energy needs (Hurwitz et al., 1978). A major portion of the maintenance energy expenditure is basal metabolism, accounting for an estimated 85% of the maintenance energy needs (Brody, 1964).

Galactomannan polysaccharide is one of the most viscous polysaccharides and is five times as viscous as starch on a weight basis (unpublished lab analysis by ChemGen). Enzyme studies (Ikegami et al, 1990; Salih et al., 1991; Almirall et al., 1995; Annison et al., 1995) indicated that as viscosity in the gastrointestinal tract increases, the rate of diffusion of solutes is reduced. Under such condition, the ability of the gut to physically mix gastrointestinal contents is compromised and may result in poor performance, higher gut-enterocyte turnover rate and elevated endogenous enzyme synthesis rates (Jacobs, 1983; Morgan et al., 1985; Jorgensen et al., 1996). Increased gastrointestinal tract size, gut-enterocyte turnover, and endogenous enzyme synthesis potentially increase bird maintenance energy requirements.

Unpublished lab analysis of dehulled soybean meal indicates a dry matter content of 1.3-1.7 % β mannan. Same lab research determined the the hull portion of the soybean contains 10-15 % β mannan on a dry matter bases. Additionally field data by ChemGen demonstrated that Hemicell supplementation to diets containing 30% soybean meal and 60% corn improved the adjusted feed conversion by 7.7 points. Such benefit may be

reduce maintenance energy requirements ,and decreased viscosity in the gastrointestinal tract. Therefore, objectives this project include quantification of Hemicell supplementation effects on bird maintenance energy and growth requirements in 1, 3, 5, and 7 week old broilers. More specifically, this study seeks to investigate the efficacy of beta-mannase supplementation of broiler diets varying in energy levels on broiler performance up to 7 weeks and to determine the partitioning of energy requirements for body weight homeostasis of broilers aged 1, 3, 5, and 7 weeks fed diets with and without Hemicell supplementation at two caloric densities.

MATERIALS AND METHODS

Floor Pen Study

This study was conducted to evaluate the effects of energy and enzyme supplementation on broiler performance and carcass parameters. Treatment format contained starter, grower, and finisher components and during these three stages, treatments were examined in a factorial arrangement with two dietary energy and two enzyme levels (Table 1). This provided a total of 4 dietary treatment profiles. Starter phase included 840 day-old CobbxCobb broiler birds. Four hundred and twenty day-old CobbxCobb birds were received from a commercial hatchery with two week intervals (840 total) and randomly distributed into 12 pens. During floor pen part of the trial, starter (0-21 days), grower (21-35), and finisher (35-49) feed consumption, individual body weight at 21, 35, and 49 day of ages, and mortalities were recorded. Individual body weights of birds were recorded whenever birds were removed from pens for chamber studies for feed conversion correction. On days 21, 35, and 49, eight predetermined birds per pen were evaluated for carcass dressing percentage and gastrointestinal tract size and weight. Because this part of the study also served as a pool for metabolic phase of the

study and eight birds from each pen were processed for GIT size and weight at the end of each phase, grower and finisher phases contained 624 and 468 birds respectively.

Metabolic Study

The treatment format contains four 5-day trial. These metabolic chamber periods were conducted on days 7-12, 21-26, 35-40, and 49-54. During these 4 periods, treatments were examined in a factorial arrangement with two energy (high and low), two enzyme (control and Hemicell®, 1 lb/ton), and three feeding levels for a total of 12 treatments. Treatment identifications for metabolic chamber trials were presented in Table 2.

At 7, 21, 35, and 49 days of the experiment, 60 birds (15 birds per treatment) were selected and randomly assigned to the feeding levels within each treatment. Maintenance feeding level was estimated by using the data collected in our lab (Mittelstaedt 1990). There were five replicates for each treatment. Birds were kept in the chambers for adaptation and then fasted 36 hours for initial BMR data. Following first BMR period, birds were fed based on their dietary treatments and feeding levels (fasting, maintenance, and *ad libitum*). A second BMR was performed on these birds following feeding period. They were fasted for 36 hours and BMR heat production was recorded.

The chambers were checked twice daily for mortality, general conditions, temperature, lighting, water and feed conditions and any unanticipated events were documented. After completion of the chamber trials, all birds will be sacrificed and stored in plastic bags at –20 °C for scanning and body composition evaluation.

Data Analysis

The data were analyzed using ordinary least squares (SAS, 1991). The model included dietary energy and Hemicell®, and feeding levels as main effects. Interaction between main effects was included in the model. Mean separation was accomplished using Least Significant Difference (Steel and Torrie, 1960). Regression technique was used to estimate feed consumption for body weight homeostasis. Carbon dioxide production and O_2 consumption were regressed against elapsed time with equations for each individual chamber being integrated to compute CO_2 production and O_2 consumption for the fed and BMR time periods.

