COMPENSATORY AND ACCELERATED GROWTH IN BROILERS: EFFECTS OF FEED RESTRICTION LEVEL AND INITIATION TIME

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COMPENSATORY AND ACCELERATED GROWTH IN BROILERS: EFFECTS OF FEED RESTRICTION LEVEL AND INITIATION TIME

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TABLE OF CONTENTS

Chapter		Page
I	INTRODUCTION	1
	Industry Imposed Feed restrictions	4
	Organ development	5
	Composition of gain	6
	Justification	6
II	REVIEW OF LITERATURE	7
	Introduction	7
	What is Growth?	7
	Accelerated versus compensatory growth	8
	Methods of Feed Restriction	10
	Potential mechanisms responsible for Compensatory Growth Mechanisms	13
	Digestibility	19
	Factors Influencing Occurrence of Compensatory Growth in broilers	20
	Effects of Compensatory Growth on bird Performance	25
	Body (carcass) parts and internal organs	33
	Hematochemistries chemistries	35
	Application and Benefit to the Malawi Poultry Industry	42
III.	MATERIALS AND METHODS	44
IV.	RESULTS	54
	Compensatory growth hypothesis	54
	Body weight	58
	Feed intake and feed efficiency	65
	Feed intake and efficiency	66
	Organ weights	70
	Hematochemistry	88
	Basal metabolism	92

V Discussion..... 94

VI Summary and Conclusions..... 105
References..... 110

LIST OF TABLES

Table Page

1. Normal Blood serum values of selected blood constituents..... 38

2. Diet Composition of Basal Diet..... 48

3. Feed Allowances for birds from 7 to 49 days of age 49

4. Regression equations for predicting degree of compensatory gain as percent of initial weight (Y) of male broilers at a given initial weight..... 56

5. Regression equations for predicting weight of organs as percent of body weight (Y) of male broilers at a given weight (X)..... 57

6. Effect of varying restriction level and initiation time on live body weight (BW), BW gain, BW gain as percent of initial weight and compensatory gain of male broilers..... 59

7. Effect of varying restriction level and initiation time on feed consumption, feed efficiency and feed per initial weight of male.. 60

8. Effect of varying restriction level and initiation time on overall performance of male broilers from 7 to 49 days of age posthatch broilers..... 61

9. Effect of varying restriction level and initiation time on breast weight, breast gain, breast gain per initial weight and compensatory gain of male broilers..... 71

10. Effect of varying restriction level and initiation time on thigh weight, thigh gain, thigh gain per initial weight and compensatory gain of male broilers..... 72

11. Effect of varying restriction level and initiation time on lung weight, lung gain, lung gain per initial weight and compensatory gain of male broilers..... 73

12. Effect of varying restriction level and initiation time on viscera weight, viscera gain, viscera gain per initial weight and compensatory

gain of male broilers..... 74

13. Effect of varying restriction level and initiation time on heart weight, heart gain, heart gain per initial weight and compensatory gain of male broilers..... 75

14. Effect of varying restriction level and initiation time on pancreas weight, pancreas gain, pancreas gain per initial weight and compensatory gain of male broilers..... 76

15. Effect of varying restriction level and initiation time on large intestine weight, large intestine gain, large intestine gain per initial weight and compensatory gain of male roilers..... 77

16. Effect of varying restriction level and initiation time on cecum weight, cecum gain, cecum gain per initial weight and compensatory gain of male broilers..... 78

17. Effect of varying restriction level and initiation time on liver weight, liver gain, liver gain per initial weight and compensatory gain of male broilers..... 79

18. Effect of varying restriction level and initiation time on gizzard weight, gizzard gain, gizzard gain per initial weight and compensatory gain of male broilers..... 80

19. Effect of varying restriction level and initiation time on small intestine weight, small intestine gain, small intestine gain per initial weight and compensatory gain of male broilers..... 81

20. Effect of varying restriction level and initiation time on bursa of Fabricious weight, bursa of Fabricious gain, bursa of Fabricious gain per initial weight and compensatory gain of male broilers..... 82

21. Effect of varying restriction level and initiation time on spleen weight, spleen gain, spleen gain per initial weight and compensatory gain of male roilers..... 83

22. Effect of varying restriction level and initiation time on abdominal fat pad weight, abdominal fat pad gain, abdominal fat pad gain per initial weight and compensatory gain of male broilers..... 84

LIST OF FIGURES

Figure Page

1. Body weight gain as percent of initial body weight of ad libitum fed male broilers at the end of 3, 5 and 7 weeks of age..... 55

2. Body weight gains as percent of initial body weight of male broilers subjected to varying feed restriction levels from 1 to 3 weeks of age at the end of restriction (week 3)and refeeding periods (week 5 and week 7)..... 62

3. Body weight gains as percent of initial body weight of male broilers subjected to varying feed restriction levels from 3 to 5 weeks of age at the end of restriction (week 5)and refeeding periods (week 7)..... 63

4. Feed consumption per initial body weight of male broilers subjected to varying feed restriction levels from 1 to 3 weeks of age at the end of restriction (week 3)and refeeding periods (week 5 and week 7)..... 67

5. Feed consumption per of initial body weight of male broilers subjected to varying feed restriction levels from 3 to 5 weeks of age at the end of restriction (week 5)and refeeding periods (week 7)..... 68

6. Effect of feed restriction (85% of ad libitum feed consumption) from 1 to 3 weeks of age on breast, thigh plus leg and viscera gain as percent of initial body weights at the end of restriction (week 3) and refeeding periods (week 5 and week 7)..... 86

7. Effect of feed restriction (85% of ad libitum feed consumption) from 3 to 5 weeks of age on breast, thigh plus leg and viscera gain as percent of initial body weights at the end of restriction (week 3) and refeeding periods (week 5 and week 7)..... 87

CHAPTER I

INTRODUCTION

Compensatory growth has been reported in various studies since Osbourn and Mendel (1915, 1916) first observed that growth rate may accelerate following a period of feed restriction. Feed restriction may occur as a result of providing low protein (Lipstein et al, 1975; Moran, 1979; Hulan et al, 1980; Plavnik and Hurwitz, 1990), low caloric diets (Griffiths et al, 1977; Summers and Leeson, 1986; Leeson, 1990) and reduction of feed supplied to the birds (Pokniak and Cornejo, 1982; Pinchasov and Jensen, 1989). Indeed these practices are frequently observed in the poultry industry.

However, compensatory growth (CG) occurrence has not been widely accepted as a definable entity. Indeed (Washburn and Bondari, 1978; Fisher, 1984; Pinchasov and Jensen, 1989; Newcombe et al, 1992 and Robinson et al, 1992) failed to observe the compensatory gain effect and suggested that it does not occur. In most developing countries where humans and animals compete for food, periods of reduced feed restriction are the norm rather than the exception. Under such conditions, studies are needed to establish the existence and manipulation potential of compensatory growth.

Compensatory growth has traditionally been defined as a rapid growth rate relative to age and body weight of unsuppressed controls following a period of growth retardation (Read and White, 1977; Wilson and Osbourn, 1960). Winchester and Howe (1954) observed that retarded growth animals responded by gaining weight more rapidly per unit time when provided balanced rations than controls with only small differences in final body weight. Because many

types of stressors inhibit growth (feed restriction, heat stress, diseases, poor management), it does not automatically follow that compensatory gain will occur in every case; or that the expected final body weight will be attained.

Examination of compensatory growth literature suggests that opinions differ as to whether the accelerated growth is due to increased pure lean tissue (Jones and Farrell, 1992b), gut contents (Bondi, 1987), fat deposition (Maynard, 1947) or, possibly, a combination of these. Since accelerated growth inconsistently enables broilers to attain their anticipated body weight, Fontana et al (1992) proposed that the term compensatory growth is a misnomer. These researchers further proposed that compensatory growth be termed "accelerated growth" with or without compensatory growth occurring. As pointed out by Wilson and Osbourn (1960) and Yu and Robinson (1992), animal ability to recover from a period of undernutrition depends on the nature, severity and duration of nutrient restriction, pattern of refeeding and the animal's stage of development or time at which feed restriction is initiated. In the words of Palsson (1955), any region or part of a growing animal which has been retarded in development by restricted nutrition exhibits a great recuperative capacity if the animal is changed on to a high level of nutrition. Nonetheless, bird capacity to accelerate growth and limitations of such processes for occurrence of complete compensation need to be established.

It is generally accepted that feed restricted animals have lower body weights than ad libitum fed controls during the restriction period and in the early refeeding period. The lower body weights reduce maintenance requirements (Sheehy and Senior, 1942; Wilson and Osbourn, 1960, Graham and Searle, 1975, Allden, 1970) which when coupled with increased feed consumption during refeeding dramatically elevate nutrient supply beyond that needed for

maintenance. This elevation of available substrates would then be available to augment the growth process and improve energetic efficiency (Hahn, 1982). It has also been reported that late maturing tissues and regions of the body exhibit the most ability to recover from the ills of undernutrition (Wilson and Osbourn, 1960). As such nutrient consumption exceeding maintenance needs may impact tissues differently.

Selection for increased body weight gains by the poultry industry has resulted in the production of broilers marketed at younger ages (Gyles, 1989). Growth enhancement increases annually at approximately 40g of market weights at a fixed age (Mallard and Douaire, 1988). Unfortunately, increased broiler gains are accompanied by elevated fat deposition. Diminution of the hypothalamic satiety mechanism (Burkhart et al, 1983) has been hypothesized to account for the increased feed consumption which results in increased body weight gain and.fat deposition. Chambers et al (1981) reported that early broiler strains contained lower proportions of fat at slaughter than present day birds. The poultry industry must therefore, exert efforts to minimize fat deposition so that consumers are provided with the right product. Interestingly, unlike broiler meat consumers in the developed world who are cautious about their daily dietary caloric intake and fat content of food they eat, the trend is not as serious in the developing countries such as Malawi. With low supply of calories in their food, fat from meat is considered a desirable supplemental energy source.

Though some studies indicate that feed restricted birds attain lower final body weights than ad libitum fed birds (Washburn and Bondari, 1978; Fisher, 1984; Pinchasov and Jensen, 1989; Newcombe et al, 1992 and Robinson et al, 1992); other reports suggest that previously restricted birds do attain final weights similar to ad libitum controls (Plavnik and Hurwitz, 1985; Plavnik et al.,

1986; McMurtry et al, 1988; Plavnik and Hurwitz, 1990; Summers and Leeson, 1986) upon realimentation. It is the latter type of results that need further exploration.

INDUSTRY IMPOSED FEED RESTRICTION :

Feed restriction programs have been used to improve feed efficiency (Proudfoot, 1983; Pokniak et al, 1984; Plavnik and Hurwitz, 1985, 1988, 1990; Summers et al, 1990; Newcombe et al, 1992), reduction of ascites (Albers et al, 1990), reduction of sudden death syndrome (Duff et al, 1988; Mollison et al, 1984; Bowes, 1988; O'Sullivan et al, 1991), skeletal (leg abnormalities) and metabolic disease (Robinson et al, 1992; Classen and Ridell, 1989; Ferket and Sell, 1989);, Haye and Simons, 1978; Ridell, 1983; Ridell et al, 1983; Lilburn et al; 1989); reduction of heat stress mortality (Scheideler and Baughaman, 1993); , Francis et al, 1991; Teeter et al, 1987; McCormick et al, 1979) and reduction of percentage of abdominal fat (Mollison et al, 1984; Jones and Farrell, 1987; Plavnik and Hurwitz, 1989; Cabel and Waldroup, 1988, Leeson et al, 1992, Lilburn et al 1982).

Feed restriction programs have also been reported to mitigate growth rate induced muscle damage by reducing plasma enzymes (creatine kinase and aspartate transferase) activities which subsequently reduce muscle dysfunction, injury or muscle turnover (Hocking et al, 1993; Mitchell et al, 1992). Reports on improvements in economic returns offer another advantage of the feed restriction strategy in poultry (Sherma, 1986; Parrilla, 1984 and Proudfoot et al, 1983).

ORGAN DEVELOPMENT AND COMPOSITION OF GAIN

A differential organ response to feed restriction and refeeding would also have important implications on energetic and protein efficiency with which feed is utilized by birds undergoing compensatory growth processes. In appreciation of consumer demand for lean meat products and carcass parts other than whole broiler carcass, an assessment of compensatory feed programs on important broiler carcass parts such as breast, leg and thigh is necessary. This entails determination of relative or absolute contributions of water, lean and adipose tissue to weight gain. If compensatory weight gain observed in feed restricted-refed birds is, on the major part fat (as a compromise on overall economic yield), the final product would not be desirable. On the other hand, if compensatory weight gain has little or no negative influence on final carcass yield (particularly economic parts such as breast muscle, leg and thigh), then feed restriction programs could be advantageous. Availability of information on composition of gain, may therefore facilitate the selection an appropriate feed restriction strategy and an optimal time to slaughter birds which have had an interrupted growth path.

JUSTIFICATION

Only recently (Scheideler and Baughman, 1993 and Fontana et al, 1992), compensatory growth studies have emphasized the ability of previously restricted birds to attain similar weights to ad libitum fed controls upon refeeding (Washburn and Bondari, 1978; Plavnik and Hurwitz, 1985, Plavnik et al, 1986; McMurtry et al, 1988, Pinchasov and Jensen, 1989). Most compensatory growth

studies have not exhaustively explored occurrence of accelerated growth per se, impact of feed restriction on body organs, composition of gain, digestibility and basal metabolism. A study of differential growth of body organs would provide an insight into nutrient partitioning processes that take place during feed restriction and refeeding phases. If compensatory growth studies are strictly interpreted as bird's ability to attain final market weights as ad libitum fed controls; information related to rate of tissue growth or its composition is often disregarded.

In view of the paucity of scientific long term data and information regarding occurrence of compensatory growth, the main objective of the study reported herein was to investigate effects of feed restriction level (100%, 85%, 70% and 55% of ad libitum feed consumption) and initiation time (day 7 to 21 m-period 1 and day 21 to 35- period 2) on ability of male broilers to elicit accelerated and/or compensatory growth. Specifically, the objectives of the experiments were:

1. To assess effects of feed restriction and initiation time on body weight, body weight gain and feed efficiency of male broilers 7 to 49 days of age posthatch.

2. To evaluate the impact of feed restriction and initiation time on basal metabolism (heat production) of broilers.

3. To evaluate effects of feed restriction and initiation time on growth of selected organs (Pectoralis major and Pectoralis minor), thigh plus leg, proventriculus, liver, small intestine, pancreas, spleen, large intestine, bursa of Fabricious, abdominal fat pad, lung, cecum and gizzard) during restriction and refeeding periods.

4. To evaluate effects of feed restriction and initiation time on growth of selected blood constituents (glucose, total protein, albumin, creatine, uric acid, and triglycerides).

5. To evaluate the effect of varying feed restriction levels and initiation time on the ability of previously feed restricted broilers to elicit accelerated and compensatory growth during the refeeding period.

6. To assess the potential application and manipulation of accelerated and compensatory growth programs in the broiler industry.

CHAPTER II

REVIEW OF LITERATURE

INTRODUCTION

WHAT IS GROWTH?: Definition of growth with special attention to body composition may be critical. Maynard (1947) drew a distinction between what he termed 'true growth' and extra fat deposition. He stated that true growth is characterized by an increase in protein mass, minerals, water and fat. However, Pomeroy (1955) argued that this distinction is difficult to substantiate in practice as fatty tissues perform other needed functions other than just act as an energy source. Subcutaneous tissue, for instance, has been implicated in thermoregulation of many animals (Blaxter and Rook, 1953).

Early work on growth and development (Hammond, 1932, Palsson and Verges, 1952) led to the theory that growth occurred in waves and that early maturing parts and tissues had priority over late maturing parts. According to

this theory, feed restriction would affect various tissues differently during feed restriction and refeeding. Little and Sandland (1975) observed that fat was the tissue most affected during weight loss. However, others (Burton et al, 1974; Drew and Reid 1975; Butler-Hogg, 1984; Drouillard et al, 1991) have demonstrated that loss of weight consists of protein, fat and water. These researchers further demonstrated that the extent of loss or depletion is dependent on severity and duration of feed restriction, maturity of the animal, and diet composition.

ACCELERATED VERSUS COMPENSATORY GROWTH: Although not all restricted-refed birds do attain final body weights similar to ad libitum fed controls, accelerated growth during the refeeding period is a commonplace occurrence. It is therefore important to delienate these two terms and how they apply in feed restriction programs. Definitions of compensatory growth generally address animals that have been subjected to a period of undernutrition and thus have retarded growth, with a period of accelerated growth during subsequent realimentation. Controversy regarding the definition centers on whether the birds must catch up to be considered as exhibiting compensatory growth.

The term compensatory growth has been used to describe the accelerated or "catch-up growth" following a period of reduced feed consumption which can be mediated by management stress.

Bohman (1955) defined compensatory growth as rapid growth relative to age. Ragsdale (1934) reported that when put on a low plane of nutrition, physiological aging proceeds at a slower rate and upon realimentation, such animals tend to grow at a rate appropriate to its physiological age rather than to its chronological age. Washburn and Bondari (1978) defined compensatory

growth `*as a growth velocity above that of ad libitum fed controls*' while Yu et al (1990) defined compensatory growth as `*the rate of growth exceeding that normally observed in the same breed of chicken at the same age*'. These definitions suggest that accelerated growth occurs upon refeeding without necessarily referring to attainment of market weights similar to ad libitum fed controls. In concordance with the foregoing suggestion, Fontana et al (1992) proposed that the term "accelerated growth" better describes the increase in growth rate upon refeeding because not all previously restricted birds exhibit complete compensatory growth during the refeeding period.

Most references on compensatory growth imply that compensatory growth occurs only if previously restricted birds catch-up with ad libitum fed controls. Washburn and Bondari (1978) restricted broilers in feed intake for 7 days and showed reduced weight at market age. In this regard, no compensatory growth was reported to occur. Moran (1979) reported that broilers restricted in protein intake at an early age could compensate for weight gain and feed ratio by market age suggesting that compensatory growth occurred. Other reports (Pokniak and Cornejo, 1982; Pokniak et al, 1984; Plavnik and Hurwitz, 1985, 1989; Leeson, 1990) also suggest that *compensatory growth refers to the bird's ability to catch-up with body weights of ad libitum fed controls at market age*. Yu and Robinson (1992) suggested that the term `catch-up growth' is more precise in describing the growth observed upon refeeding following a feed restriction period than the term compensatory growth.

The foregoing discussions suggest that the term compensatory growth is ill defined and controversial. A common definition and description of the increase in growth observed in previously restricted birds during the refeeding period is required to accelerate research progress. For purposes of this writing,

compensatory growth is defined as *`the birds ability or extent to which they catch-up with ad libitum fed controls while accelerated growth refers to a more rapid growth observed following a period of feed deprivation irrespective of final body weights'.*

METHODS OF FEED RESTRICTION

Two methods of feed restriction are commonly employed in accelerated and compensatory growth studies. Though the result may be the same for both methods, little attention has been paid to carry over effects during the realimentation period.

QUALITATIVE FEED RESTRICTION : Qualitative feed restriction involves providing animals or birds with feed low in quality such as low protein (Moran, 1979) or low caloric density (Griffiths et al, 1977).

REDUCTION OF PROTEIN CONTENT: Moran (1979) observed that broilers restricted in protein intake at an early age were able to compensate for weight gain and feed:gain ratio by market age although this was accompanied by fat gain. Plavnik and Hurwitz (1990) fed a 9.4% crude protein from day 8 to day 14 post-hatch and reported a 57% reduction in feed consumption. This latter approach reduced growth rate by 41 % during the restriction period with no signs of body weight recovery at 8 weeks of age. Low protein starter diets have also been used to decrease early growth and minimise leg problems in broilers grown to heavy roaster weights (Hulan et al, 1980). Recently, Skinner et al (1991) reported that low amino acid levels (80 and 90% of those recommended by Thomas et al, 1986) increased bone ash.

However, it is important to exercise some caution when lowering dietary protein quality to avoid adverse effects on bird performance. Lipstein et al (1975) reduced dietary crude protein from 20.5% to 17.5% and observed a substantial increase in fat deposition with no diferences in live body weight from 5 to 9 weeks of age This observation has been reported by other workers (Fancher and Jensen 1989).

REDUCTION OF ENERGY CONTENT: Reduction of dietary energy content has been achieved by using the dilution technique. Griffiths et al (1977) diluted a broiler diet from 3087 kcal ME/kg to 2233 Kcal ME/kg of feed with oatmeal. These researchers reported that birds fed a low energy diet (2233 kcal ME/kg) exhibited compensatory growth when switched to a control starter diet (3087 kcal ME/kg) during the fourth week. The compensatory growth observed was exhibited as an increase in weight gain suggesting accelerated growth rate during the period when birds were placed on a higher energy diet. No differences were observed in four week body weights when the low energy diet was fed from 0 to 3 weeks of age. Leeson (1990) diluted a broiler starter diet from 22% CP and 3050 Kcal ME/kg to 10% CP and 1370 Kcal ME/kg respectively and reported that bird weights were significantly reduced at day 11 for the restricted birds. However, the body weights between restricted birds and ad libitum fed controls were not significantly different at 42 days of age indicating that compensatory growth had occurred. Recently, Zubair and Leeson (1994) reported occurrence of complete compensation at 35 days of age by birds previously subjected to a starter diet diluted with 50% oat hulls. In summary, qualitative feed restriction, when appropriately manipulated, can be used to initiate optimum accelerated and compensatory growth.

QUANTITATIVE FEED RESTRICTION : Quantitative feed restriction, as the name suggests, involves allotting less feed than recommended. Quantitative feed restriction can be cumbersome as it entails frequent feed weighing and provision of adequate feeder space to ensure similar allotment of feed among birds. If not carefully implemented, disproportionate quantities of feed will be consumed within a group and result in uneven body weight distributions and general bird performance.

Pokniak and Cornejo (1982) restricted broilers to 85%, 70% and 55% of ad libitum feed intake and noted that recovery was greatest with mild feed restriction (85% of ad libitum feed consumption). Washburn and Bondari (1978) reported that restricting birds to approximately 85% of ad libitum feed consumption for one, two and three week durations resulted in reduced final body weights at 8 weks of age. McCartney and Brown (1976) reported that significant increases (accelerated growth) in body weights were observed in birds with limited access to feed (15 minutes every 2 hours).

USE OF CHEMICALS: Anorectic substances or chemicals have also been employed to elicit feed restriction. Pinchasov and Jensen (1989) added 1.5% and 3% glycolic acid into broiler feed from 7 to 14 days of age. These researchers reported a 22% and 50% feed reduction by addition of glycolic acid at a rate of 1.5% and 3% of the diet respectively vis-a-vis ad libitum fed controls. Pharmacological dosage of tryptophan, a precursor of serotonin which inhibits feed consumption (Blundell, 1977; Lacy et al, 1986) has been reported. Leathwood (1987) reported to have a sedative effect through its effect on brain serotonin. McCormick and Denbow (1987) reported that naloxone and neltrexone (opioid antagonists) inhibited feed consumption of broiler chickens when administered at 2.5 to 10 mg/kg body weight. Use of anorectic chemical

compounds may offer a potential avenue for reducing feed intake in feed restriction programs.

POTENTIAL MECHANISMS INFLUENCING OCCURRENCE OF ACCELERATED AND COMPENSATORY GROWTH

GROWTH CURVES AND PATTERNS: Under ideal conditions, animals and birds follow a predetermined growth pattern or curve. Any stressful condition (undernutrition, disease, unfavorable environment) will therefore cause birds to deviate from their original growth path and/or pattern. Pasternak and Shalev (1983) suggested that broilers following a concave-shaped growth curve require less feed than those exhibiting a convex-shaped growth curve. A concave-shaped growth curve suggests initial slow growth followed by faster growth later in the growth phase while a convex-shaped growth curve suggests a fast growth followed by slow growth later. Analyzing data of Marks (1979), Yu and Robinson (1992) reported that commercial broilers have a convex-shaped growth curve. These reseachers also reported that feed restriction in the second week post-hatch may produce a concave-shape type of growth curve in broiler chickens which can result in better efficiency and a leaner bird without compromizing final body weight. These propositions suggest that animals or birds stressed in the earlier stages of life would exhibit a concave-shaped which is indicative of occurrence of compensatory growth upon refeeding.

LOW MAINTENANCE REQUIREMENTS: Maintenance is the state of equilibrium where an animal or bird is experiencing no fat and protein gain or loss. Under such circumstances, nutrients are only available to maintain homeostatic body

processes. Reduced maintenance requirements as a direct consequence of lower body weight and possibly reduced activity (Sheehy and Senior, 1942) has been reported in cattle. Low maintenance requirements demand less nutrient use which results in a greater fraction of net energy available for productive purposes such as accelerated growth rate (Wilson and Osbourn, 1960; Graham and Searle, 1975; Allden, 1970).

Horst et al, (1934) observed low maintenance energy expenditure in previously feed restricted rats and suggested that animals subjected to undernutrition exhibit low maintenance energy expenditure per unit body mass due to reduced activity. This suggestion assumes that reduced activity is a component of maintenance. Rosebrough et al (1986), following his studies with chickens, suggested that decreased maintenance energy expenditure is carried over from feed restriction into the refeeding phase which is consequently translated into an improvement in the efficiency of feed utilization. These scientists further suggested that restriction-refeeding systems allow the chick to consume adequate amounts of energy to maintain body weight.

Thompson et al (1982) and Schnyder et al (1982).suggested that basal metabolic rate is lower than normal during the compensatory growth period due to lower maintenance requirements. Wilson and Osbourn (1960) reported that animals subjected to feed restriction and refed later raise their basal metabolic rate per unit body weight slowly to normal to accommodate the new level of nutrition leaving more food for productive processes, especially increased growth rate. An increase in oxidative phosphorylation in liver mitochondria indicating increased enzyme activity upon refeeding after feed restriction has also been reported (Kartashov et al , 1985). In a study with hens, Voloshchenko et al (1985) observed that feed restriction resulted in a decrease in energetic

reactions but concomitantly increased the effectiveness of phosphorylation in muscle mitochondria.

In a study with sheep, Kabbali et al (1992) observed reduction in weights of visceral organs (liver, heart, kidneys and digestive tract) in the early stages of feed restriction. Similar effects of reduction of internal organ weights in sheep have also been reported by Koong et al (1985) and Marais et al (1991). It was then concluded that reduction of weights of visceral organs, particularly the metabolically active ones, results in lower maintenance costs per unit body weight; thereby improving the animal's ability to survive on limited amounts of feed. However, retardation of age-associated physiological changes in intestinal and pancreatic processes have been implicated in reduced growth rates associated with malnutrition during the early growing period (Gutierrez et al, 1991).

Koong et al (1985) observed that compensatory growth observed in sheep liver and gastro intestinal tract (GIT) was due to an increase in feed consumption, feed efficiency and consequently increased body weight gain. An increase in the growth rate of the liver and GIT was therefore suggested as a potential underlying prerequisite for the attainment of compensatory growth during the refeeding period to accommodate accelerated growth rate. Similar observations has been reported in rats (Anugwa and Pond, 1989),and swine (Pond et all, 1988). Anugwa and Pond (1989) observed that restricted rats had higher relative weights of the stomach compared to controls after a two week feed restriction insult. It was then concluded that the minimal reduction in GIT weight during feed restriction (Anugwa and Pond, 1989) and the increased weight upon realimentation was indicative of an adaptive protection of the GIT from extremities of nutrient deficits through a repartitioning of nutrients. This in

agreement with Lepkovsky and Furuta (1971) who observed that visceral organs (crop and intestine) increased in weight to meet the metabolic load imposed by the abnormally large amounts of food that were forced into the gastrointestinal tract. It was however noted that the crop and intestinal weights returned to normal with the restitution of ad libitum feeding. A relative preservation of visceral organs and tissues at the expense of muscle and adipose tissue has also been reported (Jackson, 1990).

The aforegoing discusion attest to the flexibility of internal organs and tissues to partition nutrients in varying proportions to tissues during extremes of feed consumption. How these changes impact maintenance needs and partitioning of nutrients during refeeding require further exploration so that broiler production may be maximized.

ENERGETIC EFFICIENCY OF PROTEIN AND FAT GAIN: Birds are normally fed ad libitum on balanced diets to allow them to achieve their maximum potential for lean tissue or protein deposition. However, this also entails deposition of fat which is an energetically inefficient carcass component and an undesirable product by the consumer. Maximum deposition of lean tissue is therefore necessary and of paramount importance in broiler production. In this case, an assessment of the energy cost of fat and protein deposition defined as an increment of food energy required to promote a defined increment in body fat or protein (Pullar and Webster, 1977) is desirable.

