STUDIES ON SOME POSSIBLE NUTRITIONAL ASPECTS OF SUDDEN DEATH SYNDROME AND ABDOMINAL FAT PAD DEPOSITION IN THE BROILER CHICKEN

By

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ABSTRACT

Six experiments were designed to investigate some possible nutritional aspects of Sudden Death Syndrome (SDS), a disease affecting broiler chickens and characterized by the sudden death of apparently healthy individuals. In two of the six experiments, the problem of excess abdominal fat deposition in commercial broilers was studied.

Factors including vitamin levels, growth rates, blood lipids, calorie:protein ratios and electrolyte levels were investigated with regard to SDS. The effects of feed restriction, early life caloric (fat) restriction and calorie:protein ratios on the production of abdominal fat were also studied.

SDS tended to effect male birds to a greater extent than females (Experiment 1). Growth rate appeared to influence the incidence of SDS (a rapid growth rate increased the number of birds affected) although large differences in weight gain seemed to be required to show differences in SDS mortality. Birds were observed to die from SDS as early as seven days of age with mortalities continuing until market age. From observations of birds that died from SDS, cause of death appeared to be acute heart failure.

Fortifying the diet with additional B vitamins, including biotin, and fat soluble vitamins did not affect the incidence of the syndrome (Experiment 2). In one of the six trials (Experiment 3) SDS mortality was found to be significantly decreased during the period of four to seven weeks in groups fed a high protein (decreased calorie:protein ratio) finisher diet. However, these results were not replicated in Experiment

5 and the effect of calorie:protein ratio on SDS mortality remains in doubt.

The results of Experiment 4 indicated that incorporating high levels of niacin (as nicotinic acid) in broiler diets could significantly reduce serum levels of triglycerides and cholesterol. However, in Experiment 5, these results were not duplicated, although triglyceride levels tended to be reduced by the addition of niacin to the diet. No significant effect of niacin was observed with regard to SDS mortality in Experiment 5. As blood lipid levels were also not significantly affected by niacin, a relationship between these parameters and SDS could not be established. In both Experiments 4 and 5, niacin did not affect performance parameters such as growth rate or feed conversion.

Experiment 6 provided some evidence that the NaCl content of the diet could increase the incidence of SDS during the finishing period of four to seven weeks. However as overall SDS mortality was not significantly increased the involvement of NaCl with the syndrome remains in doubt. Similarly, the addition of KCl to the diet was found to not significantly affect SDS mortality. Analysis of heart samples from SDS birds and other mortalities that occurred during the trial showed that the copper content of the SDS hearts was significantly less than that of other mortalities, suggesting the possibility that copper metabolism may be involved in the syndrome.

The results of Experiment 1 showed that limiting the energy intake of broilers by feeding 90% of ad libitum intake could significantly reduce abdominal fat deposition. Although seven week body weights were reduced by the limited feed intake, the saving in feed cost was found

to offset the loss of income due to the smaller market weights.

Elimination of added dietary fat from the starter ration (Experiment 3) for the first week of life was found not to be an effective means of reducing abdominal fat deposition measured at seven weeks of age and tended to increase the amount of abdominal fat. Similarly, increasing the protein level (decreasing the calorie:protein ratio) of the finishing diet did not significantly reduce the amount of fat in the abdominal area, although the birds fed the high protein diet tended to deposit less abdominal fat.

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ABBREVIATIONS

ALF	-	abdominal leaf fat
^{o}C	-	degree celsius
c.c.	-	cubic centimetre
cm	-	centimetre
C:P	-	calorie:protein
C.P.	-	crude protein
FFA	-	free fatty acids
g	-	gram
HDL	-	high density lipoprotein
I.C.U.	-	international chick unit
IL	-	intermittent lighting
I.U.	-	international unit
Kcal	-	kilocaloric
Kg	-	kilogram
LDL	-	low density lipoprotein
LL	-	lipoprotein lipase
ME	-	metabolizable energy
meq	-	milliequivalent
ml	-	millimetre
mM	-	millimole
N	-	normal
RDA	-	recommended dietary allowance
SDS	-	sudden death syndrome
µg	-	microgram
VLDL	-	very low density lipoprotein

INTRODUCTION

I. Sudden Death Syndrome

In recent years, broiler growers have become increasingly aware of mortality problems caused by a disease called Sudden Death Syndrome (SDS) also known as Acute Death Syndrome (ADS), Flip-Over, Heart Attack and/or Lung Edema.

SDS is characterized by the tendency for apparently healthy, rapidly growing broilers to suddenly die. No indications of the syndrome are observeable before the time of death. Characteristic post-mortem findings include edema of the lungs and contracted ventricles (heart). Little information has been reported as to possible causes or prevention of SDS.

Sudden Death Syndrome has been reported in many different countries. SDS has been found to affect between 1% and 2% of a population and may account for over one-half of the total flock mortality. Although the incidence is relatively low, because SDS can account for a significant percentage of overall mortality it is thus of economic significance and warrants further study.

II. Abdominal Fat

The presence of excess abdominal fat (greater than 2% of carcass weight) in the broiler chicken presents various problems to the industry. Abdominal fat can affect factors such as waste management, product yield, by-product composition and consumer acceptance (Heath et al. 1980). The disposal of fat contaminated water is a problem to processing plants in that pollution control, for example, requires the removal of fat from

the processing water (Kubena et al. 1974b). Consumers are often concerned with the amount of fat on the carcass as it relates to perceived quality of the product as well as the possible health risks associated with the consumption of fats. In addition, excess fat production represents wasted dietary energy.

Many factors have been implicated regarding abdominal fat and fat deposition in broilers. In reviewing the literature, Summers and Leeson (1979), found that diet composition, environment, temperature, type of housing, age and sex have influenced the amount of fat deposited by the bird. Strain differences have also been reported to be of concern (Nordstrom et al. 1978; Salleh et al. 1978).

To examine the problems of SDS and excessive fat deposition in broilers, experiments were designed to: (1) determine if various nutritional approaches could affect the incidence of SDS, (2) develop a theory to explain the syndrome, (3) evaluate the effectiveness of nutrient restriction and calorie:protein ratio on abdominal fat pad deposition.

LITERATURE REVIEW

I. Sudden Death Syndrome

A. Incidence

SDS has been reported in North America, England, Europe and Australia. Hemsley (1965) recorded mortalities due to lung edema (SDS) in a flock of 100,000 broilers and found that 0.46 percent of this population was affected. This figure represented 22.50 percent of the total recorded mortalities. Jackson et al. (1972) surveyed the mortality of nine broiler flocks comprising a total population of 69,068 birds. A total of 2,890 individuals (4.16 percent) died or were culled. The number of birds listed as "died in good condition" (SDS) was 451, representing 0.65 percent of the population or 15.60 percent of the total mortality. Volk et al. (1974), referred to SDS as "fatal syncope" and stated that 25.75 percent of the mortalities necropsied had died as a result of SDS. Brigden and Ridell (1975) reported mortality data on four flocks of broilers totalling 38,212 birds. The overall mortality was 3.80 percent whereas death due to SDS was reported as 1.13 percent (29.73 percent of the overall mortality).

Although the mortality due to SDS is relatively small it contributes a significant portion to overall mortality and therefore is of economic significance and should be investigated more thoroughly.

B. Characteristic Observations of SDS

i) Gross Appearance

Many researchers have observed that birds dying from SDS are usually found lying on their backs or sides with one or both legs

extended backwards (Fig. 1). No apparent abnormalities can be seen, the birds are in good flesh with body weights at or slightly above the flock average (Brigden and Riddell 1975; Ononiwu et al. 1979a; Hulan et al. 1980; Riddell and Orr 1980).

ii) Post-Mortem Condition

The condition most often observed upon post-mortem examination of SDS birds is edema and congestion of the lungs (Hemsley 1965; Jackson et al. 1972; Ononiwu et al. 1979a). Although lung edema is generally accepted as being an indication or factor in SDS mortality, Riddell and Orr (1980) found that congestion and edema of the lungs was not a consistent feature of SDS and if birds were examined shortly after death edema was very slight or absent. They suggested that these observed conditions may be a post-mortem artifact.

Ononiwu et al. (1979a) have listed other conditions possibly associated with SDS. Observations recorded at necropsy of SDS birds were fairly consistent and included the following: feed in the GI tract, generalized pulmonary congestion, enlarged and contracted ventricles, blood clots in the atria, dilated intestines with pale contents, slightly enlarged liver, discolored and empty gall bladder, pale kidneys and congested thyroid, spleen and thymus. Breast and thigh musculature were moist and pale. Histological examination of tissues including the adrenal glands, aorta, bone, bone marrow, Bursa of Fabricius, gizzard, proventriculus, gonads, heart, intestines, kidneys, liver, lungs, muscle, pancreas, peripheral nerves, spleen, thymus, thyroid and trachea were carried out on 142 birds that had died from SDS and compared to tissues

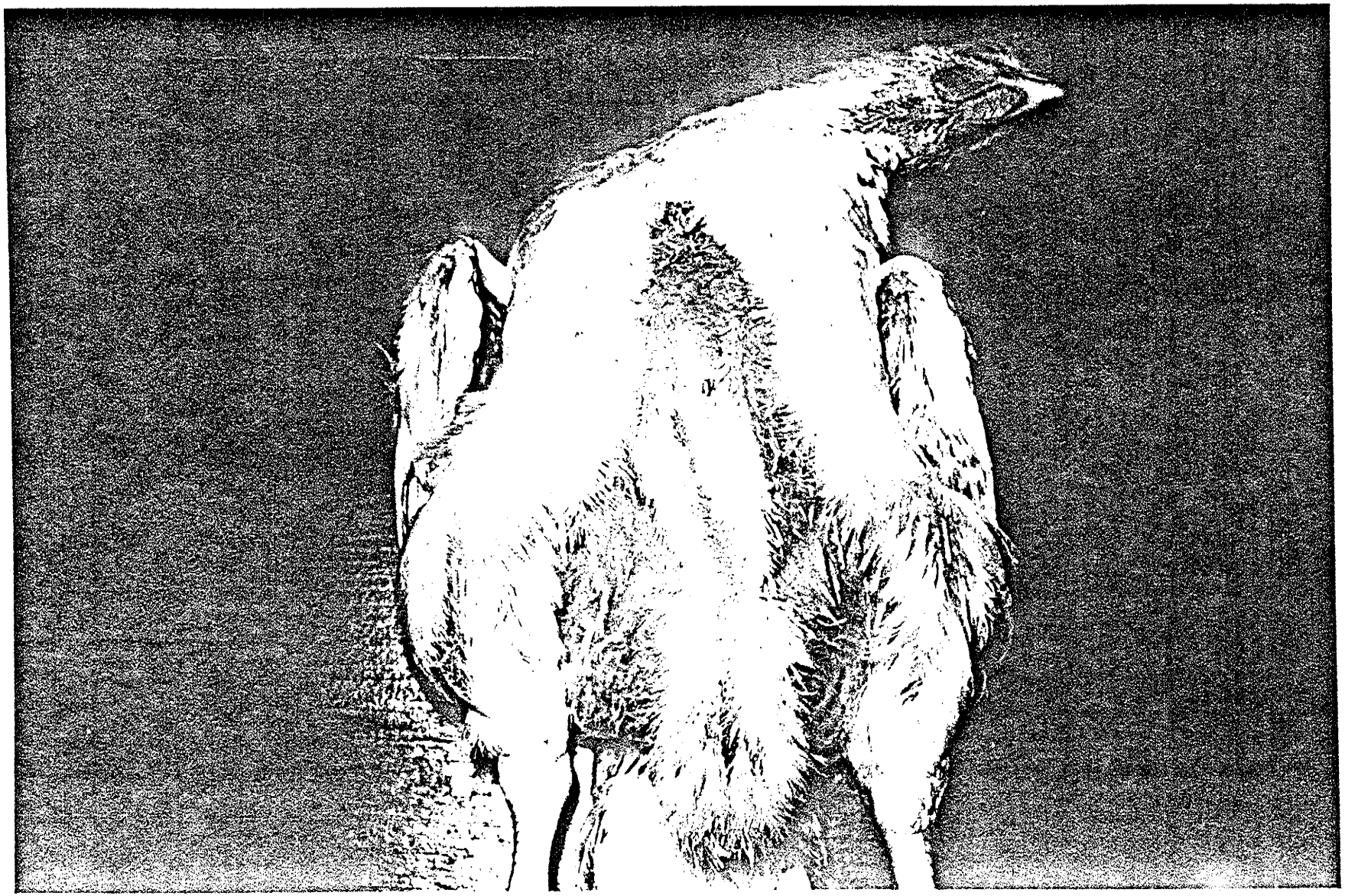

Figure 1. Characteristic position of a broiler chicken that died from Sudden Death Syndrome (SDS).

from control birds. Significant histopathological changes were found in numerous tissues. In 85% of the cases, the lungs exhibited vascular engorgement and edema of interstitial and interlobular connective tissue. Degeneration of myofibres and separation by edema was found in 70% of SDS hearts. Congestion was also observed in the sinusoidal spaces and vessels of the adrenals, thyroid and parathyroid.

Steele et al. (1982) also recorded post-mortem and histopathological findings in SDS affected birds. The appearance of the liver was found not to be a satisfactory indicator of SDS. Other tissues examined were found to have similar changes as those described by Ononiwu et al. (1979a).

The histology of the cardiac blood clots often found in SDS birds has been examined by Cassidy et al. (1975). Microscopically the specimens were classified into 3 categories; those containing fibrin and serum, those composed of erythrocytes; and those composed of fibrin and erythrocytes. Because of the shape, color, composition and lack of attachment, it was concluded that the clots were not thrombic. The authors stated that the structures were most likely blood clots of post-mortem origin.

Volk et al. (1974) did not note any marked macroscopic changes in the hearts of SDS birds. However, upon histologic examination of 630 hearts, circulatory disorders were noted with resulting regressive changes in the myocardium. Collapse of the peripheral circulation was less noticeable.

Riddell and Orr (1980) conducted chemical studies of the blood and histological investigations of the hearts of SDS birds. Examination of

heart tissues revealed no significant differences between control and SDS birds, although the authors did not rule out the possibility of ultra structural or biochemical lesions. Changes in the blood chemistry of mortalities were noted as the period of time from death to collection increased. Increases in the concentrations of potassium, phosphorous, magnesium and glucose were noted. A decrease in sodium concentration also occurred. There were no differences in these trends, however, between control and SDS birds. Elevated total serum lipid levels were found in some SDS affected birds.

C. Sex Differences and Mortality Patterns

Hemsley (1965) noted that SDS occurred more often in males and that the highest number of deaths took place when the birds were less than five or greater than seven weeks of age. A difference in incidence was also noted in relation to breed type, that is if the female parent was of New Hampshire and Rock base, the incidence of SDS in the progeny was less than if the female parent was of White Rock strain.

Jackson et al. (1972) also found that males were more susceptible to SDS stating that 72% of SDS mortalities occurred in males. Volk et al. (1974) noted that in 80% of SDS cases, the affected birds were males in apparently good condition. Brigden and Riddell (1975) found that 70% of SDS mortality occurred in males. SDS incidence was such that few birds died during the first week after which mortality increased to a peak level at three weeks of age and then declined until market age of 8 weeks. Hulan et al. (1980) reported that 70 to 80% of the mortalities in male and 20 to 25% of the mortality in female broiler chickens can be

attributed to SDS and that SDS can begin as early as the first week of life and peaks between 3 and 5 weeks of age. Gasperdone (1981) indicated that from field observations, SDS accounted for one-half of total flock mortality, the incidence was higher in males and SDS occurred as early as 10 days of age and then peaked at 4 to 5 weeks. Ononiwu et al. (1979b) also studied the differences in the incidence of SDS between males and females as well as the age at which birds were affected. Mortality attributable to SDS was reported as 37% of total mortality and as 1.0% of the total number of chicks started. Although there were more males dying from SDS than there were females, the difference was not statistically significant. Differences due to age, however, were significant, higher death rates were observed from the first to the third weeks of life than from the fourth to the seventh week. Steele et al. (1982) conducted trials involving more than 64,000 birds and found that SDS mortality accounted for an average death rate of 2.24% of the population with 74% of the mortalities comprised of male birds.

Although SDS does affect females, it appears to be most prevalent in males as most reports indicate roughly 70 to 75% of fatalities being male. SDS individuals show no apparent cause of death with mortalities beginning as early as one week and continuing until market age.

D. Possible Causes of SDS

SDS is generally thought to be as a result of acute cardiac arrest that results in the sudden death of the bird (Volk et al. 1974;

Gasperdone 1981). The possibility that cardiac clots commonly observed in SDS individuals were the immediate cause of death was discounted by Cassidy et al. (1975) in that the authors suggested the clots were of post-mortem origin. Volk et al. (1974) implied that circulatory changes with resulting regressive changes in the myocardium may be related to cardiac shock and death. Riddell and Orr (1980) suggested that ultrastructural or biochemical lesions of the heart could contribute to the syndrome and also stated that blood lipid levels may be involved.

The most detailed theory of a possible sequence of events of SDS was put forth by Ononiwu et al. (1979a). These authors stated that when a disease is acute, many pathological lesions are associated with vascular disturbances and that circulatory lesions can lead to increased permeability of peripheral vessels. Physiological stresses may result in even healthy capillaries becoming permeable. This permeability, as a result of short term increases in blood pressure, is normally reversible. If the stimulus is greater than the tolerance level, irreversible changes may take place in the vessel and the tissue it supplies. Ononiwu et al. theorized that SDS could be caused by heart damage resulting in lung edema and rendering the bird unable to breath. Fluid is lost from the circulatory system into the lung tissue causing peripheral circulatory failure and shock.

E. Reducing the Incidence of SDS

The amount of research conducted to explain or reduce the incidence of SDS is not extensive. Ononiwu et al. (1979b) investigated the effect

of continuous as opposed to intermittent lighting on the incidence of SDS. Four pens (2 male, 2 female) of 12,000 broilers each were used with all birds being exposed to continuous lighting for the first ten days of life. One pen of males and one pen of females were kept on continuous lighting until they reached market age of seven weeks. The other 2 pens were exposed to a lighting program of 3 hours light - 1 hour darkness for seven days followed by seven days of 2 hours light - 2 hours darkness. After this period through until seven weeks, the lighting regime was 1 hour light - 3 hours darkness. Continuous lighting was found to result in significantly greater mortalities due to SDS than the intermittent schedule. The authors postulated that the increased amount of light resulted in increased stress (that is, by inducing cannabalism, excitement and fighting) and that stress could thus produce a higher incidence of SDS.

Gasperdone (1981) determined the effect of various feeding schedules on growth rate and the incidence of SDS. Five feeding regimes were tested: a control program that was fed ad libitum; feed available for 16/24 hours; feed available for 8/24 hours and a program of feeding on alternate days. A fifth treatment was used in which the first kilogram of feed was low in protein, that is 17% as opposed to the 23% protein level of the other rations. Growth rates were affected by feeding schedule in that the 8 hour program, feeding on alternate days and the low protein starter ration resulted in reductions in growth rate. Total mortality and SDS mortality were found not to be related to feeding schedule although total and SDS mortalities were greatest on the low protein regime.

The effect on SDS of reduced feeding time was further investigated by Proudfoot and Hulan (1982). Differences between mash or crumble-pellet rations were also studied. Mortality of both males and females was unaffected by the various periods of feed restriction. Mortality was found to be affected, however, by the feed texture in that SDS was greater in groups fed the crumble-pellet diet. The authors stated that further research was being conducted before any conclusions could be attempted.

An additional report by these authors (Proudfoot et al. 1982) further investigated the effects of crumbled and pelleted feed on the incidence of SDS. Three different diets with identical formulation but of different textures were employed; that is a crumbled starter-pelleted finisher ration, a ground crumble-pellet ration and an all mash starter-finisher combination. SDS mortality was found to be significantly higher for birds fed the crumble-pellet or ground crumble-pellet rations compared with the all mash regime. The former groups had a SDS incidence of 1.82 percent and 1.85 percent respectively as compared to a 0.64 percent incidence in the later group. The increased SDS mortality was attributed to unidentified factor(s) involved with the crumble-pelleting process. Crumbles and pellets were produced by a steam-pressure-dry process.

Hulan et al. (1980) studied the effect of different levels of vitamins and the effects of additional vitamins (biotin, pyridoxine and thiamine) not normally added to broiler diets on the incidence of total and SDS mortality. Eight different vitamin premixes were used. The

control diet contained the standard vitamins (premix 1) usually added to practical diets at levels comparable to NRC recommendations. Premixes 2, 3 and 4 were the same as the control with the addition of biotin, pyridoxine and thiamine, respectively. These 3 vitamins were added to meet NRC requirements assuming that the ingredients of the ration contributed none of these three compounds. Premix 5 was the same as the control plus all 3 additional vitamins. Premix 6 had twice the vitamin level of the control premix. Premix 7 and 8 contained 2 and 4 times the control vitamin levels respectively as well as the extra vitamins added to premix 5. It was found that the addition of biotin, thiamine, and/or pyridoxine gave an indication of reduced total mortality although the addition of biotin alone yielded the only significant reduction. The reduced total mortality was due to a significant reduction in SDS mortality and the authors stated that further studies on the dose-response of biotin on SDS were needed.