RESULTS AND DISCUSSIONS

Effects of enzyme supplementation and energy on broiler BW, BW gain, and feed:gain ratio were presented in Table 3. Dietary enzyme supplementation did not affect starter, grower, or finisher BW. A positive response for dietary energy was noted on BW and BW gain throughout the study. High energy diet improved BW at each growth phase ($P < 0.01$). Feed conversation efficiency increased with high dietary energy during starter phase ($P < 0.05$), and numerically better during grower and finisher phases. As a result, overall feed efficiency was also significantly better for birds consuming high energy diets. Similar to our results, a study conducted by Donaldson (1985) demonstrated that increasing dietary fat at constant calorie:protein ratio improved growth and feed conversion and had no effect on body fat. Composition of the ration particularly calorie to protein ratio and energy density also profoundly affect body composition. Increasing calorie to protein ratio in diets increased fat deposition while decreasing calorie to protein ratio decreased fat deposition (Bartov et al. 1974; Farrell, 1974). Bartov *et al.* (1974) reported that increasing the energy density of the ration during the first seven days of a

broiler's life increased abdominal depot fat weight of broilers at 7 weeks of age. Increasing energy to protein ratio in diets also increased fat deposition while decreasing energy to protein ratio decreased fat deposition in broilers (Bartov *et al.*, 1974; Farrell, 1974). Rosebrough *et al.* (1992) also reported that increasing energy to protein ratios in the rations of male broiler chicks from 7 to 28 days of age decreased growth and feed consumption as a consequence of decreased plasma growth hormone and insulin-like growth factor I. The increased energy to protein ratio also was observed to increase lipogenesis and cellular energy expenditure in support of Na^+/K^+ transport.

In our study, floor pens also served as pool for metabolic chamber study and periodically 60 birds were removed from floor pens at day 7, 21, 35, and 49. Additionally, due to the limitations of the facility, pen sizes were larger than desired size and replications were limited to three. These may be the potential reasons to explain the lack of enzyme effect on broiler performance.

Effects of enzyme supplementation and energy on starter (d 21) , grower (d-35), and finisher (d-49) hot carcass weight, dressing percentage, chilled carcass weight, breast yield, percent breast yield (based on chilled carcass weight), leg quarter yield, percent leg quarter yield (based on chilled carcass weight), and abdominal fat pad weight were presented in Tables 4-6. Since BW and dressing percentage was the driving factors on carcass parameters, they will be discussed first. Neither enzyme supplementation, nor energy had an impact on starter dressing percentage. There was a enzyme x energy interaction on grower and finisher dressing percentage. Hemicell® seemed to increase dressing percentage of birds consuming low energy diets. In general, parallel to performance data, high energy diet improved breast yield and leg quarter weight. Dietary

energy also tended to increase grower (P = 0.0616) and finisher percent breast yield. Enzyme supplementation seemed to increase finisher phase percent leg quarter yield and reduce grower phase percent breast yield.

Effects of enzyme supplementation and energy on starter (d 21), grower (d-35), and finisher (d-49) broiler total GIT weight, percent GIT weight : BW, small intestine weight, percent small intestine weight : GIT weight, small intestine length, and pancreas weight were presented in Tables 7-9. High dietary energy significantly increased GIT weight at all growth phases. In addition, increasing dietary energy increased small intestine weight ($P < 0.05$ for all phases) and percent small intestine weight : GIT weight ratio (P = 0.0592 for grower and P = 0.0522 for finisher phase). Dietary energy also either numerically or statistically increased pancreas weight during grower and finisher phases indicating more enzyme secretion and better digestibility and absorption.

Dietary enzyme supplementation and energy effects on d 7, 21, 35, and 49 BW, feed required for body weight homeostasis expressed as % BW, BMR heat production, fed heat production, % body lean and fat tissue were displayed in Tables 10-13. Birds used in chamber phase did not differ in their initial BW. Feed requirement to maintain BW homeostasis was expressed as % BW feed consumption. As chickens age, the weekly proportionate increases in body weight declines and it would be expected that the relative amount of energy required for growth per unit metabolic size also declines. As the birds aged from d 7 to 49, feed requirement decreased as previously demonstrated by Mittelstaedt (1990). Mittelstaedt (1990) determined the maintenance feed required by broilers ranging in weight from 500 to 2500 g maintained on a diet of 2791 Kcal ME_n/kg and 21.5 % crude protein (CP) at 24 C and observed that the maintenance feed

requirement decreased quadratically as the weight of the birds increased. In our study, Hemicell supplementation significantly reduced feed requirement expressed as % BW at d 35 and 49 whereas energy did not have an impact. Dietary enzyme or energy supplementation did not affect basal metabolic rate or fed heat production at equalized feed consumption ($P > 0.1$). Although it was not significant, birds on high energy diets had consistently higher basal metabolic heat production throughout their growth curve. Birkelo et al. (1991) using respiration calorimetry estimated fasting heat production of beef cattle. Animals on the high plane of nutrition (2.2 x maintenance) showed a 7 % increase in their fasting heat production compared to those on low (1.2 x maintenance) plane of nutrition. Macleod et al. (1988) compared the fasting heat production and maintenance energy requirement of lean and fat lines of broilers. The lean lines showed a higher fasting heat production and maintenance energy requirement than the fat lines (996 and 812 KJ/d Vs 1058 and 887 KJ/d, respectively). The fat and lean lines had similar energy retention but differ in partitioning of retained energy into carcass fat and protein (37 Vs 27 % of retained energy stored as carcass protein and 63 Vs 73 % of retained energy stored as carcass fat in lean and fat lines, respectively). Emans (1987) reported that chicken maintenance requirement is directly proportional to body protein mass and no energy is required to maintain body lipid. Ledger and Sayers, 1977) also suggested that prior growth rate might have an impact on basal metabolic rate estimates. Neither energy nor Hemicell® supplementation affected % body lean or fat tissue at day 7, 21, and 35. Dietary energy tended to increase body fat and decrease lean mass at d 49. Study conducted by Donaldson (1985) also demonstrated that increasing dietary fat at constant calorie:protein ratio improved growth and feed conversion and had no effect on

body fat. He concluded that calorie:protein ratio affects body fat by increasing lipogenic activity as the ratio is increased. He also concluded that the depressed lipogenesis caused by dietary fat appears to be offset by increased availability of fatty acids from the diet for deposition in adipose tissue. Therefore added dietary fat did not change body fat content.

