AN ABSTRACT OF THE THESIS OF

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Title: Dietary Energy Manipulation on Fat Deposition and Metabolism in Broilers

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Harry S. Nakaue

The objective of this work was to define the effects of dietary energy intake on broiler fat deposition and metabolism. Fatty broilers are one of the major problems in the broiler industry.

The effects of changing calorie to protein (Cal/Pr) ratios, and the addition of cellulose or dried distiller's by-product (DDBP) in the diet, on abdominal fat deposition, liver lipogenesis and glucose oxidation and broiler performance indicated that the mean body weight of broilers fed narrower Cal/Pr ratio diets (124 and 143 for starter and grower, respectively) were lower ($P<.01$) than broilers fed diets with mid-Cal/Pr ratios (138 and 160) and wide Cal/Pr ratios (147 and 171). Feed conversion was better ($P<.01$) for broilers fed the diet with wide Cal/Pr ratio than the broilers fed the diet with narrow Cal/Pr ratio. Abdominal fat deposition increased ($P<.001$) with wide Cal/Pr ratio than narrow Cal/Pr ratio. No significant differences were found in in vitro liver lipogenesis and glucose oxidation when Cal/Pr ratio was widened. Mean body weight, feed conversion and abdominal fat

levels of broilers fed 5% cellulose or 10% DDBP diets were not different when compared to broilers fed diets with the same Cal/Pr ratio without these two feed ingredients.

The time-course of the deposition of abdominal fat fitted a negative exponential growth curve. The data also indicated that the highest potential for abdominal fat deposition was during the first two weeks of age. When the feed intakes of broilers were restricted for a 6 day period beginning at 1, 2, 3 and 4 weeks of age, the feed efficiencies of all the restricted broilers were significantly improved compared with broilers fed ad libitum. No significant differences were observed in mean body weight between the different treatments. The percentage of abdominal fat in broilers restricted-fed starting either at 1 or 2 weeks old for a 6-day period were lower ($P<.05$) than broilers fed ad libitum. Feed restriction from days 7 to 12 reduced the adipocyte size but not the number. Feed restriction decreased ($P<.05$) lipogenesis at 2 and 8 weeks of age. No significant effects were found in lipolysis between the restricted and ad libitum-fed broilers. These studies demonstrate that early feed restriction (days 7 to 12) reduced abdominal fat deposition which is attributed to the reduction in the adipocyte size. Smaller size adipocyte resulted from decreased activity of lipogenesis.

Dietary Energy Manipulation on Fat Deposition and Metabolism in Broilers

by

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DEDICATION

This Doctoral Dissertation is dedicated to my parents,

Shang-Chen and Yu-Rong Zhong

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PREFACE

Dr. Harry S. Nakaue, as major professor, participated in all phases of this research project. He actively guided the design, execution and analyses of all experiments.

Dr. Ching Yuan Hu not only taught the student, hand by hand, in performing the biochemical assays, but also participated all the laboratory determination reported herein. He also gave important advice in experimental design and analyses.

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Dietary Energy Manipulation on Fat Deposition and Metabolism in Broilers

CHAPTER I

INTRODUCTION

The United States' broiler industry began in 1923 when Mrs. Wilma Steele of Oceanview, Delaware started with 500 chickens and marketed 387 birds at 62 cents per pound with an average market weight of 2 pounds (Gordy, 1974). By 1926, Mrs. Steele had the housing capacity to raise 10,000 birds. Today, the U.S. broiler industry raises approximately 5 to 6 billion broilers annually, with a gross revenue of 8 to 9 billion dollars. Poultry meat per capita consumption in the U.S. has surpassed the per capita consumption of pork and beef. Americans are eating more poultry meat in some form almost everyday now which is a far cry from the "chicken every Sunday" or "chicken in every pot" slogans.

This tremendous growth was brought about by many innovative ideas that were implemented by the broiler industry. Automated systems in the houses, feed mills, and processing plants; knowledge and understanding of nutrition, disease, management and genetics; the introduction of integration and grower contracts and the U.S. Department of Agriculture inspection for wholesomeness of poultry all helped bring about the rapid growth and acceptance of poultry. All of these ideas are interrelated; however, one area that has

played a key role is genetics. In the 1950's, broiler producers were marketing a 4 pound bird when they were 12 to 14 weeks of age. Today, broiler producers are marketing a 4 pound broiler at 6 weeks of age. The geneticists have bred chickens that grow rapidly and convert feed to meat at an efficient rate (1.9 pounds of feed/pound of meat). Growth rate and efficient feed utilization were the key traits in their breeding programs, but not much attention was paid to the carcass composition of the broiler (fryers) sold at the supermarket. The consumers are now complaining that fryers sold at the supermarket are too fat. This problem of excessive carcass fat is of concern not only to the poultry producers but also to the physiologists, geneticists, nutritionists and biochemists.

Excessive accumulation of carcass fat in market broilers costs the U.S. broiler industry over 300 million dollars annually (Nelson, 1980, Rosebrough et al. 1986). Fatty broilers waste energy and feed, slower processing time in the processing plants and constant complaints from the consumers. Therefore, saving feed and reducing carcass fat content in broiler chickens are challenging tasks for the poultry scientists.

In 1989, Oregon produced 20 million broilers, and 5518.1 million were produced in the United States (Holleman, 1990). If we could reduce the abdominal fat content of broilers from 3.5% to 1%, there would have been a saving to the Oregon

broiler producers of over 1 million dollars for 1989 and much more in the United States. That does not include the labor costs for both producers and processors. Another significant benefit is that the lean chicken for human consumption will reduce the incidence of heart and blood diseases, thus promoting consumption and production.

Fat in the adipose tissue is a form of energy stored in the broilers. Dietary energy, protein, fat and some of the feed additives in the feed can affect the carcass fat content. The calorie to protein (Cal/Pr) ratio has been one of the most widely investigated dietary factors which influence the degree of fatness of broilers (Bartov, 1979; Donaldson 1955, 1985; Griffiths et al., 1977; Potter et al., 1956; Richard and Ringrose 1958; Sibbald et al., 1961). Narrowing the Cal/Pr ratio of the feed usually decreases the calorie intake, and therefore, reduces deposition of body fat. The Cal/Pr ratio of the feed can be narrowed by either decreasing the energy levels or increasing protein levels in the feed. However, the later approach is not wholly accepted by the industry because of the high cost of protein. To decrease the energy intake, two methods could be pursued: By diluting the energy content of the feed or by limiting the feed intake. In this dissertation research, the effects of Cal/Pr ratio and feed restriction on adipocyte cellularity, hepatic lipogenesis and glucose oxidation, abdominal fat accumulation and broiler performance were studied. Since cellulose and dried

distiller's by-product (DDBP) are feedstuffs that could be used in poultry industry, the effects of these feedstuffs on broiler performance and fat metabolism were examined. Proper control of feed intake will improve production efficiency by decreasing fat deposition and feed cost.

Therefore, the time-course of abdominal fat deposition, the compensatory growth after the feed restriction, and effects of timing of the feed restriction on final body weight and feed efficiency were investigated. Controlling excessive carcass fat deposition through nutritional manipulation has profound implications on the poultry industry. Hopefully, this information will be useful to the producers in improving broiler production.

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CHAPTER II

LITERATURE REVIEW: NUTRITIONAL MANIPULATION ON FAT DEPOSITION AND METABOLISM IN BROILERS

The Origin and Development of Adipose Tissue. The increase of adipose tissue is due to an increase in the number of fat cells by the multiplication of the adipocytes (hyperplasia) or by the enlargement of these cells caused by lipid accumulation within the cells (hypertrophy). The adipocytes are mature cells derived from perivascular cells (pericytes). The pericytes are progressively specialized into adipoblasts and then into fat-storing cells called preadipocytes. Then, according to nutritional conditions and endocrine control, these preadipocytes are enlarged to become adipocytes (LeClercq 1984). In commercial strains of broilers, the numbers of adipose cells increase in the abdominal fats pad until 14 weeks of age (Hood, 1982, 1984); after which the cell numbers remained constant at about 270 X 10^6 cells per fat pad. The mean size of adipose cells in the first 14 weeks increased slowly. After 14 weeks adipose cell volume increased rapidly. The distribution of cell size is frequently bimodal, with a population of small adipocytes (diameter less than 10 μm) and a population of large adipocytes. The control of lipid accumulation within the cells depend upon the balance of synthesis and degradation of

triglycerides inside the body. In the avian species, the liver is the major site for lipogenesis (Goodridge, 1968; O'Hea and Leveille, 1969), although bone marrow (Nir and Lin, 1982), adipose tissue and skin (Yeh and Leveille, 1973) do make some contribution. Saadoun and LeClercq, (1983) confirmed *in vivo* that in fat and lean line chickens, 65% and 68%, respectively, the *de novo* fatty acids came from liver synthesis. Lipolysis occurred in adipose tissues. Mobilization of triglycerides stored in fat cells is catalyzed by the enzyme hormone sensitive lipase (HSL), which hydrolyzes triglycerides to FFA and glycerol. The rate of lipolysis is dependent upon a metabolic cascade that ultimately results in the phosphorylation and activation of HSL.

The Effects of Dietary Protein on Fat Metabolism: High protein diets fed to broilers produced relatively lean carcasses in a study employing diets from 16 to 25% protein concentration (Bartov *et al.*, 1974). Summers and Leeson (1979) likewise showed that isocaloric diets, varying in protein concentration from 15.8 to 21.4%, resulted in marked decrease in the abdominal fat of broilers. Yoshida and Morimoto (1970) fed chicken diets with protein levels ranging from 9 to 79% and found carcass protein increased linearly reaching a maximum when the diets contained 31% or more protein. The need of female broilers for less protein has been reported by Siegal and Wisman (1962). Lipstein *et al.* (1975) found that lowering dietary protein below 20% affected

both performance and carcass fat levels in male broilers whereas the females gave no differential response until protein concentration was reduced to 16%. It is not clear exactly how individual amino acid deficiencies will influence fat content of the carcass. Pfaff (1977) found that a marginal methionine deficiency increased the carcass fat content of young growing chickens. The methionine deficiency also resulted in increased hepatic fatty acid synthesis *in vitro*.

Collado and Tasaki (1981) demonstrated increased hepatic lipogenesis in meat-type birds when dietary protein was reduced to 12% and suppression of lipogenesis when dietary protein concentration was raised to 30%. Yeh and Leveille (1969) studied the influence of dietary protein on hepatic lipogenesis in growing chicks; and both *in vitro* and *in vivo* studies demonstrated that the incorporation of glucose-U-^{14}C, pyruvate-2-^{14}C and acetate-1-^{14}C into liver fatty acids was decreased by elevating the dietary protein levels. They also found the activity of malic enzyme was positively correlated with the rate of lipogenesis. Increasing dietary protein from 15 to 35% depressed both *in vitro* lipogenesis and malic enzyme activity by about 75%. When Rosebrough and Steele (1985) fed broiler chicks 18, 23, and 30% dietary protein, they found the *in vitro* lipogenesis and the activity of malic enzyme was 106, 51, 21 μmoles and 8, 4 and 2 Units per 100 g BW, respectively. Uric acid is the main end product in nitrogen metabolism in

birds. Since considerable energy is required to synthesize uric acid (Buttery and Boorman, 1976), it was suggested by Bartov (1979) that high protein diets cause a reduction in carcass fat deposition by requiring the bird to expend more energy in eliminating excess nitrogen from the body. However, high dietary protein can cause the increasing secretion of growth hormone and glucagon (Martin, 1985), and some other hormones. Growth hormone will reduce the lipogenesis and increase chicken lipolysis (Harvey et al., 1977). Glucagon inhibits lipogenesis of chicken hepatocyte (Cramb et al., 1982). As a function of coordination of the change of the endogenous hormones, the fat metabolism of the chicken would be markedly changed.

The Effects of Dietary Fat on Fat Metabolism: Bartov et al. (1974) reported that the inclusion of dietary fat at a level of 6% of the diet did not increase the amount of carcass fat as long as the calorie to protein ratio remained constant but that actually a trend existed towards a lowered carcass fat content. Edwards et al. (1973) reported the effects of cottonseed oil, acidulated cottonseed soap stock, beef tallow and poultry fat on carcass composition and fatty acid composition of adipose tissue. The types of fat fed did not influence gross body composition but did influence fatty acid composition of the whole carcass and adipose tissue. Feeding beef tallow increased stearic acid and oleic acid levels at the expense of linoleic acid, thus producing a bird which

feels much firmer to tough. Pan et al. (1979) reported that replacing soya bean oil with tallow in broiler diets resulted in increased abdominal fat levels.

The increase of dietary animal fat decreased the rate of liver in vivo lipogenesis in broilers (Donaldson, 1985). When chickens were fed dietary fat ranging from 2, 4.1, 6.3 to 8.6%, the in vivo lipogenesis was 18.4, 11.6, 7.1 and 3.7 dpm X 10^{-3}, respectively. The reduced lipogenesis does not necessarily affect carcass fat content because under such conditions, a high proportion of carcass fatty acids are derived directly from dietary fat (Shapira et al., 1978). It may, however, affect the composition of carcass fat.