Steele et al. (1982) examined the effect of biotin added to the drinking water of 64,000 commercial broiler chickens on the incidence of SDS. Biotin was added at 20 and 100 µg/day which supplied 1.2 to 5 times or 6 to 25 times NRC requirements depending on the stage of growth. The amount of biotin added to the water was adjusted to compensate for increasing water consumption. Samples of feed and water were assayed for biotin on a regular basis. Radioisotopic analysis of liver biotin status was used to confirm uptake of biotin by the birds. The authors found that SDS can occur in biotin repleted chickens and that high levels of biotin in the drinking water did not prevent or reduce the degree of SDS mortality.

As few studies have had success in showing increased or decreased SDS mortality, the need for further research is apparent.

II. Fat Deposition in the Broiler Chicken

A. Abdominal Fat Pad

The fat referred to as the abdominal fat pad of the chicken may be considered the layer of fat that surrounds the gizzard and lies between the abdominal muscles and the intestines. It extends within the ischium and around the Bursa of Fabricius and cloaca, attached at this point to the abdominal muscles (Kubena et al. 1974b). More simply, Hargis and Creger (1980) defined the abdominal fat pad to be the fat within the body cavity extending around the Bursa of Fabricius.

The deposition of abdominal fat has been highly correlated with carcass fat content and has been found to be a better predictor of carcass fat than other methods such as specific gravity (Becker et al. 1981a).

B. Diet Composition and Fat Deposition

The nutritional aspects (diet composition) of fat deposition in broilers have received considerable attention and are concerned with energy, protein and fat.

i) Calorie:Protein Ratio

Although Kubena et al. (1972; 1974a) found that increasing the energy content of broiler rations while maintaining a constant calorie: protein (C:P) ratio could increase carcass fat deposition, the majority of reported research indicated that C:P ratio is of greater importance

with regard to fat deposition than is energy per se. According to Bartov (1979) high dietary energy content is not in itself the major factor of excess energy consumption and thus increased fat deposition but rather the more important consideration is the C:P ratio.

Earlier research by Fraps (1943) demonstrated the effects of C:P ratio on fattening by substituting cottonseed oil for corn in broiler diets as well as by varying the levels of protein feeds or other ingredients with lower energy values than corn. By altering the C:P ratios by these methods, Fraps was able to produce carcasses ranging from 1.4% to 16.7% fat. Similarly, Donaldson et al. (1956) tested 23 different diets of varying C:P ratios with protein content ranging from 15.16% to 27.45%. At 4 weeks of age, a significant correlation existed between C:P ratio and carcass fat content.

Kondra et al. (1962) studied the effect of strain, sex and ration formulation on the percent of edible meat obtained from broilers. Four different diets were formulated with protein levels of 18% and 22.2% paired with energy levels of 2,125 and/or 1,910 calories of productive (net) energy per kg. The results were that at both energy levels, the higher protein percentage produced carcasses with less abdominal fat.

More recently, Summers et al. (1965) investigated the effect of C:P on carcass composition by formulating rations with protein percentages ranging from 10% to 26% and with ME values of between 1,135 and 1,510 kcal/lb (2,500 to 3,225 kcal/kg). The authors found that carcass fat increased with increasing energy values and decreased with increasing protein percentages.

The effect of C:P ratios on the fatness of broilers at different ages was investigated by Bartov et al. (1974a). By using a number of feeding periods (0-5, 5-8 and 8-10 weeks) and diets with different C:P ratios, the authors reported that a relatively low C:P ratio (132 vs 192) reduced the amount of dermal and visceral fat produced when compared to a higher ratio. It was also observed that the degree of fatness could be altered within 2 weeks by switching to a ration with a significantly different C:P ratio. Yoshida et al. (1966) and Morienoto (1970) similarly reported that the effects of dietary protein on carcass fat percentages is fairly fast and/or reversible. Thomas and Twining (1971) found that after only 10 days of feeding altered dietary protein concentrations, changes in carcass fat content were apparent.

Research on the effects of the C:P ratio of the finishing diet was provided by Griffiths et al. (1977a). A commercial broiler ration was fed to all birds from 0-4 weeks. At that time, birds were grouped into a factorially arranged randomized block design utilizing 2 energy levels and 3 C:P ratios. A C:P ratio of 160, considered to be optimal for growth and feed efficiency (Bartov et al. (1974a) was used for each energy level. Values of 188 and 139 were used as high and low C:P values. At 8 weeks of age, the birds were killed and abdominal fat pad weights recorded. Energy level of the finishing diet did not affect abdominal fat pad weight. However, decreasing the C:P ratio did result in significant reductions in the amount of abdominal fat pad deposition. Neither energy level nor C:P ratio were found to influence body weight gains in this experiment.

Hargis and Creger (1980) attempted to reduce the abdominal fat pad

content of broilers by varying the protein and energy levels of both starter and finishing rations. In the first of two trials, 9 dietary treatments were used. Treatments consisted of 3 different protein levels (24, 27 and 30%) with energy levels of 2,750, 2,680 and 2,550 kcal ME/kg respectively. None of these three diets contained added fat for the first 7 days of the trial after which time fat was reintroduced (at the expense of corn) and the same energy levels maintained. In addition, at each protein level, rations containing 2,860 and 3,080 kcal ME/kg were formulated and fed from day one. Each of these additional 6 rations contained added fat (as a mixture of animal and vegetable fat). The results of this trial showed that as the energy content increased, (increased C:P ratios) percent abdominal fat also increased, regardless of the protein percentage used. An exception to this were the groups receiving the rations containing 30% protein in that the least amount of abdominal fat produced among the 3 groups was found in those birds fed the ration formulated to contain the highest energy level. When comparing the C:P ratios of the 2,750/24 diet (C:P = 115), the 2,680/27 diet (C:P = 100) and the 3,080/30 diet (C:P = 103), all of which produced reductions in abdominal fat, it is apparent that the ratios were very similar. The two other 30% protein rations that produced significantly larger amounts of abdominal fat than any of the 3 previously mentioned formulations, had C:P ratios of 85 and 95 respectively. It may be that C:P ratios have an optimum range and that a very low ratio may produce results similar to a C:P ratio that is high. With lower energy values combined with a high protein level, excess protein consumption could occur, necessitating the conversion of the excess protein to fat. The possible excess protein in-

take might explain the apparent contradiction of the 30% protein groups.

In a second trial, the authors found that the effect of C:P ratio of the finishing diet could depend on the period of fat restriction. Birds restricted for 7 days showed no difference in abdominal fat when fed one of two finishing rations with different C:P ratios. Birds restricted from 0-14 days, however, had significant differences in fat pad weights when fed the two different finishing diets in that widening the C:P ratio resulted in an increase in abdominal fat.

Although energy level alone may be a factor in abdominal fat pad production, (Kubena et al. 1972, 1974a) calorie:protein ratio appears to be the more important consideration. In general, as the C:P ratio is increased, percent abdominal fat also increases. Conversely, when C:P ratios are decreased, abdominal fat also decreases. In terms of energy, increased fat deposition due to high ratios can be explained by the bird consuming more energy than is required to meet a relatively slow growth rate. Secondly, with a slight protein deficiency the birds can meet their protein requirement by increasing consumption. In both cases, the additional energy results in greater fat deposition, (Bartov 1979). In terms of protein, reduced hepatic lipogenesis has been noted in chicks receiving a high protein diet, an effect that cannot be explained entirely on the basis of reduced carbohydrate intake due to high protein levels, (Leveille et al. 1975). Another reason why lowered C:P ratios can decrease fat content of the carcass is that a higher protein intake increases uric acid synthesis, a process that requires considerable energy, (Bartov 1979).

The ability to influence abdominal fat deposition by altering C:P

ratios is limited. As demonstrated by Hargis and Creger (1980), C:P ratios that are too low due to the addition of excess protein may increase abdominal fat. In addition, these authors experienced problems with uric acid build up in the tissues of birds fed high protein diets. A third factor is the added cost of increasing protein in a commercial ration. The lowering of ratios by decreasing the energy content of a ration is also limited in that if energy is decreased too much, reduced growth rates and poorer feed conversion result. As stated by Hargis and Creger (1980), an optimum C:P ratio that would minimize abdominal fat and maintain performance is yet to be established.

ii) Protein Quality and Fattening

Not only does the absolute level of protein in the diet (and thus the C:P ratio) of broiler chickens have an influence on performance parameters, the amino acid make-up of the protein involved can also have an effect. Methionine (Baldini and Rosenberg 1955), lysine (Schwartz et al. 1958) and arginine (Scott and Forbes 1958) have all been shown to be related to the energy requirement of the bird, in that even if the protein percent of a ration is adequate, performance will be adversely effected if the levels of these amino acids are inadequate.

A severe deficiency of one or more amino acids can have a different effect on carcass composition than that of a low protein diet or a ration with a wide C:P ratio. In the latter cases, increased carcass fat may be as a result of chicks overconsuming (Bartov 1979). A severe amino acid deficiency, however, does not cause overconsumption so that growth and the concomitant increase in percent body fat does not occur. Almquist

(1954) suggested that this may be due to amino acids (other than the one(s) that is deficient) building up in the blood and impairing appetite. This hypothesis was shown to be true for tryptophan, lysine and methionine (Fisher and Shapiro 1961) as well as for arginine, isoleucine and valine (Hill and Olsen 1963).

A marginal amino acid deficiency does not, however, retard growth but does increase fattening in chicks. Carew and Hill (1961) showed this to be true in the case of marginal methionine deficiency. The lower levels of methionine or lysine increased hepatic fatty acid synthesis (the liver being the main site of lipogenic activity in the chick) and thus carcass fat. Persad and Sedagopan (1976) found that supplemental methionine could lower body fat content.

That amino acids other than methionine can influence carcass composition has been demonstrated by numerous authors. Hill and Olsen (1963) and Coombs (1967) found that lysine can influence carcass fat in that, when compared to isocaloric diets containing lysine levels exceeding requirements, rations with 60% of recommended amounts of lysine produced a significantly fatter bird. Velu et al. (1972) discovered that adding excess lysine or isoleucine to diets containing a balanced mixture of amino acids could decrease carcass fat content. Proline, however, was found to have no effect. Contrary to these reports, Yeh and Leveille (1969) and Seaton et al. (1970) found no reduction in carcass fat when excess lysine was added to chick rations.

Glutamic acid has also been found to be a factor in carcass composition, (Velu et al. 1971). When the level of glutamic acid was increased from 10 to 16% in a diet containing low levels of essential

amino acids, carcass fat was reduced without affecting growth rate. Greater reductions in fatness were obtained, however, when the concentration of the amino acid mixture as a whole was increased. Somewhat contradictory to the effects of adding a high quality amino acid mixture, are the results of Griffiths et al. (1977a) who found that the addition of a poor quality protein source (feather meal) was as effective in reducing abdominal fat pad size as a high quality protein source (soybean meal). It may be possible, however, that because the feather meal made up a small percentage of the total protein in the feed (and that the protein percentage was in excess of requirements), the resulting amino acid balance was not of as poor a quality as suggested by the authors.

In light of the reported effects and importance of various amino acids regarding carcass composition, not only is the C:P ratio of importance but also the amino acid balance must be considered in increasing or decreasing the fat content of broiler carcasses.

iii) Dietary Fat

The effect of dietary fat on carcass composition has been investigated with regard to the amount and type of fat deposited. Donaldson et al. (1956) found that increasing the energy level of broiler diets by substituting fat for carbohydrate on a weight basis resulted in an increase in carcass fat content. This observation, however was likely due to the increased C:P ratio produced when fat replaced carbohydrate on a weight rather than a caloric basis and not necessarily due to the fat itself.

Edwards and Hart (1971) found that various lipid sources such as

lard or soybean oil could provide the sole source of non-protein energy in chick rations. No differences in carcass fat content resulted from this replacement of carbohydrate by fat although the fatty acid composition of the carcass fat was affected by the type of fat fed. Similarly, Edwards et al. (1973) tested beef tallow, poultry fat and cottonseed oil to determine the amounts and make up of carcass fat produced. The type of fat did not influence gross body composition but did affect the fatty acid composition of adipose tissue.

Griffiths et al. (1977b) fed four different levels of fat (0, 3, 6 and 9% added fat) and measured abdominal fat pad size at 8 weeks of age. The types of fat used included corn oil, poultry grease and an animal and vegetable fat blend. Each type of fat was added at each of the aforementioned percentages. All diets were formulated to be isocaloric and isoaminoacetic for methionine and lysine. At 8 weeks of age, the four different types and levels of dietary fat were found to have no effect on abdominal fat pad size.

Several other researchers (Bartov et al. 1974b; Fuller and Rendon 1977; Griffiths et al. 1977a; Bartov and Bornstein 1977) have also reported that increasing the energy content of broiler rations by adding fat has no effect on carcass fat deposition as long as the C:P ratio is kept constant.

In contrast to previously mentioned research, Deaton et al. (1981) reported that the amount of animal fat added to broiler feeds could influence abdominal fat pad size even if the C:P ratios were kept constant. Animal fat was added to rations at percentages of 4, 7 and 10%. The rations were formulated to contain 3,185 kcal ME/kg and 21% protein.

Each diet was tested at two different rearing temperatures (21°C and 29°C). At 7 weeks of age, randomly selected birds were sacrificed and abdominal fat pad weights determined. It was found that as dietary fat increased, the amount of abdominal fat increased. These increases were significant for males and females and within each temperature tested with the exception of males reared at 21°C and fed 4 and 7 percent added fat. The authors concluded that added dietary fat produces increased abdominal fat. They did not, however, compare their results or explain their conclusions with regards to other contradictory research.

The majority of the research indicates that the use of various fat sources in commercial broiler diets does not contribute to excess fat deposition providing C:P ratios are considered and that reducing the amounts of fat normally added will not contribute to significant reductions in abdominal fat.

C. Genetic Factors

The traits most often selected for in broiler chickens have included maximum body size and growth rate with little regard to factors such as carcass composition or abdominal fat pad size. That abdominal fat pad size is a highly heritable characteristic has been demonstrated by Ricard and Rouvier (1967; 1969).

Numerous studies with mice have shown that selecting for increased body weight results in animals with higher proportions of body fat, (Fowler 1958; Roberts 1965; Timon et al. 1970; Eisen et al. 1977). Contrary to these reports, Lang and Legates 1969 and Dawson et al. 1972, have demonstrated that selecting for increased body weight in

mice and rats does not always result in greater amounts of body fat being produced.

In the case of the broiler chicken, there is also some contradiction although the majority of work done regarding selection for increased weight gain and body size indicates that these factors have significantly contributed to excess carcass fat, (Ricard and Rouvier 1969; Proudman et al. 1970; Wethli and Wesséls 1973).

Siegel and Wisman (1966) suggested that selection for growth rate or feed efficiency may be complicated by the correlation of these traits with appetite. Griffiths et al. (1978), however, considered the possibility that neither growth rate nor appetite may be involved with fat deposition. Griffiths found that the amount of carcass fat was directly related to the degree of abdominal fat deposition. It was also found, however, that abdominal fat was not highly correlated with weight gain, feed intake or feed efficiency so it follows that selecting for these traits would not necessarily affect fat pad or carcass fat deposition. Pym and Solvyns (1979) similarly questioned the suggestion that selecting for growth rate affects fat deposition. An unselected control line was compared to a line selected for increased weight gain. Birds were selected for 5 generations. There was a significant difference between 9 week body weights (the selected line being heavier) but no significant difference in abdominal fat pad production was observed.

Although selection for increased weight gain may not increase fat deposition, there is evidence to suggest that fat pad size may be decreased by selecting for a decreased (improved) feed conversion ratio. Pym and Solvyns (1979) determined body composition of 9 week old broilers

selected for increased weight gain, food consumption or food conversion ratio. The results showed that there were highly significant differences between the various lines with regard to carcass composition. Those birds that had been selected for improved feed conversion had the least amount of deposited fat. Birds selected for increased feed consumption produced carcasses with the least amount of protein and the greatest percentage of fat. The lines selected for weight gain and the control line were similar in carcass composition, both being intermediate to the other two previously mentioned lines. Carcass composition was also determined when the birds reached equal weights as well as equal ages so that possible effects of body weight could be ruled out. At equal weights, the same patterns regarding carcass composition were again observed. The authors stated that their results were in accordance with the predictions of Brody (1935) that the more efficient birds would store less fat than inefficient birds.

In support of Pym and Solvyns (1979) are the results of Leclerq et al. (1980). Their studies further investigated the relationship between carcass composition and feed conversion ratio. Two lines of broiler breeders were selected for 3 generations using a criteria of the ratio of abdominal fat to live weight, that is, a high ratio ("fatty") line and a low ratio ("lean") line. Birds were selected such that there was no significant change or disparity in body weights between lines. The results showed that selecting for high or low abdominal fat content was accompanied by changes in feed conversion ratios in that ratios were better (lower) in the lean line.

Hood and Pym (1982) further studied the effects of selection for

abdominal fat with regard to cellularity of the fat pad. Four lines of broilers were used; a non selected control line (C), a line selected for weight gain (W), one selected for food consumption (F), and a line selected for improved feed conversion ratio (E). Body weight gain was greatest in line W and lowest in C with the other two lines being intermediate in weight. Percent body fat was greatest in line F and lowest in line E. Percent abdominal fat followed the same pattern.

Differences in fat pad size were accounted for by differences in cell volume and cell number. Volumes of the adipocytes were greatest in line F and smallest in line E. The number of cells was greater in lines W and F than in lines C and E. The authors concluded that both volume and number of abdominal fat cells were responsible for fat pad size at 9 weeks of age. The authors attributed differences in the number of adipocytes to different growth rates. It was further stated, however, that growth rate is unlikely to determine the final number of cells produced as cell number is an inherited trait. Pfaff and Austic (1976) reported that dietary manipulation can delay the cessation of hyperplastic activity but will not affect final cell number at maturity. In birds with similar growth rates, cell numbers will also be similar so reduction in fat pad size could only be accomplished through reduced cell volume.

Numerous researchers have reported differences in fatness of different genetic stocks of chickens, (Littlefield 1972; Farr et al. 1977; Shapira et al 1978). Griffiths et al. (1978) sampled 4 different commercial crosses at 4 and 8 weeks of age for abdominal fat pad size. It was found that only one of the four strains showed a significant difference in the amount of abdominal fat. The authors suggested that

abdominal fat does vary with the strain of bird, however differences may not have been significant due to the great within strain individual variations displayed and the relatively small sample size. Merkley et al. (1980) provided further evidence that different strains can vary in abdominal fat pad size, recording strain differences of up to 0.38 percentage points which represented 7.7 grams of fat.

Contrary results were provided by Becker et al. (1981b) who, when investigating fat levels in five current broiler strains fed commercial rations, found no differences in body weights or abdominal fat percentages.

As strain differences with regard to abdominal fat have been shown to exist, it appears that genetic selection can be used to effect reduced amounts of carcass and abdominal fat. Although the work of Pym and Solvyns (1979) indicated that selection for increased weight gain may not necessarily further increase the amount of fat in present day broilers, neither will the continued selection of fast gaining, heavy birds solve the problem of excessively fat broilers. The results of Pym and Solvyns (1979), Leclerq et al. (1980) and Hood and Pym (1982) indicate that selecting for improved feed conversion, either alone or in combination with growth rate, holds promise for the reduction of excess fat in broilers. Although selection by these means may reduce desirable performance traits such as market weight, the leaner broiler would have better feed conversion and a greater percentage of its body weight as protein, (French and Hunton 1979).

D. Sex

Although Kubena et al. (1972) found little or no difference between male and female broilers with regard to carcass fat content, the majority of researchers have found gender to be a factor responsible for the fat content of broiler carcasses.

Edwards et al. (1973) found that the female chicken, regardless of age, had a higher fat content than did the male. Contrary to an earlier study, Kubena et al. (1974b) reported that within each dietary energy level studied, the abdominal fat in females made up a greater percentage of the carcass than it did in males.

Pym and Solvyns (1979) determined body composition in broilers selected for increased body weight, feed consumption and feed conversion ratio. In all generations and in all lines, females were found to contain more carcass fat and less protein than males. The authors list values of 115 g of fat/kg of carcass for females and 98 g of fat/kg of carcass for males, measured at 9 weeks of age.

