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Effect of early nutrient restriction on performance and development of selected characteristics of gastrointestinal tract of broiler chickens

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Effect of early nutrient restriction on performance and development of selected characteristics of gastrointestinal tract of broiler chickens

by

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For the Graduate College

Iowa State University
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1994
DEDICATION

This dissertation is dedicated to the memory of my parents, Charles and Angele. Without their guidance, love, and support early in my life I would not have made it to this point today.
TABLE OF CONTENTS

ACKNOWLEDGMENTS

GENERAL INTRODUCTION

  Dissertation Organization

LITERATURE REVIEW

  Posthatching Growth and Organ Development in Birds
  Growth Patterns of Broiler Chickens
  Feed Restriction to Alter Growth Pattern
  Feed Restriction with Broilers
  Feed Restriction and Catch-up Growth
  Factors that Influence an Animal's Ability to Recover
  From the Effects of Undernutrition
  Mechanisms Involved in Catch-up Growth
  Control and Regulation of Catch-up Growth

References Cited

EFFECT OF EARLY NUTRIENT RESTRICTION ON BROILER CHICKENS.
1. PERFORMANCE AND DEVELOPMENT OF THE GASTROINTESTINAL TRACT

  ABSTRACT

  INTRODUCTION

  MATERIALS AND METHODS

  RESULTS

  DISCUSSION

  REFERENCES
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vi

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GENERAL INTRODUCTION

When an animal, whose growth has been retarded by dietary restriction, is given adequate nutrition it grows at a faster rate than an animal of the same age that had no restriction (Wilson and Osbourn, 1960). This rapid growth relative to age has been termed "compensatory growth" (Bohman, 1955) or "catch-up growth" (Prader et al., 1963).

Catch-up growth has been extensively researched in farm animals (e.g., cattle, sheep, pigs) with the objective of improving animal production efficiency. Among poultry species, market turkeys may benefit most from the exploitation of the catch-up growth phenomenon primarily because they are marketed at an older chronological age than broiler chickens, allowing more time for the recovery of a growth deficit. In fact, the application of feed restriction in the production of meat type (broiler) chickens is controversial. Feed restriction programs applied to broiler chickens have produced varied responses with respect to body weight, feed efficiency, and carcass fat of the feed-restricted chickens as compared with full-fed chickens. The reasons, however, for the relative success or failure to observe catch-up growth in broilers are still unknown.

The phenomenon of catch-up growth in broiler chickens remains complex because the physiological, nutritional, metabolic, and endocrine aspects involved are not well understood. For instance,
the gastrointestinal tract (GIT) has a major role in supporting growth during the early posthatching period, yet its role as a supply-organ system has not been extensively investigated in feed restriction studies with broilers.

The research reported in this dissertation examines the effect of early nutrient restriction on performance and development of selected characteristics of gastrointestinal tract of broiler chickens. Two experiments were conducted to address that subject. Experiment 1 was conducted to gain information on the effect of early nutrient restriction on the performance and carcass composition of broiler chickens. The second objective was to investigate the influence of early nutrient restriction and subsequent realimentation on selected characteristics (physical and chemical) of the GIT components. Experiment 2 was designed to verify whether the results obtained in Experiment 1 could be repeated with respect to performance and to the physical characteristics of GIT components. Two early feed restriction programs were evaluated in Experiment 2. Furthermore, the effect of early nutrient restriction and subsequent realimentation on activities of selected digestive enzymes was also investigated.

Dissertation Organization

The dissertation is divided into a Literature Review followed by two manuscripts and the General Conclusions.
The first manuscript has been submitted for publication to *Poultry Science* under the authorship of P. E. Palo, J. L. Sell, F. J. Piquer, M. F. Soto-Salanova and L. Vilaseca. The second manuscript will be submitted for publication to *Poultry Science* under the authorship of P. E. Palo, J. L. Sell, F. J. Piquer, L. Vilaseca and M. F. Soto-Salanova. P. E. Palo is the senior author of the two manuscripts.
Posthatching Growth and Organ Development in Birds

Growth and development of the body and its parts have been investigated with numerous animals including birds (Huxley, 1932; Laird, 1955; Lilja, 1983). In his theory of "differential growth" Hammond (1932) set forth the viewpoints that the body proportions and conformation result from differential growth gradients between the body tissues and parts and that these occur in a definite order. Chronologically the various tissues reach their maximum growth rate and mature in the following order: nervous tissue, bone, muscle, and fat. Later, Brody (1945) reviewed the findings of many workers who showed that most organisms undergo changes of form due to differential growth rates of the different organs and tissues. Recently, Lilja (1983) hypothesized and presented evidence to support the hypothesis that the rate at which posthatch growth of birds proceeds was partly determined by the distribution of growth among various organs. Later, Lilja et al. (1985) and more recently Katanbaf et al. (1988a) confirmed this hypothesis.

Growth, essentially, is a quantitative phenomenon and one of its most important properties is its rate (Lilja, 1983). The rate of growth, however, has been found to vary considerably among species. Bjornhag et al. (1979) introduced the growth rate factor which expresses the growth rate capacity of each species when the
influence of size is eliminated. Thus, by means of the growth rate factors it is possible to compare the growth of species with widely different birth weights. Indeed, Bjornhag et al. (1979) showed that birds in general grow twice as fast as mammals of equal size. Moreover, average growth rate was about four times greater in altricial than in precocial species (Ricklefs, 1985).

Growth can also be considered as an investment of primarily protein and energy. These investments create costs for maintenance. During growth, the various organs of the body can be divided into two main groups: "consuming" and "supplying" organs, according to their function (Lilja, 1983). It appeared that individuals with high growth rates (e.g. altricial avian species) were those that distributed a large share of their early growth (duration varied among species) into rapid development of "supply" organs such as the gastrointestinal tract and liver (Bjornhag et al., 1979; Lilja, 1983). Such a large early investment into these organs may have been at the expense of growth directed to "demand" organs such as muscles and feathers. Moreover, by the time this stage of development was completed, individuals with higher growth rates appeared to have a better developed digestive tract and less maintenance requirement due to less "demand" tissue than those with lower rates and hence may have better provided the rest of the body with energy during the accelerating phase of growth.
Studies on allometric growth of domestic poultry have been conducted by several researchers. Most of these studies concentrated on the development of the gastrointestinal tract (GIT) during the posthatching growth period. In all of the reports it was shown that the GIT has a major role in inducing growth during the early posthatching period. During this period, segments of the GIT increase in size and weight more rapidly in relation to body weight than other organs and tissues of chickens (Lilja, 1983; Katanbaf et al., 1988a), quail (Lilja et al., 1985), pouls (Sell et al., 1991), and certain other poultry species (Lilja, 1983). Konarzewski et al. (1990) pointed out that during the early posthatch development the assimilated energy is allocated primarily for growth of the digestive tract at the expense of all other body parts. Moreover, Katanbaf et al. (1988a) stated that the accelerated growth of the GIT of chickens immediately after hatching demonstrated the importance of organs and tissues that fulfill a supply function for achieving early body development. Indeed, each species of bird has to find an intermediate balance where the gastrointestinal tract achieves, in a short time, maximal growth in size and function without compromising body growth.
Growth Patterns of Broiler Chickens

Mathematical models have been developed to describe the posthatch growth of the whole broiler chicken in terms of its accumulation of live weight gain. Indeed, the use of equations to describe complete growth curves allow a more complete comparison of differences in growth between strains since they provide a means for visualizing the overall pattern of growth over time (Wilson, 1977). Moreover, these growth models can be used to predict the expected average weight of a group of birds at any given age within the limits of the model. Examples of nonlinear functions developed as models of poultry growth are the Gompertz, the logistic and the Von Bertalanffy models (Tzeng and Becker, 1981).

Wilson (1977) indicated that these nonlinear models are applicable to avian species and are developed under the assumption that birds fed ad libitum are capable of maximum growth. Tzeng and Becker (1981) found that the Gompertz equation was the best fit for cumulative body weight on a daily basis in male broilers between 1 and 69 days of age. The actual pattern of growth for both sexes of two modern strains of chickens fed ad libitum is shown in Figure 1. The growth curves shown (Figure 1) are sigmoid and have the following characteristics: an accelerating phase of growth from hatching, a point of inflexion in the growth curve at which growth rate is maximum, a phase where growth rate is decelerating and a
Figure 1. Growth of male and female Ross and Apollo chickens when fed ad libitum (Wilson, 1977).
limiting value (asymptote) mature weight, towards which the growth curve tends. These characteristics are also shown diagrammatically in Figure 2. Wilson (1977) pointed out that the phenomenon of sigmoid growth, however, is not peculiar to chickens but is exhibited by other poultry species, other animals, and plants.

Growth rate is one of the most important economic traits in a broiler enterprise mainly due to the reduction in feed consumption resulting from shortening the growth period (Pasternak and Shalev, 1983). Pasternak and Shalev (1983) suggested that broiler chickens having a concave-shaped growth curve (growing slowly initially and faster later) need less overall feed than those exhibiting a convex-shaped growing curve (growing faster initially and slowly later). Indeed, several researchers have indicated that intense selection pressure for body weight and growth rate in broilers has shifted the growth curve back so birds reach marketable weights at earlier ages (Marks 1979, Zelenka et al., 1986; Siegel and Dunnington, 1987). Moreover, information regarding the changes in growth patterns accompanying selection for early growth rate was reported by Marks (1979). The most dramatic of the differences in relative growth rate and feed efficiency between selected and non selected broiler populations were manifested primarily in the first 4 weeks, after which differences between the populations were not significant. Thus, the trend followed by today's commercial
Figure 2. Data, from Ross male broilers, fitted with a Gompertz function and illustrating the properties of a sigmoid growth curve (Wilson, 1977).
broilers is a convex-shaped growth curve, since they show a more rapid initial growth and subsequently a slower growth approaching market weight.

Feed Restriction to Alter Growth Pattern

Figure 3 is a schematic representation of various growth curves of broiler chickens (Leeson and Summers, 1991). Lines A, B, and C represent three potential growth curves of broilers that reach approximately 2 kg (X) at 42 days of age, yet the routes they take are dissimilar. If birds grow at a uniform rate, growth will be depicted by line B, and represents perhaps a biological ideal as far as minimizing stress is concerned, i.e. continual steady growth with no major periods of slow or rapid growth. Bird A has more rapid initial growth and subsequently, a slower growth approaching market weight. Bird C initially has a slower rate of growth followed by an accelerated growth towards market weight. Bird C will likely exhibit a superior feed conversion since its maintenance requirement will be less. Leeson and Summers (1991) indicated that the reason for this reduced maintenance requirement is that at any specific age, prior to reaching point X, the bird has a smaller body mass to maintain and so will need less feed nutrients for this purpose. Smaller birds have proportionally higher maintenance requirements, but if C is sufficiently different to A (Figure 3) the absolute quantity of nutrients going towards
Figure 3. Schematic representation of growth curves of broiler chickens (Leeson and Summers, 1991)
maintenance will be less. Totalling those reduced maintenance needs, while still achieving desired body weight (X) must result in more feed directed to growth and so improved feed efficiency. To demonstrate this theory "that different strains of broiler chickens exhibit different growth patterns and it is possible to have two strains of birds reaching market weight at identical ages, yet with significantly (and economically) different efficiencies of feed conversion," Leeson and Summers (1991) carried out detailed weighing of different strains of broilers throughout the growing period. They, invariably, observed differential growth patterns of the types shown in Figure 3 although the effects were not as dramatic as depicted in this schematic. Leeson and Summers (1991) further pointed out that with today's competitive broiler stocks there is little difference in actual growth rate, although subtle differences certainly exist with respect to feed conversion. The practical questions raised by these researchers are:

a) How do we convert a broiler that inherently has a growth curve depicted by A to that of C (Figure 3), and/or

b) How do we further improve the efficiency of bird C to perhaps D?

Plavnik and Hurwitz (1985) suggested physical feed restriction as a means to manipulate the growth curve (i.e. slow the initial growth) of the broiler chicken for a better feed efficiency. The concept developed by these workers is based on the fact that energy
restriction in many species has been shown to result in a reduction of metabolic energy loss leading to a reduced requirement for maintenance. If, during refeeding this low requirement was maintained and if growth resumed at a normal or above normal rate feed efficiency would be substantially improved leading to an economic advantage. Moreover, if by feed restriction, body weight were made to follow a more concave curve, feed efficiency would be improved due to the reduced cost for maintenance. However, this concept depends on compensatory growth to correct for the earlier growth depression, a phenomenon that remains controversial in terms of broiler production.

Feed Restriction with Broilers

Feeding techniques with possible impacts on improving the efficiency of poultry production have been researched extensively for many years. One of the techniques examined is the application of restricted feeding programs some time during the life cycle production of the bird. Restricted feeding in poultry has been practiced by limiting the birds time of access to feed, by intermittent lighting, feed availability, quantitative feed restriction, and the use of low energy and/or low protein diets.

The effects of different types of restriction-depletion feeding cycles on growth and body composition have been studied in chickens (Lepkovsky et al., 1960; Leveille and Hanson, 1965; Simon
et al., 1968; Griminger et al., 1969). Most of these studies employed methods which changed the chicken from a nibbler to meal feeding in short, discrete meals. Simon et al. (1968) examined the effect of intermittent total starvation with birds fed ad libitum only on alternative days. Growth rate was slower in all restricted groups and these studies showed that young fast growing chickens failed to grow "optimally" when subjected to meal feeding or fed alternative days. Irrespective of the type of restriction, there was marked hyperphagia during repletion but total intake was less than that of birds fed ad libitum and this could explain the reduction in weight gain. However, older birds were able to adapt to meal feeding regimens which consisted of at least five one-hour meals per day (Griminger et al., 1969).

Restricted feeding of broilers for meat production has been re-examined in recent years to improve feed efficiency and reduce excess fat. Gous (1975) studied the effect of alternate feeding and fasting in broilers fed diets differing in protein and energy content and concluded that ad libitum fed broilers outperformed restricted groups under all dietary regimens. However, under restriction there was a definite advantage in favor of a high energy-high protein diet.

Washburn and Bondari (1978) suggested that restricting the time of access of broilers to feed may prevent wastage and possible overfeeding, and take advantage of the bird's ability to compensate
in growth with better utilization of feed ingredients. Studies by Buckland (1975), McCartney and Brown (1977), Holder et al. (1977), and Beane et al. (1977) suggested that eight-week body weights of broilers with different restrictions in the time of feed access by various methods were equal to or greater than that of full-fed controls, resulting in improved feed conversion. Other reports by Proudfoot and Hulan (1982 a, b) indicated that a reduction of the daily feeding time to 16 hours per day, extending from 21 to 49 days of age, had no significant effect on performance. Moreover, broiler chickens with a daily feeding period of 8 hours per day exhibited a significant improvement for feed conversion. They observed that birds subjected to feed denial treatments developed larger crops compared with those on full feed (controls). It was postulated that feed denial might be advantageous during the starter period, an enlarged digestive system might facilitate an improved growth rate when chickens were returned to a full feeding program. In a subsequent report (Proudfoot et al., 1983), feed was denied from the broilers for either 8 or 12 hours per day from 8 to 21 or 15 to 25 days of age. They found decreased 28-day body weights for the feed denial groups. By 49 days of age the body weights of the feed denial groups were equal to those of the control group. There was an improved monetary return of 4 cents per bird on the feed denial program.
It seems that earlier studies on feed restriction with broilers have been done mostly by limiting the time during which the birds have access to feed. However, in recent years, feed restriction has been achieved by either providing a limited amount of feed or by the use of diluted diets with no time limit. Mollison et al. (1984) restricted the feed intake of broilers to 90% of ad libitum consumption from day 7 to day 49. They found decreased body weights with improved feed/gain ratios for the restricted birds at 49 days of age. Pokniak et al. (1984) compared broilers restricted to 45% of ad libitum feed intake for the first 14 or 28 days. At 56 days of age, birds that had been restricted for only the first 14 days were reported to have weights equal to the ad libitum fed group. The birds restricted for 28 days did not weigh as much as the ad libitum fed group. Pokniak et al. (1984) reported an early improvement in feed efficiency for birds that were restricted to 28 days of age, but no final improvement in feed efficiency at market age.

In the last few years, different methods of caloric restriction have been employed in attempts to reduce abdominal fat pad size in broilers. This concept is based on the assumption that such a restriction during early growth will reduce the subsequent deposition of fat by delaying hyperplasia, hypertrophy of adipocytes, or both (March and Hansen, 1977; Plavnik and Hurwitz, 1985; Plavnik et al., 1986). Indeed, several approaches, both
quantitative and qualitative, have been tried in attempts to restrict nutrient intake or caloric intake.

Griffiths et al. (1977) limited the caloric intake by feeding a low-energy diet for the first 3 weeks of age. These researchers observed no effect of limiting caloric intake on the abdominal fat content at 8 weeks of age. However, Hargis and Creger (1980) reported that feeding low-energy diets with no supplemental fat from 0 to 7 days consistently reduced fatness in 49-day-old broilers. Cherry et al. (1978) concluded that feeding a low energy starter diet could, depending on the genetic population, increase, decrease, or have no effect on abdominal fat pad size.

March and Hansen (1977) restricted nutrient intake either by diluting the diet 3:1 or 1:1 by weight with pulverized oat hulls or by fasting the birds 3 days at 1 or 10 days of age. Both programs reduced abdominal fat but also depressed body weight. Jensen et al. (1987) observed a significant increase in the abdominal fat at 7 weeks of age for birds fed a starter diet diluted with cellulose and sand (1:2 ratio) for the first 7 days.

Using quantitative feed restriction (85% of ad libitum intake) during a growing period of 14 to 42 days of age, Beane et al. (1979) observed an increase in fat pad size in male broilers at 56 days of age. Pokniak and Cornejo (1982) and Pokniak et al. (1984) limited feed intake to a certain percentage (15 to 45%) of the consumption of control birds from 8 to 23 days of age. These
researchers found no significant difference for the content of fat in the carcass at 56 days of age.

Thus, reports on the effect of feed restriction on body fat are unequivocal. It is possible that these discrepancies may be due to the type of restriction, and experimental conditions. Moreover, the inconsistencies in some cases may be ascribed to genetic differences in strains used.

Plavnik and Hurwitz (1985) used a different approach that has renewed interest in the idea of restricted feeding programs with broilers. These researchers fed male broiler chicks an amount of feed (40 kcal of ME per bird per day) calculated to only maintain their 1 week body weight for 6 days. Feed-restricted chickens had similar body weight and improved feed efficiency at 56 days of age when compared with controls that were provided *ad libitum* access to feed. Feed restriction also appeared to reduce the amount of abdominal fat. Plavnik and Hurwitz (1985) calculated the coefficient of energy requirements for maintenance. They found the maintenance energy cost of feed-restricted birds was considerably less than that of the controls during the restriction period. They went on to propose that the reduced energy requirement during feed restriction was due to a reduction of both basal metabolic rate and specific dynamic action. They suggested these factors could be responsible for the subsequent improved feed efficiency observed with the feed-restricted birds. The reduced amount of abdominal fat was suggested to be the result of the reduced number
of adipocytes due to the early inhibition of adipocyte hyperplasia caused by the severe energy restriction. Plavnik and Hurwitz (1985) also suggested that the period of restricted energy intake, that resulted in lowered body weight, was followed after reintroduction of adequate feeding, by "compensatory growth" in which the feed-restricted chickens rapidly regained the "lost weight." Indeed, Plavnik and Hurwitz (1985) reported that growth rate was less in feed-restricted birds during the first two-week period of refeeding, but by six weeks the rate of gain was greater in the feed-restricted birds versus the ad libitum fed birds. Based on a series of experiments, Plavnik and Hurwitz (1988) found that male broilers responded most favorably to a seven-day feed restriction period with what they described as adequate compensatory gain, improvement in feed efficiency, and decreased abdominal fat pad size.

A number of papers confirmed the original study of Plavnik and Hurwitz (Plavnik et al., 1986; Plavnik and Hurwitz 1988, 1989) and several abstracts (Calvert et al., 1988, 1989; McMurtry et al., 1988; Rosebrough et al., 1988; Calvert et al., 1989) provided partial corroboration of the original study of Plavnik and Hurwitz (1985). Conversely, several researchers have reported that when feed intake was restricted early in life, market age body weights of restricted broilers were not comparable to those of chicks.
eating ad libitum (Washburn and Bondari, 1978; Pinchasov et al., 1985; Cabel and Waldroup, 1990; Summers et al., 1990, Yu et al., 1990).

Other investigators (Beane et al., 1979; Mollison et al., 1984; McMurtry et al., 1988; Pinchasov and Jensen, 1989; Fontana et al., 1992) have observed significantly reduced body weights, but improved feed efficiencies in feed-restricted broilers when compared with ad libitum controls at various market ages. Calvert et al. (1989) reported that broiler chicks fed 40 kcal per bird per day for 6 or 12 days, starting at 6 days posthatch, had body weight gains similar to unrestricted controls from 21 to 56 days of age. The results of Calvert et al. (1989) indicated that once ad libitum feeding resumed, early restricted and unrestricted broilers grew at similar rates. These findings are in conflict with those of Plavnik and Hurwitz (1985) who reported compensatory growth in broilers following early feed restriction.

Recently, Summers et al. (1990) reported that broilers that were feed-restricted from 7 to 14 days of age had reduced body weights, compared with unrestricted controls at 41 days of age in one experiment, but similar weights at 42 days of age in a second experiment. In addition, while imposing various early feed restriction regimens, Yu et al. (1990) observed that male broilers restricted to 23 kcal per bird per day from 8 to 14 days had significantly lighter body weights at 56 days of age than birds
eating *ad libitum*. However, it is interesting to note that Summers *et al.* (1990) and Yu *et al.* (1990) observed similar feed efficiencies for restricted and unrestricted broilers at the conclusion of their experiments.

