BROILER ENERGY AND OXYGEN METABOLISM AND THE EFFECT OF OXYGEN CONCENTRATION AND AMBIENT TEMPERATURE ON ASCITES INCIDENCE

By

ALI BEKER YOUSUF

Bachelor of Science in Animal Science
Addis Ababa University
Addis Ababa, Ethiopia
1981

Master of Science in Animal Production
Addis Ababa University
Addis Ababa, Ethiopia
1985

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
July, 1996

BROILER ENERGY AND OXYGEN METABOLISM

AND THE EFFECT OF OXYGEN CONCENTRATION

AND AMBIENT TEMPERATURE

ON ASCITES INCIDENCE

Thesis Approved:

Robert Teeter
Thesis Adviser

F N Owens

Charles V. Maxwell

J G Breazile

Stanley Ishmaen

Thomas C. Collins
Dean of the Graduate College

ACKNOWLEDGMENTS

I would like to extend my sincere and heart felt appreciation to the USIA for the financial support they gave me during my study. My sincere appreciation also goes to my major advisor, Dr. Robert Teeter, for his advice, guidance, expertise and encouragement throughout my graduate study. My sincere thanks also goes to the members of my committee, Dr. F. Owens, Dr. C. V. Maxwell, Dr. J. E. Breazile and Dr. S. Vanhooser for their suggestions as members of my graduate committee.

A special thanks goes to my colleagues of the OSU poultry farm, Dr. Tsegaw Belay, Dr. Chester Wiernusz, Dr. Farzad Deyhim, Dr. Makonnen Lema, Chris Harjo, Jim Casson, Mr. Leon Bloom, Mrs. Afaf Melouk and Mrs. Beverly Brandon for the valuable knowledge and assistance that they provided.

I would also like to express my heart felt appreciation to my wife Etsegenet and my sons, Eskender and Girum for their love, encouragement and companion during my graduate study at Oklahoma State University. I Would also like to express my special appreciation to my parents for their unfailing love and encouragement.

TABLE OF CONTENTS

Chapter Page

LIST OF TABLES

Table Page

Chapter V

LIST OF FIGURES

Figure Page

Chapter III

Figure Page

CHAPTER I

INTRODUCTION

Modern commercial strains of broilers, that flourish in different parts of the world, are known for their high productivity. These strains however, tend to deposit excess fat even though consumer demand is for the lean product. Research studies demonstrate that improving leanness in broilers is accompanied by slowing bird growth rate, however, this increases the cost of production (Jensen, 1982).

Energy needs for maintenance, protein and fat accretion are dynamic and change continuously during the growth of an animal. Other factors such as ambient temperature and genetic potential also have an impact on energy needs (Emans, 1987; Close, 1978 and Ferell and Jenikens. 1985). Through the years, body composition of modern broiler strains has been improved through genetic manipulations that enhance growth rate along with higher body fat content (Chambers et al. 1981). This change calls for a continuous reassessment of the nutritional requirements of broilers.

Although much has been learned about the effects of dietary factors on carcass composition, applicability of results will not be absolute without giving due attention to non-dietary factors. This is mainly due to the fact that many factors and their interactions influence carcass

composition. As more information is obtained concerning the effect of dietary factors, genetic potential, environment and their interactions may provide more accurate estimates of the partitioning of energy for maintenance, fat and protein accretion.

Evaluations concerning the efficiency of animal growth have often been based on the partitioning of metabolizable energy intake between maintenance, growth and/or production functions. Partitioning of energy intake between maintenance and production functions have been a convenient and useful means to study whole animal energy metabolism. These methods have proven useful in the development of recommendations for feeding standards. The availability of knowledge on partitioning of energy needs for production into protein and fat accretion may provide more accurate data for application.

Dietary energy comprises a major cost in poultry production. Therefore, accurate and precise data describing the energy requirements of poultry are needed to formulate more efficient and less costly diets. Most studies conducted to date have considered only one or two factors affecting energy needs for a limited time during the life of a bird. Such results have very limited application to a production environment. Research that includes a number of factors and their interaction throughout the growth period of animals should enable the determination of energy

requirements at any given condition and also aid in the production of broilers to a desired carcass composition.

Another major problem hindering optimum efficiency of production of broilers is the increasing incidence of ascites syndrome. Ascites is a pathological problem characterized by fluid accumulation in the abdominal cavity. The syndrome is associated with broilers that are genetically superior in their growth rate and that in turn demonstrate high oxygen demand. An increased metabolic rate due to cold exposure increases the demand for oxygen. The inflexible lung structure of the chicken limits the ability to meet this increased O2 demand. Compensation results in increased cardiac output. A number of factors have been demonstrated to aggravate ascites, some of these are, limited ventilation in the hatchery and during brooding, high levels of CO_2, CO or ammonia in the air, low concentration of oxygen at high altitudes and a number of nutritional factors (Wideman, 1988).

During the past years, numerous investigations have been made under various circumstances in the field and research laboratory to determine the cause of ascites. The studies suggest pulmonary hypertension (PH) to be the primary cause (Hernandez, 1987; Maxwell et al. 1986; Julian et al, 1989). Yuan et al. (1990) reported that a decrease in oxygen partial pressure induces a vasoconstriction in the pulmonary circulation. Management practices such as those limiting growth rate via feed restriction has been reported

to limit ascites incidence (Acar et al. 1995). Such a practice can be successful due to slowed growth rate, but has a negative economic impact. Improving oxygen availability, practicing a sound hygiene and the use of breeds that perform well under limited oxygen supply may provide a better result. Even though low oxygen concentration and low ambient temperature were reported to aggravate pulmonary hypertension induced ascites, there is limited information on the exact levels of oxygen and temperature that induce ascites.

With this general background, this Thesis project is intended to investigate the energy and oxygen need of broilers for body weight maintenance, protein and fat accretion. It will also investigate the effect of atmospheric oxygen and ambient temperature on ascites incidence. Through these studies it is hoped that a model may be developed for the prediction of energy and oxygen needs for protein and fat accretion at any given body weight and ambient temperature.

REFERENCES

Acar, N., F.G. Sizemore, R.L. Leach, R. F. Wideman, Jr., R.I. Owen, and G.F. Barato, 1995. Growth of broilers chickens in response to feed restriction regimens to reduce ascites. *Poultry Sci.* **74**:833-843

Chambers, J.R., Gavora, J.S. and Fortin, A., 1981. Genetic changes in meat-type chickens in the last twenty years. *Canadian Journal of Animal Science* 61:555-563.

Hernandez, A. Hypoxic ascites in broilers (1987) A review of several studies done in Colombia. *Avian Dis.* **31:** 658-661.

Jensen, J. F., 1982. Quality of poultry meat-an issue of growing importance. *World's Poultry Science Journal* 38:105-111.

Julian, R. J., McMillan, I. and Quinton, M., (1989) The effect of cold and dietary energy on right ventricular hypertrophy, right ventricular failure and ascites in meat-type chickens. *Avian Pathology,* **18:** 675-684.

Maxwell, M. H., Robertson, G.W and Spence, S. (1986) Studies on an ascites syndrome in young broilers. 1. Hematology and pathology. *Avian pathol.* **15:** 511-524.

Wideman, R F Jr. (1988). Ascites in poultry. *Monsanto Nutrition Update,* **Vol. 6:** (2), pp. 1-7.

Yuan, S.J., M.L. Tod, L.J. Rubin and M.P. Blaustein (1990) contrasting effects of hypoxia on tension in rat pulmonary and mesenteric arteries. *Am.J. Physiol.* **259;** H281-H289

CHAPTER II

REVIEW OF LITERATURE

ENERGY METABOLISM SCHEMES:

Only a portion of the gross energy (GE) is actually used in the formation of animal products such as meat, eggs, milk, etc. Considerable energy losses occur in the feces and heat through oxidative processes. Blaxter (1989) estimated the efficiency of metabolizable energy corrected for nitrogen (MEn) utilization to be 45, 68, and 60 % for meat, egg, and milk production. Mittelstaedt and Teeter (Submitted) evaluated the MEn use for broiler fat gain above maintenance for gelatin, starch and corn oil. They found efficiencies for fat synthesis of 20.4, 47 and 52, respectively.

The apparent digestible energy of the ration or individual nutrients such as carbohydrates, proteins and fats may be estimated by subtracting fecal energy from gross energy. This value is termed apparent because the endogenous losses have not been accounted for (McDonald et al. 1988). Metabolizable energy (ME) losses may be estimated by subtracting gas and urinary energy loss from apparent digestible energy yielding apparent metabolizable energy. Shortly after these molecules are absorbed into the gastrointestinal tract there is an increase in heat production referred to as the heat increment of the feed

which is given off. Therefore, ME system fails to account for bird heat production affecting precise estimation of chicken true energetic efficiency. Despite this fact, Poultry producers have been using and will continue to use the ME system in ration formulation. The fact that both urine and feces are excreted as one, determination of ME is facilitated. Subtracting heat dissipation from ME provides an estimate of net energy, or the energy actually contained in tissues or utilized for work. The net energy is available to maintain body temperature, provide energy for work and other activities, stored as adipose tissue and for growth and production.

Net energy is estimated by determining retained energy (energy balance) and may be a positive or negative quantity. Two basic techniques were used by investigators to obtain this information. The balance trial, first used by Lawes and Gilbert (1861), attempts to measure the difference between energy input and output. An alternative technique is the comparative slaughter technique, which relate energy input to changes in body composition. This technique is laborious, time consuming and needs sacrifice of animals both at the beginning and at the end of a trial. In addition, the comparative slaughter technique has several potential sources of error because it is difficult to obtain a truly representative sample of birds. Davidson and Mathieson (1965) suggested that the composition of birds killed at the beginning of a trial is probably

representative of the remainder when young birds are used. However, Fraps and Carlyl (1939) and Hanlen (1939) reported that due in part to their varying fat content, variability exists within older birds.

In growing and fattening animals the measurement of energy retention has been repeatedly reported to be critical for protein and fat accretion estimation. The energy value of whole carcass can be determined by bomb calorimeter after the initial and final slaughter groups have been homogenized. Heat production can be calculated as the difference between the energy intake and the gross energy retained or using the Brouwer equation from the liters of oxygen consumed and carbon dioxide produced (Brouwer, 1965):

HP=16.18(L oxygen consumed)+(5.02 L carbon dioxide produced).

MAINTENANCE ENERGY REQUIREMENT:

Farm animals are maintained mainly for their quality products such as egg, milk, meat, etc. Such products are attained only after the dietary maintenance requirement of the animals are exceeded. To accomplish this, data on maintenance requirements are needed.

Maintenance energy is that part of the net energy consumed that is used for maintenance of body functions (basal metabolism, activity, etc.) with the animal neither translocating, losing nor gaining protein, fat, carbohydrate or mineral matter (Armsby and Moulton, 1925). According to

Bolton (1959), ME accounts about 70 % of the total energy requirement in the adult chicken. A major portion of the maintenance energy expenditure is basal metabolism. Basal metabolism accounts for approximately 85 % of the maintenance need.

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, 1961 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 .75. Brody (1945) 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 .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 .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.

As chickens age, the weekly proportionate increases in body weight declines (Marks, 1975). It would be expected that the relative amount of energy required for growth per unit metabolic size also declines. Robins and Ballew (1984) showed that 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 .882 to the age of 23-26 days for both sexes and thereafter an exponent of .627 for the females and .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 (Emans, 1987), sex (Ferrell et al. 1974 and 1977), ambient temperature (Close, 1973), and strain (Ferrell and Jenikens, 1985). 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. 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). Mittelstaedt (1990) determined the maintenance feed required by broilers ranging in weight from 500 to 2500 g maintained on a diet of 2791 Kcal MEn/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.

ENERGY NEED FOR PROTEIN AND FAT ACCRETION: The principal aim of animal production is the production of high quality protein in the form of meat, eggs, milk etc. for human consumption. To maximize accretion of protein in muscle, a considerable amount of fat also is deposited. Protein

synthesis (protein deposition, protein in milk, eggs) requires a large amount of energy. A through understanding of the relationships between the energy requirements for protein production is prerequisite for improving production efficiency.

The energy cost for fat and protein accretion is simply the increment of feed energy required to obtain a defined increment in body protein or fat. Some results of studies conducted on energy needs for maintenance, protein and fat accretion with birds and other species is indicated in Table 1.

ENERGY NEED FOR FAT DEPOSITION: The amount of energy required for fat deposition can be estimated precisely in adult animals because the protein gain is minimal and the amount of energy needed to maintain energy balance is constant. In species such as the pig and rat, the energy cost of fat deposition ranges from about 1.4 kJ ME/kJ fat deposited for feed consisting predominantly of carbohydrate to 1.15 kJ ME/kJ (Table 1)fat deposited for feed rich in triglycerides (ARC/MRC Committee, 1974). Fat accretion is often assumed to be due to a difference in total energy retained caused either by a difference in energy intake, energy expenditure or both. There is however, a possibility that differences in body composition can be caused, or at least influenced by variation in the partition of the same amount of retained energy between fat and protein.

Table 1. Energy need for body weight maintenance, protein and fat accretion compiled from various sources.

Species	BWT (kg)	Maintenance need kcal/kg$^{.75}$/d	Energy need for Protein(KJ/KJ)	Fat (KJ/KJ)	Source
Chicken,	0.175	162.9			Balnave, 1974
WL pullets	0.445	138.2			
Broiler,	0.25 - .70	129.06			DeGroote, 1968
Male	1.9 - 2.8	134.56			Shanon & Brown, 1970
Rats (lean)	0.20 - .35		2.25	1.36	Pullar & Webster, 1977
Pigs	0.20 - .35		1.54	1.15	Close & Stanier,1984
Broilers	0.40	168.99			Pinchasov, 1970
Pullets	1.06	156.14			
	2.01	147.72			
Broiler	0.68	199.09			Jones, 1994

ENERGY NEED FOR PROTEIN DEPOSITION: Pullar and Webster (1974) in their experiment on lean and obese rats that were restricted in their feed intake found that obese type rats retained a smaller proportion of energy as protein than their lean litter mates. They suggested that regulation of feed intake in the obese rats may have been directed towards the attainment of a normal rate of lean tissue growth. Webster et al. (1979) further showed that the reduced rate of protein deposition in the obese type rats is caused not by reduced protein synthesis but by an increased degradation and catabolism of protein amino acids. Amino acids therefore enter the energy metabolism pool where they may either be used for lipogenesis or oxidized with the effect of sparing other nutrients for deposition of fat. Protein retention is therefore coupled with an alteration of the distribution between body sites (Rodcliffe and Webster, 1978).

FACTORS THAT AFFECT NUTRIENT PARTITIONING INTO PROTEIN AND FAT: A number of factors have been reported by investigators to affect protein and fat accretion. Some of these factors are feed restriction, ration composition, dietary fat supplementation, rearing temperature and genotype of the broiler strain. Consumption of feed energy above the maintenance and growth requirement of the animal

results in excess energy that is converted to fat and stored.

Maintenance requirement of an animal becomes lower as food intake increases. However, if food intake is restricted, the need for maintenance predominates and part of the food protein would be used as energy source for maintenance. In a 6 day feed restriction trial Yu et al. (1990) observed that broilers fed at hourly interval show less body fat at 8 weeks of age than chicks that were fed either once a day or every other day. Reece et al. (1985) found that meal feeding of broilers increase abdominal fat compared to *ad-lib* fed control groups.

Composition of the ration particularly calorie to protein ratio and energy density also profoundly affect body composition. Haris and Creger (1980) reported that increasing the energy density of the ration during the first seven days of life increased abdominal depot fat weight at 7 weeks of age. 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).

Fat is the cheapest and most concentrated source of available energy most often used in ration formulation to increase the caloric density of a diet and subsequently improve body weight gain and feed efficiency. However, contradictory results were reported by researchers. Deaton et al. (1981) showed that as the dietary fat supplementation

is increased, the amount of abdominal fat increased. Maurice et al. (1982) found that a high fat diet during the first week has no effect on abdominal fat. Similar results were reported by Edward et al. (1973), Griffith et al. (1977) and Whitehead and Griffin (1986). Nistan et al. (1986) observed that proportional increase in dietary fat and protein decreased carcass fat deposition in high and low fat lines of broilers.

The manner in which feed is processed also contribute to the partitioning of feed energy into carcass fat and protein. Pelleting improved feed intake and increases fatness (Fisher and Wilson, 1974; Whiting and Jenson, 1983; Leclercq, 1986). Petsi et al. (1983) observed a 23 % increase in abdominal fat upon feeding crumbled low density diet. Jenson et al. (1982) indicated that mash feeding requires more consumption time and energy than pelleted feed.

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. (1971) and Bray (1983) reported that at a moderate temperature, the correlation between temperature and total body fat content is positive. Washburn (1986) compared the effect of three temperatures, 21, 27 and 32 C on abdominal

fat of broilers. There was no difference observed in the abdominal fat at these temperatures.

Genetic variation within strain in the total amount of fat was found by Fariars et al. (1983) who estimated the heritability of total body fat to be 0.48. Nir and Lin (1982) conducted an invitro test and found that chicken of heavy lines show more lipogenic activity than leaner lines.

OXYGEN METABOLISM OF BROILERS:

Atmospheric oxygen is one of the very essential nutrients where all aerobic organisms have it free of charge. All energy released from the oxidation of carbohydrates, fats and proteins is made available in the mitochondria as reducing equivalents (H or e-). These are funneled into the respiratory chain, where they are passed down a redox gradient of carriers to their final reaction with oxygen to form water and CO_2 (Murray et al. 1993).

Though O_2 is free, if its availability is limited, then the activity of respiratory chain reactions and the amount of energy released from oxidation of food nutrients will also be limited. It should also be noted that even though the H_2O and CO_2 formed during metabolism contain chemical energy, the metabolic systems in higher organisms is not able to utilize this energy.

Energy that is available to animals from oxidation of food nutrients is utilized for maintenance and production. Bolton (1959) reported that energy required for maintenance

is the largest of all energy expenditures of the adult chicken. Maintenance energy amount to approximately 70% of the total energy requirement. It is obvious therefore, that most of the oxygen that is available to animals is used to generate energy for maintenance.

Information regarding the oxygen requirement of animals is very important to livestock producers. It is well documented that availability of insufficient atmospheric oxygen can result in pulmonary hypertension (PH) and the induction of ascites in broilers (Odom, 1993).

The partial pressure of oxygen decreases as altitude increases (Table 2). Saturation of hemoglobin with oxygen decreases as the partial pressure of oxygen decreases (Sturkie, 1986). As a short term response to such a condition, the output of the heart and the number of red blood cells production increases to satisfy the high oxygen need at tissue level. A decrease in partial pressure of oxygen at higher altitude also decreases the partial pressure of oxygen at alveolar level resulting in pulmonary vasoconstriction. This inturn results in increased resistance to blood flow in the pulmonary arteries (Burton and Smith, 1969). As a compensatory response to such a condition, the heart works hard, the wall of the right ventricle dilates and valvular insufficiency develops. As a result, there will be an increase in blood pressure inside Vena Cava that convey blood from the liver and other parts of the body to the heart. This increases the pressure in

the hepatic circulation and fluid starts to ooze out into the abdominal cavity resulting in a condition known as ascites (Wideman, 1988). Freeman (1964) reported that a newly hatched chick consume 55.6 ml/hr of oxygen. Jones, (1994) found that a 3 week broiler maintained at 21 C consume 36.3 ml oxygen / $Kg^{.75}$. Determination of the oxygen requirement as influenced by bird age, size, ambient temperature and growth and the birds ability to consume O_2 under varying atmosphere may enable one to predict and/or prevent the development of ascites.

PARTITIONING ENERGY AND OXYGEN REQUIREMENT OF BROILERS:

Even though oxygen is important for oxidation of nutrients, there is limited data on the oxygen requirement of broilers. The oxygen needs of a broiler can theoretically be partitioned into maintenance and production needs in a similar manner as is done for energy. The oxygen requirement for maintenance can be directly determined by determination of fasting metabolism or indirectly from indirect calorimetery and regression equations relating weight gain to oxygen consumption. In the first method, the bird is fasted for a determined period in metabolic chambers and the oxygen consumption measured. One problem of this method is variation in activity between a fasted and fed broiler. The measurement is therefore remote from a production environment. With calorimetry, since a broiler in weight equilibrium has zero weight gain, its maintenance

oxygen requirement can be determined by regressing oxygen consumption against weight gain of broilers fed different levels of feed and solving for oxygen consumption at zero weight gain.

Limitations of this method is that gain composition is assumed to be homogeneous. Even though the broiler may not show change in body weight, it is possible that fat can be mobilized from adipose tissue and used for protein accretion. Lema (1994) observed an increase in protein gain and loss of fat in 3 and 5 week broilers that were fed at maintenance level.

FACTORS THAT INFLUENCE OXYGEN CONSUMPTION AND UTILIZATION:

There are a number of factors that affect the ability of birds to consume oxygen. Some of which are high altitude, low ambient temperature, respiratory diseases and composition of the animal.

HIGH ALTITUDE: As the altitude increases, the partial pressure of oxygen decreases. This decreases the amount of oxygen in the alveoli for exchange with blood resulting in a condition known as hypoxia. Hypoxia induces an increase in heart rate (blood flow) and respiratory frequency (Butler, 1967). Hypoxemia is followed by an increase in hemoglobin concentration, hematocrit and polycythemia (Maxwell, 1990; Yersin, 1992). An increase in the ratio of the partial

Table 2. Partial pressure of oxygen at higher altitudes.

Altitude (meters)	Barometric Pressure (mmHg)	PO_2 (mmHg)
0	760	145.88
274*	736	140.87
610	707	134.81
1220	656	124.15
1830	609	114.32
2440	564	104.92
3050	523	96.35
3660	483	87.99

*Experimental location height above sea level.
PO_2 = calculated alveolar partial pressure of oxygen
Source: calculated from Handbook of Chemistry and Physics, 39 Ed., 1952.

pressure of CO_2 to RBC concentration decrease oxygen supply (Murray, 1969).

Under low ambient temperature, metabolic activity of birds is increased through increased feed consumption to generate heat and maintain body temperature. This causes a marked increase in oxygen requirement of birds (Huchzermyer et al. 1989; Gleeson, 1986). Since the lung of chickens is fixed on the dorsal wall of the thoracic cavity, its capacity to increase in volume is limited (Scheele et al. 1991). This may limit oxygen uptake from the lung specially in modern breeds that are selected and bred for increased weight gain having an increased oxygen demand.

RESPIRATORY DISEASES: Infection of the respiratory system significantly reduces oxygen consumption of birds. Research reports indicate that diseases of the lung could damage the lung tissue and interfere with blood flow and as well as oxygen exchange (Wideman, 1986). Scheele et al.(1991) and Jones (1994) in their study on broilers found that birds most susceptible to ascites had lower oxygen consumption than their counterparts at normal or low environmental temperatures. Lower O_2 consumption of these birds may not satisfy their metabolic O_2 demand triggering all conditions leading to ascites.

ANIMAL COMPOSITION: Tissue composition (Lean/Fat) of the animal is the other factor that has an impact on the birds

oxygen requirement. As indicated in Table 1, Teeter and Wiernusz (1994) found that lean birds have elevated oxygen need than the fat ones (3.12 Vs 1.21 lit/g/bird). Owens et al. (1994) reported that in case of cattle, the mass of fat increased quadratically with empty body weight while 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 the oxygen requirement varies with body weight and composition.

Table 3. Heat and CO_2 production and O_2 consumption as influenced by gain composition in birds equalized (by covariate analysis) for quantity of protein and fat DM during days 14-42 post hatching

Protein gain (g)	Fat gain (g)	Heat prod. (Kcal/b)	O_2 cons. (Lit/b)	CO_2 prod. (Lit/b)	RQ
227.0[b]	127.2	4479.0[b]	922.9	764.6	0.83
259.4[a]	127.2[b]	5075.7[a]	1024.0	847.9	0.83
236.7	112.5[b]	4624.8	935.6	769.8	0.82
236.7	136.9[a]	4680.2	965.0	802.8	0.82

[ab]Means within a column at equalized gain category with unlike superscripts differ ($P<0.05$), b=bird
Source: Teeter and Wiernusz, 1994.

ASCITES IN POULTRY:

Ascites refers to the accumulation of plasma like edematous fluid within the abdominal cavity. This fluid originates mostly from the liver (Wideman, 1988; Shane, 1988; Julian, 1989). It is known to poultry men by several names, "water belly", "High altitude disease", or "Avian

edema". It can be distinguished from excessive abdominal fat deposition by palpating the abdomen.

Ascites was first recognized as a disease syndrome in the 1970's in broiler flocks grown at high altitudes in South Africa and South American countries, Mexico, Colombia and Peru (Huchzermyer et al. 1986; Lopez-Coello et al. 1986). Mortality due to ascites in Mexico and other South American countries averaged 6 to 15 % of all broilers (Wideman, 1988) causing a tremendous economic loss to poultry producers. Economic losses associated with ascites is due to a combination of increased mortality and condemnations at the processing plant along with reduced growth rate and feed efficiency. Lopez-Coello (1986) estimated a loss of $ 40 million in Mexican flock in 1984 due to ascites. In the 1970's, ascites was known to be prevalent in birds maintained at high altitude regions. Recently, it has been diagnosed in areas even very close to sea level. A nation wide survey report in the US indicated that mortality due to ascites varies with season, reaching its peak (2%) in the month of January (Morris, 1992). Ascites prevails all over the world where broilers are raised unless measures are taken to reduce growth rate.

CAUSES OF ASCITES:

A number of factors have been suggested by different investigators as causes of ascites. Some of the major factors are outlined below :

HIGH ALTITUDE: Constriction of pulmonary artery is induced by a lower partial pressure of oxygen at the alveoli. Eventhough work done on smooth muscles is limitting, it is believed that low concentration of ATP under hypoxic conditions causes an increase in permeability of ATP sensitive potassium channels in the pulmonary artery memberane and consequent depolarization. As a result, calcium permeability is increased to achieve constriction of the arterial smooth muscle (Hanson et al. 1993).

The partial pressure of oxygen at alveolar level is secondary to the air pressure at different altitudes. Researchers relate response of animals at high altitude to the pressure of blood flow in the pulmonary artery. As indicated by Burton et al. (1968), chickens adapted to high altitude have significantly increased blood pressure in the pulmonary arteries, and enlarged (41% increase in mass) right ventricles. He also observed that male chickens have higher pulmonary arterial pressures than females. Right ventricular enlargement is also significantly greater at high altitudes in males than in females. Olander et al. (1967) reported that chickens raised at high altitude have an increased incidence of cardiac muscle lesions. They also demonstrated incidence of right heart failure with congested combs, dilation and hypertrophy of the heart, congested and edematous lungs with microscopic damage to the lungs, liver, and kidneys.