Heath et al. (1980) compared the amounts of abdominal leaf fat removed from broiler chickens during the evisceration process. Abdominal leaf fat (ALF) was defined by the authors as the fat within the abdominal cavity which included the fat attached to the viscera and giblets and can be dissected out. The ALF, then, was greater than the fat pad alone. The fat pad accounted for 58.7% in females and 60.4% in males of the total ALF. Carcasses were divided into two groups, based on ALF as a percentage of live weight. In the low group ALF was less than 3% of live weight and in the high group ALF was greater than 4% of live weight. At 7 weeks of age, 35% of females and 21% of the males were in the high

group with 40% of the males and 24% of the females in the low group. At 8 weeks of age these differences were even more pronounced.

Becker et al. (1981a) compared abdominal fat and carcass fat percentages in 9 week old male and female broiler chickens. They also compared specific gravity as a predictor of carcass fat content to abdominal fat pad measurements. It was found that the females had a greater percentage of abdominal fat, (4.67%) compared to males (3.56%), based on abdominal fat/eviscerated carcass. Abdominal fat was highly correlated with carcass fat and was determined to be a better predictor of carcass fat than specific gravity. In a further study (Becker et al. 1981b), the sex differences with regard to the fat content of broilers were confirmed. The mean percentages of 5 different broiler strains for abdominal fat/live weight at 9 weeks were 2.9% and 3.3% for males and females respectively.

Hood (1982) investigated the cellular basis for growth of the abdominal fat pad in broilers. Although females were found to be lighter than males, they also had a higher percent of body fat at all ages tested (2, 5, 7 and 9 weeks). Males, however, were found to have a greater amount of lipids in the fat pad than females when measured at the same percentage of carcass fat. This observation does not necessarily mean that males have larger fat pads at market weight since at that time total carcass fat content is significantly lower in males. Further to this point, Kubena et al. (1974b) found that absolute quantity of abdominal fat was higher in females (29.5 g) than males (27.9 g) at 8 weeks of age. The fat pad weights were greater even though the females were lighter than the male birds.

In summary, it can be concluded that female broiler chickens contain greater percentages of carcass fat and greater percentages of abdominal fat than do male broilers. Even though females have smaller body weights at market age, the absolute amounts of carcass and/or abdominal fats are still slightly greater than the levels found in males.

E. Temperature

As well as affecting feed consumption and growth rate (high environmental temperatures can reduce both parameters), rearing temperatures can have an influence on carcass composition, (Mickleberry et al. 1966; Dale and Fuller 1980).

Increasing rearing temperature has been shown to increase carcass fat and decrease moisture content (Kleiber and Dougherty 1934; Winchester and Kleiber 1938). Scott et al. (1959) found that relatively cold environmental temperatures reduced fat deposition in ducklings. Kubena et al. (1972) found that there was a greater percent of carcass lipids in broilers raised at 29.3^{o}C than broilers exposed to 18.3^{o}C. This difference was also found when comparing birds raised at 32.2^{o}C as opposed to birds raised at 21^{o}C. Dale and Fuller (1980) found that broilers raised at a temperature of 31^{o}C had consistently (although not significantly) higher carcass lipid content than did broilers raised at 14^{o}C. The authors suggested that the lower lipid content could be explained by decreased temperatures necessitating increased energy expenditure for maintenance of body temperature. French and Hunton (1979) supported this concept, stating that high temperatures resulted in less of the birds energy intake being used to maintain body temperature.

Contrary to these reports, Adams et al. (1962) found no differences in carcass fat between chicks reared at 21°C and 29.4°C. Mickleberry et al. (1966) also compared the ether extract content of various tissues of birds raised at 21°C and 29°C. Although there was a trend towards higher ether extract in the 29°C group, no significant differences were noted. Deaton et al. (1978a) investigated abdominal fat pad production in broilers raised at 21°C and 29°C and found no significant differences due to temperature.

Although there appears to be contradiction in the literature, these differences may be explained by examining the range of temperatures that have been studied. The reports that have shown differences in the fat content of broilers as a result of temperature have generally employed a greater range in rearing temperatures. Kleiber and Dougherty (1934) raised chicks at 32°C and 21°C and found significant differences in carcass fat. Similar results were obtained by Kubena et al. (1972) who compared temperatures of 18.3°C to 29.3°C as well as 21°C to 32.2°C. Studies that failed to show differences (Adams et al. 1962; Mickleberry et al. 1966; Deaton et al. 1978a) generally employed smaller temperature ranges as well as lower maximums. It may be that relative extremes in rearing temperatures are required to show significant differences in fat content of broilers.

F. Light

The use of intermittent lighting (IL) has become more commonplace in the production of broiler chickens. In a review of the literature, Malone et al. (1980a) found that performance parameters, such as growth rate, have shown a wide variety of responses to the type of lighting

schedules used in that many authors reported increased body weights when an IL schedule was compared to continuous lighting. Other reports indicated that body weights may be decreased when broilers are reared under intermittent light. The effects of IL on the fat content of broilers have also shown varied results.

Deaton et al. (1976) compared the ether extract content of broilers raised under 4 different lighting regimes. A control group was exposed to continuous light at 12.9 lux from 0-8 weeks. The other 3 groups were as follows: 12 hours light (204.5 lux): 12 hours darkness; 12 hours light (204.5 lux): 12 hours light (12.9 lux), and 12 hours light (204.5 lux): 12 hours darkness for the first 5 weeks of life followed by 12 hours light (204.5 lux): 12 hours light (12.9 lux). The birds exposed to 12 hours light (204.5 lux): 12 hours darkness were found to have significantly greater amounts of ether extract than did the other two treatment groups. The difference between this group and the controls, however, was not significant.

A similar trial by Deaton et al. (1978b) compared percent ether extract of birds raised under either 12 hours light (237 lux): 12 hours light (7.5 lux) or continuous light (75 lux) for the first 9 days of life followed by a lighting program of 15 minutes light (7.5 lux): 105 minutes of darkness. Percent body ether extract was found to be unaffected by lighting program.

Similarly Beane et al. (1979) investigated the effects of intermittent light on various broiler performance criteria. All birds received continuous light (50 lux) for the first week of life after which four treatment groups were formed. Groups were designed as follows:

continuous light; 1 hour light: 2 hours darkness; 15 minutes light: 1 hour 45 minutes darkness; and an IL schedule with changing (weekly) light:dark periods such that initially (8-14 days) this group received 2 hours light: 1 hour darkness and by the last week the schedule was 15 min light: 1 hour 45 minutes darkness. The greatest body weights were produced by the 1 hour light: 2 hours darkness program. The authors also stated that, in general, the IL reared birds were heavier and more feed efficient. With regard to abdominal fat production, the IL groups tended towards increased deposition, especially in females.

Malone et al. (1980b) similarly found an increase in abdominal fat production in birds raised under intermittent lighting programs. In their trial, 3 different schedules were used: a control group given continuous lighting; a second group exposed to a program of 15 minutes light: 45 minutes darkness; and a third group exposed to a schedule of 2 hours light: 4 hours darkness. Both IL regimes were initiated at one week of age. At 54 days of age, significant differences in abdominal fat were found between males raised on the 15:45 program and the control group in that the IL birds produced more abdominal fat. No differences were found between males on the other IL program and the controls. Females did not show any differences in abdominal fat regardless of lighting program.

Contrary to the reports of Deaton et al. (1976), Beane et al. (1979) and Malone et al. (1980b), Malone et al. (1980a) reported evidence of decreased abdominal fat in IL reared birds. All birds were exposed to continuous light for the first 10 days of life. At this time, half of the birds were switched to an IL program of 15 minutes light: 45 minutes

darkness while the other half remained under continuous light. The IL schedule used was the same as one used in a previous report by these authors (Malone et al. 1980b). The ration fed was also the same as a diet employed in the previous trial. Both males and females were found to have consistently less abdominal fat when reared under the IL schedule, although this difference was only significant in the females.

The results of Cave (1980) support the theory that IL programs may reduce fat deposition in broilers. Performance and leaf fat content were compared between birds raised on continuous light and birds raised on continuous light for three weeks and then switched to an IL program of 1 hour light: 3 hours darkness. This program was similar to the 15 minutes light: 45 minutes darkness used by Malone et al. (1980a, 1980b) in that the ratio of light:darkness was the same (1/4 light:3/4 darkness). The absolute time differs, yet total amount of light per day (6 hours/24 hours) was the same. Body weights at 48 and 55 days were not significantly different although feed:gain ratios were better under the IL schedule. The IL regime was also found to significantly reduce the production of abdominal leaf fat.

It appears that, although there is conflicting data, the fat content of broilers can be manipulated by lighting programs. The most desirable IL program which would maintain good body weight, improve feed:gain ratio and lower fat content is yet to be consistently demonstrated.

G. Age

Although Kubena et al. (1974a) found no significant differences in the percentage of abdominal fat attributable to the age of broiler chickens, most authors have reported increases in fat content as the birds mature

(Coombs 1968; Edwards 1971).

Kubena et al. (1972) and Evans et al. (1976) found that carcass fat increased with age, beginning at one week and continuing until six weeks of age. Edwards et al. (1973) sampled broilers at 4 week intervals (from 0-20 weeks of age) and found that there was a significant decrease in moisture content and an increase in fat content with advancing age.

Bartov et al. (1974a) found that a marked increase in the fat content of broilers occurred between 7 and 9 weeks of age, especially in females. Similarly, Deaton et al. (1974) reported that percent fat content was greater in 8 week old broilers than in 7 week old birds. Griffiths et al. (1977b) found that age influenced carcass fat as the fat percent of broilers sampled at 4 weeks bore little relationship to fat levels determined at 8 weeks of age.

That abdominal fat pad size increases with age has been further demonstrated by Pfaff and Austic (1976) and Hood (1982). Increased fat pad size was shown to be as a result of hyperplasia which occurred until 15 weeks of age (Pfaff and Austic 1976) and as a result of hypertrophy.

H. Nutrient Restriction

Attempts to manipulate carcass composition (abdominal fat) by various means of nutrient restriction have produced variable results (Nelson 1980). The development of adipose tissue is a result of hyperplastic activity as well as cellular hypertrophy so a reduction in abdominal fat pad size could be accomplished by decreasing the number and/or size of the fat cells.

To be useful and effective, nutrient restriction programs should

produce a bird that is leaner without adversely affecting body weights and other economically important factors. For example, March and Hansen (1977) reduced abdominal fat in broilers via diet dilution (reduction of the energy of the ration) and also by starving the birds for up to five days post hatch. These methods, however, could not be considered practical as mortality was unacceptable and body weights were markedly lower than the control group.

Pfaff and Austic (1976) found that feeding a low energy or high protein diet to Leghorn pullets can slow hyperplastic growth but did not reduce the total number of fat cells at maturity. Similarly, hypertrophic growth was also depressed by low energy or high protein diets. Hood (1982) reported that growth of the fat pad in broilers occurred as a result of hyperplasia and hypertrophy until 14 weeks of age at which time cell numbers plateaued and hypertrophy was the sole means of further size increases.

Griffiths et al. (1977a) investigated the effect of early life caloric restriction on the productive performance and abdominal fat pad size of broiler chickens. Two different starter diets were formulated; a control containing 3,087 kcal ME/kg:24% protein and a low energy ration containing 2,233 kcal ME/kg:20% protein. Treatments consisted of a control group receiving the higher energy starter from 0-4 weeks of age and 3 groups receiving the low energy diet for 1, 2 and 3 weeks respectively, followed by the control ration until 4 weeks of age. All groups were fed the same finishing diet from 4-8 weeks of age at which time fat pad weights were determined.

Analysis of body weight data (at 3 weeks of age) revealed that the

only significant differences in weight gain was between the control group (heaviest) and the group fed the low energy ration from 0-3 weeks of age. By 4 weeks however, there were no significant differences in body weights among any of the treatments. Analysis of 4-8 week data showed no significant differences in weight gain, feed intake and feed:gain ratio. Abdominal fat pad measurements also revealed no differences among treatment groups. The authors concluded that the failure to reduce abdominal fat pad size may have been a result of the caloric restricted regimes not being of sufficient magnitude to retard adipocyte hyperplasia, or if cell numbers were reduced, the effect was nullified by cell hypertrophy when the birds were offered the high energy starter ration. An alternate suggestion was that early caloric restriction may not have any effect on avian adipocyte development. However, according to the author, the most likely reason for the failure to reduce fat pad size was that the degree of caloric restriction was not severe enough to affect hyperplasia; the fact that body weights were not reduced provided evidence for this.

Using a feed intake nutrient restriction approach, Beane et al. (1979) suggested that abdominal fat pad development could be influenced by feeding program. Nutrient restriction was accomplished by limiting the feed intake of treatment groups to 85% of a control group from 14 to 42 days of age. From 0-13 and from 43-56 days of age, feed was supplied ad libitum. Abdominal fat pads were removed and weighed at 56 days of age. Although the restricted group showed compensatory growth from 42-56 days, final body weights were significantly lower than the controls.

Contrary to the results of Griffiths et al. (1977a), restricted males

were found to have significantly larger fat pads than the full fed groups. The authors suggested that because nutrient restriction does not permanently reduce adipocyte number (Pfaff and Austic 1976), dietary influences on fat deposition would be of short duration. The feed restriction period of 4 weeks may have stimulated hyperphagia during the ad libitum feeding period to the extent that abdominal fat deposition was markedly increased. The authors also concluded that differences in the results of nutrient restriction studies could be due to differences in rations, management practices and/or genetic factors.

That genetic factors may play a role in the response of broilers to nutrient restriction has been demonstrated by Cherry et al. (1978) who found that a low nutrient density starter ration could, depending on the population, increase, decrease or have no effect on abdominal fat pad size. The authors suggested that feeding a low energy starter ration may offer some potential in reducing abdominal fat pad growth (depending on the population), most likely by delaying hypertrophy of the adipocyte.

Further experimentation with nutrient restriction was carried out by Hargis and Creger (1980). Caloric restriction was achieved by formulating starter rations that contained no added fat. Three treatment groups, plus a control group, were set up such that the fat restricted starter diet was fed for 7, 10 and 14 days respectively. After the initial starter diets, groups were offered the control ration until 4 weeks of age. Two finishing feeds were formulated, one containing 2,860 kcal ME/kg:30% protein, the other having 3,080 kcal ME/kg:30% protein. The two treatment groups that had been fat restricted for 7 and 14 days respectively, were divided such that each received both finishing

diets. A group that had been fat restricted for 10 days received only the 3,080/30 finisher ration. The control group was fed a finisher formulated to contain 3,450 kcal ME/kg:19% protein. Abdominal fat pad weights were recorded at 49 days of age.

The results showed that energy level of the finishing diet had no effect on fat pad size in the group that had been fat restricted for 7 days. This was not the case, however, in the group restricted for 14 days in that birds fed the 2,860/30 finisher had significantly less abdominal fat than birds fed the 3,080/30 finisher. When compared to the controls, all other treatment groups, with the exception of the birds restricted for 14 days and fed the 3,080/30 finisher, had significantly less abdominal fat. The treatment groups with the least amount of fat (1.23%) were those that had been fat restricted for 7 days; C:P ratio of the finisher had no effect. The control group averaged 2.62% abdominal fat while the groups restricted for 10 days or 14 days fed the 2,860/30 finisher yielded values of 1.67 and 1.62% respectively.

The authors concluded that although the optimum combination is still uncertain, the length of time a low fat starter is fed and the C:P ratio of the diet can exert a combined influence on fat pad size. They further stated that abdominal fat could be significantly reduced by not adding fat to broiler rations for the first 7 days of life. This period of fat restriction combined with a 30% protein finisher produced the greatest reduction in fat pad weights. The results of this trial are of practical significance in light of the fact that, although slightly lighter than the controls, body weights of the treatment groups were acceptable.

As the results of Cherry et al. (1978) and Hargis and Creger (1980) indicated, nutrient restriction has some potential for reducing the fat content of broilers. Although significant reductions of abdominal fat were not obtained, Griffiths et al. (1977a) also suggested that nutrient restriction offers possibilities for reducing abdominal fat deposition. Further research to determine optimum methods is required.

I. Additional Factors

Other factors affecting the fat content of broilers include cage vs floor rearing, corticosterone levels and salt consumption.

Deaton et al. (1974) reported that percent ether extracts and abdominal fat pad percent of broilers raised in cages were significantly higher than in broilers raised on the floor and attributed differences to lesser activity for birds in cages.

Bartov (1982) injected 3 and 7 week old broilers 6 times daily (680 µg/kg body weight per day) with corticosterone and found that body weight was decreased and fat pad weight increased. The increased amount of abdominal fat produced as a result of corticosterone injection at 3 weeks of age was not evident at 6 weeks of age.

Maurice and Deodato (1982) found that adding NaCl at the rate of 50-100 mM to the drinking water of broilers from 5-7 weeks of age significantly reduced abdominal fat measured at 7 weeks of age. Body weights were not affected by the salt water.

MATERIALS AND METHODS

I. Outline of Experiments

A. General

Six experiments were designed to study the incidence, possible causes, and methods of reducing the incidence of SDS in broiler chickens. Also, the problem of excess abdominal fat deposition was investigated in 2 of the 6 experiments.

All trials employed day old, vaccinated (Marek's) broiler chicks of commercial (Cobb x Cobb) parentage. With the exception of Experiment 1, (in which half of the birds were females) all trials used male birds. In Experiments 1, 2, 3, 5 and 6, birds were raised from day old in 1.54m x 4.31m floor pens with a stocking rate of $0.09m^2$ per bird (70 birds/pen). All pens were located in an environmentally controlled barn that provided continuous (24 hour) lighting at an intensity of about 5 lux at feeder level. Temperature was regulated such that birds were exposed to 35^oC temperatures for the first week, using a small canopy brooder with heat lamps. Thereafter, temperatures were lowered by approximately 3^oC per week by raising the brooder, removing one or two of the three bulbs and eventually removing the brooder until the temperature reached 21^oC. From 0-7 days, each pen was provided with a floor type trough feeder. From 7 days until the end of the trial, feed was available from tube type feeders (2 per pen, 40 cm diameter). Water was available from one 15 cm diameter cup waterer.

In Experiment 4, birds were raised in electrically heated Jamesway battery brooders. Each battery contained 10 individual wire floor pens

equipped with one trough waterer and two trough feeders as well as an overhead heat source. Temperature and lighting schedules were similar to those of birds raised in floor pens.

All rations were in mash form and, with the exception of Experiment 1 where restricted feeding was used, fed ad libitum. In all experiments, water was available ad libitum. With the exceptions of Experiments 1 and 2, which contained some modifications, all rations contained the same vitamin and mineral premixes, added at 1.00% and 0.50% of the diet respectively. The vitamin and mineral premixes are presented in Tables 1a and 1b respectively.

Daily management procedures included cleaning wet spots, stirring litter, shaking down feeders, dusting equipment and culling birds. Mortalities occurring during the first 3 days of the trials were replaced by spare birds. Subsequent mortalities were recorded by pen number, treatment number, wingband number and dead weight. All mortalities, with the exception of those from Experiment 4, were submitted for necropsy to the poultry pathology laboratory, Manitoba Department of Agriculture, and cause of death recorded.

In all experiments, average body weights for each pen were calculated by dividing the total weight of the live birds by the total number of live birds at the time of weighing. That is:

$$\frac{\text{Total live per wt.}}{\text{Total no. of live birds}} = \text{average body wt./bird}$$

Average pen gain was calculated by subtracting the average weight of live birds from the previous period (initial wt.) from the average

Table 1a. Standard vitamin premix

Ingredients	g/kg Premix
Vitamin A (500,000 I.U./g)	1.65
Vitamin D_3 (90 million I.C.U./lb)	0.44
Vitamin E (20,000 I.U./lb)	12.38
Vitamin B_{12} (60 mg/lb)	8.36
Menadione	0.11
Santoquin	25.00
DL-Methionine	50.00
Pen-strep	100.00
Amprol	50.00
Vitamin B_{58} (2-4-6-100)[1]	125.00
Animal tallow	10.00
Wheat middlings	617.06

[1]Vitamin B_{58} contains per kg:

Riboblavin	4,400 mg
Ca Pantothenate	8,800 mg
Niacin	13,200 mg
Choline chloride	220,000 mg

Table 1b. Standard mineral premix

Ingredients	g/kg Premix
Manganese oxide	33.30
Zinc oxide	2.90
Ferrous sulfate $\cdot 7H_2O$	6.20
Copper sulfate $\cdot 5H_2O$	5.10
Iodized salt	952.50

weight of live birds at the time of weighing (present wt). That is:

Average gain = Average present wt - Average initial wt

Feed:gain ratios were calculated by dividing total feed consumed per pen during the period of concern by the total weight gain of birds within a pen, including mortalities. That is, total weight gain was calculated by subtracting the initial pen weight from the present weight plus the weight of mortalities that occurred within the particular period. Therefore:

$$\text{Feed:gain} = \frac{\text{feed consumption}}{\text{total pen wt gain}}$$

All statistical analysis described (with the exception of Tukey's Test) were carried out using programs available on a Commodore PET 2001 computer. Mortality, dressing % and fat pad data were converted by arc-sin transformation before being analyzed according to the design of the particular experiment. However, for interpretation the data were reported as percentages.