**Quantitative versus qualitative feed restriction**

In recent years, some researchers have raised questions about the practicality of using quantitative feed restriction to limit nutrient intake in the broiler chickens. This physical means of nutrient restriction consists of providing a limited amount of feed during the restriction period. Summers *et al.* (1990) pointed out that in practical conditions (i.e., industry), the small quantity of feed, usually given several times during the restriction period, is difficult to distribute evenly within a flock and there is also a question regarding the micronutrients and anticoccidiostat intake of the bird subjected to a quantitative nutrient restriction. Jones and Farrell (1992a) also indicated that because of broiler behavior and feed delivery problems, the use of quantitative feed restriction in commercial practice may be of limited value. These workers observed that broiler chickens subjected to early-life nutrient restriction become excitable after 2-3 days on the restriction regimen, and in commercial practice this may result in deaths from "crushing" while feeding.
Jones and Farrell (1992a) suggested a 4-day discontinuous (i.e., restriction for 2-day followed by 2-day ad libitum feed then a further 2 day restriction) feed restriction program that provoked no excitement of the birds. Other workers have suggested diet dilution with non-nutritive materials such as alpha-floc (Summers et al., 1990), rice hulls (Leeson et al., 1991; Jones and Farrell, 1992a), or oat hulls (Zubair and Leeson, 1994a) as an alternative to the quantitative means of nutrient restriction. Jones and Farrell (1992a) pointed out that the use of a diluted diet reduced excitability and may offer a more flexible approach to feed delivery. Summers et al. (1990) observed no differences in 42-day body weight between ad libitum fed birds and birds either restricted in feed intake (75% of the ration consumed at 7 days) or fed diluted diets (15% alpha-floc) between 7 and 14 days. Jones and Farrell (1992a) observed no differences at 49 days between chickens restricted quantitatively (i.e. 4 days continuously or discontinuously 2 X 2 d) and those restricted by diet dilution by the inclusion of 65% or 60% ground rice hulls to the starter diet. Treatment effects were not observed on feed intake. Furthermore, feed conversion ratio was not affected in the birds fed on the diluted diets compared with the control birds. Recently, Zubair and Leeson (1994a) observed a complete growth compensation by 35 days of age in all treatment groups that were previously fed a diet in which 50% of the major ingredients were replaced by 50% oat
hulls. Growth compensation was attributed to the better feed efficiencies of the birds fed the diluted diet during realimentation.

Alternate forms of feed restriction have been proposed, including altering the photoperiod and the use of chemicals to suppress feed intake. Altering the photoperiod for the specific purpose of restricting feed intake in broilers has not been reported in the literature. However, Classen and Riddell (1989) noticed that chicks exposed to 6L:18D from 3 to 21 days of age ate significantly less feed compared with chicks exposed to 23L:1D. Reducing the light period to 6 hours in a 24 hour day had no effect on overall feed conversion ratio and body weight. The method of intake restriction of broiler chicks by chemical means was suggested by Fancher and Jensen (1988) who demonstrated that glycolic acid depressed feed intake in broilers in a dose-dependent manner. Later, Pinchasov and Jensen (1989) incorporated 1.5 and 3% glycolic acid into broiler feed and obtained 22% and 50% reduction, respectively, in feed intake of broilers from 7 to 14 days of age compared to controls.

*Feed restriction as an economical management alternative for the broiler industry*

Although market improvements have been made in both growth rate and feed efficiency of broiler and roaster chickens, high incidences of mortality and skeletal or metabolic diseases threaten
the viability of the industry (Robinson et al., 1992). Robinson et al. (1992) pointed out that measuring production efficiency in terms of days to market may not optimize the number of birds that are marketed nor the quality of the birds that are marketed. Several researchers have indicated that some of the problems mentioned above may be the result of early rapid growth. Robinson et al. (1992) examined the use of early short-term (7d duration) feed restriction as a management tool for use in reducing the incidence of skeletal and metabolic diseases in broiler and roaster chickens. Restricted feeding resulted in a reduction of the number of birds culled for skeletal disease, but feed-restricted birds were significantly lighter as compared with birds given ad libitum access to feed at 6 weeks and 9 weeks of age. Other benefits of early feed restriction include reduced incidences of sudden death syndrome (Mollison et al., 1984) and ascites (Albers et al., 1990). Although selection for increased body weight and rate of gain has resulted in considerably heavier commercial broilers that are marketed at progressively younger ages, there also has been a growing body of evidence suggesting a negative genetic association between growth rate and immunocompetence in chickens (Dunnington et al., 1986; Martin et al., 1988 a, b). Katanbaf et al. (1988b) challenged broilers with Escherichia coli after they were provided ad libitum access to feed, those restricted continually, and those restricted and then released to ad libitum feeding. Respective
percentages of mortality to the challenge were 20, 7 and 50. Similarly, Ballay et al. (1992) reported that the response to *Escherichia coli* inoculation was generally less severe in chicks provided restricted access to feed, and overall mortality was greatest for chicks eating *ad libitum*. Thus, despite the disadvantage of short-term feed restriction in terms of final body weight, early feed restriction programs have some merits that may outweigh this disadvantage.

*Effect of feed restriction on the development of the gastrointestinal tract*

Most studies on early nutrient restriction with broilers have been concerned for economic reasons with growth performance (e.g., body weight, feed efficiency) and carcass fat of the feed-restricted chickens as compared with full-fed chickens. Yet, there is no agreement among researchers on the response to feed restriction with respect to these performance criteria (body weight, feed efficiency, and carcass fat).

Among birds, patterns of growth of digestive organs seem to be correlated with body growth rates (i.e. rapidly growing birds exhibit early development of digestive organs, Lilja, 1983). Such observations have led to the hypothesis that growth rate may be determined in part by the relative emphasis given to tissue in the alimentary tract (Lilja et al., 1985; Konarzewski et al., 1989).
Several researchers have shown that the gastrointestinal tract has a major role in supporting growth during the early posthatching period in poultry (Lilja, 1983; Katanbaf et al., 1988a; Nitsan et al., 1991; Sell et al., 1991). Katanbaf et al. (1988a) stated that the accelerated growth of the GIT of chickens immediately after hatching demonstrated the importance of organs and tissues that fulfill a "supply" function for achieving early body development.

The role of the GIT as a supply organ system, however, has not been extensively investigated in feed restriction studies with poultry. Scott et al. (1991) suggested that limitations in the allocation of energy for the growth of the GIT may limit energy availability for growth.

It is difficult to compare data in the literature with respect to responses of the GIT to restricted feeding because of differences in the severity and duration of restriction, age of the birds when feed restriction was applied, and breed of chicken (e.g., meat or egg type). Michael and Hodges (1973) observed some atrophy of the chicken small intestine during feed restriction. These workers observed slightly shorter and thinner villi in the restricted birds as compared with controls at the end of an 8-day feed restriction period. Michael and Hodges (1973) used chickens that were 6 weeks old and limited their feed intake to 25% of that of full-fed chickens. Barash (cited by Nitsan, 1985) observed
that meal feeding was associated with increased length and reduced weight per length unit (g/cm) of the small intestine by approximately 32% and 12% in chicks fed one or two meals daily, respectively. These findings are in accord with those of Lepkovsky et al. (1960) who concluded that meal feeding increased the flexibility of the intestinal wall, thus enabling accommodation of excessive amounts of chyme and increasing the rate of absorption.

Pinchasov et al. (1985) reported that intermittent feeding was accompanied by a consistent increase in the relative weight of the liver, pancreas, and GIT including the small intestine. In the study of Pinchasov et al. (1985), male chicks of a heavy bodied strain (White Rock) were deprived of feed on alternate days from 14 through 83 days of age. Katanbaf et al. (1989) applied daily feed restriction or skip one-day or skip two-day feeding programs to meat type female breeders from 7 days through 461 days of age. These workers observed that restricting feed intake increased relative weight, and length of segments of the GIT, and relative weight of the pancreas.

After reviewing the literature, Nitsan (1985) stated that the response of the GIT to feed restriction may vary according to the type of restriction and that the magnitude of response seems to be related to the intensity of restriction. Nitsan (1985) concluded that, although the crop and gizzard are hypertrophied in all types of restriction or meal feeding, the weight of the duodenum and
small intestine was increased only when the time during which the chicks had access to the feed was limited (i.e. meal feeding) but not when feed was restricted with no time limit (i.e. feed restriction). This review by Nitsan (1985), however, referred to feed restriction studies conducted with chickens (meat and egg-type strains) that were at least 4 week-old when GIT measurements were made.

Deaton (1992) presented data showing that meal feeding of broilers significantly increased small intestine weight of broilers fed either mash or pelleted diets when compared with continuously fed broilers. Body weight, feed conversion, and mortality did not differ between the continuous-fed versus meal-fed groups. Recent research by Zubair and Leeson (1994b) indicated that, during feed restriction, weights of digestive organs (expressed as a percentage of body weight) were generally heavier for feed-restricted broilers as compared with full-fed chicks. These researchers used a feed restriction program that consisted of providing to restricted broilers 50% of the daily feed intake of the full-fed chickens from 6 to 12 days of age. Relative weights of crop, proventriculus, and gizzard were greater at 11 d of age for the feed-restricted birds as compared with the full-fed birds. No treatment effects were observed on relative weight of small intestine, large intestine, liver and pancreas at this age, but 5 days after beginning of realimentation the feed-restricted group showed greater relative weights of pancreas and liver.
Effect of feed restriction on activities of digestive enzymes

Observations indicate that newly hatched poultry have a limited capacity to digest certain carbohydrates, proteins, and lipids. In the instance of carbohydrates, Siddons (1969) found low maltase and sucrase activities in the intestinal mucosa of hatchling chicks. Similarly, Sell et al. (1989) observed low sucrase and isomaltase activities in mucosa of newly hatched poulets.

In terms of synthesis and/or secretion of digestive enzymes, the pancreas of newly hatched poulets also is immature. Krogdahl and Sell (1989) found that pancreatic amylase, protease, and lipase activities were low at hatch (approximately 20, 50, and 30% respectively, of the activities observed at 56 d of age). Subsequently, amylase activity increased rapidly through 21 d posthatching, whereas protease activity increased more slowly. Lipase activity remained low through 14 to 16 days after hatch and then increased at a moderate rate, plateauing at about 36 d of age. Escribano et al. (1988) confirmed these observations with lipase, working with young turkeys. Zelenka (1973) presented data indicating that low activities of digestive enzymes may be translated into relatively poor utilization of nutrients. He found that digestibility coefficients of organic matter, nitrogen-free extracts, and lipids were low for chicks 2 to 6 days of age. These digestibilities increased by 20 to 25% during the subsequent 8 to 14 days.
Thus, the synthesis and/or secretion of digestive enzymes of neonate poultry could be a limiting factor in studies on the effects of early nutrient restriction on the development of the digestive enzymes of poultry. The activity of digestive enzymes is affected by various factors among which are feed composition and level of feed intake (Corring, 1980). In force-fed chicks, 70% more feed than that consumed by ad libitum counterparts and in chicks pair-fed the amount consumed by ad libitum fed ones in two meals per day the level of digestive enzymes increases markedly and paralleled the increase in the feeding level (Nitsan et al., 1974; Nir et al., 1979). In feed-restricted or in meal-fed chicks the activity of digestive enzymes in the intestinal contents increased markedly after short meals (2 h) or after 24 h of feed restoration in intermittently-fed chicks. After 24 h of feed deprivation, the activity of digestive enzymes decreased sharply. In chicks fed intermittently, the synthesis of digestive enzymes on days of feed restoration was great enough to maintain high levels of enzymes in both the intestine and pancreas. This response was quite rapid since enzyme activities in both the pancreas and intestinal chyme varied daily with feeding status when chicks were exposed to day-on day-off regimen (Nir and Nitsan, 1979).

Michael and Hodges (1973) observed, during feed restriction of chickens, an increase in enzyme activities in the absorptive cells
of the small intestine. These workers concluded that the enhanced absorption of nutrients observed in semistarved animals was correlated with increased mucosal enzyme activities. However, it is not known whether this increase in enzyme activities persisted during the refeeding phase.

Corring (1980) indicated that pancreatic protease and amylase secretions, as well as their concentrations in pancreatic tissue were proportional to the amount of the substrate. For lipase, however, secretion and activity depended on dietary lipid but increases in secretion and activity were not proportional to the amount of this substrate (Lhoste et al., 1993). Hulan and Bird (1972) also reported that lipase activity in pancreatic juice was significantly augmented by increasing dietary fat intake. Moreover, studies with pouls suggested that the activity of lipase in the intestinal lumen depended on dietary fat level (Krogdahl and Sell, 1989). Low activities were observed with low fat diets whereas with high fat diets a lag period of about 3 wk (1 to 21 days posthatching) was followed by a five-fold increase in lipase activity. Corring (1980) reviewed the literature on the adaptation of digestive enzymes to the diet and indicated that when dietary restriction was not too severe, the biosynthesis of all digestive enzymes markedly decreases.
**Effect of organ size on energy utilization**

Maintenance has been defined as the feed or energy intake at which body weight or energy does not change. Maintenance requirements have been related directly to fasting heat production (ARC, 1980) and both have generally been considered to be solely functions of body size (ARC, 1980; Brody, 1945; Kleiber, 1961). However, many factors such as age, breed, sex, and environmental temperature affect fasting heat production or maintenance energy expenditures (Ferrell and Koong, 1986). Several reports have shown that fasting heat production decreases in response to decreased levels of feed intake (Marston, 1948; Graham et al., 1974, 1975; Thompson et al., 1979). Pekas (1983, 1986a, b) reported that certain gastrointestinal tissues respond to the quantity of feed intake. Indeed, fasting heat production has been shown to be closely associated with the weight of the gastrointestinal tract. Foot and Tulloh (1977) presented data suggesting that decreases in maintenance energy requirements were associated with a decrease in internal organ mass, especially the liver. Ledger and Sayers (1977) similarly noted that feed required to maintain live weight decreased with time. Their data indicated that weights of internal organs and empty digestive tract decreased in response to maintenance feeding. Reports with rats (Ferrell and Koong, 1986) and pigs (Pond, 1984) also have shown that the weights of internal organs are influenced by plane of nutrition. Visceral organs may
be involved in repartition of energy utilization for growth. The visceral organs constitute only 10% or less of total body mass, yet they play a disproportionate role in whole body energy utilization (Huntington and Reynolds, 1987; Ferrell, 1988; Yen et al., 1988). Based on data on rate of blood flow Webster (1980) estimated that heat production from liver, gut, skin, and kidney accounted for 45% of total heat production of rats at rest. Most of this heat was associated with protein synthesis, and greater rates of synthesis occurred in tissues, such as the liver and gut, than in muscle. A comparison of tissue protein synthesis in the rat is illustrated in Table 1 (Webster, 1980). Because these tissues, particularly the liver and gut, have a high rate of energy expenditure relative to their size, changes in size and metabolic rate of the tissues may have a substantial impact on maintenance requirements and, during the realimentation period of feed-restricted animals, on efficiency of body weight gain. Ferrell and Koong (1986) indicated that whole body protein synthesis decreased as the plane of nutrition declined and that this was associated with a decrease in the requirement for energy stasis. Therefore, the improved efficiency of growth observed during realimentation of feed-restricted animals is probably due to a decrease in the maintenance requirements which seems to be the result of changes in the proportion of high energy expending organs as well as in protein content of the body.
Table 1. Protein Synthesis and Deposition in 200 to 350g rats (Webster, 1980).  

<table>
<thead>
<tr>
<th></th>
<th>Skeletal Muscle</th>
<th>Liver</th>
<th>Gut</th>
<th>Skin</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Protein Content (g)</td>
<td>32.20</td>
<td>3.30</td>
<td>3.1</td>
<td>16.9</td>
<td>55.6</td>
</tr>
<tr>
<td>Protein Synthesis Rate (g/day)</td>
<td>1.78</td>
<td>2.33</td>
<td>5.28</td>
<td>2.56</td>
<td>13.0</td>
</tr>
<tr>
<td>Protein Synthesis (% of Total)</td>
<td>13.80</td>
<td>18.0</td>
<td>40.7</td>
<td>27.5</td>
<td>100.0</td>
</tr>
</tbody>
</table>
Feed Restriction and Catch-up Growth

Whether market broilers achieve total compensatory growth following an early feed restriction is open to question. Although Plavnik and Hurwitz (1985), and Plavnik et al. (1986) have suggested that compensatory growth occurs after a short period of feed restriction, Pinchasov and Jensen (1989), Cabel and Waldroup (1990), and Yu et al. (1990) have not been able to demonstrate that broilers are able to completely compensate for a growth deficit incurred during a period of restricted feeding. Another controversial aspect of early feed restriction programs with broilers has been the lack of consistent effects on abdominal fat pad or total carcass fat. To understand the differences in results, it is necessary to examine the phenomenon of catch-up growth and some of the factors that may influence the response of broiler chickens to short-term feed restriction and refeeding.

The phenomenon of compensatory growth

The preservation of constancy in the internal environment of living organisms was given the name of homeostasis by Cannon (1929). He defined it as the tendency of an organism to restore their physiologic equilibrium when forces seek to cause disequilibrium. Brody (1945) pointed out the wide application of this principle not only in maintaining constant body temperature or blood composition but also with regard to the growth and
development of animals. Brody (1945) demonstrated that growth, at least in the last stages, proceeds as if the normal condition were the mature size and that the rate of growth is proportional to that needed to reach mature weight. Therefore, an animal whose growth has been retarded exhibits, when the restriction is removed, a greater rate of growth than that which is normal in animals of the same chronological age. Waddington (1957) used the term "homeorhesis" to describe the tendency of growing organisms to return to their paths of growth after deviating from them. Later, Bauman et al. (1982) also used the term homeorhesis to denote the mechanisms by which an organism partitions nutrients to tissues to support growth or other physiologic processes. This process differs from homeostasis, which usually denotes rapid adjustments intended to maintain physiologic equilibrium. Homeostatic controls ensure, for example, that nutrient stores established in the fed state are available during fasting to maintain a constancy of the internal environment. Homeorhetic control mechanisms direct a flux of nutrients to tissues that are involved in growth or processes such as lactation. This redirection of nutrients may even proceed to the point of deranging metabolic homeostasis. Thus, homeorhetic mechanisms, are actively involved in the maintenance of the animal's genetic program for growth and development and may be an appropriate basis for the phenomenon illustrated by Brody (1945)
that animals can exhibit rapid growth after a period of undernutrition.

Osborne and Mendel (1915) were the first to observe this phenomenon with albino rats fed protein deficient diets and later fed more adequate diets. They wrote: "growth in the cases referred to, is resumed at a rate normal for the size of the animal at the time. It need not be slow, and frequently it actually exceeds the normal progress." Bohman (1955) observed this phenomenon of rapid growth rate relative to age after a period of retarded growth in beef cattle and introduced the term "compensatory growth" shortly thereafter. Wilson and Osbourn (1960) published an excellent review of research on compensatory growth in mammals and birds.

Prader et al. (1963) used a different terminology to describe the same growth phenomenon in children whose growth has been slowed by illness. These children show a greater than normal rate of growth upon correction of the disorder. Prader et al. (1963) pointed out that the rapid phase of growth may continue until the child has caught up to its pre-illness, or normal growth curve and is, therefore, called "catch-up" growth. Indeed, as early as the eighteenth century, it was observed that children have the capacity to grow faster than usual when they are recovering from malnutrition or some other growth-suppressing disorder (Tanner, 1979).
The terms "catch-up growth" and "compensatory growth" are used today interchangeably. However, researchers studying this growth phenomenon in farm animals tend to use the term compensatory growth, whereas catch-up growth is the term used when referring to humans. Those who prefer the term catch-up growth object to the use of the term compensatory growth simply because it was a term already used by zoologists to refer to the excess growth of the remaining member of a pair of organs (e.g., kidneys, testes, etc.) when one of the pair was removed (Tanner, 1981). In my opinion, catch-up growth would seem to be more precise in describing the growth observed upon refeeding after a period of feed restriction. In this review, however, the term catch-up growth is a frequent synonym of compensatory growth.

**Occurrence of compensatory growth in mammals and birds**

The phenomenon of catch-up growth is well documented in animals and humans. Many farm animals exhibit a remarkable recuperative capacity to grow after periods of underfeeding. In sheep, for example, the disadvantages experienced by twin lambs during the suckling period are partly offset when the lambs become independent of the milk supply, and the growth rate of twins begins to overtake that of singles (Hammond, 1932). In cattle, Waters (1908) demonstrated that capacity of steers to recover after periods of underfeeding. The same phenomenon has been reported in
monogastric animals such as the rat (Quimby, 1948), the pig (Kertesz and Csir, 1962), and the fowl (Wilson, 1954).

Compensatory growth is an issue of significance to animal production in many parts of the world, where seasonal and climatic fluctuations result in drastic changes in feed availability and environmental stress and, therefore, in the growth rate of animals. Under some conditions, animals undergo recurring cycles of restricted and unrestricted growth, even for a number of years, until growth ceases (Reid and White, 1977).

Wilson and Osbourn (1960) cited many references indicating that birth or hatching weights of mammals and birds do not correlate either with mature weight or postnatal growth. They indicated that enhanced postnatal growth rate of many animals, which were small at birth or hatching, could be considered as a basic example of compensatory growth. Using this example, it could be deduced that most animals have an innate ability to exhibit compensatory growth.