Studies concerning the relationship between low partial pressure of oxygen at high altitudes and ascites provide background data on conditions leading to the development of heart failure and ascites in birds. Avian species are generally more tolerant to high altitudes than are mammalian species (Schmidt - Nielsen, 1983). Domestic poultry however are an exception. Atland (1961) found that chickens had a much lower altitude tolerance than other small warm-blooded animals. Low partial oxygen pressure at high altitude results in oxygen deficiency or hypoxia. Hypoxia is defined as a condition of reduced oxygen in environmental air or in the air in the lung (Julian et al. 1986). It leads to tissue hypoxia (Wideman 1984).

Hypoxia has a great affect on cardiac output. According to the work of Burton (1968) birds at an altitude of 4167 m had pulmonary arterial blood pressure twice as great as those chickens at sea level. Pulmonary hypertension increases the work required by the heart to overcome the resistance in the pulmonary artery. Through time, this will result in dilatation of the right ventricle and can lead to heart failure. Huchzermeyer and DeRuyck (1986) investigated the relationship of high altitude and dilatation of the right ventricle as measured by ascites heart index (AHI). The AHI is the ratio of weight of the right ventricle to the weight of the total ventricle. They found a direct relationship in which the API increases with increase in altitude. Witzel et al. (1990), using week old broiler

chicks in metabolic chambers that simulate altitudes of 1980, 2590 and 2895 m observed that for every bird dying of ascites at 1980 m, two were dead at 2590 m and six at 2895 m. At 6 weeks of age the birds at the 2895 m altitude weighed 530 g less than the same age birds serving as a control. Similar results were reported by Vander Hel et al. (1988) who compared day old chicks exposed to a gas mixture with 15 % oxygen to controls at 20.9% oxygen. Ascites was noticed in the test birds around 21 days and at 32 days they weighed 600 g less than the control with a packed cell volume 50 % above that of the control birds.

High altitude and its effect on hematological values have been well documented. Maxwell et al. (1986) found that the packed cell volume, hemoglobin and red and white blood cell counts were significantly raised in 5 week old birds suffering from ascites. Hetrophiles and monocytes were also increased in these birds at the expense of lymphocytes. Hernandez (1987) and Witzel et al. (1990) found that the counts of red blood cells (RBC), percent hematocrit and concentration of hemoglobin were significantly elevated in birds maintained at high altitude.

Mirsalmi and Julian (1991) confirmed that reduced erythrocyte deformability was one of the predisposing factors that increased resistance to blood flow in the microcirculation of the lung resulting in pulmonary hypertension, heart failure and ascites in chicken. Normally, the RBC should maintain a highly deformable

character to pass through capillaries whose diameter are often less than that of the RBC. If RBC loose their deformable character, blood flow through the microcirculation will be restricted. Hakim and Macek (1988) examined erythrocyte deformability of different species of animals during hyopoxia. They found that hypoxia decreased RBC deformability.

EXCESS SODIUM IN DRINKING WATER OR FEED: Moderate levels of dietary sodium (Na) were reported to improve weight gain in broilers by increasing water consumption (Barlows et al. 1948; Eleazer and Bierer, 1964). In addition, Na causes water retention, augmenting weight gain (Julian, 1987). Numerous research publications however, have documented that Na in drinking water or feed when included at a toxic level cause ascites in young chicks and broilers (Kare et al. 1948; Mohanty and West, 1969; Paver and Robertson, 1953). Julian (1987) showed that ascites related to high dietary Na begins with an increased pressure in the venous system. This is followed by right ventricular failure and valvular (atrioventricular) insufficiency as a result of dilatation and hypertrophy of the right ventricle side of heart. High level of dietary sodium was also reported to affect red blood cell rigidity and size and could increase resistance to flow in the small blood capillaries of the lung (Eleazel and Bierer, 1964; Mirsalimi and Julian, 1991; Julian, 1987). Julian, 1992 reported that even though the effect of Na in

feed and water are additive, Na in the drinking water is more toxic than in feed. This occurs because broiler chickens consume by weight 1.5 to 2.5 times more water than feed and Na in feed or water increases water consumption.

Mammals can form concentrated urine, thereby conserving water while excreting sodium. Birds form concentrated urine. Poultry therefore, should be able to tolerate mildly hypertonic saline as drinking water. According to Martidale (1975), young and growing poultry appear to have a particular inability to excrete excess sodium in the urine (Martidale, 1975). Such natural condition was suggested to aggravate ascites in chickens.

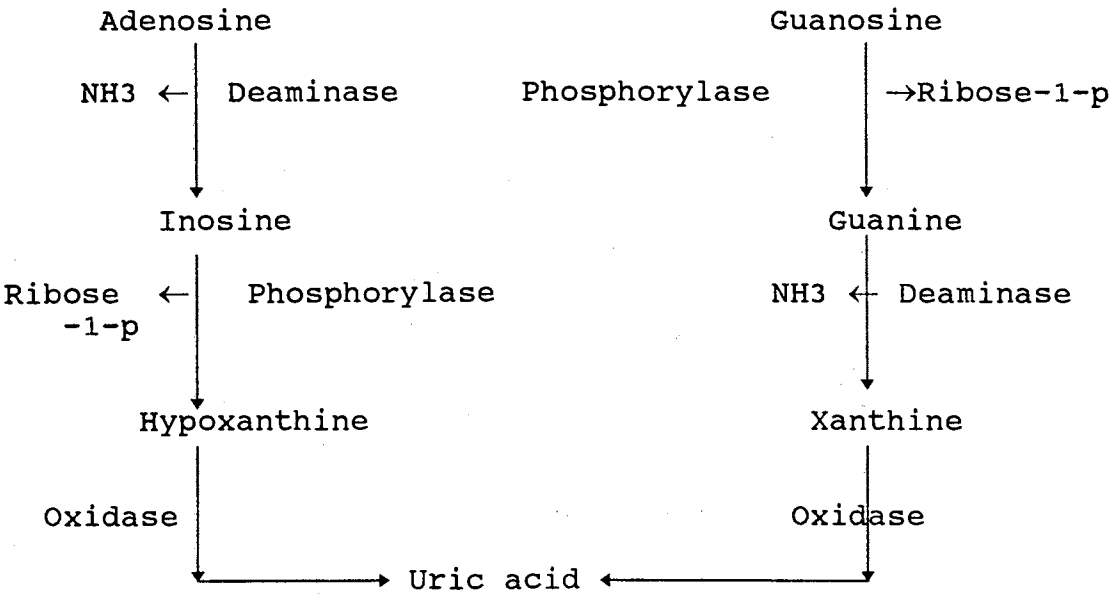
Field observations indicate that outbreaks of ascites in chicken caused by high dietary Na could be due to feed mixing error, from ingredients containing high levels of Na or the Na may have been deliberately added to water when an ion-exchange water softener is used, or the natural water supply may contain excess Na. Inconsistent results have been reported concerning the level of salt in the diet that causes ascites. For young birds 10 g NaCl /kg in the feed (Parthasarathy et al. 1979), and more than 2.5 g NaCl / liter in water (Krista et al. 1961) have been reported though under some circumstances higher levels can be tolerated (Paver and Robertson, 1953). Julian et al. (1992) reported that in meat type chickens, a threshold appears to be close to 0.12 % Na in drinking water and 0.20 % in the feed, above which there is a marked increase in Na toxicity.

The exact level that causes ascites in broilers is yet to be determined.

COLD STRESS: The incidence of ascites at both high and low altitudes has been noted to be more marked in cold weather (Hernandez, 1984; Huchzermeyer and DeRuyck, 1986; Julian et al. 1989; Shlosberg et al. 1992). Julian et al. (1989) exposed two strains of male broilers, from 2-57 days, to either high (23 C) or low (13 C) temperatures and two dietary energy densities. The incidence was much higher in the cold versus the hot temperature (40 % Vs 16.7 %). Little difference due to dietary energy density was noted (30.7 % for the high versus 27. 9 % for the low) even though the high density diet resulted in significantly faster growth. One can imagine that the increased metabolic rate induced by cold causes a marked increase in oxygen requirement. The total requirement for oxygen is higher in the cold and low oxygen concentration environments due to both the low availability of oxygen and the increased requirement of oxygen for the production of metabolic heat. This suggests that availability of oxygen had a marked influence on the incidence of ascites.

INTESTINAL AMMONIA: Recent research reports indicate that the level of intestinal ammonia have a significant influence on mortality rate due to ascites (Walker, 1994a). Intestinal ammonia is primarily the product of urea

hydrolysis by the bacterial enzyme urease. Uric acid, which is the principal form of waste nitrogen excretion from purines in the broiler, enters the gastrointestinal tract where some portion is converted to urea by bacterial enzymes.



Walker (1994a) suggested that the presence of ammonia in the intestine increases nucleic acid production in the mucosal wall resulting in a thicker intestinal wall. The extensive blood supply required by the intestines for nutrient absorption and transport is provided through a capillary bed in the mucosal wall of the intestine. Mucosal wall thickening places increased pressure on the capillary structure within the intestinal wall.

It is hypothesized that increased pressure on the capillary could restrict normal blood flow, thus increasing

intestinal hypertension, vascular congestion and decreased blood flow similar to that observed in ascites. Walker (1994b) further suggested that reducing intestinal ammonia appears to reduce the mucosal tissue turnover rate and the oxygen consumption of the portal vein drained organs, the gastrointestinal tract, spleen and pancreas which were found to consume 25 % of the whole animal oxygen consumption. Decreasing oxygen use by these tissues might be the primary driving force of the ascites syndrome.

PATHOPHYSIOLOGY OF ASCITES:

The anatomy of blood distribution in the domestic chicken is reviewed by Wideman (1984) and described by Sturkie (1976). Blood is distributed throughout the body of larger animals by a system of larger arteries, from which branch smaller distribution arteries. Distribution arteries enter the various organs and tissues, giving rise to the smallest blood vessels known as arterioles, capillaries and post capillary venules . Capillaries serve as exchange vessels for nutrients, hormones, and the waste products of cellular metabolism. Plasma fluid and nutrients exit the capillaries and bath surrounding tissues. Most of the fluid that baths the surrounding tissues returns back into the blood at the post capillary venules. The remaining fluid enters the lymphatic system. Fluid collected by the lymphatics eventually re-enters the blood at junctions of large lymphatic ducts with large veins. Anything that

disturbs the delicate balance of fluid entering and exiting across the capillary walls will result in edema of the tissues, hydropericardium and ascites. The reabsorption of the interstitial fluid by the post capillary venules and the drainage capacity of the lymphatic vessels can be blocked by a heightened venous pressure as a result of heart insufficiency. Wideman (1988) indicated that in most cases, ascites in chicken is attributed to liver, heart and/or lung damage that disturbs the delicate balance of fluid entering and exiting across the capillary walls.

LIVER DAMAGE AND ASCITES: Under normal conditions, the pressure of blood flowing inside the liver is quite low since blood flowing into the liver is supplied by a low pressure venous portal system. This venous blood normally flows through low resistance vascular channels and sinusoids, returning to the heart through veins of low pressure. The low pressure inside the liver, along with low resistance to blood flow, prevents fluid leakage through the surface connective tissue of the liver. Anything that increases pressure of outflow blood results in plasma leakage from the surface into the abdominal cavity and hence ascites. Direct damage to the liver has been reported to be associated with mycotoxins and plant toxins (Wideman, 1988).

HEART DAMAGE AND ASCITES: The use of excessive furazolidone, toxic fat (Polychlorinated biphenyl's) in the

feed and round heart disease were reported to damage heart tissue (Wideman, 1984;1988) and cause disturbance of pressure in outflow blood inside the liver. As a result, blood accumulates in the large veins leading to the heart and increase the central venous pressure. Ascites develops when the increase in central venous pressure raises the fluid pressure inside the liver, causing pressure and fluid leakage from the surface.

LUNG DAMAGE AND ASCITES: Normally, the right ventricle is a relatively weak muscle that is designed to pump blood at low pressure through low resistance blood vessels of the lungs. Lung tissue damage due to disease can result in congestive heart failure and ascites. Ploog (1973) and Wideman (1988) reported that lung damage due to disease increases pulmonary vascular resistance and shunts blood to the healthy regions of the lung for better supply of oxygen. However, widespread lung damage or oxygen deprivation can cause substantial decrease in blood flow making the heart work much harder particularly the right ventricle. This results in right ventricular enlargement and dilation followed by decreased cardiac pumping efficiency, congestive heart failure, increased central venous pressure, and pressure induced ascites.

PULMONARY HYPERTENSION INDUCED ASCITES:

Available literature on ascites caused by pulmonary hypertension (PH) induced right ventricular failure (RVF) focused on the clinical and pathological changes caused by RVF. Clinical signs resulting from PH and all pathological lesions observed in the heart and lung are the result of increased venous pressure from the RVF (Wilson et al. 1988). Reported differences in hematology and biochemistry between normal and ascitic birds are also the result of RVF rather than the cause.

CLINICAL SIGNS OF ASCITES: Broilers affected with ascites syndrome are reported to show shrunken comb and wattles. The abdomen is distended with fluid in chickens that have been affected for several days. These broilers also showed an increased respiration rate and reduced activity. It is possible that broilers may die from hypoxia before they develop ascites and would not have ascitic syndrome (Hernandez, 1979; Lopez-Coello et al. 1985; Wideman, 1984,1988; Julian, 1987). Recent reports indicate that affected chickens are of normal size but growth stops when RVF develops and those that survive are most often smaller in size (Julian, 1990; Maxwell, 1990; Fraser, 1991) compared to normal (non-ascitic) chickens..

HEMATOLOGY: An increase in blood volume in RVF has been reported by Burton and Smith (1972). A marked increase in the number of red blood cells (RBC) was also noted contributing to the increase in blood volume. Concentration of enzymes that reflect tissue damage or hypoxia from chronic congestion are elevated, but changes in electrolytes were minor (Jaeger and McGrath, 1974; Maxwell et al. 1990). Most often, the most significant changes observed in chicken are in the RBC profile. Increase in RBC numbers, hematocrit, mean cell volume and hemoglobin were reported (Maxwell et al. 1986, 1990; Hernandez, 1987; Odom et al. 1989; Witzel et al. 1990). Broilers with RVF have been shown to be hypoxaemic (Julian and Mirsalimi, 1992) although it is not clearly understood why this happens. Hypoxaemia causes an increase in RBC. Since there is plasma expansion in RVF the increased hematocrit indicates a marked increase in total RBC, many of which are young and large cells (Maxwell et al. 1986).

PATHOLOGICAL LESIONS: Depending on the stage of ascites development, a large or small quantity of yellow fluid with clots of fibrin is observed in the peritoneal cavities. The liver may be swollen and congested and irregular with edema and have fibrin adherent to the surface. There is a mild to a marked hydropericardium and occasionally epicarditis. The right ventricle, atrium and vena cava are seen distended. The lungs are congested and edematous (Huchzermeyer, 1985;

Lopez-Coello et al. 1985; Maxwell et al. 1986, 1987, 1990; Hernandez, 1987; Julian, 1987; Wilson et al. 1988; Fraser, 1991). It is possible that all broilers that die from PH may not show ascites symptoms . Death may occur suddenly before clinical signs are observed. After a short period of difficult breathing affected broilers could be seen dead on their back (Julian et al. 1987, 1989, 1990).

PATHOGENESIS OF PULMONARY HYPERTENSION:

Low atmospheric oxygen concentration causes an increase in blood cell production and hematocrit (Burton and Smith, 1969; Burton et al. 1971; Julian et al. 1986). Researchers suggest that PH and ascites incidence in broilers raised at low altitude is related to the high oxygen requirement of rapidly growing birds and the inability of the heart and lung to deliver sufficient oxygen to the tissue to maintain growth rate. High incidence of ascites is frequently observed in those flocks with superior growth rate and feed conversion (Julian, 1987, 1990). Because PH-induced ascites at high altitude is caused by low atmospheric oxygen tension and since PH can be produced experimentally using hypobaric chambers (Owen et al. 1990; Witzel et al. 1990) it is reported that PH at low altitude could be associated with decreased environmental oxygen availability in confined poultry houses without adequate ventilation (Lopez-Coello et al. 1985; Dale and Vilacres, 1986; Maxwell 1990; Dale, 1990).

Chickens have a thicker respiratory membrane than other birds, and broilers have a thicker respiratory membrane than Leghorns (Vidyadaram et al. 1990). The ability of broilers to diffuse oxygen into blood is thus not as good as that of other birds. Research on meat-type chickens indicates that fast growing broilers have a lower percentage of oxygen saturation than slow growing broilers (Peacock et al. 1988, 1989, 1990; Julian and Mirsalimi, 1992). These results suggest that some meat-type chickens are not fully oxygenating their hemoglobin at low altitude since pen oxygen levels were not low (Julian and Wilson, 1992). If fast growing meat type chickens are hypoxic, the hypoxia might have been the result of interference with respiration because of the large breast muscle mass or intra-abdominal pressure from fat and internal organs (Anderson et al. 1986; Julian and Mirsalimi, 1992).

The right atrioventricular valve of chickens is different from mammals in that the valve is composed of a muscle flap made up mainly of muscle fibers from the right ventricle wall. The anatomy of this valve makes the bird very susceptible to valvular insufficiency in case of PH (Julian, 1987, 1990). The thin RV responds very rapidly to increased workload by dilatation (stretch) and hypertrophy (Owens and Schwartz, 1982). When the RV wall hypertrophies, the right atrioventricular valve also hypertrophies. This can lead to valvular insufficiency and RVF (Julian, 1987).

Genetic difference may make some broilers more susceptible to PH-induced RVF as observed by structural and electrocardiographic studies (Asson-Batres et al. 1989; Odom et al. 1989, 1991, 1992; Martinez et al. 1992). Right ventricular hypertrophy results from increased work load by the heart causing stretching of muscle fibers (Cueva et al. 1974; Sillau et al. 1980). Pulmonary hypertension could result from either increased blood flow (increased cardiac output) or increase in pulmonary resistance to blood flow (Julian, 1993).

CAUSES OF PULMONARY HYPERTENSION:

INCREASED OXYGEN REQUIREMENT: The most important cause of increased blood flow or cardiac output is increased body oxygen requirement resulting in tissue hypoxia (Julian, 1990). Researchers suggest that an increased oxygen requirement associated with increased food intake and rapid growth to be the cause of an increased pulmonary hypertension in broiler chickens both at high and low altitudes (Hernandez, 1982; Dale and Villacres, 1987,1988; Julian et al. 1987; Odom et al. 1992).

During the past years, meat type chickens were selected for their growth rate, muscle mass and feed conversion (Julian, 1990). Due to lack of selection for proportional growth of the heart and lung, the weight of these organs is smaller relative to the body weight (Julian, 1989). There

is a possibility that the output from these organs might not satisfy the metabolic need of the animal.

LOWER AMBIENT TEMPERATURE: The increase in ascites both at high and low altitude has been noted to be more marked during low ambient temperature exposure (Hernandez, 1984; Huchzermeyer and DeRuyck, 1986; Julian et al. 1989; Shlosberg et al. 1992). Recent research shows that the increased metabolic rate induced by cold causes a marked increase in oxygen requirement and cardiac output resulting in PH. Gleeson (1986) showed a 185 % increase and Huchzermyer et al. (1989) a 32.7 % increase in oxygen requirement with cold temperature. Similar results were reported by Julian et al. (1989) and Stolz et al. (1992).

INCREASED BLOOD VOLUME: High dietary salt increases blood volume (Julian, 1987), but salt was also reported to increase RBC rigidity (Mirsalimi et al. 1992) which increases resistance to blood flow. High level of salt in the feed or drinking water was suggested to be one cause of PH and should be considered whenever there is an increased incidence of ascites in a flock (Adams et al. 1991; Julian et al. 1992).

RVF increases blood volume and the Hypoxaemia associated with RVF results in polycythaemia (Julian and Mirsalimi, 1992), which when the increased blood volume is taken into account, indicates a marked increase in RBC

number. Polycythemia increases both blood volume and viscosity if the packed cell volume increases (Burton and Smith, 1972). Increased volume and polycythaemia both increase RV workload.

INCREASED RESISTANCE TO BLOOD FLOW: Increased resistance to blood flow was indicated to be one of the major factors that causes PH induced ascites at high and low altitudes. Some of the factors that were suggested to increase resistance to blood flow are indicated below:

INCREASED RBC SIZE: Julian (1987) and Maxwell (1990, 1991) suggested increase in size of RBC to be the cause of increased resistance to flow. In fact, large RBCs are more deformable than small ones that tend to be spherical rather than biconcave (Smith et al. 1979). The hemoglobin concentration in RBC increases with age as cells become smaller and less deformable. A large nucleus would probably increase rigidity and interfere with flow (Maxwell, 1991). Young broilers have larger RBC than Leghorns (Julian, 1990) probably because the increased oxygen demand of rapid growth results in a high proportion of young cells.

REDUCED VASCULAR CAPACITY: It is indicated that lung damage is the cause of increased ascites incidence in broiler chickens with little evidence to support this idea (Wideman, 1984, 1988). In birds, the lung is more rigid than in

mammals and blood capillaries are unable to expand significantly. Even mild interstitial edema occurs with increased dietary salt (Julian, 1987b) or early PH (Julian, 1987a) could interfere with blood flow and oxygen exchange. The lung epithelium may be injured by inhaled or injected drugs and other chemicals affecting the capillary flow (Adamson et al. 1977; Cooper et al. 1986; Julian et al. 1989a).

CONTROL OF ASCITES:

Occasionally ascites outbreaks will occur as a result of mistakes in feed formulation where excess sodium is included in the feed and water, toxic fat in the diet, or inappropriate use of disinfectants while disinfecting. Persistent occurrence of ascites is mainly due to predisposing factors such as high altitude or inherent defects that contribute to respiratory and cardiovascular inadequacy in broilers. Most of the recommendations by investigators on minimizing mortality rate associated with ascites focus on identifying the cause and reducing all factors that may stress the lungs, heart and liver.

NUTRITIONAL RECOMMENDATIONS: Reducing feed intake reduces the incidence of ascites. Wideman (1988) indicated that the decrease in ascites incidence is due to decreased rate of growth and/or to decreased intake of possible toxins in feed. Feed intake and growth rates can be reduced by using

a mash feed instead of pelleted feed (DaSilvia et al. 1988) and by reducing the energy levels in the diet in combination with a feed restriction program. It appears Likely that slower growth rates place less strain on the cardiovascular and respiratory systems of broilers. Dale and Villacres (1986) and DaSilva et al. (1988) reported that changing from a pelleted to a mash type feed is effective when total ascites mortality reaches 8-16 % of the flock. The economic consequences of reduced growth rate as a result of feed restriction should be considered since a one week extension in grow out time will result in elimination of one complete flock per year from the production cycle of a broiler house (Wideman, 1988).

Excess sodium in the feed or water is known to cause ascites. Julian et al. (1992) reported that excess dietary sodium induces pulmonary hypertension in broiler chickens and particularly sodium in drinking water is more toxic than in feed although feed and water sodium are additive. Julian (1990) suggested that feed should be routinely analyzed for sodium directly and should not be estimated from the chloride level since high levels of sodium from sources other than sodium chloride may be present. If sodium is present in the water, sodium in the feed should be reduced by 2-3 times the level present in the water since water consumption is about 2-3 times that of feed consumption.

The quality of fat used in poultry feed apparently alters the incidence of ascites. Poorly refined vegetable

oil, second quality oil, or oil containing oxidized fatty acids seem to increase ascites mortality. Rotter et al. (1984) reported that diets formulated with sunflower oil significantly reduced mortality from sudden death syndrome when compared with diets formulated with tallow. In their experiment, symptoms of sudden death syndrome included edema in the lungs, liver and abdominal cavity, with liver enlargement. Apparently, the choice of fat used for feed formulation could be important in altering the incidence of ascites.

MANAGEMENT AND ENVIRONMENTAL IMPROVEMENT: Most often, air quality is quite poor at floor level but may be adequate 1-2 m above the floor in poultry houses. This factor can not be overemphasized, particularly when respiratory damage is suspected as a key factor in triggering ascites. As stocking density increases, ventilation must increase. Good litter quality must be maintained to keep ammonia and dust at low levels. Dust and ammonia can only make respiratory problems worse. It is important to prevent spillage from waterers in order to avoid problems with ammonia (Wideman, 1988). Management effort must be made to improve the quality of air at the level of the bird.

During cold weather, when poultry houses are closed tightly and ventilation is minimal, toxic fumes from brooders and heaters may accumulate at litter level especially when gas brooders are used. Carbon monoxide and

carbon dioxide have detrimental effects on the respiratory and cardiovascular systems. Julian and Wilson (1984) found increased levels of carbon monoxide near propane heaters in broiler houses during ascites outbreaks.

Cold temperatures are strongly correlated with ascites outbreaks. The overall demand for oxygen increases at cold temperatures, as the birds burn food with oxygen to generate body heat. This increased oxygen consumption places a strain on the ability of the cardio-pulmonary system to deliver oxygen to the tissues. Cold temperature also cause stress, thereby inhibiting the immune system and the resistance to disease (Wideman, 1988; Julian, 1990). Effort should be made to prevent exposure to cold and reduce wide fluctuations in poultry house temperatures.

USE OF UREASE INHIBITOR IN THE FEED: Recent research reports indicate that the use of urease inhibitor significantly reduces mortality due to ascites. Anthony et al. (1994) reported that supplementing the broiler diet with urease inhibitor resulted in a greater than 50 % reduction in cumulative mortality due to ascites. Balog et al. (1994) found that dietary supplementation of urease significantly decreased large intestine ammonia, right ventricular heart weight, hemoglobin and red blood cell count while the small intestine and liver weight were significantly increased

REFERENCES

Adams, M. H., J. N. Beasley, and P. W. Waldroup, 1991. Development of a regimen to induce onset of ascites in broiler chickens. *Poul. Sci. 70, Supp. 1, (Abst.)*, 141.

Adamson, I. Y. R., D. H. Bowden, M. G. Cote, and H. Witschi, 1977. Lung injury induced by butylated hydrotoluene. Laboratory investigation, 36, 26-32.