B. Experiment 1. Effect of feed restriction on abdominal fat pad production of broiler chickens.

Experiment 1 was designed to determine if abdominal fat pad size could be reduced by limiting the energy consumption of broilers by the use of a restricted intake feeding program. Also, this trial compared wheat and corn as grain sources as well as the effects of additional vitamins and minerals on the incidence of Sudden Death Syndrome (SDS).

Sixteen hundred eighty (1680) Cobb color-sexed, commercial type

broiler chicks were randomly distributed among 24 floor pens; 12 pens of males and 12 pens of females. Three pens of males and 3 pens of females (total of 6 pens/420 birds) were randomly assigned to 1 of 4 dietary treatments. The 4 treatment groups were as follows: (1) Control ration - wheat-soy diet, fed ad libitum. (2) Control ration - pair fed to 90% intake of group (1). (3) Control ration plus additional vitamins and minerals, fed ad libitum. (4) Corn-soy ration - fed ad libitum. Starter and finisher ration formulations are listed in Table 2 and Table 3 respectively. Vitamin and mineral premixes are presented in Table 4a and Table 4b respectively.

Starter rations were fed from 0-5 weeks of age and finisher rations fed from 5-7 weeks of age. For the first 7 days of life, all treatments were fed ad libitum, however from day 8, treatment 2 was pair fed 90% of the control group's intake. That is, the restricted group was fed according to the previous days intake of the control group.

The following data was recorded: body weights at 0, 1, 5 and 7 weeks of age. Feed consumption and weight gains were recorded and feed: gain ratios calculated for the following periods: 0-1, 1-5, 5-7 and 0-7 weeks.

At 7 weeks of age, 4 birds per pen (24/treatment) were starved overnight and slaughtered for fat pad analysis. The tissue removed and thus considered to be the abdominal fat pad was the fatty tissue surrounding the gizzard and intestines and extending around the Bursa of Fabricius. Live weights at time of slaughter, carcass weights and fat pad weights were recorded and abdominal fat pad percents (as a percentage of carcass weight) determined. Dressing percents (carcass wt/live

Table 2. Starter rations - Experiment 1

Ingredients	Diet I (Control wheat-soy)	Diet II (Fortified wheat-soy)	Diet III (Corn-soy)
	(g/kg)		
Ground corn	-	-	549.0
Ground wheat	617.8	610.9	-
Soybean meal (47.5%)	250.0	251.9	329.0
Fish meal (65%)	48.0	48.0	40.0
Tallow	42.0	42.0	40.0
Biophos	14.0	14.0	14.0
Limestone	13.0	13.0	13.0
Vitamin premix (standard)	-	-	10.0[1]
Vitamin premix (V-3)	10.0	15.0	-
Mineral premix (standard)	5.0	-	5.0
Mineral premix (fortified)	-	5.0	-
DL-methionine	0.5	0.5	-
Amprol	0.5	0.5	-
Pen-strep	1.0	1.0	-
	1000.0	1000.0	1000.0
Calculated Analysis: (Chemical Analysis)			
ME Kcal/kg	3075	3075	3075
C.P. (%)	22.96(23.65)	22.96(23.86)	22.94(23.67)
Ca (%)	1.09(1.39)	1.09(1.20)	1.06(1.18)
P (%)	0.76(0.94)	0.76(0.98)	0.72(0.90)
Methionine (%)	0.40	0.40	0.42
Lysine (%)	1.32	1.32	1.41
Methionine + cystine (%)	0.79	0.79	0.78

[1]Standard vitamin premix supplied DL-methionine, amprol and pen-strep at the same level as supplemented in Diets I and II.

Table 3. Finisher rations - Experiment 1

Ingredients	Diet I (Control wheat-soy)	Diet II (Fortified wheat-soy)	Diet III (Corn-soy)
	(g/kg)		
Ground corn	-	-	642.2
Ground wheat	695.8	690.8	-
Soybean meal (47.5%)	187.0	187.0	262.8
Fish meal (65%)	34.0	34.0	34.0
Tallow	45.0	45.0	23.0
Biophos	12.0	12.0	12.0
Limestone	11.0	11.0	11.0
Vitamin premix (standard)	-	-	10.0[1]
Vitamin premix (V-3)	10.0	15.0	-
Mineral premix (standard)	5.0	-	5.0
Mineral premix (fortified)	-	5.0	-
DL-methionine	0.5	0.5	-
Amprol	0.5	0.5	-
Pen-strep	1.0	1.0	-
	1000.0	1000.0	1000.0
Calculated Analysis:			
ME Kcal/kg	3081	3081	3081
C.P. (%)	20.10	20.10	20.10
Ca (%)	0.90	0.90	0.90
P (%)	0.68	0.68	0.65
Methionine (%)	0.34	0.34	0.39
Lysine (%)	1.07	1.07	1.19
Methionine + cystine	0.70	0.70	0.69

[1]Standard vitamin premix supplied DL-methionine, amprol and pen-strep at the same level as supplemented in Diets I and II.

Table 4a. Vitamin premix V-3 (no coccidiostat, antibiotic or methionine)

Ingredients	g/kg Premix
Vitamin A (500,000 I.U./g)	1.66
Vitamin D_3 (90 million I.C.U./lb)	0.44
Vitamin E (20,000 I.U./lb)	12.45
Vitamin B_{12} (60 mg/lb)	8.41
Menadione (Na bisulfite)	0.11
Santoquin	25.00
Vitamin B_{58} (2-4-6-100)[1]	125.00
Animal tallow	10.00
Wheat middlings	816.93

[1]Vitamin B_{58} - components are listed in Table 1a.

Table 4b. Mineral premix (fortified - 150%)

Ingredients	g/kg Premix
Manganese oxide	50.00
Zinc oxide	4.50
Ferrous sulfate $\cdot 7H_2O$	9.50
Copper, sulfate $\cdot 5H_2O$	7.75
Iodized salt	928.25

wt) were also calculated.

All data were analyzed as a 2x4 factorial arrangement of a completely randomized design. Differences between means were determined by Tukey's Test (Snedecor and Cochran 1980).

C. Experiment 2. The effects of compensatory growth and vitamin levels of broiler diets on the incidence of Sudden Death Syndrome (SDS).

This experiment was designed to examine two factors that have been associated with SDS, that is the rate of growth of the flock and the levels and types of vitamins added to broiler diets.

A total of eighteen hundred twenty (1820) day old broiler chicks were randomly distributed among 26 floor pens. Six pens were randomly assigned to each dietary treatment (420 birds/treatment), with the exception of the control treatment which consisted of 2 pens (140 birds) due to a lack of available pen space.

The control group (T_1) was fed a standard starter ration from 0-5 weeks of age followed by a standard finishing ration fed from 5-7 weeks. Ration formulations are presented in Table 5. All other (4) treatment groups were fed a low protein ration that contained 50% of the standard vitamin premix (Table 1a) from 0-14 days of age so that an initial period of growth restriction would occur. From 14-35 days of age these groups were switched to a high protein ration with the vitamin levels adjusted according to treatment, that is T_2 received the standard vitamin premix (10 g/kg of diet), T_3 received 50% of the standard premix, T_4 received the standard premix plus 3x the level of B vitamins found in the premix.

Table 5. Diet formulations - Experiment 2

Ingredients	Control starter	Restricted starter	High-protein starter	Finisher
	(g/kg)			
Ground wheat	632.0	777.7	505.0	724.6
Soybean meal (47.5%)	264.8	153.8	353.0	193.1
Fish meal (66%)	22.2	8.4	42.0	12.4
Animal tallow	40.0	21.1	57.0	28.9
Vitamin premix	10.0	5.0	10.0[1]	10.0
Mineral premix	5.0	5.0	5.0	5.0
Limestone	15.0	12.0	12.0	11.0
Biophos	10.0	16.0	16.0	15.0
DL-methionine	1.0	1.0	-	-
	1000.0	1000.0	1000.0	1000.0
Calculated analysis:				
ME Kcal/kg	3000	3000	3000	3000
C.P. (%)	22.20	18.00	26.00	19.35
Ca (%)	0.98	0.91	1.10	0.87
P (%)	0.64	0.70	0.79	0.70
Methionine (%)	0.49	0.42	0.48	0.35
Lysine (%)	1.22	0.85	1.57	0.98
Methionine + cystine (%)	0.87	0.74	0.91	0.79

[1]The amount of vitamin premix added to the ration for treatment 3 was 5g. The additional vitamins added to treatments 4 and 5 are listed in Table 6.

In addition, this group received 3x the NRC requirements for biotin, thiamin and pyridoxine. These 3 vitamins are not normally added to broiler rations. T_5 received the standard premix plus 2x the levels of vitamins A, D and E contained in the standard premix. The amounts of additional vitamins given T_4 and T_5 are listed in Table 6. All treatments were fed the standard finishing ration given to the control group from 5-7 weeks of age.

The following data was recorded: group weights at 2 and 5 weeks of age. At 7 weeks, individual bird weights were determined. Feed consumption and weight gains were recorded and feed:gain ratios calculated for the following periods: 0-2, 0-5 and 0-7 weeks. All mortalities were recorded and submitted for necropsy.

Performance and mortality data were analyzed as a completely ransomized design using a one way analysis of variance. Differences between means were determined by Tukey's Test (Snedecor and Cochran 1980).

D. Experiment 3. The effects of early life caloric (fat) restriction and calorie:protein ratios on abdominal fat pad production and the incidence of Sudden Death Syndrome (SDS) in broiler chickens.

Experiment 3 was designed to examine two nutritional aspects of abdominal fat pad deposition in broilers, that is, if the deletion of added dietary fat (as animal tallow) from the starter ration for the first 7 days of life alone or in combination with lowered C:P ratios in the finishing diet would reduce the size of the abdominal fat pad. In conjunction, the possibility that the degree of fatness of broilers

Table 6. Vitamins added to the standard premix - Experiment 2

Treatment	Vitamin	g/kg Premix
4	Biotin	0.045
	Thiamin	0.540
	Pyridoxine	0.870
	Choline	55.000
	Niacin	3.300
	Ca pantothenate	2.200
	Riboflavin	1.100
5	A (500,000 I.U./g)	1.650
	D_3 (90 million I.C.U./lb)	0.440
	E (20,000 I.U./lb)	12.370

could be related to the incidence of SDS was investigated.

Nineteen hundred sixty (1960) day old broiler chicks were randomly distributed among 28 floor pens. Seven pens were randomly assigned to each dietary treatment (4 treatments; 490 birds/treatment). The 4 treatment groups were as follows: (1) control diet - standard starter I/ finisher I regime, (2) no added fat 0-7 days (starter II) followed by the control starter/finisher diets, (3) no added fat 0-7 days (starter II) followed by the control starter/high protein finisher II, (4) control starter/high protein finisher II. Ration formulations are listed in Table 7.

The initial caloric restriction was accomplished by not adding fat to the starter diet (II) and feeding this ration from 0-7 days. Differences in the C:P ratio of the 2 finishing diets were obtained by maintaining a high protein percentage (24%) in finisher II as opposed to the lower (19%) protein level in finisher I.

The experimental period with regard to abdominal fat deposition was 49 days whereas SDS data was recorded until 56 days of age. All birds were switched from the starter to the finishing diets at 28 days of age.

The following data was recorded: group weights at 0, 1, 2, 3 and 4 weeks of age. At 7 weeks, individual bird weights were determined. Feed consumption and weight gains were recorded for the following periods: 0-1, 1-2, 2-3, 3-4 and 4-7 weeks. Feed:gain ratios were calculated for each period listed as well as from 0-4 and 0-7 weeks. Mortality data was recorded and birds submitted for necropsy.

At 7 weeks of age, 3 birds per pen (21 birds/treatment) were

Table 7. Diet formulations - Experiment 3

Ingredients	Starter I	Starter II	Finisher I	Finisher II
	(g/kg)			
Ground wheat	568.0	621.0	704.6	549.4
Soybean meal (47.5%)	305.9	336.7	175.2	294.9
Fish meal (66%)	33.2	-	21.8	41.8
Animal tallow	55.4	-	54.4	72.9
Vitamin premix	10.0	10.0	10.0	10.0
Mineral premix	5.0	5.0	5.0	5.0
Limestone	11.0	12.0	12.0	13.0
Biophos	11.0	14.8	16.0	13.0
DL-methionine	0.5	0.5	1.0	-
	1000.0	1000.0	1000.0	1000.0
Calculated Analysis:	(Chemical Analysis)			
ME Kcal/kg	3011	2770	3100	3097
C.P. (%)	24.0(27.42)	24.0(28.12)	19.0(22.69)	24.0(27.90)
Ca (%)	0.90(1.42)	0.91(1.41)	0.86(1.50)	0.90(1.67)
P (%)	0.69(0.94)	0.71(1.02)	0.70(0.95)	0.70(1.00)
Methionine (%)	0.49	0.46	0.45	0.45
Lysine (%)	1.36	1.32	0.96	1.40
Methionine + cystine (%)	0.88	0.87	0.78	0.84

selected and sacrificed for determination of abdominal fat weight and dressing percent. Birds were selected such that 7 (1/3 of the total no. selected per treatment) were of average weight, 7 were below average weight (by approximately 10%) and 7 were above average weight (by approximately 10%). All selected birds were starved overnight before being sacrificed. The tissue removed and thus considered to be the abdominal fat pad was the same as described for Experiment 1. Live weights at the time of slaughter, carcass weights and fat pad weights were recorded.

Before birds were sacrificed, caliper measurements were taken to determine if this instrument could be used to predict abdominal fat pad size in a live bird. The calipers were manufactured by the Central Instrument Service of the University of Manitoba, based on specifications provided by Pym and Thompson (1980). A diagram listing the features of the calipers is provided in Figure 2. The technique for using the calipers was the same as that described by Pym and Thompson (1980). The 3 caliper readings obtained were averaged to yield one value.

From 0-4 weeks, average body weights, weight gains, feed:gain ratios and mortalities were analyzed as a completely randomized design using a one way analysis of variance (i.e. restricted vs nonrestricted). Mortality data was considered as 2 treatments whereas the other data listed were considered as 4 treatments. Data from 4-7 and 0-7 weeks were analyzed as a 2x2 factorial arrangement of a completely randomized design.

Simple regressions were carried out to determine possible relationships between fat pad percent and carcass weight and also to determine possible relationships between average caliper reading and actual fat

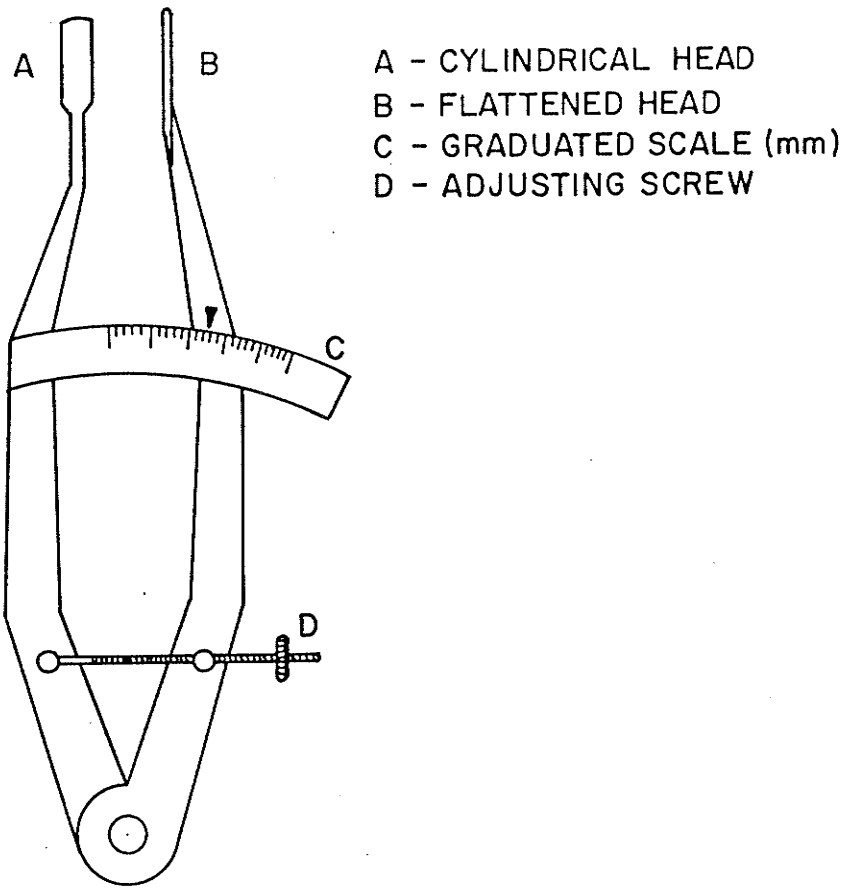

Figure 2. Scale drawing of calipers. 40% actual size.

pad weight.

Differences between means were determined by Tukey's Test (Snedecor and Cochran 1980).

E. Experiment 4. The effects of high doses of niacin in broiler diets on total serum lipids, triglycerides and cholesterol.

Experiment 4 was designed to determine if incorporating high levels of niacin (nicotinic acid) in broiler diets would lower total serum lipids, triglycerides and/or cholesterol. The effects of niacin on growth rate and feed:gain ratios were also studied.

A total of two hundred eighty (280) day old broiler chicks were randomly distributed among 3 Jamesway battery brooders. Each battery contained 10 wire floor pens, each pen capable of holding 10 birds. Seven pens were randomly assigned to each of the 4 treatment groups (4 treatments; 70 birds/treatment). The treatment groups were as follows: (1) control diet - no added niacin, (2) control diet plus 50x NRC requirement for niacin, (3) control diet plus 150x NRC requirement for niacin, (4) control diet plus 300x NRC requirement for niacin.

The control ration was the same formulation as used in Experiment 3 (Table 7, Starter I) calculated to supply 3011 Kcal ME/kg and 24% protein. Niacin was supplied as a powder (Dominion Veterinary Laboratories, Winnipeg, Manitoba) and pre-mixed with the fish meal used in the ration. The experimental period lasted until the birds were 28 days of age.

Determination of the niacin dosage was based on stated requirements of niacin for man and the chick. The recommended dietary allowance (RDA)

of niacin for young males was given as 20 mg/day (8th review edition, Food and Nutrition Board, NRC 1974) while the NRC requirement of niacin for the chick is listed as 27 mg/kg of diet (Nutrient Requirements of Poultry, 7th revised edition 1977).

In man, pharmacological doses of niacin range from 3 g to 15 g per day (Carlson 1977). A mid range dose for humans of 6 grams per day was selected as a guideline for economic reasons and also because of possible harmful side effects of larger doses. Six grams per day in humans is equivalent to 300x the RDA requirement. Therefore, the niacin requirement of the chick was multiplied by 300 to give an estimate of an equivalent dose of 8.1 g/kg of feed (300 x 27 mg/kg). Two other lower doses were also tested to determine an estimate of dose levels required to effect blood lipid parameters.

The following data was recorded: group pen weights at 0, 2 and 4 weeks of age. Feed consumption and weight gains were recorded and feed:gain ratios calculated for the following periods: 0-2, 2-4 and 0-4 weeks. Mortalities were recorded as they occurred.

At 4 weeks of age, one bird per pen was selected at random for blood sampling. All birds sampled were starved for 2 hours prior to sampling. Five c.c. of blood was taken from each bird via cardiac puncture using a 5 c.c. disposable syringe with an 18 gauge, 1.5 inch (3.75 cm) needle. The blood was then transferred to a 5 c.c. non heparanized vacutainer tube.

1) Blood Analysis Procedure

a) Sample preparation

Blood samples were centrifuged at 1,500 r.p.m. for 15-20 minutes in an Adams table top centrifuge. The serum layer was withdrawn by pipette and stored frozen (-20°C) in 2 separate 5 ml disposable culture tubes, each containing approximately equal amounts.

b) Chemical analysis

Total serum lipids were determined using the method of Frings and Dunn (1970). Serum triglycerides and cholesterol concentrations were determined using the method of Moses et al. (1975). All analyses were run in duplicate.

Average body weights, weight gains, feed:gain ratios, total serum lipids, cholesterol and triglyceride values were analyzed as a completely randomized design using a one way analysis of variance. Differences between means were determined by Tukey's Test (Snedecor and Cochran 1980).