Extended effects of dietary fat on lipogenic enzyme and fatty acid synthesis have been widely studied in rats (Clarke and Clarke, 1982; Clarke et al., 1977; Toussant et al., 1981; Iritani et al., 1980; Clarke et al., 1990). According to these studies, dietary polyunsaturated fats are potent inhibitors of hepatic fatty acid and triglyceride synthesis. Saturated and mono-unsaturated fatty acids have little or no inhibitory capability, and 20- and 22-carbon (n-3) fatty acids display the greatest inhibitory potency. The suppression of fatty acid synthesis by polyunsaturated fats is accompanied by a decrease in activities of fatty acid synthase, malic enzyme, acetyl-CoA carboxylase and glucose-6-phosphate dehydrogenase. In a recent study, Clarke et al. (1990) investigated the ability of saturated (tripalmitin), (n-9) monounsaturated

(triolein), (n-6) di-unsaturated(safflower oil), and (n-3) polyenic (fish oil) fatty acids to suppress the gene expression of fatty acid synthase and S14 (a member of the lipogenic protein family). They found that dietary polyunsaturated fats reduced, by 75-90%, the hepatic abundance of FAS and S14 mRNA. Fish oil, rich in 20- and 22-carbon polyenic fatty acids, was more effective than safflower oil, whereas tripalmitin and triolein were without effect. These data support the hypothesis that dietary polyunsaturated fats uniquely regulate the gene expression of lipogenic enzymes and that the mediator is likely a specific entity derived from the long-chain polyenic fatty acids. Unfortunately, no similar work has been found in poultry.

The Effects of Other Types of Feed on Fat Metabolism: Poultry has been fed low fiber rations for several decades because cellulose has been thought to have little, if any, energy value in broiler diets. However, there are increasing amounts of high fiber by-products from milling, brewing and distilling industries, and from other sources. Cellulose has been reported in humans to increase the fat excretion in feces (Kies et al., 1983, Kaur et al., 1985) and decrease the lipid content in blood (Behall et al., 1984, Kies et al., 1983). Kienholz (1988) reported that broiler chicks can use the nutrients in a 20% cellulose diet, if the diet is properly pelleted. No detrimental effect on growth rate was observed. Modern broilers may have a high tolerance for crude fiber.

Distiller's products are often used for chicken feeds, as well as other animal feeds, to promote growth and reduce feed cost. Whether the use of distiller's by-products will decrease fat content by decreasing dietary energy concentration, or because of the existence of unidentified factor(s) will need to be determined.

The effects of Cal/Pr Ratios on Fat Metabolism: Energy and protein levels in the feed are important to body fat content. High dietary energy level, per se, is not the major factor responsible for the excess energy consumption. The ratios of energy to protein, or of energy to balanced amino acids, are far more important regulators of carcass fat content (Bartov 1979). Therefore, the protein content at an optimal level and changes in the energy to protein ratio may alter fat deposition. Many early studies have established the relation of energy to protein (Cal/Pr) ratios and chicken carcass fat deposition (Donaldson 1955, 1985; Griffiths et al., 1977a,b; Hill and Dansky 1954; Leong et al., 1959; Potter et al., 1956; Richard and Ringrose 1958; Sibbald et al., 1961). Excessive fat deposition in the broiler is due to the extra energy supplied from the formulation of high fat rations which prompt better feed efficiency. Recently researchers have begun intensive studies on fat deposition and metabolism as affected by the nutrition of broilers. Donaldson (1985) kept dietary energy constant and protein level variable and found that when

Cal/Pr ratio was over 139, the growth rate decreased. However, Cal/Pr ratio had no effect on feed conversion, liver weight and fatty acid synthetase activity. In vivo lipogenesis increased when the Cal/Pr ratio exceeded 120. A diet with a Cal/Pr ratio of 177 produced significantly more fatty broilers. Rosebrough and Steele (1985) fed male broiler chicks ad libitum diets containing 18, 23, or 30% crude protein for the first 3 weeks and found a positive relationship between dietary protein and percent lean tissue. Broilers fed 23% dietary protein had better body lean mass, and in vitro lipogenesis was negatively related to protein level. Griffiths et al. (1977a) found two energy levels, 2970 and 3190 kcal/kg, had no significant effect on abdominal fat pad size. The decreased Cal/Pr ratio of the diet resulted in a significant reduction in the proportion of fat in the body.

The Effects of Feed Restriction on Fat Metabolism: Feed restriction of chicken and compensatory growth have been studied widely because both of these factors can improve feed efficiency and save energy by reducing metabolic energy loss. The ability to compensate for growth in animals after a period of undernutrition is called compensatory growth. Feed restriction is also important in reducing carcass fat deposition in chickens (Plavnik and Hurwitz, 1985, Rosebrough et al., 1986, 1988).

Recent studies have focused on early feed restriction.

Plavnik and Hurwitz (1985) studied the effects of duration and severity of feed restriction and the sex of the broiler on subsequent performance, weight gain, feed efficiency and fat deposition. Complete compensation of the weight depression caused by feed restriction between 6 and 28 days of age was not obtained at 56 or 63 days of age. The final body weights of males and females restricted with 40 and 35 kcal/day, respectively, beginning from the sixth to the twelfth day of age, did not differ significantly from those on the ad libitum feed. Feed efficiency was also improved in the restricted broilers more than with the ad libitum fed broilers. Washburn and Bondari (1978) found that if feed consumption of broilers was significantly depressed for 7 days between 21 and 56 days of age, weight gain was significantly depressed, with significantly poorer feed utilization and lower final body weights. An experiment from Plavnik et al. (1986) showed that broiler chickens subjected to a severe feed restriction from 7 to 13 days of age used their feed more efficiently (4%-12%) and accumulated less abdominal fat (17%-30%) compared to ad libitum-fed chickens. Rosebrough et al. (1988) fed energy at maintenance level to male broiler chickens from 6 to 12 days of age, and found the 6 day energy maintenance level (40 kcal/chick/day) decreased ($P<.05$) in vitro lipogenesis, fatty acid synthesis, activity of malic enzyme and absolute weight of abdominal fat pad as well as its proportion to body weight at 54 days when compared to ad libitum.

Therefore, the effect of early feed restriction on fat metabolism is of interest. The change in abdominal fat pad may indicate either the increase in the size of the fat cells or the increase in the number of fat cells or both. The multiplication of fat cells depends upon the anatomical location of the fat depot and the age of the animal (March and Hansen 1977). In commercial strains of birds, the numbers of adipose cells increased in the abdominal fat pad until 14 weeks of age; after which the cell number remained constant at about 270 X 10^6 cells per fat pad (Hood, 1984). Considering the changes of the abdominal fat pad in the restricted chickens, the questions of whether early feed restriction affects the multiplication of precursor adipocytes or the accumulation of lipids in the adipocyte are of interest.

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CHAPTER III

CALORIE TO PROTEIN RATIOS, CELLULOSE AND DRIED DISTILLER'S BY-PRODUCT ON BROILER PERFORMANCE AND FAT METABOLISM[1]

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Abstract

Five hundred commercial broiler strain cross (Vantress X Hubbard) chicks were reared to 7 weeks of age to study the effects of calorie to protein ratios (Cal/Pr ratios), cellulose and dried distiller's by-product (DDBP) on abdominal fat deposition, liver lipogenesis and glucose oxidation and broiler performance. Five treatments were: I. narrow Cal/Pr ratios (124 for starter and 143 for grower); II. Mid Cal/Pr ratios (138 and 160); III. wide Cal/Pr ratios (147 and 171); IV. same Cal/Pr ratios as II with 10% DDBP and V. same Cal/Pr ratios as II with 5% cellulose added. The mean body weight of broilers fed diet I was significantly ($P<.01$) lower than the weight of broilers fed diets II, III, IV and V. Feed conversion was significantly better ($p<.01$) for broilers fed diet III than those on diet I. Abdominal fat deposition increased significantly ($P<.001$) with the wide Cal/Pr ratio (diet III) over the narrow Cal/Pr ratio (diet I). No significant differences were found in *in vitro* liver lipogenesis and glucose oxidation. Mean body weight, feed conversion or abdominal fat levels were not different when 5% cellulose or 10% DDBP diets were fed.

Under the conditions of this experiment, widening the Cal/Pr ratio affected abdominal fat but not the lipogenesis and glucose oxidation. Abdominal fat levels were not affected by feeding 5% cellulose and 10% DDBP diets.

(Key words: Cal/Pr ratio, lipogenesis, cellulose, distiller's by-products, abdominal fat.)

Introduction

Excessive accumulation of carcass fat in broiler chickens costs the U.S. broiler industry over 250-300 million dollars annually (Nelson, 1980; Rosebrough and Steele, 1985). Obese broilers increase processing time and excess fat results in constant complaints from consumers. Therefore, the reduction of carcass fat content in broiler chickens is a challenging task for poultry scientists. Many early studies have established the relation of calories to protein (Cal/Pr) ratios and chicken carcass fat deposition (Donaldson 1955, 1985; Griffiths et al., 1977a; Hill and Dansky 1954; Leong et al., 1959; Potter et al., 1956; Richard and Ringrose 1958; Sibbald et al., 1961). Excessive fat deposition in the broiler is caused by high energy intake. Narrowing the Cal/Pr ratio usually decreases the calorie intake and therefore reduces deposition of body fat. Widening this ratio by maintaining the same protein content and increasing the energy level will elevate the carcass fat level. In today's poultry industry, the broiler rations contain high energy because of the use of animal fat that promotes fast growth and better feed efficiency. In rats, polyunsaturated fatty acids suppressed lipogenesis, and gene expression of lipogenic enzymes (Clarke and Clarke, 1982; Clarke et al., 1990). Donaldson (1985) reported that fat substituted isocaloric diets (same Cal/Pr ratios) decreased de novo lipogenesis.

However, the effects of widening the Cal/Pr ratio by increasing animal fat on fat metabolism in broilers have not been reported.

The purpose of this experiment was to study the effects of Cal/Pr ratios on hepatic lipogenesis and glucose oxidation, abdominal fat accumulation and broiler performance. The effects of cellulose and dried distiller's by-product (DDBP) on broiler performance and fat metabolism were examined also.

Materials and Methods

Five hundred commercial broiler strain cross (Vantress X Hubbard) chicks of equal sexes were used in this experiment. They were brooded and reared to seven weeks of age in a conventional floor pen house. One infra-red heat lamp was used per pen (1.2 X 2.4 meter) as the heat source. The floor pens were covered with 5 cm clean wood shavings. The chicks were randomly assigned to 20 pens. These 20 pens were randomly assigned to five treatments. Feed was provided ad libitum in each pen with a hanging feeder (45.7 cm diameter). Water was provided with a hanging Plasson waterer in each pen. The composition of the starter and finisher diets are listed in Table III.1 and III.2. The five treatments (4 replicates /treatment; 25 chicks /replicate) were: I. narrow calorie to protein ratio (Cal/Pr), 124 for starter and 143 for grower; II. mid Cal/Pr ratios, (138 and 160); III. wide Cal/Pr ratios (147 and 171); IV. same Cal/Pr ratios as II with 10% dried distiller's by-product[1] (DDBP) and V. same Cal/Pr ratios as II with 5% cellulose[2] added. Broilers of all treatments were fed 23% crude protein starter diets from day-old to 3 weeks and 20% crude protein finisher diets from 4 to 7 weeks of age. The higher energy levels of diets II and III were reached by

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[1]DDBP was purchased from Leo Cook Co., Tualatin, Oregon

[2]Cellulose (Solka floc B200) was product of James River Co., Berlin, New Hampshire.

adding animal fat[3]. Body weight gain and feed consumption were measured at the end of the third and seventh week of the experiment. Ten male and ten female broilers from each treatment were weighed individually and sacrificed at seven weeks of age. The abdominal fat (leaf and gizzard fat) was removed by the same individual to reduce variability. The extracted fat was then weighed.

Six birds from each treatment were selected randomly at the end of the 7th week to study the effect of treatments on in vitro hepatic lipogenesis and glucose oxidation. Broilers were sacrificed by cervical dislocation, and the livers were immediately excised and weighed. A portion of the liver was sliced with a tissue slicer. The tissue slicer has a platform with a fixed blade mounted at the edge and the depth was set at about .5 mm. Tissues were sliced horizontally by passage across the moistened platform against the knife edge. Lipogenesis and glucose oxidation were measured by a slightly modified radioactive method from Mersmann and Hu (1987). The medium used was Krebs-Ringer-bicarbonate (KRB) buffer containing 118 mM NaCl, 4.71 mM KCl, .63 mM Ca_2, 1.19 mM KH_2PO_4, 1.19 mM $MgSO_4 7 H_2O$ and 25 mM $NaHCO_3$, and routinely gassed using a gas dispersion tube for 15 min with 5% CO_2 in oxygen. The medium also contained 20 mM glucose, .5 uCi [U-

[3]Animal fat was purchased from Delaware-Darling Co., Portland, Oregon

^{14}C] glucose[4] per flask, and .1 U porcine insulin[5] per ml. There were 100 mg of tissue slices per flask. Each liver sample was incubated in triplicate in 25-ml siliconized, sealed Erlenmeyer flasks under an atmosphere of 5% CO_2 in oxygen. After 2 hrs at 39°C with reciprocal shaking at 90 strokes per min, the reaction was stopped by injecting .25 ml of 1 N H_2SO_4. The evolved CO_2 was trapped on filter papers in suspended center wells containing .2 ml hyamine hydroxide[6] during an additional 1-hr incubation. The lipids were extracted from the medium plus the tissue slices with chloroform-methanol (2:1, v/v). The washed extracts and center well were placed in a counting vial, 10 ml of counting fluid was added, and the samples were counted in a liquid scintillation spectrometer (Packard Model 2425) to determine conversion of glucose to total lipids and CO_2, respectively.