Anderson, L. S., M. Gleeson, A. L. Haigh and V. Molony, 1986. Variability in respiratory function of the domestic fowl. Research in veterinary science, 40: 292-298.

Anthony, N. B., J. M. Balog, F. B. Staudinger, C. W. Wall, R. D. Walker, and W. E. Huff, 1994. Effect of urease inhibitor and ceiling fans on ascites in broilers. 1. Environmental variability and incidence of ascites. Poultry Science 73: 801-809.

ARC/MRC Committee, 1974. Food and Nutrition Research Report. Stationary Office, London.

Armsby, H. P. and C. R. Moulton, 1925. The animal as a converter of matter and energy. Reinold publishing Corp. New York.

Asplin, F. D. and R. B. A. Carnaghan, 1961. The toxicity of certain groundnut meals for poultry and special reference to their effect on ducklings and chickens. Veterinary record, 73: 1215-1219.

Asson-Batres, M. A., M. K. Stock, J. F. Hare and J. Metcalfe, 1989. O2 effect on composition of chick embryonic heart and brain. Respiration physiology, 77: 101-110.

Balog, J. M., N. B. Anthony, C. W. Wall, R. D. Walker, N. C. Rath, and W. E. Huff, 1994. Effect of urease inhibitor and ceiling fans on ascites in broilers. 2. Blood variables, ascites scores, and body and organ weights. Poultry Science 73: 810-816.

Bartov, I., S. Bornistein, and B. Lipstin, 1974. Effect of calorie to protein ratio on the degree of fatness in broilers fed on practical diets. Br. Poult. Sci. 15:107-117.

Birkelo, C. P., D. E. Johnson and H. P. Phetteplace 1991. Maintenance requirements of beef cattle as affected by season on different plane of nutrition. J. Animal Sci. 69:1214-1222.

Blaxter, K.L., 1989. Energy Metabolism in Animals and Man. University Press, Cambridge, England.

Bolton, W., 1959. The digestibility of the carbohydrate complex of barley, wheat and maize by adult fowls. J. Agric. Sci. 46:119-122.

Bray, T., 1983. Broilers- why is temperature so important? In Gledthorpe Experimental Husbandry Farm, Poultry Booklet No. 10 pp.-9. Ministry of agriculture, Fisheries and food.

Brody, S., 1964. Bioenergetics and growth. Hafner Publishing Co. New York, New York.

Brouwer, E., 1965. Report of sub-committee on constants and factors. Energy metabolism, pp 441-443. Edit. Blaxter, K.L., London, Academic Press.

Burlacu, G., M. Baltic, and M. Peraschiv, 1970b. Efficiency of food energy utilization in growing chickens. in: Energy metabolism of Farm Animals pp. 209-212. A. Schurch and C. Wenk ed. Juris Verlag, Zurich.

Burton, R. R. and A. H. Smith, !969. Induction of cardiac hypertrophy and polycythemia in the developing chick at high altitude. Federation proceedings, 28: 1170-1177.

Burton, R. R. and A. H. Smith, 1972. The effect of chronic erythrocytic polycythemia and high altitude upon plasma and blood volumes. Proceedings of the Society of experimental biology and medicine, 140: 920-923.

Burton, R., and A. H. Smith, 1969. Induction of cardiac hypertrophy and polycythemia in the developing chick at high altitude. Fed. Proc. 28:1170-1177.

Burton, R., R. Sahara and A. H. Smith, 1971. The hematology of domestic fowl native to high altitude. Environmental physiology, 1:155-163.

Carlton, W., 1966. Experimental coal tar poisoning in the White Pecking duck. Avian Diseases, 10: 484-502.

Chudy, A., and R. Schiemann, 1971. Energetische Verwertung der Futternahrstoffe beim Huhn. In: Energettiische Futterbewertung und Energienormen, pp. 168-198. Ed. R. Schiemann, K. Nehring, L. Hoffman, W. Jentsch and A. Chudy. Berlin, VEB Deutscher Landwirtschaftsverlag.

Close, W. H., M. W. A. Verstegen, and L. E. Mount, 1973. Proc. Nutri. Soc. 32:72A.

Cueva, S., H. Sillau, A. Valenzuela, and H. Ploog , 1974. High altitude induced pulmonary hypertension and right heart failure in broiler chickens. Research in Veterinary Science, 16: 370-374.

Da Silva, J. M. L., N. Dale and J. Batista-Luchesi, 1988. Effect of pelleted feed on the incidence of ascites in broilers reared at low altitudes. Avian Diseases, 32:376-378.

Dale, N. and A. Villacres, 1986. Nutrition influences ascites in broilers. Poultry (Misset), April, 2: 40-43.

Dale, N., 1990a. Dietary factors influence ascites syndrome in broilers. Feedstuffs, December, pp. 14-16.

Davidson, J., and J. Mathieson, 1965. Observations on the utilization of dietary energy by medium-and fast-growing strains of cockerels and on their argenine and methionine requirements. BR. Poult. Sci. 6:225-233.

Deaton, J. W., J. L. McNaughton, F. N. Reece, and B. D. Lott, 1981. Abdominal fat of broilers as influenced by dietary level of animal fat. Poultry Sci. 60:1250-1253.

DeGroote, G., 1974. A comparison of a new net energy system with the metabolizable energy system in broiler diet formation, performance, and profitability. Br. Poult. Sci. 15:75.

Deighton, T., and J. C. D. Hutchinson, 1940. Studies on the metabolism of fowls. III. The determination of the comparative net energy of Sussex ground oats and white maize meal for fattening cockerels. J. Agri. Sci (Cambridge) 30:463-484.

Edwards, H. M. Jr., F. Denman, A. Abou-Ashour and D. Nugara, 1973. Carcass composition studies 1. Influence of age , sex and type of dietary fat supplementation on total carcass and fatty acid composition. Poultry Sci. 52:934-948

Emans, G. C.,1987. Growth, body composition and feed intake. World's Poultry Sci. 43:208-224.

Farrell, D. J., 1974. General principles and assumptions of calorimetry In: energy requirements of poultry pp 1-24, T. R. Morris and B. M. Freeman, British poultry Sci. Ltd., Edinberg.

Farrell, D.J., and S. Swan, 1977. Effect of temperature treatments on the heat production of starving chickens. Bri Poultry Sci.18:725-734.

Fisher., C., and B. J. Wilson, 1974. Response to dietary energy concentration by growing chickens. In: energy requirements of poultry. Edit Morris, T. R. Freeman, B. M., Br. Poult. Sci. Ltd. pp 151-184.

Fraps, G. S., and E. C. Carlyle, 1939. The utilization of the energy of feed by growing chickens. Bull. Tex. Agric. Exp. Stn. 571.

Fraser, C. M. 1991. Ascites syndrome. In: Fraser, C. M. (Ed.) The Merck Veterinary Manual, 7th edn, pp. 1628-1629 (Rahway, Merck and Co. Inc.)

Freeman, B.M., 1965. The relationship between oxygen consumption, body temperature and surface area in the hatching and young chicks. Brit. Poultry Sci. 6:67-72.

Hakim, T. S. and A. S. Macek, 1988. Effect of hypoxia on erythrocyte deformability in different species. Biorheology, 25: 857-868.

Hanlen, E.T., 1939. Some observations on the normal variations in the composition of light Sussex Cockerels. J. Agric. Sci.(Cambridge) 28:379-392.

Hanson, W.L., D.F. Boogs, J.M. Kay, 1993. Collateral ventilation and pulmonary arterial smooth muscle in the coati. J. Appl Physiol. 74: 2219-2224.

Hernandez, A., 1987. Hypoxic ascites in broilers: a review of several studies done in Columbia. Avian Diseases, 31: 658-661.

Hoffman, L., and R. Schiemann, 1971. Verdaulichkeit und Energiekennzahlen von futterstoffen beim huhn. Archiv Tierrernahrung, 21:65-81.

Huchzermeyer, F. W. and A. M. C. DE Ruyck, 1986. Pulmonary hypertension syndrome associated with ascites in broilers. Veterinary Record, 119: 94.

Huchzermeyer, F. W., 1985. Water belly-altitude disease. Poultry International, 62-66.

Huchzermeyer, F. W., 1986. Causes and prevention of broiler ascites. Poultry Bulletin, August, 346.

Huchzermeyer, F. W., and A.M.C. DeRuyck, 1986. Pulmonary hypertension syndrome associated with ascites in broilers. Vet. Res. 119:94.

Jaeger, J. J. and J. J. McGrath, 1974. Hematological and biochemical effects of simulated high altitude on the Japanese quail. Journal of Applied Physiology, 37: 357-361.

Jenson , J. F., 1982. Quality of poultry meat- an issue of growing importance. World's poultry Sci. Journal 38:105-111.

Jones, G.P.D, 1994.Energy and nitrogen metabolism and oxygen use by broilers susceptible to ascites and grown at three environmental temperatures. British poultry Science, 35:97-105.

Julian R. J. and B. Wilson, 1992. Pen oxygen concentration and pulmonary hypertension induced right ventricular failure and ascites in meat type chickens at low altitude. Avian Diseases, 36: 733-735.

Julian R. J. and S. M. Mirsalimi, 1992. Blood oxygen concentration of fast growing and slow growing broiler chickens, and chickens with ascites from right ventricular failure. Avian Diseases, 36: 730-732.

Julian, R. J. and M. Goryo, 1990. Pulmonary aspergilosis causing right ventricular failure and ascites in meat type chickens. Avian Pathology, 19: 643-654.

Julian, R. J., 1985. Cholangiohepatitis in broiler chickens. Proceedings Canadian Poultry Disease Conference, p. 16, Ottawa, Canada.

Julian, R. J., 1990. Pulmonary hypertension: a cause of right heart failure, ascites in meat type chickens. Feedstffs, January 29:19-22.

Julian, R. J., and M. Boulianne, 1988. Natural and experimental lung pathology causing pulmonary hypertension, right ventricular hypertrophy, right ventricular failure and ascites in broiler chickens. American journal of Veterinary Research, 192: (Abst. 209), 1781.

Julian, R. J., G. W. Friars, H. French and M. Quinton, 1987. The relationship of right ventricular hypertrophy, right ventricular failure, and ascites to weight gain in broiler and roaster chickens. Avian Diseases, 31: 130-135.

Julian, R. J., I. McMillan and M. Quinton, 1989. The effect of cold and dietary energy on right ventricular hypertrophy, right ventricular failure and ascites in meat type chickens. Avian Pathology, 18:675-684.

Kleiber, M., 1945. Dietary deficiencies and energy metabolism. Nutrition Abstracts and reviews 15:207-222. Kleiber, M.,1961. The fire of life. John Wiley and Sons, Inc, New York.

Kubena, L. F., J.W. Deaton, F. N. Reece and J. D. May, 1972. Body composition of chicks as influenced by environmental temperature and selected dietary factors. Poultry Sci. 51:517-522.

Lawes. J. B., and J. H. Gilbert, 1861. On the composition of oxen, sheep, and pigs and of their increase whilst fattening. Journal of the royal Agricultural Society of England 21:1-92.

Leclercq, B., J. C. Blum and J. P. Boyer, 1980. Selecting broilers for low or high abdominal fat: Initial observations. Br. Poult. Sci. 21;107-113.

Ledger, H. P.. and A. R. Sayers, 1977. The utilization of dietary energy by steers during periods of restricted food intake and subsequent realimentation. J. Agric Sci. 88:11.

Lema, M., 1994. An evaluation of broiler metabolism at three ambient temperatures with particular reference to energy and protein needs. Ph.D. thesis, Oklahoma State University.

Lopez-Coello, C., T. W. Odom and C. A. Bailey, 1987. Observations on the incidence of ascites in a commercial flock of broilers in Mexico. Poultry Science, 66, Supp. 1 (Abst.), 84.

Lopez-Coello, C., T. W. Odom and R. F. Wideman, 1985. Ascites major cause of mortality in broilers. Poultry Digest, 44:284-288.

McDonald, P., Edwards, R.A., and Greenhalgh, J.F.D., 1988. Animal nutrition, 4th Ed., Longman Scientific & Technical, New York.

Macleod, M. G., C. C. Whitehead, H. D. Griffin and T. R. Jewitt, 1987. Energy and nitrogen retention and loss in broiler chickens genetically selected for leanness and fatness. Brit. Poult Sci 29:285-292.

Marks, H. L. and G. M. Pesti, 1984. The role of protein level and diet form in water consumption and abdominal fat deposition of broilers. Poultry Sci. 63:1617-1625

Martinez, L. A., M. T. Casaubon and R. Navarro, 1992. Structure study of the heart of chickens resistant and

susceptible to ascitic syndrome. Poultry Science, 71, Supp. 1, (Abst. S91), 165.

Maxwell, M. H., 1190c. Ultrastructural abnormalities in 7-day-old broilers reared at high altitude. Research in Veterinary Science, 50: 233-239.

Maxwell, M. H., 1990a. Ascites in broilers. Poultry International, February, pp.32-38.

Maxwell, M. H., 1990b. Hematological and histopathological findings in young broilers reared in poorly and well ventilated environments. Research in Veterinary Science, 48: 374-376.

Maxwell, M. H., G. W. Robertson and S. Spence, 1986a. Studies on an ascitic syndrome in young broilers. 1. Hematology and Pathology. Avian Pathology, 15: 511-524.

Maxwell, M. H., G. W. Robertson and S. Spence, 1986b. Studies on an ascitic syndrome in young broilers. 2. Ultrastructure. Avian Pathology, 15:525-538.

Maxwell, M. H., S. Spence, G. W. Robertson, and M. A. Mitchell, 1990. Hematological and morphological responses of broiler chicks to hypoxia. Avian Pathol 19:23-40.

McCraken, K.J., and S.T.C. Weatherup, 1973. Proc. Nutri. Soc.32:66A.

Meltzer, A., 1983. Thermoneutral zone and resting metabolic rate of broilers. Br. Poult. Sci. 24:471-476.

Mirsalimi, S. M., P. J. O'Brien and R. J. Julian, 1992. Changes in erythrocyte deformability in NaCl induced right sided cardiac failure in broiler chickens. American Journal of Veterinary Research, 53:2359-2363.

Mison, B. H., 1974. An open circuit respirometer for metabolic studies on the domestic fowl: Establishment of standard operating conditions. Br. poult. Sci. 15:287-297.

Mittelstaedt, C. W., and R. G. Teeter. An evaluation of the metabolizable energy system as judged by carcass energy retention. Poultry Sci. (Submitted).

Murray, J. F., 1969. Viscosity effect on pressure-flow relations and vascular resistance in dogs lung. J. Appl. Physiol 27:336.

Murray, R. K, D. K. Granner, P. A. Mayes, and V. W. Rodwell, 1993. Harpers Biochemistry 23rd Ed. Appleton and Large Pub. Connecticut.

Nir, I. and H. Lin, 1982. The skeleton, an important side of lipogenesis in the chick. Anals of nutrition and Metabolism 26:100.

Nistan, Z., Keren-Zvi, S. Nolan, W., Weathers and P. D. Sturkie, 1978. Thermally induced peripheral blood flow changes in chickens. J. Appl. Physiol. 44:81-84.

Odom, T. W., B. M. Harges, C. Lopez-Coello, J. Arce and E. Avila, 1989. Time courses changes in electrocardiographic and hematological variables during the development of ascites in broiler chickens. Poultry Science, 68, supp.1, (Abst.), 107.

Odom, T. W., L. M. Rosenbaum and B. M. Hargis, 1992. Evaluation of vector electrocardiographic analysis of young broiler chickens as a predictive index for susceptibility to ascites syndrome. Avian Diseases, 36:78-83.

Odum, T. W., 1993. Ascites syndrome: Overview and update. Poultry digest 52(1): 14-22.

Olah, I., R. L. Taylor, Jr., and B. Glick, 1983. Ascites formation in the chicken. Poultry Sci. 62:2095-2098.

Olander, H. J., R. R. Burton and H. E. Adler, 1967. The pathophysiology of chronic hypoxia in chickens. Avian Diseases, 11: 609-620.

Owen, R. L., R. F. Wideman, Jr., A. L. Hattel and B. S. Cowen, 1990. Use of a hypobaric chamber as a model for investigating ascites in broilers. Avian Diseases, 34: 754-758.

Owens, F. N., D. R. Gill, D. S. Secrist and S. W. Coleman, 1994. Review of some aspects of growth and development of feedlot cattle. A paper presented at ADSA-ASAS Mtg. MN symposium.

Owens, G. K. and S. M. Schwartz, 1982. Alterations in vascular smooth muscle mass in the spontaneously hypertensive rat. Circulation Research, 51: 280-289.

Peacock, A. J., C. K. Pickett, K. M. Morris and J. T. Reeves, 1988. Spontaneous pulmonary hypertension in rapidly growing broiler chickens reared at sea level. American Review of Respiratory Diseases, 137, (Abst.)106.

Peacock, A. J., C. K. Pickett, K. M. Morris and J. T. Reeves, 1989. The relationship between rapid growth and pulmonary hemodynamics in the fast growing broiler chicken. American review of respiratory Diseases, 139: 1524-1530.

Peacock, A. J., C. K. Pickett, K. M. Morris and J. T. Reeves, 1990. Spontaneous hypoxaemia and right ventricular hypertrophy in fast growing broiler chicken reared at sea level. Comparative Biochemistry and Physiology, 97A: 537-541.

Penney, D. G., S. B. Davidson, R. B. Gargulinski and T. M. Caldwell-Ayre, 1988. Heart and lung hypertrophy, changes in blood volume, hematocrit and plasma renin activity in rats chronically exposed to increasing carbon monoxide concentrations. Journal of Applied Toxicology 8: 171-178.

Pesti, G. M., T. S. Whiting and L. S. Jenson, 1983. The effect of crumbling on the relationship between dietary density and chick growth, feed efficiency and abdominal fat pad weights. Poultry Sci. 62:490-494.

Pullar, J. D., and A. J. F. Webster, 1974. The energy cost of fat and protein deposition in the rat. Bri. J. Nutri. 37:355-363.

Robbins, K. R. and J. E. Ballew, 1984. Energy utilization for maintenance and gain in broilers and Leghorns at two ages 63:1419-1424.

Scheele, C. W., W. DeWit, M. T. Frankenheus, and P. F. G. Vereijken, 1991. Ascites in broilers. 1. Experimental factors evoking symptoms related to ascites. Poultry Sci. 70: 1069-1083.

Schiemann, R.,1963. Deutsche Akademie der landw. Sitzungsberichte 12:39.

Shlosberg, A., G. Pano, V. Handji, and E. Berman, 1992. Prophilactic and therapeutic treatment of ascites in broiler chickens. British Poultry Science 33: 141-148.

Sibbald, I.R., J. D. Summers, and S. J. Slinger, 1960. Factors affecting the metabolizable energy content of poultry feeds. Poultry Sci. 39:544-556.

Sillau, A. H., S. Cueva, and P. Morales, 1980. Pulmonary arterial hypertension in male and female chickens at 3300 m. Pflugers Archiv, 386: 269-275.

Smith, J. E., N. Mohandas, and S. B. Shohet, 1979. Variability in erythrocytes deformability among various mammals. American Journal of Physiology 236: H725-H730.

Snyder, G. K., 1971. Influence of temperature in hematocrit on blood viscosity. American Journal of Pathology 65: 1667-1672.

Stolz, J. L., L. M. Rosenbaum, D. Jeong and T.W. Odom, 1992. Ascites syndrome mortality and cardiological responses of broiler chickens subjected to cold exposure. Poultry Science 71, Supp. 1 (Abst) 12: 4.

Swayne, D. E., A. Shlosberg and R. B. Davis, 1986. Salt poisoning in turkey poults. Avian Diseases 30: 847-852.

Tasaki, I., and H.Sakuri, 1969. Studies on the energy metabolism in the fowl. Mem. Lab. Anim. Nutr. Nagoya Univ 4:1.

Teeter, R. G. and C. J. Wiernusz, 1994. New management approaches will provide opportunity. Feedstuffs, Feb.7, 14-16.

Thorbek, G.,1970. Publs. Eur. As. Anim. Prod. 13:120.

Van Der Hel, W., A. M. Henken, J. Viser and M. T. Frankenhuis, 1988. Induction of ascites by low environmental oxygen pressure. In Environment and animal Health, Proceedings of the 6th International Congress on Animal Hygiene. Skara, Sweden, p. 575-

Vidyadaran, M. K., A. S. King, and H. Kasim, 1990. Quantitative comparisons of lung structure of adult domestic fowl and red jungle fowl, with reference to broiler ascites. Avian Pathology 19: 51-58.

Waker, R. D., 1994a. New concept in ascites physiology. Poultry International, April.

Walker, R. D., 1994b. Intestinal ammonia and ascites mortality. Poultry international, May.

Wideman, R. F. Jr., 1988. Ascites in poultry. Nutrition update, 6:2, 1-7 (Monsanto Co. St. Louis, Missouri, USA).

Wilson, J. B., R. J. Julian and I. K. Barker, 1988. Lesions of right heart failure and ascites in broiler chickens. Avian Diseases 32: 246-261.

Witzel, D. A., W. E. Huff, L. F. Kubena, R. B. Harvey, and M. H. Elissalde, 1990. Ascites in growing broilers: a research model. Poultry Science 69: 741-745.

Yersin, A. G., W. E. Huff, L. F. Kubena, M. H. Elissalde, R. B. Harvey, D. A. Witzel and L. E. Giroir, 1992. Change in hematological blood gas and serum biochemical variables in broilers during exposure to simulated high altitude. Avian Diseases 36: 189-196.

CHAPTER III

ENERGY AND OXYGEN NEEDS FOR WEIGHT MAINTENANCE AND ACCRETION OF PROTEIN AND FAT BY COMMERCIAL BROILERS AT DIFFERENT TEMPERATURES[1]

A. Beker and R. G. Teeter[2], Oklahoma State University, Stillwater, OK 74078.

Running Head: Energy and Oxygen Requirements

[1] Agricultural experiment station, Oklahoma State University, Stillwater, OK 74078

[2] To whome correspondance should be addressed.

ABSTRACT 1. An experiment was conducted using 6 age groups of commercial broilers housed at 7 different ambient temperatures. Birds were housed in open circuit respiratory chambers such that the energy and oxygen requirements for body weight maintenance, protein and fat accretion, as well as exponents that may be used to convert live weight to metabolic body size could be determined.

2. Energy (KCal/$bwt^{.67}$/d) and oxygen (ml/$bwt^{.67}$/d) needs for body weight maintenance decreased as body weight was increased from 100 to 2400 g. Oxygen use by broilers decreased as temperature was increased.

3. Averaged across body weight, energy (Kcal) and Oxygen (ml) use above maintenance per Kcal gained as protein were 1.49, 300 and as fat were 1.31 and 165, respectively.

4. Estimates of metabolic body size exponents ranged from 0.24 to 0.98, being influenced strongly by body weight and ambient temperature. By tradition, the exponent .66 has been used to convert body weight to metabolic body size for homeotherms under thermoneutral conditions. The .66 value for birds at lower temperature is a gross underestimate.

5. Equations developed for this research to predict maintenance energy and oxygen use as well as to estimate requirements for protein and fat accretion should aid energetics research.

INTRODUCTION

Ascites, a physiological syndrome characterized by fluid accumulation in the broilers abdominal cavity, results in significant economic losses on a global scale. The syndrome is widely thought to be the result of pulmonary hypertension (PH) that is in turn mediated by insufficient oxygen supply or high altitude (Dale and Villacres, 1986; Julian, 1987; Beker et al., 1995), cold stress (Julian, 1989b; Shlosberg et al., 1992), toxic substances (Julian, 1991) and other nutritional factors such as high energy diet and sodium level (Hernandez, 1987; Julian, 1989). Expanding the Knowledge base concerning broiler oxygen and energy need requirements for maintenance and tissue gain should enhance therapeutic development to alleviate ascites as conditions predisposing birds to ascites would be better understood. Furthermore, knowledge related to broiler oxygen need could be used in the development of poultry house ventilation models and the selection-development of more efficient broiler strain.

Basal metabolic rate (BMR), the rate of heat production by a resting animal in a thermoneutral environment in post absorptive state, serves as the baseline for measuring energy increments associated with activities such as feeding, muscular work and maintaining body temperature (Brody, 1945). Even though the relationship between BMR and

body weight is exponential, there is no agreement among researchers on which specific exponent to use within a species (Brody, 1945; Kleiber, 1965; Haron and Meltzer, 1983). The superior growth performance of modern broiler strains was suggested by Kunzel and Kunzel, (1976) to be related to a reduced BMR which would spare more energy for growth. A clearer understanding of the energy needs of broiler for maintenance and for gain may help to select birds that are more efficient.

Metabolic body size $W^{3/4}$, traditionally has been used to express animal energy needs for maintenance and gain. The ratio of maintenance energy need to body weight decreases as the animal grows (Brody, 1945). Several researchers have determined the broilers maintenance heat production as a function of metabolic body size. Some workers have raised weight to the .75 power (Close, 1978; Hurwitz et al., 1979; Close and Stainer, 1984). Brody (1964) suggested that the .66 power is more appropriate for poultry. Meltzer (1983) proposed that .88 be used for broilers of both genders up to 26 days of age and thereafter to use .63 for females and .43 for males. Such differences in proposed exponents have been explained via differences in carcass composition (Emans, 1987) and ambient temperature (Close, 1978) or strain (Ferrell and Jenkins, 1985). Any exponent for converting live weight to metabolic weight must consider the importance of such factors. In addition, Kleiber (1961) noted that the production was altered

drastically by temperature. Because the thermoneutral temperature for birds at maintenance is unknown, a range in temperatures must be tested in research studies of maintenance energy requirements.

The study reported herein was designed to quantify the amounts of energy and oxygen used by broilers at various weights or ages for maintenance of body weight and for protein and fat accretion at several different ambient temperatures. The most appropriate exponent for converting live weight to metabolic body size was calculated.