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Table 1. Starter, grower, and finisher diets high and low in energy with similar ME/Crude protein ratio used in the study

	Starter		Grower		Finisher	
	Low energy	High energy	Low energy	High energy	Low energy	High energy
Ingredients	(%)	(%)	(%)	(%)	(%)	(%)
Corn	60.38	54.81	68.91	63.75	71.80	68.40
Soybean meal	35.18	37.76	27.05	29.25	22.58	24.30
Fat, vegetable	0.90	3.81	1.06	3.91	2.52	4.75
Lysine	0.06	0.03	0.05	0.02	0.05	0.02
L-methionine	0.18	0.19	0.08	0.09	0.08	0.07
Dicalcium phosphate	1.79	1.84	1.27	1.36	1.33	1.11
Calcium carbonate	0.85	0.86	1.05	1.02	1.09	0.84
Choline chloride	0.05	0.05	0.05	0.06	0.05	0.05
Copper sulphate	0.03	0.03	0.03	0.03	0.03	0.03
Salt	0.42	0.46	0.29	0.32	0.31	0.27
Vitamin premix[1]	0.05	0.05	0.05	0.06	0.05	0.05
Mineral premix[2]	0.10	0.10	0.10	0.12	0.10	0.10
Santoquin®[3]	0.01	0.01	0.01	0.01	0.01	0.01
Calculated analysis						
ME, kcal/kg	2950.42	3050.97	3050.13	3150.40	3150.42	3250.07
Crude protein, %	21.84	22.60	18.71	19.31	16.83	17.36
ME/CP ratio	135.08	135.02	163.06	163.13	187.20	187.25
Methionine	0.57	0.58	0.44	0.45	0.41	0.41
Lysine	1.21	1.25	0.97	1.00	0.85	0.86
Sulphur amino acids	0.88	0.91	0.70	0.73	0.65	0.65
Calcium	1.00	1.03	0.89	0.91	0.92	0.75
Available phosphorus	0.45	0.46	0.34	0.36	0.35	0.31

[1]Supplied per kilogram of diet: vitamin A, 38,500 IU; vitamin D_3, 11,000 IU; vitamin E, 55 IU; vitamin B_{12}, .066 mg; riboflavin, 33 mg; niacin, 165 mg; d-pantothenic acid, 55 mg; menadione, 11 mg; folic acid, 3.3 mg; pyridoxine, 13.75 mg; thiamin, 6.66 mg; d-biotin, 0.28 mg.

[2]Supplied per kilogram of diet: manganese, 120 mg; zinc, 100 mg, copper, 10 mg, iodine, 2.5 mg; calcium, 135 mg.

[3]Santoquin is an ethoxyquin compound, a chemical preservative produced by Novus Int., and its recommended uses include addition as a preservative mixed in the final feed, inclusion as an ingredient in pre-mixes and concentrates, and addition to suitable fats or other liquids prior to addition to the feed

Table 2. Dietary treatments for metabolic chamber trials at days 7, 21, 35, and 49

Treatments	Energy*, kcal/kg	Beta mannanase**	Feeding Levels***
1	High	+	0 (Fasting)
2	High	+	Maintenance
3	High	+	*Ad libitum*
4	High	-	0 (Fasting)
5	High	-	Maintenance
6	High	-	*Ad libitum*
7	Low	+	0 (Fasting)
8	Low	+	Maintenance
9	Low	+	*Ad libitum*
10	Low	-	0 (Fasting)
11	Low	-	Maintenance
12	Low	-	*Ad libitum*

*High-low energy levels for starter, grower, and finisher phages will be 2950-3050, 3050-3150, and 3150-3250 respectively.

**Beta mannanase (Hemicell®) will be added 1 lb/ton feed

***Maintenance and 2x Maintenance will be calculated based on previously published data (Mittelstaedt 1990)

Table 3. Effects of enzyme supplementation and energy on broiler BW, BW gain, and feed:gain ratio.