Data were expressed as means of treatments except where noted. Treatment effects within the experiment were determined by one-way ANOVA with a level of statistical significance of 5%. Rankings of the means utilized Duncan's Multiple Range test for significance (Duncan, 1955).

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[4]Cat. No. 29756-9, Sigma Chemical Co., St. Louis, MO 63178

[5]Cat. No. I-3505, Sigma Chemical Co., St. Louis, MO 63178

[6]Methylbenzethonium Hydroxide, Cat. No. M-1756, Sigma Chemical Co., St. Louis, MO 63178

Table III.1. Experimental starter diets

Ingredients	Diets I	II	III	IV	V
Corn, Yellow, %	57.16	55.08	54.40	51.19	47.67
Animal fat, %	-	6.13	10.0	5.60	9.80
Soybean meal (47.5% CP), %	37.37	31.08	21.79	25.55	25.06
Fish meal (65% CP), %	-	5.00	8.00	5.00	8.00
M & B meal (49.5% CP), %	-	-	5.00	-	3.06
Def. phosp (32% Ca), %	1.80	1.10	-	.95	-
Dr. limestone, %	.80	.91	.08	.99	.68
Salt, %	.30	.30	.30	.30	.30
Vitamin mix*, %	.25	.25	.25	.25	.25
Mineral mix#, %	.05	.05	.05	.05	.05
d,l-Methionine (98%), %	.17	.13	.13	.12	.13
Distil. by-prod., %	-	-	-	10.00	-
Cellulose, %	2.00	-	-	-	5.00
Calculated analyses:					
Crude protein, %	23.00	23.00	23.00	23.00	23.00
ME, Kcal/kg	2856	3186	3389	3182	3184
Cal/Pr ratios	124	138	147	138	138
Ca, %	1.00	1.00	1.00	1.00	1.00
Avail. Phos., %	.45	.45	.60	.60	.49
Meth., %	.53	.54	.55	.54	.54
Meth. + Cyst., %	.93	.93	.93	.93	.93

*Supplies per kilogram of feed: vitamin A, 4125 IU; vitamin D_3, 1375 ICU; vitamin E, 1.4 IU; vitamin K, .69 mg; vitamin B_{12}, 6.7 ug; riboflavin, 4.1 mg; pantothenic acid 6.7 mg; niacin, 27.5 mg; choline, 275 mg; folic acid, .28 ug.

#Supplies per kg of feed: calcium, 97.5 mg; manganese, 60 mg; iron, 20 mg; iodine, 1.2 mg; zinc, 27.5 mg; cobalt, .02 mg; copper, 2 mg.

Table III.2. Experimental finisher diets

Ingredients	Diets I	II	III	IV	V
Corn, Yellow, %	62.85	64.59	57.63	60.25	52.66
Animal fat, %	-	3.69	9.35	3.46	8.40
Soybean meal (47.5% CP), %	27.08	26.76	28.05	21.40	28.98
M & B meal (49.5% CP), %	3.00	3.00	3.00	3.00	3.00
Def. phosp (32% Ca), %	.66	.65	.67	.51	.69
Gr. limestone, %	.92	.66	.63	.73	.62
Salt, %	.30	.30	.30	.30	.30
Vitamin mix*, %	.25	.25	.25	.25	.25
Mineral mix#, %	.05	.05	.05	.05	.05
d,l-Methionine	.06	.06	.06	.05	.07
Distil. by-prod., %	-	-	-	10.00	-
Cellulose, %	4.84	-	-	-	5.00
Calculated analyses:					
Crude protein, %	20.00	20.00	20.00	20.00	20.00
ME, Kcal/kg	2863	3193	3413	3193	3193
Cal/Pr ratios	143	160	171	160	160
Ca, %	1.00	.90	.90	.90	.90
Avail. Phos., %	.40	.40	.40	.40	.40
Meth., %	.38	.38	.38	.38	.38
Meth. + Cyst., %	.72	.72	.72	.72	.72

*Supplies per kilogram of feed: vitamin A, 4125 IU; vitamin D_3, 1375 ICU; vitamin E, 1.4 IU; vitamin K, .69 mg; vitamin B_{12}, 6.7 ug; riboflavin, 4.1 mg; pantothenic acid 6.7 mg; niacin, 27.5 mg; choline, 275 mg; folic acid, .28 ug.

#Supplies per kg of feed: calcium, 97.5 mg; manganese, 60 mg; iron, 20 mg; iodine, 1.2 mg; zinc, 27.5 mg; cobalt, .02 mg; copper, 2 mg.

Results and Discussion

The 7 week mean body weights of males, females and combined sex in the narrow Cal/Pr ratios treatment were significantly ($P<.01$) lower than those of mid-Cal/Pr, wide Cal/Pr, mid-Cal/Pr with 5% cellulose and mid-Cal/Pr with 10% dried distiller's by-product (DDBP) (Table III.3). The feed conversion was significantly ($P<.01$) improved when the Cal/Pr ratio was wider. There were no significant differences observed in feed conversion among the dietary treatments with same Cal/Pr ratios.

The percent abdominal fat of narrow Cal/Pr ratio broilers was significantly lower ($P<.05$) than that of mid or wide Cal/Pr ratio fed broilers (Table III.4). There were no differences in abdominal fat deposition between treatments with mid- and wide Cal/Pr ratios. The addition of 5% cellulose lacked significant reduction of abdominal fat content. The adding of DDBP seems to reduce the abdominal fat; however, a significant difference was observed when 10% DDBP was added (Table III.4).

Data on the *in vitro* hepatic lipogenesis and glucose oxidation are presented in Table III.5. No statistical differences were observed because of the large variation in the data.

Table III.3 The effects of Calorie to protein (Cal/Pr) ratios, distiller's dried by-products and cellulose on broiler chicken performance at 7 weeks of age

Treatments	Mean body wts (kg)[1] Males	Females	M + F	Feed Body Wt.
Narrow Cal/Pr	2.42[a]	1.99[a]	2.19[a]	2.41[a]
Mid Cal/Pr	2.65[b]	2.30[b]	2.45[b]	2.24[b]
Wide Cal/Pr	2.88[c]	2.30[b]	2.55[b]	2.03[c]
Mid Cal/Pr +10% DDBP	2.62[b]	2.29[b]	2.45[b]	2.22[b]
Mid Cal/Pr +5% Cellulose	2.73[b]	2.28[b]	2.47[b]	2.16[b]
F	16.12**	18.20**	26.74**	45.21**

1. Mean values in each column with different superscripts are significantly different (P<.001).

Table III.4. The effects of Calorie to protein (Cal/Pr) ratios, Distiller's dried by-product and cellulose on percentage broiler chicken abdominal fat at 7 weeks of age

Treatments	Abdominal Fat (%)[1]		
	Males	Females	M + F
Narrow Cal/Pr	1.81[a]	2.02[a]	1.91[a]
Mid Cal/Pr	2.62[ab]	3.62[b]	3.09[b]
Wide Cal/Pr	3.02[b]	3.69[b]	3.36[b]
Mid Cal/Pr +10% DDBP	2.49[ab]	3.49[b]	2.99[b]
Mid Cal/Pr +5% Cellulose	2.86[ab]	3.50[b]	3.18[b]
F	5.55**	8.88**	10.65**

1. Mean values in each column with different superscripts are significantly different (P<.001).

The ratio of energy to protein, or of energy to balanced amino acids, is a far more important regulator of carcass fat content (Bartov 1979). The results from this study show that when dietary protein levels were kept constant (23% for starter and 20% for grower) and the Cal/Pr ratios increased from 143 to 160 and 171, the percent abdominal fat in broilers increased from 1.91 to 3.09 and 3.36%, respectively (Table III.4). Increasing the energy content of the diet with the addition of fat at the expense of carbohydrate, without significantly altering Cal/Pr ratio did not affect carcass content of broilers (Griffiths et al. 1977b; Fuller and Rendon 1977). In Treatments II, IV and V (Cal/Pr ratios were the same), added dietary fat levels increased from 5.6, 6.13 to 9.8% for the starter and 3.46, 3.69 and 8.40% for finisher diet. The percent abdominal fat did not significantly change. In Treatment III, the Cal/Pr ratios for starter and finisher diets were 147 and 171, respectively, produced by adding dietary fat. No significant increase in abdominal fat was observed when compared to Treatment II (Cal/Pr ratios = 138 and 160). Jensen et al.(1970) observed that metabolizable energy derived from fat appears to be more efficiently utilized for tissue energy gain than metabolizable energy from other sources. This also may explain that the feed efficiency and growth rate increased in this experiment when dietary energy levels were enhanced by adding dietary fat.

Poultry has been fed low fiber rations for several decades. However, there is available from time to time high fiber by-products from the milling, brewing and distilling industries for inclusion in poultry feeds. Cellulose (a fibrous material) has been thought to have little, if any, energy value in poultry diets. Cellulose has been reported in humans to increase the fat excretion in feces (Kies et al., 1983, Kaur et al., 1985) and decrease the lipid content in blood (Behall et al., 1984, Kies et al., 1983). In this study, broilers fed 5% cellulose did not show a significant decrease in abdominal fat deposition compared to broilers fed the same Cal/Pr ratio diet (Table III. 4). Growth rate and feed efficiency were not influenced either (Table III. 3). Kienholz (1988) reported that broiler chicks could use the nutrients in a 20% cellulose diet without detrimental effect on growth rate if the diet was properly pelleted. Modern broilers may have a high tolerance for crude fiber.

Distiller's dried by-products have been recognized for a long time to have the ability to promote chicken growth, but their effect on fat deposition has not been clarified. This study shows that when feeding a 10% distiller's dried by-product diet no differences in broiler body weight or feed efficiency were observed when compared to the control group. There were no significant differences either in abdominal fat deposition or in liver in vitro lipogenesis compared to the control group (Treatment II).

The extended effects of dietary fat on lipogenic enzyme and fatty acid synthesis have been widely studied in rats (Clarke and Clarke, 1982; Clarke et al., 1977; Toussant et al., 1981; Iritani et al., 1980; Clarke et al., 1990). According to these studies, dietary polyunsaturated fats are potent inhibitors of hepatic fatty acid and triglyceride synthesis which suppresses the gene expression of fatty acid synthase and S14 (a member of the lipogenic enzyme family). These researchers found that dietary polyunsaturated fats reduced the hepatic abundance of FAS and S14 mRNA by 75-90%. High dietary fat intake inhibited lipogenesis found in chickens. Donaldson (1985) reported that in vivo lipogenesis and lipogenic enzyme activity was reduced when the dietary fat level was increased. No significant differences in lipogenesis among the dietary treatments in this study were observed because of the high variation of the data (Table III. 5). However, it is clear that the lipogenesis decreased when dietary fat levels were increased (Figure III.1). According to the fatty acid profile of the diets and animal fat fed to the broilers (Table III.6), only small amount of polyunsaturated fatty acids existed in the diets (.08, .08 and .18%, respectively, for narrow, mid, and wide Cal/Pr ratios diets). The changing of di-unsaturated fatty acids is small (1.61, 1.66, and 2.10% for narrow, mid, and wide Cal/Pr ratios, respectively). The enhancement of dietary fat content is due to increase of saturated fatty acids (.63, 2.24 and

4.58% for narrow, mid, and wide Cal/Pr ratios, respectively) and mono-unsaturated fatty acids (.66, 2.62 and 4.97% for narrow, mid-, and wide Cal/Pr ratios, respectively). It suggested that monounsaturated or saturated fatty acids might suppress lipogenesis in chickens. The response of glucose oxidation to dietary fat intake is shown in Figure III.1. The glucose oxidation in liver also tended to decrease when fat intake increased. This may represent the decrease of energy requirement for lipogenesis in liver.

Changing the Cal/Pr ratio may not be necessary to alter the abdominal fat content. Widening Cal/Pr ratios by increasing dietary fat improved feed efficiency and growth rate. The significant increase of abdominal fat only existed between the narrow and wide Cal/Pr ratio groups. There was a decrease of lipogenesis and glucose oxidation when the Cal/Pr ratio was wide, which may reflect better utilization of animal fat in the diets by the broilers. The addition of 5% DDBP or 10% cellulose without changing the Cal/Pr ratio did not affect either abdominal fat deposition or broiler performance.