MATERIALS AND METHODS

GENERAL BIRD HANDLING AND FEEDING:

Three groups of commercial 1-day old broiler chicks of a single strain (Cobb-500) were obtained at 7 day intervals. Upon arrival, chicks were reared in sawdust covered floor pens until they reached 3, 5, 7, 14, 21, 35, 42, and 49 days of age. Ambient temperatures tested for chicks at 3 and 5 days included 28, 30 and 32; temperature for chicks at 7, 14 and 21 days of age were 24, 26, 28, 30, and 32C. For 35, 42 and 49 day old chicks, test temperatures were 18, 22, and 26C. Birds utilized in the study were selected randomly from the various age groups and transferred to open circuit respiratory chambers described previously by Wiernusz and Teeter (1993) and Belay and Teeter (1993). The 36 respiratory chambers were housed within 3 different thermostatically controlled rooms so that ambient

temperature could be controlled precisely. Feed and water were available for *ad-libitum* consumption the first day for adaptation to the chamber. Following this chamber adaptation period, but prior to collection of metabolic data, chicks were deprived of feed for 10 h, and weighed; the experiment was initiated by administering an appropriate amount of feed (Table 1) to each bird (*ad- libitum* or 0, 5, 10 % of initial body weight). Each day's feed was divided into two equal portions which were provided at 12 h intervals for 48 h. The feeding phase of each experimental period birds was followed by a 10 h period of feed deprivation after which final weight was recorded.

Excreta were allowed to accumulate during the assay period in fecal collection trays at the bottom of each metabolic chamber. Excreta were weighed and frozen (-20 C) until analyzed for energy, carbon and nitrogen content.

BIRD HOMOGENIZATION AND ANALYSIS: A preliminary test was conducted to determine if nitrogen would be lost from samples autoclaved at 115.6 C and 0.75 Atm. pressure. For this test, minced broiler breast meat was analyzed for N before and after unautoclaving using a micro kjeldahl procedure (Karasawa, 1989). Results indicated that the autoclaving did not impact (P=.81) tissue N content averaging 13.8% on DM basis.

Five chicks from each age group were sacrificed via cervical dislocation at the start of each experimental

period; these were stored in polyethylene bags at -20 C until analyzed. At the end of the experiment, all chicks were sacrificed by cervical dislocation and similarly held at -20 C. Carcasses were homogenized to simplify sampling for laboratory assays. Carcasses from chicks less than 14 days were placed in loaf pans, covered with aluminum foil, weighed and autoclaved for 8 hours. For chicks older than 14 days, carcasses were thawed and feathers were cut to a maximum of 2 cm length prior to autoclaving for 12 h. Following autoclaving, each carcass was cooled to room temperature and homogenized using a blender. These samples were stored in a labeled polyethylene bag and held at - 20 C until analyzed.

For measuring carbon (C) and nitrogen (N) contents, samples of feed, chicken and excreta were dried at 55 C for 72 h and ground using a coffee grinder. Duplicate samples were analyzed for C and N using a Leco-2000 (Moline, Illinois, Parr Co.) infrared C and N analyzer. Percentages of dry matter (DM), fat (ether extract) and ash were analyzed according to the procedures of AOAC (1990). Gross energy was determined using a bomb calorimeter.

OXYGEN CONSUMPTION AND CO_2 PRODUCTION: The differences in concentrations of O_2 and CO_2 between air entering and leaving each chamber were regressed against time, $time^2$ and $time^3$ to establish polynomial equations describing O_2 consumption and CO_2 production. These regressed values

against rate of air passage through each chamber use to calculate O_2 and CO_2 production. Heat production was calculated from the liters of O_2 consumed and CO_2 produced according to the Brouwer (1965) equation: HP(Kcal/d) = 16.18 (O_2 L/d) + 5.02 (CO_2 L/d).

ENERGY AND OXYGEN NEEDS FOR MAINTENANCE: Heat production (energy use) and O_2 use were estimated for each bird in body weight-ambient temperature and feed intake group. Means for each body weight-temperature group were calculated and maintenance needs were calculated by regression as follows:

MEI / MWT = M + G / MWT.

TO2C / MWT = M + G / MWT.

HP / MWT = M + G / MWT.

Where MEI, TO2C, and HP are metabolizable energy (Kcal /$bwt^{.67}$/d), total oxygen consumption (ml/$bwt^{.67}$/d), and heat production (Kcal/$bwt^{.67}$/d), respectively; MWT = metabolic body weight ($bwt^{.67}$); Men = maintenance energy (Kcal/$bwt^{.67}$/d); Mo = maintenance oxygen need (ml/$bwt^{.67}$/d); Mhp=heat production at maintenance (Kcal/$bwt^{.67}$/d) and G is weight gain (g) per day.

According to these models, the intercept (M) of each equation provides an estimate for homeostasis; the slope (G) provides an estimate for gain at each body weight and ambient temperature.

PROTEIN AND FAT ACCRETION: Carbon and nitrogen balance were determined based on C and N contents of feed, tissues and feces. Protein and fat accretion for birds of each weight and ambient temperature combination were calculated as the difference between final and initial tissue composition.

ENERGY AND OXYGEN USE FOR PROTEIN AND FAT ACCRETION

Amounts of metabolizable energy and oxygen used for each unit energy of protein and of fat were estimated by regressing metabolizable energy and oxygen intake above maintenance against energy of protein and fat accrued at each body weight-ambient temperature combination for broilers fed above 10 % of their body weight using the following equations:

$$MEIAM = aEp + bEf$$

$$OIAM = cEp + dEf$$

Where MEIAM is metabolizable energy (Kcal/$bwt^{.67}$/d) intake above maintenance, Ep and Ef are rates of energy (Kcal/ $bwt^{.67}$ /d) of protein and fat accrued, respectively. OIAM is oxygen consumed(ml/$bwt^{.67}$/d) above maintenance.

According to this model, the values for a and b equal the energy requirements for protein and fat synthesis expressed as Kcal/Kcal retained. similarly, the values of c and d give the oxygen requirements for protein and fat synthesis expressed as ml/kcal. The reciprocals of a and b

are equal to the efficiencies of use of ME above maintenance for synthesis of protein and fat, respectively.

METABOLIC BODY SIZE: The ambient temperature-body weight effects on exponential conversion to metabolic body size for birds weighing 1.46 to 2.4 kg at 18 C was estimated by regressing log heat production against log body weight (Brody, 1945). Heat production at thermoneutral (TN) was estimated for each weight group birds ranging in weight from 0.11 to 2.4 Kg at the three ambient temperatures considered and taking the lowest heat production. Exponent was estimated by regressing log heat production at TN against log body weight. Similar procedure was followed in estimating exponent at 26 C.

RESULTS

MAINTENANCE ENERGY AND OXYGEN NEEDS:

Maintenance energy requirements for 3 and 5 day old broiler chicks were higher than the metabolizable energy (ME) intake by these chicks during the study. This may be due to the use of nutrients from the yolk sac which inverts into the abdominal cavity. Therefore, maintenance energy need data for these chicks was examined separately.

To examine the effects of ambient temperature and body weight on the maintenance energy, O_2 as well as HP at maintenance for each age group of birds separately, 2-dimensional plots are presented in Figures 1 to 12. Oxygen

use for maintenance (ml/bwt$^{.67}$/d) of all age groups decreased as ambient temperature was increased (Figures 1 and 7). Similar results were noted for energy expenditures for maintenance (kcal/wt$^{.67}$/d) as shown in Figures 2 and 8 and HP(Kcal/bwt$^{.67}$/d) at maintenance for all ages of birds (Figures 3 and 9).

Two-dimensional plots of maintenance energy expenditure, O_2 use as well as HP at maintenance against body weight are displayed in Figures 4 to 6. Maintenance energy (Kcal/bwt$^{.67}$/d), O_2 (ml/bwt$^{.67}$/d) and HP at maintenance (KCal/bwt$^{.67}$/d) regressed across weight of broiler decreased as body weight increased.

A test for fitness of linear and quadratic models was performed; a quadratic fit appeared to reflect the maintenance requirement more closely.

To examine the interaction between ambient temperature and body weight, maintenance energy and O_2 expenditures and HP at maintenance are plotted in 3-dimensional plots in Figures 13 to 20. In young chicks, oxygen use for body weight maintenance (ml/bwt$^{.67}$/d) showed that oxygen use per unit weight decreased as body weight of the broilers was increased from 42 g to 75 g (Figure 13). As the ambient temperature was decreased from 30 to 18 C or increased from 31 to 32 C, a increasing trend in oxygen use was noted. This shows that the thermoneutral zone for this chicks lies between 30 to 31 C. Similar trend was noted for heat production at maintenance (Figure 14). Oxygen use for body

weight maintenance and heat production at maintenance for these group of chicks at any given ambient temperature-body weight can be estimated by the predictive equations:

$$MO2PWD=1258.909742-12.321786W+0.303105W^2 -77.311012T+1.2844752T^2+0.111144WT.$$

where MO2PWD is oxygen use(ml/bwt$^{.67}$/d); W is metabolic body weight (bwt$^{.66}$) and T is ambient temperature (C).

$$HPKCWD=6.954073-0.094450W+0.002761W^2 -0.413274T+0.006867T^2+0.000405WT.$$

where HPKCWD is heat production (Kcal/bwt$^{.66}$/d) at maintenance; W is metabolic body weight (bwt$^{.66}$) and T is ambient temperature(C).

Energy needs for body weight maintenance (Kcal/ bwt$^{.66}$/d) of broilers aged 1 to 3 weeks showed that energy need per unit metabolic weight decreased as body weight of the broilers was increased from 100 g to 650 g (Figure 16). As the ambient temperature was decreased from 28 to 24 C or increased from 30 to 32, a increasing trend in maintenance energy need was noted. This shows that the thermoneutral zone for this chicks lies between 28 to 30 C. Oxygen use for maintenance per metabolic weight (Figure 15) and heat production at maintenance per metabolic weight (Figure 17) decreased as body weight of the broilers increased from 100 to 650 g. Similarly, oxygen use and heat production of this group of birds decreased as the ambient temperature of increased from 24 to 32 C. Oxygen use for body weight

maintenance, energy need for maintenance and heat production at maintenance for these group of chicks at any given ambient temperature-body weight can be estimated by the predictive equations:

$$MO2PWD=349.792833-1.828569W+0.005628W^2 -14.650178T+0.198061T^2+0.023203WT.$$

where MO2PWD = oxygen use(ml/bwt$^{.66}$/d); W is metabolic body weight (bwt$^{.66}$) and T is ambient temperature (C).

$$MEPWD=1.052607-0.004623W+0.00003878W2 -0.034812T+0.000492T2-0.000072595WT.$$

where MEPWD = energy need for body weight maintenance (Kcal/bwt$^{.66}$/d), W =metabolic body weight (bwt$^{.66}$) and T = ambient temperature (C).

$$HPKCWD=1.706405-0.008614W+0.00002812W^2 -0.072243T+0.000993T^2+0.000100WT.$$

where HPKCWD = heat production (Kcal/bwt$^{.66}$/d) at maintenance; W is metabolic body weight (bwt$^{.66}$) and T = ambient temperature(C).

Oxygen need for body weight maintenance of broilers aged 5 to 7 weeks (Figure 18) also showed an inverse relationship where the oxygen need per unit metabolic body weight decreased as body weight of the broilers was increased from 1100 g to 2400 g. As the ambient temperature was decreased from 26 to 18 C, oxygen use and energy need for maintenance as well as heat production at maintenance increased. Oxygen use for body weight maintenance, energy need for maintenance and heat

production at maintenance for these group of birds at any given ambient temperature-body weight can be estimated by the predictive equations:

$$MO2PWD=103.245413-1.155883W+0.002605W^2 +3.460825T-0.159324T^2+0.010637WT.$$

where MO2PWD is oxygen use(ml/bwt$^{.66}$/d); W is metabolic body weight (bwt$^{.66}$) and T is ambient temperature (C).

$$MEPWD=0.496544-0.006811W+0.000013153W2 +0.015206T-0.000794T2+0.000126WT.$$

where MEPWD is energy need for body weight maintenance (Kcal/bwt$^{.66}$/d), W =metabolic body weight (bwt$^{.66}$) and T = ambient temperature (C).

$$HPKCWD=0.499854-0.005576W+0.000012821W^2 +0.016382T-0.000749T^2+0.000048451WT.$$

where HPKCWD is heat production (Kcal/bwt$^{.66}$/d) at maintenance; W is metabolic body weight (bwt$^{.66}$) and T = ambient temperature(C).

ENERGY AND OXYGEN NEED FOR PROTEIN AND FAT ACCRETION:

The energy requirement for body weight gain did not appear to be impacted by ambient temperature (P=.1) and averaged 2.8 Kcal/g/d.

Because the energy need for body weight gain was not affected by ambient temperature, ME intake above maintenance (Kcal/bwt$^{.66}$/d) and O_2 consumption (ml/bwt$^{.66}$/d) above maintenance were regressed against energy content of protein and fat gained. The following regression equations were obtained:

MEIAM=1.64Ep + 1.43Ef. R^2=.80.

O2CAM=300Ep + 165Ef. R^2=.81.

Where MEIAM and O_2CAM are ME intake and oxygen consumption above maintenance (Kcal/bwt$^{.67}$/d; ml/ bwt$^{.67}$/d), respectively; Ep=energy retained as protein (Kcal) and Ef=energy retained as fat (Kcal).

The coefficients, 1.64 and 1.43 Kcal indicate the energetic efficiencies (kcal needed per kcal of protein and fat deposited) were 0.61 and 0.70, respectively; 316 and 175 ml oxygen were consumed for each Kcal of protein and fat accreted, respectively.

Energy need for maintenance and gain determined using our predictive equations and data of NRC, 1994 is indicated in Table 3. Values determined are within the range of estimates proposed from previous investigators (Shanon and Brown, 1970; Pinchasov, 1970; Jones, 1994).

METABOLIC BODY WEIGHT CONVERSION:

Table 3 shows exponents most suitable to convert live weight to metabolic body size based on body weight and ambient temperature. Values determined ranged from 0.24 to 0.67. Chicks with body weight of 1.46 to 2.4 Kg and under cold stress had the lowest exponent (0.24); Chicks with a body weight of 0.11 to 2.4 kg and at 24 C had an exponent of 0.67. The 0.67 value is in agreement with the value suggested by Brody (1964) for birds of all age group.

DISCUSSION

A increase in maintenance oxygen use and heat production at maintenance of chicks (Figure 13 and 14) as the ambient temperature was decreased from 30 to 28 C or increased from 31 to 32 suggests that chicks under extreme ambient temperature are subject to increased oxygen demand for oxidation of feed nutrients and generate more heat.

As seen in Figures 16, maintenance energy expended for weight maintenance of broiler chicks increased as the ambient temperature was increased from 30 to 32 C or decreased from 28 to 24 suggesting that birds outside the zone of thermoneutrality were less efficient energetically. An increase in energy expenditure for maintenance of broilers as the ambient temperature was increased from 30 to 32 C suggest that birds under heat stress increase their breathing rate to dissipate more heat and cool down.

A increase in maintenance oxygen need of chicks (Figure 15) as the ambient temperature was decreased from 28 to 24 C suggests that chicks under cold stress are subject to increased oxygen demand for oxidation of feed nutrients and generate more heat. It is anticipated that such a condition is the cause of pulmonary hypertension induced ascites in broilers. An increase in energy expenditure for maintenance of broilers as the ambient temperature was increased from 30 to 32 C suggest that birds under heat stress increase their breathing rate to dissipate more heat and cool down.

Heat production by chicks (Figure 17) increased as ambient temperature was decreased from 28 to 24 C indicating that the bird's metabolic activity was increased under such circumstances to maintain body temperature. Similarly, as ambient temperature was increased from 30 to 32 C, heat production increased. This might be due to the fact that the birds increased their metabolic activity to generate energy for panting.

A increase in oxygen use and energy need for maintenance as well as heat production at maintenance per unit metabolic weight (Figures 18, 19 and 20) of birds weighing over 1400g as the ambient temperature was decreased from 26 to 18 suggests that birds under cold stress increase their metabolic activity to maintain body temperature.

Maintenance energy need reported by Hurwitz et al. (1980) was slightly higher than those we measured. This may be because they averaged maintenance energy need over all age group.

Results of ME needs above maintenance (Kcal) for a Kcal of protein accreted found in this study was lower than the value reported by Pullar and Webster, (1977) for rats but similar to values reported by Close and Stainer, (1984) for 14 day-old weaned piglets. Accretion is the difference between synthesis and degredation. Whenever this rate is higher, efficiency of ME use is greater. Pullar and Webster's higher value could be due to the increment of heat production per unit energy of protein accreted with the

semisynthetic diet they have used. The efficiency of ME use above maintenance use for protein synthesis that we determined was similar with the value reported by Hoffmann and Schiemann(1971).

Exponents determined to convert live body weight to metabolic body size ranged from 0.24 to 0.67. This range suggests that metabolic body size is influenced by body weight and ambient temperature. The value of 0.67 obtained for chicks having body weights of 0.11 to 2.4 Kg and at 26 C is in agreement with the value suggested by Brody (1964) for birds of all age groups. However, the low exponent (0.24) obtained for birds with body weight of 1.46 to 2.4 Kg at 18 C indicates that caution should be made in using the same exponent for birds under cold stress. Exponents for young birds appear dynamic and can deviate substantially.

Table 1. Composition of experimental diet used in the study

Ingredients	g/kg
Corn, ground yellow	509.0
Soybean meal (49% CP)	382.0
Fat (animal and vegetable)	65.0
Dicalcium phosphate	17.8
Limestone	12.0
Salt	4.5
Vitamin mix[A]	2.5
Methionine (99%)	2.2
Trace mineral mix[B]	1.0
Ethoxyquin	0.2
Energy	13.59 MJ ME/kg
Crude protein	233 g/kg

[A]Mix supplied the following per kilogram of diet: Vitamin A, 5.25 mg; cholecalciferol, 0.125 mg; vitamin E, 0.025 mg; vitamin B12, 0.03 mg; riboflavin, 15 mg; niacin, 75 mg; d-panthothenic acid, 25 mg; choline, 705.5 mg; menadione, 5 mg; folic acid, 1.5 mg; pyridoxine, 6.25 mg; thiamine, 3.03 mg; d-biotin, 0.127 mg.
[B]Mix supplied the following per kilogram of diet: Manganese, 120 mg; zinc, 100 mg; copper, 10 mg; iodine, 2.5 mg; calcium, 135 mg; iron, 75 mg; selenium, 0.15 mg.

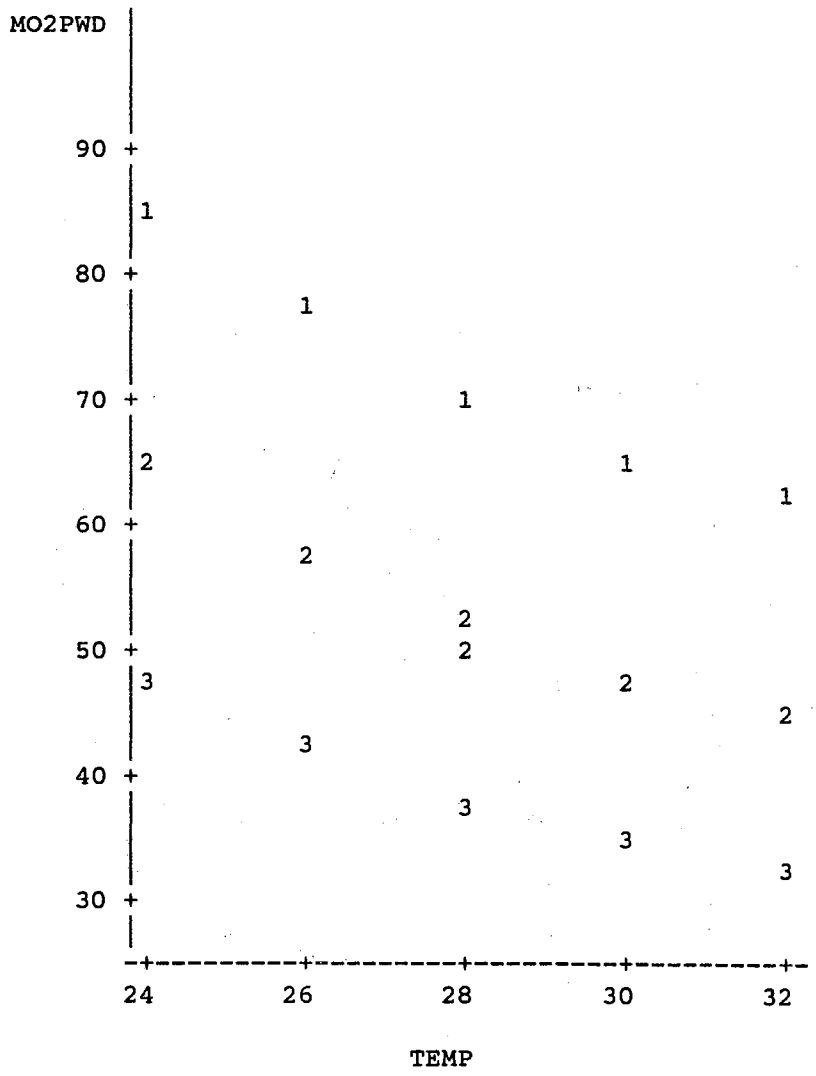


Figure 1. Oxygen (ml/gmwt/d) use for maintenance of broilers aged 1 to 3 weeks in relation to ambient temperature.

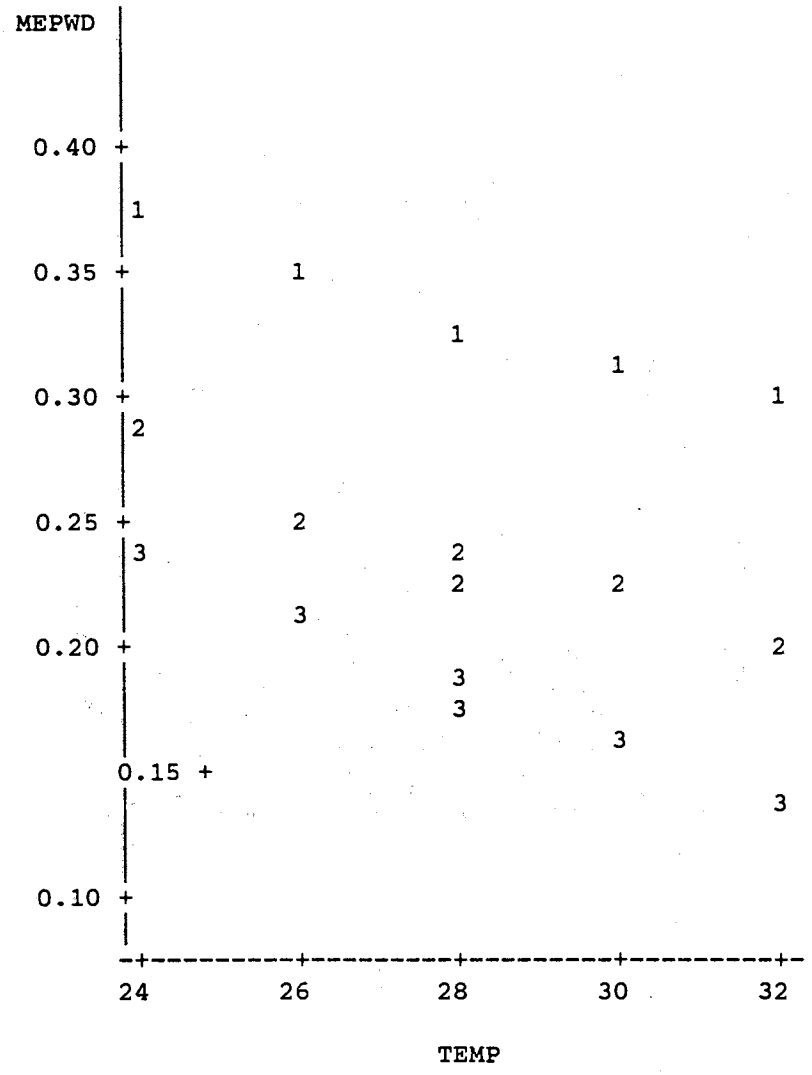


Figure 2. Maintenance energy (KCal/gmwt/d) need of broilers aged 1 to 3 weeks in relation to ambient temperature.

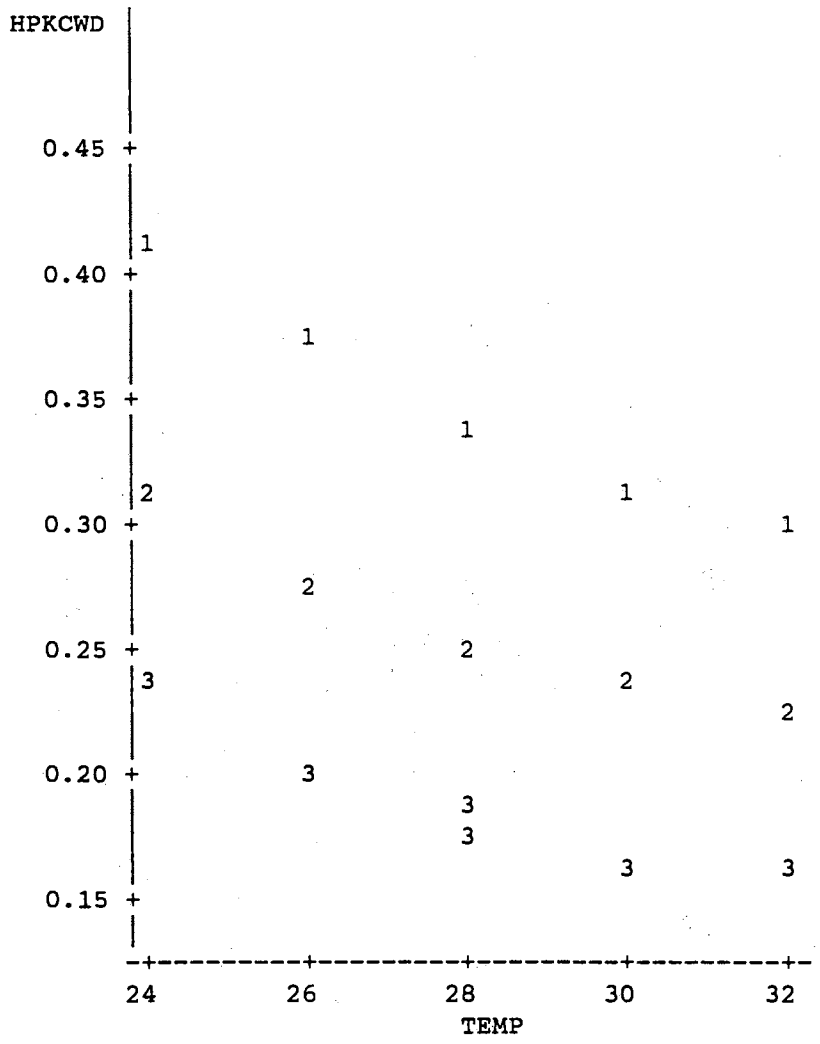


Figure 3. Heat production(KCal/gmwt/d) at maintenance of broilers aged 1 to 3 weeks in relation to ambient temperature.