Treatment		Variables									
			Starter			Grower			Finisher		Overall
Hemicell®	Energy	BW (g)	BW gain (g)	Feed:Gain (g:g)	BW (g)	BW gain (g)	Feed:Gain (g:g)	BW (g)	BW gain (g)	Feed:Gain (g:g)	Feed:Gain (g:g)
-	High	632a	592a	1.458	1710	1060a	1.820	2713	976b	2.405	1.869
-	Low	559b	519b	1.580	1569	972c	1.903	2592	1030a	2.323	1.931
+	High	611a	572a	1.464	1711	1052a	1.875	2762	1049a	2.334	1.875
+	Low	582b	543b	1.564	1607	1009b	1.861	2532	916c	2.520	1.958
Hemicell®											
-		596	556	1.519	1639	1016	1.862	2652	1003	2.364	1.900
+		597	557	1.514	1659	1031	1.868	2647	983	2.421	1.916
Energy											
High		622	582	1.461	1710a	1057	1.847	2737a	1012	2.370	1.872
Low		571	531	1.572	1588b	991	1.882	2562b	973	2.421	1.945
Source of Variation		probabilities									
Hemicell		NS	NS	NS	NS	NS	NS	NS	NS	NS	NS
Energy		0.0001	0.0001	0.0038	0.0001	0.0001	NS	0.0001	0.0216	NS	0.0433
Hemicell x Energy		0.0267	0.0271	NS	NS	0.0500	NS	0.0674	0.0001	NS	NS

[a-c] Means in a column with common superscript do not differ significantly ($P < 0.05$)

Table 4. Effects of enzyme supplementation and energy on starter broiler (d 21) hot carcass weight, dressing percentage, chilled carcass weight, breast yield, percent breast yield (based on chilled carcass weight), leg quarter yield, percent leg quarter yield (based on chilled carcass weight), and abdominal fat pad weight

Treatments		Variables							
Hemicell®	Energy	Hot carcass weight (g)	Dressing percentage (%)	Chilled carcass weight (g)	Breast yield (g)	Percent breast yield (%)	Leg quarter yield (g)	Percent leg quarter yield (%)	Abdominal fat pad weight (g)
-	High	416.8	60.7	439.6	81.2	18.43	146.6	33.56	4.84
-	Low	411.6	61.9	432.8	80.2	18.52	143.2	33.14	4.60
+	High	425.0	61.2	446.2	81.9	18.39	148.2	33.29	4.98
+	Low	393.1	61.0	412.7	73.0	17.79	136.7	33.27	5.17
Hemicell®									
-		414.2	61.3	436.2	80.7	18.47	144.9	33.35	4.72
+		409.1	61.1	429.4	77.5	18.09	142.5	33.28	5.08
Energy									
High		420.9a	60.9	442.9a	81.5a	18.41	147.4a	33.42	4.91
Low		402.4b	61.4	422.7b	76.6b	18.15	140.0b	33.20	4.88
Source of Variation		probabilities							
Hemicell		0.5413	0.7557	0.4410	0.1422	0.2112	0.4334	0.8527	0.3264
Energy		0.0322	0.3670	0.0247	0.0277	0.4024	0.0222	0.5251	0.9324
Hemicell x Energy		0.1411	0.1647	0.1582	0.0900	0.2989	0.2403	0.5861	0.5852

[a-c] Means in a column with common superscript do not differ significantly ($P < 0.05$)

Table 5. Effects of enzyme supplementation and energy on grower broiler (d 35) hot carcass weight, dressing percentage, chilled carcass weight, breast yield, percent breast yield (based on chilled carcass weight), leg quarter yield, percent leg quarter yield (based on chilled carcass weight), and abdominal fat pad weight

Treatments		Variables							
Hemicell®	Energy	Hot carcass weight (g)	Dressing percentage (%)	Chilled carcass weight (g)	Breast yield (g)	Percent breast yield (%)	Leg quarter yield (g)	Percent leg quarter yield (%)	Abdominal fat pad weight (g)
-	High	1148.4a	69.7a	1194.2a	252.6a	21.29	394.6a	33.12	18.53
-	Low	1030.9b	68.6b	1074.4b	225.4b	20.73	357.5b	33.25	16.59
+	High	1113.7a	68.8b	1155.8a	238.0ab	20.49	379.3ab	32.90	18.04
+	Low	1115.2a	69.4ab	1163.7a	233.5b	19.95	389.1a	33.54	19.61
Hemicell®									
-		1089.7	69.1	1134.3	239.0	21.00a	376.1	33.19	17.56
+		1114.5	69.1	1159.8	235.8	20.22b	384.2	33.22	18.83
Energy									
High		1131.1	69.3	1175.0	245.3	20.89	387.0	33.01	18.28
Low		1073.1	69.0	1119.1	229.5	20.34	373.3	33.40	18.10
Source of Variation		probabilities							
Hemicell		0.2221	0.9363	0.2121	0.5727	0.0077	0.2892	0.9116	0.2137
Energy		0.0050	0.3721	0.0075	0.0038	0.0616	0.0961	0.2293	0.8954
Hemicell x Energy		0.0065	0.0038	0.0044	0.0461	0.9807	0.0064	0.4647	0.1275

[a-c] Means in a column with common superscript do not differ significantly ($P < 0.05$)

Table 5. Effects of enzyme supplementation and energy on grower broiler (d 35) hot carcass weight, dressing percentage, chilled carcass weight, breast yield, percent breast yield (based on chilled carcass weight), leg quarter yield, percent leg quarter yield (based on chilled carcass weight), and abdominal fat pad weight