Protein in the body is associated with six times its own weight of water (Wilson and Osbourne, 1960) resulting in low calorific value per unit body weight. Consequently, protein accretion is relatively more efficient than fat accretion (Wilson and Osbourne, 1960). On the other hand, Mallard and

Douaire (1988) reported that protein deposition is more efficient than fat deposition because a gram of protein is accompanied by three times as much water. With regard to protein synthesis, McDonald et al (1988) reported that energetic efficiency for protein synthesis is more efficient (.88) than the energetic efficiency for fat synthesis (.81). These calculations were based on the ratio of energy retained by an amino acid with a molecular weight of 100g (2437 kJ) to the total energy expended (2777 kJ) for protein. For fat synthesis, the energetic efficiency was based on the ratio of energy stored (32037 kJ) to energy expended (39344 kJ in 1 molecule of tripalmitin. Being an energetically dense tissue, calories required for deposition of lean tissue are much less per unit increase in weight than required for fat deposition.

From the foregoing discussion, it can therefore be concluded that a net synthesis of lipid or fat in adipose tissue is a drain on dietary nutrients, particularly glucose and fatty acids (Forbes, 1988) which would in turn be compromised by an increase in feed intake. However, it is important to realize that deposition of fat and protein is a function of both synthesis and breakdown. Proteins have a continuous turnover which means that the calorimetric efficiency of protein deposition stated at .55 for non-rumunants and .40 for ruminants (McDonald et al, 1988) is much lower than the theoretical efficiency of protein synthesis. McDonald et al (1988) also reported that fat synthesis is much slower than protein synthesis. It was therefore concluded that calorimetric efficiency of fat deposition is relatively close to the theoretical efficiency of fat synthesis with values of 0.7 to .75 commonly quoted for all species (McDonald et al, 1988).

However, it should be borne in mind that energy cost of fat deposition can be precisely measured in adult animals since energy retention as protein is small and the amount of metabolizable energy required to maintain energy balance

does not differ much between successive measurements made of metabolic heat production at different levels of ME intake (Pullar and Webster, 1977). For protein deposition, assessment of energy cost is more difficult because of low energy deposition as protein during rapid growth relative to that deposited as fat or dissipated as heat (Pullar and Webster, 1977). In a study with rats, Pullar and Webster, concluded that values for energy costs of protein (2.25 kJ) and fat (1.36 kJ) deposition per kJ of protein and fat respectively are arbitrary in nature since they do not describe the total costs of synthesis, but simply relates depostion to increments of ME.

INCREASED FEED CONSUMPTION UPON REFEEDING: Availability of substrates for growth and physiological processes is vital for occurrence of compensatory growth. An increase in feed intake relative to body size has also been observed in birds during the realimentation period. Plavnik and Hurwitz (1990) observed that previously restricted broilers consumed more feed than controls during the feed restriction period and exhibited catch-up growth. In humans, this increase in feed intake maybe associated with an increase in appetite which drops off upon attainment of body weight for a given height (Ashworth and Milliward, 1986).

In discussing homeostatic mechanisms, Brody (1945) stated that appetite is closely associated with maintaining a constant body weight. It therefore seems feasible that appetite should be one of the mechanisms responsible for ensuring that normal weight is attained or eventually achieved in restricted animals once the restraint is removed. Bondi (1987) suggested that an increase in space of the abdominal cavity may be responsible for the increase in feed intake of restricted-refed animals.

IMPROVED FEED EFFICIENCY: Improvement in feed efficiency following feed restriction has been attributed to improved metabolic efficiency associated with maintaining a smaller body during early growth (Dickerson, 1978). Changes in intermediary metabolism have been shown to occur in underfed animals which elicit a reduction in basal metabolic rate. Subsequently, there is an increase in the animals' net food efficiency (Bondi, 1987). Hypertrophy of the gastrointestinal tract (GIT) and an increase in digesta/gut fill greater than in animals not previously underfed has been reported (Bondi, 1987). This increases apparent efficiency of refed animals to gain body weight as measured by gain tofeed ratio.

DIGESTIBILITY

Digestibility of feed for broilers can be influenced by a number of factors such as genotype (Jorgensen et al, 1990, Leenstra and Pit, 1988, Soorensen et al, 1983), sex, age and method of determination (Doeschate et al, 1993). However, the influence of age on digestibility has been controversial. Age has been reported to have no influence (Sorensen et al, 1983; McNab and Shannon, 1972), increase (Wallis and Balnave, 1984) or decrease (Haakansson et al, 1978) metabolizability or digestibility of organic matter. Doeschate et al (1993) reported that apparent metabolizability increases only during the last part of the growth phase.

Contrary to findings of Doeschate et al (1993) that female broilers showed higher (3%) digestibility coefficients than males, sex has been reported to have no influence on digestibility (Leenstra and Pit, 1988; Wallis and Balnave, 1983;

Sorensen et al, 1983). The higher digestibility reported by Doeschate et al, (1993) was also related to a higher feed conversion ratio and the where the efficiency with which digested feed was converted to feed was in female than male birds. Since availability of an adequate amounts of dietary substrates is one of the prerequisites for occurrence of accelerated growth, studies on effects of feed restriction on digestibility of feed are required.

FACTORS INFLUENCING OCCURENCE OF ACCELERATED AND COMPENSATORY GROWTH IN BROILERS

Inconsistent compensatory growth results have been reported by various feed restriction programs. These inconsistencies could be attributed to many influencing factors such as breed, sex, age, stage of development and time at which feed restriction is imposed, degree of feed restriction (length and amount-severity, age at time of rehabilitation or refeeding, season and environment (Yu and Robinson, 1992). These factors, acting singly or in combination ultimately affect the growth rate, body composition and general performance of birds/animals in restricted-refed type experiments. An understanding of factors that influence performance of birds under feed restriction programs is therefore a prerequisite towards a thorough comprehension of the compensatory growth phenomena and the ability to benefit from its manipulation.

STRAIN OF THE CHICKEN: Not all studies use similar strains or breeds of chickens. It is therefore probable that divergent results reported in compensatory growth literature could be attributed to genetic makeup. Cherry et al (1978) observed that fast growing broilers exhibit little catch-up growth in contrast to

slower growing broilers. Contrary to these observations, Plavnik et al (1986) observed complete that catch-up growth was elicited by Ross male broilers which is a fast growing. Considerable compensatory growth accompanied by an improvement in efficiency of feed utilization has also been reported in fast growing broilers (Beane et al (1979). On the other hand, Jones and Farrell (1992a) reported that genotype (low intermediate or high fat strains of broilers) had no influence on the bird's ability to demonstrate compensatory growth. Since male broilers grow faster and are generally heavier than female broilers, use of unsexed birds (Jones and Farrell, 1992b) may have contributed to the differences and confounded final results.

SEX OF THE BIRD: It is well established that growth rates of male are higher than female broilers under a similar environments (Fisher, 1984). It therefore follows that the responses of male and female broilers to various management programs may be different (McMurtry et al, 1988). Plavnik and Hurwitz (1985; 1990) reported that male broilers, unlike female broilers, exhibited compensatory growth after a period of feed restriction. On the other hand, Jones and Farrell (1992b) reported complete body weight recovery with unsexed broilers. Therefore, studies using unsexed broilers may produce varying results.

DEGREE AND EXTENT OF FEED RESTRICTION: The degree to which an animal can recover from feed restriction is dependent upon the duration and extent of feed restriction. Increasing the extent or severity of restriction decreases the bird's ability to recover (Wilson and Osbourn, 1960). Feeding below maintenance level is detrimental to the bird's well being and production of runts may occur (Barnes and Miller, 1981). As recommended by Plavnik and

Hurwitz (1985), feed restriction programs should at least meet maintenance requirements in order to optimize compensatory growth.

Plavnik and Hurwitz (1985), Plavnik et al (1986) and McMurtry et al (1988) provided birds with 1.5 x $BW^{2/3}$ Kcal ME/day (35-40 Kcal ME per bird per day) to meet maintenance requirements by providing about 35% of ad libitum feed consumption. Plavnik and Hurwitz (1985) and Yu et al, (1990) reported that this level of restriction provided more nutrients than required to the birds' maintenance and hence expected compensatory growth to occur. However, no significant body weight recovery or accelerated growth was observed in birds subjected to a similar level of feed restriction (Calvert et al, 1987; Pinchasov and Jensen, 1989, Robinson et al, 1992). Less severe feed restriction levels have been reported to adversely impact body weight recovery (Deaton et al, 1973; Washburn and Bondari, 1978 and Beane et al, 1979). Although failure to exhibit compensatory growth that ultimately leads to full body weight (BW) recovery suggests that the 1.5 x $BW^{2/3}$ kcal ME/day level of restriction did not provide maintenance requirements during feed restriction, other factors such as length of feed restriction and strain of birds used may have contributed to the divergent results discussed above.

The 1.5 X $BW^{2/3}$ Kcal ME feed restriction was meant to provide approximately 35% of ad libitum feed intake (Plavnik and Hurwitz, 1985). However, Jones and Farrell (1989) reported that this feeding level provided about 50% of the birds ad libitum intake using Australian broilers. Jones and Farrell (1989) reported that 3.1 kJ/g$BW^{2/3}$ per day restriction provided approximately 20% of ad libitum feed intake and was enough to meet the requirements of the bird for body weight stasis during the restriction period.

Differences in genetic makeup of birds used in the experiments may play a role in determination of what feeding levels provide maintenance requirements.

AGE AT INITIATION AND LENGTH OF FEED RESTRICTION: In addition to the degree of feed restriction, the duration of feed restriction and age at which feed restriction is initiated play a significant role in the occurrence of compensatory growth. An interaction between length (period) of feed restriction and severity (Wilson and Osbourn, 1960) could therefore be critically important to ensure significant body weight recovery of previously restricted broilers during the refeeding phase. McMurtry et al (1988) suggested that male broilers should be feed restricted for no more than seven days and female broilers for no more than five days. Jones and Farrell (1989) reported that short restrictions of less than four days allows the bird to fully recover after feed restriction. Feed restriction through dietary dilution with cereal hulls from 4 to 11 days of age has also been reported to allow body weight recovery at six weeks of age (Leeson, 1990). Comparing 6 and 12 day restriction periods, Plavnik and Hurwitz (1986) noted that the 12 day restriction period had more detrimental effects on eight week body weight than the 6 day restriction period.

Plavnik and Hurwitz (1988) recommended initiation of feed restriction at 3 and 5 days for male and female broilers respectively so that no compromise is made for lost weight. Yu et al (1990) reported that restricting the growth of broilers for one week produced a growth curve similar to controls upon refeeding although final body weights were dissimilar. Jones and Farrell (1992b) interpreted failure of Yu et al (1990) birds' to elicit compensatory growth to be influenced by length of feed restriction. Other researchers have suggested that fast growth tends to decline late in the growth phase. Arafa et al (1983) applied

feed restrictions during the last three weeks of life (5-8 weeks) and no compensatory growth was observed. It was therefore concluded that late restrictions leaves the bird with inadequate time to fully recover from the effects of restricted feeding.

These studies suggest that prolongation of the growing period would be required for compensatory growth to occur. Robinson et al (1992) observed that prolongation of refeeding time from 7 to 9 weeks of age resulted in no significant differences in body weight between restricted and ad libitum fed birds. Under such circumstances, an assessment as to whether the prolonged time may overshadow any financial returns or savings is warranted.

FEED INTAKE DURING THE REFEEDING PHASE: The actual amount of feed consumed by birds upon refeeding has a significant impact on the occurrence of compensatory growth. No complete recovery of previously feed restricted birds should be anticipated unless adequate amount of feed equal to or slightly above that of ad libitum fed birds is provided during the refeeding phase. Lower feed consumption by seven-day restricted birds than ad libitum fed controls has been implicated in the reduced growth rate observed in restricted birds (Yu et al, 1990). Increased feed consumption (64g more feed than the controls) during the refeeding period following a 77% feed restriction level (of ad libitum fed birds) has been shown to produce full body weight recovery (Plavnik and Hurwitz, 1990). The ability to consume feed and an elevation in nutrient supply could therefore be a limiting factor in occurrence of compensatory growth

EFFECTS OF COMPENSATORY FEED RESTRICTION PROGRAMS ON BIRD PERFORMANCE

When growth is not interrupted, body weight and other body parameters such as lean mass, metabolic rate and body fat increase from the time of conception, or shortly after, along curves that proceed from a sigmoid fashion to an asymptotic value at maturity (Webster, 1980). As such, body weight and other parameters of birds subjected to feed restriction may be different from those of ad libitum controls at maturity unless compensatory gain occured.

FINAL BODY WEIGHT: As discussed above, effects of feed restriction on bird performance have produced inconsistent results. Using feed restriction levels to provide maintenance requirements (1.5 x $BW^{2/3}$ kcal ME/day), Plavnik and Hurwitz (1985) Plavnik et al, 1986 and McMurtry et al, (1988) reported that birds exhibited compensatory growth when birds were provided with ad libitum access to feed. Moran (1979) observed that broilers restricted in protein intake at an early age were able to compensate for body weight and feed efficiency at market age. However, he reported that much of the compensatory gain was fat deposition which was higher than that of ad libitum fed controls. Broilers restricted in nutrient intake from 8 to 23 days was also reported to show full body weight recovery at 8 weeks of age (Pokniak and Cornejo, 1982; Pokniak et al, 1984).

However, others were unable to obtain full body weight recovery at slaughter (Washburn and Bondari, 1978; Mollison et al (1984); Pinchasov and Jensen, 1989, Robinson et al, 1992, Jones and Farrell, 1992a, 1992b, Deaton et al, 1973, Beane et al, 1979, Yu et al, 1990, Newcombe et al, 1992). Arafa et al

(1983) reported that energy restriction in the last ten days of finishing had little or no influence on dressing percentage or cooked carcass weight. Teeter and Smith (1985) observed that a 25% reduction in feed intake of broilers from 28 to 39 days of age resulted in a 30% weight gain reduction. A more severe (50%) feed restriction has been observed to result in a 50% weight gain reduction in broilers subjected fro a 49 day period (Washburn, 1990). It therefore seems plausible to conclude that the an interaction of factors mentioned above may be responsible for the controversial results of feed restriction programs as reported in literature. For instance, failure to obtain compensatory growth by Pinchasov and Jensen (1989) versus occurrence of compensatory growth by Plavnik and Hurwitz (1985) may be related to the length of refeeding phase and type of birds used. Plavnik and Hurwitz (1985) terminated their experiment at 8 weeks of age while Pinchasov and Jensen (1989) terminated theirs at 7 weeks of age. Birds in the former experiment had, therefore, more time for full compensation of lost weight to occur.

FEED CONSUMPTION: Nutrient availability governs bird ability to recover body weight following a period of undernutrition. A severe feed restriction followed by refeeding with a diet low in nutritional value will likely result in poor ration consumption and extend the period required for recovery if it occurs. As discussed earlier, an increase in nutrient availability is a requisite for occurrence of compensatory growth. Several factors have been reported to impact increased in feed consumption during refeeding.

GASTRO INTESTINAL TRACT: Pokniak and Cornejo (1982) reported that liver and digestive tract (absolute weights) were greater for the control than for the restricted birds at 23 days but the differences were overcome at 56 days. It was

also noted that feed intake for the restricted birds was less than that of the control. It was therefore suggested that the increase in feed intake of the restricted birds was accomodated by an increase in growth of the digestive tract. Reduced capacity of the digestive tract in sheep has been implicated in lower feed intake (Ledin, 1983).

RATION COMPOSITION: Dilution of feed as a qualitative method of feed restriction has been reported to impact feed intake. Leeson (1990) reported that dilution of diet by rice hulls resulted in a higher feed consumption by birds, possibly in a bid to maintain nutrient requirement. Although a reduction in body weight was reported at 11 days, the final body weights and overall feed efficiency were similar between ad libitum fed controls and feed restricted broilers at 42 days of age. An increase in feed intake of diets diluted with alpha floc has been reported (Summers et al, 1990). These observations are in agreement with the proposition by Wilson and Osbourn (1960) that increased appetite following refeeding may be responsible for any improvement in growth and feed efficiency associated with compensatory growth.

Despite an increase in feed consumption observed in these studies, savings in feed have been reported in feed restricted birds. Sanz et al (1985) observed that feed restriction of heavy broilers to 72.2% of ad libitum feed intake saved up to 3.6 kg of feed per bird for the whole production cycle. With a big flock, and where compensatory growth occurs, such savings would have a significant impact on economic returns.

CARCASS FAT CONTENT AND ABDOMINAL FAT PAD SIZE: Many factors such as breed or genotype of birds (Holsheimer and Veerkamp, 1992), form of feed (Leclercq, 1986), housing type (Deaton et al (1973), age (Kubena et al,

1974), sex (Deyhim et al, 1992), and protein to energy ratio (Holsheimer and Veerkamp, 1992), have been reported to affect birds' carcass composition; particularly body fat. Use of nutritional manipulation techniques such as feed restriction has been identified as one of the avenues through which a reduction in fat content of broilers can be attained (Plavnik and Hurwitz, 1985, 1990; Plavnik et al, 1986; McMurtry et al, 1988)

Apart from providing the consumer with a desired product, lean carcasses also reduce processing costs. Losses due to excess fat deposition in broiler chickens have been estimated at $250-300 million annually (Rosebrough et al, 1986). Reduction of fat deposition in broilers may also reduce cleaning costs and pollution problems associated with waste water disposal in factories (Fisher, 1984). Additionally, fat gain is energetically inefficient (Fisher, 1984) and birds with higher skin fat content have reportedly fewer pin feathers and more blemishes on the carcass (Quarles et al, 1968), hence reducing carcass quality.

Moran (1979), reported that a greater portion of compensatory gain observed in broilers subjected to protein restriction imposed at an early age was composed of fat. However, similar carcass fat content was reported for birds subjected to nutrient intake restriction and those on ad libitum feed consumption (Pokniak and Cornejo, 1982; Pokniak et al, 1984). Restriction of energy intake from 0 to 3 weeks was reported to have no significant effect on abdominal fat pad weight when measured at 8 weeks of age (Griffiths et al, 1977). On the other hand, providing birds with 35% and 50% of ad libitum feed intake (Jones and Farrell, 1987) and energy reduction in the finisher phase (Arafa et al, 1983) has been reported to reduce abdominal fat pad of broilers. Reduction in abdominal fat pad weight have also been reported to occur through quantitative dietary restriction (Mollison et al ,1984) at a young age. A 50% (12g) reduction

in abdominal fat pad weight with a 4% (100g) reduction in 17 week body weight versus ad libitum fed controls have been observed elsewhere (Lilburn et al,1982).

However, other scientists have reported contradictory results. Wilson and Osbourn (1960) indicated that birds had a 6 -24 % increase in fat upon refeeding after a period of feed restriction. Beane et al (1979) restricted broiler feed intake to 85% of ad libitum fed birds from 14 to 42 days of age and observed that abdominal fat pad weights at 56 days of age were heavier than those of ad libitum fed controls as recently reported (Newcombe et al, 1992).

PROTEIN AND FAT ACCRETION: The effect of energy balance and protein deposition on the birds ability to elicit compensatory growth has been discussed. Jones and Farrell (1992b) reported that the success of feed restriction program to allow compensatory growth is triggered by negative energy balance observed in broilers during feed restriction when birds mobilize stored energy while, protein accretion continues. This leads to a loss of body fat accompanied by decreased adipocyte size. These researchers further reported that adipocyte hyperplasia commences upon realimentation marking the on set of compensation for body fat gain. Fisher (1984) suggested that restriction during early growth might reduce fat cell hyperplasia, thereby limiting the potential for adipose tissue accretion.

Sustenance of protein deposition during the feed restriction phase has been implicated for the rapid compensatory growth (Jones and Farrell, 1992b). This suggests that adequate protein deposition during the restriction phase is necessary if compensatory growth is to occur in previously restricted birds. If

true, then the degree of restriction should markedly impact the birds' ability to compensate for reduced growth.

ADIPOSE TISSUE CELLULARITY: Growth of adipose tissue is basically due to hypertrophy or hyperplasia. Compared to other species, few studies on the effect of feed restriction on cellularity of avian adipose tissue and dynamics of adipocyte growth have been reported in literature. Fisher (1984) suggested that feed restriction might facilitate manipulation of carcass composition by reducing adipocyte hyperplasia in early growth, thereby limiting the potential for growth of fat later in growth. Jones and Farrell (1992b) suggested that feed restriction programs reduce body fat by causing a delay in adipocyte hyperplasia. These researchers reported that mean adipocyte diameter and volume tended to decrease with application of feed restriction. They further reported that hyperplasia continues during feed restriction but adipocytes remain smaller.

Diet dilution employed as a form of feed restriction at an early age has been reported to reduce adipocyte hyperplasia (Cherry et al, 1984). Ballam and March (1979) reported that underfeeding birds (layers) from 0 to 14 weeks of age increased adipocyte number, but reduced adipocyte weight at 14 and 42 weeks posthatch. Hood (1982), proposed that both hypertrophy and hyperplasia occur up to 14 weeks of age in abdominal fat pad of broiler type chickens after which only hypertrophic adipose tissue accretion occurs. This was consistent with the observation that DNA-deoxyribose content of adipose tissue in pullets reached a plateau at 12 to 15 weeks of age (Pfaff and Austic, 1976). The observations of Ballam and March (1979), based on layers, may not have a direct application in broilers which are normally marketed at 7 weeks.

It has generally been observed that nutrient restriction does not permanently reduce adipocyte cellularity (Pfaff and Austic, 1976; March and Hansen, 1977), suggesting that dietary influences on abdominal fat deposition would be expected to be of short duration. This then, consequently, allows a stimulation of a hyperphagic rebound of adipocytes which may increase abdominal fat deposition, a fact making restriction-refeeding time intervals critical. Higher fat accretive rates and consequently fatter carcasses have been reported in steers (Coleman et al, 1993) and pigs (McMeekan, 1940; Mersmann, 1987) reared on a compensatory gain progams probably due to availability of excess caloric intakes beyond that needed for maintenance and maximal protein accretion (Just, 1984). In consonance with the above observations, a preponderance of adipose tissue over lean tissue has been reported in humans (Jackson, 1990).

LIPOGENESIS: An increase in lipogenesis upon refeeding has been implicated to be responsible for the increase in fat deposition. Rosebrough et al (1986) observed an eighty fold increase in lipogenic activity in broilers two days into the refeeding phase. It has been postulated that enzyme activities are related to corresponding changes in in-vitro lipogenesis. The lipogenic-enzyme capacity of chick liver has been reported to be responsible for influencing the degree of fatness in broilers since O'Hea and Leveille (1969b) have shown that 90 to 95 % of total fatty acid synthesis in chicks occurs in the liver. Shapira et al (1978) noted that hepatic lipogenesis is increased in birds which are overfed. Whether enzymes per se regulate lipogenesis or merely reflect changes in metabolic flux through lipogenic pathways (Calabota et al, 1983) is yet to be fully elucidated.

Because strains differ in their ability to deposit fat, it is pertinent to consider the genetic makeup of the birds in assessing the effects of feed

restriction on carcass fat. Different genetic stocks of chickens (Littlefield, 1972; Farr et al, 1977; Griffiths et al, 1978) have been reported to differ in fat accretion rates. Shapira (1978) reported that White Rock chickens, a heavy breed of chicken, had higher lipogenic enzyme activities and consequently deposited more fat than light breed cross birds (Newhampshire and Leghorn).

MEASUREMENT OF BODY COMPOSITION

In a bid to assess the composition of birds as they grow (at different ages), the comparative slaughter technique (alternatively referred to as serial slaughter technique) is normally employed (Davidson and Mathieson, 1964, McDonald et al, 1988). This technique can be relatively accurate and demands no elaborate apparatus though it is laborious (McDonald et al, 1988) and time consuming. The carcass composition of the birds is determined by slaughtering a representative sample of the initial bird or animal population at the beginning and end of the experiment. A relationship is then obtained between liveweight of the birds or animals and their carcass composition. This is then used to predict the initial carcass composition of the test group. Opinions differ on accuracy of the comparative slaughter technique. Davidson and Mathieson (1964) suggested that the composition of birds killed at the beginning of a trial is probably representative of the remainder when very young birds are used. On the other hand, Fraps and Carlyle (1939) and Hanlen (1939) reported variability exists within older birds especially due to their varying fat content. Utmost care should therefore be exercised to ensure that samples are thoroughly homogenized so that only true representative samples of the carcass are used.

BODY (CARCASS) PARTS AND INTERNAL ORGANS

BODY (CARCASS) PARTS: There is currently an increase in retailing of specific body parts to consumers. Chicken sold in parts such as breast, leg, thigh, leg plus thigh, giblets, neck and wings provides the consumer with a wide range of choices. Katanbaf et al (1989) reported that a shift from marketing whole broilers to cut-up and further processed products is responsible for the renewed interest in growth patterns of individual organs and muscle masses of chickens. Parrilla (1984) reported that restricting meat-type ducks to 90 or 95 % of ad libitum consumption resulted in no significant difference in percentages of thigh, drumstick, wing and neck. However, the proportion of breast meat in fully fed birds was greater than in restricted birds. Hester et al (1990) also observed no differences in weights of drumsticks and wings when turkeys were provided with 74 % of the amino acid requirement from 6-12 weeks. Breast muscle weights were lighter for the restricted turkeys vis~a~vis ad libitum fed turkeys at 20 weeks of age. This was in contrast to thigh weights which were higher than those of full-fed turkeys. It was concluded that turkeys fed low compensatory growth diet partition a greater proportion of weight gains on thigh muscle rather than breast muscle. This is unfortunate in that the breast, the most highly prized carcass portion, is adversely affected by feed restriction. Whether a similar trend is/can be observed in broilers remains to be further explored. However, Katanbaf et al (1989) reported that relative weights of breast, legs, heart and liver were not were not affected by feeding restriction.

Visceral organs (intestinal tract, lung ,spleen, pancreas and kidneys) of Cobb x Cobb broilers consist about 9.56 % and total giblets (heart, liver, gizzard, and neck) consist 9.80% of total body mass of a Cobb x Cobb broiler weighing

1.86 kg (Anonymous, 1987). Katanbaf et al (1989) reported that restricted feeding increased relative weights and lengths of segments of the gastrointestinal tract and pancreas. Anugwa and Pond (1989) reported that accelerated growth of liver and gastrointestinal tract of rats undergoing rehabilitation after a period of feed restriction was associated with greater feed intake and higher efficiency of feed utilization. This supports the concept of enhanced energetic efficiency during realimentation (Wilson and Osbourn, 1960).

A question that arises is whether visceral organs modulate body and organ growth during periods of feed restriction and upon realimentation or not. During feed restriction, Plavnik and Hurwitz (1983) observed proportionate reductions in size of the small intestine, lung, heart, spleen and kidney to body weight. It was then speculated that the size of the intestine and other organs change to accommodate growth rate as modified by feed restriction. On the other hand, Anugwa and Pond (1989) reported that an increase in rate of growth for the liver and gastrointestinal tract is a possible underlying requisite for compensatory growth of the whole body during realimentation folowing feed restriction. These observations are in accord with those of Crompton and Walters (1979) and Moran (1979). Pokniak and Cornejo (1982) restricted birds to 15, 30 and 45 % of control intake from 8 to 23 days of age and observed that liver and digestive tract weights were significantly lower than those for ad libitum fed birds at 23 days of age. However, differences in these organ weights, like final body weigjhts and carcass fat, between restricted and control birds were eliminated following refeeding at 56 days of age. These observations were ascribed to compensatory growth.

HEMATOCHEMISTRIES

Hematochemistry is an increasingly useful aid in zootechnical and veterinary research It has been used to assess pathological and metabolic alterations of certain blood constituents (Melluzi et al, 1992). Level of blood metabolites is influenced by age (Meluzzi et al, 1992; Strurkie, 1976; Ross et al, 1978); sex (Lewandowski, 1986); rearing management (Cerolini et al, 1986), genetic makeup (Lewandowski et al, 1986); macro and micro-environment (Meluzzi et al, 1992); physiological status, pathological factors (Melluzzi et al, 1992) and feeding regime (Meluzzi et al, 1992; Lewandowski et al, 1986). Since final values are influenced by many factors, a definition of a range of values or limits to serve as reference values for each blood constituent is recommended (International Federation of Clinical Chemistry, 1978). Values for selected serum blood chemical components of normal ad libitum fed male chicken are presented in Table 1.

HANDLING OF BLOOD SAMPLES AND ACCURACY OF RESULTS: Methods of sampling and obtaining blood samples and method of analysis (Davidson, 1979; Dorner, 1981; Lewandowski, 1986; Melluzi et al, 1992) are critical in hematochemistries. Improper handling of blood will definitely cause numerous analytical inaccuracies, particularly hemolysis. Hemolysis interferes with calorimetric, enzymatic and chemical-reaction based processes used by laboratories to obtain chemical profiles (Lewandowski et al, 1986). Hemolysis has been reported to increase values obtained for lactate dehydrogenase, aspartate aminotransferase and potassium (Tietz, 1976). Hemolysis can be prevented by using clean dry equipment; by drawing the blood gently through

the needle; by inverting the tube to mix blood rather than shaking; by applying a slight and gentle rather than rough and excessive rimming of the clot; by avoiding overcentrifugation and refrigerating only after clot has occurred (Lewandowski et al, 1986).