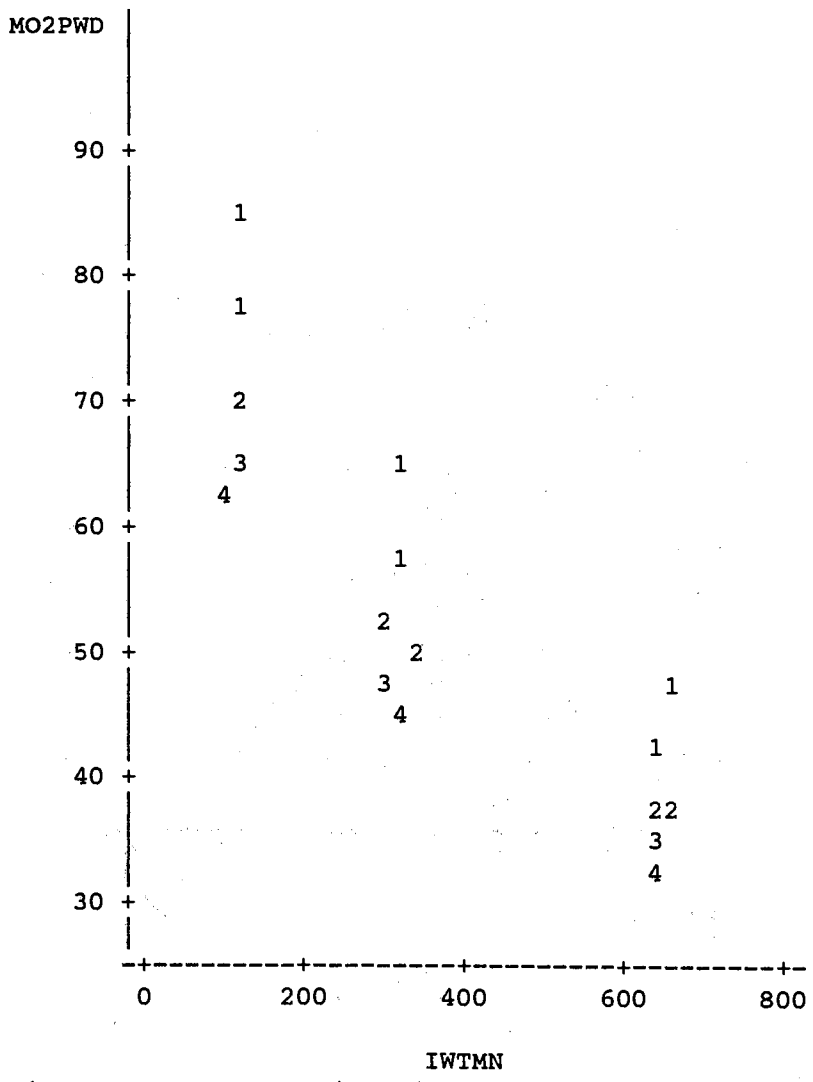


Figure 4. Oxygen (ml/gmwt/d) use for maintenance of broilers aged 1 to 3 weeks in relation to body weight

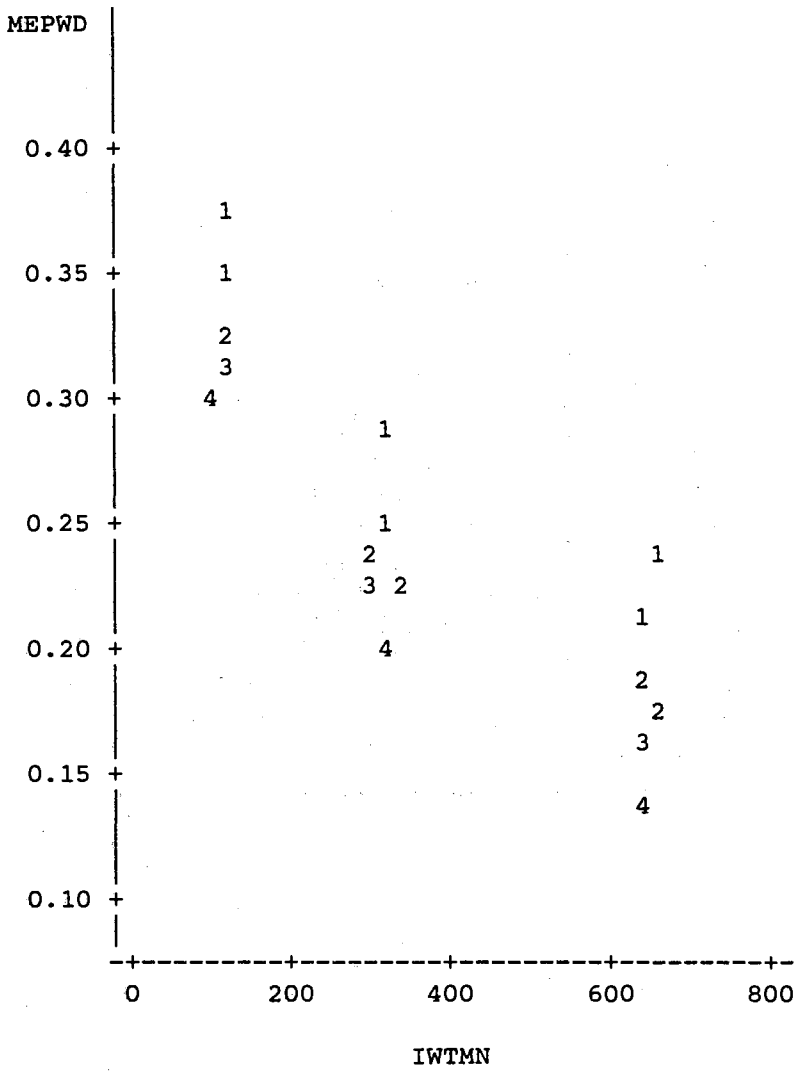


Figure 5. Maintenance energy (KCal/gmwt/d) need of broilers aged 1 to 3 weeks in relation to body weight.

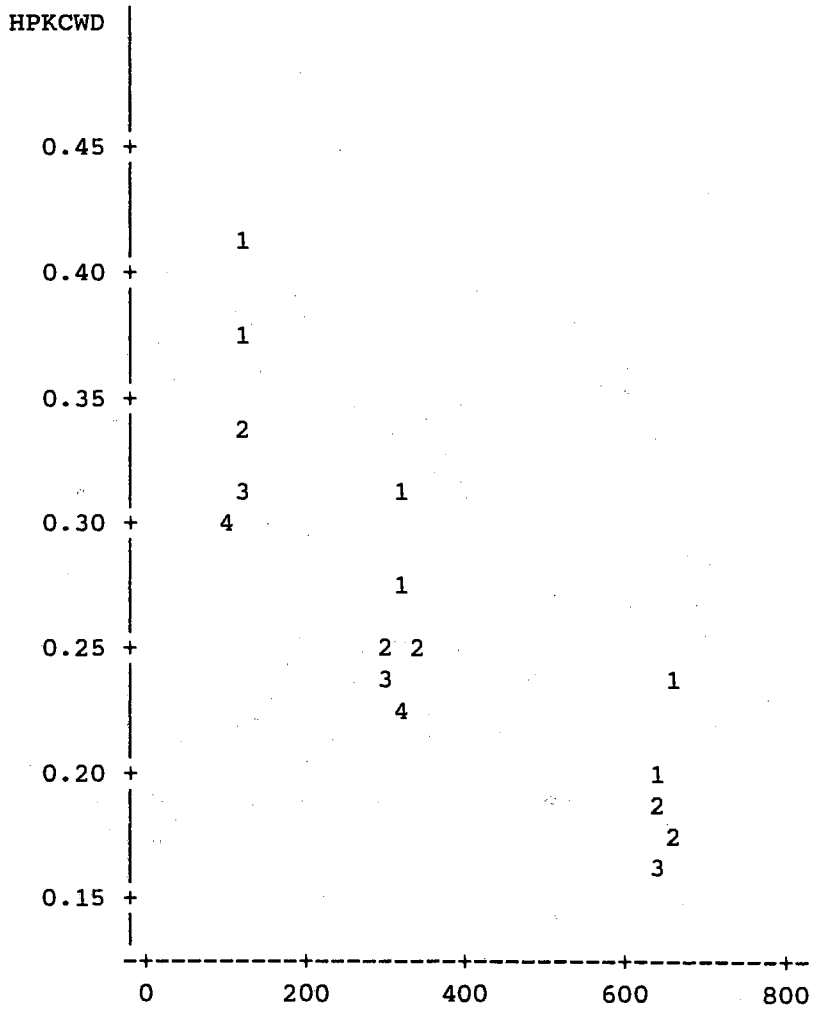


Figure 6. Heat production (KCal/gmwt/d) at maintenance of broilers aged 1 to 3 weeks in relation to body weight.

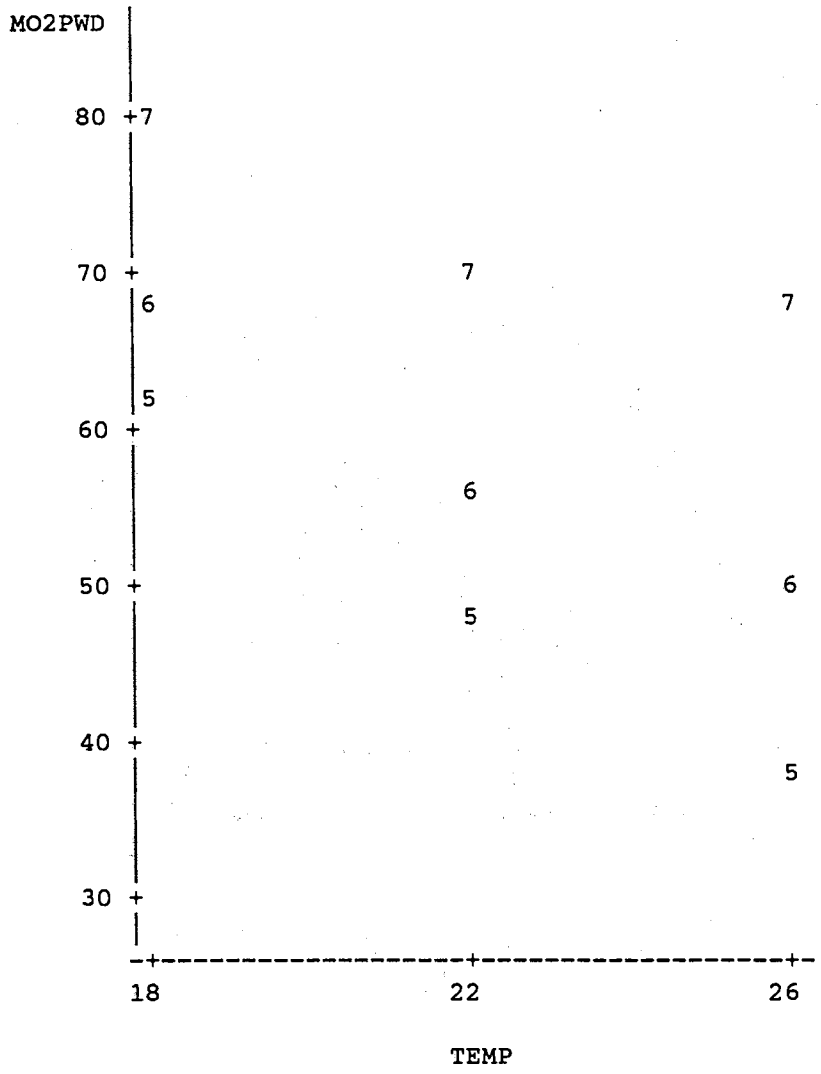


Figure 7. Oxygen (ml/gmwt/d) use for maintenance of broilers aged 5 to 7 weeks in relation to ambient temperature.

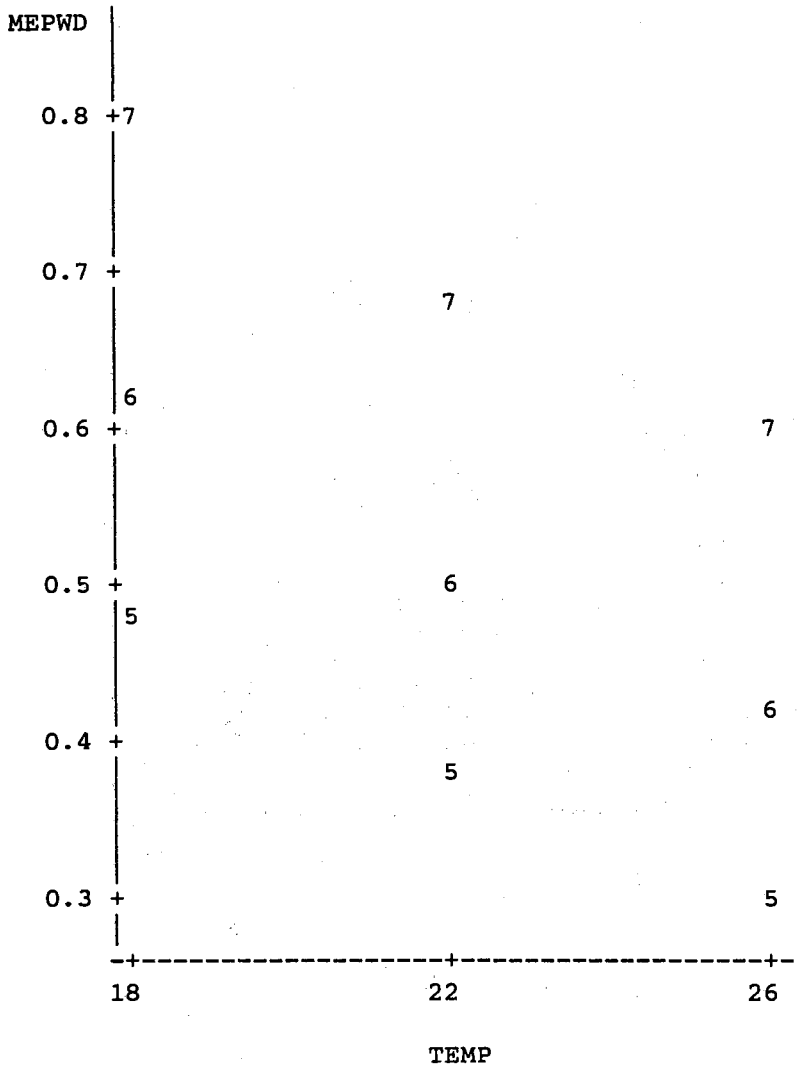


Figure 8. Maintenance energy (KCal/gmwt/d) need of broilers aged 5 to 7 weeks in relation to ambient temperature.

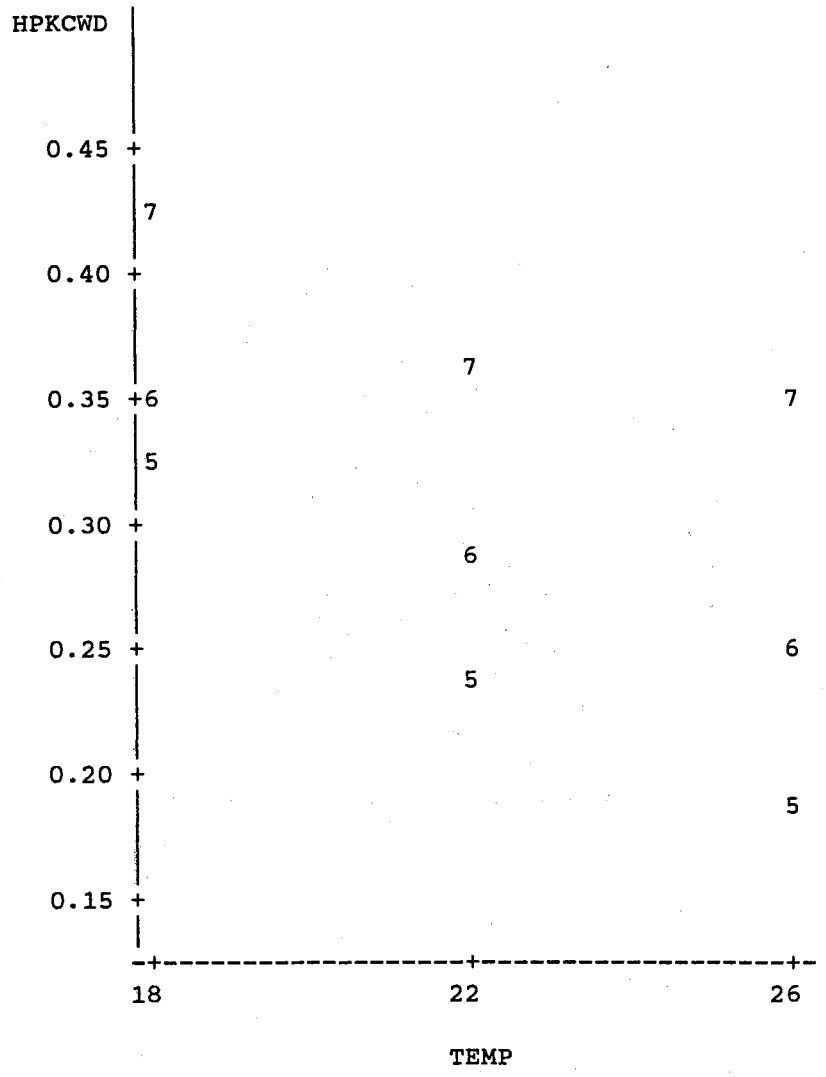


Figure 9. Heat production(KCal/gmwt/d) at maintenance of broilers aged 5 to 7 weeks in relation to ambient temperature.

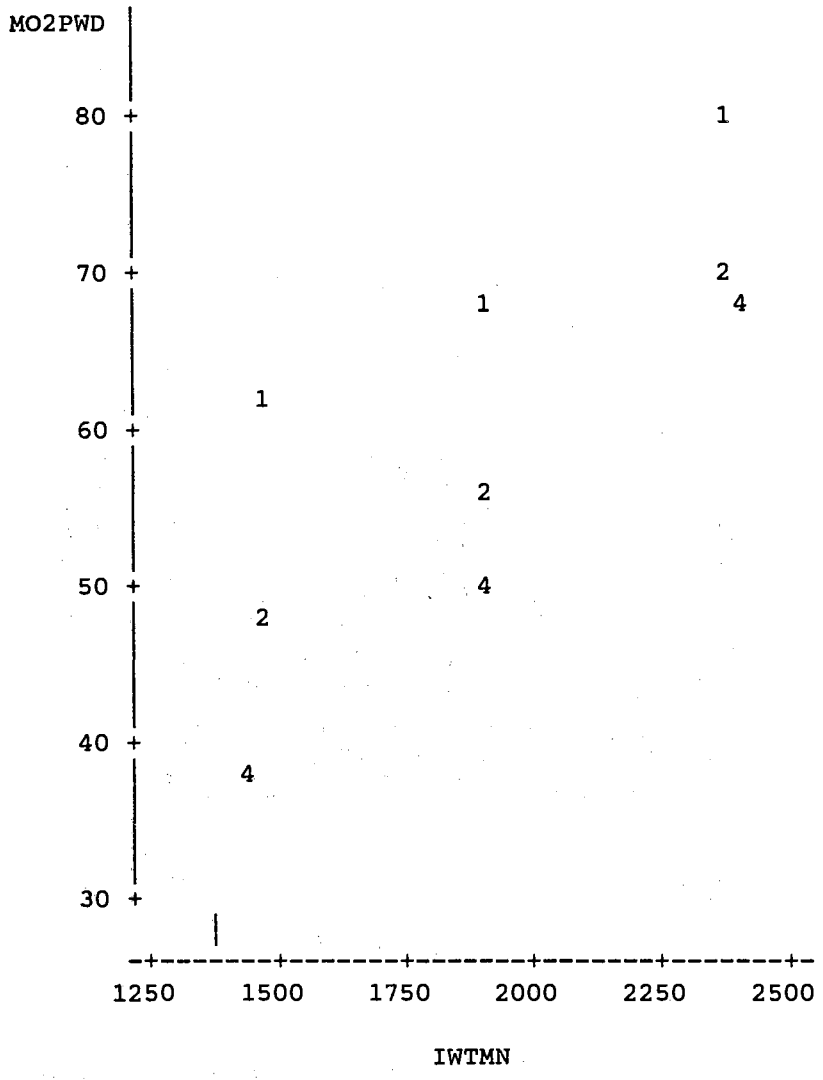


Figure 10. Oxygen (ml/gmwt/d) use for maintenance of broilers aged 5 to 7 weeks in relation to body weight

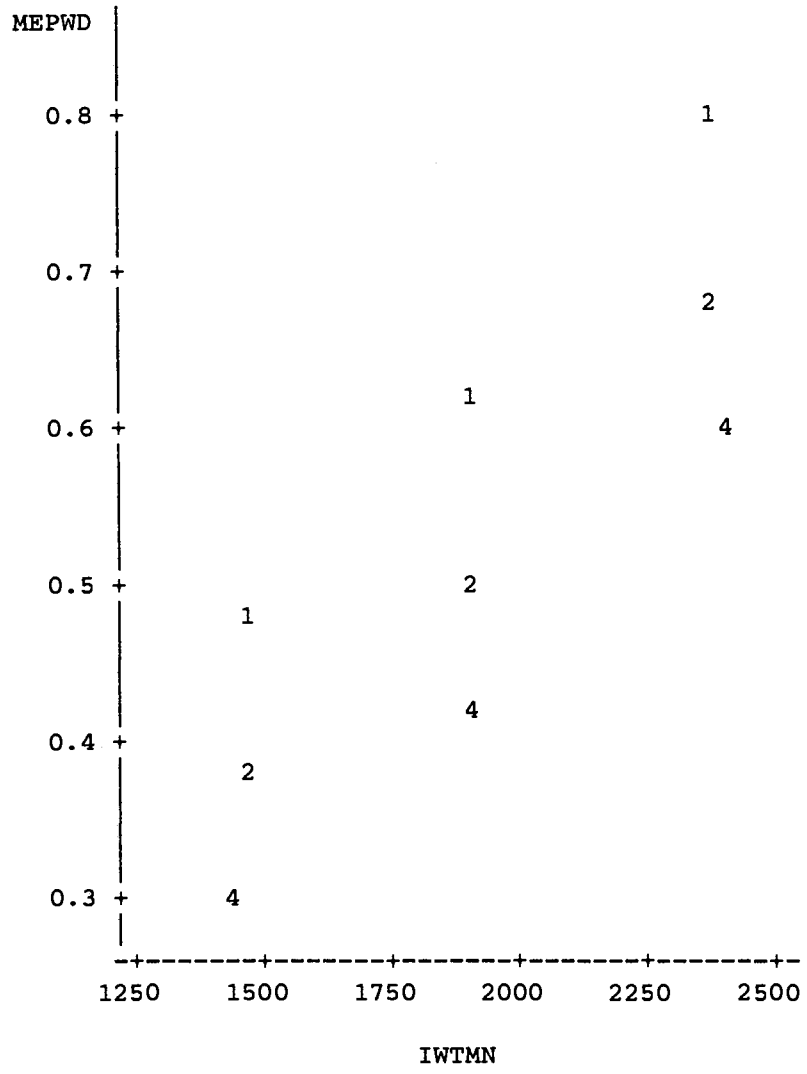


Figure 11. Maintenance energy (KCal/gmwt/d) need of broilers aged 5 to 7 weeks in relation to body weight.

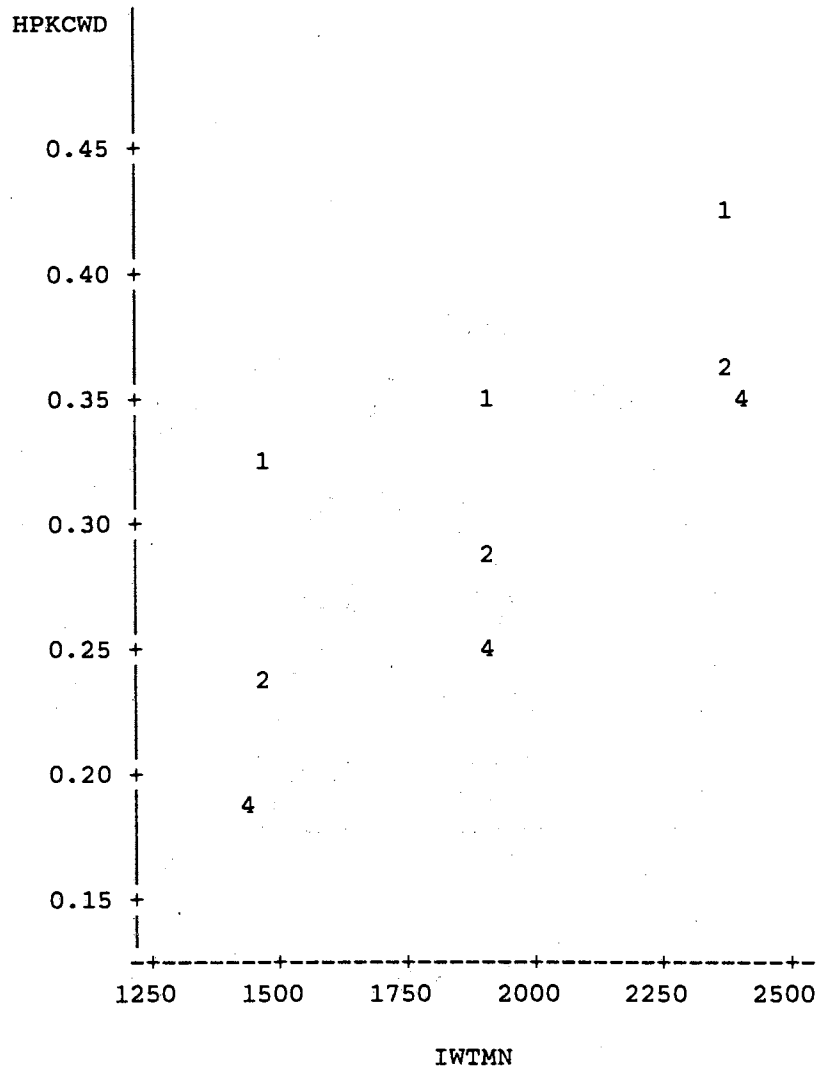


Figure 12. Heat production (KCal/gmwt/d) at maintenance of broilers aged 5 to 7 weeks in relation to body weight.