F. Experiment 5. The effects of niacin and calorie:protein ratios on the incidence of Sudden Death Syndrome (SDS).

Experiment 5 was designed to determine if high levels of dietary niacin alone or in combination with a high protein diet would affect the incidence of SDS.

Nineteen hundred sixty (1960) day old broilers were randomly distributed among 28 floor pens. Seven pens were randomly assigned to each of the 4 treatment groups (490 birds/treatment). The 4 groups were

as follows: (1) standard starter/finisher diets, (2) standard starter/high protein finisher, (3) standard starter plus 150x NRC niacin requirement/standard finisher plus 150x NRC niacin requirment, (4) standard starter plus 150x NRC niacin requirement/high protein finisher plus 150x NRC niacin requirement. The dietary formulations for the starter and finisher diets were those used in Experiment 3 (Table 7). Niacin was added to the diet at the expense of wheat at the rate of 4.1 g/kg of feed (0.41%). Mixing was accomplished as described in Experiment 4.

The experimental period lasted 49 days and the following data was recorded: group weights at 4 weeks of age and individual weights at 7 weeks of age. Feed consumption and weight gains were recorded and feed:gain ratios calculated for the periods of 0-4, 4-7 and 0-7 weeks. All mortalities were recorded and submitted for necropsy.

At 4 and 7 weeks of age, one bird per pen (7 birds/treatment) was randomly selected and 5 c.c. of blood collected via cardiac puncture as described in Experiment 4. The samples were transferred into 10 c.c. heparanized Vacutainer tubes and then packed in ice before they were transported to be centrifuged.

The analysis procedures for sample preparation, storage and determination of total lipids, triglycerides and cholesterol were those described in Experiment 4. In this trial, however, plasma was used to minimize contamination of the samples by red blood cells during centrifuging. The analyses used are effective for serum or plasma.

From 0-4 weeks, average body weights, weight gains, feed:gain ratios,

total lipids, triglycerides, cholesterol and mortalities were analyzed as a completely randomized design using a one way analysis of variance (i.e. niacin vs no added niacin). Performance data from 0-4 weeks was considered as 4 treatment groups whereas mortalities and blood parameters were considered as 2 treatment groups. All data from 4-7 and 0-7 weeks was analyzed as a 2x2 factorial arrangement of a completely randomized design. Differences between means were determined by Tukey's Test (Snedecor and Cochran 1980).

G. Experiment 6. The effects of sodium and potassium levels of broiler diets on the incidence of SDS.

Experiment 6 was designed to determine if sodium and/or potassium levels in the diet would influence the incidence of SDS in broilers.

Twelve hundred sixty (1260) day old broilers were randomly distributed among 18 floor pens. Six pens were randomly assigned to 1 of 3 dietary treatments (420 birds/treatment). The 3 treatment groups were as follows: (1) control diet, (2) control diet plus 1.3 x Na content of the control, (3) control diet plus 1.3 x K content of the control.

The control diet was the same formulation as the starter/finisher rations used in Experiment 3 (Table 2, Starter I/Finisher I). As Mongin and Sauveur (1977) found that the acid-base balance (calculated as $Na_{meq} + K_{meq} - Cl_{meq}$) could affect growth rates, sodium and potassium were added as chlorides so that the net acid base balance would be the same among all treatment groups. Both compounds were added in powder form as part of the mineral premix.

The amounts of NaCl or KCl added to the diets was determined by

first calculating the amounts of Na and K found in the starter and finisher rations to be used. Sodium and potassium percentages for each ingredient in the ration were obtained from NRC tables (Nutrient Requirements of Poultry, 7th revised edition 1977). The total amounts of Na and K were thus determined. The calculated value was then increased in the ration by 30% by the addition of NaCl or KCl. Calculated and determined Na, K and Cl values for the starter and finisher diets are listed in Tables 8 and 9 respectively. As percentages of the diets, NaCl was added at 0.23% (starter) and 0.21% (finisher) while potassium was added to the starter and finisher diets at levels of 0.51% and 0.40% respectively.

The experimental period was 49 days with birds being switched from the starter to the finisher ration at 28 days of age. The following data was recorded: group weights at 4 weeks and individual bird weights at 7 weeks of age. Feed consumption and weight gain were recorded and feed:gain ratio calculated from 0-4, 4-7 and 0-7 weeks of age. From approximately the beginning of the 4th week until the end of the 6th week, the hearts of SDS birds and the hearts of other mortalities (including culls) were removed at the time of post-mortem for the determination of Ca, K, Na and Cu levels.

1. Heart Analysis

a) Sample preparation

After the hearts were removed, they were placed in labelled plastic bags and stored frozen ($-20^{o}C$). Prior to chemical analysis, all hearts were lyophilized for approximately 36 hours in a Virtis 10-324 freeze-dryer. When completely dried, all samples were ground in a Janke

Table 8. Calculated and determined Na, K and Cl levels of starter diets - Experiment 6

Treatment		Na (g/kg)	Na (meq/kg)	K (g/kg)	K (meq/kg)	Cl (g/kg)	Cl (meq/kg)	(Na + K) - Cl (meq/kg)
1	Calculated	2.97	129	8.82	226	3.44	97	258
	Determined	2.50	109	10.80	277	4.70	132	254
2	Calculated	3.87	168	8.82	226	4.84	136	258
	Determined	5.30	230	10.70	274	9.20	259	245
3	Calculated	2.97	129	11.47	294	5.86	165	258
	Determined	4.40	191	13.50	346	10.80	304	233

Table 9. Calculated and determined Na, K and Cl levels of finisher diets - Experiment 6

Treatment		Na		K		Cl		(Na + K) - Cl
		(g/kg	meq/kg)	(g/kg	meq/kg)	(g/kg	meq/kg	(meq/kg)
1	Calculated	2.80	121	6.95	178	3.50	99	200
	Determined	5.40	235	8.40	215	8.20	231	219
2	Calculated	3.62	157	6.95	178	4.77	134	201
	Determined	7.20	313	7.80	200	11.80	333	180
3	Calculated	2.80	121	9.04	232	5.41	152	201
	Determined	4.7	204	11.3	290	8.6	242	252

and Kunkel A10-5 stainless steel grinder and placed into polyethylene screw top vials.

b) Chemical analysis

Heart samples were weighed into silica dishes and ashed at 550°C for 4 hours. Upon cooling, 10 ml of 5N HCl and 5 drops of concentrated HNO_3 were added to each dish. The dish was then placed on a hot plate and brought to a gentle boil for 10 minutes. After cooling, the sample was filtered into a 50 ml volumetric flask through acid-rinsed Whatman #541 filter paper and made up to volume. Determinations were done on these solutions or dilutions thereof as necessary, using an Instrumentation Laboratory model 551 atomic absorption spectrophotometer. Instrument parameters were as recommended by the manufacturer.

i) Copper

Copper standards were prepared by diluting a stock solution (1000 µg Cu/ml; Fisher Scientific, catalogue no. So-C-194) with an appropriate amount of 0.5N HCl. Standards were made up in the range of 0.0 to 1.00 µg Cu/ml and samples and standards were analyzed in the absorbance mode at 10x scale expansion.

ii) Calcium

Standards were made up in the range of 0 to 20.0 µg Ca/ml with the analysis conducted using the concentration mode. Standard solutions were prepared by adding 5 ml of a lanthanum stock solution (25 µg Ca/ml) and diluting to 25 ml by the addition of 0.5N HCl. The lanthanum stock solution was prepared by dissolving 58.65 g of La_2O_3 (99.99%) in 250 ml

HCl and then diluted with HCl to 1 litre.

iii) Potassium

Standards were made up in the range of 0.0 to 2.00 µg K/ml with the analysis conducted using the concentration mode. Standard solutions were prepared by diluting a stock solution (1000 µg K/ml; Fisher Scientific, catalogue no. So-P-351) with the appropriate amount of 0.5N HCl.

iv) Sodium

Sodium standards were prepared by dilution of a stock solution (1000 µg Na/ml; Fisher Scientific, catalogue no. So-S-139) with the appropriate amount of 0.5N HCl. Standards were made up in the range of 0 to 2.00 µg Na/ml and the analysis conducted using the concentration mode.

2. Feed Analysis

a) Sample preparation

Feed samples were ground through a 1 mm screen in a Wiley Mill and stored in plastic bags prior to chemical analysis.

b) Chemical analysis

i) Sodium and potassium

Analysis of the feed samples for Na and K was done by the same procedure as for heart analysis.

ii) Chlorine

Preparation of the sample solution was by the method for chlorine in plants, Official Methods of Analysis of the Association of Official Chemists, 13th edition (1980).

Concentration of chloride was then determined using a Fisher Accumet pH ion (Model 750) equipped with a Coleman chloride electrode (Model 3-802) and using Cl standards of 0, 10, 100 and 1000 µg Cl/ml.

All performance and mortality data were analyzed as a completely randomized design using a one way analysis of variance. Differences between means were determined using Tukey's Test (Snedecor and Cochran 1980).

A simple T-test was used to compare mean mineral contents of the hearts of SDS and cull birds.

RESULTS AND DISCUSSION

I. Effect of feed restriction on abdominal fat pad production of broiler chickens. (Experiment 1).

RESULTS

A. Mean body weights and weight gains

Mean weight gains and body weights are for the 7 week experimental period and are presented in Table 10. Mean squares from analysis of variance tables are reported in Appendix 1. Within all treatment groups and during all periods tested, females were found to gain significantly ($P<0.01$) less body weight than males. All of the following values represent the treatment average for males and females.

After the initial 7 days of the trial, during which all treatment groups were fed ad libitum, a significant ($P<0.01$) treatment effect was observed. The corn fed birds gained more weight than the wheat fed birds, averaging 7 day body weights of 136 g as compared to weights that ranged from 124 g to 129 g.

At 5 weeks of age, significant ($P<0.01$) differences in body weight persisted. The group fed 90% of the basal wheat ration had average body weights of 1,028 g as compared to 1,200 g and 1,216 g for the basal and fortified wheat rations respectively. The corn fed birds were significantly ($P<0.01$) heavier than any of the other 3 groups with average body weights of 1,275 g.

From 5-7 weeks of age only the restricted group had a significantly ($P<0.01$) greater gain than any of the other treatments. However, overall weight gain from 0-7 weeks in the restricted treatment was still significantly ($P<0.01$) less than the other groups. There was no treatment

Table 10. Mean body weights and weight gains of male and female broilers - Experiment 1

Parameters	Period (weeks)	Sex	Dietary treatment T_1 (Basal wheat)	T_2 (Restricted)	T_3 (Fortified wheat)	T_4 (Corn)
Mean body weight (g)	0	Male	49	49	49	49
		Female	48	48	48	48
		Average	48.5	48.5	48.5	48.5
	1	Male	128±3.7	134±1.4	132±4.2	141±2.1
		Female	124±1.7	127±2.2	129±1.6	136±1.3
		Average	126_A[1]	131_A	131_A	139_B
	5	Male	1299±12.6	1097±12.2	1321±15.7	1353±15.6
		Female	1101±4.7	958±10.2	1110±8.3	1197±8.1
		Average	1200_A	1028_B	1216_A	1275_C
	7	Male	2037±46	1892±12	2015±28	2087±27
		Female	1695±31	1028±14	1683±10	1781±12
		Average	1866_{AB}	1760_C	1849_B	1934_A
Mean weight gain (g)	0-1	Male	79±3.4	85±1.1	83±4.2	92±2.0
		Female	76±1.7	79±2.1	81±1.6	88±1.3
		Average	78_A	83_A	82_A	90_A
	1-5	Male	1171±9.0	963±13.6	1189±15.4	1212±15.3
		Female	977±5.3	831±12.1	981±7.0	1061±8.8
		Average	1074_B	897_C	1085_B	1137_A
	5-7	Male	738±41.5	795±3.9	694±17.5	734±12.4
		Female	594±31.2	670±4.0	573±4.4	584±10.8
		Average	666_B	733_A	634_B	659_B
	0-7	Male	1988±46	1843±12	1966±28	2038±27
		Female	1647±31	1580±14	1635±10	1733±12
		Average	1818_{AB}	1712_C	1801_B	1886_A

[1]Means within a row not followed by the same subscript are significantly different at (A, B) $P<0.01$.

[2]All data listed showed significant ($P<0.01$) differences between males and females.

difference in weight gain during this period between birds fed the basal wheat and fortified wheat ration. Similarly, no significant difference was observed between the corn fed birds and those fed the basal wheat ration. The overall body weight gains averaged 1,818 g, 1,801 g, 1,712 g and 1,886 g for the basal wheat, wheat fortified group, wheat restricted and the corn fed groups respectively.

The 7 week mean body weight of the restricted group was significantly ($P<0.01$) less than all other treatments. The corn fed birds were significantly ($P<0.01$) heavier than the birds fed the fortified wheat diet, however no difference was observed between the corn and basal wheat groups. Similarly, there was no treatment difference between the mean weights of birds fed the basal or fortified wheat diets. Average final body weights for the basal group, restricted group, fortified group and corn fed group were: 1,866 g, 1,760 g, 1,849 g and 1,934 g respectively.

B. Feed consumption

Feed consumption and feed:gain ratios for the 7 week trial are presented in Table 11 while mean squares from analysis of variance tables are listed in Appendix 1.

For all periods tested and within each treatment group, feed consumption was significantly ($P<0.01$) less for females than males.

No significant differences among treatment groups were observed during the initial week when all birds were fed ad libitum. From the end of the first week through the fifth week, the group fed 90% of ad libitum intake consumed significantly ($P<0.01$) less feed than all other groups. The amount of feed consumed was calculated to have been 86% of

Table 11. Mean feed consumption and feed:gain ratio of male and female broilers - Experiment 1

Parameters	Periods (weeks)	Sex	Dietary treatment[2] T_1	T_2	T_3	T_4
Mean feed consumption (g/bird)	0-1	Male	102±2.2	105±0.8	101±3.8	102±1.7
		Female	97±1.5	102±2.7	98±1.3	97±0.6
		Average	99.5_A[1]	103.5_A	99.5_A	99.5_A
	1-5	Male	2027±25.4	1749±12.8	2060±11.1	2053±28.2
		Female	1791±7.5	1542±12.5	1777±3.2	1865±6.0
		Average	$1909±0_A$	1645.5_B	1918.5_A	1959.0_A
	5-7	Male	1845±53.8	1770±2.0	1830±11.1	1802±20.2
		Female	1586±31.1	1515±6.7	1522±14.5	1582±10.1
		Average	1715.5_A	1642.5_A	1676.0_A	1692.0_A
	0-7	Male	3974±76	3624±11	3991±2.2	3957±49
		Female	3474±33	3159±9	3397±11	3544±11
		Average	3724_A	3391.5_B	3694_A	3750.5_A
Feed:gain	0-1	Male	1.30±.04	1.22±.01	1.22±.02	1.11±.01
		Female	1.29±.02	1.29±.01	1.23±.01	1.11±.01
		Average	1.30_C	1.26_{BC}	1.23_B	1.11_A
	1-5	Male	1.73±.01	1.82±.02	1.73±.02	1.69±.01
		Female	1.83±.00	1.86±.02	1.81±.01	1.76±.02
		Average	1.78_B	1.84_C	1.77_{AB}	1.73_{CA}
	5-7	Male	2.51±.10	2.23±.01	2.64±.05	2.45±.03
		Female	2.68±.01	2.26±.02	2.65±.04	2.71±.03
		Average	2.59_B	2.25_A	$2/65_B$	2.59_B
	0-7	Male	2.00±.03	1.97±.01	2.03±.02	1.94±.01
		Female	2.11±.02	1.97±.01	2.03±.02	1.94±.01
		Average	2.06_B	1.99_A	2.06_B	1.99_A

[1]Means within a row (average) not followed by the same subscript are significantly different at (A, B) $P<0.01$.

[2]With the exception of 0-1 feed:gain, all data listed showed significant ($P<0.01$) differences between males and females.

the intake of the birds fed the basal wheat ration. No treatment differences were observed among the 3 treatments offered feed ad libitum.

During the final 2 weeks of the trial, there was no statistically significant difference in feed intake among the 4 treatment groups. The restricted group did consume less feed than the other treatments and the difference was approaching significance at $P<0.05$. The restricted group was found to have consumed 96% of the amount of feed ingested by the birds fed the basal ration, 6% more than should have been allotted.

Overall feed consumption (1-7 weeks) was not significantly different among the 3 full fed treatment groups. The restricted birds consumed significantly ($P<0.01$) less feed than all other groups during this period. This group was found to have consumed 91% of the amount of feed ingested by the birds offered the basal wheat ration.

C. Feed:gain ratio

With the exception of the first week of the trial, all other periods and within each treatment group, males were found to have significantly ($P<0.01$) better feed conversion than females. All of the following values represent the treatment averages of males and females.

During the initial week of the trial, the corn fed birds showed significantly better feed conversion, yielding a feed:gain ratio of 1.11 as compared to the wheat fed birds which produced ratios ranging from 1.23 to 1.29.

The corn diet maintained its superior feed:gain ratio (1.73) during the period of 1-5 weeks ($P<0.05$). There was, however, no significant

difference between the basal wheat or fortified wheat ration yielding feed:gain ratios of 1.78 and 1.77 respectively. The restricted group was the most inefficient, resulting in a feed:gain ratio of 1.84 which was significantly ($P<0.01$) poorer than all other treatments.

From 5-7 weeks, the wheat restricted group had significantly ($P<0.01$) better feed conversion than any of the 3 full fed groups among which there were no significant differences. Feed:gain ratios for the restricted group, basal group, fortified group and the corn fed group were 2.24, 2.59, 2.65 and 2.58 respectively.

Overall feed conversion (0-7 weeks) was found to be significantly ($P<0.01$) better in both the restricted and corn fed birds as compared to the basal wheat and fortified wheat groups. Respective ratios were 1.99, 1.98, 2.06 and 2.05.

D. Dressing percent and fat pad percent

Mean dressing percentages, fat pad percentages, total mortality and SDS mortality for the 7 week trial are presented in Table 12. Mean squares from analysis of variance tables are reported in Appendix 2.

Within all treatment groups, females were found to have significantly ($P<0.05$) higher dressing percentages than males with differences ranging from 0.31% to 1.75%. A significant ($P<0.01$) treatment effect was also observed in that the restricted birds exhibited a reduced dressing percentage (67.71%) when compared to the 3 full fed groups. Dressing percents for the latter groups were not significantly different ranging from 69.04% to 69.89%.

A significant ($P<0.05$) effect of sex was observed for fat pad percent

Table 12. Mean dressing percentage, fat pad percentage and mortality of male and female broilers - Experiment 1

Parameters	Period (weeks)	Sex	T_1	T_2	T_3	T_4
Dressing percent[2,4]	7	Male	68.7±2.7	66.8±2.8	68.9±1.3	69.5±1.1
		Female	59.4±1.3	68.6±2.3	69.2±2.0	70.3±0.8
		Average	69.1_A[1]	67.7_B	69.0_A	69.9_A
Fat pad percent[3]	7	Male	1.87±0.5	1.27±0.5	1.84±0.5	1.70±0.6
		Female	2.24±0.7	2.06±0.8	2.49±0.7	2.19±0.8
		Average	2.06_{ab}	1.67_a	2.17_{ab}	1.95_{ab}
Total no. of[5] mortalities (%)	0-7	Male	11(5.24)	6(2.86)	9(4.29)	13(6.19)
		Female	4(1.90)	5(2.38)	7(3.33)	2(0.95)
		Male & female	$15(3.57)_A$	$11(2.62)_A$	$16(3.81)_A$	$15(3.57)_A$
No. of SDS mortalities (%)	0-7	Male	6(2.86)	4(1.90)	5(2.38)	4(1.90)
		Female	1(0.48)	4(1.90)	3(1.43)	0(0.00)
		Male & female	$7(1.66)_A$	$8(1.90)_A$	$8(1.90)_A$	$4(0.95)_A$

[1]Means within a row not followed by the same subscript are significantly different at (A,B) $P<0.01$ (a,b) $P<0.05$.

[2]Significant differences ($P<0.05$) were present between males and females in all treatments.

[3]Significant differences ($P<0.01$) were present between males and females in all treatments.

[4]Calculated by dividing carcass wt. by live wt. Carcass wt. included the neck but not the feet, head or giblets.

[5]Includes culled birds.

with females of each treatment having the greater percent of abdominal fat. Differences between males and females ranged from 0.37% to 0.79%.

A treatment effect ($P<0.05$) was also observed for fat pad percent. The restricted group produced less abdominal fat than with the basal wheat or fortified wheat fed groups with average values being equal to 1.67%, 2.06% and 2.17% respectively. The restricted group also had less abdominal fat pad production than the corn fed birds (1.95%), although the difference was not significant. Similarly there was no significant difference between the corn fed birds and the basal wheat and fortified wheat groups.