SERUM PROTEIN: Allison (1955) pointed out that total protein and albumin values are good indicators of protein reserves in the animal. Normal values vary with the type of bird being tested, but falls within 3 to 5g/dl for Lewandowski et al (1986) or from 5.2 to 6.9 g/dl for.Mitruka (1981). Levels of serum total protein is influenced by many factors. Hypoproteinemia has been associated with parasitism, chronic hepatic and renal diseases, stress and starvation (Bush and Smith, 1980; Dolensek and Otis, 1973) while hyperproteinemia may indicate dehydration, infection (Dolensek and Otis, 1973, Lewandowski et al, 1986). Dietary protein depletion manifests as hypoproteinemia and hypoalbuminemia in chickens (Leveille and Sauberlich, 1961). Leveille and Sauberlich (1961) reported that increasing dietary protein beyond that required for growth resulted in an increase in total serum protein. Age has been reported to influence serum total protein values. Increased total protein values have been observed with increasing age (Melluzzi et al, 1992, Sturkie, 1976; Ross et al, 1978; Brandt et al, 1951). Morgan and Glick (1972) reported that total serum protein increased two fold from 2.68 gm % at 1 week of age to 4.43 gm % at 12 weeks of age. Anabolic hormones (testosterone, growth hormones) have been reported to effect an increase in total plasma proteins due to their anabolic effects (Kaneko, 1989b) while catabolic ones(thyroxine, cortisol) decrease total plasma protein (Strurkie, 1951). An increase total protein concentration has been associated with hot environments (Meluzzi et al, 1992).

Table 1. Normal blood serum values of selected blood constituents

Constituent	Mean Values[1]	Mean value[2]	Range of
Creatinine (mg/dl)	-	1.38	0.90-1.85
Uric acid (mg/dl)	3.9	5.28	2.47-8.08
Glucose (mg/dl)	148	162	152-182
Sodium (mEq/l)	149	153	148-163
Pottasium (mEq/l)	5.3	5.06	4.60-6.50
Calcium (mg/dl)	8.2	2.58	1.3-3.80
Total Protein (g/dl)	-	6.10	5.20-6.90
Albumin (g/dl)	-	2.81	2.10-3.45

[1]Teeter and Belay (1993)

[2]Mirtuka (1981)

SERUM ALBUMIN: Albumin is the largest (Galvin , 1980) and most prominent individual protein fraction in avian serum. It is synthesized in the liver and catabolized by all metabolically active tissues and varies with species (Kaneko, 1989b). A drop in plasma protein in diseased birds is due to decreased albumin levels (Galvin, 1980). Albumin is a reservoir of proteins and transport of amino acids (Grimminger and Scanes, 1986). It is the most osmotically active (75% of plasma osmotic activity) plasma protein because of its abundance and small size (Kaneko, 1989b). Since serum albumin is reported to bind and transport anions, cations, fatty acids, amino acids and thyroid hormones, hypoalbuminemia would definitely affect blood concentrations of all these albumin-transported compounds (Lewandowski, 1986). Meluzzi et al (1992) reported that albumin concentrations increased in winter with a significant interaction between season and age. Prealbumin, which does not occur in all domestic animals, has been reported to occur in birds for binding of thyroxine and transport (Kaneko, 1989b).

SERUM GLUCOSE: Blood glucose concentration is dependent on a number of factors and the concentration at any time is the net result of an equilibrium between the rates of entry and removal of glucose in the circulation (Kaneko, 1989a). Glucose in the blood may come from dietary glucose, hepatic production from fructose and galactose, amino acids (gluconeogenesis) and from glycogen. Dietary carbohydrates have been reported to enter the blood stream within 15 minutes of feeding (Hill, 1971). Removal of glucose is governed by utilization rate by tissues as an energy source or conversion to other products such as glycogen, pentoses and lipids (Kaneko, 1989a). Blood glucose concentration itself partially governs the rate of glucose utilization and therefore, in a sense, autoregulatory where the liver occupies a central position.

Belo et al (1976) observed that plasma glucose levels and glucose turnover rates were constant during fasting. Brady et al (1978) observed that plasma glucose in the chicken remained stable after fasting. Using tracer studies, they observed that tricarbon units originally derived from glucose are reincorporated into glucose molecule and concluded that little glucose sparing adaptation exists during short term starvation in chickens. Belo et al (1976) suggested that rate of glucose utilization in the chicken is rapid and that substantial recycling of glucose carbon occurs in the fasted chicken. Hazelwood (1986) reported that short term starvation causes immediate mobilization of hepatic carbohydrate reserves to liberate free glucose to the plasma to support metabolic needs of certain tissues. He also reported that short term starvation (1 to 8 days) does not decrease glucose utilization in chickens per unit body weight as compared to mammals. He then concluded that greatest energy loss during starvation is due to fat depletion and to some extent protein mobilization.

On the other hand, Chamblee and Morgan (1982) reported that serum glucose levels were lowered within three hours in chickens deprived of feed. Langslow et al (1970) reported that compensatory gluconeogenesis, protein and fatty acid catabolism increase greatly during periods of starvation. In contrast to glucose values in Table 1 (152 -182 mg/dl), different ranges, 200 - 500 mg/dl (Lewandowski et al, 1986) and 180 -250 mg/dl (Hazelwood, 1986) have been reported. A twofold increase in glucose levels in serum is common in stressed birds (Lewandowski et al, 1986). Hypoglycemia may be caused by starvation, malnutrition (hypovitaminosis) and disease (Chandra et al, 1983, Galvin, 1980). Prolonged contact of glucose with the clot has been implicate in serum glucose loss at a rate of 5% per hour. During starvation, small birds become hypoglycemic within 24 hours. However, chickens are more resistant to fasting

hypoglycemia than other animals such as rats (Houpt, 1958). Hyperglycemia has been reported to occur immediately after feeding (Kumar and Gupta, 1981; Simon and Rosselin, 1979) and during hyperthermia (Kumar and Gupta, 1981). Blood glucose values have been reported to vary with age, time of the day and state of captivity (Lewandowski et al, 1986). In conclusion, maintenance of normal glucose levels with low diets necessitate that enhanced gluconeogenesis and enzymes associated with amino acid catabolism and glucose could be of critically important.

URIC ACID: Uric acid is the primary catabolic product of protein, nonprotein nitrogen and purines in birds (Lewandowski et al, 1986) excreted by the kidney by tubular excretion. Elevated serum uric acid (above 2-15 mg/dl) range values have been associated with renal disease, starvation, age and captivity (Lewandowski et al, 1986). This range is, however, different from that reported in Table 1 above. Contradictory effects of fasting on uric acid levels have been reported. Evans and Scholz (1971) reported that plasma uric acid levels during adaptation to protein carbohydrate feed.

SERUM CALCIUM SODIUM AND POTASSIUM: Lewandowski et al (1986) reported that calcium values range from 8 to 12 mg/dl while Mirtuka (1981) reported that normal values from avaraged from other literature range from 9.0 to 23.7 mg/dl (Table 1). Elevated calcium values have been associated with excesses of Vitamin D_3 (Rosskopf et al, 1984, Tietz, 1976). Renal disease have also been reported to cause hypocalcenemia due to hypoalbuminemia or due to reduced calcium reabsorption (Lewandowski et al, 1986). When different studies were compared, Lewandowski al (1986) reported that normal serum sodium and

potassium values range from 130 to 170 mEq/l and 2.5 to 6.0 mEq/l respectively. On the other hand values in Table 1 above have ranges of 148 to 163 mEq/l and 4.6 to 6.50 mEq/l respectively as normal. The body's state of water deprivation have been reported to affect sodium and potassium levels. Chamblee and Morgan (1982) observed that serum sodium level was higher in birds deprived of water within one and half hours while serum potassium and phosphorus were unchanged. However, serum sodium levels returned to normal within one and half hours upon refeeding. On the contrary, Siegel (1968) reported that increased water consumption resulted in an increase in sodium levels and attributed this as an attempt by the bird to remove excess serum sodium. Hemolysis or failure to remove serum from the clot increases serum potassium and decreases sodium hence invalidating the determinations.

TRIGLYCERIDES: Many factors influence serum triglycerides levels. Circulating blood lipids are derived from intestinal absorption, synthesis or mobilization from carcass fat (Grimminger, 1986). Increased feed intake has been reported to result in increased blood lipids and adipose tissue. With adequate nutrients available in the body, circulating triglycerides are spared which results in increased fat deposition (Grimminger, 1986). Melluzi et al (1992) reported that Hybro birds had higher triglycerides levels than Arbor acres suggesting influence of breed and genetics of serum triglycerides. These researchers further reported that males had higher triglyceride levels than females. Male chickens selected for increased fatness had greater concentrations of triglycerides after an overnight (16 hr) fasting than those selected for leanness ((Hermier et al, 1984). The increase in triglyceride levels was noted two hours

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following refeeding. In conclusion, serum constituents levels are influenced by many factors one of which is feed restriction.

APPLICATION AND BENEFIT TO THE MALAWI POULTRY INDUSTRY

In most of the developing countries such as Malawi, protein and energy feeds are scarce (Safalaoh, 1992) due to lower crop production yields caused by a myriad of factors including vagaries of weather, pests and diseases. Scarcity of feed precipitates animal feed prices that are economically prohibitive for the average poultry producer. Competition between humans and animals (monogastric animals in particular) for feedstuffs (grains and protein sources) will likely become keener in the future. In an attempt to prevent exacerbation of the already frail poultry feed situation, more efficient use of available feedstuffs and nutrients in critical supply poses a significant challenge to poultry nutritionists. Feed restriction programs and associated advantages could potentially alleviate the burden of feed shortages faced by the poultry industry. Under such circumstances, a program which utilizes low quality feeds (as a feed restriction strategy) at the appropriate time on the growth curve, followed by standard diets without adversely compromising final body weights offers an avenue to enhance poultry production

Since protein feed ingredients are in shortest supply and costly components of chicken diets in Malawi and most of the developing countries, use of protein restriction programs could be advantageous. As reported by Barnes and Miller (1979), dietary protein restriction at an early age improves feed efficiency upon realimentation with a standard diet. This improved efficiency has remarkable financial implications. Use of low protein diets in the

early stages of growth will be less costly than standard commercial diets since starter diet is the most expensive broiler diet hence increase financial returns on investment. Using an optimizing technique referred to as the `reduced- gradient method', Talpaz et al (1988) observed that an 8 to 10 % savings in production expenses above the best free feeding dynamic plan was possible in feed restricted broilers while simultaneously keeping total production unchanged. However, conceivably so, market input and output prices of ingredients would affect this optimizing technique.

It is encouraging to note that there is currently a surge of interest in exploring use of unconventional feedstuffs in broiler production. Haustein et al, (1992) reported that broilers fed an unconventional chicken feed (Lemna gibba - duckweed) for the first three weeks of life followed by standard diets exhibited compensatory growth. D'mello et al (1985), Ologhobo et al (1993) have also investigated use of jackbean (Canavalia ensiformis) and Ambadi (Hibiscus cannabinus) meal in broiler diets respectively. Further research in use of such feed stuffs may consequently result in identification of low quality feeds which can be potentially be used in conjunction with standard diets in poultry production. Use of low quality diets would be used as a form of feed restriction which would finally result in compensatory growth of birds upon refeeding with standard diets. In the long run, reduction in production costs and freeing of other feedstuffs for human consumption may occur. With no other potential option left for more effective use and management of low quality and expensive feed concentrates, the success of compensatory growth feeding programs may therefore be pivotal in the advancement of the poultry industry in countries where standard poultry diets are an exception other than the norm.

CHAPTER III

MATERIALS AND METHODS

The Cobb X Cobb male broilers used in this study were fed a corn-soybean based ration. One ration, consisting of a mash diet containing 22.3% CP and 3252 Kcal ME/Kg. (Table 2) was used throughout the experiment.

Water was provided for ad libitum consumption. Male broilers were raised on rice hull liter from 0 to 7 days following normal brooding procedures. At seven days of age, eighty birds were randomly selected for similar body weight and wing banded. The birds were then transferred to individual 47 x 26 cm wire floored cages housed in an environmentally controlled room where supplemental heat was provided as recommended under normal brooding procedures.(Anonymous, 1987). On day 28, the temperature was then maintained at 24±1^{o}C until 49 days of age when the experiment was terminated. Lighting was provided continuously.

On day 7, birds were divided into two groups and randomly assigned to four feeding levels (100% (ad libitum); 85%, 70% and 55% of ad libitum feed consumption) as shown in Table 3. Group one (Treatments I, II, III, and IV) was restricted from 7 to 21 days while group two (Treatments V, VI, VII and VIII) was restricted from 21 to 35 days of age (Table 3). Upon compeletion of the first experiment, the pens were cleaned and disinfected before commencement of a replicate experiment one week later. Because treatment 5 was similar to treatment 1 through out the course of the experiment, this treament (5) was analysed as treatment 1 (see statistical analysis below).

Table 2. Diet composition of basal diet

Ingredient	Percent
Ground Corn	62.14
Soybean Meal	30.95
Tallow	2.90
Dicalcium phosphate	2.00
Limestone	1.00
Salt	0.40
Vitamin premix[2]	0.25
Trace mineral mix[3]	0.10
DL-Methionine	0.25
Monensin	0.01
Total	100.00
Calculated analysis	
ME	3252 kcal/kg
CP	22.30 %

[2]Mix supplied per kilogram of diet : Vitamin A, 14109 IU; Cholecalciferol, 5291 ICU; Vitamin B12, 0.014 mg; Riboflavin, 8.82 mg; Niacin, 26.5 mg; D-pantothenic acid, 28.2 mg; Choline, 705.5 mg; menadione, 1.16 mg; folic acid, 1.176 mg; Pyridoxine, 3.52 mg; Thiamine, 3.52 mg; D-biotin, 176 mg.
[3]Mix supplied per kilogram of diet : Ca, 160 mg; Zn, 100 mg; Mn, 120 mg; Cu, 10 mg; I, 2.5 mg

Table 3. Feed allowances for birds restricted from 7 to 21 days of age

Treatment	Day 7 to 21	Day 21 to 35	Day 35 to 49
I	100	100	100
II	85	100	100
III	70	100	100
IV	55	100	100
V	100	100	100
VI	100	85	100
VII	100	70	100
VIII	100	55	100

FEED INTAKE.

Mean feed intake for ad libitum fed birds (TRT I) was taken daily as a basis for amounts of feed to be provided for birds restricted from day 7 to 21 and birds restricted from day 21 to 35. The previous day's mean feed intake for ad libitum fed birds (AL birds) was used to calculate the amount of feed for the 85%, 70% and 55% feed restriction levels. For the restricted birds, half the amount of feed was given to the birds in the morning and the other half in the afternoon to alleviate possibilities of meal feeding effects. Total weekly feed consumption was then calculated by adding daily feed intakes together.

BIRD WEIGHTS.

Individual bird weight measurements were taken weekly. To avoid confounding bird weights with gut contents, birds were fasted for a minimum of 8 hours before weighing the next day. Three birds from each treament were then randomly selected for slaughter to determine organ weights at different ages (day 21, 35 and 49).

BASAL METABOLISM - HEAT PRODUCTION (HP)

At the end of each restriction and refeeding period, three birds per treatment were randomly selected for determination of HP. Measurements for group one birds (Trt I, II, III, and IV) were taken on days 21, 35 and 49 while those for group two birds were taken on days 35 and 49. Preparation of birds for measurement of gas and heat production measurements were done using

procedures of Wiernuz and Teeter (1993) as follows: birds were administered with a Ketaset Ketamine HCL, USP (Fort Dodge Laboratories, Inc., Fort Dodge, IA, 50501, USA) intramuscular injection at 11 mg/kg of body weight and halothane in oxygen (Floutec 3, Serial # 31242, Cyprane, North America Inc, Tonawanda, NY 14150, USA). Following anesthesia, birds were then gently placed on the surgery table with their ventral side up. Feathers were removed from the abdomen (30 cm^2) and the skin was aspectically cleansed using Novalsan Surgical Scrub-chlorhexidine (Fort Dodge Laboratories, Inc, Fort Dodge, IA, 50501, USA) for a rapid and residual antimicrobial effect. One to 5 ml of Nolvalsan Surgical Scrub was applied to the area and washed with cotton wool for two minutes following recommended procedures. A 1.5 cm incision was then made lateral to the cloaca, on the abdomen past the skin and fat tissue using a single Rib-Back Carbon steel sterile Surgical Blade (Bard-Parker, Becton Dickinson and Company, Lincoln Park, NJ 07035, USA) for implantation of radiotelemetry temperature transmitters (Mini-Mitter Telemetry System, Sun River, OR 97709, USA) in the abdominal cavity. The incision was immediately sutured with a sterile 3-0 ethilon black monofilament nylon non-absorbable surgical suture, USP (Ethicon Inc, Sommerville, NJ 08876, USA). The adjacent skin and fat tissue were sutured together with a minimum of six sutures per incision. After suturing, the operated surface area and surrounding skin were lightly and gently coated with Vaseline Petroleum Jelly to prevent further irritation and alleviate pain. The birds were finally injected with an antibiotic (Penicillin) as a prophylactic measure against infections. The birds were then allowed a twenty four hour recovery and adjustment period in individual respiratory chambers (51 x 34 x 41 cm) before undergoing a 24 hour fast.

RESPIRATORY CHAMBERS: Chambers were constructed of clear 63.5 mm acrylic plexiglass and fitted with Hart watering cups. Water supplying the drinking cups was supplied via a 1000 ml graduated cylinder. The chamber floor (51 x34 cm) was constructed of wire mesh suspended 9 cm above a 51 x 34 cm excreta collection pan containing 4 cm of mineral oil. The mineral oil was used to prevent voided excreta moisture from interacting with the chamber environment. Each compartment was fitted with a 3 cm fan (Radio Shack Cooling Fan Catalog # 273-244, Stillwater, OK 74075, USA) located at the top central part. This fan was used to mix air a prequisite for uniform gas sampling. Temperature probes (Model ES-060 Omnidata International, Logan, UT 84321) were used to monitor chamber temperature.

AIR SUPPLY: Air was delivered to the chambers as compressed air (Gradner-Denver, Quincy, IL. 62305) at 7% RH and at 24^{o}C through individual 64 mm diameter polythene transparent tubes. Each tube was passed through a computer monitored and controlled heat exchanger for proper air temperature (room temperature) before utilization by the birds in the chambers. A pressure regulator released flow at a constant pressure from the air compressor and microwaves were used to direct the desired flow rate through each chamber. Air flow was monitored through an electronic mass flow meter (Omega Engineering, Stamford, CT 06907). The air was then mixed using a 3 cm fan as explained under respiratory chambers above.

DATA ACQUISITION SYSTEM: Chamber and data measurements were controlled and monitored using a Workhose Data Acquisition and Control System (Omega Engineering, Stamford, CT 06907). Gas concentrations (O_2

and CO_2) and RH quantifications, flow rate and ambient temperature were recorded once for each chamber every 12 minutes.

OXYGEN (O_2) AND CARBON DIOXIDE (CO_2): Oxygen and CO_2 concentrations were determined five times per hour per chamber using oxygen and carbon dioxide analyzers (Ametek, Pittsburg, PA 15238) with a ±.2% and ±.03% accuracy respectively. Relative humidity was monitored by a relative humidity probe (Omnidata International, Logan, UT 84321) with a ±1% accuracy. Oxygen and carbon dioxide consumption were estimated by multiplying chamber air flow rate (litters/minute) by differential gas concentration between reference and test chambers.

HEAT PRODUCTION (HP): Because of problems with the oxygen analysers, only carbon dioxide values were used for determination of HP. Blaxter (1989) reported that HP can be estimated by considering heat produced per amount of oxygen consumed or carbon dioxide produced. He reported that production of one liter of carbon dioxide produces heat averaging 24 kJ (5.74 kcal) per liter per hour. Using this approach, HP was therefore estimated as follows:

$$HP\ (kcal/hr/BW^{.66} = CO_2\ (liters/hour/BW^{.66}) * 5.74\ kcal$$

HEMATOCHEMISTRY

In order to reduce variation, all operations regarding drawing of blood, like dissection of carcasses and separation of internal organs, was carried out by one person. Upon completion of the 8 hour gas measurement recording,

blood was immediately drawn from each bird through the brachial vein and drained into a polythene tube. The blood was allowed to clot at room temperature for about 20 minutes. The clot was then slightly and gently rimmed before centrifugation (IEC Model CI Centrifuge, International Equipment CO., 300 2nd Avenue, Needham Heights, MA 02149) which was carried out within an hour after drawing of blood (Lewandowski et al, 1986). Following centrifugation, the serum was immediately frozen at (-20^{o}C) until assay time for triglycerides (TG), total protein (TP), albumin (ALB), uric acid (URIC), creatinine (CRE), glucose (GLU) ,sodium (NA), calcium (CA) and potassium (K) using a Cobas Mira analyzing machine (Model Serial # 24-3037A, Cobas Mira Diagnostics, Roche, Roche Analytical Instruments Inc., Nutley, NJ 07110).

ORGAN WEIGHT MEASUREMENTS

Comparison of bird organ weights at different ages was done using the comparative slaughter technique (David and Mathieson, 1964; McDonald et al, 1988). After drawing of blood, birds were euthanatized by cervical dislocation in as humane a manner as possible. Rigor mortis was complete in all birds when dissection was commenced. The carcass was then dissected for removal and separation of the following organs: outer and inner right and left breast muscle (Pectoralis major and Pectoralis minor, respectively); proventriculus, gizzard, pancreas, small intestine, large intestine, cecum, bursa of Fabricius, left and right lungs, spleen, right and left thigh plus leg, abdominal fat pad (leaf fat surrounding the cloaca and abdominal muscles excluding fat around the gizzard) and liver. All organs were then weighed to the nearest .01g.

To avoid confounding of final organ weights, proventriculus, small intestine, large intestine and cecum were weighed after removal of contents by gently squeezing the organs between the thumb and forefinger while the gizzard was cut open, washed with tap water and blotted with absorbent paper towels. Organs weights were subsequently absolute weights and as percent of live body weight. Organ weight gains were expressed as percent of initial body weight for assessment of compensatory gain of each organ during the refeeding period.

Organs of each carcass were then pooled together (including feathers), placed in an aluminium pan, covered with aluminium foil and frozen. Carcasses were then thawed, weighed and autoclaved (American Sterilizing Company, Type DS 2036, Serial # 223711, Erie, PA, U. S. A.) for 12 hours at a maximum input pressure of 60 psi. After autoclaving, carcasses were cooled, weighed and ground using a domestic grinder (Regal La Machine - Electronic, 68b0 food preparation Machine, Model LM6, VB-VA). Ground carcasses were then thoroughly mixed to a homogenous consistency, placed in freezer bags and frozen at -20C until carcass analysis.

DIGESTIBILITY

Chromic oxide was added to the diet at .03% as a non-absorbable marker for determination of digestibility. Feed samples and fecal material from each bird were collected for a period of three days and thoroughly mixed before a grab sample was taken for drying in an oven at 120°F. Fecal samples were then ground to pass through a 1 mm sieve. Samples were ashed in a muffle furnace at 500°C for 6 hours. The ash was subsequently used for determination of

chromium content (AOAC, 1985) which was used for determination of Digestibility coefficients (DC) as follows:

DC=(g chromic oxide/kg feces-g chromic oxide /kg feed)÷(g chromic oxide feces)

STATISTICAL ANALYSIS

Data were analyzed using the General Linear Models (GLM) procedure of SAS® software (SAS Institute, 1985). The t-test was used to separate significant means within treatment effects. Regression analysis was used to estimate the the degree of accelerated and compensatory growth of previously restricted birds during the refeeding period. The backward elimination procedure of SAS® software (SAS, 1985) was to develop regression equations for predicting blood constituents at a given age and weight. Because treatment 1 and 5 were similar during the whole experimental period, data for treatment 5 were analysed as treatment 1 resulting in a total of seven treatments. The following model was used to analyze data :

$$Y_{ijk} = \mu + T_i + W_k + (TW)_{ik} + e_{ijk}$$

where Y_{ijk} = a j^{th} observation on the k^{th} day (k=7, 21, 35, 49) in the ^{ith} treatment

μ = overall mean

T_i = effect of the i^{th} treatment

W_k = effect of the k^{th} restriction period

$(TW)_{ik}$ = interaction between T_i and W_k and

e_{ijk} = random error term

CHAPTER IV

RESULTS

Occurrence of compensatory growth, interpreted as bird's ability to elicit final body weights (BW) similar to ad libitum fed controls has traditionally been used as a measure of success for feed restriction programs (Plavnik and Hurwitz, 1985, Griffiths et al, 1977; Pokniak and Cornejo, 1982). As discussed above, methodology for measurement of occurrence of compensatory growth has basically looked at ability of restricted birds to catch-up with ad libitum fed broilers in final body weight. In this study, two terms, accelerated and compensatory growth were used to delineate different effects of feed restriction programs on bird performance during the refeeding period.

COMPENSATORY GROWTH HYPOTHESIS: Birds undergoing compensatory growth are associated with an increase in growth during the refeeding period. In order to evaluate the ability of previously restricted birds to compensory growth, regression equations (Table 4) were developed to predict the rate of live body and organ weight gain as percent of initial weight during the refeeding period. The pattern of live body weight gain as percent of initial weight for ad libitum fed broilers is presented in Figure 1. Because initial organ weights of birds killed at the end of restriction and refeeding periods were not known, regression equations (Table 5), were developed to predict intial organ weight as percent of body weight.