MAINTENANCE OXYGEN REQUIREMENT
OF 3 AND 5 DAY OLD BROILERS

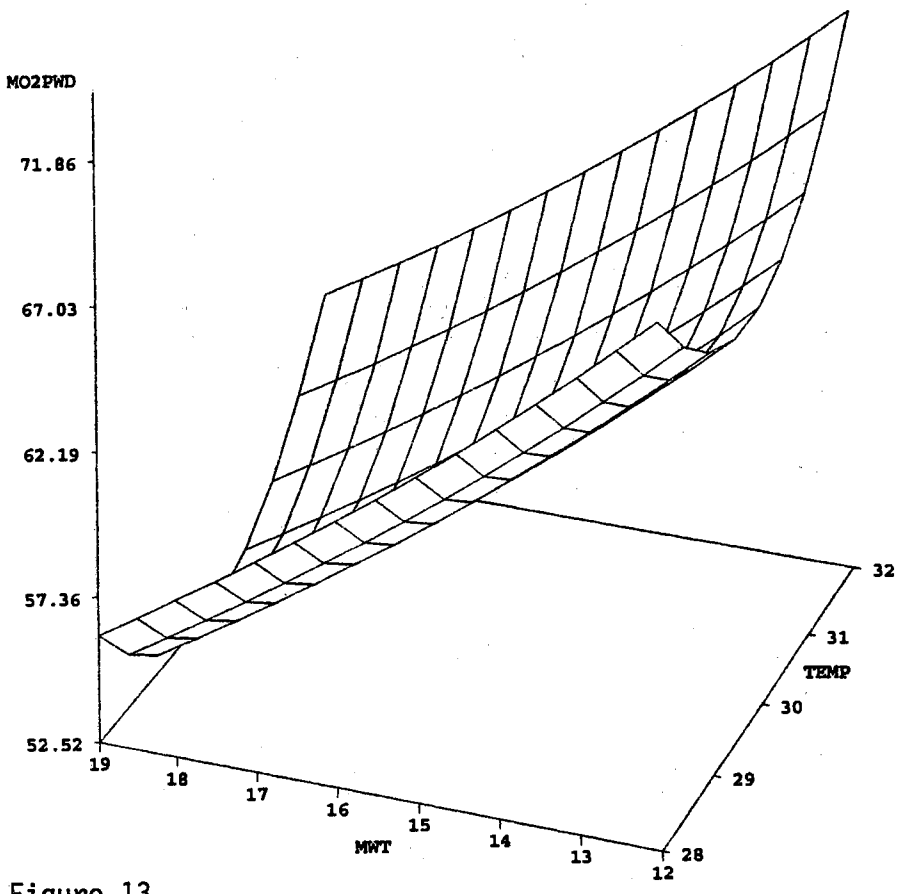


Figure 13

MO2PWD=MAINT. OXYGEN (ml/mwt/d). MO2PWD=1223.456202−14.198711W+0.395008WW
W=MWT=METABOLC WEIGHT. −73.897624T+1.234299TT+0.078121WT
T=TEMP=AMBIENT TEMP (C).

HEAT PRODUCTION AT MAINTENANCE
OF 3 AND 5 DAY OLD BROILERS

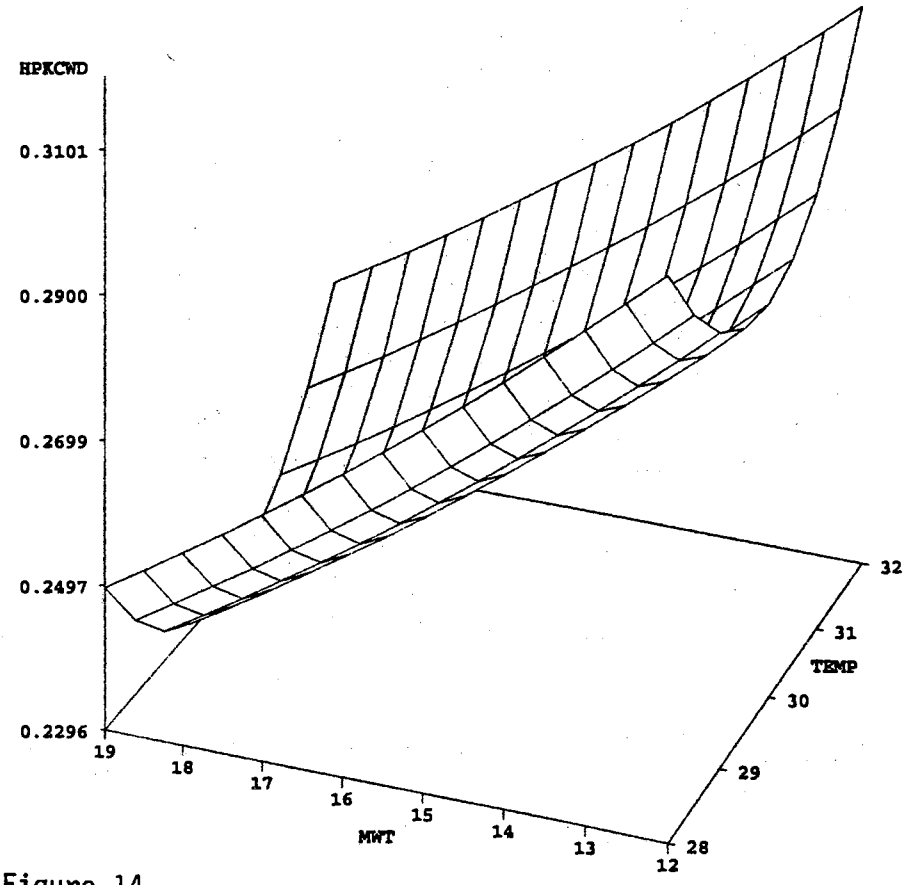


Figure 14

HPKCWD=HEAT PROD. (Kcal/mwt/d).
W=MWT=METABOLIC BODY WEIGHT.
T=TEMP=AMBIENT TEMP (C).

HPKCWD=5.800831−0.061568W+0.001702WW
−0.352352T+0.005857TT+0.000385WT

MAINTENANCE OXYGEN REQUIREMENT

OF 1 TO 3 WEEK OLD BROILERS

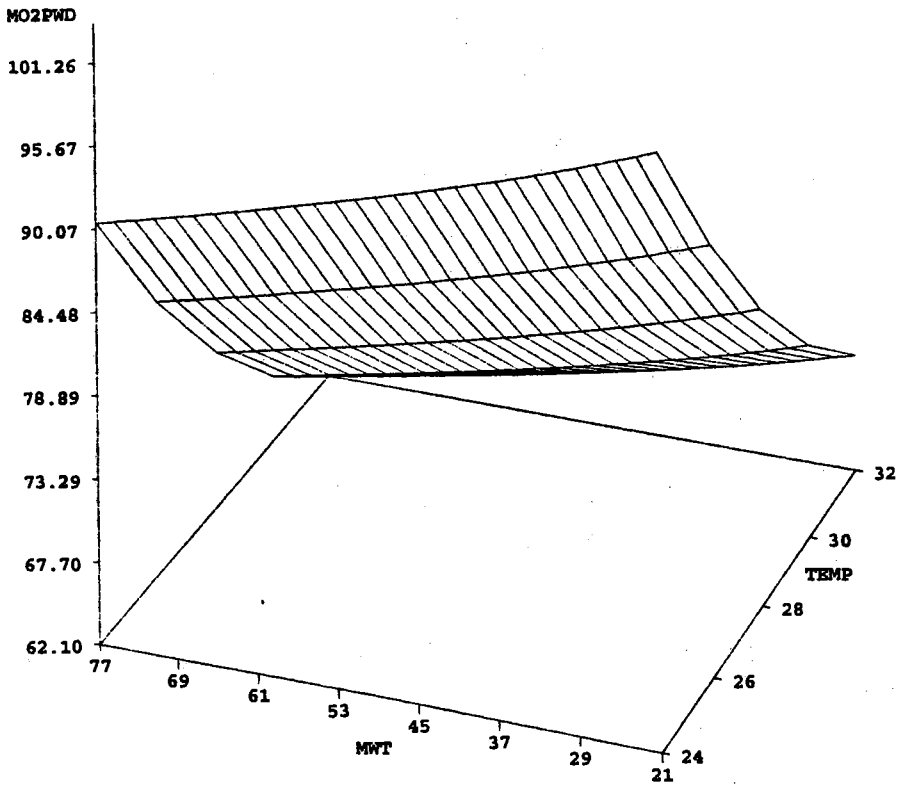


Figure 15

MO2PWD=MAINT. OXYGEN (ml/mwt/d).
W=MWT=METABOLC WEIGHT.
T=TEMP=AMBIENT TEMP (C).

MO2PWD=375.388080−1.872051W+0.004027WW
−16.285888T+0.217145TT+0.031527WT

MAINTENANCE ENERGY REQUIREMENT
OF 1 TO 3 WEEK OLD BROILERS

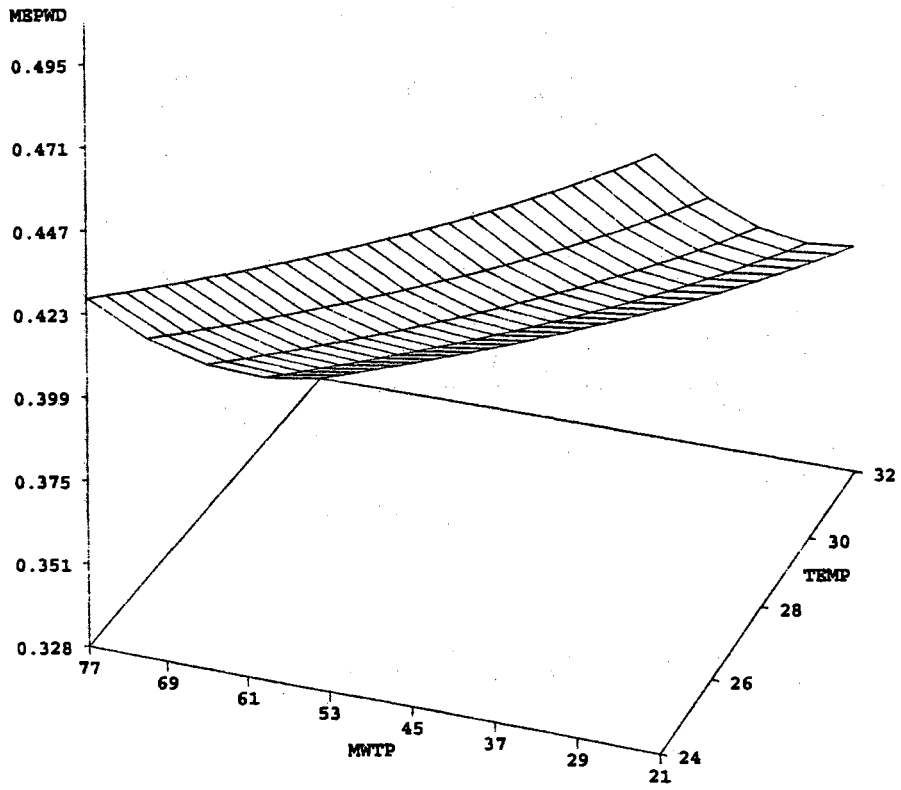


Figure 16

MEPWD=MAINT. ENERGY (Kcal/mwt/d).
W=MWT=METABOLIC BODY WEIGHT.
T=TEMP=AMBIENT TEMP (C).

MEPWD=1.196256−0.007362W+0.000044619WW
−0.037886T+0.000455TT−0.000004035WT

HEAT PRODUCTION AT MAINTENANCE
OF 1 TO 3 WEEK OLD BROILERS

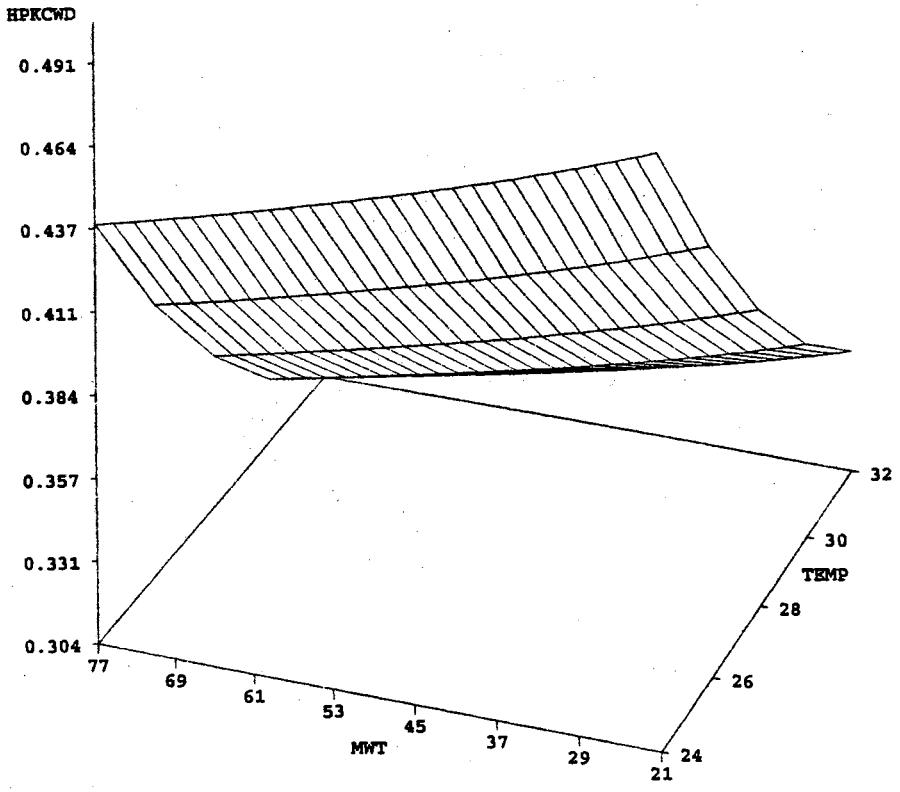


Figure 17

HPKCWD=HEAT PROD. (Kcal/mwt/d).
W=MWT=METABOLIC BODY WEIGHT.
T=TEMP=AMBIENT TEMP (C).

HPKCWD=1.830566−0.008816W+0.000020006WW
−0.080168T+0.001084TT+0.000142WT

MAINTENANCE OXYGEN REQUIREMENT

OF 5 TO 7 WEEK OLD BROILERS

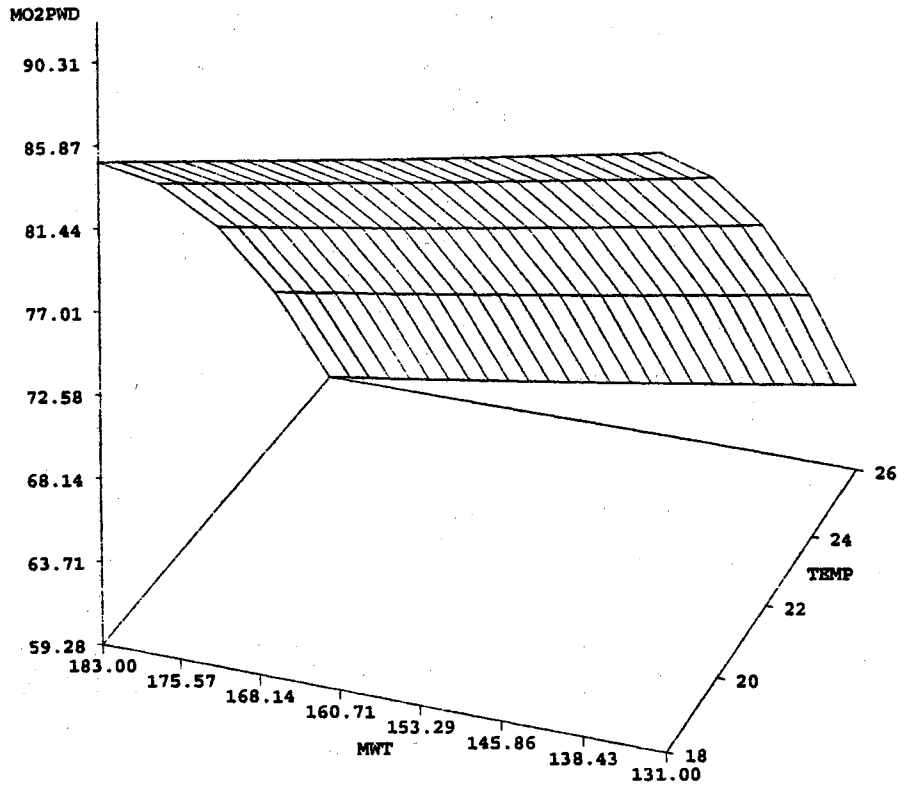


Figure 18

MO2PWD=MAINT. OXYGEN (ml/mwt/d).
W=MWT=METABOLC WEIGHT.
T=TEMP=AMBIENT TEMP (C).

MO2PWD=103.361775−1.154889W+0.002596WW
+3.443481T−0.159283TT+0.010726WT

MAINTENANCE ENERGY REQUIREMENT
OF 5 TO 7 WEEK OLD BROILERS

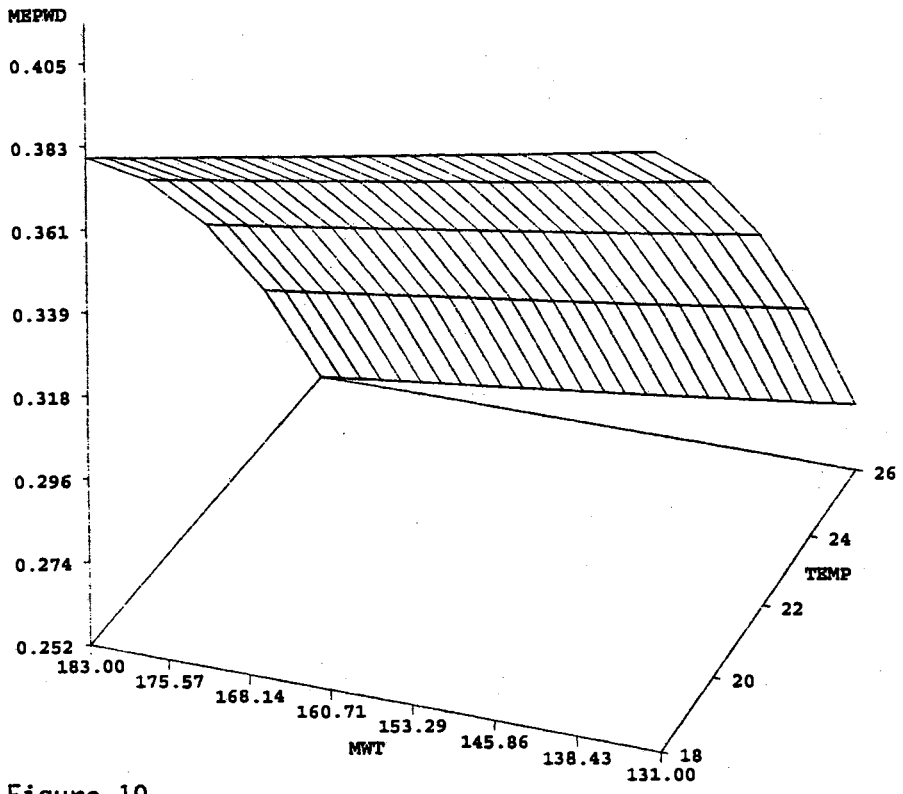


Figure 19

MEPWD=MAINT. ENERGY (Kcal/mwt/d).
W=MWT=METABOLIC BODY WEIGHT.
T=TEMP=AMBIENT TEMP (C).

MEPWD=0.506733−0.006897W+0.000013209WW
+0.014913T−0.000801TT+0.000130WT

HEAT PRODUCTION AT MAINTENANCE

OF 5 TO 7 WEEK OLD BROILERS

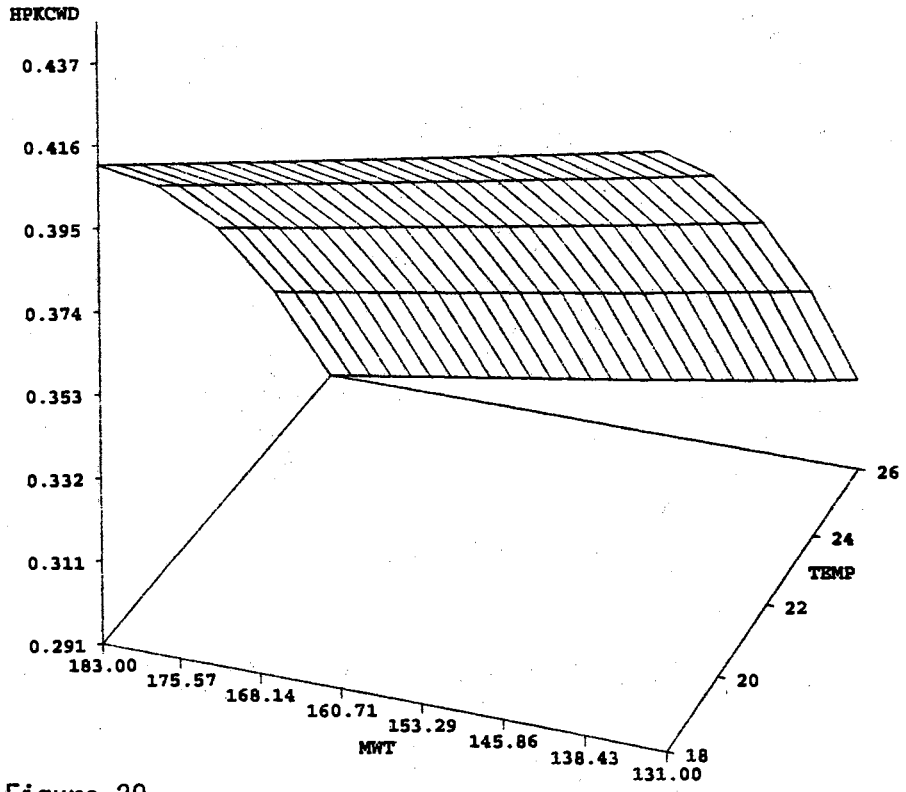


Figure 20

HPKCWD=HEAT PROD. (Kcal/mwt/d).
W=MWT=METABOLIC BODY WEIGHT.
T=TEMP=AMBIENT TEMP (C).

HPKCWD=0.500551−0.005571W+0.000012776WW
+0.016290T−0.000749TT+0.000048942WT

Table 2. Estimates of energy need for maintenance (MEm) and gain (MEg) using NRC, 1994 data.

BWT (g)	AMBIENT TEMP.(C)	ME Cons. (KCal/b/d)	MEm (KCal/b/d)	MEg (KCal/b/d)	METGR (KCal/g)
152	30	61.71	44.06	17.65	3.93
376	30	132.57	74.28	58.30	4.14
686	28	222.57	123.42	99.15	5.03
1085	28	322.29	222.94	99.35	5.65
1576	22	439.29	222.66	216.63	6.26
2088	22	521.57	295.58	225.99	7.13
2590	22	586.00	416.11	169.89	8.17

ME Cons.=metabolizable energy consumption.

Mem=maintenance energy need .

Meg=metabolizable energy need for weight gain.

METGR=Metabolizable energy to gain ratio.

Table 3. Exponents to convert live weight to metabolic body size of broilers at different weights and ambient temperatures.

Body weight (Kg)	Ambient temp	Exponent
1.46 to 2.4	18 C (lowest)	0.24
0.11 to 2.4	TN	0.62
0.11 to 2.4	26 C	0.67

REFERENCES

Beker, A., S. L. Vanhooser and R. G. Teeter, 1995. Effect of oxygen level on ascites incidence and performance in broiler chicks. Avian Diseases 39:285-291.

Brody, S. 1945. Bioenergetics and Growth. Haffner press, New York, p 371.

Brouwer, E., 1965. Report of sub-committee on constants and factors. In: Energy metabolism, pp. 441-443. K. L. Blaxter, ed. Academic Press, London.

Close, W. H., 1978. The effects of plane of nutrition and environmental temperature on the energy metabolism of the growing pig. Br. J. Nutr. 40:433-438.

Close, W.H. and M.W. Stanier, 1984. Effects of plane of nutrition and environmental temperature on the growth and development of the early-weaned piglets.

Dale, N., and A. Villacres, 1986. Influence of dietary density, calorie:protein ratio and supplemental fat on the incidence of ascites in broilers. Poultry Science, 65, Supp. 1, (abst.),163-164.

Emans, G. C., 1987. Growth, body composition and feed intake. World's poultry Science 43:208-224.

Ferrell, C. L., and T. G. Jenkins, 1985. Cow type and the nutritional environment: Nutritional aspects. J. Anim. Sci. 61-127.

Hernandez, A., 1987. Hypoxic ascites in broilers: a review of several studies done in Columbia. Avian Diseases, 31: 658-661.

Hoffmann, L. and R. Schiemann, 1973. In: Energy requirements of poultry, 1974, British Poultry Science Ltd, pp61-71.

Hurwitz, S., M. Weiselberg, U. Eisner, I. Bartov, G. Riesenfeld, M. Sharvit, A. Niv and S. Bornstein, 1980. The energy requirements and performance of growing chickens and turkeys as affected by environmental temperature. Poultry Science 59: 2290-2299.

Julian, R. J., G. W. Friars, H. French and M. Quinton, 1987. The relationship of right ventricular hypertrophy, right ventricular failure, and ascites to weight gain in broiler and roaster chickens. Avian Diseases, 31: 130-135.

Julian, R. J., I. McMillan and M. Quinton, 1989b. The effect of cold and dietary energy on right ventricular hypertrophy, right ventricular failure and ascites in meat type chickens. Avian Pathology, 18:675-684.

Julian, R.J., 1991. Poisons and toxins. Diseases of poultry, 9th edn, pp 863-884, Ames, Iowa State University.

Kleiber, M., 1961. The fire of life. John Wiley and Sons, Inc., New York.

Meltzer, A. 1983. Thermoneutral zone and resting metabolic rate of broilers. British Poultry Sci. 24:471-476.