Factors that Influence an Animal's Ability to Recover from the Effects of Undernutrition

The ability of animals to compensate in growth after a period of undernutrition has been reviewed by Wilson and Osbourn (1960), and these researchers listed six factors that influence the ability of animals to recover from growth retardation. These factors are:

1. The nature of undernutrition
2. The severity of undernutrition
3. The duration of the period of undernutrition
4. The stage of development at the commencement of undernutrition
5. The relative rate of maturity of the species
6. The pattern of realimentation

The nature of undernutrition

An animal's growth can be retarded by restricting any component of its diet. In most feed restriction studies, however, either the protein and/or the energy contents were restricted. Restriction is achieved by dietary manipulation (proportional changes of ingredients in the diet) or by limiting feed intake. The ability to recover may, in certain instances, depend on whether the energy or the protein content of the diet has been limiting weight gain. The papers reviewed by Wilson and Osbourn (1960) indicate that complete recovery is possible after quite severe restriction of either the energy or protein content of an animal's diet. Nevertheless, it seems possible that very severe protein restriction may have a more harmful effect than very severe energy restriction. They speculated that there is little reserve protein in animals and, consequently, active tissues could be depleted and irreparably damaged. Thus the severity of undernutrition is also
an important factor to consider with respect to an animal's ability to exhibit compensatory growth.

The severity and duration of the period of undernutrition

In experiments where the plane of nutrition is varied, the severity of the feed restriction may be increased by greatly reducing feed intake or by extending the duration of restriction over a long period. Hogan (1929), Joubert (1954), and Osbourn and Wilson (1960) agree that the rate of growth following restriction is directly proportional to the severity of the restriction when it is imposed by severely depressing feed intakes, although Hogan (1929) considered growth to be independent of the duration of restriction.

Pratt and McCance (1960) found that bone development in cockerels was reduced by severe feed restriction in a period as short as 7 days. The dwarfing effect became progressively worse as the duration of severe feed restriction was increased. Evidence suggests that animals with stunted bone growth do not recover as well as animals that suffer from retardation of soft tissue development. After reviewing the literature on the effects of severity and duration of undernutrition, Wilson and Osbourn (1960) concluded that realimentation following short periods of restriction resulted in increased growth rates compared with unrestricted control animals. Longer periods of restriction,
however, diminished this recovery and may result in normal weight being achieved at a much later chronological age, or sometimes in a permanent stunting of the animal. Finally, it seems that the more severe the restriction, the greater is the initial rate of gain immediately after realimentation. This phenomenon was first reported by Clarke and Smith (1938) with rats. These researchers induced retarded growth by restricting the calorie and mineral intakes by 50% for 3 weeks, and they found that the realimented rats gained in weight so rapidly that by 9 weeks their weights exceeded those of the controls. They termed this phenomenon "over-compensation". These results were later confirmed by Quimby (1948). Both of these experiments evaluated compensatory growth on the basis of body weight. Wilson and Osbourn (1960) indicated that the increase in weight in the realimented groups may be partly due to increased gut content, and/or increases in the fat content of the body. Wilson and Osbourn (1960) concluded that there may be a fundamental difference in response to realimentation of (i) animals restricted in such a manner that they lost weight during the period of undernutrition, (ii) animals restricted so that they maintained weight constant during the period of undernutrition, and (iii) animals only mildly restricted so that they made small weight gains during the period of undernutrition. Therefore, it is difficult to make comparisons among different experiments which have employed different modes of restriction.
The stage of development at the commencement of undernutrition

The stage of development at which the undernutrition commenced has a profound effect on the degree of compensatory growth, in terms of rate of weight gain or the ability of an animal to fully recover from the growth restriction and achieve its intended mature body size. Compensatory growth is influenced by the normal growth pattern of an animal, which is determined by the distribution of growth among various tissues and organs. Hammond (1932) suggested that chronologically various tissues reach their maximum growth rate and mature in the following order: nervous tissue, bone, muscle, and fat. Later, he proposed that the same order exists among body tissues for their priority for nutrients. Thus, the plane of nutrition imposed at certain stages of growth influences the extent of development of the various body parts differently depending upon their nutrient priorities and needs relative to supply.

Wilson and Osbourn (1960) cited several workers whose work suggested that undernutrition in the earlier stages of growth was more detrimental to an animal than restriction at a later stage, and that the ability to recover and to reach normal mature size was consequently reduced. On the other hand, if the undernutrition began too late in the growth phase of an animal, then the degree of compensatory growth in terms of rate of weight gain would be less
than if the undernutrition began earlier (Williams et al., 1974; Williams and Hughes, 1975).

Critical periods of development

Enesco and Leblond (1962) proposed a hyperplasia-hypertrophy model of tissue growth. According to this model, tissue growth proceeds in three distinct phases. Initially there is proliferation (hyperplasia) of the component cells of the tissue. Before cell division ceases, the tissue enters a second phase in which the dividing cells begin to enlarge. Finally, once the final adult complement of cells is established, cell division ceases and tissue growth results only from enlargement of existing cells (hypertrophy). Enesco and Leblond (1962) proposed this model on the basis of studies in growing rats, measuring tissue DNA as an index of cell number (since the amount of DNA per nucleus remains fixed) and the ratio of DNA to tissue weight as an index of cell size (the ratio of nucleus to cytoplasm). Their studies suggested that in the first 17 days of postnatal life in rats, all tissues enlarged by increasing their DNA content while the ratio of DNA to tissue remained constant. After 34 to 48 days of life, tissue weight increased steadily and DNA remained fixed. Between these two periods, tissue growth seemed to result from increases in both cell number and size. Using mathematical analysis, Laird (1966)
confirmed that this growth pattern was comparable with that of other mammals and birds.

Although the hyperplasia-hypertrophy model has been useful in understanding the "critical periods" of vulnerability to undernutrition, it has not been universally accepted. In contrast to Enesco and Leblond (1962), Sands et al. (1979) reported that multiple tissues in the rat showed an increase in cell size early in development, and cell proliferation continued in many tissues as long as the tissue was growing.

The response of orderly growth to nutritional deprivation was brought into focus by the work of Widdowson and McCance (1975), and McCance (1976). These investigators formulated a hypothesis of critical periods of sensitivity to the effects of malnutrition. They observed that growth was stunted when rat pups were nutritionally deprived by manipulation of litter size during suckling. This stunting could not be overcome by restoration of normal nutritional intake. Subsequent studies in both rats and pigs revealed that this process was critically dependent on the duration of nutritional deprivation and the developmental stage of the animal at its onset (McCance, 1975).

Winick and Noble (1966) studied the effects of nutritional deprivation at various ages in rats and provided evidence that the state of nutrition during the neonatal period influences cell proliferation and consequently determines the ultimate size of the
animal and its organs. Food restriction for the first 21 days of life resulted in an overall decrease in cell number of most tissues, but cell size remained normal. Provision of adequate nutrition after weaning at 21 days did not restore normal organ growth presumably because of a deficit of cells. This same phenomenon was evident when malnutrition was induced between weaning and 42 days, except that the brain and lung were able to resume normal growth with restoration of proper nourishment. In these tissues, malnutrition had not reduced the total cell number but had reversibly decreased cell size. When malnutrition was induced between 65 and 86 days of age, almost all organs underwent a reversible decrease in cell size only. In short, the critical period hypothesis holds that nutritional deficit of a tissue at a time coincident with its phase of maximal cellular proliferation will irreversibly alter the ultimate growth potential of that tissue by impairing cell division. Altered nutrition after the critical period produces changes in cell volume but not in cell number. This process is reversed by normalization of nutrient supply because tissues mature at different rates, and the long-term effects of a nutritional insult occur in those tissues that are at a susceptible developmental period during the time of deprivation (Winick and Brasel, 1980).

Although useful for understanding the differential sensitivity of tissue to nutritional alterations, the critical period
hypothesis depends upon the triphasic model of growth proposed by Enesco and Leblond (1962). Wharton (1976) has cautioned that the critical period hypothesis may be less applicable to deranged nutrition in postnatal life than it is in fetal life when the potential impact of restrictive influences is much greater.

The relative rate of maturity of the species

The relative rate at which an animal matures is of importance when dealing with both inter-breed and intra-breed differences (Wilson and Osbourn, 1960). There is very little information, however, that specifically evaluates the effect of the relative rate of maturity on compensatory growth.

However, a few reports suggest that slower maturing animals are more capable or recovering from earlier undernutrition than faster maturing animals. This characteristic may explain the differences in results observed among researchers with respect to body weight recovery in feed-restricted chickens. Winchester and Howe (1955) feed-restricted cattle having the same initial weight with equal severity and duration. The genetic differences between these two animals was revealed by the growth rates of their control twins which were 1.52 and 2.26 pounds /day respectively. The recovery indices were 23% and 12% respectively, suggesting that the slower maturing animal was able to make a more rapid recovery than the faster maturing animal. Wilson and Osbourn (1960) indicated
that if a breeder aims at early maturity, then selection for this characteristic will lead to the development of a population which will be handicapped by a period of undernutrition. Indeed, restriction at very early ages may be disadvantageous for broilers with the potential to grow rapidly or those reared on high-nutrient density feeds (Marks, 1990; Washburn, 1990). This relationship is not consistent, however, within all species. Auckland (1972) presented data with turkeys that suggest the opposite is true: fast maturing strains were more able to compensate from early growth restriction than slower maturing strains.

The pattern of realimentation

Bohman (1955) showed that the higher the plane of nutrition during realimentation, the more rapid and the greater the recovery in weight of cattle. Because the rate of compensatory growth was greatest immediately after realimentation, it is possible that a continuous 100 days undernutrition would best be made good by splitting up this restrictive period into five periods of restriction each of 20 days duration. These restrictive periods would be interspersed with five periods of realimentation, during each of which, compensatory growth might be expected to be maximal (Wilson and Osbourn, 1960). Studies with chickens tend to support this concept. Osbourn and Wilson (1960) feed-restricted chickens using two periods of restriction of 10 to 14 days duration.
separated by a period of 18 days of *ad libitum* feeding. These birds were compared with a second group which were subjected to continuous mild restriction for the same total period and which received the same total quantity of feed. Both groups were then realimented on *ad libitum* feeding until they reached equal weights. A control group of birds was fed *ad libitum* throughout the experiment. The birds that were on the discontinuous feed restriction treatment showed a greater relative growth rate after final realimentation than the mildly restricted group. However, the efficiency of feed conversion, measured in terms of units of weight gain per unit of feed consumed, was essentially the same for all three groups.

**Mechanisms Involved in Catch-up Growth**

The ability possessed by animals to recover from the growth deficit sustained during a period of undernutrition has been presented earlier. This section presents some of the processes by which catch-up growth is achieved. The recovery of an animal whose growth has been retarded by dietary restriction is brought about following realimentation in two ways: (a) a prolongation of the time taken to reach a mature weight; and, (b) an increase in the rate of gain in weight during the realimentation period, especially during the early stages of realimentation.
Prolongation of the growth period

Several researchers presented data showing that animals that had been feed-restricted, either for one long continuous period or discontinuously for shorter periods continue growing long after normal animals achieved their mature weight (Osborne and Mendel, 1915; Wilson and Osbourn, 1960). Ragsdale (1934) suggested that the phenomenon of catch-up growth, once the restriction is lifted, is due to the disturbance between the chronological and physiological aging whereby the latter proceeds at a slower rate under conditions of nutrient restriction. Other reports, however, indicated that animals subjected to severe undernutrition early in life and then realimentated stopped growing at a fixed chronological age regardless of their size (Widdowson and McCance, 1963 with rats; Lister and McCance, 1965 with guinea pigs; Lister and McCance, 1967 with pigs). That age was the same as that of growth cessation by their ad libitum fed litter mates. On the other hand, McCay et al. (1935) observed a considerable increase in the weight of feed-restricted rats long after the normal life span of their litter-mates that were given ad libitum access to feed. Similarly, cockerels severely feed-restricted during the first 6 months and then realimentated, grew well at an age after which their ad libitum fed broodmates had already reached their full genetic stature and yet despite this, the feed-restricted cockerels
failed to achieve the same body weight than their *ad libitum* fed counterparts (Lister et al., 1966).

*Increase in rate of weight gain*

Increased rate of body weight gain following a period of growth restriction is the most frequent observation associated with the catch-up growth phenomenon. Some workers suggested that the increased rates of weight gain of the realimentated animals are not true increases in body tissue weight and mass but rather reflect an increase in gut content and/or increase in amount of fat deposition as a consequence of the realimentation. Maynard (1947) drew a distinction between what he termed "true growth" and fat deposition within an animal. He stated that true growth was characterized by an increase in mass of protein, minerals, and water, but not by an increase of fat. Several mechanisms have been proposed to explain the increase rate of body weight during the realimentation period.

*Change in gut content weight*

McMeekan (1940) showed that realimentated pigs had the same eviscerated carcass weight gain as *ad libitum* fed pigs; however, these pigs had a higher rate of live weight gain which was attributed to an increase in gut content. Similarly, Drew and Reid (1975) showed that gut weight increased in realimentated sheep. There is some evidence that increased gut contents exaggerates the
rate of gain; nevertheless, many feed-restricted animals still exhibit a greater degree of growth during realimentation than their ad libitum fed counterparts.

Digestibility of feed

The energy content of digested feed, expressed as a percentage of gross energy intake, increased as the gross energy intake decreased (Waters, 1908; Quimby, 1948). Mitchell and Hamilton (1932) and Blaxter and Graham (1955) showed that this decrease in digestibility with increasing intake can only be demonstrated in the nitrogen-free extract and other ether extract components of the ration. Indeed, this change in digestibility has been related to the rate of passage of feed through the gut, rather than to the level of intake per se (Blaxter et al., 1955). It has also been reported that decreased digestibility occurs only at exceptionally high and low energy intakes. This would seem to be supported by the relationship of digestibility to the rate of passage through the gut (Forbes et al., 1928, 1931). Other researchers indicated that the improved digestibility observed in animals on a low plane of nutrition can also extend into the period of realimentation (Quimby, 1948; Burton, 1970). Some authors, however, could not find a difference in digestibility during prolonged restriction and refeeding. Sheely and Senior (1942) conducted extensive digestibility trials during restriction and realimentation periods.
They found digestibility of hay, for growing cattle to be the same during both periods. From these varying results, it cannot be definitely stated that an animal is any more efficient at digesting feed during either a period of feed restriction or of realimentation.

**Appetite**

Under *ad libitum* feeding conditions, increased appetite is probably the major factor involved in growth deficit recovery. Several researchers presented data showing a significant increase in the appetite of feed-restricted animals during realimentation (Sheely and Senior, 1942; Winchester and Howe, 1955 with cattle; Quimby, 1948 with rats; Osbourn and Wilson, 1960 with chickens.) Similarly, Ashworth (1969) and Ashworth and Millard (1986) observed that catch-up growth in children was always associated with increased appetite which dropped off abruptly once the "normal" body weight for a given height was reached.

It has been suggested that the development of the alimentary tract was only slightly retarded by undernutrition and was related to chronological age rather than to physiological age (Wilson and Osbourn, 1960). Thus, animals subjected to undernutrition have the physical capacity to ingest the same or a greater bulk of feed as compared with *ad libitum* fed animals. On the other hand, several workers did not observe any change in appetite with realimentation.
Meyer and Clawson (1964) presented sheep and rat data that showed no increase in feed capacity or appetite during realimentation. Stuedemann et al. (1968) also observed similar results with cattle.

The conflicting results in the literature concerning appetite and realimentation suggest that other factors influence the role of appetite in the catch-up growth phenomenon. There is some evidence that the appetite and control mechanisms of feed intake have a profound effect on growth rate but at this time the mechanisms are poorly understood.

**Efficiency of growth**

In rats, feed efficiency was observed to improve after short-term periods of feed restriction (Meyer and Clawson, 1964). Similarly, studies on fasted and refed rats showed that they utilized ingested calories for weight gain five-fold more efficiently than controls (Bjorntorp and Yang, 1982). Decreased thermogenesis during refeeding was postulated to account for this increased efficiency. Several workers indicated that animals generally utilize feed more efficiently and gain more weight relative to the amount of feed consumed following a period of feed restriction than they do when they are given *ad libitum* access to feed throughout their growth period (Levitsky et al., 1976; Boyle et al., 1978; Boyle et al., 1981). In fact, the feed efficiency
response is one of the key factors that has maintained interest in the exploration of the catch-up growth phenomenon in farm animals, especially in terms of economics.

**Maintenance requirements and basal metabolic rate**

Sheely and Senior (1942) suggested that feed-restricted animals make greater weight gains upon realimentation than animals given *ad libitum* access to feed because their maintenance requirements are less. Brody (1945) related this to metabolic rate, metabolic body size (kg BW\(^{-2}\)) and possibly also to reduced motor activity. Thus, a greater proportion of net energy from a diet is available for productive processes and, thus, catch-up growth occurs. Wilson and Osbourn (1960) reported that several workers confirmed the finding that basal metabolism declines as energy intake is reduced (Blaxter and Wood, 1951 with calves; Quimby, 1948; Horst et al., 1934 with rats). Moreover, Forbes et al. (1934) and Kriss et al. (1934) presented data showing that heat increment decreased in response to reduced feed intake. Thus, animals seem to adapt to undernutrition by reducing their energy needs as basal metabolic rate declines and by decreasing the proportion of ingested energy dissipated as heat because of a decline in heat increment. Both adaptations contribute to increased efficiency of dietary energy utilization and, if they persist during realimentation, may partially explain the observed
increased weight gain per unit feed consumption. Other studies showed that the reduction in basal metabolic rate observed during feed restriction persisted during realimentation but that metabolic rate gradually returned to normal (Quimby et al., 1948; Cumming and Morrison, 1960). Animals that have been subjected to feed restriction and then realimentated only slowly increase their basal metabolic rate to the normal rate for the higher plane of nutrition (Wilson and Osbourn, 1960). This would result in a greater proportion of nutrients being available for productive purposes, especially growth, and would result in an increased growth rate compared with that shown by animals given ad libitum access to feed. This improved efficiency, however, would only be temporary and would decrease gradually throughout the period of realimentation. A recent report by Jones and Farrell (1992b) indicated an increased heat production upon realimentation of chickens previously restricted in feed intake. Furthermore, Zubair and Leeson (1994b) reported reduced basal metabolic rate (BMR) in feed-restricted chickens during the restriction period as compared with full-fed broilers. This reduced BMR, however, did not carry over into the refeeding period. These authors concluded that a reduced metabolic rate during realimentation does not play a role in the ability of birds to utilize feed more efficiently or to undergo catch-up growth.
Control and Regulation of Catch-up Growth

Present hypotheses to explain control and regulation of catch-up growth are purely speculative. The two current hypotheses proposed to explain the control mechanisms of catch-up growth are the "central control" hypothesis (Mosier, 1986) and the "peripheral control" hypothesis (Winick and Noble, 1966; Pitts, 1986). The "central control" hypothesis suggests that the body has a set-point for body size appropriate for a particular age and that this control resides in the central nervous system. Mosier and Jansons (1971) experimentally verified the importance of the central nervous system in the regulation of catch-up growth by showing that abnormal growth occurred after bilateral brain irradiation in rats. They postulated that brain structures lateral to the pituitary and hypothalamus played a role in the regulation of both normal and catch-up growth. The "peripheral control" hypothesis suggests that the control of body size resides in the tissues, where cell number or more accurately DNA determines the extent of growth following a period of undernutrition or illness.

A regulatory role for hormones, growth factors, or neurotransmitters in the process of catch-up growth seems likely but direct evidence for this has not been forthcoming. Dickerson and McAnulty, (1975) suggested that at the level of the whole organism, catch-up growth seems to reflect either the effects of growth-accelerating influence(s) or the repression of inhibitory
influence(s). At the level of the individual tissues, however, the process is complex and the component body parts catch-up at different rates. Furthermore, Williams et al. (1974, 1975) found that the rate of catch-up growth is influenced by the normal growth pattern: specifically, catch-up is the capacity of the organism to monitor its actual size relative to a preprogrammed, "intended" size to induce accelerated growth after release from the inhibitory influence and then to limit growth so that no more than the intended size is attained (Forbes, 1974; Mosier, 1978; Tanner, 1981). Leibel (1977) proposed that a hormone such as insulin could serve as a monitor of body size by "reading" the body adipose mass. He suggested that changes in the total adipose cell surface area might result in increases or decreases in the density of insulin receptors and, consequently, in altered ratios of insulin to some key metabolite, perhaps one important in neurotransmitter synthesis and in the regulation of appetite. In support of such a "radar" system, Roza et al. (1982) found that somatomedin-C/insulin-like growth factor I (Sm-C/IGF-I) correlated well with body mass. Other studies from the same laboratory showed that a preparation rich in Sm-C/Igf-I caused a decrease in food intake during 24 hours when infused into the cerebral ventricles of rats (Tannenbaum et al., 1983). If somatomedins play a role in appetite suppression, it is possible that the increased appetite often observed during catch-up growth may cease when body mass becomes appropriate for the
physiological age. Furthermore, somatomedin concentrations are a highly sensitive index of body nitrogen balance (Clemmons et al., 1981).

Few studies have addressed the role of circulating hormones in the process of catch-up growth. After rats are fasted, changes in growth hormone and somatomedin seem to be independent of actual catch-up growth because they return to normal whether catch-up growth occurs or not (Mosier, 1978). Other reports indicated that circulating growth hormone concentrations are typically elevated after prolonged energy deficiency (Daughaday et al., 1975; Blum et al., 1985) or prolonged protein deficiency (Scanes et al., 1981) which is paradoxical for animals with reduced growth rates. Thus, there is little evidence that hormones and growth factors alter the growth failure caused by undernutrition. Furthermore, the capacity for catch-up growth after the nutritional insult is removed is not readily explained by the action of hormones.