The following computations were used for determination of degree of compensatory or accelerated growth:

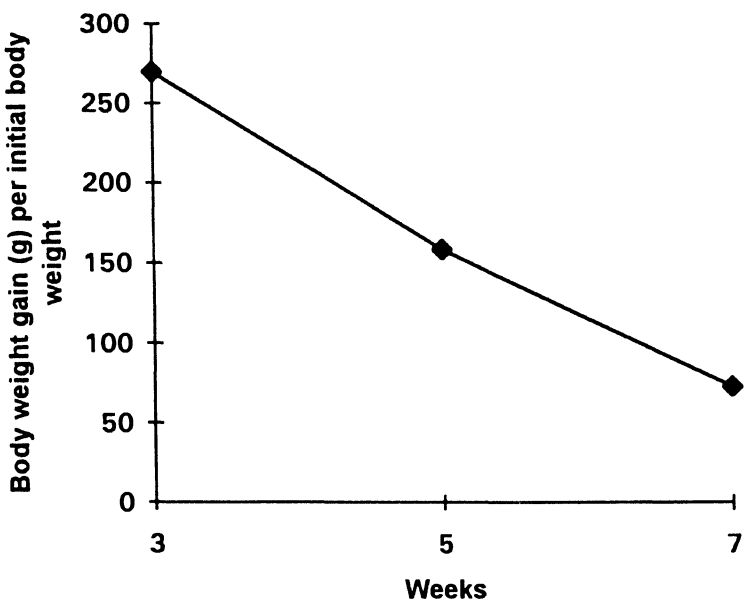

Figure 1. Body weight gain as percent of initial body weight of ad libitum fed male broilers at the end of 3, 5 and 7 weeks of age

Table 4. Regression equations for predicting degree of compensatory gain as percent of initial weight (Y) of male broilers at a given initial weight (x)

Variable (Y)	Regression equation	R^2
Livegain	$3.746671 - .005328x + .000002349x^2$	.97
Total breast	$.328994 - .000305x + .000000102x^2$	.88
Total thigh plus leg	$.472314 - .000417x + .000000136x^2$	.98
Abdominal fat pad	$.000733 + .000016761x$	.96
Viscera	$.324096 - .00063x + .000000325x^2$	.92
Total lung	$.012511 - .000014473x + .0000000091842225x^2$	.80
Heart	$.021949 - .000040056x + .000000017632978x^2$	.93
Liver	$.080687 - .00014x + .000000064482899x^2$	.93
Gizzard	$.06533 - .000105x + .000000043833358x^2$	.79
Large intestine	$.003650 - .000002370x + .0000000004643411x$	.88
Small intestine	$.084491 - .000196 + .0000006638613x^2$	.95
Pancreas	$.008641 - .000015378x + .0000000087280293x^2$	.95
Cecum	$.012544 - .000020035x + .000000010171503x^2$	.92
Bursa of Fabricious	$.007354 - .000011029x + 4.4904366x^2$	.49
Spleen	$.003287 - .000003254x .0000000011684729x^2$	.48
Carcass Protein	$44.664039 + .033735x - .000016908x^2$	.95
Carcass Fat	$.102296 + .000262x - .00000006351761x^2$	.92

Viscera = combination of proventriculus, spleen, pancreas, lungs, gizzard, small intestine, large intestine, Bursa of Fabricious, heart, liver and proventriculus

Total breast= Pectoralis major + Pectoralis minor

x^2 denotes a quadratic relationship between weight and organ weight as percent of body weight

$$GIWT=\{(A-B)/B\ )\}*100$$

$$CG=GIWT-PGIWT$$

where A = body or organ weight at the end of restriction/refeeding period

B = body or organ weight at the beginning of restriction/refeeding period

GIWT= Live body weight or organ gain as percent of B

PGIWT=Predicted liveweight or organ gain per initial weight (Table 5)

CG = degree of accelerated or compensatory growth

BODY WEIGHT

During the restriction period, feed restricted (FR) birds had had significantly lower ($P<.0001$) body weights than ad libitum fed birds (Table 6). Treatment effects were also reflected in significantly lower (<.0001) live body weight gains of FR birds than control birds. Body weight gain during the first two weeks (period 2) following restriction (period 1) were not affected by dietary treatment (Table 6) for birds restricted up to 70% of ad libitum feed consumption However, when body weight gain from 21 to 25 days (period 2) was expressed as percentage of initial body weight at 35 days of age (end of period 2),

Table 6. Effect of varying feed restriction level and initiation time on live body weight (BW), BW gain, BW gain as percent of initial weight and compensatory gain of male broilers

TRT	1	2	3	4	5	6	7	SEM[1]	Probability TRT	PD	TRT x PD
Body weight (g)											
PD 1	550^{a}	460^{b}	413^{a}	375^{c}	552^{a}	543^{a}	551^{a}	20	.0001	.3539	.6939
PD 2	1370^{a}	1140^{b}	1223^{b}	1301^{a}	1097^{b}	1039^{ab}	865^{b}	64			
PD 3	2469^{a}	2380^{ab}	2376^{ab}	2342^{ab}	2262^{ab}	2228^{ab}	2130^{b}	78			
Live body weight gain (g)											
PD 1	376^{ab}	330^{b}	280^{c}	238^{c}	412^{a}	401^{a}	402^{a}	43	.2218	.0001	.0001
PD 2	820^{ab}	680^{b}	810^{ab}	926^{c}	545^{c}	496^{b}	314^{c}	64			
PD 3	1099^{b}	1240^{b}	1153^{ab}	1041^{b}	1165^{ab}	1189^{a}	1265^{a}	56			
Body weight gain (% of initial weight)											
PD 1	270^{a}	257^{a}	212^{b}	175^{b}	294^{a}	281^{a}	272^{a}	13	.0708	.0001	.0001
PD 2	150^{d}	147^{c}	196^{b}	246^{a}	98^{e}	91^{e}	57^{f}	12			
PD 3	80^{d}	108^{d}	94^{d}	80^{d}	106^{c}	114^{b}	146^{a}	10			
[2]Compensatory gain (%)											
PD 2	.10^{b}	-14^{b}	8.8^{b}	76^{a}	-	-	-	.002	.0001	.0001	.0001
PD 3	-9c	-117^{d}	-108^{c}	-141^{e}	32^{b}	51^{ab}	59^{a}	.003			

[a-f] Means within a row with unlike superscripts under common subheading differ

PD= period; PD 1= day 7 to 21; PD 2 = day 21 to 35; PD 3= day 35 to 49
Treatment 1, 2, 3 and 4 = ad libitum feed consumption; 85% of ad libitum feed consumption , 70% of ad libitum feed consumption and 55% of ad feed consumption respectively in period 1 and then on ad libitum feed consumption during periods 2 and 3.
Treatments 5, 6 and 7= ad libitum feed intake from 7 to 21 days and then fed 85%, 70% and 55% of ad libitum feed consumption from 21 to 35 days; and then on ad libitum feed intake from 35 to 49 days
[1] SEM=pooled standard error of the mean; [2]Calculated as gain per initial initial weight- predicted gain per initial weight (Table 5)

Table 7. Effect of feed restriction and initiation time on feed consumption, feed efficiency and feed per initial weight of male broilers

TRT	1	2	3	4	5	6	7	SEM[1]	Probability TRT	PD	TRT X PD
Feed (g)											
PD 1	787^{a}	568^{b}	527^{bc}	476^{c}	760^{a}	773^{a}	765^{a}	72	.0072	.0001	.0001
PD 2	1539^{ab}	1221^{bcd}	1419^{abc}	1581^{a}	1132^{cd}	1008^{d}	916^{d}	75			
PD 3	2161^{ab}	1997^{b}	2250^{ab}	2086^{ab}	2218^{ab}	2415^{a}	2329^{ab}	73			
Gain :feed ratio (g:g)											
PD 1	.55^{ab}	.58^{a}	.53^{ab}	.49^{b}	.54^{ab}	.52^{ab}	.53^{ab}	.03	.0430	.0001	.0744
PD 2	.54^{b}	.60^{ab}	.57^{ab}	.62^{a}	.44^{c}	.48^{c}	.35^{d}	.01			
PD 3	.47	.51	.48	.48	.52	.52	.54	.02			
Feed per initial body weight (g)											
PD 1	5.50^{a}	4.38^{c}	3.97^{bc}	3.49^{c}	5.43^{a}	5.38^{a}	5.17^{a}	.23	.0001	.0001	.0001
PD 2	2.89^{c}	3.10^{c}	3.76^{b}	4.89^{a}	2.04^{d}	1.82^{de}	1.67^{e}	.16			
PD 3	1.54^{c}	1.57^{c}	1.74^{bc}	1.57^{c}	2.03^{b}	2.51^{a}	2.74^{a}	.13			
Digestibility Coefficients											
PD 1	.67	.68	.68	.70	.71	.70	.71	.02	.0506	.0601	.0531
PD 2	.71	.73	.71	.73	.70	..71	.74	.03			
PD 3	.71^{a}	.68^{b}	.69^{a}	.72^{a}	.65^{b}	.66^{b}	.73^{b}	.04			

[a-e] Means within a row with unlike superscripts under common subheading differ

PD= period; PD 1= day 7 to 21; period = day 21 to 35; period 3= day 35 to 49

Treatment 1, 2, 3 and 4 = ad libitum feed consumption; 85% of ad libitum feed consumption , 70% of ad libitum feed consumption and 55% of ad feed consumption respectively in period 1 and then on ad libitum feed consumption during periods 2 and 3; Treatments 5, 6 and 7= ad libitum feed intake from 7 to 21 days; 85%, 70% and 55% of ad libitum feed consumption respectively from 21 to 35 days and then on ad libitum feed intake from 35 to 49 days; [1] SEM=pooled standard error of the mean; [2] Calculated as gain per initial initial weight- predicted gain per initial weight (Table 5)

Table 8. Effect of feed restriction and initiation time on overall performance of male broilers from 7 to 49 days of age posthatch

TRT	1	2	3	4	5	6	7	SEM[1]	Probability TRT	PD	TRT xPD
BW (g)	2469^{ab}	2242^{ab}	2376^{ab}	2342^{ab}	2262^{ab}	2228^{ab}	2130^{a}	84	.0001	.0001	.0001
BW gain (g)	2328^{a}	2103^{ab}	2232^{ab}	2192^{ab}	2123^{ab}	2082^{ab}	1983^{b}	133	.0001	.0001	.0461
Gain/initial BW	16.5^{a}	15.1^{abc}	15.4^{ab}	14.7^{ab}	15.3^{abc}	14.3^{bc}	13.6^{c}	.56	.0001	.0001	.0001
Feed (g)	4440^{a}	4023^{b}	4133^{b}	4026^{b}	4194^{b}	4177^{b}	3942^{c}	87	.0001	.0001	.0001
Feed/initial BW	31.6^{a}	28.9^{ab}	28.6^{ab}	27.0^{b}	30.1^{ab}	28.8^{ab}	27.0^{b}	.83	.0001	.0001	.0001
Gain/Feed	.52	.52	.55	.54	.51	.50	.50	.001	.0001	.0001	.0001

[a-c] Means within a row with unlike superscripts under common subheading differ

BW=body weight ; PD 1= day 7 to 21; period = day 21 to 35; period 3= day 35 to 49

Treatment 1, 2, 3 and 4 = ad libitum feed consumption; 85% of ad libitum feed consumption , 70% of ad libitum feed consumption and 55% of ad feed consumption respectively in period 1 and then on ad libitum feed consumption during periods 2 and 3.
Treatments 5, 6 and 7= ad libitum feed intake from 7 to 21 days; 85%, 70% and 55% of ad libitum feed consumption respectively from 21 to 35 days and then on ad libitum feed intake from 35 to 49 days; [1] SEM=pooled standard error of the mean; [2] Calculated as gain per initial initial weight- predicted gain per initial weight (Table 5)

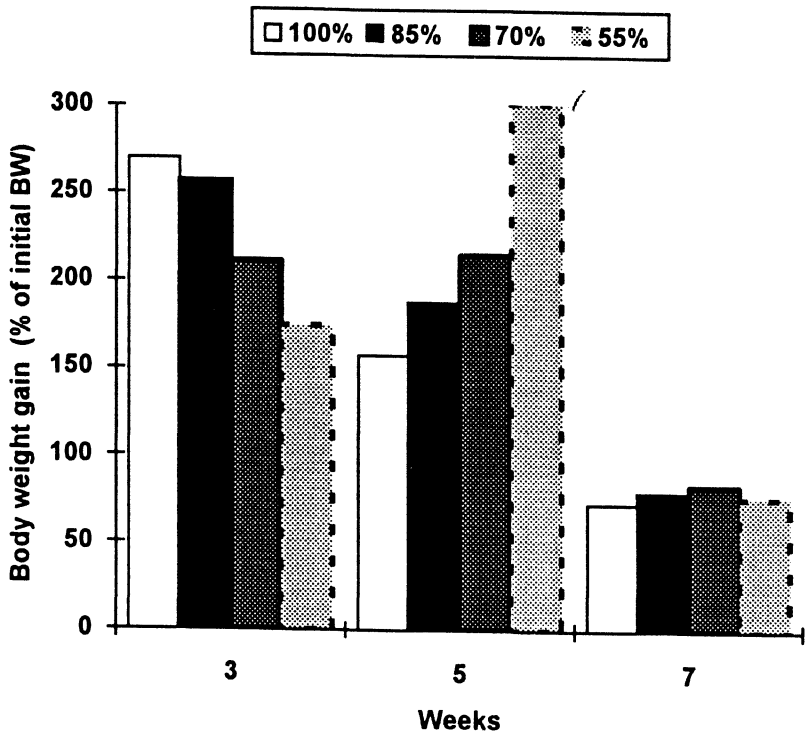

Figure 2. Body weight gain as percent of initial body weight of male broilers subjected to varying feed restriction levels from 1 to 3 weeks of age at the end of restriction (week 3) and refeeding periods (week 5 and week 7).

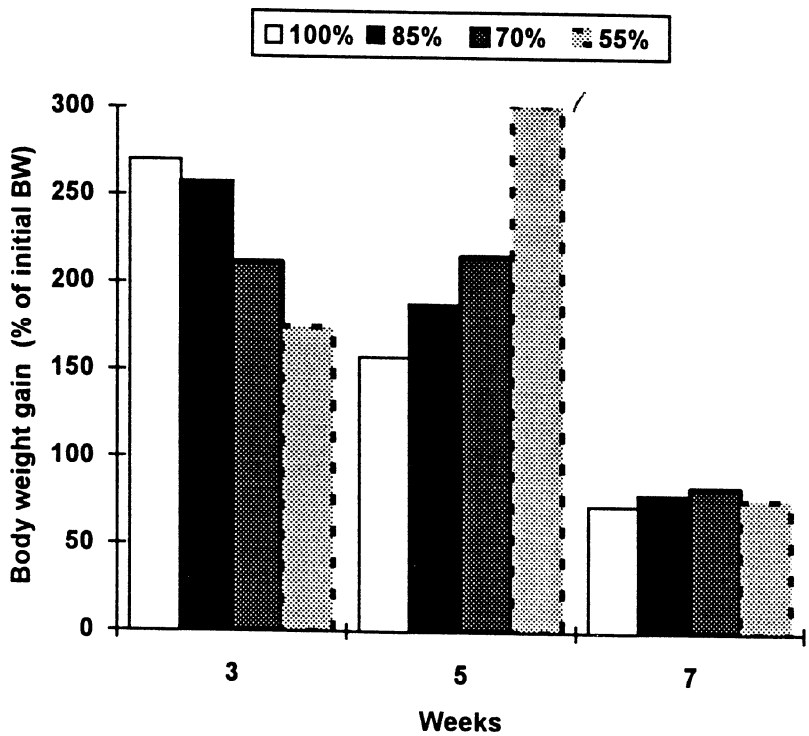

Figure 2. Body weight gain as percent of initial body weight of male broilers subjected to varying feed restriction levels from 1 to 3 weeks of age at the end of restriction (week 3) and refeeding periods (week 5 and week 7).

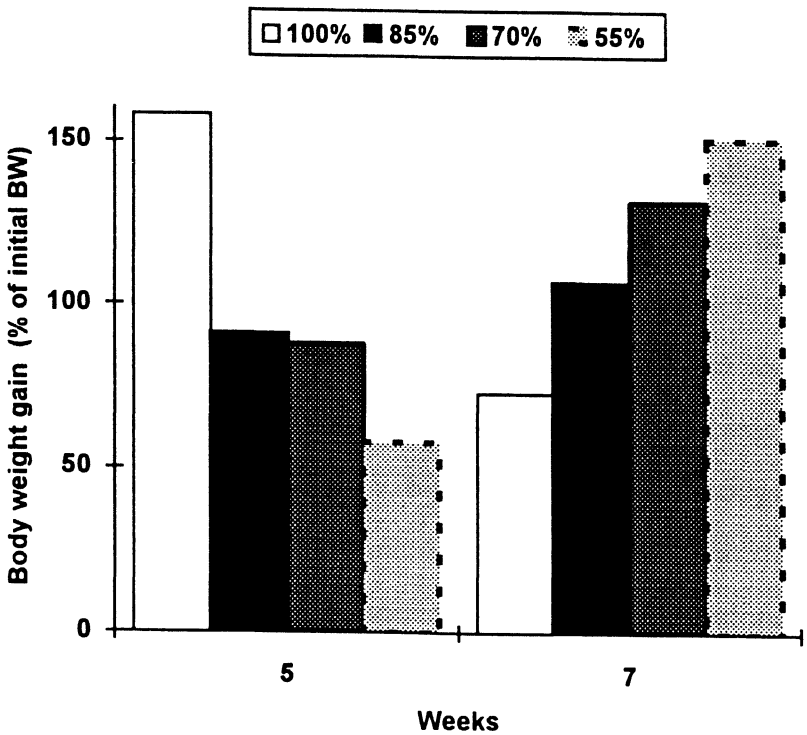

Figure 3. Body weight gain as percent of initial body weight of male broilers subjected to varying feed restriction levels from 3 to 5 weeks of age at the end of restriction (week 5) and refeeding periods (week 7)

significant (P<.0001) treatment effects were observed. The increase in body weight gain as percent of initial weight is clearly illustrated in Figure 2. This suggests that FR birds attempted to recover lost weight during the restriction period by eliciting accelerated growth. No treatment effects were observed for body weight gain as a percentage of initial weight during the later part of the refeeding phase (period 3). However, the trend to exhibit accelerated growth immediately following restriction was exhibited by birds restricted from 21 to 35 days of age (period 2, Trts 5, 6 and 7). Degree of body weight gain as a percent of initial weights tended to increase with degree of feed restriction imposed. This is reflected in the calculated degree of compensatory gain (Table 6). Birds restricted to 55% level had a significantly higher (P<.0001) compensatory gain than other treatments. The compensatory gain was higher for birds restricted in period 1 than those restricted in period 2. No compesatory growth was elicited

during period 3 by birds restricted in period 1. This suggests that birds previously subjected to feed restriction elicit most of the accelerated growth immediately following feed restriction other than later (Figure 2). This suggestion is further substantiated by the fact that birds restricted in period 2 exhibited compensatory growth immediately upon realimentation in period 3 (Figure 3). The ability of previously restricted birds to elicit complete compensation for lost weight observed in this study have been reported by other researchers (Plavnik and Huwitz, 1985, 1989; Plavnik et al, 1986); Pokniak and Cornejo, 1982). These results suggest that birds ability to elicit compensatory and accelerated growth is affected by degree of feed restriction and age at which feed restriction is initiated as evidenced by the significant treatment and period interaction (P<.0001).

On overall body weight performance (Table 8), birds previously restricted from 7 to 21 days of age (period 1) had final body weights similar to ad libitum fed birds while birds restricted later (period 2, Trts 5, 6 and 7) had lower (P<.0001) final bird weights than ad libitum fed birds. However, ad libitum fed birds showed a tendency to exhibit higher body weight as percentage of initial

body weight than FR birds. This observation is in concordance with that of Arafa et al (1983) who reported that subjecting broilers to feed restriction from 21 days of age resulted in lower final body weights when compared to ad libitum fed controls. These results suggest that although previously restricted birds may exhibit high rates of accelerated growth, final body weights may be adversely affected depending on the level of feed restriction imposed and age at which the restriction is imposed. In this regard, since not all birds which exhibit accelerated growth do attain weights similar to ad libitum fed controls, using the term compensatory growth to describe the increase in body weight gain as absolute amounts or as percent of initial BW may be misnomer. In view of these findings, and as suggested by others (Fontana et al, 1992), it is suggested that an increase in body weight gain of previously restricted broilers be defined in terms of accelerated growth which may not necessarily result in final body weights similar to control birds.

FEED INTAKE AND FEED EFFICIENCY

As anticipated, feed restricted birds had significantly lower (P<.0072) feed intake (Table 7) than ad libitum fed birds during the restriction period. This trend was observed through out the duration of the whole experiment (Table 8) There

was also a significant treatment by period interaction (P<.0001) in which birds restricted in period 2 had higher feed intakes than those restricted in period 1. the increase in feed consumption for birds restricted in period 2 may have been initiated as a compensatory response following restriction in period 2. When expressed as feed intake per initial body weight (Table 7), feed restriction level had a significant (P<.0001) effect on the ability of birds to elicit accelerated growth by consuming more feed. It was observed that the more severe the degree of feed restriction, the higher the feed intake per initial body weight during the refeeding period. The propensity to increase feed intake per initial body weight declined with age as evidenced by lower values in period 3 (Figure 4). Birds restricted to 85% of ad libitum feed consumption in period 1 or 2 showed the least ability to increase feed consumption during the first two weeks of feed restriction when compared to other restriction levels. There was a treatment and period interaction (P<.0001) in which feed intake per initial weight during the first two weeks following restriction was higher for birds restricted in period 1 (Table 7 and Figure 4) than those restricted in period 2 (Table 7 and Figure 5). Feed efficiency for birds restricted to 55% of ad libitum feed consumption broilers was lower (P<.0001) than that of AL broilers during the restriction period as reported by a other researchers (Fontana et al, 1992; Plavnik and Hurwitz, 1985; 1989; Plavnik et al, 1986, McMurtry et al, 1988; Pinchasov and Jensen, 1989). However, feed efficiency was similar between

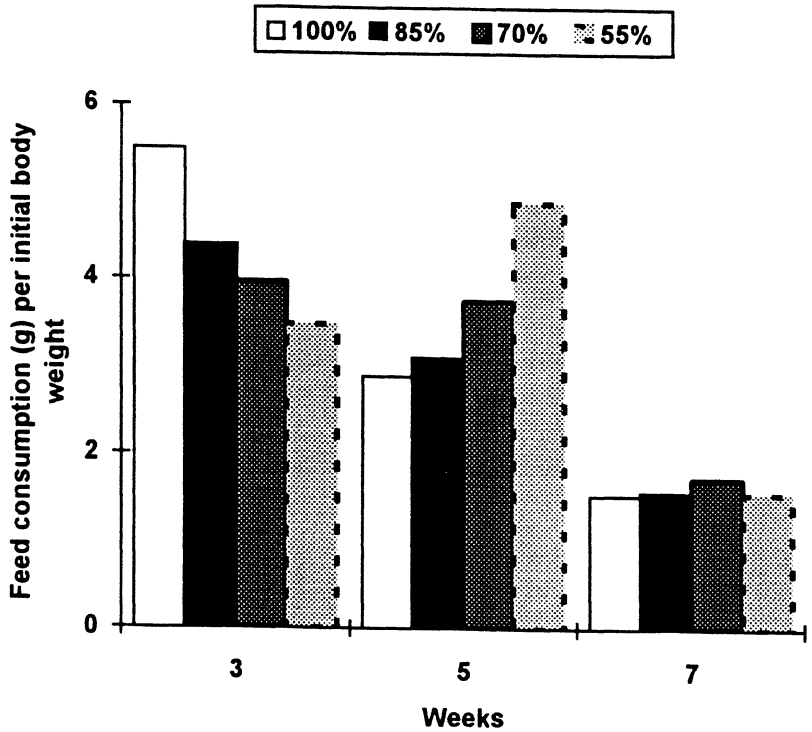

Figure 4. Feed consumption per initial unit body weight of male broilers subjected to varying feed restriction levels from 1 to 3 weeks of age at the end of restriction (week 3) and refeeding periods (week 5 and week 7)

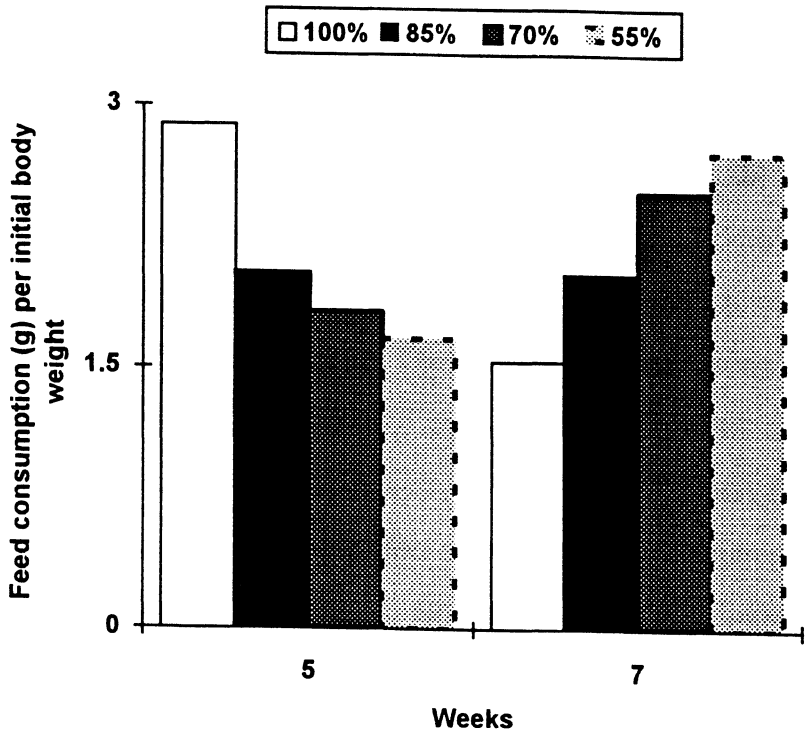

Figure 5. Feed consumption per initial unit body weight of male broilers subjected to varying feed restriction levels from 3 to 5 weeks of age at the end of restriction (week 5) and refeeding periods (week 7).

birds restricted to 85% and 70% of ad libitum feed consumption. No differences were seen in feed efficiency during period 3 (Table 7). Ad libitum fed birds had a significantly higher (P<.0001) feed intake (Table 8) than all restricted birds during the 7 to 49 day experimental period. No differences were observed in feed intake per initial body weights for the whole experimental period between restricted (85%, 70% of ad libitum feed consumption) and ad libitum fed birds. However, overall feed intake per initial body weight of birds restricted to 55% during period 2 was significantly lower (P<.0001) than ad libitum fed controls. On an overall basis, ad libitum fed birds consumed significantly more feed (P<.0001) than other treatment groups. No treatments effects were observed in digestibility coefficients of broilers during period 1 and 2 (Table 7). However, birds restricted to 85% and 70% of ad libitum feed intake during period 2 had lower (P<.0506) digestibility coefficients than other treatments in period 3. Due to an increase in feed intake, passage rate may have been accelerated suggesting less time for digestion. Contrary to this proposition, Teeter and Smith (1985) reported that time available for digestion and nutrient does not vary with feed intake. These results suggest that the ability of birds to elicit accelerated or compensatory growth is manifested in an increase in gain per initial body weight of restricted broilers which which, in turn, may be attributed to improved feed efficiency and an increase in feed intake relative to body weight (Tables 6 and 7, Figure 4 and 5). Results of improved feed efficiency by previously restricted birds have been reported by a number of workers (Plavnik and Hurwitz, 1985, 1991; Plavnik et al, 1986; Cabel and Waldroup, 1990). The ability of previously restricted birds to elicit compensatory growth have potential implications in practical poultry production. These results imply that early feed restriction programs may be potentially used as a tool to reduce the amount of feed provided to birds during

the early growth phase. In this study, actual reduction of feed intake ranged from 6 to 12% of ad libitum fed birds. Reduction of feed intake early in the growing phase have a profound impact on production costs since broiler starter diets are generally more expensive than grower and/or finisher diets. For the birds which did not compensate for lost body weight during the restriction period, prolongation of the refeeding period would be required. Whether additive effects of improved feed efficiency and reduction in amount of starter diet consumed would offset added costs associated with such a prolongation of production time for previously restricted birds needs further exploration.