Treatments		Variables							
Hemicell®	Energy	Hot carcass weight (g)	Dressing percentage (%)	Chilled carcass weight (g)	Breast yield (g)	Percent breast yield (%)	Leg quarter yield (g)	Percent leg quarter yield (%)	Abdominal fat pad weight (g)
-	High	1148.4a	69.7a	1194.2a	252.6a	21.29	394.6a	33.12	18.53
-	Low	1030.9b	68.6b	1074.4b	225.4b	20.73	357.5b	33.25	16.59
+	High	1113.7a	68.8b	1155.8a	238.0ab	20.49	379.3ab	32.90	18.04
+	Low	1115.2a	69.4ab	1163.7a	233.5b	19.95	389.1a	33.54	19.61
Hemicell®									
-		1089.7	69.1	1134.3	239.0	21.00a	376.1	33.19	17.56
+		1114.5	69.1	1159.8	235.8	20.22b	384.2	33.22	18.83
Energy									
High		1131.1	69.3	1175.0	245.3	20.89	387.0	33.01	18.28
Low		1073.1	69.0	1119.1	229.5	20.34	373.3	33.40	18.10
Source of Variation		probabilities							
Hemicell		0.2221	0.9363	0.2121	0.5727	0.0077	0.2892	0.9116	0.2137
Energy		0.0050	0.3721	0.0075	0.0038	0.0616	0.0961	0.2293	0.8954
Hemicell x Energy		0.0065	0.0038	0.0044	0.0461	0.9807	0.0064	0.4647	0.1275

[a-c] Means in a column with common superscript do not differ significantly ($P < 0.05$)

Table 7. Effects of enzyme supplementation and energy on starter broiler (d 21) total GIT weight, percent GIT weight : BW, small intestine weight, percent small intestine weight : GIT weight, and small intestine length

Treatments		Variables				
Hemicell®	Energy	Total GIT weight (g)	GIT Weight : BW (%)	Small intestine weight (g)	Small intestine weight : GIT weight (%)	Small intestine length (cm)
-	High	63.15	10.321	30.80	48.810	158.3
-	Low	59.07	10.109	28.34	47.877	150.7
+	High	62.84	10.106	30.61	48.681	154.5
+	Low	57.42	10.383	27.58	47.894	154.8
Hemicell®						
-		61.11	10.215	29.57	48.343	154.5
+		60.13	10.245	29.10	48.288	154.7
Energy						
High		63.00a	10.214	30.71a	48.746	156.4
Low		58.24b	10.246	27.96b	47.885	152.8
Source of Variation		probabilities				
Hemicell		0.4538	0.9021	0.5190	0.9370	0.9189
Energy		0.0005	0.8956	0.0003	0.2156	0.1759
Hemicell x Energy		0.6279	0.4093	0.7117	0.9202	0.1555

[a-c] Means in a column with common superscript do not differ significantly ($P < 0.05$)

Table 8. Effects of enzyme supplementation and energy on grower broiler (d 35) total GIT weight, percent GIT weight : BW, small intestine weight, percent small intestine weight : GIT weight, small intestine length, and pancreas weight

Treatments		Variables					
Hemicell®	Energy	Total GIT weight (g)	GIT Weight : BW (%)	Small intestine weight (g)	Small intestine weight : GIT weight (%)	Small intestine length (cm)	Pancreas weight (g)
-	High	107.02	6.570	53.05	49.564	151.6	4.076
-	Low	99.97	6.735	48.32	48.371	146.5	3.816
+	High	106.31	6.669	53.27	50.084	150.3	4.346
+	Low	104.58	6.603	50.91	48.645	148.8	4.024
Hemicell®							
-		103.49	6.653	50.68	48.967	149.1	3.945
+		105.44	6.636	52.09	49.365	149.6	4.185
Energy							
High		106.67	6.620	53.16a	49.824	151.0	4.211
Low		102.27	6.669	49.61b	48.508	147.7	3.920
Source of Variation		probabilities					
Hemicell		0.3808	0.8763	0.2791	0.5658	0.7738	0.2909
Energy		0.0503	0.6432	0.0073	0.0592	0.0723	0.1998
Hemicell x Energy		0.2593	0.3126	0.3911	0.8662	0.3574	0.8941

[a-c] Means in a column with common superscript do not differ significantly ($P < 0.05$)

Table 9. Effects of enzyme supplementation and energy on finisher broiler (d 49) total GIT weight, percent GIT weight : BW, small intestine weight, percent small intestine weight : GIT weight, small intestine length, and pancreas weight

Treatments		Variables					
Hemicell®	Energy	Total GIT weight (g)	GIT Weight : BW (%)	Small intestine weight (g)	Small intestine weight : GIT weight (%)	Small intestine length (cm)	Pancreas weight (g)
-	High	148.34	5.463ab	70.97	47.885	157.0	4.970
-	Low	142.01	5.661a	67.39	47.273	157.5	4.757
+	High	150.50	5.593a	73.44	48.799	158.9	5.102
+	Low	136.11	5.263b	64.14	47.060	152.8	4.559
Hemicell®							
-		145.17	5.562	69.18	47.579	157.3	4.863
+		143.30	5.428	68.79	47.929	155.8	4.831
Energy							
High		149.42a	5.528	72.20	48.342	158.0	5.036a
Low		139.06b	5.462	65.76	47.166	155.1	4.658b
Source of Variation		probabilities					
Hemicell		0.5433	0.1587	0.8322	0.5587	0.5552	0.8508
Energy		0.0011	0.4883	0.0007	0.0522	0.2427	0.0063
Hemicell x Energy		0.2173	0.0100	0.1447	0.3766	0.1921	0.2444