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EFFECT OF EARLY NUTRIENT RESTRICTION ON BROILER CHICKENS

1. PERFORMANCE AND DEVELOPMENT OF THE GASTROINTESTINAL TRACT

A paper submitted to *Poultry Science*

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**ABSTRACT**

An experiment was conducted to determine the effects of early nutrient restriction on performance and development of the gastrointestinal tract (GIT) of broiler chickens. Four hundred male broiler (Ross X Ross) chicks raised in floor pens were assigned to two treatment groups. One group was given *ad libitum* access to feed from 1 to 48 d of age. The second group was feed restricted from 7 to 14 d of age to an energy intake of 1.5 X BW$^{0.7}$ kcal ME/d, where BW is the body weight in grams, and then given *ad libitum* access to feed from 14 to 48 d. Body weight and feed intake were determined weekly. At 49 d of age, birds were processed for carcass yield, abdominal fat pad measurement, and body composition analysis. Broilers were also sampled at 7, 14, 21, and 41 d of age for proventriculus, gizzard, small intestine (duodenum, jejunum, and ileum), pancreas, and liver weights and for intestinal length measurements. Total DNA, protein:DNA, and RNA:DNA ratios of livers and jejuna were determined as indexes of
changes in cell size and number. Feed-restricted broilers failed to catch up to the Control birds in BW at 48 d of age but were superior (P < .01) in overall feed efficiency. No treatment effects were observed on breast meat yields or abdominal fat. Moreover, percentage carcass fat, crude protein, ash, and dry matter were not significantly affected by restricted feeding. Body weight and weights of gastrointestinal organs were reduced (P < .01) by feed restriction at 14 d of age. Restricted feeding, however, did not decrease the relative weights of organs, except for liver. Feed restriction also resulted in a reduction (P < .01) of liver cell number and size and a decrease in jejunum cell number. All organs recovered normal weight on refeeding and all cellular constituent ratios (e.g., RNA:DNA, RNA:protein, and protein:DNA) returned to normal by 41 d of age. Absolute and relative weights of supply organs (e.g., proventriculus, gizzard, small intestine, liver, and pancreas) were less affected by feed restriction and responded more quickly to refeeding than the whole body.

(Key words: broiler, nutrient restriction, performance, gastrointestinal tract)
When an animal, whose growth has been retarded by dietary restriction, is given adequate nutrition, it grows at a faster rate than an animal of the same age that had no restriction (Wilson and Osbourn, 1960). This rapid growth relative to age has been termed compensatory (Bohman, 1955) or catch-up growth (Prader et al., 1963). Most of the studies on early nutrient restriction with broilers have been concerned, for economic reasons, with growth performance (e.g., BW and feed efficiency) and carcass fat of the feed-restricted chickens as compared with full-fed chickens. Feed restriction programs applied to broiler chickens have produced varied responses with respect to these performance criteria (BW, feed efficiency, and carcass fat). In reviewing the literature, Yu and Robinson (1992) reported that factors such as the severity, timing, duration of feed restriction, feed intake during the period of refeeding, sex, or strain may affect the subsequent ability of broiler chickens to recover from a growth deficit. The reasons, however, for the relative success or failure to achieve full BW recovery following realimentation of the feed-restricted broiler chickens are still unknown. The phenomenon of catch-up growth in broiler chickens remains complex because the physiological, nutritional, metabolic, and endocrine aspects involved are not well understood. For instance, the role of the gastrointestinal tract
as a supply-organ system has not been extensively investigated in feed restriction studies with broilers. The gastrointestinal tract has a major role in supporting growth during the early posthatching period, and limitations in the allocation of energy for the growth of the gastrointestinal tract may limit energy availability for growth (Scott et al., 1991). Zubair and Leeson (1994) indicated that during feed restriction, weights of digestive organs (expressed as a percentage of BW) were generally heavier for feed-restricted broilers than for full-fed chickens.

The present study was conducted to obtain additional information on the effect of early nutrient restriction on the performance and carcass composition of broiler chickens. The second objective of our study was to examine the influence of early nutrient restriction and subsequent realimentation on the development of the gastrointestinal tract in relation to the growth of the whole body.

MATERIALS AND METHODS

Experimental Design

One-day-old male broiler (Ross X Ross) chicks obtained from a commercial hatchery were kept in floor pens and fed a broiler starter diet (Table 1) to 7 d of age. At this age, 16 groups of 25
chicks each were formed such that group weights were similar and each group constituted an experimental unit. Groups were randomly assigned to one of two treatments (8 replicate groups for each treatment) that consisted of providing feed for ad libitum intake from 7 to 48 d of age (Control) or restricting feed intake from 7 to 14 d to an energy intake of $1.5 \times \text{BW}^{67}$ ME kcal/d, where BW is the body weight in grams, then providing ad libitum access to feed from 14 to 48 d (Restricted). This feed restriction program is similar to that used by Plavnik and Hurwitz (1985). Chicks in both treatments were fed practical corn-soybean meal starter (1 to 21 d of age), grower (21 to 41 d of age), and finisher (41 to 48 d of age) diets that met or exceeded the National Research Council (1984) nutrient recommendations (Table 1).

Performance: Body Weight Gain, Feed Efficiency, and Carcass Traits

Body weight and feed efficiency (FE), calculated as feed to gain ratio, were measured at 14, 21, 28, 35, 41, and 48 d of age. At the end of the experiment (Day 49), four chickens were randomly selected from each pen and processed for carcass yield, abdominal fat pad measurement, and body composition analysis. Feed was withdrawn from these chickens, but water was provided for approximately 16 h before the chickens were processed at the Meat Laboratory, Iowa State University. Chickens were weighed individually, stunned electrically, and killed by exsanguination.
Carcasses were deplumed after a 2-min scald at 60 C and then were eviscerated. During evisceration, abdominal fat pads, including adipose tissue around the gizzard, were removed and weighed. After evisceration, carcasses were placed in ice water and chilled for 21 to 22 h. Chilled carcasses were removed from ice water, drained, and weighed. Yields of deboned breast meat were determined and the carcass, breast meat, and fat pad of the four broilers from each pen were placed in plastic bags and frozen at -20 C.

Carcass Composition

Frozen chicken carcasses were cut into sections by using a meat band saw\(^1\) and passed three times through a Buffalo N066BX, worm gear meat-grinder equipped with a .7-cm die. A sample of approximately 300 g was taken from each replicate batch consisting of four ground chicken carcasses. Two 10-g subsamples from each of these samples were placed in an oven at 70 C for 48 h to determine dry matter. Another 70-g subsample was pulverized by freeze grinding with liquid nitrogen and a commercial Waring blender\(^2\). The 70-g subsamples were subsequently lyophilized and oven dried. This sample preparation was done to minimize error due to bone chunks.

\(^1\)Model 50-12 Hobart Manufacturing Co., Des Moines, IA 50318.

\(^2\)Model 313L92, Waring Products Division, Dynamics Corp. of American, New Hartford, CT 06057.
Dry, pulverized samples were taken in duplicate for analysis of ether extract, CP (N X 6.25), and ash contents (Association of Official Analytical Chemists, AOAC, 1980).

Selected Characteristics of Gastrointestinal Tract: Physical Characteristics

On day 1 and 7 of age, 8 chicks were sampled for baseline measurements. Two chicks were chosen randomly from each pen for sampling at 14, 21, and 41 d of age. Because of a conflict of dates to sample birds, BW and feed consumption were recorded at 41 d instead of 42 d of age. Chicks were weighed and killed by cervical dislocation, and the small intestine, liver, and pancreas were excised and weighed. The small intestine was divided into three segments: duodenum (from ventriculus to pancreo-biliary ducts), jejunum (from pancreo-biliary ducts to yolk stalk), and ileum (from yolk stalk to ileo-cecal junction). Length of the segments of small intestine was measured. Segments were flushed with 10 to 20 mL of cold dionized water, and the weight of empty segments was recorded. Jejuna and livers were pooled by pen, placed in plastic bags, and frozen in liquid nitrogen. Samples were stored at -20 C until prepared for analysis. Weights of the proventriculus and gizzard were recorded after ingesta was removed.
Chemical Characteristics

Liver and jejunal samples (500 mg from each pooled sample) were homogenized with quantities of cold deionized water that resulted in a concentration of 50 mg of jejunum or liver tissue per mL of homogenate. Homogenizing was done with a Polytron at a speed of 6 (moderate) for 45 s. Aliquots of the jejunal and liver homogenates were taken for immediate determination of protein (Lowry et al., 1951). Deoxyribonucleic acid (DNA) and ribonucleic acid (RNA) were extracted by using a modified Schmidt-Thannhauser method as recommended by Munro and Fleck (1969). Deoxyribonucleic acid was measured by the modified diphenylamine method (Giles and Myers, 1965) with calf thymus DNA used as a standard. The RNA content was determined by the orcinol method (Munro and Fleck, 1966) with Bakers yeast RNA as a reference standard. Total organ DNA, protein:DNA, and RNA:DNA ratios were calculated to serve as indexes of changes in organ cell size and number.

Statistical Analysis

Data obtained were analyzed by analysis of variance (SAS Institute, 1985) to determine the effect of dietary treatments. Statistical analysis of percentage data was done after arc sine transformation (Snedecor and Cochran, 1980).

RESULTS

Performance: Body Weight and Feed Efficiency

At the end of the restriction period, BW of the Restricted group was significantly less (P < .01) than that of the Control group (Table 2). Indeed, BW of Restricted birds was 49% of that of the Control group at 14 d of age (170 vs. 349 g per broiler); then after allowing free access to feed, the BW difference between Restricted and Control groups decreased to 10% at 48 d of age (2,666 vs. 2,930 g per broiler). However, the difference in absolute BW between the Restricted and Control birds increased slightly (264 g) at 48 d of age as compared with 14 d (179 g).

Differences in BW gains during the various experimental periods from 14 to 35 d of age were significant between treatments groups but not for the periods of 35 to 41 or 41 to 48 d of age (Table 2). However, overall BW gain (7 to 48 d of age) of the Control group was significantly greater (P < .01) than that of the Restricted group (2,800 vs. 2,536 g broiler). During the restriction period (7 to 14 d of age), feed intake of the Restricted birds was 30% of that of those provided feed for ad libitum consumption (Table 3). A week later, (21 d of age) after allowing Restricted birds free access to feed, feed intake of the Restricted birds was 20% less than that of the Controls. Differences in feed intake between treatment groups diminished with
age, and no significant differences were noted from 41 to 48 d of age. At the end of the restriction period, feed:gain ratio of Restricted birds was significantly greater (P < .01) than that of the Control birds (Table 3). After allowing free access to feed, the feed:gain ratios of the Restricted birds were significantly less from 14 to 48 days of age. Consequently, Restricted birds were superior (P < .01) in overall feed conversion from 7 to 48 days of age. Overall mortality was low during the experiment, averaging 2 and 2.5% for Control and Restricted groups, respectively.

Selected Carcass Traits

Dressing percentage and carcass weights were significantly greater (P < .01 and P < .05, respectively) for the Control group than the Restricted group at 49 d of age (Table 4). Quantitative yields of breast meat and abdominal fat per carcass were significantly reduced by restricted feeding, but no treatment effects were observed for percentage yields of breast meat or abdominal fat pad. Proximate composition of the broiler carcasses is shown in Table 5. No treatment effects were observed for percentage carcass fat, crude protein, ash, and dry matter.
Digestive Organs: Physical Characteristics

Absolute weights of liver, pancreas, proventriculus, gizzard, and small intestine (duodenum, jejunum, ileum) of the Control birds were significantly greater than those of the Restricted birds at 14 and 21 d of age, but not at 41 d of age. Of the visceral organs studied, feed restriction had the most pronounced effect on liver weight. Liver weights of Restricted chickens were only 41% of those of the Controls at the end of the restriction period. At this age, liver weights relative to BW (g/100 g BW) of the Restricted birds also were less (P < .01) than those of Control chickens. However, after allowing ad libitum feed consumption, no significant differences were observed at 21 and 41 d of age (Figure 1). Relative weight of the pancreas was not affected by restricted feeding at 14 d of age. But at 21 and 41 d of age Restricted birds had greater (P < .02 and P < .01, respectively) pancreas weight (Figure 1). No differences were observed in relative weights of the proventriculus at 14 d and 21 d of age. At 41 d, however, relative weights of the proventriculus were significantly (P < .01) greater in the Restricted birds (Figure 2). Relative weights of gizzards of Control birds were less (P < .01) than those of Restricted birds at 14 and 21 d but not at 41 d of age (Figure 2).

The relative weights and densities (grams of weight per centimeter) of duodenum, jejunum, and ileum are shown in Table 6.
The relative proportion of BW, constituted by the small intestine (duodenum, jejunum and ileum), decreased from 7 d age through 41 d of age. For instance, the relative weight of the jejunum was 3.59% at 7 d of age but only 1.11 and 1.17% (for Control and Restricted birds, respectively) at 41 d of age. In contrast, during the same period, densities (grams per centimeter) of intestinal segments increased from .130 (jejunum) at 7 d of age to .373 and .337 for Control and Restricted birds, respectively, at 41 d of age.

Relative weight of the small intestine (duodenum, jejunum, and ileum) was not affected by restricted feeding at 14 d of age. But after realimentation, Restricted birds had greater duodena (P < .01), jeuna (P < .02), and ilea (P < .01) weights than Control birds at 21 d of age. By 41 d of age, significant differences were not observed between treatments. Small intestine (duodenum, jejunum, and ileum) density was significantly reduced at the end of the restriction period, and also 1 wk after restricted birds were allowed free access to feed. No treatment effects were observed at 41 d of age.

Chemical Characteristics: Liver Protein, RNA, and DNA

At the end of the restriction period, Restricted birds had greater (P < .01) concentration of protein and DNA in the liver (milligrams per gram of wet tissue) than Controls, but RNA concentration was not affected by treatments at 14, 21, and 41 d of age.
age. No differences were observed between treatments for DNA and protein concentration in livers at 21 d of age, but at 41 d of age Control birds had greater (P < .01) protein concentration in livers than Restricted birds. At this age, however, significant differences were not observed between treatments for DNA concentration (Figure 3).

Total protein, RNA, and DNA concentration of the liver were significantly less in feed-restricted birds at 14 d of age and 1 wk after the Restricted birds were allowed free access to feed. No treatment effects, however, were observed for total RNA and DNA at 41 d of age (Table 7). The reduced amounts of protein, RNA, and DNA in livers of Restricted birds corresponded with the decreased liver weights of this treatment group as compared with Control birds. Restricted birds had a greater (P < .03) relative liver DNA content (milligrams per 100 grams of BW) following feed restriction but a lower relative RNA content. Even though, Control birds had greater total liver protein at 14 and 41 d of age, significant differences were not observed between treatments for relative protein content of livers at 14, 21, and 41 d of age (Table 8). Liver RNA:DNA, RNA:protein, and protein:DNA ratios were significantly reduced by feed restriction at 14 d of age (Table 9), but no treatment effects were detected for these ratios at 21 and 41 d of age.
Jejunal Protein, RNA, and DNA

Restricted feeding reduced (P < .01) protein, RNA, and DNA concentration of jejuna (milligrams per gram of wet tissue) at 14 d of age. No differences were observed between treatments at 21 and 41 d of age (Figure 4). Total jejunal protein, RNA, and DNA was significantly greater in Control birds than in Restricted birds at 14 and 21 d but not at 41 d of age. The greater total jejunum protein, RNA, and DNA in the Control birds parallels their greater jejunum weight and body weight at 14 and 21 d of age (Table 7). Restricted birds had significantly lower (P < .01) relative protein, RNA, and DNA (milligrams per 100 grams of BW) contents of jejuna at the end of the restriction period. No treatment effects were observed at 21 and 41 d of age. The RNA:DNA ratio of the jejunum was less (P < .01) in the Restricted birds than in the Controls at 14 d of age but not at 21 or 41 d of age. The RNA:protein and protein:DNA ratios at 14, 21, or 41 d of age were not affected by restricted feeding (Table 9).

DISCUSSION

Broilers restricted in feed intake from 7 to 14 d of age failed to catch up in BW at 48 d to broilers given ad libitum access to feed from 1 to 48 d, but Restricted broilers were
superior in overall feed efficiency. No effects of feed restriction were observed on percentage yields of breast meat and abdominal fat pad. Similar observations have been reported for BW and abdominal fat pad percentage (Pinchasov and Jensen, 1989; Cabel and Waldroup, 1990; Summers et al., 1990) and for feed efficiency (Plavnik and Hurwitz, 1985; Plavnik et al., 1986; Fontana et al., 1992). However, our results are not in agreement with reports by Plavnik and Hurwitz (1985) and Plavnik et al. (1986) with respect to final BW and abdominal fat as a result of early feed restriction. In their original report, Plavnik and Hurwitz (1985) applied feed restriction to 7-d-old male broiler chicks (114 g BW) for 6 d. During this period, Restricted birds received 40 kcal per bird per d. Significant differences were not observed for final BW between Restricted broilers (2,257 g per broiler) and those given ad libitum access to feed (2,275 g per broiler) at the end of the 56 d growout period. Restricted feeding also resulted in a lower abdominal fat pad percentage and an overall improved feed efficiency. More recently, Plavnik and Hurwitz (1989) reported that male broilers that were restricted for 6 d, starting at 6 d of age, had BW at 55 d of age of 2,533 g per broiler versus 2,560 g per broiler for the chickens provided ad libitum access to feed. In the current study, 7-d-old male broiler chicks (130 g BW) were restricted for 7 d to an energy intake of approximately 40 kcal per
bird per d by using the formula of Plavnik and Hurwitz (1985), and this feed restriction reduced 48-d BW (2,930 and 2,666 g per broiler for Control and Restricted groups, respectively).

Moreover, no treatment effects were observed on abdominal fat pad percentage. Differences between our results and those reported by Plavnik and Hurwitz (1985, 1989) with respect to final BW may be attributed to broiler strains, growout duration, and type of diets fed (e.g., ME and protein contents). For instance, Plavnik and Hurwitz (1985) fed starter and grower diets that were lower in ME (2,900 and 3,000 kcal per kilogram, respectively) and protein (20.9 and 18.8%, respectively) contents than those fed in the present study. Even though it might be difficult to compare the broiler strain (Ross X Ross) of the present study with the one (White Rock) used by Plavnik and Hurwitz (1985) with respect to growth rate, the data suggest that a slower growing broiler strain was used by these workers. These data are supported by the report of Yu et al. (1990), who obtained greater BW performance at 56 d of age (2,982 and 2,684 grams per broiler for full-fed and feed-restricted broilers, respectively) than those of Plavnik and Hurwitz (1985). No effects of feed restriction were observed for percentage abdominal fat by Yu et al. (1990), who used a feed restriction program similar to the one applied by Plavnik and Hurwitz (1985).
Feed restriction programs with broilers have produced various responses in terms of catch-up growth, feed efficiency, and abdominal fat pad weight. For instance, Pinchasov and Jensen (1989) applied feed restriction (65 kcal ME per bird per g) to 7-d-old male broilers for 7 days. Final body weights were 2,239 g and 2,099 g per broiler, respectively, for Controls and Restricted chickens at 49 d of age. Restricted feeding adversely affected 49-d BW but no treatment effects were observed for abdominal fat pad.

Cabel and Waldroup (1990), using a feed restriction program similar to the one used by Plavnik et al. (1986), reported final body weights of 2,010 g and 1,900 g per broiler at 49 d of age for full-fed and restricted male broilers, respectively. These workers used diets similar to those of the present study with respect to ME and protein contents. Final BW was affected adversely by restricted feeding, but treatment effects were not observed on abdominal fat pad percentage. Restricted broilers were superior, however, in overall feed efficiency. The current results agree in many respects (BW, feed efficiency, abdominal fat-pad percentage) with those of Fontana et al. (1992, 1993) who used a feeding program (40 Kcal of ME per bird per day) similar to the one of Plavnik and Hurwitz (1985). These workers observed lighter BW but improved feed efficiency in feed-restricted groups as compared with the group given ad libitum access to feed at 14, 28, and 49 d of
age. No treatment effects were observed on abdominal fat pad percentage at these ages. As pointed out by Yu and Robinson (1992) in their review paper, factors such as the severity, timing, duration of feed restriction, feed intake during the period of refeeding, sex, or strain may affect the subsequent ability of broiler chickens to recover from a growth deficit.

Wilson and Osbourn (1960) suggested that increased appetite following refeeding is mainly responsible for improved growth and feed efficiency associated with compensatory growth. Even though Control birds had greater (absolute) feed intake than Restricted birds from day 14 to 41 d of age, our results suggest that the Restricted birds showed a greater feed intake relative to their BW after they were given free access to feed. For instance, 1 week after refeeding, BW of the Control and Restricted birds were 714 g and 508 g per broiler, respectively. During the same week, however, the Control and the Restricted groups consumed 534 g and 431 g per broiler, respectively. Moreover, Restricted birds had significantly lower feed:gain ratios than controls during the same period and up to 48 d of age.

In the present study, no treatment effects were observed on percentage carcass fat, crude protein, ash, and dry matter. These observations support recent findings by Fontana et al. (1993), who observed no differences in carcass fat, and carcass protein between feed-restricted and full-fed broilers at 28 and 49 d of age.
In the current study, BW and organ weights of the Restricted birds were significantly less than those of the Controls at the end of the restriction period. Gastrointestinal organs, however, responded more quickly to realimentation than the whole body. For instance, by 41 d of age, no differences were observed between treatments for organ weights, but at this age, Restricted birds were still significantly lighter than the Controls. These responses may be a result of more nutrients being used by Restricted birds to maintain the gastrointestinal system at the expense of the demand tissues (e.g., legs, and breast muscle).