ORGAN WEIGHTS

No treatment effects were observed on (breast, Table 9), lung (Table 11); viscera (a combination of proventriculus, liver, pancreas, small intestine, large intestine, total lung, cecum, bursa of Fabricious, gizzard, spleen (Table 12), heart, (Table 13); pancreas (Table 14); large intestine, (Table 15); cecum (Table 16); liver (Table 17); gizzard (Table 18); bursa of Fabricious (Table 20) and spleen (Table 21) weights during the 7 to 21 day (period 1) restriction. Total breast (Table 9) and thigh plus leg weight (Table10) of ad libitum fed birds was higher ($P<.0001$) than that of all restricted birds. Treatment effects during the period 1 restriction were also observed for small intestine weights (Table 19) where birds restricted to 55% of ad libitum feed consumption had lower ($P<.0001$) weights than all other treatments. Ad libitum fed birds had significantly higher breast (Table 9), thigh (Table 10), lung (Table 11), viscera (Table 12), heart (Table 13), large intestine (Table 15), cecum (Table, 16), liver (Table, 17), small intestine (Table 19), spleen (Table 21) weights than birds subjected to 55% feed restriction level in period 2. No treatment effects were

Table 9. Effect of feed restriction and initiation time on breast weight, breast gain, breast gain per initial weight and compensatory gain of male broilers

TRT	1	2	3	4	5	6	7	SEM[1]	Probability TRT	PD	TRT x PD
Breast (g)											
PD 1	47^{a}	46^{a}	38^{a}	33^{b}	43^{a}	45^{a}	45^{a}	9	.0001	.0001	.0001
PD 2	146^{a}	134^{abc}	120^{abc}	128^{ab}	109^{bc}	105^{bc}	89^{c}	6			
PD 3	332^{a}	276^{b}	293^{ab}	254^{b}	280^{ab}	273^{b}	258^{b}	7			
Breast (% of body weight)											
PD 1	9.4^{bc}	10.3^{abc}	9.5^{bc}	8.3^{c}	11.8^{a}	11.6^{a}	11.7^{a}	.1	.0007	.0001	.13
PD 2	10.7	10.5	9.8	9.9	10.3	10.0	10.8	.08			
PD 3	13.6^{abc}	11.9^{ab}	12.5^{ab}	11.1^{b}	12.4^{ab}	12.3^{ab}	12.1^{ab}	.7			
Breast gain (g)											
PD 1	40^{a}	39^{a}	32^{a}	26^{b}	40^{a}	39^{a}	40^{a}	6	.19	.0001	.0001
PD 2	92^{ab}	86^{ab}	78^{bc}	96^{a}	66^{c}	62^{cd}	47^{d}	7			
PD 3	138^{abc}	1195^{abc}	136^{abc}	104^{d}	147^{ab}	155^{a}	159^{a}	6			
Breast gain (% of initial body weight)											
PD 1	29.1	31.4	24.1	19.5	28.2	29.2	28.7	.02	.68	.0001	.0001
PD 2	17.4^{c}	19.2^{bc}	20.7^{bc}	30.0^{a}	12.0^{d}	11.2^{de}	8.6^{e}	..02			
PD 3	9.8^{b}	9.2^{c}	10.6^{c}	7.8^{c}	13.5^{b}	16.2^{ab}	18.7^{a}	.03			
[2]Compensatory gain (%)											
PD 2	-.2.12^{b}	-2.08^{b}	-2.19^{b}	5.87^{a}	-2.13^{b}	-2.11^{b}	-2.12^{b}	.009	.0001	.0001	.0001
PD 3	-9.43^{b}	-1.25^{b}	-1.13^{b}	-1.57^{c}	-.73^{a}	.72^{a}	2.13^{a}	.012			

[a-e] Means within a row with unlike superscripts under common subheading differ

PD= period; PD 1= day 7 to 21; period = day 21 to 35; period 3= day 35 to 49
Treatment 1, 2, 3 and 4 = ad libitum feed consumption; 85% of ad libitum feed consumption , 70% of ad libitum feed consumption and 55% of ad feed consumption respectively in period 1 and then on ad libitum feed consumption during periods 2 and 3; Treatments 5, 6 and 7=ad libitum feed intake from 7 to 21 days; 85%, 70% and 55% of ad libitum feed consumption repectively from 21 to 35 days and then on ad libitum feed intake from 35 to 49 days; [1] SEM=pooled standard error of the mean; [2]Calculated as gain per initial initial weight- predicted gain per initial weight (Table 5)

Table 10. Effect of feed restriction and initiation time on thigh weight, thigh gain, thigh gain per initial weight and compensatory gain of male broilers

									Probability		
TRT	1	2	3	4	5	6	7	SEM[1]	TRT	PD	TRT x PD
Thigh (g)											
PD 1	75^{a}	78^{a}	67^{a}	66^{b}	74^{a}	73^{a}	75^{a}	27	.00011	.0001	.0001
PD 2	236^{a}	205^{ab}	190^{abc}	202^{abc}	178^{bc}	189^{abc}	143^{c}	30			
PD 3	465	425	464	420	418	412	393	27			
Thigh (%)											
PD 1	15.4	17.4	16.5	16.5	17.9	18.1	18.4	.9	.4805	.0002	.3665
PD 2	17.3	16.1	15.5	15.6	17.0	18.2	17.2	.10			
PD 3	19.1	18.4	19.9	18.5	18.4	18.4	18.5	.7			
Thigh gain (g)											
PD 1	57	61	48	47	56	55	57	10	.62	.0001	.0001
PD 2	149^{ab}	131^{abc}	124^{bcd}	152^{a}	98^{d}	109^{cd}	63^{e}	15			
PD 3	192^{bc}	184^{c}	212^{abc}	181^{c}	217^{abc}	234^{ab}	240^{a}	10			
Thigh gain/body weight (%)											
PD 1	42	48	37	35	41	42	40	3	.68	.0001	.0001
PD 2	28.2^{b}	29.4^{b}	32.6^{b}	47.3^{a}	17.8^{c}	19.8^{c}	11.6^{d}	2			
PD 3	13.5^{d}	14.1^{b}	16.6^{cd}	13.6^{cd}	19.9^{c}	24.4^{b}	28.3^{a}	2			
[2]Compensatory gain (%)											
PD 2	-.71^{b}	-1.90^{b}	-.85^{b}	12.14^{a}				.013	.0001	.0001	.0001
PD 3	-14.9^{c}	-17.8^{cd}	-15.56^{c}	-20.8^{d}	1.7^{b}	4.5^{ab}	6.7^{a}	.012			

[a-d] Means within a row with unlike superscripts under common subheading differ

PD= period; PD 1= day 7 to 21; period = day 21 to 35; period 3= day 35 to 49
Treatment 1, 2, 3 and 4 = ad libitum feed consumption; 85% of ad libitum feed consumption , 70% of ad libitum feed consumption and 55% of ad feed consumption respectively in period 1 and then on ad libitum feed consumption during periods 2 and 3
Treatments 5, 6 and 7=ad libitum feed consumption from 7 to 21 days, 85%, 70% and 55% of ad libitum feed consumption repectively from 21 to 35 days and then on ad libitum feed intake from 35 to 49 days ; [1] SEM=pooled standard error of the mean; [2] Calculated as gain per initial initial weight- predicted gain per initial weight (Table 5)

Table 11 . Effect of feed restriction and initiation time on lung weight, lung gain, lung ain per initial weight and compensatory gain of male broilers

TRT	1	2	3	4	5	6	7	SEM[1]	Probability TRT	PD	TRT x PD
Lung (g)											
PD 1	2.36	2.82	2.58	2.33	2.33	2.34	2.38	1.1	.018	.0001	.0029
PD 2	6.06^{a}	4.83^{b}	4.96^{b}	4.84^{b}	4.19^{b}	4.15^{b}	4.5^{b}	1.5			
PD 3	14.67	12.39	11.26	10.94	8.98	10.37	12.68^{a}	1.3			
Lung (%)											
PD 1	.47^{ab}	.63^{a}	.63^{a}	.63^{a}	.40^{b}	.44^{ab}	.57^{ab}	.01	.17	.0001	.66
PD 2	.44^{ab}	.38^{b}	.37^{b}	.37^{b}	.40^{b}	.40^{b}	.54^{a}	.008			
PD 3	.6^{a}	.54^{a}	.5	.48^{a}	.40^{a}	.46^{a}	.59^{a}	.06			
Lung gain (g)											
PD 1	1.49	2.00^{c}	1.75	1.47	1.46	1.47	1.51	.88	.1702	.0001	.2900
PD 2	3.63^{a}	2.78^{ab}	3.37^{a}	3.63^{a}	1.76	1.71^{c}	2.08^{bc}	.81			
PD 3	6.05^{ab}	5.36^{ab}	5.17^{ab}	4.27^{b}	4.59^{ab}	5.89^{ab}	7.68^{a}	.66			
Lung gain per initial weight (%)											
PD 1	1.1	1.6	1.3	1.1	1.0	1.1	1.2	.2	.23	.0001	.0018
PD 2	.68^{bc}	.63^{cd}	.92^{ab}	1.12^{a}	.32^{e}	.31^{e}	.38de	.1			
PD 3	.42^{b}	.42^{b}	.40^{b}	.32^{ab}	.42^{b}	.6^{ab}	.92^{a}	.1			
[2]Compensatory gain (%)											
PD 2	.06^{ab}	.16^{b}	.08^{ab}	.24^{a}				.002	.0001	.0001	.0001
PD 3	.30^{abc}	.38^{bc}	.41^{abc}	-.54^{c}	-.35^{bc}	-.10^{b}	.23^{a}	.001			

[a-e] Means within a row with unlike superscripts under common subheading differ

PD= period; PD 1= day 7 to 21; period = day 21 to 35; period 3= day 35 to 49; Treatment 1, 2, 3 and 4 = ad libitum feed consumption; 85% of ad libitum feed consumption , 70% of ad libitum feed consumption and 55% of ad feed consumption respectively in period 1 and then on ad libitum feed consumption during periods 2 and 3; Treatments 5, 6 and 7= ad libitum feed consumption from 7 to 21 days; 85%, 70% and 55% of ad libitum feed consumption res pectively from 21 to 35 days and then on ad libitum feed intake from 35 to 49 days; [1] SEM=pooled standard error of the mean; [2] Calculated as gain per initial initial weight- predicted gain per initial weight (Table 5)

Table 12. Effect of feed restriction and initiation time on viscera[1] weight, viscera gain, viscera gain per initial weight and compensatory gain of male broilers

									Probability		
TRT	1	2	3	4	5	6	7	SEM[1]	TRT	PD	TRT x PD
Viscera (g)											
PD 1	55	59	50	47	54	55	54	8	.0003	.0001	.0001
PD 2	103[a]	88[ab]	95[ab]	91[ab]	86[b]	83[b]	81[b]	14			
PD 3	184[a]	165[ab]	174[ab]	165[ab]	148[ab]	147[b]	148[b]	9			
Viscera (% of body weight)											
PD 1	11.0[a]	13.12[a]	12.2[a]	12.0[a]	7.1[b]	7.0[b]	7.8[b]	.7	.0001	.0001	.0001
PD 2	7.5[b]	6.9[b]	7.8[ab]	7.0[b]	8.1[b]	8.1[b]	9.7[a]	.9			
PD 3	7.4	7.3	7.4	7.3	6.6	6.6	7.0	.69			
Viscera gain (g)											
PD 1	34	38	29	26	33	34	34	5	.4595	.0001	.0001
PD 2	54[ab]	49[b]	64[ab]	68[a]	21[c]	19[c]	17[c]	6			
PD 3	76	71	74	66	74	84	85	5			
Viscera gain per initial weight (%)											
PD 1	24.5	30.7	22.2	19.4	24.3	24.5	24.5	2.1	.393	.0001	.0001
PD 2	10.1[c]	11.2[c]	17.1[b]	21.2[a]	3.9[d]	3.5[d]	3.0[d]	2.1			
PD 3	5.3[c]	5.5[c]	5.8[c]	5.0[c]	6.8[bc]	8.7[ab]	10.1[a]	1.8			
[2]Compensatory gain (%)											
PD 2	1.96[d]	.34[c]	3.76[b]	5.78[a]				.23	.0001	.0001	.0001
PD 3	-2.23[c]	-5.97[d]	-5.96[d]	-9.54[c]	4.20[b]	6.77[a]	7.68[a]	.12			

[a-d] Means within a row with unlike superscripts under common subheading differ

PD= period; PD 1= day 7 to 21; period = day 21 to 35; period 3= day 35 to 49

Treatment 1, 2, 3 and 4 = ad libitum feed consumption; 85% of ad libitum feed consumption , 70% of ad libitum feed consumption and 55% of ad feed consumption respectively in period 1 and then on ad libitum feed consumption during periods 2 and 3; Treatments 5, 6 and 7= ad libitum feed consumption from 7 to 21 days; 85%, 70% and 55% of ad libitum feed consumption from 21 to 35 days and then on ad libitum feed intake from 35 to 49 days; [1] SEM=pooled standard error of the mean; [2]Calculated as gain per initial initial weight- predicted gain per initial weight (Table 5)

Table 13. Effect of feed restriction and initiation time on heart weight, heart gain, heart gain per initial weight and compensatory gain of male broilers

TRT	1	2	3	4	5	6	7	SEM[1]	Probability TRT	PD	TRT x PD
Heart (g)											
PD 1	3.31^{ab}	3.55^{a}	2.82^{a}	2.96^{a}	3.30^{ab}	3.28^{ab}	3.32^{ab}	.9	.121	.0001	.0001
PD 2	5.68^{ab}	6.12^{ab}	7.47^{a}	5.36^{ab}	6.25^{ab}	4.87^{b}	4.09^{b}	.7			
PD 3	10.2	9.67	10.96	10.42	8.58	8.60	7.79	.64			
Heart (% of body weight)											
PD 1	.69^{a}	.70	.60	.7	.68	.66	.69	.06	.0001	.0001	.0001
PD 2	.41^{b}	.49^{ab}	.60^{a}	.41^{b}	.60^{a}	.47^{ab}	.50^{ab}	.07			
PD 3	.41^{b}	.44	..47	.5	.38	.4	.4	.07			
Heart gain (g)											
PD 1	2.31	2.61	1.85	1.96	2.30	2.28	2.32	.6	.5165	.0001	.0001
PD 2	2.77^{bc}	3.51^{ab}	5.1^{a}	4.03^{ab}	2.71^{bc}	1.34^{cd}	.58^{e}	.7			
PD 3	4.21	4.18	4.32	4.42	4.03	4.90	4.57	.4			
Heart gain (% of initial body weight)											
PD 1	1.7	2.1	1.4	1.5	1.7	1.7	1.7	.2	.6812	.0001	.0013
PD 2	.52^{bc}	.80^{b}	1.35^{a}	1.25^{a}	.49^{bc}	.24^{cd}	.11^{d}	.2			
PD 3	.29^{b}	.34^{ab}	.33^{ab}	.33^{ab}	.37^{ab}	.51^{ab}	.51^{ab}	.1			
[2]Compensatory gain (%)											
PD 2	-.04	.03	.41	.16				.001	.0001	.0001	.0001
PD 3	-.23^{b}	-.46^{c}	-.49^{c}	-.68^{d}	.43^{a}	.53^{a}	.47^{a}	.0008			

[a-d] Means within a row with unlike superscripts under common subheading differ

PD= period; PD 1= day 7 to 21; period = day 21 to 35; period 3= day 35 to 49

Treatment 1, 2, 3 and 4 = ad libitum feed consumption; 85% of ad libitum feed consumption , 70% of ad libitum feed consumption and 55% of ad feed consumption respectively in period 1 and then on ad libitum feed consumption during periods 2 and 3; Treatments 5, 6 and 7= ad libitum feed consumption from 7 to 21 days; 85%, 70% and 55% of ad libitum feed consumption from 21 to 35 days and then on ad libitum feed intake from 35 to 49 days; [1] SEM=pooled standard error of the mean; [2] Calculated as gain per initial initial weight- predicted gain per initial weight (Table 5)

Table 14 . Effect of feed restriction and initiation time on pancreas weight, pancreas as percent of body weight, pancreas gain, pancreas gain per initial weight and compensatory gain of male broilers

									Probability		
TRT	1	2	3	4	5	6	7	SEM[1]	TRT	PD	TRT x PD
Pancreas (g)											
PD 1	1.34	1.71	1.53	1.41	1.33	1.32	1.34	3.2	.1686	.0001	.0003
PD 2	2.73	2.17	2.17	2.45	1.92	2.53	2.14	.3			
PD 3	3.97	4.41	4.55	4.14	3.78	3.67	3.65	.35			
Pancreas (% of body weight)											
PD 1	$.26^{b}$	$.38^{a}$	$.36^{a}$	$.36^{a}$	$.17^{ab}$	$.19^{b}$	$.20^{b}$	.003	.0001	.0001	.0001
PD 2	.20	.18	.19	.19	$.18^{ab}$	.24	.26	..002			
PD 3	.16	.19	.18	.18	.17	.16	.17	.002			
Pancreas gain (g)											
PD 1	.79	1.19	.99	.86	.78	.77	.79	.13			
PD 2	1.52^{ab}	1.17^{abc}	1.41^{abc}	1.84^{a}	$.62^{c}$	1.23^{abc}	$.84^{bc}$	.28	.7114	.0001	.0121
PD 3	1.62	1.89	1.97	1.61	1.88	2.10	2.09	.19			
Pancreas gain (% of initial body weight)											
PD 1	.56	.94	.75	.64	.55	.56	.54	.07	.2349	.0001	.0027
PD 2	$.29^{bc}$	$.28^{bc}$	$.37^{b}$	$.57^{a}$	$.11^{c}$	$.22^{bc}$	$.15^{c}$	.07			
PD 3	$.11^{c}$	$.14^{c}$	$.15^{bc}$	$.12^{c}$	$.17^{a}$	$.22^{a}$	$.25^{a}$	.06			
[2]Compensatory gain (%)											
PD 2	$-.00^{b}$	$-.07^{b}$	$-.04^{b}$	$.11^{a}$				.0002	.0812	.0001	.0009
PD 3	$-.17^{c}$	$-.22^{c}$	$-.22^{c}$	$-.31^{d}$	$-.06^{b}$	$.02^{a}$	$.06^{a}$	.0003			

[a-c] Means within a row with unlike superscripts under common subheading differ

PD= period; PD 1= day 7 to 21; period = day 21 to 35; period 3= day 35 to 49

Treatment 1, 2, 3 and 4 = ad libitum feed consumption; 85% of ad libitum feed consumption , 70% of ad libitum feed consumption and 55% of ad feed consumption respectively in period 1 and then on ad libitum feed consumption during periods 2 and 3; Treatments 5, 6 and 7= ad libitum feed consumption from 7 to 21 days; 85%, 70% and 55% of ad libitum feed consumption from 21 to 35 days and then on ad libitum feed intake from 35 to 49 days; [1] SEM=pooled standard error of the mean; [2] Calculated as gain per initial initial weight- predicted gain per initial weight (Table 5)

Table 15. Effect of feed restriction and initiation time on large intestine weight, large intestine gain, large intestine gain per initial weight and compensatory gain of male broilers

TRT	1	2	3	4	5	6	7	SEM[1]	Probability TRT	PD	TRT x PD
Large intestine (g)											
PD 1	.85	.82	.75	.80	.84	.85	.83	.16	.0001	.0001	.0001
PD 2	1.33^{b}	1.51^{ab}	1.34^{b}	1.78^{a}	1.41^{b}	1.38^{b}	1.36^{b}	.17			
PD 3	3.3^{a}	2.43^{b}	2.90^{ab}	2.65^{b}	2.41^{b}	2.31^{b}	2.45^{b}	.12			
Large intestine (% of body weight)											
PD 1	.17^{a}	.18^{a}	.18^{a}	.20^{b}	.11^{b}	.11^{b}	.13^{b}	.01	.0001	.0001	.0001
PD 2	.10^{c}	.12^{bc}	.11^{bc}	.14^{ab}	.13^{ab}	.13^{ab}	.16^{a}	.01			
PD 3	.14^{a}	.11^{b}	.12^{ab}	.12^{ab}	.10^{b}	.10^{b}	.11^{ab}	.01			
Large intestine gain (g)											
PD 1	.45	.45	.37	.40	.44	.46	.43	.13			.0001
PD 2	.62^{b}	.89^{b}	.92^{b}	1.34^{a}	.27^{c}	.24^{c}	.23^{c}	.09			
PD 3	1.37	1.04	1.26	1.04	1.20	1.30	1.39	1.11			
Large intestine gain (% of initial body weight)											
PD 1	.33	.36	.27	.30	.32	.33	.31	.004	.0023	.0001	.0001
PD 2	.12^{c}	.20^{b}	.25^{b}	.40^{a}	.04^{c}	.04^{c}	.04^{c}	.003			
PD 3	.10^{bc}	.08^{c}	.10^{bc}	.08^{c}	.11^{bc}	.14^{ab}	.16^{a}	.004			
[2]Compensatory gain (%)											
PD 2	-.13^{c}	-.06^{bc}	-.03^{b}	.12^{a}				.002	.0001	.0001	.0001
PD 3	-.15^{b}	-.19^{bc}	-.17^{bc}	.21^{c}	-.05^{a}	-.04^{a}	-.03^{a}	.0001			

[a-c] Means within a row with unlike superscripts under common subheading differ

PD= period; PD 1= day 7 to 21; period = day 21 to 35; period 3= day 35 to 49

Treatment 1, 2, 3 and 4 = ad libitum feed consumption; 85% of ad libitum feed consumption , 70% of ad libitum feed consumption and 55% of ad feed consumption respectively in period 1 and then on ad libitum feed consumption during periods 2 and 3; Treatments 5, 6 and 7= ad libitum feed consumption from 7 to 21 days; 85%, 70% and 55% of ad libitum feed consumption from 21 to 35 days and then on ad libitum feed intake from 35 to 49 days; [1] SEM=pooled standard error of the mean; [2] Calculated as gain per initial initial weight- predicted gain per initial weight (Table 5)

Table 16. Effect of feed restriction and initiation time on cecum weight, cecum gain, cecum gain per initial weight and compensatory gain of male broilers

TRT	1	2	3	4	5	6	7	SEM[1]	Probability TRT	PD	TRT x PD
Cecum (g)									.0003	.0001	.0001
PD 1	1.68	2.17	1.87	1.92	1.68	1.65	1.56	.73			
PD 2	4.44^{a}	3.55^{b}	3.35^{bc}	4.08^{ab}	2.23^{d}	2.30^{d}	2.62^{cd}	.55			
PD 3	6.43	6.39	6.11	6.48	6.51	6.71	6.78	.55			
Cecum (% of body weight)											
PD 1	.34	.48	.46.	.50	.26	.28	.32	.03	.0001	.0001	.0001
PD 2	.32^{a}	.28^{ab}	.27^{ab}	.31^{a}	.21^{b}	.22^{b}	.31^{a}	.04			
PD 3	.25	.28	.26	.29	.29	.30	.32	.03			
Cecum gain (g)											
PD 1	1.01	1.53	1.21	1.24	1.00	.88	.86	.30	.4975	.0001	.0001
PD 2	2.67^{ab}	2.04^{b}	2.29^{b}	3.07^{a}	.32^{d}	.38^{d}	.70^{c}	.21			
PD 3	2.67^{b}	2.74^{b}	2.57^{b}	2.72^{b}	3.46^{ab}	3.80^{a}	4.04^{a}	.23			
Cecum gain (% of initial body weight)											
PD 1	.73	1.20	.93	.91	.72	.68	.57	.10	.1642	.0001	.0001
PD 2	.50^{b}	.46^{b}	.60^{b}	.95^{a}	.06^{c}	.07^{c}	.13^{c}	.09			
PD 3	.18^{c}	.21^{c}	.19^{c}	.21^{c}	.32^{b}	.40^{ab}	.48^{a}	.08			
[2]Compensatory gain (%)											
PD 2	.02^{ab}	-.09^{b}	-.04^{b}	.24^{a}				.08	.0001	.0001	.0001
PD 3	-.27^{c}	-.37^{cd}	-.39^{d}	-.47^{d}	.03^{b}	.13^{ab}	.12^{a}	.19			

[a-d] Means within a row with unlike superscripts under common subheading differ

PD= period; PD 1= day 7 to 21; period = day 21 to 35; period 3= day 35 to 49

Treatment 1, 2, 3 and 4 = ad libitum feed consumption; 85% of ad libitum feed consumption , 70% of ad libitum feed consumption and 55% of ad feed consumption respectively in period 1 and then on ad libitum feed consumption during periods 2 and 3; Treatments 5, 6 and 7= ad libitum feed consumption from 7 to 21 days; 85%, 70% and 55% of ad libitum feed consumption from 21 to 35 days and then on ad libitum feed intake from 35 to 49 days; [1] SEM=pooled standard error of the mean; [2] Calculated as gain per initial initial weight- predicted gain per initial weight (Table 5)

Table 17 . Effect of feed restriction and initiation time on liver weight, liver gain, liver gain per initial weight and compensatory gain of male broilers

									Probability		
TRT	1	2	3	4	5	6	7	SEM[1]	TRT	PD	TRTXPD
Liver (g)											
PD 1	12.86	15.19	12.15	11.44	12.66	12.23	12.89	4.1	.2806	.0001	.0001
PD 2	23.40	22.73	22.78	20.62	20.87	19.24	19.86	3.2			
PD 3	54.17	46.88	57.11	50.71	40.49	40.36	43.49	4.1			
Liver (% of body weight)											
PD 1	2.59^{abc}	3.38^{a}	3.02^{a}	2.91^{ab}	1.84^{c}	1.81^{c}	2.14^{bc}	.31	.0001	.0001	.0173
PD 2	1.71^{ab}	1.84^{ab}	1.94^{ab}	1.59^{ab}	1.98^{ab}	1.85^{ab}	2.38^{a}	.29			
PD 3	2.16	2.17	2.42	2.23	1.79	1.80	2.03	.22			
Liver gain (g)											
PD 1	8.77	11.30	8.17	7.33	8.51	8.11	8.81	2.1	.7723	.0001	.3108
PD 2	12.05^{ab}	12.49^{ab}	15.39^{a}	15.49^{a}	7.61^{bc}	5.99^{c}	6.67^{c}	3.1			
PD 3	22.41	20.18	24.57	21.79	20.20	23.06	25.48	2.3			
Liver gain (% of initial body weight)											
PD 1	6.38	8.90	6.30	5.51	6.33	8.02	6.41	.76	.5075	.0001	.003
PD 2	2.25^{c}	2.88^{bc}	4.26^{ab}	4.79^{a}	1.38^{c}	1.08^{c}	1.22^{c}	.61			
PD 3	1.54^{b}	1.57^{b}	1.91^{b}	1.63^{b}	1.85^{b}	2.39^{ab}	3.0^{a}	.43			
[2]Compensatory gain (%)											
PD 2	$.21^{b}$	$.11^{b}$	$.83^{a}$	$.79^{a}$				.003	.0001	.0001	.1142
PD 3	$-.36^{c}$	-1.36^{cd}	-1.09^{cd}	-2.11^{d}	1.27^{b}	2.00^{ab}	2.47^{a}	.003			

[a-c] Means within a row with unlike superscripts under common subheading differ

PD= period; PD 1= day 7 to 21; period = day 21 to 35; period 3= day 35 to 49

Treatment 1, 2, 3 and 4 = ad libitum feed consumption; 85% of ad libitum feed consumption , 70% of ad libitum feed consumption and 55% of ad feed consumption respectively in period 1 and then on ad libitum feed consumption during periods 2 and 3; Treatments 5, 6 and 7= ad libitum feed consumption from 7 to 21 days; 85%, 70% and 55% of ad libitum feed consumption from 21 to 35 days and then on ad libitum feed intake from 35 to 49 days; [1] SEM=pooled standard error of the mean; [2] Calculated as gain per initial initial weight- predicted gain per initial weight (Table 5)

Table 18 . Effect of feed restriction and initiation time on gizzard weight, gizzard gain, gizzard gain per initial weight and compensatory gain of male broilers

									Probability		
TRT	1	2	3	4	5	6	7	SEM[1]	TRT	PD	TRT x PD
Gizzard (g)									.0001	.0001	.0001
PD 1	12.78	12.32	10.27	9.59	12.77	12.69	12.81	1.23			
PD 2	22.52	18.53	20.18	20.50	19.86	20.26	18.53	2.11			
PD 3	34.12	32.43	30.35	32.06	32.41	29.59	29.48	1.11			
Gizzard (% of body weight)											
PD 1	2.51^{a}	2.68^{a}	2.51^{a}	2.45^{a}	2.50	1.45	2.54	.13	.0001	.0001	.0001
PD 2	1.64^{bc}	1.46^{c}	1.63^{bc}	1.57^{bc}	1.88^{ab}	1.94^{ab}	2.22^{a}	.08			
PD 3	1.42	1.41	1.29	1.42	1.43	1.64	1.39	.09			
Gizzard gain (g)											
PD 1	7.51^{a}	7.29^{a}	5.13^{ab}	4.29	7.47	6.31	7.21	1.22	.8122	.0001	.0001
PD 2	11.54^{a}	10.28^{a}	13.20^{a}	15.40^{a}	7.09^{b}	7.57^{b}	5.72^{b}	.81			
PD 3	14.16	14.00	12.87	12.85	16.40	16.81	16.75	.91			
Gizzard gain (% of body weight)											
PD 1	5.39^{a}	5.70^{a}	3.91^{ab}	3.21^{b}	5.11^{a}	4.35^{ab}	4.97^{ab}	.42	.3382	.0001	.0001
PD 2	2.15^{c}	2.30^{c}	3.43^{b}	4.76^{a}	.69^{d}	.77^{d}	.47^{d}	.24			
PD 3	1.01^{c}	1.08^{c}	0.99^{c}	.96^{c}	1.51^{b}	1.75^{ab}	1.98^{a}	.32			
[2]Compensatory gain (%)											
PD 2	-.03^{ab}	-.40^{b}	.23^{ab}	1.16^{a}				.003	.0001	.0001	.0001
PD 3	-.1.06^{b}	-1.76^{c}	-1.90^{c}	-2.45^{d}	1.18^{a}	1.25^{a}	1.19^{a}	.001			

[a-c] Means within a row with unlike superscripts under common subheading differ

PD= period; PD 1= day 7 to 21; period = day 21 to 35; period 3= day 35 to 49

Treatment 1, 2, 3 and 4 = ad libitum feed consumption; 85% of ad libitum feed consumption , 70% of ad libitum feed consumption and 55% of ad feed consumption respectively in period 1 and then on ad libitum feed consumption during periods 2 and 3; Treatments 5, 6 and 7= ad libitum feed consumption from 7 to 21 days;85%, 70% and 55% of ad libitum feed consumption from 21 to 35 days and then on ad libitum feed intake from 35 to 49 days; [1] SEM=pooled standard error of the mean; [2] Calculated as gain per initial initial weight- predicted gain per initial weight (Table 5)

Table 19 . Effect of feed restriction and initiation time on small intestine eight, small intestine gain, small intestine gain per initial weight and compensatory gain of male broilers

TRT	1	2	3	4	5	6	7	SEM[1]	Probability TRT	PD	TRT x PD
Small intestine (g)											
PD 1	15.42[a]	15.88[a]	14.03[ab]	12.96[b]	15.32[a]	15.47[a]	14.98[ab]	2.12	.0001	.0001	.0001
PD 2	26.74[a]	20.18[ab]	24.32[ab]	22.95[ab]	20.36[ab]	20.01[ab]	18.67[b]	1.89			
PD 3	40.67[a]	34.17[ab]	34.74[ab]	31.79[bc]	30.37 [bc]	29.42 [bc]	25.50[c]	1.87			
Small intestine (% of body weight)											
PD 1	3.07	3.52	3.44	3.31	1.52	1.50	1.52	.22	.0001	.0001	.0001
PD 2	1.95[ab]	1.59[b]	1.98[ab]	1.76[ab]	1.93[ab]	1.92[ab]	2.24[a]	.16			
PD 3	1.65[a]	1.51[ab]	1.48[ab]	1.39[ab]	1.34[ab]	1.32[ab]	1.21[b]	.14			
Small intestine gain (g)											
PD 1	8.47	9.25	7.26	5.98	8.36	8.54	8.00	1.85	.0028	.0001	.0001
PD 2	13.06[a]	10.98[a]	16.67[a]	17.24[a]	1.35[b]	1.01[b]	-.26[b]	1.23			
PD 3	17.01	14.91	14.54	11.33	14.96	16.68	13.98	1.6300			
Small intestine gain (% of initial body weight)											
PD 1	6.11	7.28	5.52	4.49	5.72	5.85	5.48	.007	.2022	.0001	.0001
PD 2	2.43	2.51	4.38	5.32[a]	.24[c]	.18[c]	-.04[c]	.004			
PD 3	1.20[bc]	1.14[bc]	1.12[bc]	.84[c]	1.37[abc]	.17[a]	1.64[ab]	.004			
[2]Compensatory gain (%)											
PD 2	2.51	1.48	2.38	2.56				.013	.0001	.0001	.0001
PD 3	1.51[b]	-.149[b]	-.283 [b]	-1.63[c]	1.71[a]	2.07[a]	1.98[a]	.321			