E. Mortality

No significant differences for treatment or sex were observed regarding total mortality although mortality tended to be greater in males. Total mortality ranged from 2.62% to 3.81% of the total number of birds started.

SDS mortality was low and not significantly different among treatment groups. Differences in SDS mortality between males and females were not statistically significant although the difference was approaching significance at $P<0.05$ with males tending to be more susceptible to SDS. A total of 20 males (50% of total male mortality) and a total of 8 females (44.44% of total female mortality) died from SDS.

The lowest incidence of SDS was observed in the corn fed group with male plus female SDS deaths totalling 4 birds (0.95%). The other 3 groups averaged a combined male-female SDS mortality of 8 (1.90%) individuals.

DISCUSSION

Although significant differences were found between males and females with regard to performance parameters, sex differences are well documented and therefore only treatment differences are discussed.

A. Mean body weights and weight gains

At 1 and 5 weeks of age, the corn fed birds were found to be significantly heavier than the full fed birds offered the wheat based ration. The increased gains were surprising in that all diets were formulated to contain equal energy and protein and there were no differences in feed consumption.

The superior gains of the corn fed birds may have been due to differences in the fatty acid (saturated vs unsaturated) make up of the different diets resulting in differences in the amount of energy absorbed by the chick. Differences in the proportions of fatty acids would be expected as the corn diets had less added tallow (saturated fat) and would contain greater amounts of unsaturated fats due to the composition of corn oil (≈85% unsaturated; Scott 1982). Differences in absorbed energy could thus have resulted as March and Biely (1957) showed that corn oil was absorbed more readily by the chick than was beef tallow. Furthermore, Sell et al. (1976) showed that increased amounts of unsaturated fatty acids improved the absorption of saturated fats. Thus the greater amounts of unsaturated fats (specifically linoleic acid), of the corn diet allowed the chick to better utilize the tallow resulting in improved weight gains due to increased energy absorption.

At 5 weeks of age, the birds fed 90% of ad libitum intake were sig-

nificantly lighter than all other treatments with the difference in body weight being about 15%. This reduction in body weight parallels the difference in feed intake as the restricted group was found to have received 86% of ad libitum intake or 14% less than the full fed birds. Thus, a 14% reduction in intake resulted in a 15% reduction in body weights. Similar results were presented by Waldroup et al. (1976) who found a significant linear increase in weight gain as caloric intake increased.

From 5-7 weeks, the 90% group gained significantly more weight than the treatments fed ad libitum. This increased (compensatory) growth may have been due to the restricted birds receiving more feed (96% vs 90% of ad libitum intake) than was intended. The observed increase in growth rate would likely not have occurred if the birds had received less feed as the energy and protein intake would not have been sufficient to sustain the observed gains.

Overall weight gain tended to be greater in the corn fed birds although 7 week weights were not significantly heavier than the full fed wheat treatments. Although the wheat and corn diets were formulated to contain equal protein and energy, the slightly superior overall performance of the corn fed birds further indicated that differences in energy absorption may have occurred.

B. Feed consumption

Feed consumption during the first week was not significantly different among treatments. Slight errors in measurements likely caused the 1-5 week consumption and 5-7 week consumption of the restricted

birds to be slightly below (1-5 weeks) and slightly above (4-7 weeks) the desired 90% of ad libitum intake. Overall, however, the intake of the restricted birds was calculated to be almost exactly 90% of the ad libitum groups. The overfeeding of the restricted birds from 5-7 weeks may have been significant in that it resulted in heavier 7 week weights (due to compensatory growth) than would have resulted from feeding 90% of ad libitum intake.

C. Feed:gain ratios

Feed:gain ratios were significantly better in the corn fed birds than in the full fed wheat groups. The improved feed:gain may be attributed to the corn fed birds being better able to absorb fat as explained in section A. Fortifying the wheat diet with additional vitamins and minerals did not affect feed:gain indicating that the standard amounts added are adequate to attain efficient feed conversion.

The restricted birds had significantly poorer feed conversion from 1-5 weeks, likely as a result of reduced protein and energy intake as other authors have shown that reduced energy intake resulted in poor feed conversion (Forbes and Yohe 1955; Griffiths et al. 1977a). From 5-7 weeks, however, the restricted birds showed significant improvements in feed conversion due to the fact that the birds received more feed than intended and were thus able to demonstrate compensatory growth. Compensatory growth has been shown to improve feed efficiency by numerous authors including Ried and White (1978); Marks (1979) and Moran (1980).

Overall feed:gain was not significantly different between the birds

fed the basal and fortified basal rations. Similarly the corn fed birds and the restricted birds had similar feed:gain ratios overall. The fact that there was no difference in overall feed:gain between the corn fed birds and the restricted birds was due to the magnitude of the improvement in feed:gain of the restricted group from 5-7 weeks. The improved overall feed:gain ratio of the restricted birds does not agree with other reports in which improvements in feed:gain as a result of compensatory growth are cancelled by poorer feed conversion during the restricted period resulting in overall feed:gain ratios not better than the non-restricted controls (Marks 1979; Moran 1979; Cherry et al. 1978). Differences in results may be due to differences in the age when restriction was imposed, strain of bird and the severity of the restriction (Ried and White 1978). Evidence that age may have been the most important factor was provided by Beane et al. (1979) who found that the improvement in feed:gain as a result of compensatory growth that occurred from 6-8 weeks of life was great enough to significantly improve the overall 0-8 week feed:gain ratio.

D. Dressing percent and fat pad percent

The restricted group was found to have a significantly lower dressing percentage than the other treatments. Beane et al. (1979) also found that restricted feeding resulted in a significantly smaller dressing percent as compared to full fed controls and suggested that the observed differences were simply due to the fact that the restricted birds had lighter body weights. Another explanation is that restricted feeding has been found to affect the size of the GI tract. McCartney

and Brown (1977) observed that birds limited in feeding time and thus consuming less feed, were found to have significant increases in the size of the crop and proventriculus. Thus, if the weight of the GI tract is increased, dressing percent will be decreased as the GI tract is removed and is not included in the carcass weight.

Fat pad percent was found to be significantly less in the restricted group than in either of the groups fed wheat rations ad libitum. The restricted birds also tended to have less abdominal fat deposition than the corn fed birds with the difference approaching significance at $P<0.05$.

These results appear to contrast those of Beane et al. (1979) who found that birds restricted to 85% of ad libitum consumption (from 14 to 42 days of age) and then returned to full feed (from 43 to 56 days of age) produced more abdominal fat than nonrestricted controls even though 8 week weights of the restricted birds were less than weights of the non-restricted birds. Differences in the results of Experiment 1 and the results of Beane may reflect differences in the final 2 weeks of each trial in that birds in the latter trial were fed ad libitum during this time whereas in Experiment 1 the birds were restricted during the final 2 weeks. Beane suggested that the increased deposition of abdominal fat may have been due to hyperphagia after birds were returned to ad libitum feeding resulting in excess energy intake and thus increased fat deposition.

Various factors may have contributed to the restricted birds having less abdominal fat than the other treatment groups.

Differences in body weight, which reflect differences in physio-

logical age, likely contributed to differences in abdominal fat pad size. Numerous studies (Edwards and Hart 1971; Deaton et al. 1974; Evans et al. 1976) have demonstrated that as birds get older (and heavier) percent carcass protein decreases and percent fat increases. Thus, as the restricted birds were lighter at the time of slaughter, they were depositing more protein and less fat than were the nonrestricted birds.

Another factor that could have contributed to reduced abdominal fat in the restricted birds is that hyperplasia and hypertrophy of the abdominal fat pad were likely decreased. Pfaff and Austic (1976) found that low energy intakes slowed hyperplastic growth of abdominal fat. Restricted feeding could have reduced hypertrophy of the fat pad by reducing the amount of excess energy available to be stored as fat. It is also possible that if the restricted birds had been given less feed (90% of ad libitum rather than 96% of ad libitum) from 5-7 weeks and compensatory growth was eliminated, abdominal fat would have been further reduced. Compensatory growth has been shown to increase abdominal fat (Beane et al. 1979) so it is possible that this growth spurt from 5-7 weeks may have slightly increased the amount of abdominal fat in the restricted birds.

Although the restricted birds were found to deposit less abdominal fat, 7 week body weights were also significantly less than for the full fed groups. Therefore practical application of restricted intake may meet resistance from producers as they are concerned with attaining maximum body weights since the price paid to the producer is based on live weight. However, the savings in feed cost when restricted intake is

practiced can be shown to offset the loss of gross income as a result of lighter market weights. Based on current feed costs and price paid to the producer, no difference in net profit was found to exist between full fed and restricted birds. Calculations are shown in Appendix 3. Only feed costs are shown as fixed costs would not be affected.

E. Mortality

No treatment differences were observed for either total or SDS mortalities.

Feed restriction did not increase or decrease SDS incidence and thus questions the concept that growth rate and SDS are related although it may be that a greater difference in weight gain is required to produce differences in SDS mortality.

Fortification of the ration with additional vitamins and minerals had no effect on total mortality (including culls) or SDS mortality although the levels and/or vitamins and minerals used may not have been sufficient to affect the syndrome.

SDS mortality did tend to be lower in the group fed the corn diet. No explanation was apparent, although differences in the types of dietary fatty acids (saturated vs unsaturated) may have been involved.

Males were observed to be more frequently affected by SDS than females although differences were not significant. Although no explanation for sex differences can be put forward, the observation that males are more susceptible confirms reports by Hemsley (1965), Jackson et al. (1972) and Hulan et al. (1980).

II. The effects of compensatory growth and vitamin levels of broiler diets on the incidence of Sudden Death Syndrome (SDS). (Experiment 2).

RESULTS

Mean body weights, weight gains and feed:gain ratios for the 7 week experimental period are listed in Table 13. Mean squares from analysis of variance tables are presented in Appendix 4.

A. Mean body weights and weight gains

From 0-2 weeks of age, birds fed the low protein, vitamin reduced starter ration gained significantly ($P<0.01$) less body weight than the control group which received a ration containing adequate protein and vitamin levels. Two week mean body weights for the former groups ranged from 170 g to 175 g as compared to the latter group with mean weights of 216 g.

After 3 weeks (2-5 weeks of age) of being fed a protein and vitamin repleted ration (treatment 3 received increased protein but remained on a low vitamin regime) and with the exception of treatment 4, fed additional B vitamins, the restricted treatments were still significantly ($P<0.01$) lighter than the controls. Treatment 4 was not significantly different from either the controls or the other treatment groups. At 7 weeks of age, all treatment groups had average body weights slightly less than the control group but only treatment 3 had an average body weight significantly ($P<0.05$) less than the control group. Mean 7 week body weights ranged from 1,986 g (T_3) to 2,134 g (control).

B. Feed:gain ratios

During the initial 2 week period, all restricted treatments were

Table 13. Mean body weights, weight gains and feed:gain ratios of male broilers - Experiment 2

Parameters	Periods (weeks)	Dietary treatment[1]				
		T_1	T_2	T_3	T_4	T_5
Mean body weight (g)	0	44	44	44	44	44
	2	$216\pm6.0_{A}$[2]	$175\pm2.7_{B}$	$171\pm4.0_{B}$	$170\pm2.5_{B}$	$170\pm1.6_{B}$
	5	$1259\pm17.5_{A}$	$1173\pm12.2_{BC}$	$1152\pm12.2_{B}$	$1206\pm9.4_{AC}$	$1178\pm6.1_{BC}$
	7	$2134\pm4.5_{a}$	$2047\pm27.3_{ab}$	$1986\pm8.3_{b}$	$2048\pm14.7_{ab}$	$2036\pm15.1_{ab}$
Mean weight gain (g)	0-2	$172\pm5.0_{A}$	$131\pm2.7_{B}$	$127\pm4.0_{B}$	$126\pm2.5_{B}$	$126\pm1.6_{B}$
	0-5	$1215\pm17.5_{A}$	$1129\pm12.2_{BC}$	$1108\pm12.2_{B}$	$1162\pm9.4_{AC}$	$1134\pm6.1_{BC}$
	0-7	$2090\pm4.5_{a}$	$2003\pm27.3_{a}$	$1942\pm8.3_{b}$	$2004\pm14.7_{a}$	$1992\pm15.1_{a}$
Feed:gain	0-2	$1.43\pm.00_{a}$	$1.67\pm.06_{b}$	$1.68\pm.04_{b}$	$1.71\pm.06_{b}$	$1.68\pm.04_{b}$
	0-5	$1.67\pm.02_{A}$	$1.61\pm.01_{A}$	$1.62\pm.01_{A}$	$1.62\pm.01_{A}$	$1.65\pm.01_{A}$
	0-7	$1.89\pm.02_{A}$	$1.86\pm.03_{A}$	$1.89\pm.02_{A}$	$1.89\pm.01_{A}$	$1.88\pm.02_{A}$

[1]Dietary treatments were as follows: T_1 - control; T_2 - restricted, standard vitamin levels; T_3 - restricted, 50% of standard vitamin levels; T_4 - restricted, additional B vitamins; T_5 - restricted, additional fat soluble vitamins.

[2]Means within a row not followed by the same subscript are significantly different at (A,B) $P<0.01$ (a,b) $P<0.05$.

significantly less efficient than the controls. The feed:gain ratio of the control group was 1.43 as compared to the other treatments where values ranged from 1.67 to 1.71. Calculated from 0-5 weeks, feed:gain was not significantly different among all groups with ratios ranging from 1.61 to 1.67. Overall (0-7 weeks), feed conversion was not significantly different among treatments with values ranging from 1.86 to 1.89.

C. Mortality

Total and SDS mortality figures from the 7 week period are presented in Table 14. Mean squares from analysis of variance tables are listed in Appendix 5.

No treatment differences were observed in total mortality for 0-2, 0-5 and 0-7 weeks of age. Total mortality by 7 weeks ranged from 3.11% to 4.33% of the total number of chicks started per treatment.

SDS mortality was similar to total mortality in that there were no treatment differences for either 0-2, 0-5 or 0-7 weeks. The lowest overall SDS mortality was in treatment 5 (increased levels of vitamins, A, D and E) with 1.66% of the initial population dying from SDS. The greatest mortality was observed in the control group in which 2.86% of the birds died from SDS.

Table 14. Mortality data, male broiler chickens - Experiment 2

Parameters	Periods (weeks)	Dietary treatment[1]				
		T_1 (control)	T_2	T_3	T_4	T_5
Total no. of mortalities (%)	0-2	1 (0.71)	2 (0.47)	6 (1.42)	3 (0.71)	3 (0.71)
	0-5	4 (2.86)	6 (1.42)	9 (2.14)	8 (1.90)	8 (1.90)
	0-7	6 (4.33)	17 (4.07)	14 (3.35)	14 (3.35)	13 (3.11)
Total no. of SDS mortalities (%)	0-2	1 (0.71)	2 (0.47)	3 (0.71)	1 (0.23)	1 (0.23)
	0-5	3 (2.14)	4 (0.95)	6 (1.43)	5 (1.18)	5 (1.18)
	0-7	4 (2.86)	9 (2.14)	10 (2.40)	9 (2.15)	7 (1.66)

[1]There were no significant differences among treatments.

DISCUSSION

The purpose of Experiment 2 was to study the effects of growth rate and vitamin levels on the incidence of SDS. Differences in SDS incidence are often difficult to establish due to the relatively low number of individuals affected. As a consequence it was determined that it would be desirable to try and maximize the occurrence of SDS, that is if a degree of SDS higher than reported averages could be induced, possible treatment differences would be more significant.

As SDS has been associated with rapid growth rate and has been reported to peak between 3 and 5 weeks of age (Hulan et al. 1980) an attempt was made to induce a period of rapid (compensatory) growth during the suggested critical period. During this period, vitamin levels and types were varied to determine if SDS could be influenced by vitamin supplementation.

A. Mean body weights and weight gains

The results of Experiment 2 showed that after 2 weeks of protein and vitamin restriction, body weights of the restricted birds were approximately 20% less than the average weights of the nonrestricted controls. Although reduced vitamin supplementation may have contributed somewhat, the slower growth rate may be attributed to the low protein content of the ration fed. Even when energy level is adequate, insufficient protein has been shown to reduce performance (Hill and Dansky 1950; Peterson et al. 1954; Matterson et al. 1955; Donaldson et al. 1956). Furthermore, Moran (1979) and Marks (1979) both observed similar growth depression when chicks were fed a low protein diet from 0-2 weeks of age. The

later author reported differences of 20%, the restricted birds being lighter than the controls.

The levels of B vitamins in the ration fed to the restricted groups from 2-5 weeks seemed to affect the ability of the bird to exhibit compensatory growth in that the group fed additional B vitamins was the only treatment not significantly lighter than the controls by 5 weeks of age. Conversely, the smallest 5 week body weights were observed in the group fed 50% of standard vitamin levels. By 7 weeks, this group was the only treatment significantly ($P<0.05$) lighter than the controls. In addition, this group tended to be lighter (by approximately 3%) than all other treatments, although the difference was not significant.

Final body weights were not significantly improved by increasing the level of B vitamins or fat soluble vitamins indicating that the standard premix supplied both types of vitamins at adequate levels. The low vitamin group tended to be lighter indicating that additional vitamins were required to support maximum growth rates.

The birds in this experiment did show some ability to compensate for early growth restriction induced by a low protein diet. The failure to fully compensate may have been due to the severity of the restriction (i.e. protein level and duration) and the age at which it was imposed. Ried and White (1978) stated that the severity of undernutrition and the length of time a deficient ration is fed affects the ability to compensate while Marks (1979) suggested that early life restriction is more detrimental to compensatory growth than is restriction later in life. Full compensation may also have occurred if the birds had been given a longer period of time to recover.

B. Feed:gain ratio

Feed conversion from 0-2 weeks was negatively affected by the restriction imposed. Feed conversion from 0-5 weeks however, was not significantly different between restricted and nonrestricted birds, indicating that the restricted birds used feed more efficiently than the controls from 2-5 weeks of age.

Marks (1979) and Moran (1979) reported similar trends with the improvement in feed:gain following a period of protein restriction lasting up to 3 weeks. Marks suggested that improvements in feed conversion could be due to a reduction in feed consumption when birds were returned to a ration containing adequate protein. This author also stated that as the restricted birds were lighter, their maintenance requirements were less and feed:gain was thus improved.

As 0-7 week feed:gain was not significantly different among treatments, the use of early restriction does not appear to either increase or decrease overall feed efficiency. Initial decreases in feed efficiency are countered by improvements once the restriction is removed with the net result being a final feed efficiency not significantly different from that of a nonrestricted control.

C. Mortality

The incidence of SDS over the 7 week period ranged from 1.66% to 2.86%. These percentages are within the ranges reported by Brigden and Riddell (1975). The attempt to induce a higher than normal incidence of SDS by inducing a period of rapid compensatory growth was thus unsuccessful.

The importance of growth rate may be questioned in that there were

no significant differences in SDS mortalities between restricted and non-restricted groups from 0-2 weeks, that is the comparatively slow growth of the restricted birds did not reduce the incidence of SDS when compared to that of the controls. It may be that continued differences in growth rate over a longer period of time may have resulted in differences in SDS mortalities. However, even if SDS could be reduced by slowing growth rates, this approach would be uneconomical due to increased costs associated with a longer growing period.

Hulan et al. (1980) reported that additional B vitamins, specifically biotin, significantly reduced the incidence of SDS and suggested that further research with biotin was required.

The results of Experiment 2 contradict those of Hulan et al. (1980). The addition of biotin and/or other B vitamins did not reduce the incidence of SDS. Furthermore, reducing the content of these vitamins did not result in any increase in SDS mortality. Additional evidence that biotin does not affect SDS has since been provided by Steele et al. (1982). Thus the possibility that biotin or other B vitamins can reduce SDS is unlikely.

The lowest incidence of SDS was found to be in the treatment supplemented with vitamins A, D and E. The small number of birds in this study and the incidence of SDS may not have been great enough to show a significant reduction and the possible involvement of fat soluble vitamins may warrant further investigation.

III. The effects of early life caloric (fat) restriction and calorie: protein ratio on abdominal fat pad production and the incidence of Sudden Death Syndrome in broiler chickens. (Experiment 3).

RESULTS

A. Mean body weights and weight gains

Mean body weights, weight gains and feed:gain ratios during the 7 week period are presented in Table 15 while mean squares from analysis of variance tables are reported in Appendix 6 and 7 (0-4 week data and 4-7 week data respectively).