Our results suggest also that the supply organs of Restricted birds need to catch up and eventually exceed those of the Controls before the whole body might catch up. An increased rate of growth of the visceral organs has been suggested as a possible underlying requisite for compensatory growth of the whole body of rats during realimentation following feed restriction (Anugwa and Pond, 1989). Upon realimentation, weekly BW gains of the Restricted birds were significantly smaller than those of Controls, but between 35 and 41 and 41 and 48 d of age, Restricted birds had gains equal to those of Control birds. The 35 to 41 d period also coincides with the disappearance of treatment effects for organ weights.

Even though the relative weights of the segments of the small intestine were not affected by restricted feeding, their densities
(weight/length) were significantly decreased at 14 d of age, indicating that the intestinal mucosa of restricted birds was thinner than that of controls. These observations agree with reports of Michael and Hodges (1973), showing some atrophy of the chicken small intestine during feed restriction. These workers observed slightly shorter and thinner villi in the Restricted birds as compared with Controls at the end of the 8-d feed restriction period. Michael and Hodges (1973), used chickens that were 6-wk-old and limited their feed intake to 25% of that of full-fed chickens.

In the current study, restricted feeding had no effect at 14 d of age on relative weight of small intestine (duodenum, jejunum, and ileum), pancreas, and proventriculus, but absolute and relative weights of liver were decreased. Restricted feeding also resulted in a greater relative gizzard weight at 14 d of age. These observations agree with those of Zubair and Leeson (1994) with respect to relative weights of the small intestine and pancreas as a result of early feed restriction. Zubair and Leeson (1994) used a feed restriction program that consisted of providing to restricted broilers 50% of the daily feed intake of the full-fed chickens from 6 to 12 d of age. Relative weights of crop, proventriculus, and gizzard were greater at 11 d of age for the restricted birds than with the full-fed birds. No treatment
effects were observed on relative weights of small intestine, large intestine, liver, and pancreas at this age but, 5 d after realimentation, the feed-restricted group showed greater pancreas and liver relative weights. The feed restriction program described herein was more severe than the one used in the study of Zubair and Leeson (1994) and this may explain the decrease in liver weight (absolute and relative) following restricted feeding in the present study.

After reviewing the literature, Nitsan (1985) stated that the response of the gastrointestinal tract to feed restriction may vary according to the type of restriction, and the magnitude of response seems to be related to the intensity of restriction. Nitsan (1985), concluded that although the crop and the gizzard are hypertrophied in all types of restrictions or meal feeding, the weight of the duodenum and small intestine was increased only when the time during which the chicks had access to the feed was limited (i.e., meal feeding) but not when feed was restricted with no time limit (i.e., feed restriction). This review by Nitsan (1985), however, referred to feed restriction studies conducted with chickens (meat and egg strains) that were at least 4 wk old when gastrointestinal tract measurements were made. It is difficult to compare our results with those obtained by other workers (Pinchasov et al., 1985; Katanbaf et al., 1989) because of differences in feed
restriction programs, breed of chickens, and length of growout periods. For instance, in the studies of Pinchasov et al. (1985), White Rock male chicks were deprived of feed on alternate days from 14 throughout 83 d of age, whereas daily feed restriction or skip-1-d or skip-2-d feeding programs were applied to meat type female breeders from 7 to 461 d by Katanbaf et al. (1989). Nevertheless, both groups of workers reported that restricted feeding (i.e., intermittent or alternate-day feeding) increased relative weights of gastrointestinal tract components.

Restricted feeding in the current research resulted in a decrease of total liver and jejunal protein, RNA, and DNA at the end of the restriction period and also for 7 d after allowing restricted birds free access to feed. Treatment effects were observed only at 14 d of age for relative (milligrams per 100 g BW) RNA and DNA contents, but relative liver protein was not affected by restricted feeding during the experiment. The decrease in total liver protein in the Restricted birds was a result of lighter liver weight rather than to changed protein concentration. Restricted birds had significantly greater liver DNA and protein concentrations at the end of the restriction period. Similar observations have been made by Burrin et al. (1988) in rats after nutrient deprivation. These workers, however, did not observe any treatment effect for protein concentration although RNA
concentration was significantly greater in fed than in fasted rats. The reduced total jejunal protein of the present study was attributed to both decreased weight and decreased protein concentration of jejuna at the end of the restriction period.

The ratio of protein to DNA can be used as an estimate of cell size (Winick and Noble, 1966). Restricted feeding significantly reduced liver cell size but jejunal cell size was not affected at 14 and 21 d of age. In our study, ratios of RNA:DNA and RNA:protein were significantly smaller in liver of Restricted chickens at the end of the restriction period than in Controls. These smaller ratios suggest that Restricted birds experienced a decrease in liver protein synthetic activity during feed restriction. In overall protein metabolism, the decrease in liver weight and protein:DNA indicates the significance of the liver as a labile source of protein that can be degraded to supply amino acid to peripheral tissues during nutrient deprivation (Burrin et al., 1988). Refeeding resulted in normal ratios of the cellular constituents, RNA:DNA, RNA:protein, and protein:DNA, and recovery of normal weight in all organs. In the present study, liver and jejenum water contents were not determined. However, there is some evidence from the literature (Harrison, 1953; Steiner et al. 1968), indicating that the loss of water and protein primarily account for
the decrease in small intestine and liver weights observed in rats following starvation. Plavnik and Hurwitz (1985) described the feed restriction program evaluated herein as a severe one. Therefore, it is plausible that some of the effects we observed on liver and small intestine were similar to those seen in short-term nutrient deprivation studies, (i.e., feed withdrawal).

Winick and Noble (1966) suggested that growth failure because of malnutrition, (i.e., caloric restriction) depends on the age at the onset of malnutrition, and the ability to recover also may be dependent on the cellular type (cell number or cell size) of growth failure produced. A reduction in cell number (in early stage of growth) results in permanent stunting, whereas reduction in cell size (in later stage of growth) may facilitate recovery of normal stature after refeeding. Thus, growth failure caused by caloric restriction is reversible as long as cell division has not been affected. Winick and Noble (1966) pointed out, however, that the duration of cell division varies in different organs; therefore, differential effects on organ recovery are possible following refeeding. Although restricted feeding in the current study resulted in a reduction of liver and jejunal cell number (i.e., decrease in total organ DNA) at 14 d of age, permanent stunting of these two organs was not observed and, upon realimentation, these organs returned to normal weight by 41 d of age. These results
seem to agree with reports indicating that cell proliferation continues in many tissues as long as the tissue is growing (Sands et al., 1979). Therefore, it is plausible that the feed-restricted birds might have caught up in BW with the full-fed birds if the refeeding period had extended beyond 49 d of age.

REFERENCES


TABLE 1. Composition and calculated analysis of the corn-soybean meal diet

<table>
<thead>
<tr>
<th>Ingredients and composition</th>
<th>Starter 1 to 21 d</th>
<th>Grower 21 to 41 d</th>
<th>Finisher 41 to 48 d</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ground corn</td>
<td>52.57</td>
<td>61.92</td>
<td>67.89</td>
</tr>
<tr>
<td>Soybean meal</td>
<td>35.11</td>
<td>27.82</td>
<td>23.32</td>
</tr>
<tr>
<td>Animal-vegetable fat</td>
<td>6.00</td>
<td>4.41</td>
<td>3.32</td>
</tr>
<tr>
<td>Menhaden fish meal</td>
<td>3.00</td>
<td>3.00</td>
<td>3.00</td>
</tr>
<tr>
<td>Limestone</td>
<td>1.29</td>
<td>1.20</td>
<td>1.11</td>
</tr>
<tr>
<td>Dicalcium phosphate</td>
<td>1.23</td>
<td>.99</td>
<td>.76</td>
</tr>
<tr>
<td>Mineral premix1</td>
<td>.30</td>
<td>.30</td>
<td>.30</td>
</tr>
<tr>
<td>Vitamin premix2</td>
<td>.30</td>
<td>.30</td>
<td>.30</td>
</tr>
<tr>
<td>DL-methionine (98%)</td>
<td>.20</td>
<td>.06</td>
<td>----</td>
</tr>
</tbody>
</table>

Calculated composition

<table>
<thead>
<tr>
<th></th>
<th>Starter</th>
<th>Grower</th>
<th>Finisher</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dry matter</td>
<td>90.67</td>
<td>90.47</td>
<td>90.35</td>
</tr>
<tr>
<td>ME&lt;sub&gt;n&lt;/sub&gt;, kcal/kg</td>
<td>3,200</td>
<td>3,200</td>
<td>3,200</td>
</tr>
<tr>
<td>CP</td>
<td>23</td>
<td>20</td>
<td>18</td>
</tr>
<tr>
<td>Methionine</td>
<td>.58</td>
<td>.42</td>
<td>.33</td>
</tr>
<tr>
<td>TSAA</td>
<td>.93</td>
<td>.72</td>
<td>.60</td>
</tr>
<tr>
<td>Lysine</td>
<td>1.53</td>
<td>1.16</td>
<td>1.01</td>
</tr>
<tr>
<td>Calcium</td>
<td>1.00</td>
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<td>.80</td>
</tr>
<tr>
<td>Available phosphorus</td>
<td>.45</td>
<td>.40</td>
<td>.35</td>
</tr>
</tbody>
</table>

<sup>1</sup>Supplied per kilogram of diet: Mn, 70 mg; Zn, 40 mg; Fe, 37 mg; Cu, 6 mg; Se, .15 mg; NaCl (I), 2.60 g.

<sup>2</sup>Supplied per kilogram of diet: vitamin A (retinyl acetate), 5,000 IU; cholecalciferol, 1,500 I.U.; vitamin E (dl-α-tocopheryl acetate), 15 IU; vitamin B<sub>12</sub>, 11 μg; menadione sodium bisulfite, 1.8 mg; riboflavin, 2.7 mg; pantothenic acid, 7 mg; niacin, 75 mg; choline, 509 mg; folic acid, 550 μg, biotin, 75 μg.
TABLE 2. Effect of early nutrient restriction on body weight and body weight gain of broiler chickens

<table>
<thead>
<tr>
<th>Treatment</th>
<th>7 d</th>
<th>14 d</th>
<th>21 d</th>
<th>28 d</th>
<th>35 d</th>
<th>41 d</th>
<th>48 d</th>
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</thead>
<tbody>
<tr>
<td></td>
<td>(g/chick)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Control</td>
<td>130</td>
<td>349</td>
<td>714</td>
<td>1,188</td>
<td>1,788</td>
<td>2,312</td>
<td>2,930</td>
</tr>
<tr>
<td>Restricted</td>
<td>130</td>
<td>170</td>
<td>508</td>
<td>954</td>
<td>1,509</td>
<td>2,028</td>
<td>2,666</td>
</tr>
<tr>
<td>SEM</td>
<td>1</td>
<td>2</td>
<td>4</td>
<td>10</td>
<td>15</td>
<td>21</td>
<td>25</td>
</tr>
</tbody>
</table>

Source of variation | Probability
Treatment          | .70    | .01   | .01   | .01   | .01   | .01   | .01   |

Source of variation | Probability
Age period          |         |       |       |       |       |       |       |
| 7 to 14 d         | 219     | 365    | 474   | 600   | 524   | 618   | 2,800 |
| 14 to 21 d        | 40      | 338    | 446   | 555   | 519   | 638   | 2,536 |
| 21 to 28 d        | 1       | 3      | 9     | 10    | 11    | 19    | 25    |
| 28 to 35 d        | 10      | 11     | 19    | 25    |       |       |       |
| 35 to 41 d        | 1       | 3      | 9     | 10    | 11    | 19    | 25    |
| 41 to 48 d        | .01     | .01    | .04   | .01   | .76   | .47   | .01   |
| 7 to 48 d         | .01     | .01    | .04   | .01   | .76   | .47   | .01   |

*Means of 8 pens of 20 broilers each.
2Control = full-fed throughout; Restricted = feed intake restricted from 7 to 14 d of age to maintenance requirements of 7-d-old chicks weighing 130 g each.
### TABLE 3. Effect of early nutrient restriction on feed intake and feed efficiency of broiler chickens

<table>
<thead>
<tr>
<th>Treatment</th>
<th>Age period (g intake/chick)</th>
<th>SEM</th>
<th>Source of variation</th>
<th>Probability</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>7 to 14 d</td>
<td>14 to 21 d</td>
<td>21 to 28 d</td>
<td>28 to 35 d</td>
</tr>
<tr>
<td>Control</td>
<td>291</td>
<td>534</td>
<td>676</td>
<td>1,117</td>
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<tr>
<td>Restricted</td>
<td>86</td>
<td>431</td>
<td>564</td>
<td>993</td>
</tr>
<tr>
<td>Source of variation</td>
<td>Treatment</td>
<td>.01</td>
<td>.01</td>
<td>.01</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Source of variation</th>
<th>Probability</th>
</tr>
</thead>
<tbody>
<tr>
<td>Feed efficiency(^2)</td>
<td></td>
</tr>
<tr>
<td>(g:g)</td>
<td></td>
</tr>
<tr>
<td>Control</td>
<td>1.32</td>
</tr>
<tr>
<td>Restricted</td>
<td>2.23</td>
</tr>
<tr>
<td>Source of variation</td>
<td>Treatment</td>
</tr>
</tbody>
</table>

\(^1\) Means of 8 pens of 20 broilers each.

\(^2\) Control = full-fed throughout; Restricted = feed intake restricted from 7 to 14 d of age to maintenance requirements of 7-d-old chicks weighing 130 g each.

\(^3\) Feed efficiency was adjusted for mortality by using the gains of the dead birds in the calculations.
<table>
<thead>
<tr>
<th>Treatment</th>
<th>Eviscerated Carcass (g)</th>
<th>(% of live weight)</th>
<th>Breast meat (g)</th>
<th>(% of carcass weight)</th>
<th>Abdominal fat pad (g)</th>
<th>(% of carcass weight)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td>2,060</td>
<td>73.16</td>
<td>379</td>
<td>18.4</td>
<td>64</td>
<td>3.11</td>
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<tr>
<td>Restricted</td>
<td>1,854</td>
<td>72.42</td>
<td>349</td>
<td>18.8</td>
<td>51</td>
<td>2.75</td>
</tr>
<tr>
<td>SEM</td>
<td>19</td>
<td>.24</td>
<td>4</td>
<td>.13</td>
<td>4</td>
<td>.18</td>
</tr>
</tbody>
</table>

Source of variation: Treatment

<table>
<thead>
<tr>
<th>Treatment</th>
<th>Probability</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td>.01</td>
</tr>
<tr>
<td>Restricted</td>
<td>.04</td>
</tr>
<tr>
<td>SEM</td>
<td>.01</td>
</tr>
<tr>
<td></td>
<td>.06</td>
</tr>
<tr>
<td></td>
<td>.02</td>
</tr>
<tr>
<td></td>
<td>.19</td>
</tr>
</tbody>
</table>

1 Means of 8 pens per treatment, 4 broilers per pen.
2 Control = full-fed throughout; Restricted = feed intake restricted from 7 to 14 d of age to maintenance requirements of the 7-d-old chicks weighing 130 g each.
3 Mean BW of processed broilers were 2,816 and 2,560 g/broiler for Control and Restricted groups, respectively.
Table 5. Effect of early nutrient restriction on carcass composition of broiler chickens

<table>
<thead>
<tr>
<th>Treatment</th>
<th>DM (%)</th>
<th>Protein (%)</th>
<th>Fat (%)</th>
<th>Ash (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td>34.28</td>
<td>45.18</td>
<td>45.24</td>
<td>7.32</td>
</tr>
<tr>
<td>Restricted</td>
<td>33.76</td>
<td>45.98</td>
<td>45.10</td>
<td>7.80</td>
</tr>
<tr>
<td>SEM</td>
<td>.41</td>
<td>.46</td>
<td>.72</td>
<td>.19</td>
</tr>
</tbody>
</table>

Source of variation: Treatment

<table>
<thead>
<tr>
<th>Treatment</th>
<th>Probability</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td>.39</td>
</tr>
<tr>
<td>Restricted</td>
<td>.25</td>
</tr>
<tr>
<td>SEM</td>
<td>.89</td>
</tr>
<tr>
<td>Means</td>
<td>.10</td>
</tr>
</tbody>
</table>

\(^1\) Means of 8 pens per treatment, 4 broilers per pen.
\(^2\) Control = full-fed throughout; Restricted = feed intake restricted from 7 to 14 d of age to maintenance requirements of 7-d-old chicks weighing 130 g each.
TABLE 6. Effect of early nutrient restriction on relative weight and weight per length of duodena, jejuna, and ilea of broiler chickens

<table>
<thead>
<tr>
<th>Treatment</th>
<th>Duodenum relative weight</th>
<th>Duodenum weight per length</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>7 d</td>
<td>14 d</td>
</tr>
<tr>
<td>Control</td>
<td>2.39</td>
<td>1.65</td>
</tr>
<tr>
<td>Restricted</td>
<td>2.39</td>
<td>1.47</td>
</tr>
<tr>
<td>SEM</td>
<td>----</td>
<td>.08</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Source of variation</th>
<th>Probability</th>
</tr>
</thead>
<tbody>
<tr>
<td>Treatment</td>
<td>---- .15 .01 .39</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Jejunum relative weight</th>
<th>Jejunum weight per length</th>
</tr>
</thead>
<tbody>
<tr>
<td>Treatment</td>
<td>(%</td>
</tr>
<tr>
<td>Control</td>
<td>3.59</td>
</tr>
<tr>
<td>Restricted</td>
<td>3.59</td>
</tr>
<tr>
<td>SEM</td>
<td>----</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Source of variation</th>
<th>Probability</th>
</tr>
</thead>
<tbody>
<tr>
<td>Treatment</td>
<td>---- .19 .02 .38</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Ileum relative weight</th>
<th>Ileum weight per length</th>
</tr>
</thead>
<tbody>
<tr>
<td>Treatment</td>
<td>(%</td>
</tr>
<tr>
<td>Control</td>
<td>2.43</td>
</tr>
<tr>
<td>Restricted</td>
<td>2.43</td>
</tr>
<tr>
<td>SEM</td>
<td>----</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Source of variation</th>
<th>Probability</th>
</tr>
</thead>
<tbody>
<tr>
<td>Treatment</td>
<td>---- .57 .01 .08</td>
</tr>
</tbody>
</table>

1Means of 8 pens per treatment, 2 broilers per pen.
2Control = full-fed throughout; Restricted = feed intake restricted from 7 to 14 d of age to maintenance requirements of 7-d-old chicks weighing 130 g each.
TABLE 7. Effect of early nutrient restriction on total DNA, RNA, and protein of livers and jejunum of broiler chickens

<table>
<thead>
<tr>
<th>Age (d)</th>
<th>Treatment</th>
<th>Liver DNA</th>
<th>Liver RNA</th>
<th>Liver Protein</th>
<th>Jejunum DNA</th>
<th>Jejunum RNA</th>
<th>Jejunum Protein</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Baseline</td>
<td>4.3</td>
<td>12.4</td>
<td>179</td>
<td>3.2</td>
<td>8.1</td>
<td>93</td>
</tr>
<tr>
<td>7</td>
<td>Baseline</td>
<td>27.3</td>
<td>61.2</td>
<td>923</td>
<td>19.8</td>
<td>43.1</td>
<td>590</td>
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<tr>
<td>14</td>
<td>Control</td>
<td>57.5</td>
<td>120.8</td>
<td>2,205</td>
<td>44.8</td>
<td>76.7</td>
<td>1,405</td>
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<td>1,022</td>
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<td>25.9</td>
<td>521</td>
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<tr>
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<td>4.8</td>
<td>2.6</td>
<td>67</td>
<td>1.7</td>
<td>3.1</td>
<td>58</td>
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<td>21</td>
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<td>209</td>
<td>4,418</td>
<td>66</td>
<td>100</td>
<td>2,186</td>
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<td>156</td>
<td>3,237</td>
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<td>76</td>
<td>1,682</td>
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<td>122</td>
<td>3</td>
<td>4</td>
<td>85</td>
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<tr>
<td>41</td>
<td>Control</td>
<td>143</td>
<td>483</td>
<td>10,189</td>
<td>86</td>
<td>183</td>
<td>3,999</td>
</tr>
<tr>
<td></td>
<td>Restricted</td>
<td>166</td>
<td>439</td>
<td>8,492</td>
<td>82</td>
<td>162</td>
<td>3,704</td>
</tr>
<tr>
<td></td>
<td>SEM</td>
<td>16</td>
<td>24</td>
<td>430</td>
<td>6</td>
<td>8</td>
<td>190</td>
</tr>
</tbody>
</table>

Source of variation

| 14 | Treatment | .01 | .01 | .01 | .01 | .01 | .01 |
| 21 | Treatment | .01 | .01 | .01 | .01 | .01 | .01 |
| 41 | Treatment | .21 | .33 | .01 | .66 | .10 | .29 |

1 Means of 8 pens per treatment, 2 broilers per pen.
2 Organ total DNA (mg) = [DNA concentration (mg/g) X organ weight (g)].
3 Organ total RNA (mg) = [RNA concentration (mg/g) X organ weight (g)].
4 Organ total protein (mg) = [protein concentration (mg/g) X organ weight (g)].
5 Control = full-fed throughout; Restricted = feed intake restricted from 7 to 14 d of age to maintenance requirements of 7-d-old chicks weighing 130 g each.
<table>
<thead>
<tr>
<th>Age (d)</th>
<th>Treatment</th>
<th>Liver DNA^a (mg/100 g BW)</th>
<th>Liver RNA^a (mg/100 g BW)</th>
<th>Liver Protein^a (mg/100 g BW)</th>
<th>Jejunum DNA (mg/100 g BW)</th>
<th>Jejunum RNA (mg/100 g BW)</th>
<th>Jejunum Protein (mg/100 g BW)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Baseline</td>
<td>10.3</td>
<td>29.5</td>
<td>425</td>
<td>7.6</td>
<td>19.2</td>
<td>222</td>
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<tr>
<td>7</td>
<td>Baseline</td>
<td>21.0</td>
<td>47.1</td>
<td>710</td>
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<td>33.1</td>
<td>454</td>
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<tr>
<td>14</td>
<td>Control</td>
<td>16.9</td>
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<td>650</td>
<td>13.3</td>
<td>22.8</td>
<td>416</td>
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<td>Restricted</td>
<td>22.3</td>
<td>29.0</td>
<td>620</td>
<td>9.6</td>
<td>15.7</td>
<td>314</td>
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<td>1.5</td>
<td>20</td>
<td>.7</td>
<td>1.2</td>
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<td>29.6</td>
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<td>14.2</td>
<td>310</td>
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Source of variation | Probability
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21 Treatment | .52 | .12 | .21 | .06 | .24 | .07
41 Treatment | .08 | .27 | .73 | .28 | .52 | .18

^aMeans of 8 pens per treatment, 2 broilers per pen.