[a-c] Means within a row with unlike superscripts under common subheading differ

PD= period; PD 1= day 7 to 21; period = day 21 to 35; period 3= day 35 to 49
Treatment 1, 2, 3 and 4 = ad libitum feed consumption; 85% of ad libitum feed consumption , 70% of ad libitum feed consumption and 55% of ad feed consumption respectively in period 1 and then on ad libitum feed consumption during periods 2 and 3; Treatments 5, 6 and 7= ad libitum feed consumption from 7 to 21 days;85%, 70% and 55% of ad libitum feed consumption from 21 to 35 days and then on ad libitum feed intake from 35 to 49 days; [1] SEM=pooled standard error of the mean; [2] Calculated as gain per initial initial weight- predicted gain per initial weight (Table 5)

Table 20 . Effect of feed restriction and initiation time on bursa of Fabricious weight, bursa of Fabricious gain, bursa of Fabricious gain per initial weight and compensatory gain of male broilers

									Probability		
TRT	1	2	3	4	5	6	7	SEM[1]	TRT	PD	TRT x PD
Bursa (g)											
PD 1	1.02	1.25	.78	1.05	1.00	.98	.99	.38	.1218	.0039	.1496
PD 2	2.15	2.29	1.92	2.17	2.08	2.10	2.28	.29			
PD 3	3.85	4.00	4.03	2.90	3.31	3.49	3.66	.43			
Bursa (% of body weight)											
PD 1	.22	.27	.19	.24	.16	.17	.20	.03	.0001	.0001	.0001
PD 2	$.15^{b}$	$.18^{b}$	$.16^{b}$	$.18^{b}$	$.19^{b}$	$.20^{b}$	$.27^{a}$	.04			
PD 3	.19	.17	.17	.13	.15	.16	.17	.02			
Bursa gain (g)											
PD 1	.78	1.03	0.55	0.82	.75	.73	.77	.23	.3291	.0001	.2634
PD 2	1.13	1.36	1.29	1.63	1.16	1.18	1.37	.21			
PD 3	1.60^{ab}	1.73^{ab}	1.81^{a}	1.05^{b}	1.59^{ab}	1.97^{a}	2.03^{a}	24			
Bursa gain (% of initial body weight)											
PD 1	.59	.63	.43	.62	.51	.50	.53	.08	.6472	.0001	.3629
PD 2	$.21^{c}$	$.30^{bc}$	$.34^{b}$	$.50^{a}$	$.21^{c}$	$.21^{c}$	$.25^{bc}$	.09			
PD 3	$.11^{bc}$	$.14^{bc}$	$.14^{b}$	$.08^{a}$	$.14^{b}$	$.21^{a}$	$.23^{a}$	.09			
[2]Compensatory gain (%)											
PD 2	$-.06^{b}$	$-.03^{ab}$	$-.03^{ab}$	$.07^{a}$				.0002	.0001	.0001	.0001
PD 3	$-.15^{b}$	$-.21^{b}$	$-.21^{b}$	$-.32^{c}$	$.08^{a}$	$.12^{a}$	$.12^{a}$	.0002			

[a-c] Means within a row with unlike superscripts under common subheading differ

PD= period; PD 1= day 7 to 21; period = day 21 to 35; period 3= day 35 to 49

Treatment 1, 2, 3 and 4 = ad libitum feed consumption; 85% of ad libitum feed consumption , 70% of ad libitum feed consumption and 55% of ad feed consumption respectively in period 1 and then on ad libitum feed consumption during periods 2 and 3; Treatments 5, 6 and 7= ad libitum feed consumption from 7 to 21 days;85%, 70% and 55% of ad libitum feed consumption from 21 to 35 days and then on ad libitum feed intake from 35 to 49 days; [1] SEM=pooled standard error of the mean; [2]Calculated as gain per initial initial weight- predicted gain per initial weight (Table 5)

Table 21. Effect of feed restriction and initiation time on spleen weight, spleen gain, spleen gain per initial weight and compensatory gain of male broilers

									Probability		
TRT	1	2	3	4	5	6	7	SEM[1]	TRT	PD	TRT x PD
Spleen (g)											
PD 1	.50	.42	.31	.32	.51	.49	.48	.21	.0001	.0001	.0001
PD 2	1.37^{ab}	1.05^{b}	1.13^{b}	1.18^{b}	1.55^{a}	1.33^{ab}	1.28^{ab}	.27			
PD 3	2.81	2.71	2.72	3.15	3.08	3.11	3.05	.20			
Spleen (% of body weight)											
PD 1	.10	.09	.07	.08	.09	.09	.09	.009	.0001	.0046	.0841
PD 2	.10^{b}	.08^{b}	.09^{b}	.09^{a}	.14^{a}	.12^{a}	.15^{b}	.008			
PD 3	.11	.11	.11	.14	.13	.14	.14	.009			
Spleen gain (g)											
PD 1	.40	.32	.21	.22	.39	.39	.38	.11	.0003	.0001	.2946
PD 2	.79^{b}	.67^{b}	.76^{b}	.88^{ab}	1.12^{a}	.90^{ab}	.85^{ab}	.09			
PD 3	1.17^{b}	1.17^{b}	1.25^{b}	1.50^{ab}	1.60^{ab}	1.76^{a}	1.82^{a}	.11			
Spleen gain (% of initial bodyweight)											
PD 1	.29	.26	.17	.17	.28	.27	.28	.03	.0925	.0001	.0025
PD 2	.14^{b}	.15^{b}	.27^{a}	.27^{a}	.20^{b}	.16^{b}	.15^{b}	.04			
PD 3	.08^{c}	.09^{d}	.09^{ac}	.11^{cd}	.15^{bc}	.18^{ab}	.21^{a}	.03			
[2]Compensatory gain (%)											
PD 2	-.04^{b}	-.05^{b}	-.01^{ab}	.04^{a}				.0002	.0001	.0001	.0899
PD 3	-.10^{b}	-.12^{b}	-.11^{b}	-.11^{b}	.03^{a}	.06^{a}	.08^{a}	.0001			

[a-c] Means within a row with unlike superscripts under common subheading differ

PD= period; PD 1= day 7 to 21; period = day 21 to 35; period 3= day 35 to 49

Treatment 1, 2, 3 and 4 = ad libitum feed consumption; 85% of ad libitum feed consumption , 70% of ad libitum feed consumption and 55% of ad feed consumption respectively in period 1 and then on ad libitum feed consumption during periods 2 and 3; Treatments 5, 6 and 7= ad libitum feed consumption from 7 to 21 days;85%, 70% and 55% of ad libitum feed consumption from 21 to 35 days and then on ad libitum feed intake from 35 to 49 days; [1] SEM=pooled standard error of the mean; [2]Calculated as gain per initial initial weight- predicted gain per initial weight (Table 5)

Table 22. Effect of feed restriction and initiation time on abdominal fat pad weight, abdominal fat pad gain, abdominal fat pad gain per initial weight and compensatory gain of male broilers

									Probability		
TRT	1	2	3	4	5	6	7	SEM[1]	TRT	PD	TRT x PD
Abdominal fat pad (g)											
PD 1	.46[a]	.21[b]	.21[b]	.13[b]	.45[a]	.47[a]	.50[a]	.27	.0486	.0001	.0192
PD 2	5.15	3.70	3.71	3.32	1.60	1.58	.41	2.1			
PD 3	33.59	23.46	23.46	26.00	24.40	23.94	25.28	3.3			
Abdominal fat pad (% of initial body weight)											
PD 1	.09	.04	.00	.00	.09	.08	.08	.002	.0462	.0001	.0486
PD 2	.37[a]	.27[ab]	.26[ab]	.25[ab]	.15[bc]	.15[bc]	.05[c]	.001			
PD 3	1.39	1.02	1.08	1.17	1.11	1.11	1.18	.002			
Abdominal fat pad gain (g)											
PD 1	.44	.28	.12	.21	.45	.46	.50	.23	.8824	.0001	.7507
PD 2	3.88[a]	2.75[a]	2.12[ab]	2.49[ab]	.82[bc]	.81[bc]	-.34[c]	1.51			
PD 3	13.92	10.08	12.94	14.19	13.53	13.43	16.03	1.7			
Abdominal fat pad gain (% of initial body weight)											
PD 1	.39	.22	.09	.16	.31	.32	.33	.002	.2388	.0001	.0059
PD 2	.74[a]	.60[a]	.53[a]	.77[a]	.15[b]	.15[b]	-.06[b]	.001			
PD 3	.99[b]	.77[b]	1.01[b]	1.09[b]	1.24[ab]	1.41[ab]	1.91[a]	.002			
[2]Compensatory gain (%)											
PD 2	-.13	-.12	-.05	.28				.0025	.0175	.1926	.9869
PD 3	.08[ab]	.09[ab]	.34[a]	.55[a]	-.57[b]	-.19[ab]	.49[a]	.0023			

[a-c] Means within a row with unlike superscripts under common subheading differ

PD= period; PD 1= day 7 to 21; period = day 21 to 35; period 3= day 35 to 49

Treatment 1, 2, 3 and 4 = ad libitum feed consumption; 85% of ad libitum feed consumption , 70% of ad libitum feed consumption and 55% of ad feed consumption respectively in period 1 and then on ad libitum feed consumption during periods 2 and 3; Treatments 5, 6 and 7= ad libitum feed consumption from 7 to 21 days;85%, 70% and 55% of ad libitum feed consumption from 21 to 35 days and then on ad libitum feed intake from 35 to 49 days; [1] SEM=pooled standard error of the mean; [2]Calculated as gain per initial initial weight- predicted gain per initial weight (Table 5)

observed for bursa of Fabricious (Table 20), gizzard (Table 18), pancreas (Table, 14) and total breast (Table 9). During the first two weeks weeks of the refeeding period, gain as percent of initial weight for all organs tended to increase with increasing degree of feed restriction. The increase in gain as percent of initial weight was higher in the refeeding period for birds restricted in period 1 than birds restricted in period 2. For birds restricted in period 1, organ weight gains as percent of initial body weights tended to decrease later in the refeeding period suggesting that gains are more pronounced early in the refeeding phase. It was also noted that compensatory gain tended to increase with increasing degree of feed restriction for most organs. When thigh plus leg, breast and viscera were compared, it was noted that thigh plus leg was the least affected by feed restriction imposed from 7 to 21 days of age (Figure 6) in terms of gain relative to initial body weight. Differences in these organs during restriction period 2 (Figure 7) were less than those in period 1 (Figure 1). This trend was true for compensatory gain during the first two weeks of the refeeding period where thigh plus leg (Table 10) and viscera (Table 12) had higher values of compensatory gain than breast muscle (Table 9). Similar to live body weight, level of compensatory gain also increased with degree of feed restriction. Higher ($P<.0001$) compensatory gain values were observed during the first two weeks of refeeding period for birds restricted during period 2 than birds restricted in period 1. These observations suggest that protein accretion was adversely impacted by feed restriction as evidenced by low potential lean tissue (breast muscle) to elicit compensatory growth. Additionally, these results suggest feed restriction imposed at an early age exhibit higher compensatory growth than that imposed late in the growing phase (day 21 to 35). It is also evident from these results that most of the compensatory gain occurs early in the refeeding phase

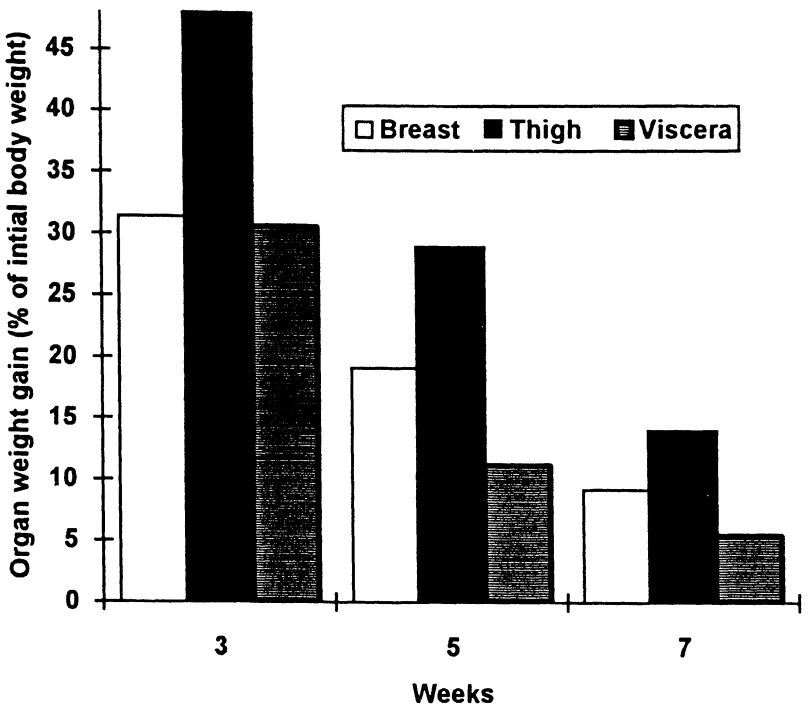

Figure 6. Effect of feed restriction (85% of ad libitum feed consumption from 1 to 3 weeks of age on breast, thigh plus leg and viscera gain as percent of initial body weight at the end of restriction (week 3) and refeeding period (week 5 and week 7)

[1]combination of proventriculus, pancreas, lung, spleen, cecum, small intestine, large intestine, bursa of Fabricious, gizzard

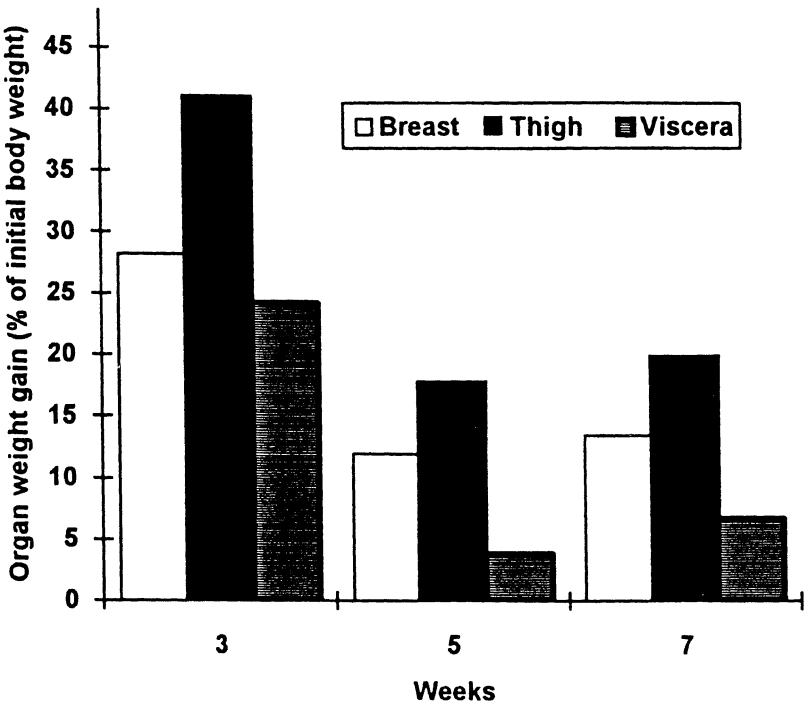

Figure 7. Effect of feed restriction (85% of ad libitum feed consumption from 3 to 5 weeks of age on breast, thigh plus leg and viscera gain as percent of initial body weight at the end of restriction (week 5) and refeeding period (week 7)

[1]combination of proventriculus, pancreas, lung, spleen, cecum, small intestine, large intestine, bursa of Fabricious, gizzard

Table 23. Effect of feed restriction and initiation time on serum glucose, triglycerides, total protein, uric acid and albumin of male broilers

									Probability		
TRT	1	2	3	4	5	6	7	SEM[1]	TRT	PD	TRT x PD
Glucose (mg/dl)											
PD 1	276[a]	240[b]	231[b]	226[c]	276[a]	276[a]	276[a]	21	.0001	.0001	.0001
PD 2	268[a]	266[a]	265[a]	237[b]	263[a]	254[ab]	225[b]	13			
PD 3	217[b]	228[b]	249[a]	256[a]	228[b]	228[b]	224[b]	15			
Triglycerides (mg/dl)											
PD 1	49[b]	54[a]	54[a]	56[a]	49[b]	49[b]	49[b]	2.11	.0001	.0001	.0001
PD 2	25[b]	24[b]	24[b]	22[b]	43[a]	44[a]	55[a]	2.13			
PD 3	19[b]	20[b]	20[b]	25[a]	23[ab]	23[ab]	23[ab]	1.23			
Total protein (mg/dl)											
PD 1	4.1[ab]	4.4[a]	3.9[b]	3.8[b]	4.1[ab]	4.1[ab]	4.1[ab]	.09	.0001	.0001	.0001
PD 2	3.1	3.3	3.0	3.1	3.4	3.4	3.4	.08			
PD 3	3.0	3.5	3.6	3.4	3.5	3.4	3.1	.09			
Uric acid (mg/dl)											
PD 1	4.53[a]	4.22[a]	3.52[b]	3.22[b]	4.53[a]	4.53[a]	4.53[a]	1.00	.0001	.0001	.0001
PD 2	4.41[b]	4.45[b]	4.76[ab]	4.73[ab]	5.2[a]	5.9[a]	6.0[a]	.009			
PD 3	3.03	3.42	3.51	3.39	3.9	3.6	3.4	.052			
Albumin (g/dl)											
PD 1	1.62[a]	1.55[a]	1.46[a]	1.34[b]	1.62[a]	1.62[a]	1.62[a]	.0001	.0002	.0561	.0001
PD 2	.99	1.04	1.05	1.04	1.2	1.2	1.2	.0002			
PD 3	.99	.99	1.01	1.11	1.1	1.0	1.0	.002			

[a-c] Means within a row with unlike superscripts under common subheading differ

PD= period; PD 1= day 7 to 21; period = day 21 to 35; period 3= day 35 to 49

Treatment 1, 2, 3 and 4 = ad libitum feed consumption; 85% of ad libitum feed consumption , 70% of ad libitum feed consumption and 55% of ad feed consumption respectively in period 1 and then on ad libitum feed consumption during periods 2 and 3; Treatments 5, 6 and 7= ad libitum feed consumption from 7 to 21 days;85%, 70% and 55% of ad libitum feed consumption from 21 to 35 days and then on ad libitum feed intake from 35 to 49 days; [1] SEM=pooled standard error of the mean; [2] Calculated as gain per initial initial weight- predicted gain per initial weight (Table 5)

Table 24. Regression equations for predicting serum metabolite levels at a given weight and age of male broilers

Constituent	Predictive Equation	R^2
Glu, mg/dl	317.689 + 114.29(A)-.433(W)-81.807(A^2)+437(A*W)	0.53
Trig, mg/dl	179.175+33.017(A)-.292(W)-14.889(A^2) +.083(A*W)	0.99
TP, g/dl	5.554+2.04 -.009(A)-1.225 (A2)+.006 (A*W)	0.87
Alb,mg/dl	2.031+.995(A)-.004(W)-.496(A^2)+.003(A*W)	0.88
Uric, mg/dl	17.153+10.904(A)-.052(W)-4.246(A^2)+.022(A*W)	0.90
Crea, mg/dl	.312	0.00
Na, mEq/l	194.16+71.64(A)-.276(W)-34.639(A^2)-002(W2)+.178(A*W)	0.77
Ca, mEq/l	11.377+4.567-.021(W)-1.669(A^2) +.008(A*W)	0.87
K, mEq/l	11.2449+9.275(A)-.036(W)-1.751(A^2)+.007(A*W)	0.92

[1]Age=weeks posthatch
Glu=glucose; Trig=triglycerides; Alb=albumin; Uric=uric acid;
Crea=creatinine; Na=sodium; Ca= Calcium; K=Potassium

treatment effects were observed in serum glucose levels in period 3 for birds restricted in period 2 when compared to their control counterparts. The hypoglycemic condition observed during the restriction period is therefore a transient and cab be eliminated by provision of adequate dietary conditions. An increase in serum glucose levels in previously restricted have been reported in cattle (Blum et al, 1985) upon refeeding.

SERUM ALBUMIN, URIC ACID, TOTAL PROTEIN : Lower ($P<.0001$) serum albumin and total protein values (Table 23) were observed for FR birds than for AL broilers during the restriction period (period 1). Lower serum values have been associated with lower protein reserves (Lewandowski et al, 1986) although others, (Nir et al, 1974), have reported no change in albumin and total protein values during starvation. No treatments effects were observed in albumin values during period 2 and period 3 suggesting that feed restriction in the later growth phase has no effect in albumin levels. This was also reflected in total protein values which were not affected by feed restriction in period 2.

Serum uric acid levels decreased with increasing degree of feed restriction during restriction period 1. However, uric acid levels were higher in restriction period 2 than in period 1 (Table 23). These observations suggest an increase in protein metabolism during the early refeeding period (Period 2) when adequate amounts of dietary protein substrates are made available to the birds. No treatment effects were observed in uric acid levels later in the refeeding phase (period 3) suggesting reduced protein metabolism. Indeed the later part of the refeeding phase is associated with adipose tissue deposition.

SERUM TRIGLYCERIDES: Feed restriction provides less food energy than the body would normally expend. As a consequence, endogenous fuels must be

oxidized to make up the energy deficit. Mobilization of lipids during starvation is a common feature. During the restriction period, serum triglyceride levels increased with increasing degree of feed restriction. Restricted birds had significantly higher (P<.0001) triglyecride levels than ad libitum fed birds probably due to reduced dietary substrate availability. This then may have resulted in mobilization of readily releasable fatty acids to bridge the energy deficits for the severely restricted birds. This situation was reversed during both refeeding periods (period 2 and period 3). This may have subsequently resulted in reduced lipolytic processes. Consistent with this speculation, a fall in non-esterified fatty acids in steers during the refeeding period (Blum et al, 1985) has been reported. Contrary to these findings, Nir et al (1974) reported that force feeding increased plasma triglyceride and free fatty acid levels in broilers.

Using backward elimination procedure (SAS, 1985), predictive equations (Table 24) for serum constituents were developed by regression analysis. These predictive equations enable estimation of to predict normal serum constituents levels of ad libitum fed birds given a particular age and weight of ad libitum fed birds. The bird used in this experiment were between 7 and 49 days of age posthatch.

BASAL METABOLISM

Because of technical problems with the oxygen analyzer in the respiratory chambers, basal metabolism was evaluated using levels of carbon dioxide production. As shown in Table 25, AL birds had higher (P<.05) total carbon dioxide production than FR broilers. This could be attributed to the fact that AL birds were heavier than FR birds. Blaxter (1989) reported that heat production can be determined using amount of carbon dioxide produced or oxygen

Table 25. Effect of feed restriction and initiation time on carbon dioxide production (CO_2) and fasting total heat production (HP) of male broilers at the end of restriction and refeeding periods

TRT	1	2	3	4	5	6	7
Total carbon dioxide production (litres/ 8hours)							
PD 1	9.44[a]	6.96[b]	6.48[c]	6.08[d]	9.44[a]	9.41[a]	9.39[a]
PD 2	11.04[a]	10.38[b]	9.35[c]	9.90[c]	9.86[c]	9.73[c]	9.35[c]
PD 3	13.68[a]	12.08[b]	11.84[c]	10.80[d]	10.32[d]	10.48[d]	10.72[d]
Total fasting HP (kcal/8 hours)							
PD 1	54.18[a]	39.95[b]	37.19[b]	34.89[b]	54.18[a]	54.17[a]	54.16[a]
PD 2	63.36[a]	59.58[b]	53.66[c]	56.82[b]	56.59[b]	55.85[b]	53.66[c]
PD 3	78.52[a]	69.33[b]	67.96[b]	61.99[c]	59.23[c]	60.15[c]	61.53[c]

[a-d] Means within a period under same treatment with unlike superscripts differ

PD= period; PD 1= day 7 to 21; period = day 21 to 35; period 3= day 35 to 49

Treatment 1, 2, 3 and 4 = ad libitum feed consumption; 85% of ad libitum feed consumption , 70% of ad libitum feed consumption and 55% of ad feed consumption respectively in period 1 and then on ad libitum feed consumption during periods 2 and 3; Treatments 5, 6 and 7= ad libitum feed consumption from 7 to 21 days;85%, 70% and 55% of ad libitum feed consumption from 21 to 35 days and then on ad libitum feed intake from 35 to 49 days

[1] SEM=pooled standard error of the mean.

consumed. Blaxter (1989) reported that 5.736138 kcal of heat are produced by production of 1 liter of carbon dioxide. Using this value, heat production by all treatment groups was calculated. Heat production for ad libitum fed birds was significantly higher ($P<.05$) than that of restricted birds (period 1 and period 2, Table 25). There was a tendency for heat production to decrease with increasing degree of feed restriction. This could be associated with lower body weights and lower feed intake during the restriction period. Lower feed intake may also have resulted in reduced specific dynamic effect. This trend continued in the refeeding phases (period 2 and period 3). Lower heat production by FR birds during the restriction and refeeding periods may reflect lower maintenance requirements due to lower body weights. When accompanied by increased feed intake per initial body weight during the refeeding periods, lower maintenance requirements may result in an increase in dietary substrate availability which is prerequisite for occurrence of accelerated weight gain or growth during the refeeding period.

CHAPTER V

DISCUSSION

BODY WEIGHT: Considerable evidence regarding occurrence of accelerated and compensatory growth by previously restricted birds as evidenced in this study exists (Plavnik and Hurwitz 1985, 1990, Plavnik et al, 1986, McMurtry et al, 1988, Jones and Farrell, 1992a, 1992b). In their study, Plavnik and Hurwitz (1985) restricted their birds form 6 to 12 days of age and exhibited compensatory growth while in this study, birds were restricted for a longer period (14 days). This observation illustrates the fact that depending on the level of feed restriction imposed, complete compensation for lost body weight may occur

with longer continuous restriction periods. However, contrary to these findings, others (Wilson and Osbourn, 1960; Jones and Farrell, 1992b) have suggested that full body weight recovery can be attained by using a number of short restriction periods rather than long continuous ones.

Time at which feed restriction is imposed had a tremendous impact on broiler performance. In this study, birds restricted from day 21 to 35 (period 2) exhibited lower final body weights than those restricted from 7 to 21 days vis~vis ad libitum fed birds. Birds restricted in period 2 has less potential to recover lost weight as evidenced by low degree of compensatory gain. Failure of birds restricted in feed intake to attain weights similar to ad libitum fed controls have been reported by a number of workers (Washburn and Bondari, 1978; Pinchasov and Jensen, 1989; Cabel and Waldroup, 1988; Summers et al, 1990; Yu et al, 1990, Beane et al, 1979; Mollison et al, 1984; McMurtry et al, 1988). Yu et al (1990) observed that restricting broilers to 23 kcal per bird per day from 8 to 14 days had significantly lower body weights at day 56 than birds on ad libitum feed consumption. The restriction imposed may have been too severe to allow full compensation. Plavnik and Hurwitz (1985) proposed that about 40 kcal ME are required per day to maintain body weight. As reported by Yu and Robinson (1992), time at which feed restriction is imposed have a significant influence on occurrence of compensatory growth. McMurtry et al (1988) suggested that feed restriction for male and female broilers should be initiated at no later than seven days and five days of age respectively. On the other hand, Plavnik and Hurwitz (1988) recommended initiation of feed restriction at 3 and 5 days for male and female broilers respectively if optimum compensatory growth is to be attained. Jones and Farrell (1989) reported that short restrictions of less than four days allows the bird to fully recover after feed restriction. Feed restriction through

dietary dilution with cereal hulls from 4 to 11 days of age has also been reported to allow body weight recovery at six weeks of age (Leeson, 1990). Comparing 6 and 12 day restriction periods, Plavnik and Hurwitz (1986) noted that the 12 day restriction period had more detrimental effects on eight week body weight than the 6 day restriction period.

The above reported studies follow the same trend as was observed in the current studies. However, the length of restriction period used in this experiment was longer than those reported by other workers discussed above. Since compensatory growth was elicited by a longer feed restriction interval employed in the current study, it is postulated, ceteris paribus, that an improvement in bird performance could be attained with shorter a restriction period, particularly if imposed early in the growing phase. Failure of some birds restricted in period 2 (21 to 35 days of age) to exhibit compensatory growth has been observed by other workers (Arafa et al (1983) who observed that restricting birds in feed intake in the last three weeks of life (5 to 8 weeks) provided birds with inadequate time to elicit full compensation of lost weight. Where full compensation is not attained at normal slaughter age, prolongation of the refeeding phase would be required to allow compensatory growth to occur. Robinson et al (1992), observed that feed restricted birds had lower weights than AL birds at 7 weeks of age but this difference in final body weights was eliminated when the production cycle was extended to 9 weeks of age. Profitablity of extending the growing period need to be handled with caution since disadvantages of increased feed costs and lower carcass turnover due to longer production cycles may attenuate advantages of compensatory feed programs.