The initial week of fat restriction resulted in a reduction ($P<0.01$) in weight gain (and consequently body weight) by 7 days of age. Restricted groups (T_2 and T_3) averaged 1 week weights of 124 g as compared to the nonrestricted groups that averaged body weights of 140 g (T_1 and T_4). For each of the following weeks, until 4 weeks of age, there were no differences ($P<0.05$) among treatments with regard to weight gain. Measured at 4 weeks, slight, although significant ($P<0.05$) differences in body weight were still present in that the early restricted groups were lighter than the nonrestricted groups. The former treatments averaged 997 g compared to the latter groups which averaged 1,019 g, a difference of 2.16%.

From 4-7 weeks of age, there were no differences ($P<0.05$) in weight gain among treatments and body weights at 7 weeks showed no treatment differences, ranging from an average of 2,441 g to 2,468 g.

Table 15. Mean body weights, weight gains and feed:gain ratios of male broilers - Experiment 3

Parameters	Periods (weeks)	Dietary treatment[1] T_1	T_2	T_3	T_4
Mean body weight (g)	0	44.0	44.0	44.0	4.0
	1	$140.0\pm1.5_A$[2]	$124.0\pm1.1_B$	$124.0\pm1.1_B$	$140.0\pm1.4_A$
	2	$333.0\pm3.4_A$	$311.0\pm1.9_B$	$311.0\pm2.6_B$	$332.0\pm3.2_A$
	3	$633.0\pm5.1_a$	$611.0\pm5.2_b$	$608.0\pm4.7_b$	$631.0\pm4.6_a$
	4	$1020.0\pm7.4_a$	$993.0\pm8.0_b$	$996.0\pm4.5_b$	$1018.0\pm7.6_a$
	7	$2451.0\pm16.7_A$	$2441.0\pm14.7_A$	$2458.0\pm15.3_A$	$2468.0\pm18.8_A$
Mean body weight gain (g)	0-1	$96.0\pm1.5_A$	$80.0\pm1.1_B$	$80.0\pm1.1_B$	$96.0\pm1.4_A$
	1-2	$193.0\pm2.2_A$	$187.0\pm2.3_A$	$187.0\pm1.4_A$	$192.0\pm1.4_A$
	2-3	$300.0\pm3.8_A$	$300.0\pm3.6_A$	$297.0\pm2.2_A$	$299.0\pm2.1_A$
	3-4	$387.0\pm4.4_A$	$387.0\pm4.9_A$	$388.0\pm2.9_A$	$387.0\pm3.5_A$
	0-4	$976.0\pm7.4_a$	$954.0\pm8.0_b$	$952.0\pm4.5_b$	$974.0\pm7.6_a$
	4-7	$1431.0\pm15.0_A$	$1443.0\pm12.1_A$	$1462.0\pm15.1_A$	$1450.0\pm12.1_A$
	0-7	$2407.0\pm16.7_A$	$2397.0\pm14.7_A$	$2414.0\pm15.3_A$	$2424.0\pm18.8_A$
Feed:gain	0-1	$1.13\pm.01_A$	$1.30\pm.02_B$	$1.30\pm.01_B$	$1.13\pm.01_A$
	1-2	$1.48\pm.02_a$	$1.44\pm.01_b$	$1.44\pm.01_b$	$1.49\pm.02_a$
	2-3	$1.57\pm.01_A$	$1.54\pm.01_A$	$1.55\pm.02_A$	$1.57\pm.01_A$
	3-4	$1.78\pm.02_A$	$1.76\pm.01_A$	$1.75\pm.01_A$	$1.79\pm.01_A$
	0-4	$1.60\pm.02_A$	$1.58\pm.01_A$	$1.58\pm.01_A$	$1.58\pm.01_A$
	4-7	$2.13\pm.01_a$	$2.11\pm.01_a$	$2.04\pm.02_b$	$2.08\pm.02_{ab}$
	0-7	$1.90\pm.01_a$	$1.89\pm.01_a$	$1.85\pm.01_b$	$1.87\pm.01_{ab}$

[1]Dietary treatments were as follows: T_1 - control, standard starter/finisher; T_2 - fat restricted/standard finishing diet; T_3 - fat restricted/high protein finishing diet; T_4 - standard starter/high protein finishing diet.

[2]Means within a row not followed by the same subscript are significantly differenc at (A,B) $P<0.01$, (a,b) $P<0.05$.

B. Feed:gain ratios

Elimination of added dietary fat resulted in significantly ($P<0.01$) poorer feed conversion after one week. Fat restricted birds (T_2 and T_3) had feed:gain ratios of 1.30 as compared to a value of 1.13 for the non-restricted controls (T_1 and T_4). During the period of 1-2 weeks, this trend was reversed and the restricted treatments had significantly ($P<0.01$) better feed conversion than the other treatments, yielding values of 1.44 and 1.49 respectively. During the periods 2-3 and 3-4 weeks, the restricted groups still exhibited slightly better feed:gain than the non-restricted treatments although differences were not significant ($P<0.05$). Overall ratios from 0-4 weeks were thus not significantly different among treatments with values ranging from 1.58 to 1.60.

During the period of 4-7 weeks a significant effect of protein level in the finishing diet on feed conversion was evident. Both high protein treatments (3 and 4) had lower ratios than the other two treatments although only treatment 3 showed a significant ($P<0.05$) improvement. The same pattern was evident concerning 0-7 week feed:gain in that treatment 3 (fat restricted/high protein finisher) had significantly ($P<0.05$) better feed conversion than either treatment 1 or 2. There were no differences ($P<0.05$) between treatments 3 and 4 or among treatments 1, 2 and 4. Values for treatments 1, 2, 3 and 4 were 1.90, 1.89, 1.85 and 1.87 respectively.

C. Mortality

Total mortality and SDS mortality were recorded from 0-8 weeks of age and are reported in Table 16. Mean squares from analysis of variance

Table 16. Mortality data of male broiler chickens - Experiment 3

Periods (weeks)	Total no. mortality[1] (%)	
	$T_1 + T_4$	$T_2 + T_3$
0-4	$45(4.59)_A$[2]	$49(5.00)_A$

	T_1	T_2	T_3	T_4
4-8[3]	$38(8.08)_A$	$26(5.56)_A$	$16(3.34)_A$	$19(4.08)_A$
0-8	$60(12.24)_A$	$49(10.00)_A$	$42(8.57)_A$	$44(8.97)_A$

	No. of SDS mortalities (%)	
	$T_1 + T_4$	$T_2 + T_3$
0-4	26(2.65)	27(2.76)

	T_1	T_2	T_3	T_4
4-8[3]	$21(4.46)_a$	$17(3.64)_a$	$8(1.72)_b$	$7(1.50)_b$
0-8	$32(6.53)_A$	$30(6.12)_A$	$22(4.48)_A$	$22(4.48)_A$

[1]Includes culled birds.

[2]Means within a row not followed by the same subscript are significantly different at (A,B) $P<0.01$, (a,b) $P<0.05$.

[3]Percents were calculated by dividing the no. of mortalities by the no. of live birds at 4 weeks of age.

tables are presented in Appendix 8.

Neither total nor SDS mortality during the initial 4 week starting period exhibited any significant differences between restricted and non-restricted treatments. A total of 45 birds (4.59%) from the nonrestricted treatments died or were culled. Of these, 26 birds (2.65% of the initial population) died as a result of SDS. Similarly, 49 birds (5.00%) from the restricted treatments died or were culled. Of these, 27 birds (2.76% of the initial population) died from SDS.

During the 4-8 week finishing period, significant ($P<0.05$) differences in SDS mortality were observed and were shown to be due to protein level of the finishing ration rather than early restriction. SDS mortalities for treatments 3 and 4 (high protein finisher) numbered 8 (1.72%)[1] and 7 (1.50%) birds respectively and 21 (4.46%) and 17 birds (3.64%) for treatments 1 and 2, respectively.

Treatments 3 and 4 also had lower total mortalities of 16 (3.34%)[2] and 19 (4.08%) respectively compared to total mortalities of 38 (8.08%) and 26 (5.56%) for treatments 1 and 2 respectively. These differences were not significant. There was, however, a significant ($P<0.05$) effect of protein on reducing total mortality due to the reduction in SDS mortality.

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[1]Percentages represent the number of SDS mortalities from 4-8 weeks/total number of live birds at the end of 4 weeks.

[2]Percentages represent the total number of mortalities from 4-8 weeks/ number of live birds at 4 weeks of age.

D. Mean carcass weights, dressing percent, fat pad weight and fat pad percent.

Table 17 lists data for average carcass weights, dressing percent, fat pad weights and fat pad weight as a percentage of carcass weight. Mean squares from analysis of variance tables are reported in Appendix 9. No significant treatment effects were observed for any of the aforementioned parameters.

There was however, a trend towards smaller percentages of abdominal fat in the treatments fed the high protein finisher diets with the calculated F value approaching significance at $P<0.05$. The high protein groups (3 and 4) yielded values of 2.35% and 2.25% respectively as compared to treatments 1 and 2 which produced values of 2.44% and 2.66% respectively.

In addition, although not significant, for both protein levels tested, the fat restricted groups tended to deposit greater amounts of abdominal fat.

Relationships between fat pad percent and carcass weight are presented in Figures 3, 4, 5 and 6; for treatments 1, 2, 3 and 4 respectively.

Treatment 1 showed no significant relationship between fat pad percent and carcass weight although the r value of 0.2869 was very close to being significant at $P<0.05$. The relationship between fat pad percent and carcass weight was significantly correlated ($P<0.01$) for treatment 2. The r value of 0.6440 indicated that 41.47% of the variation in fat pad percent was caused by carcass weight.

Table 17. Mean 7 week carcass weight, dressing percentage, fat pad weight, fat pad percent (as percent of carcass weight) - Experiment 3

Parameters	Dietary treatment[2]			
	T_1	T_2	T_3	T_4
Mean carcass weight (g)[1]	1616±46.5	1599±32.0	1599±33.5	1609±33.4
Mean dressing percent	68.0±1.1	68.3±0.3	67.8±0.3	67.7±0.3
Mean fat pad weight (g)	39.4±2.5	42.5±2.8	37.5±3.5	36.2±2.6
Mean fat pad percent	2.44±0.13	2.66±0.14	2.35±0.22	2.25±0.14

[1]Calculated by dividing carcass wt. by live wt. Carcass wt. included the neck but not the feed, head or giblets.

[2]There were no significant differences among treatments.

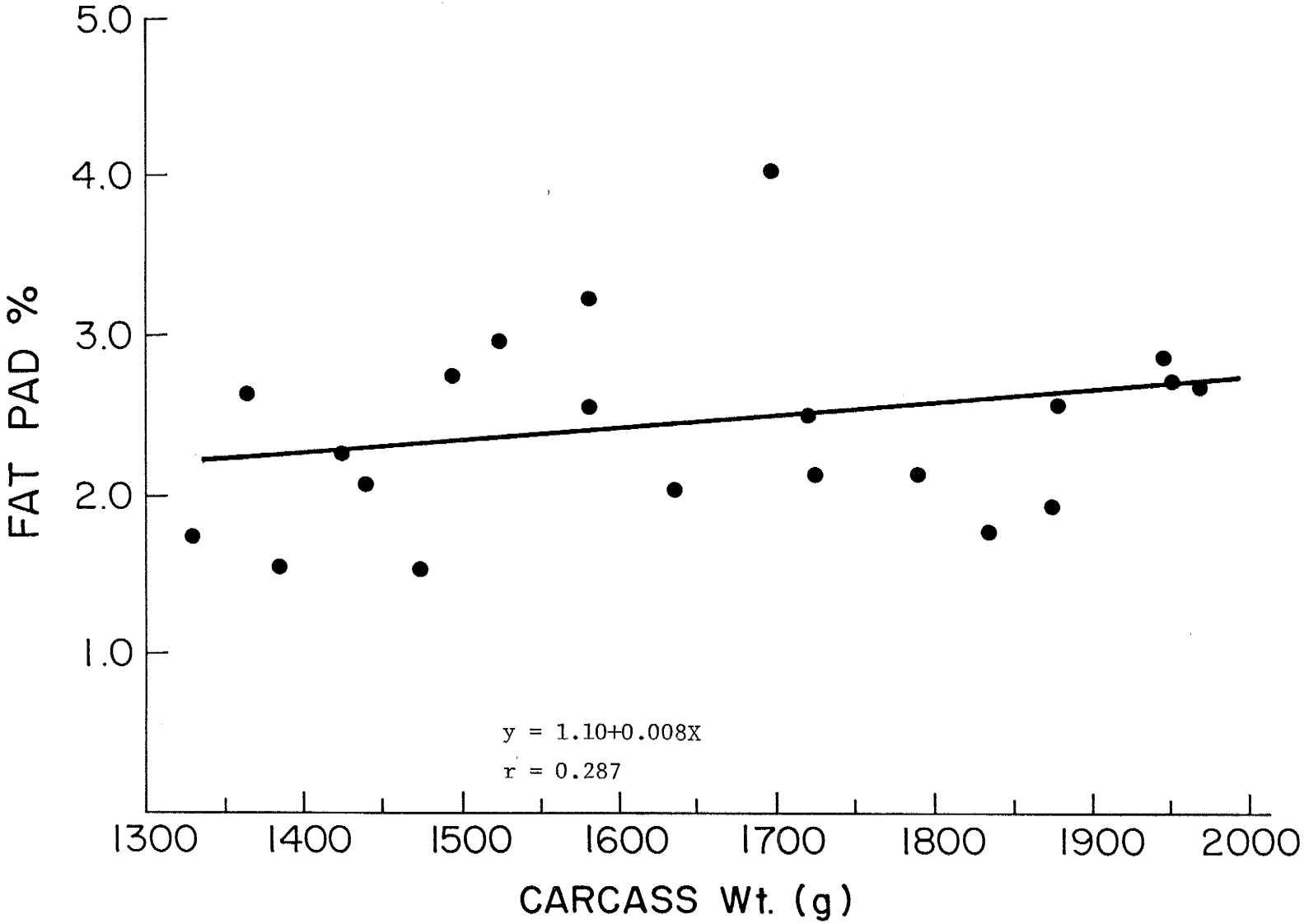

Figure 3. Relationship between fat pad percent and carcass weight (treatment 1) - Experiment 3. The data are based on 21 observations.

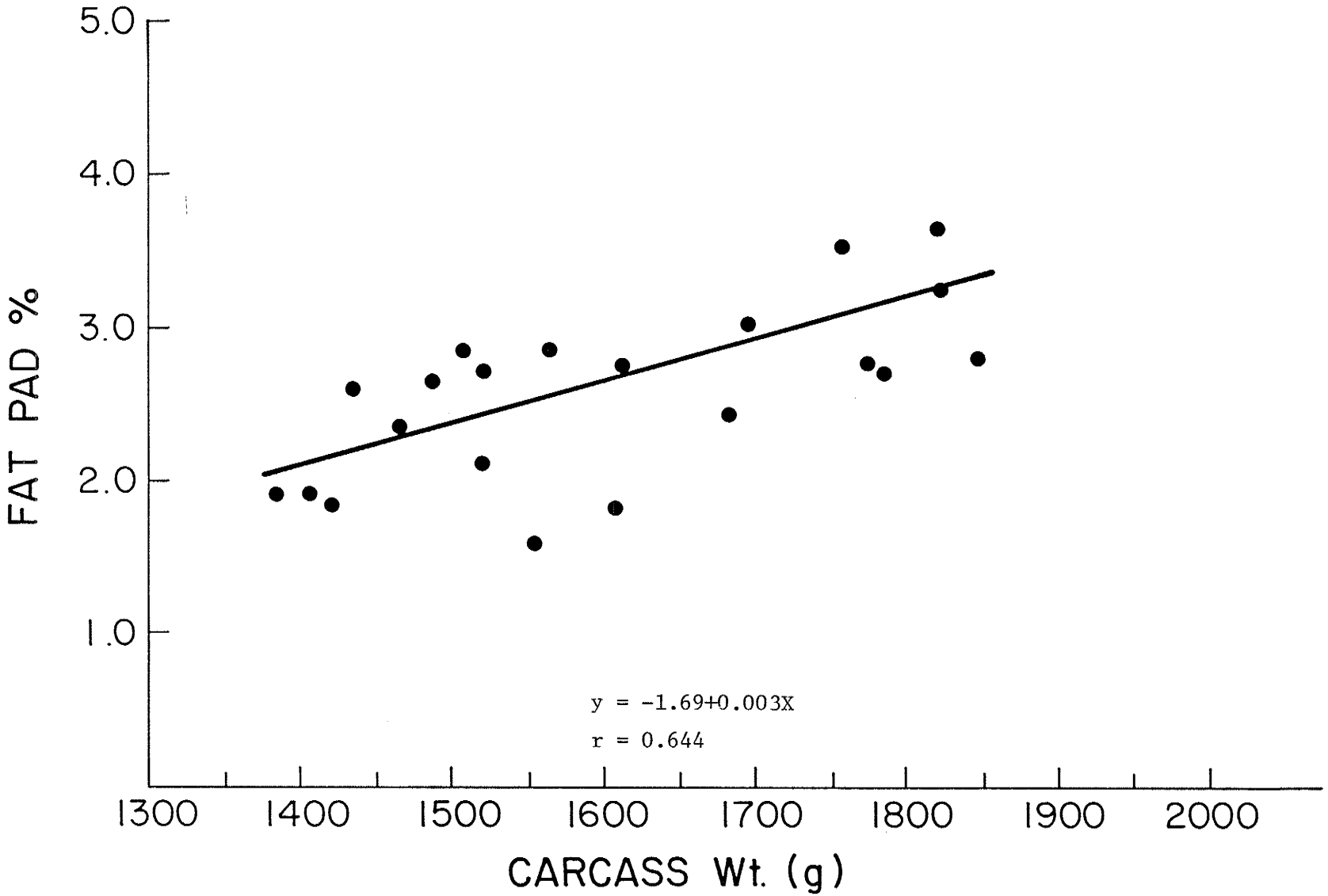

Figure 4. Relationship between fat pad percent and carcass weight (treatment 2) - Experiment 3. The data are based on 21 observations.

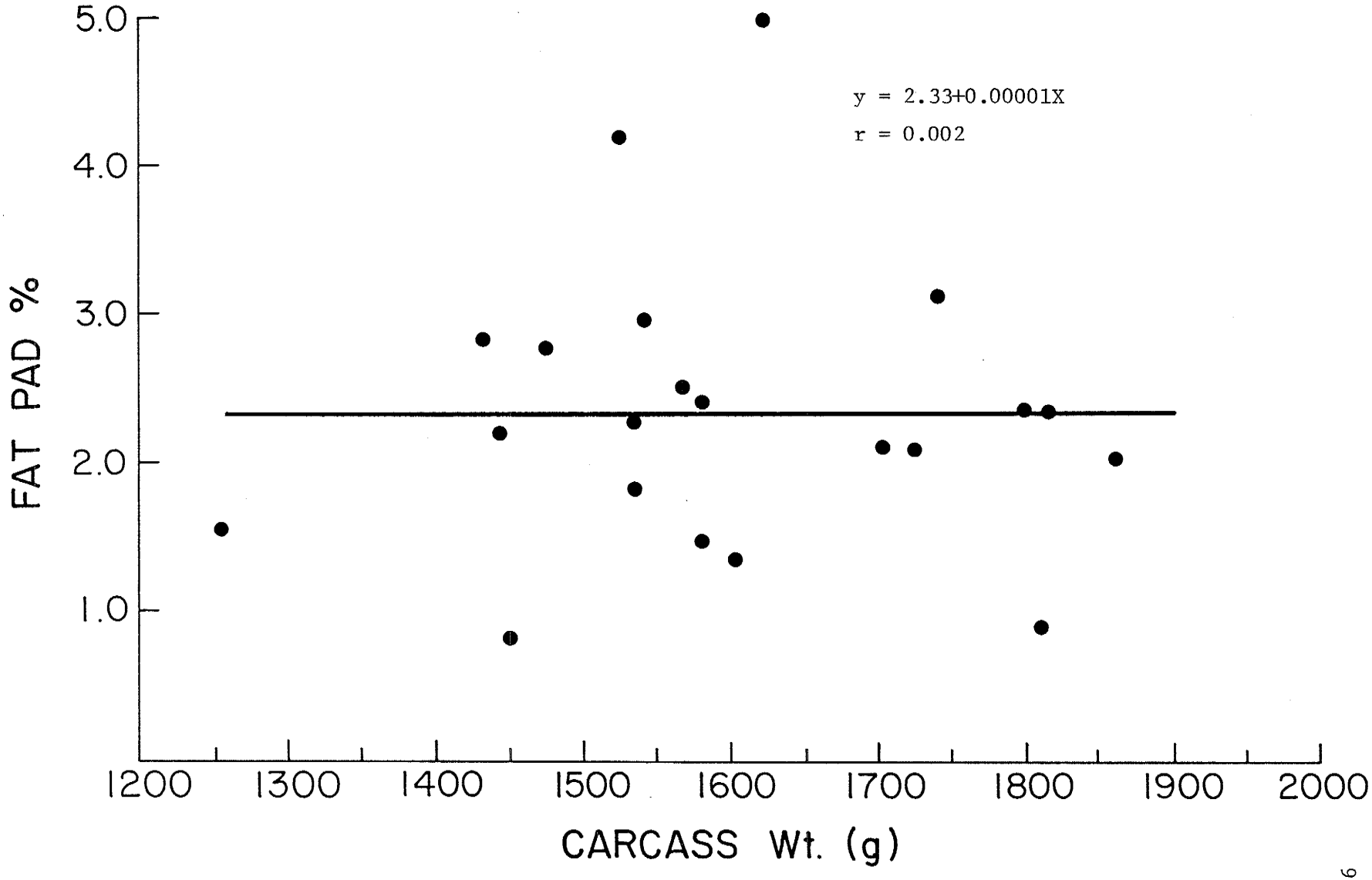

Figure 5. Relationship between fat pad percent and carcass weight (treatment 3) - Experiment 3. The data are based on 21 observations.