[a-c] Means in a column with common superscript do not differ significantly ($P < 0.05$)

Table 10. Effects of enzyme supplementation and energy on 7 d initial body weight, percent BW feed requirement for BW homeostasis, BMR heat production, fed heat production, percent lean tissue, and percent fat tissue analysis

Treatments		Variables					
Hemicell®	Energy	Initial BW (g)	Feed for maint. (% BW)	BMR HP (kcal/kg/h)	Fed HP[1] (kcal/kg/h)	Lean (% BW)	Fat (% BW)
-	High	81.0	23.47	6.15	17.03	89.64	9.06
-	Low	81.4	22.01	5.61	17.63	88.38	9.95
+	High	78.8	21.63	6.54	17.02	89.30	9.13
+	Low	76.1	25.00	5.55	19.69	89.87	8.83
Hemicell®							
-		81.2	22.74	5.87	17.33	89.01	9.51
+		77.4	23.31	6.04	18.35	89.59	8.98
Energy							
High		79.9	22.55	6.35	17.02	89.47	9.09
Low		78.7	23.50	5.58	18.66	89.13	9.39
Source of Variation		probabilities					
Hemicell		NS	NS	NS	NS	NS	NS
Energy		NS	NS	NS	NS	NS	NS
Hemicell x Energy		NS	NS	NS	NS	NS	NS

[a-c] Means in a column with common superscript do not differ significantly ($P < 0.05$)

[1] Feed consumption was used as covariant

Table 11. Effects of enzyme supplementation and energy on 21 d initial body weight, percent BW feed requirement for BW homeostasis, BMR heat production, fed heat production, percent lean tissue, and percent fat tissue analysis

Treatments		Variables					
Hemicell®	Energy	Initial BW (g)	Feed for maint. (% BW)	BMR HP (kcal/kg/h)	Fed HP[1] (kcal/kg/h)	Lean (% BW)	Fat (% BW)
-	High	478.9	16.95	6.33	8.32	90.84	7.58
-	Low	411.3	15.76	6.00	9.68	88.88	9.50
+	High	427.4	17.49	5.50	8.37	90.15	8.43
+	Low	429.2	15.95	5.43	9.08	88.75	9.88
Hemicell®							
-		445.1	16.36	6.17	9.00	89.86	8.54
+		428.3	16.71	5.47	8.72	89.45	9.15
Energy							
High		453.1	17.22a	5.91	8.34	90.49	8.00
Low		420.2	15.85b	5.71	9.39	88.82	9.69
Source of Variation		probabilities					
Hemicell		NS	NS	NS	NS	NS	NS
Energy		NS	0.0001	NS	NS	NS	NS
Hemicell x Energy		NS	NS	NS	NS	NS	NS

[a-c] Means in a column with common superscript do not differ significantly ($P < 0.05$)

[1] Feed consumption was used as covariant

Table 12. Effects of enzyme supplementation and energy on 35 d initial body weight, percent BW feed requirement for BW homeostasis, BMR heat production, fed heat production, percent lean tissue, and percent fat tissue analysis

Treatments		Variables					
Hemicell®	Energy	Initial BW (g)	Feed for maint. (% BW)	BMR HP (kcal/kg/h)	Fed HP[1] (kcal/kg/h)	Lean (% BW)	Fat (% BW)
-	High	1381.5	12.15	2.50	3.79	88.85	9.65
-	Low	1393.5	12.33	2.34	4.87	85.55	13.05
+	High	1603.0	11.47	2.26	4.62	85.25	13.30
+	Low	1425.3	11.10	2.44	4.13	84.25	14.20
Hemicell®							
-		1387.5	12.24a	2.42	4.33	87.20	11.35
+		1514.2	11.29b	2.35	4.38	84.75	13.75
Energy							
High		1492.3	11.80	2.38	4.21	87.05	11.48
Low		1409.4	11.72	2.39	4.50	84.90	13.62
Source of Variation		probabilities					
Hemicell		0.0893	0.0001	NS	NS	NS	NS
Energy		NS	NS	NS	NS	NS	NS
Hemicell x Energy		NS	NS	NS	NS	NS	NS

[a-c] Means in a column with common superscript do not differ significantly ($P < 0.05$)

[1] Feed consumption was used as covariant

Table 13. Effects of enzyme supplementation and energy on 49 d initial body weight, percent BW feed requirement for BW homeostasis, BMR heat production, fed heat production, percent lean tissue, and percent fat tissue analysis

Treatments		Variables					
Hemicell®	Energy	Initial BW (g)	Feed for maint. (% BW)	BMR HP (kcal/kg/h)	Fed HP[1] (kcal/kg/h)	Lean (% BW)	Fat (% BW)
-	High	2354.6	7.82	2.78	4.87	87.98	10.56
-	Low	2321.4	7.63	2.71	4.63	90.30	8.18
+	High	2379.6	6.27	2.82	4.82	89.06	9.44
+	Low	2369.7	6.28	2.61	3.88	88.80	9.73
Hemicell®							
-		2338.0	7.72a	2.75	4.75	89.14	9.37
+		2374.6	6.27b	2.71	4.35	88.93	9.59
Energy							
High		2367.1	7.04	2.80	4.84	88.52	10.00
Low		2345.5	6.95	2.66	4.26	89.55	8.96
Source of Variation		probabilities					
Hemicell		NS	0.0003	NS	NS	NS	NS
Energy		NS	NS	NS	NS	0.0654	0.0644
Hemicell x Energy		NS	NS	NS	NS	0.0541	NS