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Table III.5. The effects of Calorie to protein (Cal/Pr) ratios, distiller's dried by-product on broiler lipogenesis and glucose oxidation

	(nM/hr/g liver tissue/100 g bw)	
Treatments	Lipogenesis	Glucose oxidation
Narrow Cal/Pr	64.06 ± 17.54	86.42 ± 14.23
Mid Cal/Pr	53.36 ± 14.22	81.84 ± 13.85
Wide Cal/Pr	29.25 ± 8.53	52.16 ± 6.34
Mid Cal/Pr +10%DDBP	45.03 ± 23.16	69.29 ± 16.84

Table III.6. Fatty acid composition of finisher diet and animal fat

	Diets			Anim. fat
	I	II	III	
Cal/Pr ratios	143	160	171	
Total crude fat,%	3.08*	6.84	12.24	100
Added anim. fat,%	0	3.71	9.35	-
Fatty acids %				
14:0	.02	.11	.27	2.64
14:1		.02	.05	.62
16:0	.46	1.35	2.63	23.45
16:1		.17	.44	4.78
16:2	.02	.05	.13	1.12
18:0	.15	.78	1.69	16.60
18:1	.66	2.43	4.48	42.86
18:2	1.59	1.61	1.97	3.68
18:3	.081	.08	.18	.82
Unidentified	.10	.24	.41	3.45
Total	3.08	6.84	12.24	100
Among total:				
Saturated	.63	2.24	4.58	42.69
Monounsaturated	.66	2.62	4.97	48.25
Di-unsaturated	1.61	1.66	2.10	4.80
Polyunsaturated	.08	.08	.18	.82

*Diet I was calculated from diets II, III and animal fat, all which were actually assayed values.

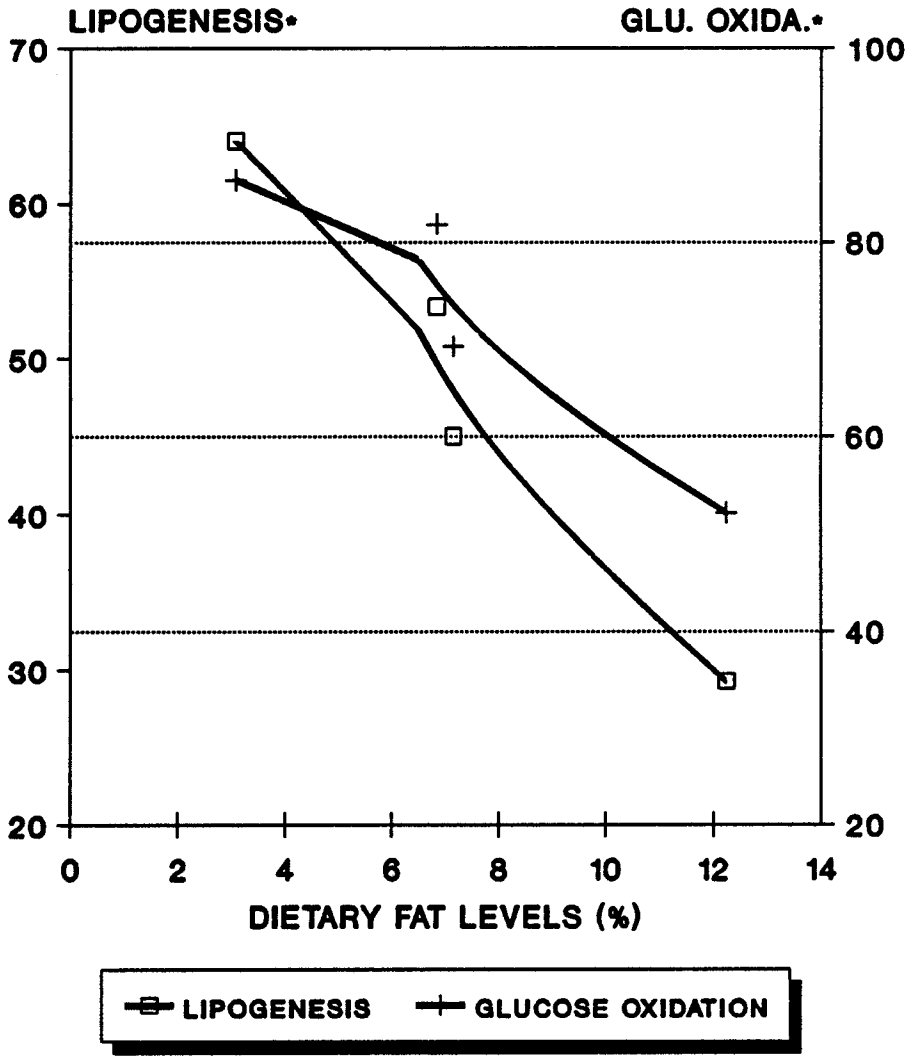

Figure III.1. The effects of dietary fat on liver lipogenesis and glucose oxidation. Unit = nM/h/g liver/100 g body weight.

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CHAPTER IV

TIME-COURSE OF ABDOMINAL FAT DEPOSITION AND EFFECTS OF TIMING OF FEED RESTRICTION IN TWO COMMERCIAL STRAINS OF BROILERS[1]

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Abstract

Peterson X Arbor Acres and Vantress X Hubbard broiler strain crosses were used to determine the rate of abdominal fat accumulation and effect of six days feed restriction starting at 7, 14, 21 and 28 days of age on abdominal fat accumulation. Broilers were sacrificed at day one and at the end of 7, 14, 21, 28, 35, 42, and 49 days of age to determine the rate of abdominal fat accumulation in the broiler. The abdominal fat levels increased as the broilers grew older. However, the highest potential for abdominal fat deposition occurred during the first 14 days of age.

When the feed intake of the broilers was restricted for a 6 day period at 7, 14, 21 and 28 days of age, feed efficiency was significantly ($P<.05$) improved compared with broilers fed ad libitum. There were no significant differences in body weights between the restricted and ad libitum-fed broilers. The abdominal fat levels in broilers restricted-fed starting at 7 and 14 days old were significantly ($P<.05$) lower than in the ad libitum fed group at 7 weeks of age. The difference was approximately 13%. Compensatory growth of the restricted broilers occurred slowly and fitted a polynomial mathematical model. Therefore, early feed restriction did improve feed efficiency and reduce abdominal fat without significantly affecting the body weight.

KEY WORDS. Abdominal fat, feed restriction, compensatory growth, broilers.

Introduction

Reducing the production costs and improving carcass quality of dressed broilers are both important factors in broiler production. High production costs reduce profitability in the broiler industry, and poor carcass quality such as excessive carcass fat makes the product (dressed bird) less desirable to the consumers. Numerous experiments were conducted on the nutrition of the early stage of broiler (Fancher and Jensen, 1988; Bartov, 1987; Plavnik and Hurwitz, 1985; Plavnik et al 1986; Rosebrough et al., 1986) to reduce the carcass fat. In these studies, the effects of protein (Fancher and Jensen, 1988) and fat (Bartov, 1987) nutrition in early life on fat accumulation in broilers were investigated. Plavnik and Hurwitz, (1985), Plavnik et al., (1986), Rosebrough et al., (1986) studied the effects of early feed restriction on fat deposition and metabolism.

Feed restriction of chickens and compensatory growth have been studied widely because both of these factors can improve feed efficiency and save energy by reducing metabolic energy loss. The ability to compensate for growth in animals after a period of undernutrition is called compensatory growth. Some recent research used early feed restriction to improve broiler carcass quality by reducing carcass fat deposition. An experiment by Plavnik et al. (1986) showed that broiler chickens subjected to a severe feed restriction from 7 to 13 days of age used their feed more efficiently (4%-12%) and

accumulated less abdominal fat (17%-30%) compared to ad libitum-fed chickens. Rosebrough et al. (1988) fed energy at the maintenance level to male broiler chickens from 6 to 12 days of age, and found the 6 day energy maintenance level (40 kcal/chick/day) decreased ($P<.05$) in vitro lipogenesis, fatty acid synthesis, activity of malic enzyme and absolute weight of abdominal fat pads as well as their proportion to body weight at 54 days when compared to ad libitum.

The investigations reported here were to determine the rate of fat deposition in broilers from day-old to 7 weeks of age and to study the effects of six-day feed restriction starting at 7, 14, 21 and 28 days of age on abdominal fat level, and the effect of compensatory growth at 7 weeks of age.

Materials and Methods

Standard rearing methods were used in these experiments. The floor pens were covered with 5 cm clean wood shavings. Feed was provided ad libitum in each pen with a hanging feeder (45.7 cm diameter). Water was provided with a hanging Plasson waterer in each pen. The diets were prepared according to NRC (1985) and shown in Table IV. 1.

Trial 1. The Time-Course of Fat Deposition. Seven hundred and forty Peterson X Arbor Acres and Vantress X Hubbard broiler strain cross chicks was used to determine the rate of abdominal fat deposition. Chicks were housed in two conventional litter floor pens. Fourteen chicks of each sex were weighed individually and sacrificed at 0, 7, 14, 21, 28, 35, 42, and 49 days of age and the abdominal fat was weighed. The percent abdominal fat was calculated to express changes of carcass fat. The percent abdominal fat at the end of a week, subtracting percent abdominal fat at the beginning of the same week, was taken as the increase of fat for the week. This experiment was repeated using Vantress X Hubbard strain cross.

Trial 2. Effects of Feed Restriction on Abdominal Fat. Nine hundred Peterson X Arbor Acres broiler strain cross chicks were used in this experiment. There were five treatments of 180 chicks per treatment. Treatment one was fed ad libitum

from day old to 7 weeks of age. Treatment two was fed 1.49 Kcal/g $BW^{2/3}$ (Plavnik and Hurwitz, 1985) of body weight daily for 6 days beginning at one week. Treatment three was fed 1.75 Kcal/g $BW^{2/3}$ of body weight daily for six days beginning at two weeks. Treatments four and five were fed 2.14 Kcal/g $BW^{2/3}$ of body weight daily for six days beginning at 3 and 4 weeks, respectively. The 180 chicks of each treatment were assigned to 4 pens (replicates). Sixteen male and sixteen female birds from each pen were sacrificed at the end of 7 weeks for the measurement of abdominal fat.

Trial 3. Compensatory Growth. Three hundred and thirty six Vantress X Hubbard strain cross chicks were used in this experiment. Half of the chicks were randomly assigned to four pens of 42 chicks per pen and fed ad libitum from day-old to 54 days of age. The other half was also randomly assigned to four pens and the energy intake of the chicks was limited to maintenance level (1.49 kcal/g $BW^{2/3}$) from 7 to 12 days, then fed ad libitum from 13 to 54 days of age. Body weights of male and female chicks in each pen were measured on day 1, 7, 13, 21, 28, 35, 42, 47 and 54. Compensatory and normal growth models were postulated by using computer.

Data were expressed as means of treatments except where noted. Treatment effects within the experiment were determined by one-way ANOVA with a level of statistical significance of 5%. Rankings of the means utilized Duncan's

Multiple Range Test for significance (Duncan, 1955). Statistical Analysis System (SAS, 1986) was used for statistical analysis and selection and fitness test of mathematical biological models for growth and rate of abdominal fat deposition.

Table IV. 1. Composition of experimental diets

Ingredients	Starter	Finisher
	%	
Corn, Yellow	55.08	64.59
Animal fat	6.13	3.69
Soybean meal(47.5% CP)	31.08	26.76
Fish meal (65% CP)	5.00	-
Meat & Bone meal (49.5% CP)	-	3.00
Def. phosp. (32% Ca)	1.10	.65
Gr. limestone	.91	.66
Salt	.30	.30
Vitamin mix*	.25	.25
Mineral mix#	.05	.05
d,l-Methionine,98%	.13	.06
Calculated analyses:		
Crude protein, %	23.00	20.00
ME, kcal/kg	3186	3193
Cal/Pr ratios	138	160
Ca, %	1.00	.90
Avail. Phos., %	.45	.40
Meth., %	.53	.38
Meth. + Cyst., %	.93	.72

*Supplies per kilogram of feed: vitamin A, 4125 IU; vitamin D_3, 1375 ICU; vitamin E, 1.4 IU; vitamin K, .69 mg; vitamin B_{12}, 6.7 μg; riboflavin, 4.1 mg; pantothenic acid 6.7 mg; niacin, 27.5 mg; choline, 275 mg; folic acid, .28 ug.

#Supplies per kg of feed: calcium, 97.5 mg; manganese, 60 mg; iron, 20 mg; iodine, 1.2 mg; zinc, 27.5 mg; cobalt, .02 mg; copper, 2 mg.

Results and Discussion

Growth rate of broiler chickens is accompanied by an increased percentage of body fat and a concomitant increase in the mass of abdominal and visceral fat. Significant correlation coefficients ($P<.01$) between percent body fat and weight of abdominal fat were reported by Hood, (1982). Yamashita et al., (1975) found a close correlation between muscle fat content and abdominal fat ($r=.94$). The weight of the abdominal fat is a useful indicator of body fatness. Therefore, the percent of abdominal fat was used in this trial.