FEED INTAKE AND EFFICIENCY: In general, feed intake was reduced with increasing degree of feed restriction during the restriction period. The ability of ad libitum fed birds to showe better efficiency than restricted birds concur with reports of other investigators (Plavnik and Hurwitz, 1985, 1989; Plavnik et al, 1986, McMurtry et al, 1988; Pinchasov and Jensen, 1989). The observed increase in feed intake during the refeeding period was necessitated to accomodate the increase in gain per initial body weight when compared to ad libitum fed birds. Not surprisingly, feed restricted birds had a superior feed efficiency than ad libitum fed birds during the refeeding period immediately following restriction (period 2 for birds restricted from 7 to 21 days and period 3 for birds restricted from 21 to 35 days) than ad libitum fed birds. This suggests that previously restricted bird have a higher ability to utilize increased levels of dietary substrates during the early part of the refeeding period. On an overall basis, no differences were observed in feed efficiencies between ad libitum fed and restricted birds.

The advantage of improved feed efficiency may therefore be attenuated by lower final body weights. This observation is in agreement with other earlier workers (Summers et al, 1990; Yu et al, 1990) who reported that feed restricted birds had lower final body weights than ad libitum fed birds albeit eliciting feed efficiencies similar to ad libitum fed birds. These observations suggest that accelerated growth observed during the refeeding period could partly be attributed to higher feed efficiency and increased feed intake relative to initial body weight. Given this proposition, the ability to consume feed could be limiting, and full expression of accelerated or compensatory growth may be dependent on a further increase in nutrient intake.

Advantages of improved feed efficiency, lower feed intake and final body weights similar to ad libitum birds by previously restricted birds may have critical economic implications in the broiler industry. Reducing feed intake from 7 to 21 days (period 1) implies that cost of starter diet, the most expensive diet for broilers (due to high protein content required), may be reduced. This means that less of the starter diet can be fed followed by adequate provision of grower and finisher diets later which are generally cheaper than the starter diet

ORGAN WEIGHTS

Variation exists in the growth rates among organs. In birds, supply organs involved in the procurement or processing of energy (intestine, liver) have been reported to grow at a different rate when compared to demand organs (e. g. muscle, feathers) that are primary users of energy (Lilja, 1983). During the restriction period, gain of the highly prized portions of total breast (Pectoralis major and Pectoralis minor) and total leg + thigh (right thigh + left thigh) of ad libitum fed birds were higher than that of restricted birds particularly those birds restricted to 55% of ad libitum feed consumption. Breast gain may have been limited by lower protein intake for protein gain. Plavnik and Hurwitz (1989) reported that birds fed a lower protein level had a slight reduction in growth rate which was overcome by accelerated growth during the refeeding period suggesting that degree of feed restriction adversely affects growth of lean tissue. As a percent of live body weight, breast muscle weight values ranged from 12 to 14%. Plavnik and Hurwitz (1991) and Zubair and Leeson (1994) have reported that breast weight as percent of carcass values ranges from 13 to 15% and 18 to 20 % respectively. Differences between values reported by these workers may

be due to strains of birds used in the those studies. The values reported in this study were lower because the whole bird, not carcass, was used for computation of the percentages. Higher values could therefore have been attained if carcass weight were used.

A majority of internal organs had lower weights than ad libitum fed birds during the restriction period. Other researchers (Plavnik and Huwirtz, 1983) have reported that intestinal weights decrease slightly during feed restriction. Reduction of small intestine weights (on absolute weight basis) during feed restriction suggest that growth of previously restricted birds may be limited by size of the intestine. Alternatively, size of the intestine may be altered to accommodate the prevailing growth rate as modified by feed restriction. A proportionate decrease in internal organs (lung, heart, kidney and spleen) have been reported (Crompton and Walters, 1979; Moran, 1979).

COMPENSATORY RESPONSE OF BROILERS

BODY WEIGHT: Unlike most compensatory growth literature, this study employed both accelerated and compensatory growth terms to delineate what happens happening during the refeeding period. Accelerated growth was used to describe an increase in body weight or organ gain without necessarily attaining final weights similar to ad libitum controls while compensatory growth referred to birds' ability to fully compensate for lost weight during the restriction period upon realimentation. Regression equations were developed to assess the degree of compensatory and /or accelerated gain for previously restricted birds. These studies indicated that accelerated growth is a common occurrence

in previously restricted birds during the refeeding period. It was noted that increasing degree of feed restriction increased degree of accelerated growth when measured as gain per initial weight. This is in agreement with (Wilson and Osbourne, 1960) who reported that the degree to which an animal can recover from feed restriction is dependent upon the extent of feed restriction. The increase in body weight gain per initial weight was more pronounced immediately following feed restriction. It was also observed that an increase in feed consumption per initial body weight may be responsible for exhibition of accelerated and compensatory growth since it was observed that previously restricted birds had higher feed intake relative to their initial weights. An overshoot in availability of dietary substrates accompanied by lower maintenance requirements could lead to partitioning of nutrients to productive processes. Complete body weight recovery have previously been reported by restricting birds to 77% of ad libitum feed consumption from 8 to 14 days of age (Plavnik and Hurwitz, 1990). Indeed, restricted birds were reported (Plavnik and Hurwitz, 1990) to consume 64g more feed than ad libitum fed birds during the refeeding period. On the other hand, lower feed consumption by restricting birds from 7 to 14 days of age have been reported to reduce growth rate (Yu et al, 1990). An elevation in appetite following feed restriction has been reported in human beings (Ashworth and Milliward, 1986).

In this study, lower maintenance requirements were associated with lower carbon dioxide production and heat production levels by previously restricted birds versus ad libitum fed birds. A reduction in heat production has been associated with reduced maintenance requirements in rats (Forsum et al, 1981).

ABDOMINAL FAT PAD: An increase in compensatory gain for abdominal fat pad increased with increasing degree of feed restriction. this is probably due to an increase in availability of dietary metabolites than required to maintain homeostasis. Most of the abdominal fat pad gains occurs late in the refeeding phase suggesting that abdominal fat pad is a late occurring tissue. This observation further suggests that part of the compensatory gain observed in previously restricted birds is fat. Moran (1979) reported that a greater proportion of compensatory gain observed in broilers subjected to protein restriction imposed at an early age was composed of fat. In this study, there were no significant differences in percent of abdominal fat content of ad libitum fed and restricted birds on a percentage basis. However, abdominal fat pad weights were higher ($P<.0486$) for ad libitum fed birds than restricted birds at the end of the experiment in period 3 suggesting that feed restriction is a potential tool for reducing absolute quantities of fat in broiler carcasses. reduction in abdominal fat content through feed restriction has been previously reported (Arafa et al, 1983; Mollison et al, 1984). Lilburn et al (1982) reported a 50% reduction in abdominal fat content of male broilers due to feed restriction. On the other hand, other workers (Beane et al, 1979; Newcomb et al, 1992) have reported that feed restricted birds had heavier abdominal fat pad weights than ad libitum fed birds. reduction of abdominal fat pad weights and fat content in general is one of the important aspects of feed restriction programs. Apart from providing the consumer with a desired product, lean carcasses also may reduce production costs of processing plants. Losses due to excess fat deposition in broiler chickens have been estimated at $250-300 million annually (Rosebrough et al, 1986). Reduction of carcass fat may also reduce cleaning costs, pollution

problems associated with waste water disposal (Fisher, 1984) and skin bl;emishes (Quarles, 1968).

BODY ORGANS: Results reported herein indicate that total breast exhibited a lower rate of gain per initial weight than those exhibited by the viscera and thigh plus leg. Lower compensatory growth of breast muscle, the most prevalent lean tissue of broiler carcass suggest that feed restriction may limit protein accretion. When compared to birds restricted from 21 to 35 days, it was noted that more accelerated growth occurred with birds restricted from 7 to 21 days. Lower rate of accelerated growth for lean tissue may be related to protein metabolism. It has been reported that breast muscle protein synthesis and nucleic acid contents are more rapid than that for leg muscles (Milliward et al, 1974). These changes in rate of synthesis are related to RNA content of tissue. Because RNA and DNA concentrations decrease with age (Hentges et al, 1983, Kang et al, 1985), rate of growth may be reduced hence the low degree of accelerated growth observed with increasing age. Acar et al (1993) reported that concentrations (per gram of fresh tissue) of breast muscle DNA and RNA decreased rapidly with age from hatching (2 mg DNA and 3 mg RNA/g) to 6 weeks of age (.29 mg DNA and 1.8 mg RNA/g) and remained unchanged later. Feed restriction may therefore impact protein synthesis in the early stages by causing a disequilibrium in DNA and RNA synthesis.

An increase in growth of organs may be associated with functional demands. Accelerated growth elicited by the liver during the first two weeks of refeeding could be necessitated to accommodate increased lipogenic activity of the previously restricted birds as depicted by increased abdominal fat pad gains during the refeeding period as discussed above.

Increases in rate of growth for gizzard and small intestine may have been necessitated to accommodate the elevation in feed intake associated with previously restricted birds. The need to process more feed nutrients to accommodate the increase in metabolic functions may also be responsible for an increase in size of these 'supply' organs. Not much accelerated growth was observed for the large intestine, lung and bursa of Fabricious later in the refeeding phase (period 2) suggesting that these are early maturing tissues.

HEMATOCHEMISTRY

Meluzzi et al (1992) reported that level of blood constituents is influenced by a number of factors including genetics, feeding regimes, environment, physiological status of the animal, sex, age, pathological factors and age. Low glucose levels observed in this study during the restriction period suggests that birds became hypoglycemic. Lewandowski (1986) reported that small birds can succumb to hypoglycemia during starvation within a 24 hour period. In contrast to these observations, Nir et al (1974) reported that force feeding broilers beyond their normal intake had no effect on plasma glucose levels. The observation that glucose levels increased upon refeeding for both groups of restricted birds, indicates that the hypoglycemic condition observed during starvation is a transient occurrence and can be eliminated by provision of adequate dietary conditions. An increase in glucose levels has been reported in cattle (Blum et al, 1985) upon refeeding following feed restriction.

The decrease in total proteion levels observed in this experiment could be associated with low protein intake. Low serum protein values have been

reported to reflect parasitism or starvation (Lewandowski, 1986). During the restriction period, values of uric acid were lower than later in the refeeding period. Lower uric acid levels (a primary catabolic product of protein, nonprotein nitrogen and purines) for feed restricted than ad libitum fed birds was corresponded to decreases in amount of protein available to restricted birds. From the clinical point of view, albumin is the largest individual protein fraction in avian serum (Galvin, 1980). Low albumin values observed in restricted birds during restriction period may therefore be an indicator of low protein reserve status (Lewandowski et al, 1986). In contrast to this suggestion, Nir et al (1974) reported that starvation caused no change in albumin and total protein values.

BASAL METABOLISM

Low heat production by restricted birds observed in this study agrees with reports of Wiernusz and Teeter (1993) who reported that heat production levels reduced with decreasing feeding levels. Wiernusz and Teeter (1993) further reported that force feeding broilers to 0%, 3%, 6% and 9% of body weight resulted in heat production of 4.8, 5.1, 5.3 and 5.9 kcal/$BW^{.66}$ respectively. Heat production tended to increase with increasing degree of feed intake during the refeeding period. This could possibly be attributed to an increase in specific dynamic effect associated with consuming large amounts of feed relative to their body weights. Further interpretation suggests that low heat production implies lower maintenance requirement. Lower maintenance requirements result in an increase in availability of dietary substrates during the refeeding period due to increased feed intake. This increased availability of nutrients augments occurrence of accelerated and/or compensatory growth.

CHAPTER VI

SUMMARY AND CONCLUSIONS

GROWTH PERFORMANCE: Results reported herein indicate that feed restriction programs have a profound impact on performance of broilers during restriction and subsequent refeeding periods. Almost all previously restricted birds exhibited considerable accelerated growth during the refeeding period. However, this accelerated growth did not automatically result in final body weights similar to ad libitum fed controls. That is elicitation of accelerated growth by previously restricted birds may not necessarily result in compensatory growth. The extent of accelerated and compensatory growth (where it occurred) were dictated by the degree of feed restriction and the initiation time as reported by earlier workers (Wilson and Osbourne, 1960, Jones and Farrell, 1989, Pokniak and Cornejo, 1982, Plavnik and Hurwitz, 1985).

As advocated by earlier workers who recommended initiation of feed restriction at seven days of age (Jones and Farrell, 1989; Summers et al, 1990; Plavnik and Hurwitz, 1985; Pokniak and Cornejo, 1982) or earlier than seven days (Plavnik and Hurwitz; 1990;), the current study confirms that feed restriction when imposed at an early age (7 to 21 days posthatch) can result in better accelerated and/or compensatory growth than if imposed later in the growth phase (day 21 to 35). Indeed, Plavnik et al (1986) reported that broilers restricted in feed intake from 6 to 12 days of age had slightly greater final body weights than ad libitum fed controls.

Exhibition of accelerated and compensatory growth by previously restricted birds illustrates the tenacity with which the capacity to grow can be maintained unimpaired in spite of earlier underfeeding and malnutrition. The accelerated and compensatory growth attained could be attributed to an increase in feed intake per initial weight observed during the refeeding period which makes available more dietary substrates available for productive processes. Accelerated and compensatory growth are further enhanced by the fact that previously restricted birds have lower metabolic rates than ad libitum fed controls as indicated by lower carbon dioxide and heat production. This then suggests that lower maintenance requirements for previously restricted birds than ad libitum fed controls may contribute to birds' ability to compensate for lost weight during the refeeding period. Consequently, the lower maintenance requirement augments dietary substrate availability which is triggered by an increase in feed intake upon realimentation.

The current study further suggests that protein accretion is adversely affected by feed restriction as evidenced by reduced ability of breast muscle to gain weight when compared to other tissues such as thigh plus leg. It is also apparent that internal organs (viscera) are least affected by feed restriction levels probably because they are early maturing. It is also apparent from this study that feed restriction canb be used to reduce absolute quantities of abdominal fat although there may be no differences on a percentage basis.

In summary, this study has revealed

1. that previously restricted birds do exhibit accelerated growth following a period of feed restriction irrespective of age at initiation.

2. that degree of feed restriction has a significant impact on birds ability to exhibit compensatory growth. It is suggested that restriction birds to no more than 70% of ad libitum feed consumption can produce optimum results.

3. that birds restricted in feed intake at an earlier age (day 7 to 21) have a greater ability to elicit accelerated and compensatory growth than birds restricted later in the growth phase (day 21 to 35).

4. that accelerated and/or compensatory growth observed in previously restricted birds upon refeeding is attributed to an increase in feed intake per initial body weight (which results in an overshoot in availability of dietary substrates), improved feed efficiency and lower maintenance requirements associated with lower bird weights.

5. that breast muscle have a lower ability to exhibit accelerated growth than thigh plus leg suggesting that protein accretion is adversely affected by feed restriction.

6. that feed restriction is a potential tool for reducing abdominal fat content of broilers.

POTENTIAL FOR APPLICATION IN THE BROILER INDUSTRY: Where compensatory growth occurs, feed restriction programs are a potential panacea to many countries faced with irregular and low quality poultry feed supplies such as Malawi. Reduction in amount of feed used in the production cycle, particularly the starter diet, offer some potential economic advantages. Being the most expensive broiler diet, low quantities of starter diet used can result in reduced feed costs. Another potential exploration is use of low quality feed or by feed products. Potentially, broiler producers can use a deferred feeding program

where low quality feed (and presumably cost) during the early stages of growth followed by provision of standard broiler grower and finisher diets later. Use of low quality feed would then act as a method of feed restriction. Since use of quantitative feed restriction has been described as cumbersome (Yu and Robinson, 1992), use of dietary dilution technique would offset this disadvantage. Zubair and Leeson (1994) reported that replacing major ingredients of a starter diet with 50% oat hulls resulted in complete growth compensation at 35 days of age due to improved efficiency. For Malawi, use of maize bran to dilute standard starter diets offers an immediate potential exploration and application of compensatory feeding regimes.

FURTHER RESEARCH: The present day controversy circumscribing the compensatory growth phenomena demands further research. The uncertainty regarding occurrence of compensatory growth necessitates the realization that far reaching dogmatic statements regarding accelerated and compensatory growth may be scarcely justifiable until experiments have been extended to include all factors and variables with a tangential bearing to occurrence of compensatory growth.

Although variables such as organ growth, blood constituents, basal metabolism (heat production) and general growth performance investigated in the present study provide an insight into the accelerated and compensatory growth phenomena, further research is necessary. Conceivably, certain tissues and metabolic processes may be permanently impaired without furnishing evidence by any of the criteria used in the present study. It is therefore, suggested that measurements in accelerated growth and compensatory growth studies be extended to include protein and fat synthesis mechanisms, hormonal

and enzymatic reactions, environmental conditions, composition of individual organs and different broiler strains. Different degrees of feed restriction may further be explored. In summary, nutritional and physiological studies are needed to better describe and understand the compensatory growth phenomena.

REFERENCES

Acar, N., E. T. Moran, Jr and D.R. Mulvaney, 1993. Breast muscle development of commercial broilers from hatching to twelve weeks of age. *Poultry Sci. 72: 317-325.*

Albers, G, Barranon, Zurita and Ortiz, 1990. Correct feeding prevents ascites. *Poultry Misset 6:22-23*

Allden, W. G., 1970. The effects of nutritional deprivation on the subsequent productivity of sheep and cattle. *Nutr. Abstr. Rev. 40:1167-1184*

Allison, J. B. 1955. Biological Evaluation of Proteins. *Physio. Rev. 35:664*

Anonymous, 1987. Components of a broiler . Cobb Broiler Manual. p46. Cobb-Vantress, Inc.Siloam Springs, Ar 72761, U. S. A.

Anugwa, F. O. I. and W. G. Pond, 1989. Growth of whole body and organs of growing rats during feed restriction and subsequent realimentation. *Growth, Age and Dev. 53:167-174*

Arafa, A. S., M. A. Boone, D. M. Janky, H. R. Wilson, R. D. Miles and R. H. Harms, 1983. Energy restriction as a means of reducing fat pads in broilers. *Poultry Sci. 62:314-320.*

Ashworth, A and D. J. Millward, 1986. Catch-up growth in children. *Nutr. Rev. 44:157-163.*

Association of Official Analytical Chemists (AOAC) 1980. Methods of Analysis. 13th ed. Assoc. Off. Anal. Chem. Washington, DC.

Ballam, G. C. and B. E. March, 1979. Adipocyte size and number in mature broiler-type female chickens subjected to dietary restriction during the growing period. *Poultry Sci. 58: 940-948*

Barnes, B. A and B. F. Miller, 1981. Protein restriction and growth in roaster chicks. *Poultry. Sci. 60: 336-341*

Beane, W. L., J. A. Cherry, and W. D. Weaver, Jr. 1979. Intermittent light and restricted feeding of broiler chickens. *Poultry Sci. 58:567-571*

Belo, P. S., D. R. Rosmos and G. A. Leveille, 1976. Blood metabolites in the fed and fasted chicken. *J. Nutr. 106: 1135-1143.*

Blaxter, K. L. 1989. Energy metabolism in Animals and man. Cambridge University Place, Cambridge, England.

Blaxter, K. L. and J. A. F. Rook. 1953. The heat of combustion of the tissues of cattle in relation to their chemical composition. *Br. J. Nutr. 7:83-91*

Blum, J. W., W. Schnyder, P. L. Kunz, A. K. Blom, H. Bickel and A. Schurch, 1985. Reduced and compensatory growth: Endocrine an metabolic changes during feed restriction and refeeding in steers. *J. Nutr. 115:417-424*

Blundell, J. E. 1977. Is there a role for serotonin in feeding? *Int. J. Obesity 1:15-42.*

Bohman, V. R. 1955. Compensatory growth of beef cattle: the effect of hay maturity. *J. Anim. Sci. 14:249-255*

Bondi, A. 1987. Animal Nutrition. John Wiley and Sons.

Bowes, W. L., N. J. Julian , S. Leeson and T. Stirtzinger, 1988. Effect of feed restriction on fed efficiency and incidence of sudden death syndrome in broiler chickens. *Poultry. Sci. 67:1102-1104*

Brady, L. J., D. R. Rosmos, P. S.Brady, W. G. Bergen and G. A. Leveille, 1978. The effects of fasting on body composition, glucose turnover, enzyme and metabolites in the chicken. *J. Nutr.108: 648-657.*

Brandt, L. W., R. E. Clegg and A. C. Andrews, 1951. The effect of age and degree of maturity on the serum proteins of the chicken. *J. Biol. Chem. 191: 105-111*

Brody, S. 1945. Bioenergetics and Growth. Reinhold Publishing Company, New York

Brody, S. 1926. Growth and Development with special reference to domestic animals. *Res. Bull. Mo. Agric. Exp. St. No. 103, pp40.*

Bukhart, C. A., J. A. Cherry, P. B. Siegel, and H. P. Van Krey, 1983. Genetic selection for growth rate alters hypothalamic satiety mechanisms in chickens. *Poultry Sci. 60: 54-62*

Bush, M and E. E. Smith, 1980. Clinical chemistry and hematology as diagnostic aids in zoological medicine. In The Comparative Pathology of Zoo Animals. (Montali R. J. and Migaki, G. eds): Washington, D. C. Smithsonian Institution Press, pp 615-620.

Butler-Hogg, B. W. 1984. Growth pattern in sheep. Changes in chemical composition of the empty body and its constituent parts during weight loss and compensatory growth. *J. Agric. Sci. (Camb.) 103:17*

Burton, J. H., M. Anderson, and J. T. Reid, 1974. Some biological aspects of starvation. The effect of weight loss and regrowth on body composition in sheep. *Br. J. Nutr. 32:515*

Cabel, M. C. and P. W. Waldroup, 1990. Effect of different nutrient-restriction programs early in life on broiler performance and abdominal fat content. *Poultry. Sci. 69:652-660.*

Cabel, M. C. and P. W. Waldroup, 1988. Comparison of different nutrition restriction programs for abdominal fat reduction in broilers. *Poultry. Sci. 67(Suppl. 1):6 (Abstr.)*

Calabota, D. F., J. A. Cherry, P. B. Siegel and E. M. Gregory, 1983. Lipogenesis and lipolysis in normal and dwarf chickens from lines selected for high and low body weight. *Poultry Sci. 62:1830-1837.*

Calvert, C. C., J. P. McMurtry, R. W. Rosebrough and R. G. Campbell, 1987. Effect of energy level on the compensatory growth response of broilers following early feed restriction. *Poultry Sci. 66: (Suppl.):75*

Cerolini, S. L., G. Cavalchini, A. Baldi and P. Mariani, 1986. Pullets in cage vs floor: effect on laying performance. Pages 800-804. In Proceedings 7th European Poultry Conference, Paris, France.

Chambers, J. R., J. S. Gavora and A. Fortin. 1981. Genetic changes in meat-type chickens in the last twenty years. .

Chamblee, T. N. and G. W. Morgan, 1982. Effects of short term fasting on physiological parameters in broiler chickens. *Poultry Sci. 61:1372 (Abstr.).*

Chandra, M. 1983. Renal and biochemical changes produced in broilers by high protein, high calcium urea containing, and vitamin A deficient diets. *Avian Dis. 28. 1*

Cherry, J. A., I. Nir, D. E. Jones, E. A. Dunnington, Z. Nitsan and P. B. Siegel, 1987. Growth associated traits in parental and F1 populations of chickens under different feeding programs. 1. Ad libitum feeding. *Poultry Sci. 66:1-9*

Cherry, J. A., W. J. Swartworth and P. B. Siegel, 1984. Adipose cellularity studies in commercial broiler chicks. *Poultry Sci. 63: 97-108*

Classen, H. L. and C. Ridell, 1989. Photoperiodic effects on performance and leg abnormalities in broiler chickens. *Poultry. Sci. 68:873-879.*

Coleman, S. W., B. C. Evans and J. J. Guenther, 1993. Body and carcass composition of Angus and Charlois steers as affected by age and nutrition. *J. Anim. Sci. 71:86-95*

Crompton, D. W. T. and D. E. Walters, 1979. A study of the groth of the alimentary tract of the young cockerel. *Br. Poultry Sci. 20:149-158*

Davidson, I. and J. B. Henry, 1979. Clinical Diagnosis by Laboratory Methods. W. B. Saunders Company, Philaldephia, PA, USA.

Davidson, J., J. Mathieson, R. B. Williams and A. W. Boyne, 1964. Effects of animal fat and low ratios of protein to metabolizable energy of the utilization of dietary energy by medium and fast growing strains of poultry. *J. Sci. Fd. Agric., 15:316-325*

Deaton, J. W., F. N. Reece, L. F. Kubena, B. D. Colt, and J. D. May, 1973. The ability of the broiler chicken to compensate for early growth depression. *Poultry Sci. 52:262-265*

Deyhim, F, R. E. Moreng and E. W. Kienholz , 1992. The effect of testosterone propionate on growth of broiler chickens. *Poultry sci. 71:1921-1926*

Dickerson, G. E. 1978. Animal size and efficiency: basic concepts. *Anim. Prod. 27:367-379*

D'mello, J. P. F., T. Acamovic and A. B. Walker, 1985. Nutritive value of jackbeans (*Canavalia ensiformis*) for young chicks. *Tropical Agriculture (Trinidad) 62:145-150.*

Doeschate, R. A. H. M., C. W. Scheele, V. V. A. M. Schreurs, J. D. Van der Klis, 1993. Digestibility studies in broiler chickens: Influence of genotype, age, sex and method of determination. *Br. Poult. Sci. 34:1917-1924*

Dolensek, E. P and U. S. Ottis. 1973. Clinical Pathology in Avian species. *Vet. Cli. North Am. 3:159-163*

Dorner, J. L. 1981. Effects of in vitro hemolysis on equine serum values. Amer. *J. Vet. Res. 42: 1519-1522*

Drew, K. R. and J. T. Reid, 1975. Compensatory growth in immature sheep. I. The effects of weight loss and realimentation on the whole body composition. *J. Agric. Sci.(Camb.)85:193*

Drouillard, J. S., T. J. Klopfenstein, R. A. Britton, M. L. Bauer, S. M. Gramlich, T. J. Wester, and C. L. Ferrell, 1991. Growth, body composition, and visceral organ mass and metabolism in lambs during and after metabolizable protein and energy restrictions. *J. Anim. Sci. 69:3357*

Duff, S. R. I. and B. H. Thorp, 1988. Further studies on collateral ligaments of knee and intertarsal joints in domestic fowl. *J. Comp. Pathology 98:461-469*

Evans, R and R. Scholz, 1971. Metabollic responses of chicks during adaptation to a high protein carbohydrate free diet. *J. Nutr. 101:127-1143*

Fancher, B and L. S. Jensen, 1989. Influence on performance of three to six week old broilers of varying dietary protein contents with supplementation of essential amino acid requirements. *Poultry Sci.68:113-123*

Farr, A. J., J. A. Herbert and W. A. Johnson, 1977. Studies on the effects of dietary energy levels and commercial broiler strains on live bird, dry carcass and abdominal fat weights. *Poultry Sci. 56:1713*

Ferket, P. R. ans J. L. Sell, 1989. Effect of early protein restriction on large turkey toms. I. Performance characteristics and leg weakness. *Poultry Sci. 68: 676-686.*

Fisher, C. 1984. Fat deposition in broilers. Pages 437-470 in: Fats in animal nutrition. J. Wiseman, ed. Proc. Easter School in Agricultural Science, University of Nottingham (37th), Butterworth, London, UK.

Fontana, E. A. ,W. D. Weaver, Jr, B. A. Watkins and D. M. Denbow, 1992. Effect of feed restriction on growth, feed conversion and mortality on broiler chickens. *Poultry. Sci. 71:1296-1305*

Forbes, J. M. Relationships between feed intake, energy balance and adiposity. In Leanness in Domestic Birds (Leclercq, B and Whitehead, C. C. ed). pp97-108. Butterworths

Forsum, E., P. E. Hillman, and M. C. Nesheim, 1981. Effect of energy restriction on total heat production, basal metabolic rate and dynamic action of food in rats. *J. Nutr. 111: 1691-1697*

Francis, C. A., M. G. Mc Leod and J. E. M. Anderson, 1991. Alleviation of acute heat stress by food withdrawal or darkness. *Br. Poult. Sci. 32:219-225*

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

Galvin, C. Laboratory diagnostivc aids in pet bird practice. Proceedings of the American Animal Hospital Association, South Bend, IN. p41.

Graham, N. McC, and T. W. Searle, 1975. Studies of weaned sheep during and after a period of weight stasis. I. Energy and nitrogen utilization. *Austr. J. Agric Res. 26:343-353*

Griffiths, L. , S. Leeson and J. D. Summers, 1978. Studies on abdominal fat with four commercial strains of male broiler chicken. *Poultry Sci. 57:1198-1203.*

Griffiths, L., S. Leeson and J. D. Summers, 1977. Fat deposition in broilers: effect of dietary energy to protein balance, and early life caloric restriction on productive performance and abdominal fat pad size. *Poultry. Sci. 56: 638-646*

Grimminger, P and C. G. Scanes, 1986. Protein Metabolism. In Avian Physiology, ed P. Sturkie. p326. Springer-Verlag, New York Inc. New York.