^bRelative DNA concentration = (organ total DNA (mg) + BW (g)) X 100.

^cRelative RNA concentration = (organ total RNA (mg) + BW (g)) X 100.

^dRelative protein concentration = (organ total protein (mg) + BW (g)) X 100.

^eControl = full-fed throughout; Restricted = feed intake restricted from 7 to 14 d of age to maintenance requirements of 7-d-old chicks weighing 130 g each.

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Source of variation | Probability
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1 Means of 8 pens per treatment, 2 broilers per pen.
2 Control = full-fed throughout; Restricted = feed intake restricted from 7 to 14 d of age to maintenance requirements of 7-d-old chicks weighing 130 g each.
FIGURE 1. Relative weights of livers and pancreases (g/100g BW) of C and feed R broilers. Control = full-fed throughout; Restricted = feed intake restricted from 7 to 14 d of age to maintenance requirements of 7-d-old chicks weighing 130 g each. Asterisks indicate that means differ significantly (P < .05).
FIGURE 2. Relative weights of proventriculus and gizzard (g/100 g BW) of C and feed R broilers. Control = full-fed throughout; Restricted = feed intake restricted from 7 to 14 d of age to maintenance requirements of 7-d-old chicks weighing 130 g each. Asterisks indicate that means differ significantly (P < .05).
Proventriculus

Gizzard

Controlling factors:
- CONTROL
- RESTRICTED

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FIGURE 3. DNA, RNA and protein (milligrams per gram of wet tissue) of livers of C and feed R broilers. Control = full-fed throughout; Restricted = feed intake restricted from 7 to 14 d of age to maintenance requirements of 7-d-old chicks weighing 130 g each. Asterisks indicate that means differ significantly (P < .05).
FIGURE 4. DNA, RNA and protein (milligrams per gram of wet tissue) of jejunum of C and feed R broilers. Control = full-fed throughout; Restricted = feed intake restricted from 7 to 14 d of age to maintenance requirements of 7-d-old chicks weighing 130 g each. Asterisks indicate that means differ significantly (P < .05).
EFFECT OF EARLY NUTRIENT RESTRICTION ON BROILER CHICKENS

2. PERFORMANCE AND DIGESTIVE ENZYME ACTIVITIES

A paper submitted to Poultry Science

Pierre E. Palo, Jerry L. Sell, F. Javier Piquer, Lluis Vilaseca and Maria F. Soto-Salanova

ABSTRACT

An experiment was conducted to determine the effect of two early nutrient restriction programs on performance, selected characteristics of the gastrointestinal tract (GIT), and activities of digestive enzymes of broiler chickens. Three hundred and sixty male broiler (Ross X Ross) kept in floor pens were assigned to three groups. The control group (C) was given ad libitum access to feed from 1 to 48 d of age. Another group was restricted from 11 to 14 d (R4) to an energy intake of .74 x BW$^{0.67}$ and a third group was restricted from 7 to 14 d (R7) of age to an energy intake of 1.5 x BW$^{0.67}$ kcal ME per day. Then, both restricted groups were given ad libitum access to feed through 48 d. Body weight, and feed intake were determined weekly and selected carcass characteristics were measured at 48 d of age. Broilers also were sampled at 7, 14, 21, and 42 d of age to obtain data on components of the GIT and activities of selected digestive enzymes. Feed-restricted groups were significantly lighter (P < .01) at 14 and
48 d of age than the C group but were superior in overall feed efficiency.

No treatment effects were observed for percentage yields of breast meat and abdominal fat pad. Absolute weights of GIT components were significantly reduced by feed restriction at 14 d of age. But, GIT components increased in weight more quickly after refeeding than did the whole body. Restricted groups had significantly reduced (P < .01) specific activities of jejunal alkaline phosphatase, and pancreatic amylase, trypsin, and lipase as compared with the C group at 14 d of age but not at 21 and 42 d of age. Relative activities for jejunal maltase and sucrase were significantly greater (P < .01) at 21 d of age in the R4 and R7 groups as compared with the C group. The data show that feed restriction resulted in transient changes in organs and activities of digestive enzymes, suggesting a functional adaptation of these organs to feed restriction.

(Key words: broiler, nutrient restriction, performance, digestive enzymes)

INTRODUCTION

Recent research with broilers (Palo et al., 1994) has shown that relative weights of gastrointestinal tract (GIT) components (e.g., proventriculus, gizzard, small intestine, and pancreas) are
not adversely affected during or after a severe early nutrient restriction program. GIT components responded more quickly to refeeding than the whole body. Feed-restricted chickens, however, failed to catch up in BW with those given ad libitum access to feed to 48 d of age whereas treatment effects were not observed for absolute weight of GIT components by 41 d of age. Realimentation of the feed-restricted chickens resulted in transient increases in the relative weight of GIT components and improved feed efficiency. The nature and the physiological bases for these transient changes in organ weights in relation to ingestion and utilization of nutrients (i.e., improved feed efficiency) are not known.

The present study was conducted to investigate the effect of two early nutrient restriction programs on performance and activities of the digestive enzymes of broilers. Thus, activities of selected jejunal (maltase, sucrase, and alkaline phosphatase) and pancreatic (amylase, trypsin, and lipase) enzymes were measured during early nutrient restriction and subsequent realimentation of broiler chickens. Similar to our previous work (Palo et al., 1994) the current study also reexamines the influence of early nutrient restriction on selected characteristics of GIT components.
Experimental Design

One-day-old male broiler (Ross X Ross) chicks obtained from a commercial hatchery were kept in floor pens and fed a broiler starter diet to 7 d of age. At this age, 18 groups of 20 chicks were formed such that group weights were similar. Each group constituted an experimental unit. Groups were randomly assigned to one of the three treatments (6 replicate groups for each treatment). The treatments consisted of 1) providing feed for ad libitum intake from 7 to 48 d of age (C); 2) restricting feed intake from 11 to 14 d to an energy intake of .74 X BW$^{67}$ ME kcal per d (Jones and Farrell, 1992a) and then feeding ad libitum through 48 d of age (R4); and 3) restricting feed intake from 7 to 14 d to an energy intake of 1.5 X BW$^{67}$ kcal ME per day (Plavnik et al., 1985) then feeding ad libitum from 14 to 48 d (R7).

All chicks were fed practical corn-soybean meal starter (1 to 21 d), grower (21 to 42 d of age), and finisher (42 to 48 d) diets that met or exceeded National Research Council (1984) nutrient recommendations. The composition of the diets fed is shown in Table 1.
Performance

Body weight gain and feed efficiency (FE), calculated as feed to gain ratio, were measured at 14, 21, 28, 35, 42, and 48 d of age. At the end of the experiment (Day 49) four chickens were randomly selected from each pen and processed for carcass yield and abdominal fat pad measurements. Feed was withdrawn from these chickens, but water was provided for approximately 16 hours before the chickens were processed at the Meat Laboratory of Iowa State University. The processing procedures were described previously (Palo et al., 1994). During evisceration, abdominal fat pads including adipose tissue around the gizzard were removed and weighed. After evisceration, carcasses were placed in ice water, drained, and weighed. Yields of deboned breast meat were determined.

Selected Characteristics of GIT

At 7 d of age, ten chicks were sampled for base line measurements. Two chicks were chosen randomly from each pen for sampling at 14, 21, and 42 d of age. Chicks were weighed and killed by cervical dislocation. The GIT was excised as described previously (Palo et al., 1994). Weights of the proventriculus and gizzard were recorded after ingesta was removed. Liver was
excised, and weighed. The pancreas was removed from the duodenal loop, placed in preweighed vials and frozen in liquid nitrogen. The jejunum (segment between pancreas-biliary ducts and yolk stalk) was removed, and length was measured. The jejunum was flushed with 10 to 20 mL of cold, deionized water, and empty segment weight recorded. Ten cm of the middle portion of the jejunum were separated, pooled by pen, placed in preweighed vials, and frozen in liquid nitrogen. Samples were stored at -20 C until prepared for analysis.

Enzymes Analysis

Before analysis, samples were thawed and homogenized with quantities of cold deionized water that resulted in a concentration of 50 mg jejunum per mL of homogenate or 100 mg pancreas per mL of homogenate. Homogenizing was done by using a Polytron at a speed setting of 6 (moderate) for 30 sec. Aliquots of homogenates of jejunums and pancreata were taken for determination of protein concentration (Lowry et al., 1951) and enzyme assays. Jejunums were assayed for maltase (EC 3.2.1.20) and sucrase (EC 3.2.1.48) activities by using maltose and sucrose, respectively, as substrates (Dahlquist, 1964). Jejunal alkaline phosphatase (EC 3.1.3.1), was determined using Sigma kits (Sigma 104). Aliquots of pancreas homogenates were analyzed for amylase (EC 3.2.1.1), trypsin (EC 3.4.21.4), and lipase (EC 3.1.1.3) activities. Amylase
activity was determined by using the Phadebas blue starch procedure (Ceska et al., 1959). Trypsin activity was determined by the method of Erlanger et al. (1966). Lipase activity was determined by a modified procedure of Nitsan et al. (1974) as described by O'Sullivan et al. (1992), and was expressed as milligrams of naphthol released in 60 min at 37 C.

**Statistical Analysis**

Statistical analysis within each age was performed according to the General Linear Models of SAS (SAS Institute, 1985). When treatment effects were significant (P < .05), orthogonal contrasts were used to determine differences among means. Analysis of percentage data was done after arc sine transformation (Snedecor and Cochran, 1980).

**RESULTS**

**Performance: Body Weight Gain and Feed Efficiency**

Mean BW of chicks at the end of the restriction period were 387, 218, and 184 g per chick for C, R4 and R7 groups, respectively. As a result, the C group had a significantly greater

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1Kinematica PT 10-35, Brinkman Instruments, Westburg, NY 11590.

2Sigma Chemical Co., St. Louis, MO 63178-9916.
(P < .01) BW gain as compared with the Restricted groups at 14 d of age (Table 2). Significant differences in BW gain were not observed between the C and the R4 groups at 21 d of age, and through 48 d of age. The R7 group gained less BW (P < .01) than the C group through 28 d of age and then gains of these two groups were equal. The R4 group had greater (P < .01) BW gains than the R7 group at 14, and 21 d of age, but not at 28, 35, 42, and 48 d of age. Significant differences were observed in 48-d BW between the C (2,714 g per broiler) and the Restricted groups (2,528 and 2,462 g per broiler for R4 and R7, respectively). At this age, however, the difference between the R4 and R7 groups was not significant.

From Day 7 to 10, the R4 group consumed 37 grams of feed per bird per day (data not shown), and gained approximately 29 grams per bird per d. But during the feed restriction period (Day 11 to 14) they lost 3 g of BW per bird per d while receiving 9 g of feed per chicken daily. Consequently, the net gain of the R4 group from Day 7 to 14 was 74 g per bird. Chickens in the R7 group were fed 13 g of feed per chicken per d and gained approximately 6 g per bird daily during the restriction period (Day 7 to 14). The C group consumed 45 g of feed per bird, and gained 35 g per d per chicken during the same period.
Feed efficiencies of the Restricted groups were significantly poorer (P < .01) than that of the C group during the restriction period (Table 3). After allowing free access to feed, the feed efficiencies of the Restricted groups were significantly better than that of the C group (P < .01) at 21, 28, and 35 d of age, but not at 42, and 48 d of age. Restricted groups, however, were superior (P < .01) in overall feed efficiency as compared with the C group. The R7 group had a significantly (P < .01) better feed efficiency as compared with the R4 group at 14 d of age. One week after refeeding, significant differences were not observed between the Restricted groups, but at 28 d, the R7 group had a significantly better feed efficiency than the R4 group. No significant differences were observed between the Restricted groups at 35 or 48 d. Overall mortality was low during the experiment, averaging 2% for each treatment group.

Selected Carcass Traits

Treatment effects were not observed for dressing percentage, percentage yields of breast meat, or abdominal fat pad at 49 d (Table 4). Carcass weights and quantitative yields of breast meat were significantly (P < .01) reduced by restricted feeding. The R4 group had significantly less (P < .01) abdominal fat pad weight per carcass as compared with the C group (52 g vs 64 g), but differences (P > .07) were not observed between the R4 and R7
groups that had 52 and 57 g of abdominal fat pad per carcass, respectively. Indeed, significant differences were not observed between the Restricted groups for any of the carcass traits measured.

**Digestive Organs: Physical Characteristics**

Absolute weights of liver, pancreas, proventriculus, gizzard, and jejunum of the C birds at the end of the restriction period were significantly greater than those of the feed-restricted birds (data not shown). One week after refeeding, the C group still had significantly greater proventriculus, gizzard, and jejunum weights than the Restricted groups but no significant differences were observed for pancreas weight at this age. By 42 d of age, no significant differences in organ weights were observed between the Restricted and C groups, nor were differences observed between Restricted groups during the experiment. At the end of the restriction period, relative liver weights (grams per 100 g BW) were significantly decreased by restricted feeding (Figure 1), but, by 21 d of age, no significant differences were observed between the Restricted and the C groups. Differences were not observed between Restricted groups for liver weight at 14, 21, and 42 d of age. Feed-restricted groups had a significantly (P < .01) greater relative gizzard weight than the C group at 14 and 21 d of age but not at 42 d of age (Figure 2).
The R7 group had greater (P < .01) relative proventriculus weights than the C group at 14 and 21 d but not at 42 d of age (Figure 2). Significant differences were not observed, however, between the R4 and the C group at the end of the restriction period, but at 21 d of age, the R4 group had a greater (P < .04) proventriculus weight. By 42 d of age, no significant differences were observed between these two groups.

At the end of the restriction period, significant differences were not observed in relative weights of jejunum (Table 5) between the R7 and the C groups, but 1 wk after refeeding, relative weights of the jejunum of the R7 group were greater (P < .02) than those of the C group. By 42 d of age, no significant differences were observed between these two groups. No treatment effects were observed between the Restricted groups at 14, 21, and 42 d of age. No significant differences were observed in relative weight of jejunum at 14 d of age between the R4 and the C groups. But 1 wk after refeeding, the R4 group had greater (P < .01) jejunum relative weight as compared with the C group. At 42 d of age, no significant differences were observed between these two groups.

Restricted feeding resulted in lower densities (grams per centimeter) of jejunum (P < .01) in the R7 group as compared with the C group at 14 and 21 d of age but not 42 d of age. The C group had greater jejunum (Table 5) densities than the R4 group at the end of the restriction period, but by 21 d of age, no significant
differences were observed between these two groups. No significant differences were observed in jejunum density between the Restricted groups at 14, 21, and 42 d.

Selected Digestive Enzymes: Jejunal Maltase (M) and Sucrase (S)

No treatment effects were observed for maltase and sucrase activities at 14, 21, and 42 d of age (Tables 6 and 7, respectively). At the end of the restriction period, however, the C group had significantly (P < .01) greater total activities (μ moles substrate hydrolyzed per jejunum per hour) of M and S as compared with the Restricted groups (data not shown). Although total activities of jejunal M and S were significantly greater (P < .01) at 14 d of age for the C group than for the Restricted groups, no treatment effects were observed in the relative activities (units of activity per 100 g BW) of these enzymes. One week after Restricted groups were allowed free access to feed, relative jejunal M (Table 6) and S (Table 7) activities were significantly greater (P < .01) in the Restricted groups as compared with the C group, but by 42 d of age, no significant differences were observed between the Restricted and the C groups. No treatment effects were observed for M and S specific activities at 14, 21, and 42 d of age (Figure 3). No differences in M or S activities were observed between the Restricted groups.
Jejunal Alkaline Phosphatase (AP)

Restricted groups had lower (P < .01) total (Figure 3), relative, and specific activities (Table 8) of jejunal AP at the end of the restriction period than the C group. At 21, and 42 d of age, no significant differences in jejunal AP were observed between the Restricted and the C groups. At the end of the restriction period, jejunal AP specific activity of the Restricted groups was only 62% of the control value. From 14 to 21 d, there was a 28% decrease in AP specific activity of the C group, whereas there were 13 and 17% increases for R4 and R7 groups, respectively. No differences were observed in jejunal AP activities between the Restricted groups at 14, 21, and 42 d of age.

Pancreatic Amylase (A), Trypsin (T) and Lipase (L)

At the end of the restriction period, the C group had greater (P < .01) pancreatic A, T, and L activities per weight of tissue and relative to BW as compared with the Restricted groups (Tables 9, 10, and 11, respectively). However, no differences among treatments were observed at 21 and 42 d of age. The C group also had greater (P < .01) relative pancreatic A (Table 9), T (Table 10), and L (Table 11) activities than the Restricted groups at 14 d of age. But at 21 d of age, no significant differences were observed between the Restricted and the C groups for relative
pancreatic A and L. At this age, however, the R7 group had greater (P < .01) relative pancreatic T activity than the C group. By 42 d of age, no differences were observed between these two groups.

No significant differences in relative activities of pancreatic A, T, and L were observed between the R7 and the R4 groups at 14, 21, and 42 d of age. Restricted feeding also resulted in lower specific activities (P < .01) of pancreatic A, T, and L at 14 d of age, but not at 21 or 41 d of age (Figure 4).

**DISCUSSION**

In the present study, chickens were restricted either from 11 to 14 d to an energy intake of .74 X BW\(^{67}\) kcal ME per day (R4) or from 7 to 14 d of age to 1.5 X BW\(^{67}\) kcal ME per day (R7), and then, both groups were given *ad libitum* access to feed through 48 d.

Feed-restricted groups (R4 and R7) were significantly lighter than the C group at 14 and 48 d of age but were superior in overall feed efficiency. These findings agree with our previous results (Palo *et al.*, 1994) and other reports (Pinchasov and Jensen, 1989; Cabel and Waldroup 1990; Fontana *et al.*, 1992) showing that broiler chickens severely restricted in feed intake early in life were unable to catch up in BW with Controls by 48 d of age. Once again, as in our previous study (Palo *et al.*, 1994), the results are not in agreement with reports by Plavnik and Hurwitz (1985) and Plavnik *et al.* (1986) with respect to final BW and abdominal fat as a
result of the 7-d feed restriction program. Moreover, our results are not in agreement also with reports by Jones and Farrell (1992a) with respect to performance data of the 4 d restriction group. Jones and Farrell (1992a) feed restricted three Australian strains of broiler chicks to an energy intake of .74 X BW\(^{0.67}\) kcal ME/d for 4 d starting at 7 d of age. This feed restriction program provided 20% of ad libitum intake and was calculated to meet the requirement for BW stasis during the feed restriction program. Following realimentation, Jones and Farrell (1992a) reported no significant differences in BW at 49 d of age between feed-restricted chickens (1,891 g per broiler) and full-fed ones (1,896 g per broiler). No treatment effects were observed for feed intake and feed conversion ratio. However, weight of abdominal fat pad was significantly decreased by restricted feeding when considered on a g per kg BW basis.

In the current study, feed restriction was applied to 11-d-old broiler male chicks through 14 d of age by using the formula of Jones and Farrell (1992a). This feed restriction program also provided 20% of feed intake of the C group (45 g per broiler daily), but resulted in broilers that were significantly lighter (2,528 g) than the C group (2,714 g) at 48 d of age. Abdominal fat pad percentage was not affected by dietary treatment. Neither of the feed restriction programs tested herein maintained BW status during the restriction period. The R4 group lost 3 g BW per
broiler daily whereas the R7 group gained 6 g BW per broiler daily during their respective restriction periods. No significant differences were observed between the Restricted groups in BW or feed efficiency. The differences in performance data (final BW, feed efficiency, and fat pad weight) between the current study and the study of Jones and Farrell (1992) may be attributed to differences in strains and sex of broilers used or type of diets fed. Jones and Farrell (1992a) fed unsexed broiler chicks a starter diet containing 2,990 kcal ME/kg and 22% protein through 28 d of age, then, a finisher diet containing 19% protein and 2,990 kcal ME/kg was fed through 49 d of age. Yu and Robinson (1992) pointed out that factors such as the severity, timing, and duration of feed restriction, feed intake during the period of refeeding, and sex or strain have a significant effect on the subsequent ability of the broiler chicken to recover from a growth deficit. Feed restriction programs applied to relatively slow growing broiler strains (as in Jones and Farrell, 1992a study) may have resulted in a faster recovery in BW as compared with the fast growing strain used in the present study.