Shlosberg, A., Zadikov, I, Bendheim, U, Handji, V, and Berman, E. (1992) The effects of poor ventilation, low temperature, type of feed and sex of bird on the development of ascites in broilers. *Avian Pathol.* **21**:369-382.

Pullar, J.D. and A.J.F. Webster, 1977. The energy cost of fat and protein deposition in the rat. BR. J. NUTR., 37:355-363.

CHAPTER IV

ATMOSPHERIC OXYGEN CONCENTRATION EFFECTS ON BROILER GROWTH, ASCITES INCIDENCE, SERUM CHEMISTRY AND HEMATOLOGY[1]

A. Beker, S. L. Vanhooser, and R. G. Teeter[2]
Department of Animal Science
Oklahoma Animal Disease Diagnostic Laboratory
Oklahoma State University
Stillwater, Oklahoma 74078-0425

Running Head: Broiler ascites, oxygen, serum chemistry.

[1] Agricultural experiment Station, Oklahoma State University, Stillwater, OK 74078.
[2] To whome correspondance should be addressed.

ABSTRACT 1. An experiment was conducted using commercial broiler chicks maintained at 26.9 C to evaluate the effect of atmospheric oxygen concentration (13.6, 20.6 ml/100ml indicated as % hereafter) on performance, ascites incidence, hematology and serum chemistry.

2. Necropsy, performed on all birds that died, indicated that ascites occurred in birds held at 13.6 % atmospheric oxygen by day 5 of the study. The lower oxygen concentration decreased body weight gain, survivability, white blood cell count and serum glucose but increased($P<.01$)blood hematocrit, hemoglobin and serum triglycerides.

3. Lactate dehydrogenase (LDH) values were greater ($P<.05$) for chicks maintained at 20.6 % than at 13.6 % atmospheric oxygen and in birds with than without ascites at 20.6% atmospheric oxygen. No effects of ascites were noted ($P>.05$) for ALT, AST, glucose, triglycerides, uric acid, creatinine, total protein or albumin. Data presented corroborate the previously reported effect that altitude exacerbate ascites and provide a serum chemistry base for future reference.

INTRODUCTION

Ascites causes broiler mortality especially at higher altitudes. Mortality rates exceeding 25 % have

been reported in these regions (Lopez-Coelo, 1985). However, the syndrome has grown to be a major economic problem to the world's poultry industry at all altitudes (Lopez-Coelo, 1985; Brake and Garlich, 1996). Diagnosis of the syndrome relies on accumulation of an amber or clear plasma-like fluid in the abdominal cavity (Dale and Villacres, 1986; Huchzermeyer and De Ruyck, 1986; Wideman, 1988).

The ascites syndrome is caused by pulmonary hypertension due to oxygen deficit caused by rapid growth or an increased metabolic rate due to either cold, high dietary salt, inadequate ventilation in poultry houses and/or toxicants (Anderson et al. 1986; Julian et al. 1986; Wideman, 1988; Brake and Garlich, 1996).

Low ambient temperature stress presumably elevates the incidence of ascites by increasing the metabolic rate. Huchzermeyer et al. (1989) reported that cold exposure markedly elevated the oxygen requirement of chicks. Julian et al. (1989) and Stolz et al. (1992) observed that pulmonary hypertension increased in birds exposed to a cold environment. Moye et al. (1969) further suggested that cold stress increases blood viscosity by decreasing blood volume; this could elevate pulmonary hypertension and induce ascites. Little work has examined other blood constituents. The increased basal metabolic rate (BMR)

at a low ambient temperature could have profound effects on bird serum chemistries. The purpose of this study was to simulate two altitudes under mild cold stress to examine their effect on ascites incidence, growth performance, and blood chemistry.

MATERIAL AND METHODS

GENERAL:

Three hundred and sixty, one-day-old male commercial broiler chicks were weighed, divided into 24 groups of 15 chicks each and allocated to respiratory chambers in a completely randomized experimental design. The respiratory chambers utilized have been described previously (Wiernusz and Teeter, 1993; Belay and Teeter, 1993). All chicks were given *ad-libitum* access to Starter ration (Table 1) and drinking water.

ENVIRONMENT:

Environmental conditions included two oxygen concentrations(13.6 and 20.6 ml/100ml); these were achieved by dilution of ambient air (20.6%) with nitrogen gas. Ambient temperature was maintained at 26.9 C.

VARIABLES:

Mortality was monitored four times each day. Dead chicks were weighed and immediately examined for gross heart, liver and lung lesions. Upon completion of the 15 day study, live chicks were weighed; blood samples for serum chemistry and hematology were collected via cardiac puncture from all remaining chicks; chicks then were sacrificed by cervical dislocation, and necropsied. The necropsy procedure was as follows: Each liver, lung, and heart was removed and weighed. The ascites heart index (AHI; right ventricular weight/total ventricular weight) was determined and expressed as a percentage of the final body weight (FBW). Tissue specimens of the heart, lung, and liver were fixed in 2.5% glutaraldehyde solution, sectioned and stained with hematoxylin and eosin for microscopic examination.

Blood for hematological evaluation was collected and analyzed for leukocytes (WBC) and erythrocytes (RBC) by the hematocytometer method. Hemoglobin (HGB) was measured as cyanomethemoglobin (J. T. Baker System 9000, Allentown, PA). Hematocrits were determined by microcentrifugation. Serum concentrations of inorganic phosphorus, calcium, sodium, potassium, creatinine, total protein, albumin, glucose and uric acid as well as the activity of alanine aminotransferase (ALT),

aspartate aminotransferase (AST) and lactate dehydrogenase (LDH) were determined using Roche kits[3] for phosphorus (No.44031), calcium (No.44033), creatinine (No.44905), total protein (No.44903), albumin (No.44902), glucose (No.44557),Uric acid (No.44124), alanine aminotransferase (No.44644), aspartate aminotransferase (No.44645), and lactate dehydrogenase (No.43623). In all instances, serum variables were measured using a Cobas Mira wet chemistry analyzer[4] with sodium and potassium values assayed using the sodium and potassium selective electrode module (No.44498) of the Cobas Mira.

BREATHING AIR SUPPLY AND ANALYSIS: Compressed dried air (7 % RH; 20.6 % O_2) was delivered to the control chicks and those designated as high altitude (7% RH; 13.6% O_2). The 13.6 % atmospheric oxygen concentration was created by diluting dried air with N_2 at the ratio of air to N_2 of 8.55:1.45. It was necessary to provide low RH air to the chick chamber in order to hold the chamber at a RH below 75 %. The concentration of O_2 and CO_2 in air exiting each bird's chamber was monitored twice each day using Ametek[5] oxygen (accuracy +.02%) and carbon dioxide (accuracy +.03%) analyzers, respectively.

---

[3]Hoffmann-LaRoche, Nutley, NJ 07042.
[4]Roche Diagnostics Systems Inc., Montclair, NJ 07042-5199.
[5] Ametek

STATISTICAL ANALYSIS

Data for all response variables were subjected to analysis of variance using the General Linear Model procedure of SAS(SAS, 1985). First, effects of oxygen concentration on performance and ascites incidence were measured. Then, because chicks with ascites were detected at both oxygen concentration chambers, subsequent measurements were analyzed as a 2 by 2 factorial experiment with oxygen and ascites as the two factors (birds in each chamber with and without ascites at the two atmospheric oxygen concentrations were used as experimental units). When an interaction between oxygen concentration and ascites presence was detected, individual least square means were contrasted (Steel and Torrie, 1960). Least squares means are presented in all tables.

EXPERIMENTAL RESULTS

Birds receiving 13.6% oxygen exhibited a higher ($P<.01$) ascites incidence than birds at 20.6% oxygen. The first case of ascites mortality was observed among the chicks maintained at 13.6% oxygen on day 5 of the 15-day experiment. At necropsy, chicks with ascites exhibited enlarged heart with dilation of the atria and right ventricle; lungs were moist and exuded foamy fluid when cut.

The liver was congested and often covered with a thin layer of clotted proteinaceous fluid. The measurable quantity of ascitic fluid in the abdominal cavity ranged from 0.2 ml to 19 ml. No such lesions were observed in chicks without ascites.

The principal histopathologic lesion in the lung was dilation of the parabronchi and associated atria with hypertrophy of the parabronchial smooth muscle. Atelectasis of the adjacent air capillaries and marked congestion also were present. Random parabronchi and groups of air capillaries contained proteinaceous fluid or strands of proteinaceous material and occasionally hemorrhage.

In the myocardium, particularly the atria, atrophy and vacuolation of myofibers and interstitial edema were observed; liver exhibited individualization and atrophy of the centrilobular hepatocytes with dilation of the sinusoids. Occasionally, coagulative necrosis of centrilobular hepatocytes was observed. Compared to chicks at 13.6% oxygen, chicks housed at 20.6% oxygen had greater ($P<.01$) weight gain and survival. Total feed disappearance was similar; however, accuracy of feed intake and the gain to feed ratio is questionable due to excessive feed waste. The survivability observed for chicks maintained at 20.6% oxygen (54%) was lower than expected, presumably due to the low ambient temperature employed.

Because ascites occurred under both atmospheric conditions, we obtained measurements from ascitic and nonascitic chicks at each of the two oxygen concentrations. When an interaction was detected, individual means were compared using least square analysis of variance. Organ weights are presented in Table 3. Chicks at 13.6% oxygen with ascites exhibited greater ($P<.05$) right ventricle (RV) weight and ascites heart index (AHI) than chicks in all other groups. Chicks with ascites had smaller lung($P<.03$) and a tendency ($P<.07$) for smaller livers than nonascitic chicks at both atmospheric oxygen concentrations. Chicks at 13.6% oxygen had heavier lung($P<.01$) and greater($P<.03$) ascites heart index (AHI) than chicks at 20.6% oxygen.

Results of blood cell analyses are presented in Table 4. Regardless of ascites presence, red blood cell concentration and hemoglobin concentrations were greater ($P<.05$) and WBC was less ($P<.05$) with the 13.6% oxygen concentrations.

Serum chemistries are presented in Table 5. The only differences detected were that glucose was higher and triglyceride concentrations were lower for chicks at 20.6% than at 13.6% oxygen ($P<.01$). An interaction between ascites and oxygen concentration was detected in which lactate dehydrogenase (LDH) was lower ($P<.05$) for normal chicks at 20.6% oxygen than for chicks with

ascites at 20.6% oxygen or chicks without ascites.

Serum mineral concentrations are presented in Table 6. No effects of ascites were detected. However, chicks at 13.6% oxygen had lower ($P<.01$) sodium and lower ($P<.04$) calcium but greater ($P<.01$) phosphorus concentrations in blood serum compared with chicks at 20.6% oxygen.

DISCUSSION

The elevated ($P<.01$) ascites incidence for chicks maintained at 13.6% oxygen (Table 1) is consistent with earlier observations that ascites incidence is greater under high altitude conditions. Previous workers have suggested that this condition is related to pulmonary hypertension (Hernandez, 1987; Julian et al, 1986) and cold stress (Bendheim et al. 1992; Shlosberg et al. 1992). The elevated incidence of ascites (64.47) observed in chicks at the simulated higher altitude therefore was likely aggravated by the cool ambient temperature that we utilized. The lower weight gains and decreased survival presumably reflect an imbalance between metabolic oxygen demand and consumption ability. The reduced growth rate for ascitic chicks is consistent with findings reported by of Maxwell et al. (1986).

The high mortality observed in chicks maintained under both atmospheric oxygen concentrations probably

was induced by the cold stress imposed. Temperature was maintained at 26.9 C compared to 30 C recommended for growth and survival of 2 week old chicks.

Ascitic chicks at 13.6% oxygen, as judged by abdominal tract fluid presence, also exhibited the greatest AHI ((Right ventricle/total ventricle)*100). An increase in this ventricular weight ratio is consistent with compensatory dilation of the right ventricle as a result of increased pulmonary arterial pressure (Burton et al. 1967; Cueva et al. 1974; Hernandez, 1987). The decreased liver and lung weights (4.83 and 0.99% of BWT) of ascitic chicks may have been due to hepatocellular atrophy and degeneration.

Increased lactate dehydrogenase (LDH) of chicks with ascites at normal altitude (20.6 % oxygen) might have been the result of lung damage. Low glucose concentration detected in chicks maintained at lower atmospheric oxygen concentration might be due to a change in hormonal balance as a result of the stress condition where the concentration of growth hormone is elevated. Decrease in triglyceride concentration of chicks at the lower atmospheric oxygen concentration is the result of fat mobilization from adipose tissue due to their low feed consumption.

Changes in hematology were noted in previous studies of ascites. Hernandez(1987) reported that hemoglobin (HGB) and hematocrit (HCT) were increased

by over 40% in ascitic broilers at 2,638 m above sea level. Maxwell (1990) reported that there was an outbreak of ascites near sea level during which HGB, HCT and RBC increased by 24, 26 and 22% respectively. Shlosberg et al.(1992) reported that cold stress increases packed cell volume that can lead to increased resistance to blood flow, pulmonary hypertension and cardiac arrest. In the present study, HCT and HGB both were increased by the lower oxygen concentration while no independent effect of ascites was detected. An increase in HCT and in HGB concentration acts as a compensatory mechanism to the stimulus of reduced oxygen saturation at higher altitude to increase the oxygen carrying capacity of the blood (Yersin et al. 1992). Perhaps of even greater importance, white blood cell concentrations were reduced by over 40% by the lower oxygen concentration. With a lower oxygen concentration and higher respiration rate, deeper breathing may increase uptake by pathogens and air-born toxins by the lungs. Combined with decreased immune status, higher amounts of toxins will be absorbed which may initiate damage to the heart and vascular system that is associated with ascites.

An increase in inorganic phosphorus and a decrease in total serum calcium of birds at higher altitude presumably could be due to hypoxia increasing

glucocorticoid activity. Hypoxia is a potent stimulus for the release of catecholamines from the sympathetic nervous system and the adrenal medulla into the circulation (Cheung, 1989); this disturbs electrolyte homeostasis at the cellular and subcellular levels and results in accumulation of calcium and loss of phosphorus (Yersin et al. 1992).

In conclusion, our results indicate that exposing chicks to 13.6% oxygen concentration and 26.9 C markedly reduces growth rate, induces ascites, elevates mortality, and alters chick serum chemistry and hematology. Although the low atmospheric oxygen and cold stress condition employed induced a severe oxygen deficit, the rapidity of the development of ascites suggests that oxygen availability in the time period immediately following hatching plays a significant role in the incidence of ascites. Further research is needed to quantify the critical concentrations of oxygen, ambient temperature and age that exacerbates ascites.

Table 1. Composition of the experimental ration employed

Ingredients	g\kg
Corn, ground	523.2
Soy meal (49%)	369.3
Fat (animal and vegetable)	64.6
Dicalcium phosphate	17.7
Limestone	11.9
Salt	4.5
Vitamin mix[A]	2.5
Methionine (99%)	2.2
Trace mineral mix[B]	1.0
Ethoxyquin	0.2
Energy (MJ ME/kg)	13.39
Crude protein (g/kg)	225

[A]Mix supplied the following per kilogram of diet: Vit A, 5.25mg; cholecalciferol, 0.125mg; vitamin E, 0.025mg; vitamin B12, 0.03 mg; riboflavin, 15 mg; niacin, 75 mg; d-panthothenic acid, 25 mg; cholin, 705.5 mg; menadione, 5 mg; folic acid, 1.5 mg; pyridoxine, 6.25 mg; thiamin, 3.03 mg; d-biotin, 0.127 mg.

[B]Mix supplied the following per kilogram of diet: Manganese, 120 mg; zinc, 100 mg; copper, 10 mg; iodine, 2.5 mg; calcium, 135 mg; iron, 75 mg; selenium, 0.15 mg.

Table 2. Atmospheric oxygen concentration effects on ascites incidence and performance of broiler chicks.

	Oxygen concentration (%)			
Measurement	13.6	20.6	SE	P>F
Birds, no	180	180		
Ascites(%)	64.47	35.53	0.04	0.0001
Weight gain(g)	64.13	105.02	2.93	0.0001
Feed cons (g)	221.07	219.61	17.66	0.9540
Survival (%)	30.00	54.44	0.03	0.0001

Table 3. Atmospheric oxygen concentration effects on chick organ weight with and without ascites symptoms.

Measure	Oxygen concentration(%)				Asc	*Ox*	*Int*
	13.6		20.6		P<	*P<*	*P<*
	Without ascites	With ascites	Without ascites	With ascites			
Birds,No	48	53	62	25			
RV	0.27±0.018	0.37±0.017	0.28±0.016	0.29±0.026	.01	*.10*	*.03*
Heart	1.43±0.05	1.53±0.05	1.40±0.04	1.43±0.070	.25	*.29*	*.53*
Lung	1.07±0.029	0.99±0.027	0.85±0.025	0.79±0.040	.03	*.01*	*.77*
Liver	5.43±0.18	4.83±0.17	5.24±0.16	5.23±0.250	.07	*.83*	*.13*
AHI	0.30±0.012	0.38±0.011	0.28±0.01	0.32±0.016	.01	*.03*	*.04*

Measure=measurement, Asc=ascites, OX=oxygen, Int=interaction, FBW=final body weight, RV=right ventricle; +AHI=Ascites heart index ((right ventricle/total ventricle)*100),BW= final body weight

Table 4. Atmospheric oxygen concentration effects on chick hematology with and without ascites symptoms.

Measurement	Oxygen concentration(%)				*Asc*	*Ox*	*Int*
	13.6		20.6				
	Without ascites	With ascites	Without ascites	With ascites	*P<*	*P<*	*P<*
Birds, No							
RBC ($10^6 mm^{-3}$)	1.86±0.10	2.07±0.13	1.82±0.09	1.78±0.14	*.47*	*.28*	*.29*
HCT (%)	36.61±1.80	40.81±2.38	33.78±1.59	31.50±2.54	*.65*	*.02*	*.13*
HGB ($g100ml^{-1}$)	7.76±0.38	8.76±0.51	7.31±0.34	7.06±0.54	*.41*	*.05*	*.17*
WBC ($10^3 mm^{-3}$)	11.12±2.69	10.11±3.55	17.91±2.37	21.17±3.80	*.72*	*.01*	*.50*

Asc=ascites, OX=Oxygen, Int=interaction, RBC=red blood cells, HGB=haemoglobin, HCT=hematocrit, WBC=white blood cells

Table 5. Atmospheric oxygen concentration effects on chick serum chemistry with and without ascites symptoms.

	Oxygen concentration(%)						
	13.6		20.6		*Asc*	*Ox*	*Int*
Measurement	Without ascites	With ascites	Without ascites	With ascites	*P<*	*P<*	*P<*
Birds, No	*21*	*8*	*21*	*6*			
ALT(µg/l)	2.74±0.49	1.63±0.75	2.71±0.47	1.67±0.87	*.11*	*.99*	*.96*
AST(µg/l)	322.5±25.43	275.5±41.20	302.4±25.43	303.0±47.58	*.48*	*.79*	*.51*
LDH(µg/l)	2868±351	2466±608	1763±255	3280±527	*.12*	*.09*	*.05*
GLU(mg/dl)	86±14.61	99±20.67	215±13.07	210±20.67	*.83*	*.01*	*.60*
TRIG(mg/dl)	112.2±8.09	105.9±13.10	80.2±8.09	75.5±15.13	*.62*	*.01*	*.94*
URIC(mg/dl)	7.07±1.44	4.8±2.15	5.4±1.36	6.8±2.49	*.75*	*.69*	*.34*
Crea(mg/dl)	0.09±0.02	0.05±0.03	0.09±0.02	0.10±0.04	*.54*	*.52*	*.46*
TP(g/dl)	3.42±0.14	3.38±0.22	3.36±0.16	3.50±0.25	*.85*	*.95*	*.66*
ALB(g/dl)	1.51±0.10	1.38±0.15	1.48±0.11	1.46±0.17	*.56*	*.99*	*.69*

Asc=ascites, OX=oxygen, Int=interaction, ALT=alanine aminotransferase, AST=aspartate aminotransferase, LDH=lactate dehydrogenase, Glu=glucose, Trig=triglyceride, Uric=uric acid, Crea=creatinine, TP=total protein, ALB=albumin

Table 6. Atmospheric oxygen concentration effects on chick serum minerals with and without ascites symptoms.

Measurement	Oxygen concentration(%)				*Asc*	*Ox*	*Int*
	13.6		20.6				
	Without ascites	With ascites	Without ascites	With ascites	*P<*	*P<*	*P<*
Birds, No	*0 to 19*	*1 to 8*	*5 to 15*	*1 to 6*			
Ca(mg/dl)	9.35±0.53	9.98±0.81	10.94±0.59	10.93±1.03	*.63*	*.04*	*.68*
P(mg/dl)	11.88±0.80	10.35±1.20	5.03±0.87	5.64±1.38	*.58*	*.01*	*.33*
Na(mmol/l)	142.7±0.98	143.9±1.59	146.9±0.98	147.0±1.84	*.61*	*.01*	*.72*
K(mmol/l)	ND	9.50±1.45	8.76±0.65	9.60±1.45	*.62*	*.96*	-

Asc=ascitES, OX=oxygen, Int=interaction, Ca=calcium, P=phosphorus, Na=sodium, K=potassium, ND=not determined

REFERENCES

Anderson, L. S., Gleeson, M., Haigh, A.L. and Molong, V. (1986) Respiratory responses of the domestic fowl to low level carbon dioxide exposure. *Res. Vet. Sci.* **40:** 99-104.

Bendheim, U., Berman, E., Zadikov, I. and Shlosberg, A. (1992) The effect of poor ventilation, low temperatures, type of feed and sex of bird on the development of ascites in broilers. Production parameters. *Avian Pathology* **21:** 383-388.

Burton, R.R., Besch,E.L., and Smith, A.H., (1967). Effect of chronic hypoxia on the pulmonary arterial blood pressure of the chicken. *Am. J. Physiol.* **214**:1438-1445.

Cheung, C.Y., (1989). Direct adrenal medullary catecholamine response to hypoxia in fetal sheep. *J. Neurochem.* **52**:148-153.

Cueva.S., Sillau, H., Valenzuela, A., and Ploog, H, (1974). High altitude inducedpulmonary hypertension and right heart failure in broiler chickens. Res. Vet. Sci. **16**:370-374.

Dale, N. M. and Villacres, A, (1986). Dietary factors affecting the incidence of ascites. *Proceedings of the Georgia nutrition conference*, **pp** 79-85.

Hernandez, A. Hypoxic ascites in broilers (1987) A review of several studies done in Colombia. *Avian Dis.* **31:** 658-661.

Huchzermeyer, F. W. and De Ruyck, A.M.C. (1986) Pulmonary hypertension syndrome associated with ascites in broilers. *Veterinary Record,* **119:**94.

Huchzermeyer, F. W., Van Der Colf, W. J. And Guinane, P. R., (1989) Broiler ascites: increased oxygen demand with cold may explain high winter incidence. *(SAPA) Poultry Bulletin,* September, **474** and **483.**

Julian, R. J., McMillan, I. and Quinton, M., (1989b) The effect of cold and dietary energy on right ventricular hypertrophy, right ventricular failure and ascites in meat-type chickens. *Avian Pathology,* **18:** 675-684.

Julian, R.J., Friars, G.W., French, H. and Quinton, M. (1986) The relationship of right ventricular

hypertrophy, right ventricular failure, and ascites to weight gain in broiler and roaster chickens. *Avian Dis* **31:**130-135.

Lopez-Coello, C., Odom, T.W and Wideman, R. F (1985) Ascites major causes of mortality in broilers. *Poultry Digest* **Vol. 44**, No. 521, pp. 284-288.

Maxwell, M. H., Robertson, G.W and Spence, S. (1986a) Studies on an ascites syndrome in young broilers. 1. Hematology and pathology. *Avian pathol*. **15:** 511-524.

Maxwell, M. H., Spence, S., Robertson, G.W. and Mitchell, M.A. (1990). Hematological and morphological responses of broiler chicks to hypoxia. *Avian pathol* **19:** 23-40.

Moye, R.J., Washburn, K.W. and Huston, T.M. (1969) Effects of environmental temperature on erythrocyte numbers and size. *Poultry Science* **48:**1683-1686.

SAS Institute (1985) SAS user's guide. Version 5 Edition. SAS Inst. INC., Cary, NC.

Shlosberg, A., Zadikov, I, Bendheim, U, Handji, V, and Berman, E., (1992). The effects of poor ventilation, low temperature, type of feed and sex of bird on the development of ascites in broilers. *Avian Pathol*. **21:**369-382.

Steel, R.D.G., and Torrie, (1960). Principles and procedures of statistics. McGrew Hill Book Co., NY.

Stolz, J.L., Rosenbaum, L.M., Jeong, D. And Odom, T.W. (1992). Ascites syndrome mortality and cardiological responses of broiler chickens subjected to cold exposure. *Poultry Science,* **71,** Supp. 1, (Abst. 12), 4.

Wideman, R.F.Jr,(1988). Ascites in poultry. *Monsanto Nutrition Update,* **Vol. 6:** (2), pp. 1-7.

Wiernusz, C. J. and R. G. Teeter, (1993). Feeding effects on broiler thermobalance during thermoneutral and high ambient temperature exposure. Poultry Sci. **72:**1917-1924.

Yersin, A.G., Huff, W.E., Kubena, L.F., Elissalde, R.B., Harvey, R.B., Witzel, D.A., and Giroir, L.E., (1992). Changes in hematological, blood gas, and serum biochemical variables in broilers during exposure to simulated high altitude. *Avian Diseases* **36:**189-196.

CHAPTER V

OXYGEN CONCENTRATION, AMBIENT TEMPERATURE AND METAPROTERENOL EFFECTS ON ASCITES INCIDENCE AND BROILER PERFORMANCE[1]

A. Beker, S. L. Vanhooser, and R. G. Teeter[2]
Department of Animal Science
Oklahoma State University
Stillwater, Oklahoma 74078-0425

Running Head: Broiler ascites, oxygen, Metaproterenol.

[1] Agricultural experiment station, Oklahoma State University, Stillwater, OK 74078
[2] To whom correspondance should be addressed

ABSTRACT 1. Two experiments were conducted to evaluate the effects of oxygen concentration, ambient temperature and the prophylactic use of metaproterenol on performance, ascites incidence, hematology and carcass composition of commercial broilers.