Trial 1. Two commercial broiler strain crosses (Peterson X Arbor Acres and Vantress X Hubbard) were used to study the time-course of abdominal fat deposition. Peterson X Arbor Acres chicks were used to repeat the experiment. The growth and abdominal fat data from these two broiler strains were not statistically different between the strain crosses, therefore, the data were pooled and shown in Table IV.2 and Figure IV.1. Growth rate and abdominal fat levels increased with increasing age to 7 weeks (Table IV.2). The growth curve kept linearly increasing even at the 7 weeks of age. This may indicate that broilers still have high potential for fast growth at marketing time (7 week). Similar work reported by Hood, (1982), indicates this growth potential could last to 11 weeks of age.

Table IV.2. Time-course of growth and abdominal fat deposition of broilers from day-old to 7 weeks of age

	weeks of age							
	0	1	2	3	4	5	6	7
	Mean Body weight (g)							
Male	39.7	124	342	696	1089	1520	2021	2531
Female	37.3	120	317	580	923	1278	1682	2103
Average	38.5	123	329	638	1006	1399	1852	2317
	Percent abdominal fat (%)							
Male	-	.76	1.08	1.32	1.69	1.86	1.96	1.91
Female	-	.61	1.30	1.47	1.89	2.10	2.46	2.58
Average	-	.69	1.19	1.39	1.79	1.98	2.21	2.25

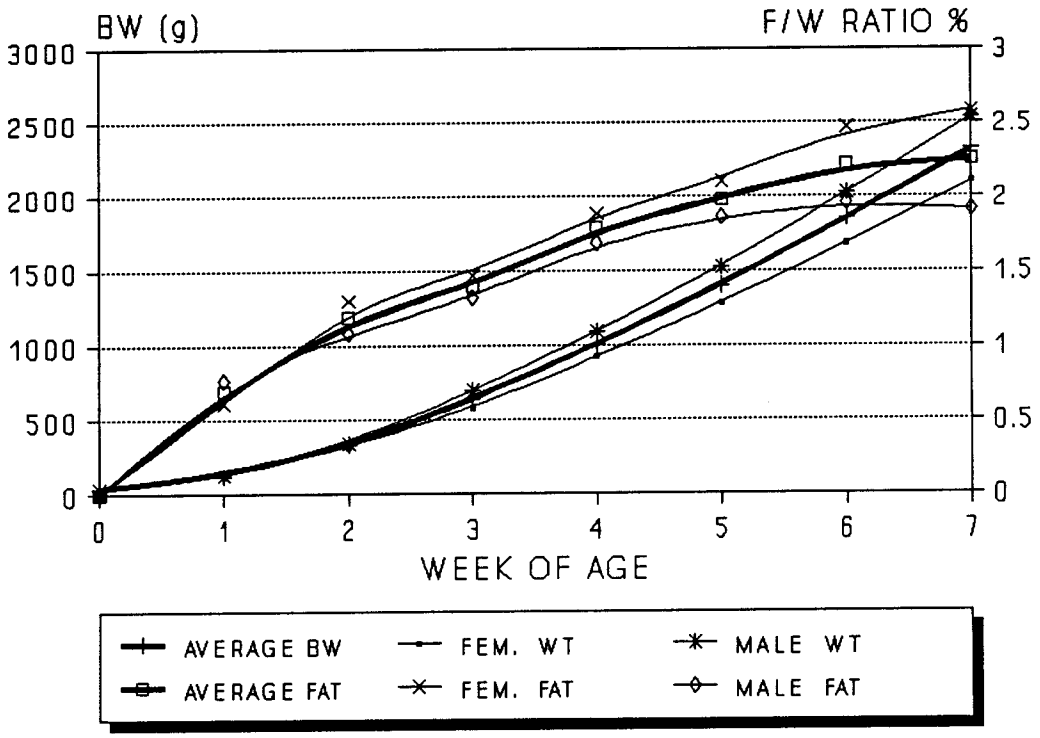

Figure IV.1. The time-course of percent abdominal fat and growth of commercial broilers. Data were pooled from two strain crosses of broilers repeated twice. The percent abdominal fat upon days of age followed a negative exponential growth curve, but the growth curve fits a cubic polynomial model.

The percent abdominal fat of both sexes of broilers increased with increasing body weight. However, their increasing curves were different from their corresponding growth curves (Figure IV.1). A model using SAS (1986) to describe the relation between percent abdominal fat and days of age was tried. Because of the nonlinear relationship between percent abdominal fat and days of age, their logarithmic, exponential, square, cubic and high power functions were considered and followed a forward selection, backward selection, selection by R-Square and Maximum R-Square improvement procedure (program is shown in Appendix A). The percent abdominal fat upon days of age followed a negative exponential growth curve (SAS, 1986):

Female $F = 3.1007(1 - e^{-.0340T})$ $(P<.0001)$

Male $F = 2.1621(1 - e^{-.0510T})$ $(P<.0001)$

Where, F is the percent abdominal fat in percentage, e is the Euler number (2.71828 to infinite) and T represents days of age. In contrast to these negative exponential growth curves, the gain of the body weight (pooled data) fits a cubic polynomial model:

$$W = 25.1448+5.2344T+1.3591T^2- .0108T^3 \quad (R^2=.931, P<.001)$$

Here W represents body weight (gm) and T is the time in days.

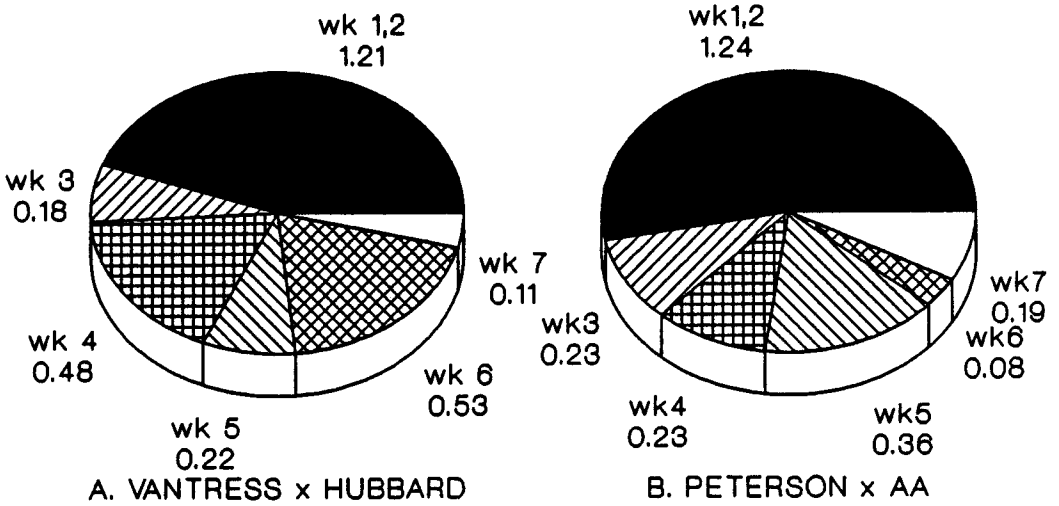

Figure IV.2. Weekly change of percent abdominal fat for two commercial strain crosses of broilers. We calculated by subtracting percent abdominal fat at the beginning of the week from percent abdominal fat at the end of same week. Both these two commercial strains of broiler chickens had a high potential in fat deposition in their first two weeks.

The negative exponential curves of percent abdominal fat of both sexes showed that percent abdominal fat increased rapidly at an early age and decreased when the broilers reached 6 weeks of age. The weekly increase of the percent abdominal fat was calculated by subtracting the percent abdominal fat at the beginning of the week from the percent abdominal fat at the end of same week. As shown in Figure IV.2, both broiler strain crosses had high potential for fat deposition in their first two weeks of age. This phenomenon may partially explain why early nutrition of the broilers is important for the control of carcass fat deposition for the remainder of the growing period (Fancher and Jensen, 1988; Bartov, 1987; Rosebrough et al., 1986).

Trial 2. Washburn and Bondari (1978) found that if feed consumption of broilers was significantly depressed for as little as one week at any time over a 3 to 8 week age period, weight gain would be significantly depressed with a significant decrease in efficiency of feed utilization and lower final body weights. Plavnik and Hurwitz (1985) and Plavnik and Hurwitz, (1986) studied the effects of feed restriction for 6, 10, 14, and 28 days on broiler performance and fat deposition. The 6-day feed restriction could significantly reduce the abdominal fat without significantly affecting body weight. The purpose of this experiment was to verify at which age the 6-day feed restriction could achieve

the best feed efficiency and growth rate.

As shown in Table IV.3, the feed efficiency of broilers restricted at 1, 2, 3 and 4 weeks of age was significantly ($P<.05$) better than broilers fed ad libitum. Feed restriction improved feed utilization. There were no significant differences between treatments for mixed sex and male body weights. However, the female body weights of the ad libitum group were heavier ($P<.05$) than the restricted treatments. The percent abdominal fat of broilers restricted at 1 and 2 weeks of age was lower ($P<.05$) than that of ad libitum-fed broilers. The difference between broilers fed ad libitum and restricted at 1 wk of age is about 13%. This difference is mainly from the female broilers rather than male broilers as shown in this Table IV.3. Plavnik and Hurwitz (1985) and Plavnik et al. (1986) reported that broiler chickens subjected to a severe feed restriction from 7 to 13 days of age used their feed more efficiently (4%-12%) and accumulated less abdominal fat (17%-30%) than ad libitum fed broilers.

Table IV.3. The effects of feeding restriction in different week of ages on abdominal fat deposition and broiler performance at 7 weeks of age

	Ad libitum	Restricted at wks of age			
		1	2	3	4
Feed Effi.	2.047[a]	1.956[b]	1.953[b]	1.939[b]	1.965[b]
Live Body Weight (kg)					
Male	2.367[a]	2.311[a]	2.322[a]	2.313[a]	2.290[a]
Female	2.019[a]	1.913[b]	1.932[b]	1.923[b]	1.918[b]
M+F mix	2.190[a]	2.098[a]	2.126[a]	2.139[a]	2.112[a]
Abdominal Fat (%)					
Male	3.233[a]	3.040[a]	3.001[a]	3.277[a]	3.127[a]
Female	3.443[a]	2.852[b]	2.856[b]	2.914[b]	2.905[b]
M+F mix	3.335[a]	2.929[b]	2.933[b]	3.102[ab]	3.023[ab]

[a,b]. Mean values in each row with different superscripts are significantly different ($P<.05$).

Table IV.4. The comparison of feed cost and dollar value of the broilers fed ad libitum and restricted at 1 wk of age[1]

Feeding system	Feed consumed (kg)	Feed cost ($)	Bird value ($)	Diff. ($)	Comparison ($)
Ad libitum	4.48	.90	1.30	.40	
Restricted	4.10	.82	1.25	.43	.03

1. Calculation is on a per bird basis and assumed other costs are same for these two groups.

From the data described above, feed restriction at 1 week of age is of practical interest to the broiler industry. Compared to ad libitum-fed broilers, feed restriction at 1 week of age significantly improved feed efficiency and resulted in a significant reduction in abdominal fat deposition, without significantly affecting the average body weight of broiler chickens at 7 weeks of age. Feed cost (20 cents/kg) and dollar value received for the broilers (59.4 cents/kg) for these two groups were calculated and presented in Table IV.4. Broilers restricted at 1 week of age could net 3 cents more per bird compared with ad libitum-fed broilers. If this method is practiced, a company which processes a million birds per week may net approximately $30,000 a week or 1.56 million dollars per year. The dressed broilers would be leaner also.

Trial 3. The purpose of this trial was to investigate compensatory growth after early feed restriction (7 to 12 days of age). As shown in Figure IV.3, during the 6 day feed restriction (7 to 12 days of age) period, growth of the broilers was delayed. The compensatory growth was also slow for the following several weeks. When broilers were weighed at the end of each week, statistical differences ($P<.05$) in live body weight between the restricted and ad libitum-fed broilers existed for the first 47 days and no difference ($P>.05$) at 54 days of age in this trial. However, in trial 2,

no statistical difference was observed at 49 instead of 54 days of age. The delayed growth noted in this experiment has also been observed by Plavnik and Hurwitz (1985), Plavnik and Hurwitz, (1986), and Reid and White (1977).

To develop a mathematical model for the compensatory growth, a growth curve which was after disturbance from the steady state (Nelder, 1961) was followed. However, the data failed to fit this model. The method of comprehensive selection was used (program used for selection is shown in Appendix 2). The following equations were found to fit the growth curve of the restricted and ad libitum-fed broilers:

(1) $W = .0291 + .0296T + .0004T^2 - .1082 * \ln(T)$ ($R^2 = .994$, $P < .0001$)

(2) $W = .0206 + .0099T + .0013T^2 - .00001T^3$ ($R^2 = .996$, $P < .0001$)

W represented body weight in kg and T is the age in days. Equation (1) is for the restricted broilers and (2) for the ad libitum-fed broilers. The difference between these two models indicates that the restricted broilers grew differently from the normal-fed birds, which may represent the compensatory growth.