Gutierrez, J. M., D. Alvarez, F. Diaz and S. Fernandez, 1991. Influence of nitrogen balance and intestinal glucose absorption on growth of rats recovery from early undernutrition. *Nutr. Res.11:1427-1437*

Gyles, R. 1989. Poultry, People and Progress. *Poultry. Sci.68:1-8*

Haakansson, J and S. Eriksson, 1978. The influence of feed energy level on feed consumption, growth and development of different organs of chicks. Swedish University of Agricultural Sciences, *Report, 59: 1-54*

Hanlen, E. T. 1939. Some observations on the normal variations in composition of Light Sussex cockerels. *J. Agric. Sci. (Camb.) 28:379-392*

Hahn, G. L., 1982. Compensatory growth in livestock: Influences on environmental Criteria. Proc. of the 2nd Intl.. Livestock environment Symp.:285-294 ASAE SP-03-82, Amer. Soc. Agric. Engin., St Joseph, MI.

Harris, P. M. and E. M. Widdowson, 1978. Deposition of fat in the body of the rat during rehabilitation after early undernutrtition. *Br. J. Nutr. 39: 201-211.*

Haustein, A. T., R. H. Girman, P. W. Skillicorn, V. Guevara, F. Diaz, V. Vergara, A. Gastanaduy and J. B. Gilman, 1992. Compensatory growth in broiler chicks fed on Lemna gibba. *Br. J. Nutr.68:329-335*

Haye, U., and P. C. M. Simons, 1978. Twisted legs in broilers. *Br. Poult. Sci. 19:549-557*

Hazelwood, R. L. 1986 Carbohydrate metabolism. In Avian Physiology, ed. P. N. Sturkie, Cornell University Press Ithaca, New York, p313.

Hentges, E. J., D. N. Maple, D. A. Roland, Sr and J. P. Pritchett, 1983. Growth and in vitro protein synthesis in two strains of chickens. *J. Anim. Sci. 57:320-327*

Hermier, D.; M. J. Chapman and B. Leclercq, 1984. Plasma lipoprotein profile in fasted and refed chickens of two strains selected for high or low adiposity. *J. Nutr. 114: 1112-1121.*

Hester, P, K. K. Kruger, M. Jackson, 1990. The effect of compensatory growth on carcass characteristics ofmale turkeys. *Poultry Sci. 69:1743-1748.*

Hill, K. J. 1971. the physiology of digestion. In physiology and Biochemistry of the domestic fowl Vol. 1 eds D. J. Bell and B. M. freeman. pp25-49 Academic Press, London.

Hocking, P.M., M. H. Maxwell and M. A. Mitchell, 1993. Welfare assessment of broiler breeder and layer females subjected to food restriction and limited access to water during rearing. *Br. Poult. Sci. 34:443-458*

Hocking, P. M., D. Waddington, M. A. Walker and A. B. Gilbert, 1989. Control of the development of the follicular hierachy in broiler breeder pullets by food restriction during rearing. *Br. Poult. SCi. 30: 161-174*

Holsheimer, J. P. and C. H. Veerkamp, C. H. 1992. Effect of dietary energy, protein and lysine content on performance and yields of two strains of male broiler chicks. *Poultry Sci. 71:872-879*

Hood, R. L. 1982. The cellular basis for growth of the abdominal fat pad in broiler-type chickens. *Poultry. Sci. 61: 117-121*

Horst, K., L. B. Mendel and F. G. Benedict, 1934. The influence of previous diet, growth and age upon basal metabolism of the rat. *J. Nutr. 8: 139-162*

Houpt, T. R., 1958. Effects of fasting on blood sugar levels in baby chicks of varying ages. *Poultry Sci. 37:1452-1459*

Hulan, H. W., F. G. Proudfoot, D. Ramey and K. B. McRae, 1980. Influence of genotype and diet on general performance of chickens reared to roaster weight. *Poultry Sci 59:748-757*

International Federation of Clinical Chemistry, 1978. The theory of reference values. Part 1. The concept of reference values. *Clin. Chim. Acta 87:459F-475F.*

Jackson, A. A. 1990. Protein requirements for catch-up growth. *Proc. Nutr. Soc. 49:507-516.*

Jackson, S., J. D. Summers and S. Leeson, 1982. Effect of dietary protein and energy on broiler carcass composition and efficiency of nutrient utilization. *Poultry Sci.61:2224-2231*

Jones, G. P. D. and D. J. Farrell, 1992a. .Early life feed restriction of broiler chickens I. Methods of application, amino acid supplementation and the age at which restrictions should commence. *Br. Poultry. Sci. 33:579-587*

Jones, G. P. D. and D. J. Farrell, 1992b. Early life food restriction of broiler chickens. II. Effects of food restrictions on the development of fat tissue. *Br. Poult. Sci. 33:589-601*

Jones , G. P. D. and D. J. Farrell, 1989. Reducing body fat in broilers and some physiological consequences. *South African Journal of Animal Science. 19:179-183*

Jones, G. P. D., A. Yang and D. J. Farrell, 1989. Advances in reducing body fat in broilers by nutritional and chemical means. In Recent Advances in Animal Nutrition in Australia 1989. Farrell, D. J. ed p332.

Jones, G. P. D. and D. J. Farrell, 1987. Reducing body fat in broilers: preliminary results. Recent advances in Animal Nutrition in Australia 1987. D. J. Farrell, ed. pp211-221

Jorgensen, H., P. Sorensen and S. A. Svensson, 1990. Protein and energy metabolism in broiler chickens selected for either body weight gain or feed efficiency. *Br. Poultry Sci. 31: 517-524*

Just, A. I. 1984. Nutritional manipulation and interpretation of body compositional differences in growing swine. *J. Anim. Sci. 58:740*

Kabbali, A., W. L. Johnson, D. W. Johnson, R. D. Goodrich and C. E. Allen, 1992. Effects of undernutrition and refeeding on weights of body parts and chemical components of growing Moroccan Lambs. *J. Anim. Sci. 70:2859-2865*

Kaneko, J. J., 1989a. Carbohydrate Metabolism and its Diseases. In: Clinical Biochemistry of Domesticv Animals, 4th Edition. pp45-81 Academic Press.

Kaneko, J. J., 1989b. Serum Proteins and the Dysproteinemias. In Clinical Biochemistry of Domesticv Animals, 4th Edition. pp143-163 Academic Press.

Kang, C. W., M. L. Sunde, and R. W. Swick, 1985. Growth and protein turnover in the skeletal muscles of broiler chicks. *Poultry Sci. 64: 370-379*

Katanbaf, M. N., E. A. dunnington and P. B. siegel, 1989. Restricted Feeding in Early and Late Feathering chickens. 3. Organ Size and Carcass Composition. *Poultry Sci. 68: 359-368.*

Kartashov, N. I., V. V. Lupashko, N. Batyuzheskii and N.G. Maneilo, 1985. Oxidative phosphorylation in liver mitochondria of line B7 feed-restricted hens. *Sbornik-Nauchnykh-trudov-Khar'kovskogo-sel'skokhozyaistvennogo-Instituta 316: 49-54*

Koong, L. J., C. L. Ferrell and J. A. Nienaber, 1985. Asessment of interrelationships among levels of intake and production, organ size and fasting heat production in growing animals. *J. Nutr. 115:1383.*

Kubena, L. F., B. D. Lott, 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*

Kumar, S and Y. K. Gupta, 1981. Seasonal and experimental studies on blood glucose of *Psittacula kramelli. Pavo. 19: 1-10*

Lacy, M. P., H. P. Van Krey, P. A. Slaves and D. M. Denbow, 1986. Tryptophan's influence on feeding and body temperature in the fowl. *Poultry Sci. 65: 1193-1196*

Langslow, D. R., E. J. Butler, C. N. Hales and A. W. Pearson, 1970. The response of plasma insulin, glucose and non-esterified fatty acids to various hormones, nutrients and drugs in the domestic fowl. *J. Endocrinology. 46:243*

Leathwood, P. D., 1987. Tryptophan availability and serotonin synthesis. *Proc. Nutr. Soc. 46:143-156.*

Leclercq, B. 1986. Energy requirements of avian species. In Nutrientrequirements of poultry and nutritional research. Fisher, C and Boorman, K. N eds). pp125-141, Butterworths, London.

Ledin, A. 1983. Compensatory growth and carcass composition in lamb and rabbits. Effect of energy concentration, restricted feeding and realimentation. Report 120. Sculleries Lantbruksuniversitet. Dissertation. Swedish University of Agricultural Sciences, Uppsala, Sweden.

Leenstra, F. R. and R. Pit, 1988. Consequences of selection for feed conversion in broiler chicks, In : Korver, S, H. A. M. Van der Steen and J. A. M. Van Arendonk (eds). Advances in Animal Breeding: Proceedings of the World Symposium in Honour of Professor R. D. Politiek, pp 160-161, Wageningen, Pudoc).

Leeson, S. 1990. Broiler growth patterns: the implications of differential growth of broiler chickens. Part 2: consideration of carcass quality. *Canada Poultry-man (Feb.), pp25-26*

Leeson, S, J. D. Summers and L. J. Caston, 1992. Response of broilers to feed restriction or diet dilution in the finisher period. Poultry. Sci. 71:2056-2064

Lepkovsky, S and F. Furuta, 1971. The Role of Homeostasis in Adipose Tissue upon the Regulation of Food Intake of White Leghorn Cockerels. *Poultry Sci. 573-577*

Leveille, G. A. 1979. In vivo fatty acid and cholesterol synthesis in fasted and fasted-refed chicks. *J. Nutr. 98:367-372.*

Leveille, G. A. and H. E. Sauberlich, 1961. Influence of dietary protein level on serum protein components and cholesterol in the growing chick. *J. Nutr. 74: 500-504*

Lewandowski, A., T. W. Campbell and G. J. Harrison, 1986. Clinical chemistries. In Clinical Avian Medicine and Surgery, Harrison and Harrison, Eds. W. B. and Sanders Co.

Lilburn, M. S.,T. J. Lauterio, K. Ngiam-Rilling, and J. H. Smith, 1989. Relationships among mineral balance in the diet, early growth manipulation, and incidence of tibial dyschondroplasia in different strains of meat type chickens. *Poultry Sci. 68:1263-1273*

Lilburn, M. S., R. M. Leach, Jun, E. G. Buss and R. J. Martin, 1982. The development chatacteristics of two strains of chickens selected for differences in mature abdominal fat pad size. *Growth, 46:171-181*

Lipstein, B., S. Bornstein and I. Bartov, 1975. The replacement of some of the soyabean meal by the first limiting amino acifds in practical broiler diets 3. effects of protein concentrations and amino acid supplementation in broiler finisher diets on fat deposition in the carcass. *Br. Poult. Sci. 16:627-635*

Little , D. A. and R. L. Sandland, 1975. Studies on the distribution of body fat in sheep during continuos growth and following nutritional restriction and rehabilitation. *Aust. J. Agric. Res. 26:363*

Littlefield, L. H., 1972. Strain differences in quantity of abdominal fat in broilers. *Poultry Sci. 51:1829*

Mallard, J and M. Douaire, 1988. Strategies of selection for leanness inmeat production. In Leanness in Domestic Birds: Genetic, Metabolic and Hormonal Aspects. B. Leclercq and C. C. Whitehead, Eds.pp3-23, Butterworths, London.

Marais, P. G., H. J. van der Merwe, J. E. du Toit, 1991. The effect of compensatory growth on feed intake, growth rate, body composition and efficiency of feed utilization in Dorper sheep. *South African J. Anim. Sci. 21:80-88*

March, B. E. and G. Hansen, 1977. Lipid accumulation and cell multiplication in adipose bodies in White Leghorn and broiler-type chicks. *Poultry Sci. 61:1137-1146*

Marks, H. L. 1979. Growth rate and feed intake of selected and non selected broilers. *Growth 43:80-90*

Maynard, L. A. 1947. Animal Nutrition. 2nd edition. New York. McGraw Hill.

McCartney, M. G. and H. B. Brown, 1976. The effects of feed restriction time on the growth and feed consumption of broiler males. *Poultry Sci. 56:713-715*

McCormick, J. F. and D. M. Denbow, 1987. The effects of opioid antagonists on ingestive behavior in the domestic fowl. *Pharmaco. Biochemi. Behav. 27:25-33*

McDonald, P; R. A. Edwards and J. F. D. Greenhalgh, 1988. Animal Nutrition. 4th Edition. Longman Scientific and Technical

McMeekan, C. P. 1940. Growth and Development in the Pig, with special reference to carcass quality characteristics. III. Effect of Plane of nutrition on the form and composition of the bacon pig. *J. Agric. Sci.(Camb.) 30:511*

McMurtry, J. P., R. Rosebrough, I. Plavnik and A. Cartridge, 1988. Influence of early plane of nutrition on enzyme systems and subsequent tissue deposition. in :G. L. Steffens and D. S. Rhymes (eds) :Biomechanisms regulating growth and development. ed Belleville Symposia on Agric. Res. 12, The Netherlands.

McNab, J. M. and D. W. F. Shannon 1972. Studies of the process of digestion in the fowl: dry matter and total nitrogen. *Br. Poultry Sci. 13: 495-502*

Mellen, W. J., F. W. Hill and H. H. Dukes, 1954. Studies on the energy requirements of chickens. Effect of dietary energy level on the basal metabolism. *Poultry Sci. 33:791*

Meluzzi, A., G. Primiceri, R. Giordani and G. Fabris, 1992. Determination of Blood Constituents Reference Values in Broilers. *Poultry Sci. 71:337-345*

Mersmann, H. J., M. D. mcNeil, S. C. Seidman and W. G. Pond, 1987. Compensatory growth in finishing pigs after feed restriction. *J. Anim. Sci. 64: 752-764*

Milliward, D. J., D. O. Nanyelugo, W. P. T. James, and P. J. garlick, 1974. Protein metabolism in skeletal muscle: the effect of feeding and fasting on muscle RNA, free amino acids and plasma insulin concentrations. *Br. J. Nutr. 32:127-142*

Mitchell, M. A., P. J. Kettlewell and M. H. Maxwell, 1992. Indicators of physiological stress in broiler chickens during road transportation. *Animal Welfare 1:91-103*

Mirtuka, B. M. and H. M. Rawnsley, 1981. Clinical, biochemical and hematological reference values in normal experimental animals and normal humans. 2nd edition

Mollison, B., W. Guenter, and B. R. Boycott, 1984. Abdominal fat deposition and sudden death syndrome in broilers: effects of restricted intake, early life calorie (fat) restriction, and calorie:protein ratio. *Poultry Sci. 63:1190-1200*

Morgan, G. W. Jr and B. Glick, 1972. A quantitative study of serum proteins in bursectomised and irradiated chickens. *Poultry Sci. 51: 771-778*

Moran, E. T., Jr. 1979. Carcass quality changes with the broiler chickens after dietary protein restriction during the growing phase and finishing period compensatory growth. *Poultry Sci. 58:1257-1270*

Mosier, H. D. Jr. 1986. The control of catch-up growth. *Acta Endocrinol. 113:1-7*

Newcombe, M, S. H. Fitz-Coy and J. M. Harter-Dennis, 1992. The effect of feed restriction and *Eimeria maxima* infection with or without medication on growth and feed intake in broilers. *Poultry Sci. 71:1442-1449*

Nir, I, N. Shapira, Z. Nitsan and Y. Dror, 1974. Force feeding effects on growth, carcass and blood composition in th young chick. *Br. J. Nutr. 32: 229-239*

O'Hea, E. K. and G. A. Leveille, 1969a. Lipid biosynthesis and transport in domestic chick (*Gallus domesticus*). *Comp. Biochem. Physiol. 30:149-159*

O'Hea, E. K. and G. A. Leveille, 1969b. Influence of fasting and refeeding on lipogenesis and enzymatic activity of pid adipose tissue. *J. Nutr. 99:345-352*

Ologhobo, A. D., D. F. Apata and A. Oyejide, 1993. Utilization of raw jackbean (*Canavalia ensirformis*) and jackbean fractions in diets for broiler chicks. *Br. Poult. Sci. 34:323-338*

Osbourn, T. B and L. B. Mendel, 1916 Acceleration of growth after retardation. *Amer. J. Physiology. 40:16*

Osbourn, T. B and L. B. Mendel, 1915. The ressumption of growth after long continuous failure to grow. *J. Biol. Chemistry 23:439-454*

Osbourn, D. F. and P. N. Wilson, 1960. Effects of different patterns of allocation of a feed restricted quantity of food upon the growth and development of cockerels. *J. Agric. Sci. 54: 278-289*

during and
O'Sullivan, N. P., E. A. Dunnington and P. B. Si 18-355
characteristics of early and late featherin
feeding regimes. *Poultry. Sci. 70: 1323-1*

in
of
Ozelci, A., D. R. Romsos and G. A. Leveille, .
restriction on subsequent body weight gain and bu
rats. J. *Nutr. 108:1724-1732.*

Palsson, H. 1955. Conformation and body composition. Progress in the physiology of Farm Animals. (J. Hammond, ed). Butterworths Publications, London

Palsson, H and J. Verges, 1952. Effects of the plane of nutrition on growth and the development of carcass quality in lambs. *J. Agric. Sci. 42:1*

Parrilla, E. T. 1984. Performance of meat type ducklings on different levels of feed restriction. *Annals Trop.Rres. 6:42-52.*

Pasternak, H and B. A. Shalev, 1983. Genetic-economic evaluations of traits in a broiler enterprise:reduction of food intake due to increased growth rate. *Br. Poultry. Sci. 24: 531-536.*

Pfaff, F. E. and R. E. Austic, 1976. Influence of diet on adipose tissue accumulation in the pullet. In Proc. 1974 Cornell Nutr. Conf. pages 82-86.

Pinchasov, Y and L. S. Jensen, 1989. Comparison of physical and chemical means of feed restriction in broiler chicks. *Poultry Sci. 68:61-69*

Plavnik, I and S. Hurwitz, 1991. Response of broiler chickens and turkey poults to food restriction of varied severity during early life. *Br. Poult. Sci.68:1118-1125*

Plavnik, I and S. Hurwitz, 1990. Performance of broiler chickens and turkey toms subjected to feed restriction or to feeding of low protein or low sodium diets at an early age. *Poultry Sci. 69: 945-952*

Plavnik, I and S. Hurwitz, 1988. Early feed restriction in chicks: effect of age, duration and sex. *Poultry Sci. 67:383-390*

Plavnik, I, J. P. McMurtry and R. W. Rosebrough, 1986. Effects of early feed restriction in broilers. 1. Growth performance and carcass composition. *Growth 50:68-76.*

Plavnik, I and S. Hurwitz, 1985. The performance of broilers chicks during and following severe feed restriction at an early age. *Poultry Sci. 64:348-355*

Plavnik, I and S. Hurwitz, 1983. Organ weights and body composition in chickens as related to energy and amino acid requirements: effects of strain, sex and age. *Poultry Sci. 62:152-163.*

Pokniak, J. A,, M. S. Avaria and S. B. Cornejo, 1984. Productive performance and changes in carcass composition of broilers under an initial energy protein restriction and subsequent refeeding. *Nutr. Rep. Int. 30:1377-1383*

Pokniak, J. A., and S. B. Cornejo, 1982. Effects of energy and protein undernutrition on productive performance and carcass, liver and digestive tract composition of broiler males (1). *Nutr. Rep. Int. 26:319-327.*

Pomeroy, R. W. 1955. The effect of a sub-maintenance diet on the composition of the pig. *J. Agric. Sci. 31:50-73*

Proudfoot, F. G., H. W. Hulan, and K. B. McRae, 1983. The effect of feed denial in starter diets on the performance of broiler chickens. *Poultry Sci. 62:1915-1917*

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

Pym, R. A. E. and J. F. Dillon, 1974. Restricted food intake and reproductive performance of broiler breeder pullets. *Br. Poult. Sci. 15: 245-259*

Quarles, C. L., T. W. Burr, J. H. MacNeil and G. O. Bressler, 1968. The effects of varying levels of hydrolysed animal and vegetable fat upon growth and carcass characteristics of broilers. *Poultry Sci. 47: 1764*

Ragsdale, A. C. 1934. Growth standards for Dairy Cattle. *Res. Bull. Mo. Agric. Exp. St. No.336. pp12*

Read, J. D. and O. T. White, 1977. The phenomenon of compensatory growth. Proc. Cornell Nutr. Conf. 16-27

Ridell, C,. 1983. Pathology of skeleton and tendons of broiler chickens reared to roaster weights. I. Crippled chickens. *Avian Dis. 27:950-962*

Ridell, C,. M. W. King and K. R. Gunasekera, 1983. Pathology of skeleton and tendons of broiler chickens reared to roaster weights. II. Normal chickens. *Avian Dis. 27:980-991*

Robinson, F. E., H. L. Classen, J. A. Hanson and D. K. Onderka, 1989. Short term feed restriction for broiler and roaster chickens. 68th Annual Feeders day Report, pp45-46 , University of Alberta, Alberta, Canada.

Robinson, F. E., H. L. Classen, J. A. Hansen and D. K. Onderka, 1992. Growth performance, feed efficiency and the incidence of skeletal and metabolic disease in full-fed and feed restricted broiler and roaster chickens. *J. Appl. Poult. Res. 1:33-41*

Rosebrough, R. W. N. C. Steele, J. P. McMurtry and I. Plavnik, 1986. Effect of early feed restriction in broilers. II. Lipid Metabolism. *Growth 50:217-227*

Ross, J. G., G. Christie, W. G. Halliday and R. M.Jones, 1978. Haematological and blood chemistries "comparision vaues" for clinical pathology in poultry. *Vet. Rec. 102:29-31*

Rosskopf, W. J. and R. W. Woerpel, 1984. Clinical experience with avian laboratory diagnostics. *Vet. Clin. North Amer. (Small Anim. Prac.) 14: 2*

Safalaoh, A. C. L. 1992. A Review of Poultry Production in Malawi: Constraints and Possible Solutions. *Tizame 3: 15-22* (A Malawi National Commission for UNESCO Publication, Lilongwe, Malawi)

Sanz, M, M. Smith and M. Hernandez, 1985. Comparative study of different feeding systems of heavy breeders. Effect of stage of growth. *Revista-Avicultura. 29:241-250*

SAS Institute, 1985. SAS® User's Guide. Statistics. 1985 Edition. SAS Institute, Inc. Cary, North Carolina.

Scheideler, S. F. and G. R. Baughman, 1993. Computerized early feed restriction program for various strains of broilers. *Poultry. Sci 72:236-242*

Schnyder, W, H. Bickel and A. Schurch, 1982. Energy metabolism during retarded and compensatory growth of Braunvieh steers. In Proceedings, 9th Symposium of the European Association for Animal Production on Energy Metabolism in Farm Animals, Lillehammer, Norway. A. Ekern and F. Sundstol, eds. pp96-99. Euro. Assoc. Anim. Prod. Public. No. 29

Shapira, N. , I. Nir and P. Budowski, 1978. Response of lipogenic enzymes to overfeeding in liver and adipose tissue of light and heavy breeds of chicks. *Brit. J. Nutr. 39:151-157.*

Sheehy, E. J. and B. J. Senior , 1942. Storing cattle at different levels of nutrition. J. Dep. Agric. *Eire.39:245-262.*

Sherma, M. L., 1986. Effect of feed restriction during the growing period on the subsequent laying performance of White Leghorn pullets. *Indian J. Poult Sci. 21:124-127*

Siegel, H. S. 1968. Adaptation of Poultry. In adaptation of Domestic Animals, ed E. S. E. Hafez, pp292-309, Lea and febiger, Philaldephia, PA, USA.

Simon, J and G. Rosselin, 1979. Effect of intermittent feeding on glucose insulin relationship in the chicken. *J. Nutr. 109: 631-641*

Skinner, T. T., J. N. Beasley and P. W. Waldroup, 1991. Effects of dietary amino acid levels on bone development in broiler chickens. *Poultry Sci. 70:941-946.*

Sorensen, P., A. Chwalibog and B. O. Eggum, 1983. Protein and energy metabolism in two lines of chickens selected for growth on high or low protein diets. *Br. Poultry Sci. 24: 237-250*

Sturkie, P. D. 1976. Avian Physiology. 3rd Ed. Springer-Verlag, New York, NY.

Sturkie, P. D. 1951. *Endocrinology 49: 565.*

Summers, J. D. and S. Leeson, 1986. Influence of nutrient density on feed consumption, weight gain and gut capacity of broilers, leghorns and turkeys reared to 26 days of age. *Anim. Feed Sci . Technol. 16:129-141*

Summers, J. D., D. Spratt and J. L. Atkinson, 1990. Restricted feeding and compensatory growth for broilers. *Poultry Sci. 69:1855-1861*

Talpaz, H, S. Hurwitz, J. R. de la Torre and P. Sharpe, 1988. Economic Optimization of a Growth Trajectory for Broilers. *Poultry Sci. 67: 382-390.*

Teeter, R. G. and T. Belay, 1993. A Report for the Coperative research Project between Church and Dwight Company, Inc and Oklahoma State University. Stilwater, Oklahoma State University.

Teeter, R. G. and M. O. Smith, 1985. Feed intake effects upon gain, yield and ration digestibility in broilers force fed five feed intakes. *. oultry Sci. 64: 2155-2160*

Teeter, R. G., M. O. Smith, S. Sangiah and F. B. Mather. 1987. Effects of feed intake and fasting duration upon body temperature and survival of thermostressed broilers. *Nutr. Rep. Intl. 35:531-537*

Tiez, N. W. 1976. Fundamentals of Clinical Chemistry. W. B. Saunders and Company. Philaldephia, PA, USA.

Thomas, O. P., A. I. Zuckerman, M. Farran and C. B. Tamplin, 1986. Updated amino acid requirements of broilers pp79-83 in: Proceedings of the Maryland Nutrition Conference, University of Maryland, College Park, MD.

Thompson, E. F., H. Bickel, A. Schurch, 1982. Growth performance and metabollic changes in lambs and steers after mild nutritional restriction. *J. Agric. Sci. 98:183-194*

Timmers, K and J. L. Knittle, 1980. Effects of undernutrion and refeeding on enzyme activities and rates of glucose metabolism in rat's epididymal adipose tissue. *J. Nutr. 110:1176-1184*

Voloshchenko, M. V., A. V. Chechetkin and V. A. Luk'yanov, 1985. Functional characteristics of oxidative phosphorylation activity of muscle mitochondria in laying hens during their various stages of development and in relation to nutritional factors. *Sbornik NauchnykhtrdovKhrkovskogolskokhoz yais tvenogo -Instituta 316:54-65*

Wallis, I. R. and D. Balnave, 1984. The influence of environmental temperature, age and sex on thedigestibity of amino acids in growing broiler chickens. *Br. Poultry Sci. 25: 401-407.*

Washburn, K. W. 1991. Efficiency of feed utilization and rate of passage through the digestive system. *Poultry Sci.70: 447-452*

Washburn, K. W. 1990. Effect of restricted feeding on fatness, efficiency, and the relationship between fatness and efficiency in broilers. *Poultry Sci. 69 502-508.*

Washburn, K. W and K Bondari, 1978. Effects of timing and duration of feed restricted feeding on compensatory growth in broilers. Poultry. Sci. 57:1482-1487

Webster, A. J. F., 1980. The energetic efficiency of growth. *Livestock Production Science 7: 243-252.*

Wiernurz, C. J. and R. G. Teeter, 1993. Feeding Effects on Thermobalance during Thermoneutral and high Ambient Temperature Exposure. *Poultry Sci. 72:1917-1924*

Wilson, P. N. and Osbourn, D. F. 1960. Compensatory growth after undernutrition in mammals and birds. *Biol. Rev. 35:324-363.*

Wilson, P. N. 1960. Effect of plane of nutrition on growth and development of East Africa dwarf goats. III : The effect of plane of nutrition and sex on the carcass composition of the kid at two stages of growth- 10 lb liveweight and 30 lb liveweight. J. *Agric. Sci. 54:105-134.*

Winchester, G. F. and P. E. Howe, 1954. Some effects on beef cattle of interrupted growth. I. Results of the 1950-1951 identical twin steer trials. USDA res. Service.

Yu, M. W. and F. E. Robinson, 1992. The application of short term feed restriction in broiler chicken production. Albers, G, Barranon, Zurita and Ortiz, 1990. Correct feeding prevents ascites. *Poultry Misset 6:22-23*

Yu, M. W., F. E. Robinson, M. T. Clandinin and L. Bodnar, 1990. Growth and body composition of broiler chickens in response to diferent regimens of feed restriction. *Poultry Sci:1:147-154*

Zubair, A. K. and S. Leeson, 1994. Effect of varying period of early nutrient restriction on growth compensation and carcass characteristics of male broilers. *Poultry Sci. 73: 129-136*

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