[a-c] Means in a column with common superscript do not differ significantly ($P < 0.05$)

[1] Feed consumption was used as covariant

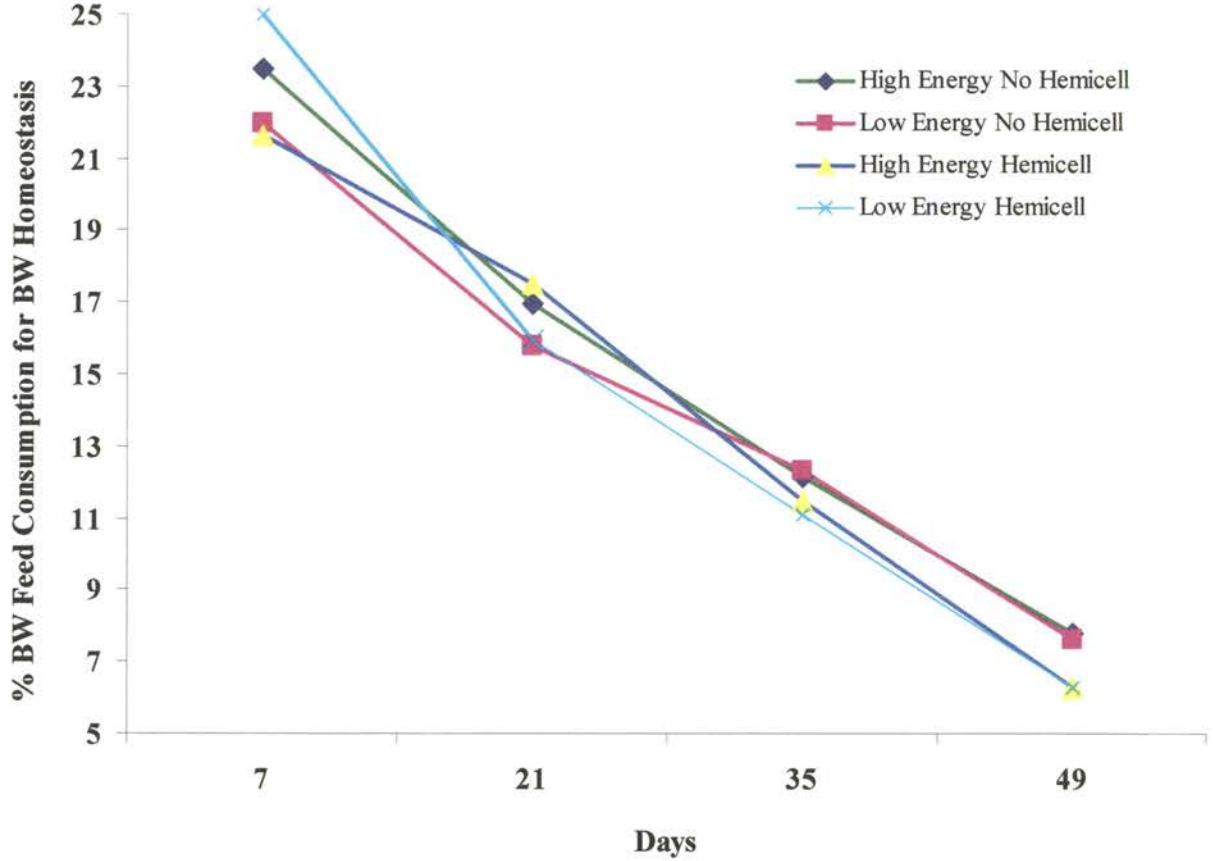

Figure 1. Effects of Energy and Hemicell on Maintenance feed requirement expressed as % BW feed consumption