In summary, commercial broilers have a high rate of abdominal fat deposition in the first two weeks of age. Feed restriction for 6 days at different ages showed that only restricted broilers at 1 week of age could reduce abdominal fat, improve feed efficiency and not statistically affect the final body weight. Compensatory growth after the early feed restriction happened at a slow rate.

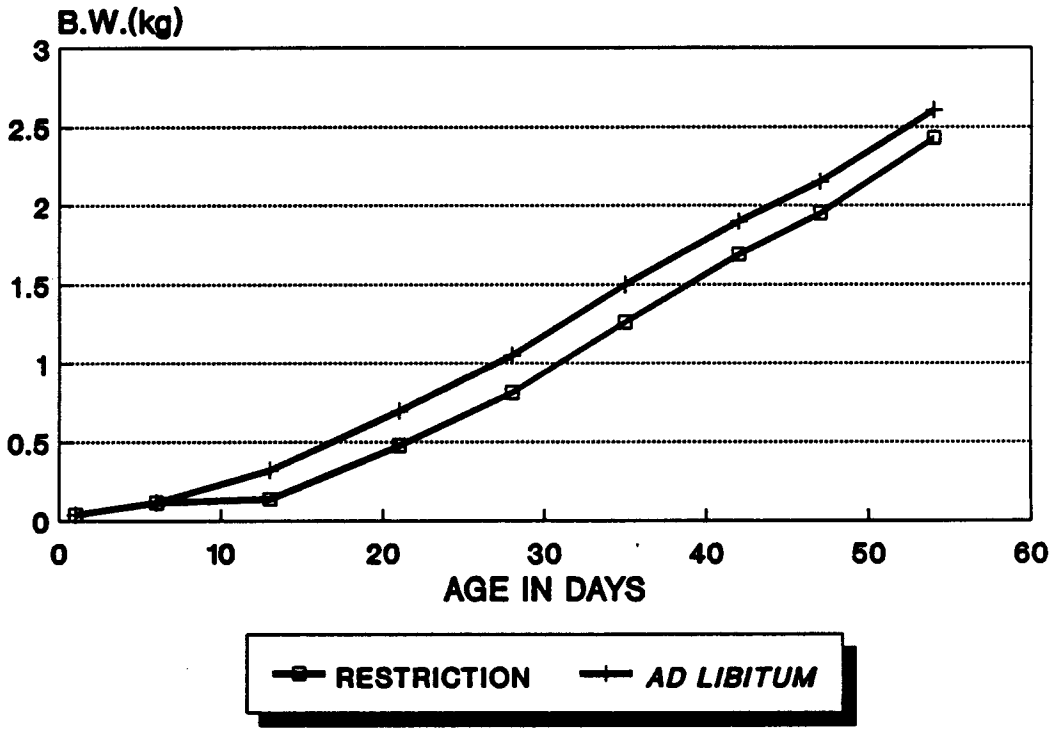

Figure IV.3. The compensatory and normal growth curves for the restricted and ad libitum-fed broilers. The former fits a model of $W = .0291 + .0296T + .0004T^2 - .1082 * \ln(T)$, ($R^2 = .994$, $P < .0001$) and the later fits the equation of $W = .0206 + .0099T + .0013T^2 - .00001T^3$, ($R^2 = .996$, $P < .0001$).

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CHAPTER V

THE EFFECT OF EARLY FEED RESTRICTION ON ADIPOCYTE CELLULARITY AND FAT METABOLISM IN BROILER CHICKENS

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Abstract

Peterson X Arbor Acres broiler strain cross chicks were either fed ad libitum from 0 to 54 days of age (DOA) or restricted-fed (1.49 Kcal/g $BW^{2/3}$ of body weight) daily from 7 to 12 days of age and then full-fed from 13 to 54 days of age to determine the effect on adipocyte cellularity, lipolysis, liver lipogenesis and glucose oxidation.

The number and volume of adipocytes were higher and smaller, respectively, in the feed restricted broilers than in the ad libitum-fed broilers.

Lipolysis (μM free fatty acid released/10^6 cells) was not significantly different ($P>.05$) between the treatments. Lipolysis significantly ($P<.05$) increased with the addition of isoproternal (10^{-4} M) and theophylline (10^{-3} M) for both treatments but no significant difference was observed between the two treatments.

Lipogenesis (μMole C^{14}-glucose converted/g liver) of the feed-restricted broilers was significantly ($P<.05$) lower than that of ad libitum-fed broilers at 2 and 7 weeks of ages. No significant difference in glucose oxidation was found between the treatments.

Under the condition of this experiment, limited-fed broilers at an early age had decreased liver lipogenesis, which may have led to less abdominal fat accumulation.

KEY WORDS: Adipocyte, broilers, lipogenesis, lipolysis, feed restriction.

Introduction

Selecting commercial broiler strain cross chickens for rapid growth rate has resulted in the increase of adipose tissue. The increase of adipose tissue is due to an increase in the numbers of fat cells by adipocyte multiplication (hyperplasia) or enlargement of these cells by lipid accumulation and mobilization within the cells (hypertrophy). In commercial strain of broilers, the number of adipose cells increased in the abdominal fat pad until 14 weeks of age (Hood, 1982, 1984), after which the cell numbers remained constant at about 270 X 10^6 cells per fat pad. The mean size of adipose cells in the first 14 weeks increased slowly. After 14 weeks adipose cell volume increased rapidly. The distribution of cell size is frequently bimodal with a population of small adipocytes (diameters less than 10 μm), and a population of large adipocytes. The influence of nutrition during early life on adipocyte numbers during the growth period is difficult to study because of the limitation of techniques in measuring cell numbers that have diameters less than 10 μm.

The control of lipid accumulation within the cells depends upon the balance between synthesis and degradation of triglyceride inside the body. In the avian species, the liver is the major site for lipogenesis (Goodridge, 1968a; O'Hea and Leveille, 1969), although bone marrow (Nir and Lin, 1982), adipose tissue and skin (Yeh and Leveille, 1973) make some

contribution. Saadoun and LeClercq, (1983) confirmed *in vivo* that in fat and lean line chickens, 65% and 68%, respectively, of *de novo* fatty acids came from liver synthesis. Lipolysis occurred in adipose tissues. Mobilization of triglyceride stored in the fat cell is catalyzed by the enzyme hormone sensitive lipase (HSL), which hydrolyses triglycerides to FFA and glycerol. The rate of lipolysis is dependent upon a metabolic cascade that ultimately results in the phosphorylation and activation of HSL.

Our research (Chapter IV) and some early studies (McMurtry *et al.*, 1988; Plavnik and Hurwitz, 1985, 1986; Anonymous, 1988; Rosebrough *et al.*, 1986, 1988) have shown that broiler chickens subjected to an early feed restriction utilized their feed more efficiently, decreased lipogenic capability and accumulated less abdominal fat compared to *ad libitum* fed broilers. Considering the changes in the abdominal fat pad in the restricted broilers, this study was designed to investigate the effects of early feed restriction on broiler adipose cellularity and hepatic lipolysis, lipogenesis and glucose oxidation in liver tissue.

Materials and Methods

Peterson X Arbor Acres broiler strain cross chicks were used in this experiment. Infra-red heat lamps were used in each pen, as the heat source. The floor pens were covered with 5 cm clean wood shavings. Feed was provided ad libitum in each pen with a hanging feeder (45.7 cm diameter). Water was provided with a hanging Plasson waterer in each pen. The chicks were randomly assigned to two treatments. Broiler chicks in one treatment were fed ad libitum from day-old to 8 weeks of age. The energy intake of the chicks in the second treatment was limited to maintenance level (1.49 kcal/B.W.$^{2/3}$) from 7 to 12 days, then the chicks were full fed from 13 to 56 days of age. The diets were prepared according to NRC (1985) and shown in Table V.1.

In Vitro Lipogenesis and Glucose Oxidation. Six broilers from each treatment were selected randomly and sacrificed by cervical dislocation. The livers were rapidly excised and weighed. A portion of the liver was sliced by using a tissue slicer which had a platform with a fixed blade mounted at the edge with a depth set at about .5 mm. The tissue was sliced horizontally by passage across the moistened platform against the knife edge. Lipogenesis and glucose oxidation were measured by a slightly modified radioactive method of Mersmann and Hu (1987). Krebs-Ringer-bicarbonate (KRB) buffer medium

Table V.1. Composition of experimental diets

Ingredient	Starter	Finisher
	%	
Corn, Yellow	55.08	64.59
Animal fat	6.13	3.69
Soybean meal(47.5% CP)	31.08	26.76
Fish meal (65% CP)	5.00	-
Meat & Bone ml (49.5% CP)	-	3.00
Def. phosp (32% Ca)	1.10	.65
Gr. limestone	.91	.66
Salt	.30	.30
Vitamin mix*	.25	.25
Mineral mix#	.05	.05
d,l-Methionine,98%	.13	.06
Calculated analyses:		
Crude protein, %	23.00	20.00
ME, kcal/kg	3186	3193
Cal/Pr ratios	138	160
Ca, %	1.00	.90
Avail. Phos., %	.45	.40
Meth., %	.53	.38
Meth. + Cyst., %	.93	.72

*Supplies per kilogram of feed: vitamin A, 4125 IU; vitamin D_3, 1375 ICU; vitamin E, 1.4 IU; vitamin K, .69 mg; vitamin B_{12}, 6.7 μg; riboflavin, 4.1 mg; pantothenic acid 6.7 mg; niacin, 27.5 mg; choline, 275 mg; folic acid, .28 ug.

#Supplies per kg of feed: calcium, 97.5 mg; manganese, 60 mg; iron, 20 mg; iodine, 1.2 mg; zinc, 27.5 mg; cobalt, .02 mg; copper, 2 mg.

containing 118 mM NaCl, 4.71 mM KCl,.63 mM Ca_2, 1.19 mM KH_2PO_4, 1.19 mM $MgSO_4 \cdot 7H_2O$ and 25 mM $NaHCO_3$, and was routinely gassed using a gas dispersion tube, for 15 min with 5% CO_2 in oxygen. The medium also contained 20 mM glucose, .5 μCi [U-^{14}C] glucose[7] per flask and .1 U porcine insulin[8] per ml. There were 100 mg of tissue slices per flask. Incubations were in triplicate in 25-ml siliconized, sealed Erlenmeyer flasks under an atmosphere of 5% CO_2 in oxygen. After 2 hrs at 39 C with reciprocal shaking at 90 strokes per min, the reaction was stopped by injection of .25 ml of 1 N H_2SO_4. The evolved CO_2 was trapped on filter papers in suspended center wells containing .2 ml hyamine hydroxide[9] during an additional 1 hr incubation. The lipids were extracted from the medium plus the tissue slices with chloroform-methanol (2:1, v/v). The washed extracts and center well were placed in a counting vial, 10 ml of counting fluid were added, and the samples were counted in a liquid scintillation spectrometer (Packard Model 2425) to determine the conversion of glucose to total lipids and CO_2, respectively.

In Vitro Lipolysis. Another six male broilers were killed by

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[7]Cat. No. 29756-9, Sigma Chemical Co., St. Louis, MO 63178

[8]Cat No. I-3505, Sigma Chemical Co., St. Louis, MO 63178

[9]Methylbenzethonium Hydroxide, Cat. No. M-1756, Sigma Chemical Co., St. Louis, MO 63178

cervical dislocation and the abdominal fat pads were rapidly excised and weighed. Unless otherwise indicated, lipolysis was measured in adipose tissue slices in a system somewhat similar to that for lipogenesis measurement (Mersmann and Hu, 1987; Hu et al., 1988). The medium of KRB (1/2 Ca^{++} concentration) contained 5.56 mM glucose, .56 mM ascorbate and 6% BSA medium. The basal lipolysis was measured in triplicate with no exogenous hormone added. Stimulated lipolysis was measured in triplicate in the presence of 10^{-4} M isoproterenol bitartrate, a beta-adrenergic agonist, and 10^{-3} M theophylline, a phosphodiesterase inhibitor. Incubations were for 2 hr at 39 C with reciprocal shaking at 90 strokes per min. The reaction was stopped by chilling the flasks on ice. The medium was filtered through cheesecloth, frozen at -20 C and later analyzed for free fatty acid concentration by extraction and microtitration (Kelley, 1965).

Adipose Tissue Cellularity. Adipocyte size and number were determined according to a procedure similar to that reported by Etherton et al. (1977), and Hu et al. (1988). Abdominal adipose tissue pieces of about 100 mg were rinsed three times in .9% NaCl at 37 C and transferred to a glass vial containing 3 ml of 50 mM collidine-HCl buffer (pH 7.1) and 5 ml of 3% osmium tetroxide[10] in collidine buffer. After 96 hr of

[10]EM Corp., Boston, MA 02167

fixation, the osmium solution was removed by aspiration and replaced by two 12-ml washes of .9% NaCl, each for 24 hrs. Finally, the saline was replaced by 10 ml of 8 M urea and left at room temperature for 48 hrs. Following dispersion with urea, adipocytes were washed through a 250-μm nylon screen[11], trapped on a 10-ml nylon screen and suspended using .01% Triton X-100 in .9% NaCl, pH 10. After appropriate dilution, adipocytes were counted and sized using a Coulter Counter[12] with a 400-μm aperture tube. Diameter distributions were calculated by pooling cell numbers in each 10-μm diameter range, and plotted as the average percentage of adipocytes present in a 10-μm diameter range.