2. In experiment 1, both a low oxygen concentration (17.6 ml/100ml) and a low ambient temperature reduced ($P<.01$) rate of weight gain. Ascites incidence was greater ($P<.03$) for chicks maintained at the lower oxygen concentration indicating that oxygen deprivation plays a role in ascites induction. Heart weight was greater ($P<.01$) in chicks maintained at the cooler temperature. Right ventricular weight was increased ($P<.05$) in chicks at either the low oxygen concentration or the low temperature. White blood cell counts were elevated by the low oxygen concentration.

3. In the second study, low atmospheric oxygen again reduced ($P<.01$) rate of weight gain. However, addition of metaproterenol to drinking water reduced ($P<.05$) the depression in growth and incidence of ascites. Metaproterenol supplementation reduced both heart weight ($P<.02$) and right ventricle weight ($P<.01$). Fat accretion was less ($P<.01$) while ash content was greater ($P<.03$)at the lower oxygen concentration. Metaproterenol supplementation tended to increase ($P<.06$) ash content of dry matter. Results indicate that low atmospheric oxygen concentrations increase the

incidence of ascites and that the bronchodilator metaproterenol alleviates ascites.

INTRODUCTION

The ascites syndrome, which is characterized by fluid accumulation in the abdominal cavity, is a major health concern in the poultry industry around the world. Although precise estimates are not available, economic losses in the United States alone likely exceed one million dollars annually (Odum, 1993). Though the exact cause of ascites is not known; numerous environmental factors have been implicated including inadequate ventilation (leading to ammonia and carbon dioxide accumulation), high altitude (low oxygen concentration), and cold stress (Wideman, 1988; Julian et al. 1989; Scheele et al. 1991; Odum, 1993). These factors, either directly or indirectly, can induce the pulmonary hypertension (PH) syndrome leading to ascites.

Available literature on PH-induced ascites suggests that a low partial pressure of oxygen at the alveolar level causes constriction of the pulmonary artery. This in turn causes the right ventricle of the heart to pump harder, thereby forcing more blood to the lung via the pulmonary artery. The elevated blood flow increases pulmonary blood pressure and enhances the ventricular muscle size (Hernandez, 1984; Huchzermeyer

and De Ruyck, 1986; Wideman, 1988; Julian et al. 1989; Odum, 1993).

Wideman (1988) detected a strong correlation between low ambient temperatures and ascites incidence. Scheele et al. (1991) using two lines of broilers selected on the basis of feed conversion efficiency found that low ambient temperature caused metabolic dysfunction. Gleeson (1986) and Huctzermeyer (1989) reported that a cold-induced increase in metabolic rate increased the oxygen requirement and cardiac output resulting in pulmonary hypertension (HP) induced ascites. Olander (1967) and Burton et al. (1968) in their studies with broilers maintained at high altitude found that the right ventricle was enlarged and that the liver and lungs had microscopic damage. Similar findings were reported by Beker et al.(1995).

Pulmonary hypertension, though mediated by many factors, is exacerbated by constriction of the bronchioles. Metaproterenol is a beta adrenergic bronchodilator that lowers both pulmonary and systemic vascular resistance in man (Westling, 1979). If this compound is active in broilers, it might reduce PH and the mortality from ascites. The objectives of the studies reported herein were to examine the interrelationships between atmospheric oxygen concentration, ambient temperature, and supplementation of metaproterenol via drinking water on

chick performance, hematology, organ weights and ascites incidence.

MATERIALS AND METHODS

GENERAL:

Two experiments were conducted concurrently to examine effects of atmospheric oxygen concentration (17.6 vs 20.6 ml/100ml), ambient temperature (26.7 vs 32.2 C), and the prophylactic use of metaproterenol (0 vs 2 mg/kg water) administered via drinking water. Variables monitored included ascites incidence and chick performance. Commercial broiler chicks were used in both experiments. Chicks had free access to water and feed (Table 1). In all cases birds were housed in respiratory chambers (Wiernusz and Teeter, 1993). Our method to reduce O_2 content of ambient air has been described previously (Beker et al. 1995). Breathing air O_2 and CO_2 concentration were measured each 6 h during the experiment to assure that oxygen was maintained at the desired concentration. Both O_2 consumption and CO_2 production also were determined. In both experiments, mortality was monitored every 12 h. Dead chicks were removed, weighed and necropsied. Each experiment was terminated on day 11. Upon termination of the experiments, all remaining chicks were weighed; they were bled via cardiac puncture since these chicks were small and it proved difficult to collect blood

from wing veins. Chicks were sacrificed by cervical dislocation. At necropsy, each chick was examined for gross lesions of ascites, heart enlargement, pulmonary edema and abnormal accumulation of fluid in the abdominal cavity. The heart, lungs and liver were removed, and weighed; organ weights as a percentage of body weight were calculated. Ascites heart index (AHI) was determined by dividing the weight of the right ventricular wall by the weight of the total ventricle, AHI = 100RV/TV. Blood for hematological evaluation was collected and analyzed for leukocytes (WBC) and erythrocytes (RBC) by the hematocytometer method. The packed cell volume (PCV) was determined by microcentrifugation. Hemoglobin was measured as cyanomethemoglobin (J.T. Baker System 9000, Allentown, PA).

EXPERIMENT 1:

In the first experiment, 125 one day-old Commercial chicks were weighed, divided into 11 groups of 15 chicks each and allocated to respiratory chamber compartments at random. Two atmospheric oxygen concentrations (17.6% and 20.6%) and two ambient temperatures (26.7C and 32.2C) were used in this experiment with an unequal number of replications per treatment. The treatments employed and number of replications of each are indicated in Table 1. The

Oxygen and carbon dioxide differential concentrations between incoming and outgoing respiratory chamber gases were regressed against time, time^2 and time^3 to establish polynomial equations describing the data. The regressed values were multiplied by air flow to calculate O_2 use and CO_2 production as described previously (Wiernusz and Teeter, 1993; Belay and Teeter, 1993).

EXPERIMENT 2:

In the second experiment, 125 chicks (Commercial broilers) at one day of age were divided into 11 groups of 15 chicks each and allocated to the open circuit respiratory chambers at random. Two atmospheric oxygen concentrations (17.6 and 20.6 ml/100ml) were examined without or with metaproterenol (2mg/Kg water) added to drinking water. Temperature was maintained at 26.7 C throughout the experiment. Treatments and number of replications employed are indicated in Table 2. Representative birds were taken from the second experiment for carcass analysis; these were placed in polyethylene freezer bags, and frozen at -20 C for body composition analysis. Prior to analysis, each frozen carcass was weighed, placed in a loaf pan, covered with aluminum foil, and autoclaved at 115 C and 0.75 Atm. pressure overnight. After cooling to room temperature, autoclaved samples were homogenized as described by

McDonald (1993). Representative samples were analyzed for DM, N, fat and ash according to AOAC (1980) procedures.

STATISTICAL ANALYSES

Data for all response variables were subjected to analysis of variance using the General Linear Models procedure of SAS (SAS, 1985). When a significant F statistic was noted by ANOVA, treatment means were compared using least squares analysis of variance (Steel and Torrie, 1960).

RESULTS

EXPERIMENT 1:

Ambient temperature (26.7 vs 32.2 C) effects on broiler performance at the two atmospheric O_2 concentrations (17.6% vs 20.6%) examined are presented in Table 4. No interaction of temperature and O_2 concentration was noted for performance ($P>.05$). Both the low atmospheric O_2 (17.6%) and cold stress (26.7 C) reduced body weight gain ($P<.01$). The mortality rate was quite high for chicks in the low oxygen-cold stressed group (80%) suggesting that this particular combination was particularly severe. Ascites incidence was higher ($P<.03$) for chicks maintained at 17.6% O_2.

The lower temperature resulted in increased ($P<.01$) weight of the heart. Right ventricular weight

of the heart of chicks at both the low temperature and the low O_2 concentration was increased (P<.05) compared to those at 32.2 C and 20.6 % O_2 (Table 5). AHI was greater (P<.04) for chicks at the lower oxygen concentration. Red blood cell count (RBC), hemoglobin concentration (HGB), and hematocrit (HCT) were not significantly (P>.05) impacted by either oxygen concentration or ambient temperature (Table 6). However, WBC concentration was greater (P<.02) for chicks at the lower oxygen concentration (Table 6).

EXPERIMENT 2.

The effects of two oxygen concentrations and metaproterenol supplementation on performance are presented in Table 7. No interaction between O_2 and Metaproterenol supplemetation for performance was detected (P>.05). Final weights and weight gains were lower (P< 0.02) for chicks at the lower oxygen concentration. Metaproterenol supplementation reduced (P< .02) ascites incidence, completely prevented ascites in chicks at the higher oxygen concentration. The weights of the heart and right ventricle were decreased (P< .01) by metaproterenol supplementation (1.5 vs 1.8 and .31 vs .40, respectively) for birds at lower oxygen concentration (Table 8). White blood cell counts tended to be higher (P< .07)for chicks at the lower oxygen concentration. Red blood cell

concentration, hemoglobin and hematocrit were not significantly impacted by either oxygen concentration or metaproterenol supplementation.

The effects of metaproterenol supplementation at the two oxygen concentrations and their interaction on protein, fat and ash contents of birds are presented in Table 9. Decreasing the oxygen concentration from 20.6 to 17.6 % reduced final weight, final percentage of fat and grams of fat. Ash content was increased by the lower oxygen concentration or metaproterenol ($P< .05$). At 17.6 % oxygen, chicks that were supplemented with metaproterenol had higher ($P< .05$) protein deposition while no metaproterenol effect was noted at 20.6% atmospheric oxygen. Supplementation of drinking water with metaproterenol numerically increased oxygen use but this difference was not significant (Table 10).

DISCUSSION

Cool temperatures and high altitudes have been reported to increase the incidence of ascites and reduce performance of chicks (Huchzermeyer et al. 1988; Julian et al. 1989b; and Maxwell et al. 1986). In experiment 1, both low atmospheric oxygen and cold stress severely reduced growth rate; in addition, the low temperature reduced the gain:feed ratio of broilers. Cold stress has been reported to increase

the metabolic demand for oxygen (Gleeson, 1986; Huchzermeyer, 1989). A low oxygen concentration further aggravated the oxygen deficit resulting in poor gain and oxygen use. Maxwell et al.(1986) reported that ascitic birds had a reduced growth rate. The significantly higher ascites incidence in chicks maintained at low oxygen concentration suggests that oxygen availability increased ascites development and reduced growth rate of broiler chicks.

In the second experiment, the prophylactic use of metaproterenol (2 mg/kg) via drinking water reduced ($P<.02$) ascites incidence. Metaproterenol is a sympathomimetic drug that relaxes bronchial smooth muscles. It also reduces both pulmonary and systemic vascular resistance and increases cardiac output through increasing stroke volume (Nelson, 1986). Both of these factors would be expected to help prevent ascites and permit greater oxygen consumption. In this study metaproterenol tended to increase oxygen consumption (0.55 Vs 0.51 and 0.55 Vs 0.53 L/h/bird) for chicks at the two oxygen concentrations.

Stewart and Muir (1982) using chickens selected for low and high oxygen consumption observed that strains consuming less oxygen were leaner and had less fat in dry matter. In agreement with their observation, chicks at the lower oxygen concentration in the present study had less fat accretion and a lower

percentage fat in the carcass. Perhaps limiting oxygen availability altered nutrient metabolism which resulted in leaner birds or the leaner birds may simply reflect the reduced final weight.

In conclusion, low atmospheric oxygen concentration and low ambient temperatures reduced rate of growth of birds. In addition, a low oxygen concentration increased the incidence of ascites. Prophylactic use of metaproterenol reduced the incidence of ascites. The success of this bronchodilator should stimulate further investigation into therapeutic regimens to prevent ascites.

Table 1. Treatments employed in experiment 1.

Treatment	oxygen Conc.(%)	ambient temp.	Metaproterinol Supp.	Reps,no.
1	20.6	26.7	-	3
2	17.6	26.7	-	3
3	20.6	32.2	+	3
4	17.6	32.2	+	2

Table 2. Treatments employed in experiment 2.

Treatment	oxygen Conc.(%)	ambient temp.	Metaproterinol Supp.	Reps,no.
1	17.6	26.7	+	2
2	17.6	26.7	-	3
3	20.6	26.7	+	3
4	20.6	26.7	-	3

Table 3. Composition of diet used in the study

Ingredients	g/kg
Corn, ground	523.2
Soy meal (49%)	369.3
Fat (animal and vegetable)	64.6
Dicalcium phosphate	17.7
Limestone	11.9
Salt	4.5
Vitamin mix[A]	2.5
Methionine (99%)	2.2
Trace mineral mix[B]	1.0
Ethoxyquin	0.2
Energy	13.39 MJ ME/kg
Crude protein	225g/kg

[A]Mix supplied the following per kilogram of diet: Vit A, 5.25mg; cholecalciferol, 0.125mg; vitamin E, 0.025mg; vitamin B12, 0.03 mg; riboflavin, 15 mg; niacin, 75 mg; d-panthothenic acid, 25 mg; cholin, 705.5 mg; menadione, 5 mg; folic acid, 1.5 mg; pyridoxine, 6.25 mg; thiamine, 3.03 mg; d-biotin, 0.127 mg.

[B]Mix supplied the following per kilogram of diet: Manganese, 120 mg; zinc, 100 mg; copper, 10 mg; iodine, 2.5 mg; calcium, 135 mg; iron, 75 mg; selenium, 0.15 mg.

EXPERIMENT 1.

Table 4. Ambient temperature effects on chick performance at two atmospheric oxygen concentrations in Experiment 1.

	Ambient temperature (C)				ANOVA		
	26.7		32.2				
	O_2 concentration(%)		O_2 concentration(%)			P<	
Param	17.6	20.6	17.6	20.6	O_2	T	O_2*T
FWT (g)	42.2±10.2	89.9±10.2	84.2±10.2	109.1±12.5	0.0106	0.0062	0.5375
Gain (g)	27.3±6.2	53.9±5.0	48.3±5.0	73.1±6.2	0.0023	0.0006	0.9040
Feed (g)	64.9±17.3	86.2±14.2	48.9±14.2	84.2±17.3	0.2445	0.4587	0.9058
Eff.	0.52±0.2	0.55±0.2	0.85±0.3	0.88±0.2	0.9646	0.0963	0.9542
Asc. (%)	20.0±0.1	11.1±0.1	31.1±0.1	3.3±0.1	0.0258	0.6372	0.3878

Ox=oxygen, Param=parameter, FWT=final weight, gain=weight gain, Feed=feed consumption Eff=gain:feed, Asc=ascites.

Table 5. Ambient temperature effects on chick organ weight as a percentage of body weight and ascites heart index at two atmospheric oxygen concentrations in Experiment 1.

	Ambient temperature (C)				ANOVA		
	26.7		32.2				
	O_2 concentration(%)		O_2 concentration(%)			P<	
Param	17.6	20.6	17.6	20.6	O_2	T	O_2*T
Liver	5.7±0.005	6.5±0.004	5.3±0.004	5.7±0.008	0.46	0.31	0.88
Lung	.91±.0008	.94±.0008	1.06±.0007	.73±.0001	0.45	0.95	0.12
Heart	1.8±.001	1.8±.001	1.5±.001	1.4±.002	0.37	0.01	0.95
RV	.40±.0003	.39±.0003	.33±..0002	.25±.0005	0.035	0.02	0.29
AHI	30.4±.02	29.1±.023	35.3±.022	29.2±.041	0.035	0.17	0.68

Ox=oxygen, Param=parameter (% body weight), RV=right ventricle, T=temperature, AHI=Ascites heart index ((RV/total ventricle) x 100)).

Table 6. Ambient temperature effects on chick blood cells at two atmospheric oxygen concentrations in Experiment 1.

	Ambient temperature (C)				ANOVA		
	26.7		32.2			P<	
	O_2 concentration(%)		O_2 concentration(%)				
Param	17.6	20.6	17.6	20.6	O_2	T	O_2*T
RBC_{6} ($10^{6}mm^{-3}$)	1.84±.21	1.47± .21	1.94± .21	1.37± .41	0.22	0.88	0.77
HCT(%)	33.6±3.22	25.9±3.22	33.4± .22	22.0±6.45	0.09	0.63	0.89
HGB ($g100ml^{-1}$)	7.6±.69	5.85± .69	7.83± .69	5.3±1.39	0.09	0.87	0.87
WBC_{3} ($10^{3}mm^{-3}$)	11.9±1.55	5.20±1.55	9.7±1.55	3.8±3.11	0.01	0.22	0.96

RBC=red blood cell, HCT=packed cell volume, HGB=hemoglobin, WBC=white blood cell Param=parameter.

Table 7. Atmospheric oxygen concentration effects on chick performance with and without drinking water supplementation with metaproterinol (Experiment 2).

	O_2 concentration (%)				ANOVA P<		
	17.6		20.6				
Param	-M	+M	-M	+M	O_2	M	O_2*M
FWT(g)	64.3±5.15	80.4±10.2	90.8±10.2	109.1±12.5	0.01	0.01	0.54
Gain(g)	27.3±5.15	43.4±5.15	53.8±4.21	54.4±7.3	0.01	0.06	0.41
Feed(g)	64.9±35.1	116±35.1	86.2±28.7	61.8±49.7	0.76	0.99	0.49
Asc.(%)	20.0±0.05	2.2±0.05	11.1±0.05	0	0.33	0.02	0.57

O_2=oxygen, Param=parameter, FWT=final weight, gain=weight gain, Feed=feed consumption,-M=without metaproterinol, +M=with metaproterinol.

Table 8. Atmospheric oxygen concentration effects on chick organ weight with and without drinking water supplementation with metaproterinol (Experiment 2).

	O_2 concentration (%)				ANOVA P<		
	17.6		20.6				
Param	-M	+M	-M	+M	O_2	M	O_2*M
Liver	5.7±0.005	5.4±0.005	6.5±0.005	5.4±0.006	0.63	0.19	0.75
Lung	.91±.0008	.93±.0008	.94±.0008	.77±.0001	0.82	0.44	0.61
Heart	1.8±.001	1.5±.001	1.8±.001	1.5±.002	0.54	0.01	0.87
RV	.40±.0004	.31±.0004	.39±.0003	.27±.0005	0.08	0.01	0.48
AHI	30.4±.02	27.4±.02	29.1±.02	25.1±.03	0.24	0.23	0.66

O_2=oxygen, Param=parameter (% body weight), RV=right ventricle, M=metaproterenol, AHI=Ascites heart index ((RV/total ventricle) x 100)).

Table 9. Atmospheric oxygen concentration effects on chick blood cells with and without drinking water supplementation with metaproterinol (Experiment 2).

Param	O_2 concentration (%)				ANOVA P<		
	17.6		20.6				
	-M	+M	-M	+M	O_2	M	O_2*M
$RBC_{(10^6 mm^{-3})}$	1.84±0.21	1.64±0.30	1.47±0.21	1.89±0.42	0.64	0.35	0.33
HCT(%)	33.6±3.91	28.8±5.54	25.9±3.9	28.5±7.83	0.37	0.38	0.74
$HGB_{(g100ml^{-1})}$	7.6±0.78	6.6±1.10	5.9±0.78	7.0±1.56	0.38	0.41	0.53
$WBC_{3(10^3 mm^{-3})}$	11.9±1.74	6.1±2.46	5.2±1.74	5.9±3.49	0.07	0.19	0.38

RBC=red blood cell, HCT=packed cell volume, HGB=hemoglobin, WBC=white blood cell, Param=parameter, M=metaproterenol, O_2=oxygen.

Table 10. Atmospheric oxygen concentration effects on chick protein, fat and ash content with and without drinking water supplementation with metaproterinol (Experiment 2).

	O_2 concentration (%)				ANOVA P<		
	17.6		20.6				
Parameter	-M	+M	-M	+M	O_2	M	O_2*M
Bird, NO	6	6	6	6			
Fwt(g)	64.3±5.15	80.4±10.2	90.8±10.2	109.1±12.5	0.01	0.01	0.54
Protein(%)	15.49±0.01	16.77±.01	16.93±.01	15.75±.01	0.62	0.01	0.20
Protein(g)	9.96±0.57	13.48±.57	15.37±.57	17.18±.57	0.01	0.84	0.49
Fat(%)	25.04±.02	23.67±.02	27.82±.02	27.31±.02	0.01	0.27	0.61
Fat(g)	16.10±1.94	19.03±1.94	25.26±1.94	29.80±1.98	0.01	0.12	0.43
Ash(%)	8.22±.78	8.58±1.10	7.74± .78	8.15±1.56	0.03	0.05	0.91

M=metaproterenol, O_2=oxygen, FWT=final weight.

Table 11. Atmospheric oxygen concentration effects on oxygen consumption, carbon dioxide production, respiratory quotient and heat production of broiler chicks with and without drinking water supplementation with metaproterenol during day 10-11 (Experiment 2).

	O_2 concentration (%)				ANOVA P<		
	17.6		20.6				
Parameter	-M	+M	-M	+M	O_2	M	O_2*M
O_2 use (L/h/b)	0.51±.003	0.55±.004	0.53±.0002	0.55±.0002	0.76	0.30	0.77
Air use (L/h/b)	2.90	3.12	2.57	2.67	0.01	0.28	0.65
CO_2 made (L/h/b)	0.43±.0003	0.44±.0004	0.44±.0002	0.45±.0002	064	0.61	0.88
RQ	0.84±.03	0.80±.04	0.83±.02	0.82±.02	0.91	0.44	0.46
HP (Kj/h/Kg)	34.91±2.01	39.15±2.84	33.56±1.42	35.2±1.42	0.35	0.27	0.74

O_2=oxygen, Rq=respiratory quotient, M=metaproterenol, HP=heat production.

REFERENCES

Association of Official Analytical chemists (1980) Official Methods of Analysis of the Association of Analytical Chemists. 13th ed. *Association of Official Analytical Chemists*, Washington, DC.

Beker, A., Vanhooser, S.L. and Teeter, R.G.(1995). Effect of oxygen level on ascites incidence and performance in broiler chicks. *Avian Diseases* **39**:285-291.

Burton, R.R and Smith, A.H (1969). Induction of cardiac hypertrophy and polycythemia in the developing chick at high altitude. *Federation Proceedings*, **28**: 1170-1177.

Gleeson, M. (1986). Respiratory adjustments of the unanaesthetized chicken Gallus domesticus to evaluate metabolism elicited by 2,4-dinitrophenol or cold exposure. *Comparative Biochemistry and Physiology* **83a:** 283-289.

Hernandez, A., 1984. Hypoxic ascites in broilers. A review of several studies done in Colombia. Avian Dis. 31:658-661.

Huchzermeyer, F.W. and DE Ruyck, A.M.C. (1986). Pulmonary hypertension syndrome associated with ascites in broilers. *Veterinary Record* **119:** 94.

Huchzermeyer, F.W., DE Ruyck, A.M.C., and Van Ark, H. (1988). broiler pulmonary hypertension syndrome. III. Commercial broiler strains differ in their susceptibility. *Ondersteport Journal of Vetrinary Research* **55:** 5-9.

Huchzermeyer, F. W., VAN DER Colf, W. J. And Guinane, P. R., (1989) Broiler ascites: increased oxygen demand with cold may explain high winter incidence. *(SAPA) Poultry Bulletin,* September, 474 and 483.

Julian, R.J., McMillan, I. and Quinton, M. (1989b). The effect of cold and dietary energy on right ventricular hypertrophy, right ventricular failure and ascites in meat type chickens. *Avian Pathology* **18:** 675-684.

McDonald, K.R (1993). Broiler performance: An evaluation of metabolic considerations related to stress tolerance and tissue accretion methodology. *MSc. thesis*, Oklahoma State University.

Maxwell, M.H., Robertson, G.W. and Spence, S. (1986). Studies on ascitic syndrome in young broilers. 1. Hematology and pathology Avian. *Pathology* **15:** 511-524.

Odum, T. W. (1993). Ascites syndrome: overview and update. *Poultry Digest*, January pp 14-22.

Olander, H. j., Burton, R.R. and Adler, H.E. (1967). The pathophysiology of chronic hypoxia in chickens. *Avian Diseases*, **11:** 609-620.

SAS (1985). SAS users guide. *Statistical Analysis System Inst., Inc.,* Cary, NY

Scheele, C.W., DeWit, W., Frankenhuis, M.T and Vereijken, P.F.G (1991). Ascites in broilers. !. Experimental factors evoking symptoms related to ascites. *Poultry Science*, **70:** 1069 - 1083.

Steel, R. D. G., and Torrie, J. H.(1960). *Principles and Procedures of Statistics*. McGrew Hill Book Co., NY.

Stewart, P. A., and Muir, W. M.(1982) The effect of varying protein levels on carcass composition and nutrient utilization in two lines of chickens divergently selected for oxygen consumption. *Poultry Science* **61:** 1-11.

Westling, H. (1979) Circulating effects of ß2-receptor agonists in man. *Acta Pharmacol Toxicol*. **44** (Suppl. 11):36.

Wideman, R F Jr (1988) Ascites in poultry. *Nutrition update*, August, **Vol. 6:** #2

Wiernusz, C. J. and Teeter, R.G. 1993. Feeding effects on broiler thermobalance during thermoneutral and high ambient temperature exposure. *Poultry Sci.* **72**:1917-1924.

VITA

ALI BEKER YOUSUF

Candidate for the Degree of

Doctor of Philosophy

Thesis: BROILER ENERGY AND OXYGEN METABOLISM AND THE EFFECT OF OXYGEN CONCENTRATION AND AMBIENT TEMPERATURE ON ASCITES INCIDENCE

Major field: Animal Nutrition

Biographical:

Personal Data: Born in Harar, Ethiopia, April 8, 1956,the son of Beker Yousuf and Zebida Habib.

Education: Graduated from Medhanealem Comprehensive Secondary School, Harar in June, 1977; received Bachelor of Science degree in Animal Science and Master of Science degree in Animal Production from Addis Ababa University in 1981 and 1985, respectively; completed the requirements for the Doctor of Philosophy degree from Oklahoma State University, Stillwater, Oklahoma in July, 1996.

Professional Experience: Head, Foggera Cattle Research Station, Debrezeit Agricultural Research Center, 1981 - 1982. Participated in the International Poultry Husbandry and Animal Feed Technology training, Barnveld, the Netherlands in 1988. Worked as Lecturer and Researcher at Alemaya University of Agriculture, Department of Animal Science, 1983 to present.