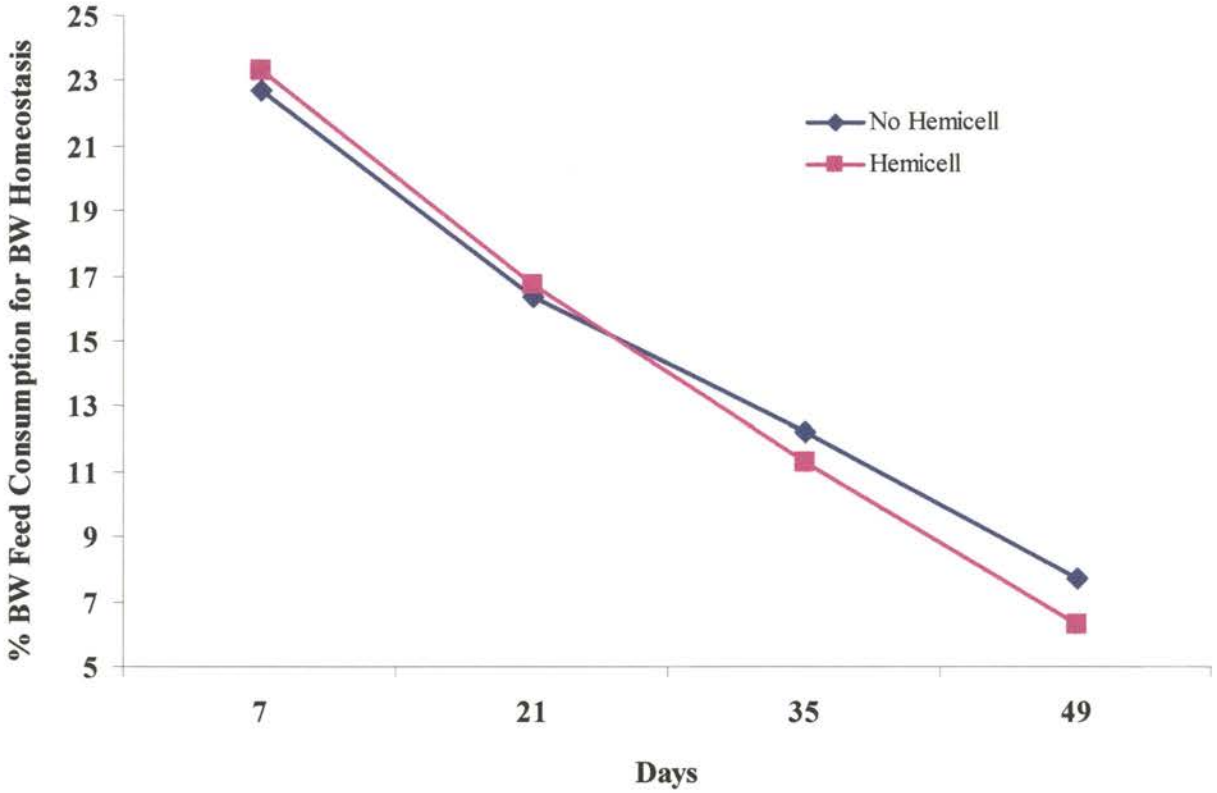

Figure 2. Hemicell Effect on Maintenance feed requirement expressed as % BW feed consumption

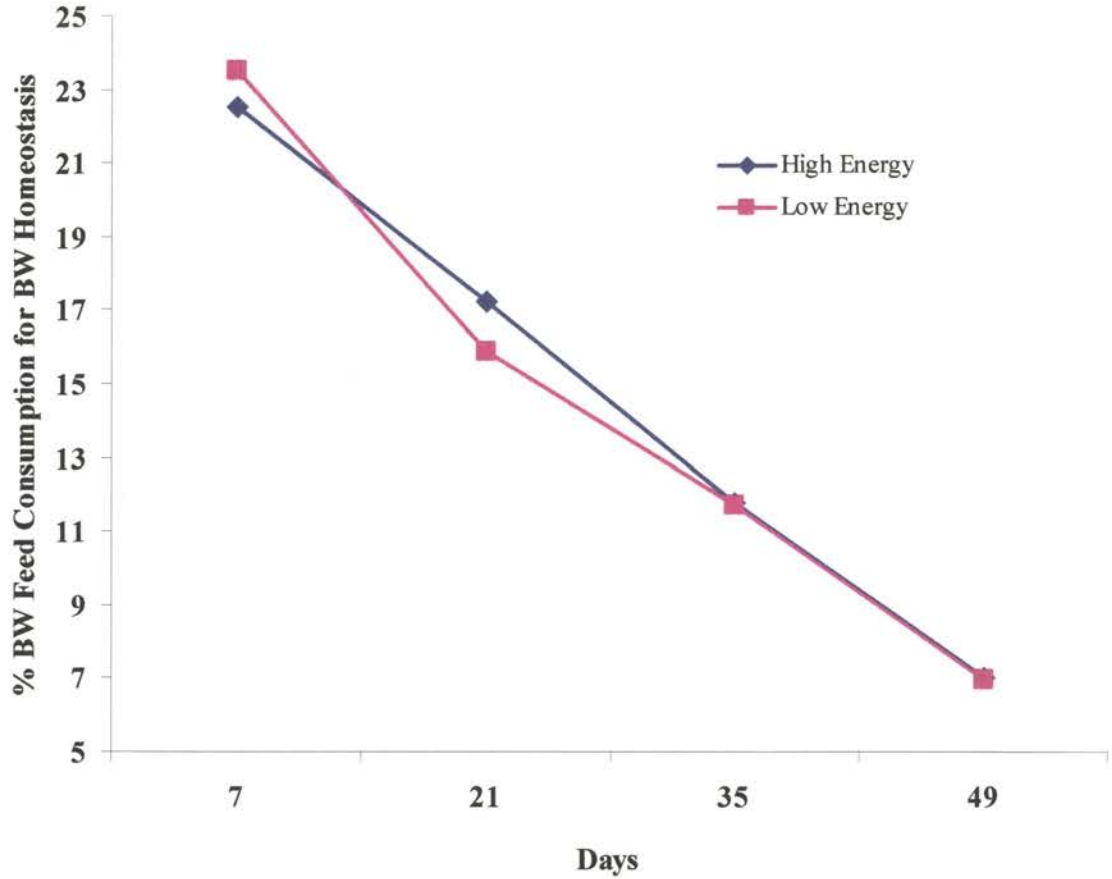

Figure 3. Energyl Effect on Maintenance feed requirement expressed as % BW feed consumption

VITA

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Doctor of Philosophy

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