Data were statistically analyzed by the paired t-test (Steel and Torrie, 1980) and a level of 5% was recognized as statistically significant.

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[11]Small Parts Inc., Miami, FL 33238

[12]Model ZM, Coulter Electronics, Inc., Hialeah, FL 33010

Results and Discussion

The effects of early feed restriction on adipocyte cellularity of broilers at 4 and 6 weeks of age are shown in Table V.2. There was no difference between the ad libitum and the feed restricted groups in adipocyte numbers for whole abdominal fat pads at 4 and 6 weeks of age. However, the adipocyte numbers on a per gram tissue basis in the feed restricted broilers were significantly higher ($P<.05$) than the broilers full-fed at 4 weeks of age. The average adipocyte numbers per fat pad were 105×10^6 and 178×10^6 at 4 and 6 weeks of age, respectively.

The average fat cell diameter of restricted broilers were smaller ($P>.05$) than those of control broilers. When the solid volume of the fat cells was calculated using their diameters, there was about 10% difference between these two groups. The average adipocyte volume was 120 to 185 $\mu m^3 \times 10^3$ for these two ages. Both the values for adipocyte volume and number are comparable to the data of Hood (1982) and March et al. (1984). However, the adipocyte number and volume from this experiment were larger than those reported by Hood (1982) and March et al. (1984), which implies that today's broilers have both a faster growth rate and more total carcass fat.

Table V.2. The effect of ad libitum and early feed restriction of broilers on adipocyte number and size in the fat pad[1]

Feed System	Cell No.($X10^6$) g tissue	Cell No.($X10^6$) fat pad	Cell size Diameter(μm)	Cell size Vol($\mu m^3 X10^3$)
	4 weeks of age			
Ad libi.	5.51±.88[a]	106.4±19.8[a]	61.3±3.6[a]	125.6±7.1[a]
Restr.	6.64±.72[b]	109.0±25.7[a]	59.1±2.9[a]	112.2±5.2[a]
	6 weeks of age			
Ad libi.	4.08±.75[c]	178.0±29.5[b]	70.6±2.5[b]	190.5±5.8[b]
Restr.	4.49±1.4[c]	178.9±22.3[b]	68.7±5.0[b]	178.3±11.9[b]

1. Mean values plus standard errors in each column for each age period with different superscripts are significantly different (P<.05).

The hypothesis that adipocyte number is determined early in life and remains constant throughout adulthood has been suggested in rats and mice. The proliferative activity in adipose tissue ceased by about 10 weeks of postnatal, because no increase in adipocyte numbers were observed after that time (Greenwood et al. 1970; Hirsch and Han, 1969; Johnson and Hirsch, 1972; Knittle and Hirsch, 1968). In broilers, hyperplasia is an important phenomenon during the first week of growth, and can be observed until 12 to 15 weeks of age (Pfaff and Austic, 1976; Hood, 1982; and March et al., 1984). In this experiment, a 50% increase in total adipocyte numbers, and a 40% enhancement in adipocyte volume, from 4 to 6 weeks of age, respectively, were observed. This indicated that both hyperplasia and hypertrophy are important during the growth period in today's broilers.

Early studies fostered the concept of a critical period for determination of adipocyte number that occurs early in the growth period. The control of hyperplasia or hypertrophy in early nutrition is of interest. Knittle and Hirsch (1968) reported that undernutrition of rats prior to weaning resulted in reduced adipocyte number and this persisted into adulthood, even after the rats were returned to ad libitum chow feeding. In pullets, Pfaff and Austic (1976) suggested that adiposity in pullets may be influenced by nutritional conditions during the growth period. Low energy diets given prior to sexual maturity tended to reduce the number of fat cells during the

laying period. March and Hansen (1977) studied the effect of early fasting on later broiler adipocyte multiplication. Broilers were fasted for three days, either immediately after hatching or at 10 days of age, and the uptake of ^{3}H-thymidine into adipocytes as the rate of cell multiplication was measured. If feeding was begun after the first 3 day fast, multiplication of adipocytes in the retroperitoneal body occurred within 20 hr at a reduced rate but rapidly attained a normal rate compared to the full-fed chicks. If fasting was imposed on 10 day old broiler chicks, refeeding did not immediately stimulate adipocyte multiplication but there was a rapid repletion of the lipid lost from the cells. Data from this experiment showed that early feed restriction did not affect the adipocyte numbers at 4 and 6 weeks of age. There was a reduction of adipocyte volume for the restricted broilers. From the distribution of adipocytes (Figure V.1 and V.2), a shift of adipocyte distribution in the restricted broilers at both 4 and 6 weeks of age was observed. There were more small adipocytes in the restricted broilers, and more large cells in the full-fed broilers. This suggests that the decreased fat content in the early restricted broilers can be attributed to a decrease in cell size rather than cell numbers.

The decrease of adipocyte volume in the restricted broilers could be due to either the decrease of lipogenesis or the increase of lipolysis. However, no information was found

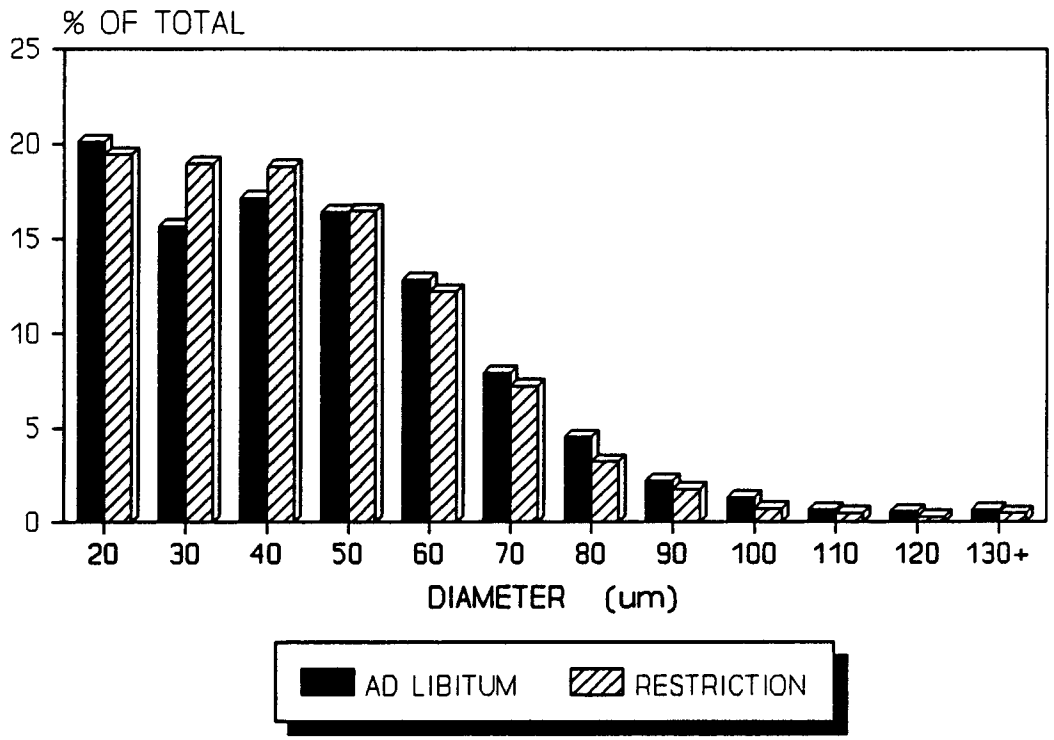

Figure V.1. The effect of ad libitum and early feed restriction on adipocyte distribution at 4 weeks of age. There were more small adipocytes in the restricted chickens, and on the contrary, more large cells in the ad libitum-fed broiler chickens

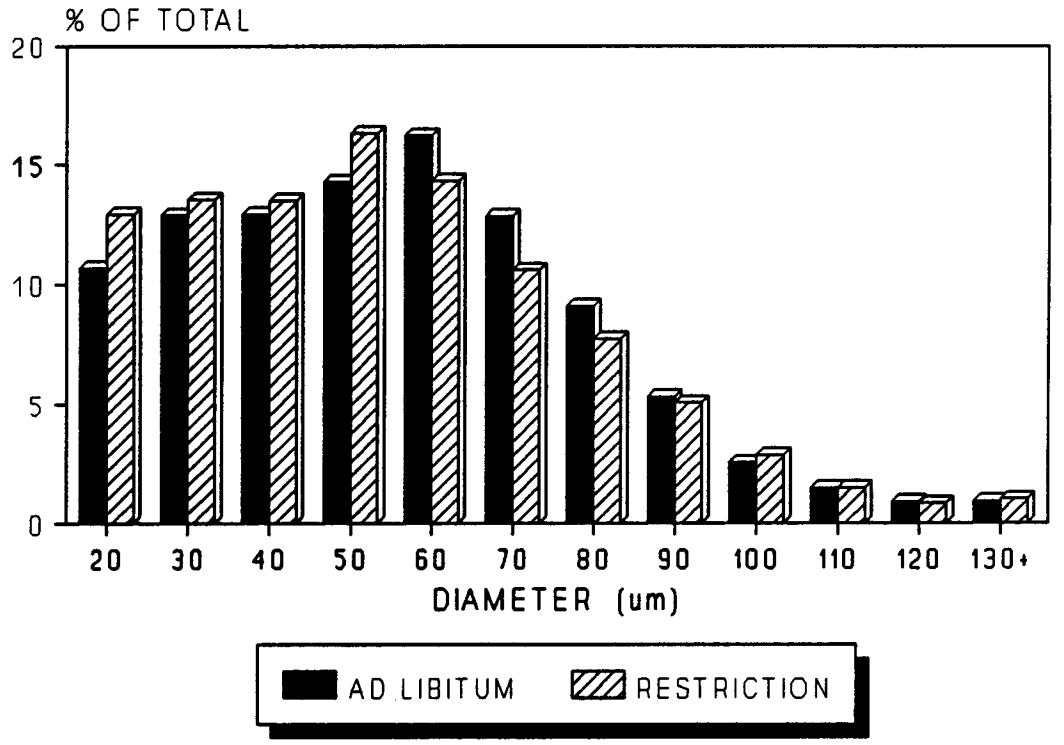

Figure V.2. The effect of ad libitum and early feed restriction on adipocyte distribution at 6 weeks of age. Similar to Figure V.1., there were more small adipocytes in the restricted chickens, and on the contrary, more large cells in the ad libitum-fed broiler chickens. This suggested that the decreased fat content in the early restricted chickens can be attributed to a decrease in cell size rather than cell numbers.

on the effect of early feed restriction on lipolysis in chickens. The data from this research showed that the effect of early feed restriction on lipolysis was small (Table V.3). There was no significant difference in lipolysis between restricted and full-fed broilers on a per million adipocyte basis ($\mu M/hr/10^6$ cell) in each age. However, the difference was significant on a per gram adipose tissue basis ($\mu M/hr/g$). Because the adipocyte numbers of restricted and full-fed broilers were the same (Table V.2), one would expect no difference in the total rate of lipolysis with these two groups of broilers. It is of interest to point out that the rate of lipolysis increased when the broilers grew older (6 weeks compared to 4 weeks of age).

Epinephrine, norepinephrine, and adrenocorticotropin stimulated lipolysis in birds only at very high concentrations (Carlson et al. 1964, Langslow, 1972). On the contrary, glucagon displayed a very potent action on lipolysis in chickens (Goodridge, 1968; Langslow and Hales, 1969; Langslow 1973). In this study, when comparing non-hormone and hormone stimulation, the addition of 10^{-4} M isoproternal and 10^{-3} M theophylline did significantly ($P<.05$) increase lipolysis in the chicken fat tissue (Table V.3). However, the responses of the adipose tissue lipolysis of the full-fed broilers to the added hormones were similar to that of restricted broilers on a per million fat cell basis at both ages. Kitabgi et al. (1976) reported that chicken adipocytes had significantly

Table V.3. The effect of ad libitum and early feed restriction of broiler on in vitro lipolysis in abdominal fat pad at 4 and 6 weeks of age[1]

Feed system	Non-hormone		Hormone stimulation	
	μM/g/h	μM/h/10^6 cell	μM/g/h	μM/h/10^6cell
	4 weeks of age			
Ad libitum	3.30±.56[a]	.61±.12[a]	4.07±.50[a]	.76±.18[a]
Restri.	3.74±1.2[a]	.56±.15[a]	4.68±1.1[b]	.70±.11[a]
	6 weeks of age			
Ad libitum	4.03±.14[b]	1.02±.18[b]	4.80±.42[c]	1.22±.30[b]
Restri.	4.46±.13[c]	1.04±.33[b]	4.73±1.3[c]	1.11±.37[b]

1. Mean values in each column for each age period with different superscripts are significantly different (P<.05).

higher capacity for the accumulation of cyclic AMP than rats. This may explain the fact that lipolysis was less responsive to exogenous hormones in these broilers.