Feed restriction, in contrast to superalimentation, reduces gastrointestinal organ size in pigs (Pekas, 1986a, 1986b). Weight of gastrointestinal organs (gizzard, proventriculus, jejunum, pancreas, and liver) of the Restricted groups were significantly less at the end of the restriction period, but by 42 d of age,
there were no significant differences between the Restricted and the C groups. These results confirm our previous work (Palo et al., 1994) showing that gastrointestinal components respond more quickly to realimentation than the whole body. Again, it seems that supply organs need to catch up and eventually exceed those of the Controls before the whole body might catch up. Restricted feeding adversely affected the density (weight per length) of the jejunum. There is no indication, however, from the present study of which tissue component of the small intestine is most affected by restricted feeding. Studies with pigs (Pekas, 1983) have shown, however, that the responses of the small intestine to feed restriction are more specific to the mucosa. Pekas (1986a, 1986b) carried out morphometric measurements that involved the total cross section of the small intestine (circumsection) and found that mucosa accounted for 88% of the weight change of the small intestine, musculature accounted for 10% and the remainder (of tissue) accounted for 2%. In previous research, Pekas, (1983) indicated that mucosal responses to feed restriction were associated with responses of the villi. These findings with pigs, suggest that intestinal functions associated with the mucosa and epithelium are affected in proportion to the level of feed intake and to the duration of the feed restriction program. For instance, broilers given ad libitum access to feed had greater densities of the jejunum than feed-restricted broilers at the end of the
restriction period (14 d of age). But, 1 wk after realimentation, no significant differences were observed between the C and R4 groups, whereas the R7 group still had lower jejuna densities as compared with the C group. Moreover, the 4-d feed restriction program may be considered more severe than the 7-d feed restriction program in terms of the amount of feed served (9 g vs 13 g per broiler) to chickens during the restriction period. But, because of its shorter duration (4 d) this feed restriction program (R4) resulted in a faster recovery of the jejunum density (21 d of age) following realimentation as compared with the 7-d feed restriction program.

The increase in the relative weight of jejuna of the Restricted groups was accompanied by an increase in the relative activities of maltase and sucrase 1 wk (21 d of age) after Restricted groups were allowed free access to feed. This increase in relative activity of digestive enzymes could be the result of a greater mechanical stimulation of the intestinal wall during the first week of realimentation of the Restricted groups. Nitsan et al. (1974) indicated that the synthesis of enzymes of the digestive tract (i.e., small intestine and pancreas) is not predominantly controlled by the amount and concentration of substrates or products in the gastrointestinal tract but can be a result of mechanical and humoral stimulation caused by the passage of greater
amount of chyme through the digestive tract. During the first week of realimentation, feed engorgement was observed in the Restricted groups. Therefore, a greater mechanical stimulation of the intestinal wall might have occurred during this period in the Restricted groups, resulting in increased synthesis and secretion of intestinal enzymes (i.e., maltase, sucrase). Force-feeding studies with chicks have shown an increase in over-all secretion of digestive enzymes (pancreas and small intestine) that parallel the increase in feed intake. Moreover, despite an increased absolute weight of the pancreas and intestinal chyme, specific activities were the same in the force-fed and ad libitum fed groups, except for a greater activity of intestinal amylase (Nitsan et al., 1974).

In the current study the C group had significantly greater specific and relative jejunal AP activities as compared with the Restricted groups at the end of the restriction period. Then 1 wk after realimentation (21 d of age) a marked decrease in the AP activity of the C group (as compared with d 14) was observed whereas activity of the Restricted groups remained the same as at 14 d. The latter may correspond with a maintenance of a high level of nutrient absorption from the intestine of the Restricted groups. Our results are not totally in agreement with those reported by Michael and Hodges (1973). In the study of Michael and Hodges (1973), intestines from the feed-restricted birds showed some
atrophy, the villi being slightly shorter and thinner than normal after 8 d of restricted feeding. In contrast with our results, an increase in the activity of alkaline phosphatase, acid phosphatase, and leucine naphthylamidase was observed in the absorptive cells. These workers concluded that the enhanced absorption of nutrients in semi-starved animals was correlated with increased mucosal enzyme activities. However, it is not known whether this increase in enzyme activities persisted during the refeeding phase. Michael and Hodges (1973) used chickens that were 6-wk old and limited their feed intake to 25% of that of full-fed chickens.

The reduced feed intake of the Restricted groups during the restriction phase of the current study was accompanied by a significant decrease in relative and specific activities of pancreatic amylase, trypsin, and lipase as compared with the C group, suggesting an adaptation of these pancreatic enzymes to substrate levels. These observations agree with report by Corring, (1980) indicating that pancreatic protease and amylase secretions, as well as their concentrations in pancreatic tissue, were proportional to the amount of the substrate. For lipase, however, secretion and activity depend on dietary lipid but increases in secretion and activity is not proportional to the amount of this substrate (Lhoste et al., 1993). Hulan and Bird (1972) also reported that the level of lipase activity in pancreatic juice was significantly augmented by increasing dietary
fat intake. Moreover, studies with poults suggest that the activity of lipase depend on dietary fat levels (Krogdahl and Sell, 1989). Low activities were observed with low fat diets whereas with high fat diets a lag period of about 3 wk was followed by a five-fold increase in lipase activity. Corring (1980) reviewed the literature on the adaptation of digestive enzymes to the diet and indicated that when dietary restriction is not too severe, the biosynthesis of all digestive enzymes markedly decreases.

Our results suggest the presence of some compensatory mechanisms developed in the jejunum and pancreas after the restriction phase that might partly explain the overall improved feed efficiency of the Restricted groups. However, these compensatory mechanisms did not allow the Restricted groups to catch up with the C group in BW by 48 d of age. Another possible explanation of the observed improvement in feed efficiency of the Restricted groups is the decrease in maintenance energy expenditure during and after the restriction period that has been reported by several investigators. It has been suggested that restricted feeding reduces the maintenance requirements by reducing the loss of metabolic energy (total heat production) the basal metabolic rate, and the specific dynamic action of food (Mitchell, 1962; Griffiths et al., 1977; Apfelbaum, 1978; Forsum et al., 1981). This decrease in maintenance energy expenditure could be carried over into subsequent ad libitum refeeding and may be translated
into an improvement in the efficiency of feed utilization. But, a recent report by Jones and Farrell (1992b) does not support this hypothesis. These workers observed an increased heat production upon realimentation of chickens previously restricted in feed intake. Furthermore, Zubair and Leeson (1994) recently observed a reduced basal metabolic rate (BMR) in feed-restricted chickens as compared with full-fed broilers during the restriction period. This lower BMR, however, did not carry over into the refeeding period. From these observations, Zubair and Leeson (1994) concluded that altered metabolic rate during realimentation does not play a role in the ability of the chickens to undergo growth compensation and improved feed efficiency. These workers, rather, suggested that greater feed intake relative to BW and its associative digestive adaptations seem to be contributing factors to growth compensation.

Our results show that neither of the quantitative nutrient restriction programs tested resulted in complete BW recovery by 48 d of age. Both feed restriction programs could be characterized as severe ones, therefore, a mild quantitative feed restriction seems to be a more appropriate means to achieve nutrient restriction without the accompanying reduction in final BW observed in the present study. This conclusion is supported by the report of Plavnik and Hurwitz (1991) showing that broilers and turkeys subjected to mild nutrient restriction, that allowed for only 60 to
75% of normal growth during the restriction period, had greater final BW than controls that ate ad libitum. These workers suggested that this may offer an economic advantage over a continuous ad libitum feeding regimen, whereas our results show that those evaluated in the current research probably would not.

REFERENCES


TABLE 1. Composition and calculated analysis of the corn-soybean meal diet

<table>
<thead>
<tr>
<th>Ingredients and composition</th>
<th>Starter 1 to 21 d</th>
<th>Grower 21 to 42 d</th>
<th>Finisher 42 to 48 d</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ground corn</td>
<td>52.74</td>
<td>61.78</td>
<td>66.05</td>
</tr>
<tr>
<td>Soybean meal</td>
<td>35.02</td>
<td>27.97</td>
<td>26.86</td>
</tr>
<tr>
<td>Animal-vegetable fat</td>
<td>6.00</td>
<td>4.44</td>
<td>4.06</td>
</tr>
<tr>
<td>Menhaden fish meal</td>
<td>2.97</td>
<td>3.00</td>
<td>----</td>
</tr>
<tr>
<td>Limestone</td>
<td>1.29</td>
<td>1.20</td>
<td>1.24</td>
</tr>
<tr>
<td>Dicalcium phosphate</td>
<td>1.23</td>
<td>.99</td>
<td>1.19</td>
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<tr>
<td>Mineral premix</td>
<td>.30</td>
<td>.30</td>
<td>.30</td>
</tr>
<tr>
<td>Vitamin premix</td>
<td>.30</td>
<td>.30</td>
<td>.30</td>
</tr>
<tr>
<td>DL-methionine (98%)</td>
<td>.15</td>
<td>.02</td>
<td>----</td>
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</table>

Calculated composition

<table>
<thead>
<tr>
<th>Dry matter</th>
<th>ME(^{\text{\textsuperscript{1}}}) kcal/kg</th>
<th>CP(^{\text{\textsuperscript{1}}})</th>
<th>Methionine</th>
<th>TSAA</th>
<th>Lysine</th>
<th>Calcium</th>
<th>Available phosphorus</th>
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<tbody>
<tr>
<td>90.64</td>
<td>3,200</td>
<td>23</td>
<td>.53</td>
<td>.93</td>
<td>1.35</td>
<td>1.00</td>
<td>.45</td>
</tr>
<tr>
<td>90.47</td>
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<td>20</td>
<td>.38</td>
<td>.72</td>
<td>1.17</td>
<td>.90</td>
<td>.40</td>
</tr>
<tr>
<td>90.39</td>
<td>3,200</td>
<td>18</td>
<td>.32</td>
<td>.64</td>
<td>1.00</td>
<td>.80</td>
<td>.35</td>
</tr>
</tbody>
</table>

\(^{1}\)Supplied per kilogram of diet: Mn, 70 mg; Zn, 40 mg; Fe, 37 mg; Cu, 6 mg; Se, .15 mg; NaCl (I), 2.60 g.

\(^{2}\)Supplied per kilogram of diet: vitamin A (retinyl acetate), 5,000 IU; cholecalciferol, 1,500 I.U.; vitamin E (dl-\(\alpha\)-tocopheryl acetate), 15 IU; vitamin B\(_{2}\), 11 µg; menadione sodium bisulfite, 1.8 mg; riboflavin, 2.7 mg; pantothenic acid, 7 mg; niacin, 75 mg; choline, 509 mg; folic acid, 550 µg; biotin, 75 µg.
TABLE 2. Effect of early nutrient restriction on body weight gain and 48-d BW of broiler chickens

<table>
<thead>
<tr>
<th>Treatment</th>
<th>7 to 14 d</th>
<th>14 to 21 d</th>
<th>21 to 28 d</th>
<th>28 to 35 d</th>
<th>35 to 42 d</th>
<th>42 to 48 d</th>
<th>48-d BW</th>
</tr>
</thead>
<tbody>
<tr>
<td>C³</td>
<td>243*</td>
<td>383</td>
<td>461</td>
<td>540</td>
<td>530</td>
<td>415</td>
<td>2,714</td>
</tr>
<tr>
<td>R4</td>
<td>74</td>
<td>381</td>
<td>444</td>
<td>531</td>
<td>530</td>
<td>423</td>
<td>2,528</td>
</tr>
<tr>
<td>R7</td>
<td>40</td>
<td>360</td>
<td>434</td>
<td>521</td>
<td>528</td>
<td>435</td>
<td>2,462</td>
</tr>
<tr>
<td>SEM</td>
<td>----</td>
<td>2</td>
<td>5</td>
<td>9</td>
<td>15</td>
<td>24</td>
<td>33</td>
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</table>

Source of variation Probability

<table>
<thead>
<tr>
<th>Treatment</th>
<th>7 to 14 d</th>
<th>14 to 21 d</th>
<th>21 to 28 d</th>
<th>28 to 35 d</th>
<th>35 to 42 d</th>
<th>42 to 48 d</th>
<th>48-d BW</th>
</tr>
</thead>
<tbody>
<tr>
<td>C vs R4</td>
<td>.01</td>
<td>.01</td>
<td>.01</td>
<td>.36</td>
<td>.99</td>
<td>.78</td>
<td>.01</td>
</tr>
<tr>
<td>C vs R7</td>
<td>.01</td>
<td>.92</td>
<td>.05</td>
<td>----</td>
<td>----</td>
<td>----</td>
<td>.01</td>
</tr>
<tr>
<td>R4 vs R7</td>
<td>.01</td>
<td>.01</td>
<td>.01</td>
<td>----</td>
<td>----</td>
<td>----</td>
<td>.01</td>
</tr>
</tbody>
</table>

1Mean BW of chicks at 7 d of age was 144 g/chick.
2Mean BW of chicks at 14 d of age were 387, 218, and 184 g/broiler for C, R4, and R7 groups, respectively.
C³ = Full fed throughout; R4 = Feed intake restricted from 11 to 14 d of age to maintenance requirements of 11-d-old chicks weighing 230 g each; R7 = Feed intake restricted from 7 to 14 d of age to maintenance requirements of 7-d-old chicks weighing 144 g each.
Means of 6 pens of 20 broilers, each.
TABLE 3. Effect of early nutrient restriction on feed efficiency (feed:gain)\(^1\)
of broiler chickens

<table>
<thead>
<tr>
<th>Treatment</th>
<th>7 to 14 d</th>
<th>14 to 21 d</th>
<th>21 to 28 d</th>
<th>28 to 35 d</th>
<th>35 to 42 d</th>
<th>42 to 48 d</th>
<th>7 to 48 d</th>
</tr>
</thead>
<tbody>
<tr>
<td>C(^2)</td>
<td>1.28(^3)</td>
<td>1.40</td>
<td>1.80</td>
<td>1.91</td>
<td>2.14</td>
<td>2.60</td>
<td>1.97</td>
</tr>
<tr>
<td>R4</td>
<td>1.51</td>
<td>1.27</td>
<td>1.68</td>
<td>1.81</td>
<td>2.09</td>
<td>2.61</td>
<td>1.84</td>
</tr>
<tr>
<td>R7</td>
<td>2.35</td>
<td>1.27</td>
<td>1.64</td>
<td>1.81</td>
<td>2.07</td>
<td>2.49</td>
<td>1.86</td>
</tr>
<tr>
<td>SEM</td>
<td>.04</td>
<td>.01</td>
<td>.01</td>
<td>.02</td>
<td>.03</td>
<td>.06</td>
<td>.02</td>
</tr>
</tbody>
</table>

Source of variation | Probability
-------------------|------------
Treatment           | .01        | .01        | .01        | .01        | .16        | .34        | .01      |
C vs R4             | .01        | .01        | .01        | .01        | ----        | ----        | .01      |
C vs R7             | .01        | .01        | .01        | .01        | ----        | ----        | .01      |
R4 vs R7            | .01        | .75        | .01        | .69        | ----        | ----        | .72      |

\(^1\)Means of 6 pens of 20 broilers, each.
\(^2\)C = full fed throughout; R4 = feed intake restricted from 11 to 14 d of age to
maintenance requirements of 11-d-old chicks weighing 230 g each. R7 = feed intake restricted
from 7 to 14 d of age to maintenance requirements of 7-d-old chicks weighing 144 g.
\(^3\)Feed efficiency was adjusted for mortality by using the gains of the dead birds in the
calculations.
TABLE 4. Effect of early nutrient restriction on carcass characteristics of broiler chickens at 49 d of age

<table>
<thead>
<tr>
<th>Treatment</th>
<th>Eviscerated Carcass (g)</th>
<th>(g) (% of live weight)</th>
<th>Breast meat</th>
<th>(g) (% of carcass weight)</th>
<th>Abdominal fat pad</th>
<th>(g) (% of carcass weight)</th>
</tr>
</thead>
<tbody>
<tr>
<td>C²</td>
<td>2,038³</td>
<td>74.10</td>
<td>386</td>
<td>18.93</td>
<td>64</td>
<td>3.14</td>
</tr>
<tr>
<td>R4</td>
<td>1,833</td>
<td>73.59</td>
<td>331</td>
<td>18.04</td>
<td>52</td>
<td>2.80</td>
</tr>
<tr>
<td>R7</td>
<td>1,798</td>
<td>73.12</td>
<td>328</td>
<td>18.21</td>
<td>57</td>
<td>3.16</td>
</tr>
<tr>
<td>SEM</td>
<td>25</td>
<td>.31</td>
<td>8</td>
<td>.30</td>
<td>3</td>
<td>.12</td>
</tr>
</tbody>
</table>

Source of variation | Probability
-------------------|-------------------
Treatment           | .01               | .12               | .01               | .12               | .01               | .10                     |
C vs R4             | .01               | ----              | .01               | ----              | .01               | ----                     |
C vs R7             | .01               | ----              | .01               | ----              | .07               | ----                     |
R4 vs R7            | .32               | ----              | .77               | ----              | .18               | ----                     |

¹Means of 6 pens per treatment, 4 broilers per pen.
²C = full fed throughout; R4 = feed intake restricted from 11 to 14 d of age to maintenance requirements of 11-d-old chicks weighing 230 g each; R7 = feed intake restricted from 7 to 14 d of age to maintenance requirements of 7-d-old chicks weighing 144 g each.
³Mean BW of processed broilers were 2,750, 2,491, and 2,458 g/broiler for C, R4, and R7 groups, respectively.
TABLE 5. Effect of early nutrient restriction on relative weight and weight per length of jejunum of broiler chickens

<table>
<thead>
<tr>
<th>Treatment</th>
<th>Jejunum relative weight (g/100 g BW)</th>
<th>Jejunum weight per length (g/cm)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>7 d</td>
<td>14 d</td>
</tr>
<tr>
<td>C</td>
<td>3.15</td>
<td>2.23</td>
</tr>
<tr>
<td>R4</td>
<td>3.15</td>
<td>2.08</td>
</tr>
<tr>
<td>R7</td>
<td>3.15</td>
<td>2.20</td>
</tr>
<tr>
<td>SEM</td>
<td>----</td>
<td>.08</td>
</tr>
</tbody>
</table>

Source of variation Probability

<table>
<thead>
<tr>
<th>Source of variation</th>
<th>Probability</th>
</tr>
</thead>
<tbody>
<tr>
<td>Treatment</td>
<td>---- .37 .01 .83 ---- .01 .03 .73</td>
</tr>
<tr>
<td>C vs R4</td>
<td>---- ---- .01 ---- ---- .01 .18 ----</td>
</tr>
<tr>
<td>C vs R7</td>
<td>---- ---- .02 ---- ---- .01 .01 ----</td>
</tr>
<tr>
<td>R4 vs R7</td>
<td>---- ---- .45 ---- ---- .07 .14 ----</td>
</tr>
</tbody>
</table>

1C = full fed throughout; R4 = feed intake restricted from 11 to 14 d of age to maintenance requirements of 11-d-old chicks weighing 230 g each; R7 = feed intake restricted from 7 to 14 d of age to maintenance requirements of 7-d-old chicks weighing 144 g each.

2Means of 6 pens per treatment, 2 broilers per pen.
### TABLE 6. Effect of early nutrient restriction on jejunal maltase activity of broiler chickens

<table>
<thead>
<tr>
<th>Treatment</th>
<th>Activity 7 d</th>
<th>Activity 14 d</th>
<th>Activity 21 d</th>
<th>Activity 42 d</th>
<th>Relative activity 7 d</th>
<th>Relative activity 14 d</th>
<th>Relative activity 21 d</th>
<th>Relative activity 42 d</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>(μmoles of maltose hydrolyzed/100 mg jejunal tissue hourly)</td>
<td>(μmoles of maltose hydrolyzed/100 g BW hourly)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>C²</td>
<td>83</td>
<td>167³</td>
<td>170</td>
<td>216</td>
<td>2,590</td>
<td>3,699</td>
<td>3,011</td>
<td>2,130</td>
</tr>
<tr>
<td>R4</td>
<td>83</td>
<td>186</td>
<td>202</td>
<td>249</td>
<td>2,590</td>
<td>3,862</td>
<td>4,122</td>
<td>2,543</td>
</tr>
<tr>
<td>R7</td>
<td>83</td>
<td>173</td>
<td>207</td>
<td>266</td>
<td>2,590</td>
<td>3,792</td>
<td>4,091</td>
<td>2,744</td>
</tr>
<tr>
<td>SEM</td>
<td>---</td>
<td>9</td>
<td>13</td>
<td>21</td>
<td>---</td>
<td>200</td>
<td>253</td>
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Source of variation

<table>
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<tbody>
<tr>
<td>Treatment</td>
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<tr>
<td>C vs R4</td>
</tr>
<tr>
<td>C vs R7</td>
</tr>
<tr>
<td>R4 vs R7</td>
</tr>
</tbody>
</table>

¹Relative activity = [((Activity/g of tissue/h) * (jejenum wt)) + BW] / 100.
²C = full fed throughout; R4 = feed intake restricted from 11 to 14 d of age to maintenance requirements of 11-d-old chicks weighing 230 g each; R7 = feed intake restricted from 7 to 14 d of age to maintenance requirements of 7-d-old chicks weighing 144 g each.
³Means of 6 pens per treatment, 2 broilers per pen.
# TABLE 7. Effect of early nutrient restriction on jejunal sucrase activity of broiler chickens

<table>
<thead>
<tr>
<th>Treatment</th>
<th>Activity 7 d</th>
<th>14 d</th>
<th>21 d</th>
<th>42 d</th>
<th>Relative activity 7 d</th>
<th>14 d</th>
<th>21 d</th>
<th>42 d</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>(μmoles of sucrose hydrolyzed/100 mg jejunal tissue hourly)</td>
<td>(μmoles of sucrose hydrolyzed/100 g BW hourly)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>C²</td>
<td>13.5</td>
<td>22.9³</td>
<td>28.5</td>
<td>43.0</td>
<td>425</td>
<td>506</td>
<td>506</td>
<td>426</td>
</tr>
<tr>
<td>R4</td>
<td>13.5</td>
<td>24.8</td>
<td>31.2</td>
<td>44.8</td>
<td>425</td>
<td>515</td>
<td>638</td>
<td>459</td>
</tr>
<tr>
<td>R7</td>
<td>13.5</td>
<td>23.4</td>
<td>34.3</td>
<td>50.6</td>
<td>425</td>
<td>512</td>
<td>678</td>
<td>519</td>
</tr>
<tr>
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<td>----</td>
<td>1.4</td>
<td>2.4</td>
<td>3.9</td>
<td>----</td>
<td>29</td>
<td>44</td>
<td>45</td>
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Source of variation

<table>
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<tr>
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</tr>
</thead>
<tbody>
<tr>
<td>Treatment</td>
</tr>
<tr>
<td>C vs R4</td>
</tr>
<tr>
<td>C vs R7</td>
</tr>
<tr>
<td>R4 vs R7</td>
</tr>
</tbody>
</table>

¹Relative activity = \[((Activity/g of tissue/h) X (jejunal wt)) + BW\] X 100.
²C = full fed throughout; R4 = feed intake restricted from 11 to 14 d of age to maintenance requirements of 11-d-old chicks weighing 230 g each; R7 = feed intake restricted from 7 to 14 d of age to maintenance requirements of 7-d-old chicks weighing 144 g each.
³Means of 6 pens per treatment, 2 broilers per pen.
TABLE 8. Effect of early nutrient restriction on jejunal alkaline phosphatase activity of broiler chickens

<table>
<thead>
<tr>
<th>Treatment</th>
<th>Activity (Sigma Units(^4))</th>
<th>Relative activity(^1) (Sigma Units/100 g BW)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>7 d</td>
<td>14 d</td>
</tr>
<tr>
<td>C(^3)</td>
<td>3.45</td>
<td>6.60(^4)</td>
</tr>
<tr>
<td>R4</td>
<td>3.45</td>
<td>4.05</td>
</tr>
<tr>
<td>R7</td>
<td>3.45</td>
<td>3.80</td>
</tr>
<tr>
<td>SEM</td>
<td>----</td>
<td>.50</td>
</tr>
</tbody>
</table>

Source of variation | Probability
--- | --- | --- | --- | --- | --- | --- |
Treatment | .01 | .53 | .23 | --- | .01 | .06 | .41 |
C vs R4 | ---- | .01 | ---- | ---- | ---- | ---- | ---- |
C vs R7 | ---- | .01 | ---- | ---- | ---- | ---- | ---- |
R4 vs R7 | ---- | .72 | ---- | ---- | ---- | ---- | ---- |

\(^1\)Relative activity = [(Activity/g of tissue) X (jejunum wt)) + BW] X 100.
\(^2\)Sigma units express the activity per 5 mg of jejunal tissue for 10 min at 37 C.
\(^3\)C = full fed throughout; R4 = feed intake restricted from 11 to 14 d of age to maintenance requirements of 11-d-old chicks weighing 230 g each; R7 = feed intake restricted from 7 to 14 d of age to maintenance requirements of 7-d-old chicks weighing 144 g each.
\(^4\)Means of 6 pens per treatment, 2 broilers per pen.
TABLE 9. Effect of early nutrient restriction on pancreatic amylase activity of broiler chickens

<table>
<thead>
<tr>
<th>Treatment</th>
<th>Activity</th>
<th>Relative activity$^1$</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>7 d</td>
<td>14 d</td>
</tr>
<tr>
<td></td>
<td>(µmoles of substrate hydrolyzed/g of pancreatic tissue per minute)</td>
<td>(µmoles of substrate hydrolyzed/100 g BW per minute)</td>
</tr>
<tr>
<td>C$^2$</td>
<td>2,365</td>
<td>2,008$^3$</td>
</tr>
<tr>
<td>R4</td>
<td>2,365</td>
<td>1,546</td>
</tr>
<tr>
<td>R7</td>
<td>2,365</td>
<td>1,312</td>
</tr>
<tr>
<td>SEM</td>
<td>----</td>
<td>102</td>
</tr>
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</table>

Source of variation: Probability

<table>
<thead>
<tr>
<th>Treatment</th>
<th>Probability</th>
</tr>
</thead>
<tbody>
<tr>
<td>Treatment</td>
<td>.01 .97 .70</td>
</tr>
<tr>
<td>C vs R4</td>
<td>.01 ---- ----</td>
</tr>
<tr>
<td>C vs R7</td>
<td>.01 ---- ----</td>
</tr>
<tr>
<td>R4 vs R7</td>
<td>.13 ---- ----</td>
</tr>
</tbody>
</table>

$^1$Relative activity = (((Activity/g of tissue/min) X (pancreas wt)) / BW) X 100.
$^2$C = full fed throughout; R4 = feed intake restricted from 11 to 14 d of age to maintenance requirements of 11-d-old chicks weighing 230 g each; R7 = feed intake restricted from 7 to 14 d of age to maintenance requirements of 7-d-old chicks weighing 144 g each.
$^3$Means of 6 pens per treatment, 2 broilers per pen.
### TABLE 10. Effect of early nutrient restriction on pancreatic trypsin activity of broiler chickens

<table>
<thead>
<tr>
<th>Treatment</th>
<th>Activity (μmoles of substrate hydrolyzed/g of pancreatic tissue hourly)</th>
<th>Relative activity1 (μmoles of substrate hydrolyzed/100 g BW hourly)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>7 d</td>
<td>14 d</td>
</tr>
<tr>
<td>C</td>
<td>17.1</td>
<td>15.6</td>
</tr>
<tr>
<td>R4</td>
<td>17.1</td>
<td>10.9</td>
</tr>
<tr>
<td>R7</td>
<td>17.1</td>
<td>11.0</td>
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<tr>
<td>SEM</td>
<td>----</td>
<td>.8</td>
</tr>
</tbody>
</table>

Source of variation | Probability
--- | ---
Treatment | .01 | .24 | .41 | .01 | .04 | .13
C vs R4 | .01 | ---- | ---- | ---- | .01 | .61 | ----
C vs R7 | .01 | ---- | ---- | ---- | .01 | .01 | ----
R4 vs R7 | .96 | ---- | ---- | ---- | .78 | .05 | ----

1Relative activity = \(((\text{Activity/g of tissue/h}) \times (\text{pancreas wt})) + \text{BW}) \times 100.

C = full fed throughout; R4 = feed intake restricted from 11 to 14 d of age to maintenance requirements of 11-d-old chicks weighing 230 g each; R7 = feed intake restricted from 7 to 14 d of age to maintenance requirements of 7-d-old chicks weighing 144 g each.

Means of 6 pens per treatment, 2 broilers per pen.
TABLE 11. Effect of early nutrient restriction on pancreatic lipase activity of broiler chickens

<table>
<thead>
<tr>
<th>Treatment</th>
<th>Activity (mg of naphtol released/g of pancreatic tissue hourly)</th>
<th>Relative activity (mg of naphtol released/100 g BW hourly)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>7 d</td>
<td>14 d</td>
</tr>
<tr>
<td>C²</td>
<td>62.9</td>
<td>66.4³</td>
</tr>
<tr>
<td>R4</td>
<td>62.9</td>
<td>30.7</td>
</tr>
<tr>
<td>R7</td>
<td>62.9</td>
<td>19.4</td>
</tr>
<tr>
<td>SEM</td>
<td>----</td>
<td>8.4</td>
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</tbody>
</table>

Source of variation

<table>
<thead>
<tr>
<th></th>
<th>Probability</th>
</tr>
</thead>
<tbody>
<tr>
<td>Treatment</td>
<td>---- .01</td>
</tr>
<tr>
<td>C vs R4</td>
<td>---- .01</td>
</tr>
<tr>
<td>C vs R7</td>
<td>---- .01</td>
</tr>
<tr>
<td>R4 vs R7</td>
<td>---- .36</td>
</tr>
</tbody>
</table>

¹Relative activity = [(Activity/g of tissue/h) X (pancreas wt)) + BW] X 100.
²C = full fed throughout; R4 = feed intake restricted from 11 to 14 d of age to maintenance requirements of 11-d-old chicks weighing 230 g each; R7 = feed intake restricted from 7 to 14 d of age to maintenance requirements of 7-d-old chicks weighing 144 g each.
³Means of 6 pens per treatment, 2 broilers per pen.
FIGURE 1. Relative weights of livers and pancreases (g/100 g BW) of C and R broilers. C = full-fed throughout; R4 = feed intake restricted from 11 to 14 d of age to maintenance requirements of 11-d-old chicks weighing 230 g each; R7 = feed intake restricted from 7 to 14 d of age to maintenance requirements of 7-d-old chicks weighing 144 g each. Bars that have no common letters within an age are significantly different (P < .05).
FIGURE 2. Relative weights of proventriculus and gizzard (g/100 g BW) of C and R broilers. C = full-fed throughout; R4 = feed intake restricted from 11 to 14 d of age to maintenance requirements of 11-d-old chicks weighing 230 g each; R7 = feed intake restricted from 7 to 14 d of age to maintenance requirements of 7-d-old chicks weighing 144 g each. Bars that have no common letters within an age are significantly different (P < .05).
FIGURE 3. Specific activities of jejunal maltase, sucrase, and alkaline phosphatase of C and R broilers. C = full-fed throughout; R4 = feed intake restricted from 11 to 14 d of age to maintenance requirements of 11-d-old chicks weighing 230 g each; R7 = feed intake restricted from 7 to 14 d of age to maintenance requirements of 7-d-old chicks weighing 144 g each. Specific activities are expressed in n moles of maltose hydrolyzed/mg protein/hourly, in n moles of sucrose hydrolyzed/mg protein/hourly, and in Sigma Units/mg protein for maltase, sucrase, and alkaline phosphatase, respectively. Bars that have no common letters within an age are significantly different ($P < .05$).
Specific activities of pancreatic amylase, trypsin, and lipase of C and R broilers. C = full-fed throughout; R4 = feed intake restricted from 11 to 14 d of age to maintenance requirements of 11-d-old chicks weighing 230 g each; R7 = feed intake restricted from 7 to 14 d of age to maintenance requirements of 7-d-old chicks weighing 144 g each. Specific activities are expressed in μ moles of substrate hydrolyzed/mg protein per minute, in μ moles of substrate hydrolyzed/mg protein/hourly, and μg of naphthol released/mg protein/hourly for amylase, trypsin, and lipase, respectively. Bars that have no common letters within an age are significantly different (P < .05).
GENERAL CONCLUSIONS

The research reported herein had two objectives: (1) to investigate the effect of early nutrient restriction on growth performance (e.g., body weight and feed efficiency), and carcass fat of broiler chickens, and (2) to examine the influence of early nutrient restriction and subsequent realimentation on the development of selected characteristics of the gastrointestinal tract in relation to the growth of the whole body.

The results show that neither of the quantitative nutrient restriction programs tested resulted in complete body weight recovery by 48 days of age (34 days of realimentation), but feed-restricted birds were superior in overall feed efficiency. Moreover, no effects of feed restriction were observed on percentage yields of breast meat, and abdominal fat pad. Indeed, both feed restriction programs can be characterized as severe ones, therefore, a mild quantitative feed restriction (50 to 75% of the control intake) might be a more appropriate mean to achieve nutrient restriction without the accompanying reduction in final body weight observed in the present research. Whether market broilers achieve total catch-up growth following an early feed restriction is still open to question. Evidence provided by the present research and by other workers seems to answer "No" - but again factors such as the severity of restriction, timing, duration
of feed restriction, feed intake during the period of refeeding, sex, and strain of broilers may affect the subsequent ability of broiler chickens to recover from a growth deficit.

The present research also indicates that despite the decrease in absolute weight of GIT components at the end of the restriction period, relative weight (expressed as a percentage of body weight) of these organs are not adversely affected during a severe early nutrient restriction program. Moreover, realimentation of the feed-restricted chickens resulted in transient increases in the relative weight of GIT components, and improved feed efficiency. GIT components responded more quickly to refeeding than the whole body. Our data suggest that the supply organs of the feed-restricted birds need to catch up and eventually exceed those of the control before the whole body might catch up in weight.

The greater feed intake relative to body weight and its associative digestive adaptations seem to be contributing factors to the relative greater body weight gain and improved feed efficiency during realimentation.

In the broiler production industry it would be prudent not to expect total catch-up growth after a short-term feed restriction but to accept some reduction in body weight. Despite this disadvantage, the potential of short-term feed restriction programs as a management tool, directed at decreasing the incidence of skeletal and other metabolic diseases is very promising. The
financial cost of slightly depressed weights of broilers that are feed restricted may be offset by improved feed efficiency, reductions in culling rates and mortality, and improved bird welfare.
APPENDIX A. PROCEDURES FOR RIBONUCLEIC ACID AND DEOXYRIBONUCLEIC ACID DETERMINATION

Reagents
1. Cold Distilled water
2. 1.2 N Perchloric acid (HClO₄)
3. 0.6 N Perchloric acid (HClO₄)
4. 0.2 N Perchloric acid (HClO₄)
5. 0.3 N Hydroxide Potassium (KOH)

Extraction of Nucleic Acids From Animal Tissues (Modified Schmidt-Thannhauser Procedure).
1. Make a 1:20 (weight:volume) homogenate of the tissues (jejunum, liver ...) in ice cold distilled water.
2. Pipette 5 mL (=250 milligrams wet weight) of this cold aqueous homogenate of tissue into a 15 mL centrifuge tube.
3. Add 2.5 mL ice-cold 0.6 N Perchloric acid (PCA) and mix.
4. After mixing the solution, allow it to stand 10 min. at 0°C (ice).
5. Centrifuge for 15 min. at 4000 g and pour off supernatant fraction (acid soluble fraction) and discard.
6. Wash (resuspend the residue in PCA solution and centrifuge) the precipitate twice with 5 mL of 0.2 N ice-cold PCA.
7. Drain off excess acid from the last wash by inverting the tube briefly over filter paper.
8. Add 4 mL of 0.3 N KOH and incubate the mixture in water bath for 1 hr at 37 C (to cause the hydrolysis of RNA).

9. After incubation, cool the alkaline solution in ice and add 2.5 mL of cold 1.2 N PCA. (This precipitates protein and DNA).

10. After standing 10 min. in ice to allow complete precipitation, centrifuge down the precipitate for 15 min. at 4000 g.

11. Decant the supernatant (RNA) fraction.

12. Wash the precipitate twice with 5 mL of 0.2 N PCA and add the washings to the RNA fraction. (The supernatant and washings are combined.)

13. Save the supernatant for RNA analysis by the orcinol method.

**DNA SEPARATION**

14. Suspend pellet (precipitate) in 8 mL of 1.5% PCA by stirring with a glass rod.

15. Incubate sample in water bath at 90 C for 20 min (with occasional stirring).

16. Remove tubes from water bath and add 0.5 mL of 70% PCA, cool tubes in ice for 15 min.

17. Centrifuge down the precipitate for 15 min at 4000 g.

18. Decant the supernatant (DNA) fraction.
19. Wash (resuspend the residue in PCA and centrifuge) the precipitate twice with 5 mL of 1.5% PCA and add the washings to the DNA fraction (the supernatant and washings are combined).

20. Save the supernatant for DNA estimation by the diphenylamine method of Giles and Myers (1965).

DNA Assay

Reagents

1. 1 N Perchloric solution (10% PCA)
2. Aqueous acetaldehyde (made fresh)
   1.6 mg acetaldehyde/mL
3. 5 mM NaOH
4. Diphenylamine Reagent.
   4% diphenylamine in glacial acetic acid
   (4 grams DPA in 100 mL glacial acetic acid)
   (This stock may be stored at 4 C for several weeks in a well-stoppered bottle)
5. DNA Standard
6. Stock solution: 20 mg of calf thymus DNA/50 mL 5 mM NaOH
7. Working standard solutions.
   Combine 20 mL of DNA stock solution and 20 mL of 1 N PCA (10% PCA) and heat at 70 C for 15 min.
   Both the stock and working standards are stored at 4 C.
Procedure

1. Pipet 2 mL DNA sample obtained from tissue separation procedure into test tubes.
2. Pipet 2 mL of 1 N PCA into test tube to be used as a reference (blank).
3. Pipet 2 mL of the standards which are at a concentration of 40, 80, 120, 160, and 20 μg/mL into test tubes to be used as standards.
4. Pipet 2 mL of diphenylamine reagent into sample test tubes then add 0.1 mL of aqueous acetaldehyde (1.6 mg/mL).
5. Incubate tubes in water bath at 30 C overnight (16-18 hr).
6. The absorbance is read using a spectrophotometer set at 595 nm.

RNA Assay

Reagents

1. Ferric chloride Reagent
   100 mg/100 mL concentrated HCl
2. Orcinol Reagent (make fresh)
   1 g orcinol/100 mL ferric chloride Reagent
3. 3 mM NaOH
4. RNA Standard (Yeast RNA)
   10 mg/100 mL 3 mM NaOH
Procedure

1. Pipet 1 mL RNA sample from tissue separation procedure into a test tube.
2. Pipet 1 mL of distilled water into test tube.
3. Pipet 2 mL distilled water into test tube to be used as a reference (blank).
4. Pipet 2 mL of orcinol reagent into test tube containing sample.
5. Pipet 2 mL of the standard at the concentrations of 20, 40, 60, 80, and 100 µg/mL into test tubes to be used as standards.
6. Put test tubes in boiling water for 35 min (evaporation may be minimized by employing glass stoppered tubes or by covering the mouths of the tubes with carefully cleaned marbles).
7. Remove tubes from boiling water and cool for 15 min.
8. The percent absorbance is read using a spectrophotometer set at 670 nm.
APPENDIX B. LIPASE ASSAY

1. Reagents
   a. Phosphate buffers (stock)
      1) $X = 0.2M \text{NaH}_2\text{PO}_4$ (28.4 g NaH$_2$PO$_4$ in 1000 mL dH$_2$O)
      2) $Y = 0.2M \text{Na}_2\text{HPO}_4 \cdot 7\text{H}_2\text{O}$ (53.6 g Na$_2$HPO$_4$ 7H$_2$O in 1000 mL)
   b. Phosphate buffer (working) : 39X + 61Y + 100 mL dH$_2$O
   c. Taurocholic acid - 890 mg / 100 mL dH$_2$O (store at 4 C)
   d. Substrate (keep at 4 C at all times)
      1) Stock - 100 mg 2-Naphthyl laurate / 100 mL acetone (store in freezer)
      2) Working substrate - (make fresh daily)
         a) 35 mL dH$_2$O + 10 mL phosphate buffer.
         b) Add 5 mL stock 2-Naphthyl laurate very slowly to the above, using a burette. Keep the burette tip under the surface.
         c) Add slowly enough for the cloudiness to dissipate once it appears. If 2-Naphthyl laurate comes out of solution (milky) begin again!
         d) Only make enough standard for 12 samples. Remake throughout the day as necessary for all the samples being assayed.
179
e. Color reagent - 40 mg O-dianizidine tetrazotized in 10 mL of cold dH$_2$O. (make fresh daily). Keep at 4°C at all times.
f. Standard - 10 mg 2-Naphthol / 100 mL dH$_2$O (store at 4°C)
   1) Dissolve naphthol in 100 mL dH$_2$O using a magnetic stirrer.
   2) Add up to 5 drops of 5 N NaOH to help it dissolve.
   3) Be patient. It takes about 20 min. to fully dissolve.
      Do not heat.
g. 40% TCA (Trichloroacetic acid).

2. Samples Preparation (note: keep samples cold at all times).
   a. Homogenize in 10 vol. of cold dH$_2$O (ex - 1g sample:9 mL dH$_2$O).
      1) Cut samples into small pieces prior to homogenization.
      2) Place the sample in dH$_2$O.
      3) Homogenize on ice, using short times; 20-30 sec. total.
         Clean all tissue from blades.
   b. Centrifuge for 20 min. at 30000 G (16000 rpm).
   c. Dilute supernatant$^1$ - 10X for pancreas, 5X for intestine
      cont. (1 - after storage if stored)
d. Store undiluted supernatant at -20 C for other enzyme assays.

4. Lipase Assay

a. Test tube setup

1) Label 3 centrifuge tubes (plastic) per sample: one sample, one standard and one blank.

2) Do not do more than 30 samples at a time.

b. Steps

1) Add .5 mL Taurocholate to all test tubes.

2) Add sample to sample and standard tubes. (vol. determined by the dilution series)

3) Add .5 mL buffer to standard tubes.

4) Add 1.3 mL dH₂O to standard and dH₂O (in the same amount as sample used) to the blank tubes.

5) Add .2 mL of 2-Naphthol to the standards only.

6) Bring to temperature in 37 C water bath. (takes about 6 min).

7) Add 2 mL substrate to sample and blank tubes. Vortex and then incubate for 10 min.

8) Add .5 mL of color reagent to all tubes. Let sit for 2 min.

9) Add .5 mL of TCA to all tubes.
10) Add 5 mL of ethyl acetate (do this under the hood). Invert each tube to fully mix the layers. Taking precautions not to cross contaminate from tube to tube.

11) Centrifuge 2000 rpm for 10 min.

12) Read upper fraction at 540 nm

c. Calculations

1) Activity = mg naphthol released in 10 min at 37 C

2) \[
\frac{\text{O.D. sample} - \text{O.D. blank}}{\text{O.D. standard}} = a
\]

3) Standard O.D. = .02 mg naphthol in total volume
   \[a \times .02 = \text{mg naphthol in total volume}\]

4) .5 mL diluted sample in total volume (9 mL)
   \[
   \frac{\text{mg naphthol}}{.5 \text{ mL}} = \text{mg naphthol/mL of diluted sample}
   \]

5) \[(\text{mg naphthol/mL of diluted sample}) \times \text{dilution factor.}\]

Dilution factor = dilution when homogenized \times \text{dilution of supernatant} \times \text{dilution when sample is added to buffer.}\]