Lipogenesis of restricted broilers was significantly lower ($P<.05$) than that of ad libitum-fed broilers at both 2 and 8 weeks of age (Table V.4). At two weeks of age, lipogenesis of ad libitum-fed group broilers was about 2.5 times higher on a per gram liver basis and 8 times higher on a whole liver basis than for the restricted broilers (Table V.4 and Figure V.3). At 8 weeks of age, the lipogenesis of ad libitum-fed broilers was larger by 2.2 to 2.8 times than the restricted broilers on a per gram tissue and per liver basis, respectively (Figure V.3). These findings concur with the data of Rosebrough et al. (1986). These researchers restricted broiler chicks from days 6 to 12 and measured lipogenesis ([2-^{14}C] sodium acetate incorporated into lipids). Lipogenesis of restricted broilers was much lower than in the ad libitum-fed broilers. After the 6-days feed restriction (day 6 to 12), the ad libitum refeeding enhanced the lipogenesis of these broiler chicks until 18 days of age compared to the ad libitum-fed broilers. At 27 days of age, the lipogenesis of the restricted broilers was 2 times less than ad libitum-fed broilers. By pooling these data, we can infer that early feed restriction could induce high activity of lipogenesis immediately after ad libitum refeeding because of compensation 6 days after the refeeding. This compensation

Table V.4. The effect of ad libitum and early feed restriction of broiler on in vitro hepatic lipogenesis at 2 and 8 weeks of age[1]

Feed system	2 week of age		8 week of age	
	nM/h/g tiss.	nM/h/liver	nM/h/g tiss.	nM/h/liver
Ad libitum	356.3±184[a]	3585.7±1850[a] (10.0 g)[2]	1553.0±681[a]	72347±30242[a] (48.6 g)[2]
Restric.	148.05±93[b]	470.5±104.6[b] (3.7 g)[2]	703.1±248[b]	26112±9248[b] (37.1 g)[2]

1. Mean values in each column with different superscripts are significantly different (P<.05).

2. Average liver weight.

disappeared and the lipogenesis of the early restricted broilers was reversed due to the accelerated growth and extra energy requirement. The data for hepatic glucose oxidation of the ad libitum-fed and restricted broilers are listed in (Table V.5). Glucose oxidation was similar on a per gram liver tissue basis. However, on the per chicken liver basis, the glucose oxidation rates were much higher ($P<.05$) in the full-fed broilers than in the restricted broilers at both 2 and 8 weeks of age. This may indicate that the ad libitum broilers had a higher energy consumption rate for lipogenesis or for other activities in liver metabolism.

In summary, early feed restriction (7 to 12 days of age) in broilers reduced carcass fat at 7 weeks of age (market time), which may be attributed to a reduction of adipocyte volume (Table V.2). Hyperplasia and hypertrophy still existed at both 4 and 6 weeks of age. However, early feed restriction did not affect the multiplication of the adipocytes. The slight reduction of adipocyte volume with feed restriction may represent a smaller accumulation of lipids inside the cells, which may be due to the decrease of lipogenesis (Table V.4). The effects of feed restriction on lipolysis were small and may not contribute to the decrease of obesity of these broilers. The rate of glucose oxidation concur with the changes of lipogenesis, which may indicate the utilization of glucose for fatty acid synthesis.

Table V.5. The effect of ad libitum and early feed restriction of broiler on in vitro hepatic glucose oxidation at 2 and 8 weeks of age

Feed System	2 weeks of age		8 weeks of age	
	μM/h/g	μM/h/liver	μM/h/g	μM/h/liver
Ad libitum	1.6±.25	15.9±4.0[a]	2.2±.7	111.2±33.4[a]
Restric.	1.6±.34	6.6±1.9[b]	1.7±.5	62.9±15.9[b]

Mean values in each column with different superscripts are significantly different (P<.05).

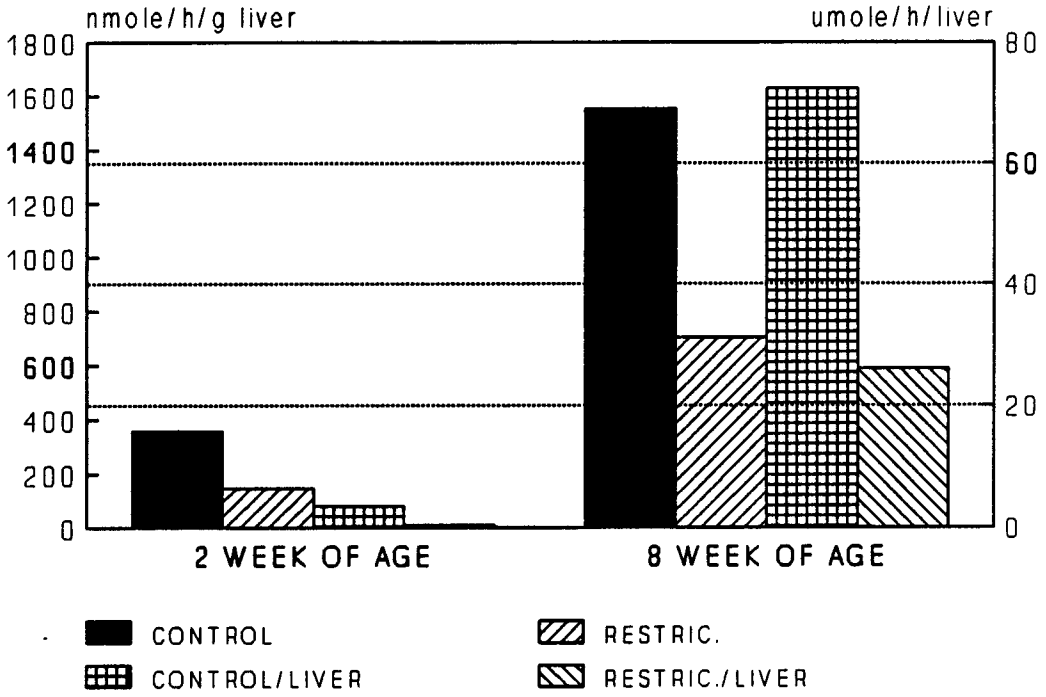

Figure V.3. The effects of ad libitum and early feed restriction on in vitro lipogenesis. At two wk of age, the lipogenesis of ad libitum-fed broilers was about 2.5 times higher on the per gram liver basis and 8 times higher on a whole liver basis. At 8 wk of age the difference got smaller. It was about 2.2 to 2.8 times lower for restricted broilers on a per gram tissue and per liver basis, respectively.

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CHAPTER VI

CONCLUSION

This research has demonstrated that excessive accumulation of abdominal fat is due to intake of excessive dietary energy. Decreased energy intake to reduce broiler fatness can be accomplished by restricting their daily energy needs from 7 to 12 days of age or by limiting the energy concentration of the feed.

The different Cal/Pr ratios in the diets did affect the abdominal fat deposition and metabolism. However, changing Cal/Pr ratio may not be necessary in altering the abdominal fat content if dietary animal fat is used in the diets which could inhibit de novo lipogenesis. Widening Cal/Pr ratio by increasing the addition of dietary fat improved feed efficiency and growth rate. A significant increase in abdominal fat existed with the wide Cal/Pr ratio group when compared to the narrow. There was a decrease of lipogenesis and glucose oxidation when the Cal/Pr ratio was wide, which may reflect the efficient utilization of animal fat from the diets. The addition of 5% DDBP or 10% cellulose, without changing Cal/Pr ratio, neither affected abdominal fat deposition nor broiler performance.

The best time for feed restriction which could reduce abdominal fat content without affecting the final body weight

was in the first two weeks of age. The change of percent abdominal fat fitted a negative exponential growth curve. Feed restriction for 6 days starting at different ages showed that feed efficiency of all restricted chickens was significantly improved when compared to ad libitum-fed broilers. Restricted broilers at 1 week of age reduced abdominal fat, improve feed efficiency and did not affect the final body weight at seven weeks of age. Compensatory growth after the early feed restriction occurred but at a slow rate.

The mechanism of the reduction of abdominal fat after the 6-days early feed restriction (7 to 12 days of age) was examined (Chapter V). Early feed restriction starting at 7 days of age for 6 days reduced carcass fat at 7 weeks of age (market time) which may be attributed to a reduction of adipocyte volume. Hyperplasia and hypertrophy still existed at both 4 and 6 weeks of age. However, early feed restriction did not affect the multiplication of the adipocytes. The reduction of adipocyte volume represented a smaller accumulation of lipids inside the cells, which may be due to the decrease of lipogenesis in the restricted broilers. Lipogenesis was significantly ($P<.05$) lower in the restricted broilers than in the ad libitum-fed broilers at 2 and 7 weeks of ages. No significant difference were observed in glucose oxidation and lipolysis between the restricted and ad libitum-fed broilers. However, hormone stimulation (isoproternal, 10^{-4}

M and theophylline, 10^{-3} M) significantly ($P<.05$) increased lipolysis for both treatments, but no significant difference was observed in response to the hormone stimulation between the ad libitum and restricted treatments. Lipolysis may not influence the reduction of abdominal fat in restricted broilers.

The 6-day feed restriction starting at 7 to 12 days of age was the best feeding scheme, which significantly improved feed efficiency and reduced carcass fat without significantly affecting final body weight. Decreased abdominal fat of the restricted-broilers was attributed to the reduced accumulation of fat inside the adipocytes which resulted in the small size of these cells. The reduced activity of lipogenesis in restricted-fed broilers may have contributed to the contraction of the adipocyte size. Therefore, feed restriction at an early age affected the abdominal fat level by decreasing lipogenesis.

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APPENDICES

APPENDIX A. PROGRAM FOR ESTABLISHING THE MODEL OF TIME-COURSE OF FAT DEPOSITION FOR TWO STRAINS OF COMMERCIAL BROILERS USING THE SAS 'NEGATIVE EXPONENTIAL GROWTH CURVE:Y=B0*(1-EXP(-B1*X))' PROCEDURE (SAS INST., 1986)

```
DATA KINETICS;
INPUT T F I M;
CARDS;
     (ENTER DATA, I.E. --7 .59322 7 .754, etc)
;
PROC NLIN BEST=10 METHOD=MARQUARDT;
PARMS b0=0 to 2 by .5 b1= .01 to .09 by .01;
MODEL F=b0*(1-exp(-b1*T));
DER. b0=1-exp(-b1*T);
DER. b1=b0*T*exp(-b1*T);
OUTPUT OUT=B P=FHAT R=FRESID;
PROC PLOT DATA=B;
PLOT F*T='a' FHAT*T='P' / OVERLAY VPOS=25;
PLOT FRESID*T / VREF=0 VPOS=25;
RUN;
PROC NLIN BEST=10 METHOD=MARQUARDT;
PARMS b0=0 to 2 by .5 b1= .01 to .09 by .01;
MODEL M=b0*(1-exp(-b1*I));
DER. b0=1-exp(-b1*I);
DER. b1=b0*I*exp(-b1*I);
OUTPUT OUT=B P=FHAT R=FRESID;
PROC PLOT DATA=B;
PLOT M*I='a' MHAT*I='P' / OVERLAY VPOS=25;
PLOT MRESID*I / VREF=0 VPOS=25;
RUN;
 (SAME PROGRAM FOR MALE AND FEMALE BROILERS).
```

SYMBOLS

T=DAYS OF AGE OF FEMALE BROILERS
F=ABDOMINAL FAT OVER BODY WEIGHT RATIOS (%) OF FEMALE BROILERS
I=DAYS OF AGE OF MALE BROILERS
M=ABDOMINAL FAT OVER BODY WEIGHT RATIOS (%) OF MALE BROILERS
P=MODEL PREDICTION

APPENDIX B. PROGRAM FOR SELECTING THE GROWTH MODELS OF NORMAL AND RESTRICTED BROILERS USING THE SAS FORWARD, BACKWARD, SELECTION BY R-SQUARE AND MAXIMUM R-SQUARE IMPROVEMENT PROCEDURE (SAS INST., 1986)

```
DATA GROWTH;
INPUT T W Y;
  t2=t*t;
  t3=t*t*t;
  t4=t*t*t*t;
  logt=log(t);
  expt=exp(t);
cards;
;
PROC REG;
 MODEL w=logt t expt t2 t3 t4 / selection=forward;
 MODEL w=logt t expt t2 t3 t4 / selection=backward;
 MODEL w=logt t expt t2 t3 t4 / selection=maxr;
 MODEL w=logt t expt t2 t3 t4 / selection=rsquare cp;
RUN;
PROC REG;
 MODEL w=t t2 t3;
 MODEL w=logt t t2;
RUN;
OUTPUT OUT=B p=X;
PROC PLOT DATA=B;
PLOT X*t;
RUN;
```

(SAME PROGRAM FOR RESTRICTED AND AD LIBITUM-FED BROILERS).

SYMBOLS

T=DAYS OF AGE OF THE TESTED BROILERS
W=BODY WEIGHT OF RESTRICTED BROILERS
Y=BODY WEIGHT OF AD LIBITUM-FED BROILERS
P=MODEL PREDICTION