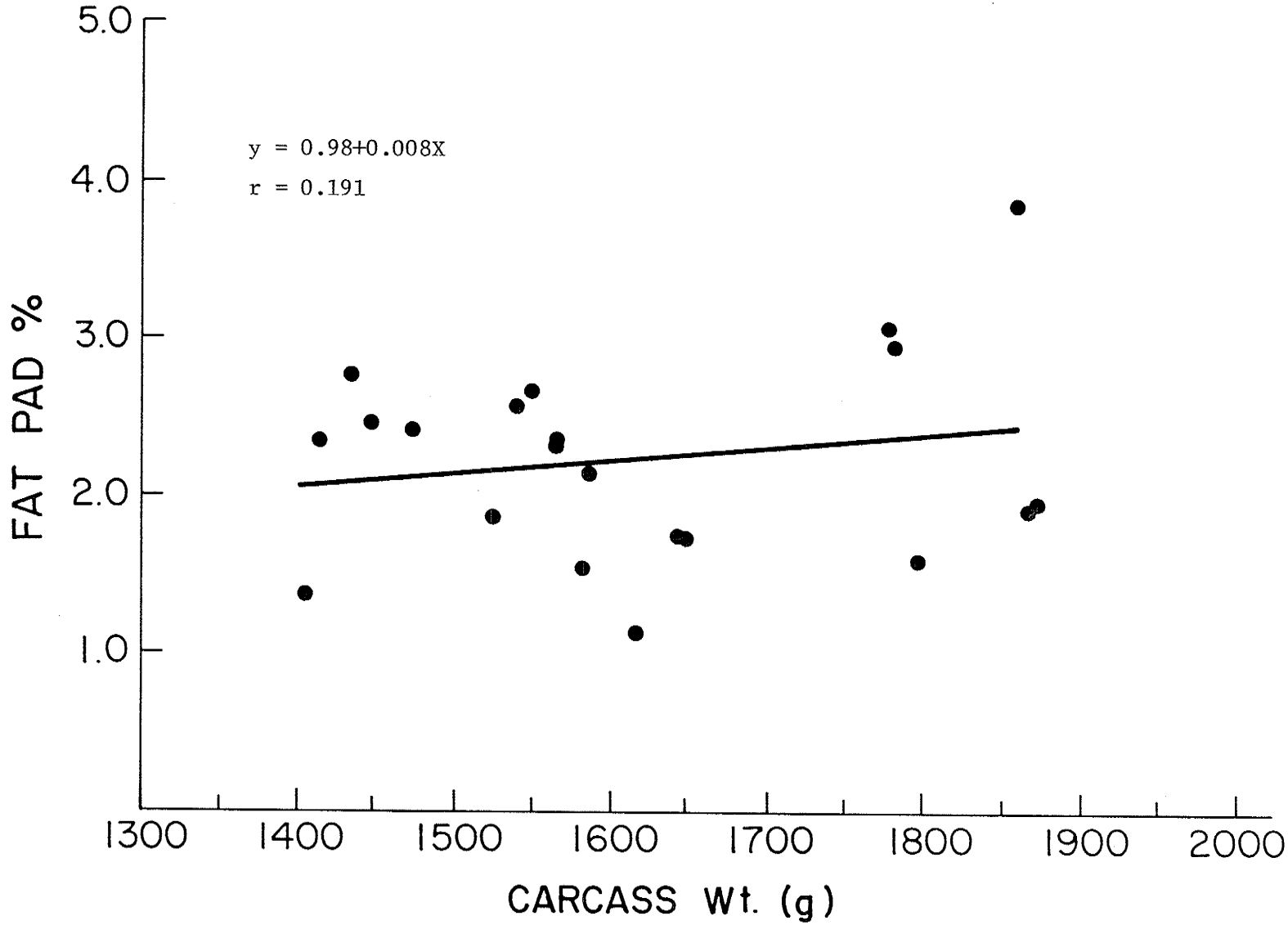

Figure 6. Relationship between fat pad percent and carcass weight (treatment 4) - Experiment 3. The data are based on 21 observations.

Neither treatment 3 nor treatment 4 exhibited any relationship between fat pad percent and carcass weight.

E. Caliper prediction of fat pad size

A significant ($P<0.01$), although small, correlation was observed between fat pad size (weight) and mean caliper reading. The correlation coefficient was $r = 0.3875$ indicating that 15% of the differences in caliper readings could be attributed to differences in fat pad size. The relationship between mean caliper reading and fat pad weight is presented in Figure 7.

DISCUSSION

A. Mean body weights and weight gains

The most dramatic effects of fat restriction on growth rate occurred during the first week of the experiment. The reduced growth rate of the restricted birds was likely due to the lower energy content of the ration resulting in chicks not being able to consume enough feed to meet their energy (and possibly protein) requirement. Scott et al. (1982) stated that the chicken can adjust feed consumption to attain maximum growth rate if dietary energy values range between 2,800 and 3,400 kcal ME/kg. The calculated energy of the fat deficient ration was 2,770 ME kcal/kg and consequently fell slightly short of the acceptable minimum. It is likely that protein deficiency was not a major contributing factor in reducing growth as the 24% protein content of this ration is considered adequate in starter rations containing over 3,200 kcal ME/kg, (Scott et al. 1982).

A third factor that may have contributed to a relatively slow rate

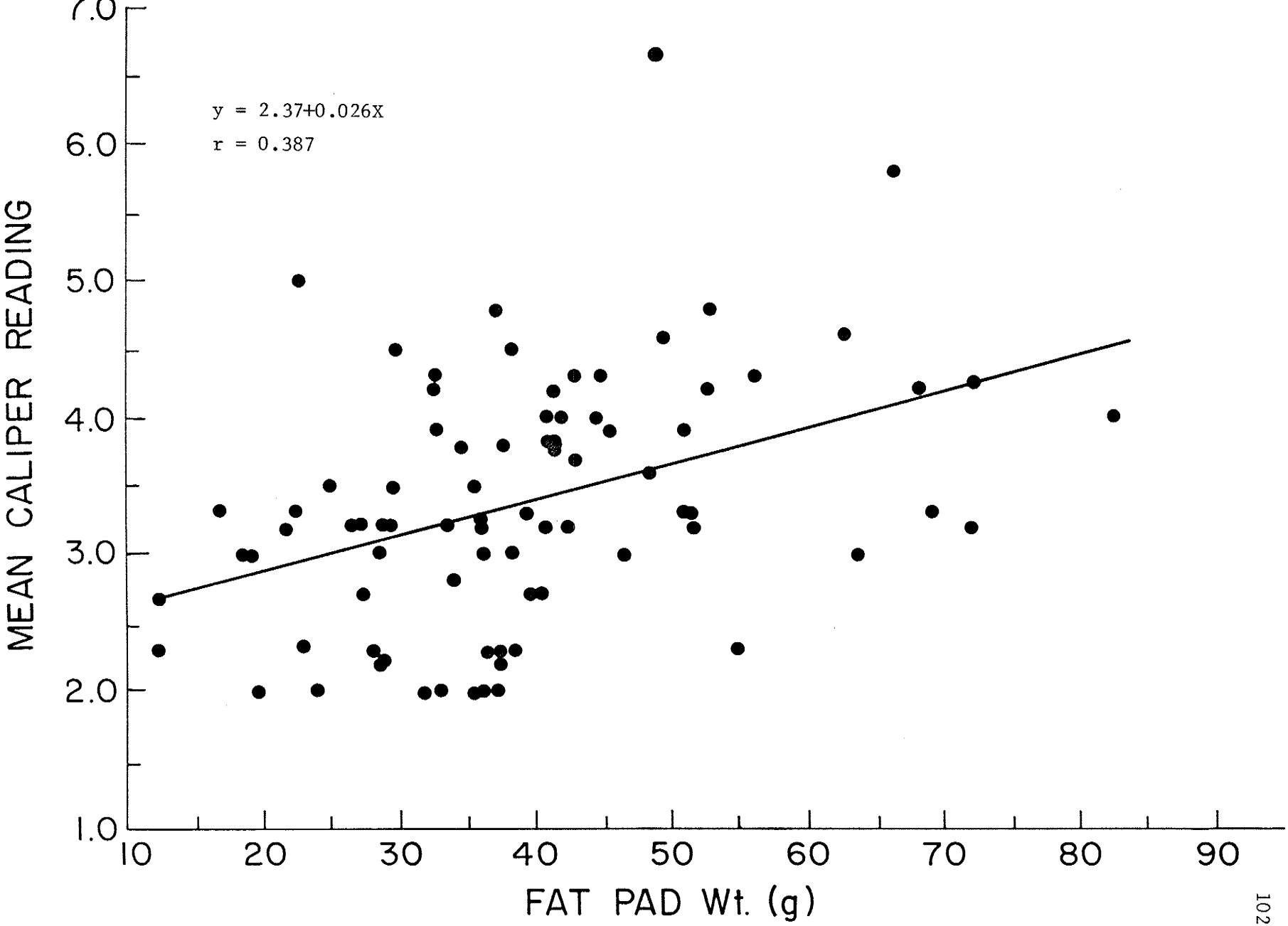

Figure 7. Relationship between mean acliper reading and fat pad weight - Experiment 3. The data are based on 84 observations.

of gain is that the fat restricted ration would have been marginal in linoleic and arachadonic acids, as wheat and soybean meal are not good sources of these compounds, and a deficiency of these essential fatty acids can impair growth (Scott et al. 1982).

From 1-4 weeks of age, rate of gain was not significantly different among treatments. Body weights of the restricted birds measured at 4 weeks were still significantly less than weights of the nonrestricted birds, however, the differences were not as great as at the end of the initial week. By 7 weeks of age there were no significant treatment differences.

The fact that final body weights were not significantly different indicated that the birds in this experiment were capable of compensatory growth. Marks (1979) stated that early restriction programs may not be practical in broilers as the chicken reaches market weight at an early age and there may not be sufficient time for the birds to fully compensate from the effects of early growth restriction. Ried and White (1978) suggested that the severity of undernutrition and the length of time it is imposed also affect the ability of the bird to compensate. The results of Experiment 3 showed that reducing the fat (and thus energy) content of the diet for one week still allows the bird to compensate within 6 weeks following the removal of the restriction. Further evidence that the length of the restricted periods is critical to the ability of the chick to compensate was provided by the results of Experiment 2 in which the birds were restricted for 2 weeks and did not fully compensate by 7 weeks of age.

B. Feed:gain ratios

The deletion of added dietary fat from the ration resulted in significantly poorer feed conversion during the first week of the trial. Reduced feed efficiency was attributed to the low energy content of the fat restricted ration. Sunde (1956) showed that a high protein ration low in energy reduced feed efficiency. Carew and Hill (1958) and Dam et al. (1959) reported that heat increment was reduced when fat was added to the diet and the energy saved contributed to improved feed:gain. Numerous other authors have reported that low energy content results in poorer feed conversion. Forbes and Yohe (1955) and Matterson et al. (1955) stated that adequate dietary energy levels improved the utilization of dietary protein while Waldroup et al. (1976) found that feed:gain improved with increasing energy values up to a level of 3,630 kcal ME/kg after which point no further improvements in feed:gain occurred.

When the restricted birds were switched to a ration containing added fat (and thus increased energy) feed conversion was found to be significantly better from 1-2 weeks than that of the nonrestricted birds. Although not significant, the improved feed conversion tended to continue from 2-4 weeks. Overall (0-4 weeks) the feed:gain ratio was not significantly ($P>0.05$) different among treatments as the poor initial feed conversion was compensated for by the improvements in feed:gain from 1-4 weeks of age. The overall pattern of feed:gain is similar to reports of Marks (1979) and Moran (1979) who found that the net result of dietary restriction was that overall feed:gain ratios were not significantly different between restricted and nonrestricted birds.

From 4-7 weeks, protein level was found to significantly affect feed:

gain ratios in that the high protein groups (narrow C:P ratio) were found to be more efficient than the lower protein (wide C:P ratio) groups. The observation that increased C:P ratio results in poor feed utilization is supported by the results of Scott et al. (1955) who reported decreased feed efficiency when C:P ratios were increased even when the energy level was adequate to maintain growth rates. Similar effects of protein (C:P ratio) on feed conversion have since been reported by Bartov et al. (1974a), Waldroup et al. (1976) and Griffiths et al. (1977a).

C. Mortality

During the starting period of 0-4 weeks, no treatment differences in total or SDS mortality were observed suggesting that fat restriction for the first 7 days of life did not affect the thriftiness of the bird. Hargis and Creger (1980) employed fat restriction for up to 14 days and did not report any increase in mortality attributable to fat restriction. The diets used in this trial did, however, contain substantial percentages of corn so an essential fatty acid deficiency that could affect mortality, (Scott et al. 1982) was less likely to occur.

Similarly, SDS incidence from 0-4 weeks was not affected by fat restriction. Because reduced growth rates only occurred during the first week and SDS does not begin before 7 days of life (Hulan et al. 1980; Gasperdone 1981), the effect of depressed growth or SDS would not have been apparent. Since rate of gain was not significantly different from 1-4 weeks of age between restricted and nonrestricted birds differences in SDS incidence due to differences in growth rate would not have been expected.

From 4-8 weeks of age causes of mortality, other than SDS, were not significantly affected by protein level or fat restriction.

There was, however, a significant reduction in SDS mortality attributed to the increased protein level of the finisher diet. Similar research on the effect of increased protein in the finisher diet on SDS incidence has not been reported.

The reduction in SDS mortality seen in this trial may have been related to the observation that the birds fed the high protein finisher tended to be leaner than the birds fed the lower protein ration. It may be hypothesized that differences in fat metabolism and deposition could be related to SDS. Riddell and Orr (1980) found elevated total plasma lipids in some SDS individuals suggesting the possibility of altered fat metabolism. Furthermore, a reduction in fat content of the bird may reduce stress on the heart and subsequently affect SDS mortality. Finally, high protein diets have been found to reduce serum cholesterol levels (Yeh and Leveille 1973) and it may be that elevated serum cholesterol or other lipid parameters are involved in SDS.

D. Dressing percent and fat pad percent

Fat restriction from 0-1 week was found to have no effect on dressing percent. Differences would not be expected as 7 week body weights were similar. Changes in the size of the GI tract, sometimes associated with nutrient restriction, (McCartney and Brown 1977) and found to influence dressing percent likely did not occur and thus did not influence dressing percents.

Fat pad percentages measured at 7 weeks of age were not significantly

affected by either early fat restriction or by protein level (C:P ratio), although some trends were evident. Birds fed the high protein finisher tended to deposit less abdominal fat. Further evidence that high protein levels affected fat deposition was provided by regression analysis. In groups fed the lower protein finisher (although only Treatment 2 showed a significant correlation) percent abdominal fat tended to increase as the carcass weight increased. In groups fed the higher protein level, no relationship was shown between fat pad percent and carcass weight. The observation that in the low protein groups abdominal fat percent tended to increase with increasing carcass weight indicated that as the birds grew larger less protein and more fat was being deposited. Feeding a high protein finisher however, allowed the birds to deposit comparatively more protein and less fat even as body weight increased.

Much of the published research on fat deposition indicated that increasing the protein level (decreasing the C:P value) of the ration reduced the deposition of abdominal fat (Summers et al. 1965; Bartov et al. 1974a; Griffiths et al. 1977a). The fact that increased protein levels did not significantly reduce abdominal fat deposition in Experiment 3 may be explained in terms of body weight and individual variation of fat pad percents.

The tendency for fat deposition to increase with age and consequently, in the case of broilers, with weight (Edwards and Hart 1971; Hood 1982) may be responsible for the effect of protein on fat deposition in Experiment 3 not being significant. Bartov et al. (1974a) found that differences in abdominal fat percent that were present at 7 and 8 weeks of age were no longer apparent by 9 weeks of age and stated that C:P

ratios could only affect fat deposition to a certain age (weight). Average 7 week body weights from Experiment 3 were comparable to average 10 week weights listed by Bartov and considerably heavier than 7 or 8 week weights reported by authors that have found reductions in fat pad deposition attributed to increased protein levels, (Griffiths et al. 1977a; Hargis and Creger 1980). Thus the birds sampled in Experiment 3 were physiologically older than those tested in experiments cited and likely past the physiological point suggested by Bartov where C:P ratio does not affect fat deposition. The tendency for the high protein groups to have less abdominal fat at 7 weeks of age may have been reflecting significant differences that were present at an earlier age and a lighter body weight.

There was also a tendency for the fat restricted birds to deposit more abdominal fat than the nonrestricted birds, that is within each protein level tested, the restricted birds were found to deposit slightly greater amounts of abdominal fat than the nonrestricted birds. These results contrast with those of Hargis and Creger (1980) who found that the elimination of added dietary fat from 0-7 days resulted in a significant decrease in abdominal fat production. This discrepancy may be explained in terms of compensatory growth.

In Experiment 3, there was no significant difference among treatments with regard to 7 week body weights indicating that the birds in this trial were capable of compensatory growth. Hargis and Creger (1980) found that the restricted birds in their study were significantly lighter at 7 weeks than the nonrestricted birds and thus did not demonstrate compensatory growth. Deaton et al. (1973) found that birds showing compensatory

ability had greater fat deposition at market age than did birds that were not restricted. Similarly, Beane et al. (1979) reported increased abdominal fat in male birds that demonstrated compensatory growth even when the final body weights were significantly less than nonrestricted controls.

Therefore it appears that reductions in abdominal fat deposition by eliminating added dietary fat from the ration may depend on the strain of bird. Cherry et al. (1978) also found that growth restriction could increase or decrease abdominal fat depending on the population. Although these authors did not show a significant relationship between abdominal fat and compensatory growth, it seems likely that elimination of added dietary fat for 7 days will not decrease abdominal fat deposition in birds that demonstrate compensatory ability. However, in slower growing birds that cannot fully compensate for early growth depression, fat restriction may prove an effective method of reducing abdominal fat deposition.

E. Caliper predictions of abdominal fat

Genetic selection as a method of reducing abdominal fat in broilers has not been used extensively as direct selection requires the slaughter of birds to determine abdominal fat content and the use of sib-selection (Pym and Thompson 1980). Therefore, a method to determine abdominal fat that does not sacrifice the individual tested would be beneficial to breeding programs aimed at reducing fat deposition.

The calipers used in Experiment 3 were designed and employed according to descriptions provided by Pym and Thompson (1980). These authors reported a significant correlation ($r = +0.80$) between the amount

of abdominal fat present in the bird and caliper measurements. Mirosh and Becker (1983) modified the design of Pym and Thompson and also found a significant correlation ($r = +0.54$) between fat pad size and caliper measurements taken at 48 days of age.

The results obtained in Experiment 3 showed a much smaller correlation ($r = +0.39$) than those of Pym and Thompson (1980) and Mirosh and Becker (1983). The calipers used in Experiment 3 were very stiff and insensitive making it difficult to determine when to stop squeezing the caliper together. Measurements were thus difficult to duplicate. Hand fatigue was also a problem. An improved design that would make the instrument more sensitive and flexible might have improved the effectiveness of the calipers.

Mirosh and Becker (1983) suggested other factors that can affect caliper predictions of abdominal fat. Accuracy may be affected by differences in abdominal skin thickness and membranes covering the intestines, which are often in the line of measurement.

The caliper technique does seem to hold promise as a predictor of abdominal fat although further refinements in design and interpretation of measurements are required.

IV. The effects of high doses of niacin in broiler diets on total serum lipids, triglycerides and cholesterol. (Experiment 4).

RESULTS

Mean body weights, weight gains and feed:gain ratios during the 4 week experimental period are presented in Table 18. Mean squares from analysis of variance tables are reported in Appendix 10.

A. Mean body weights and weight gains

The addition of niacin (nicotinic acid) to the diet at levels of 50, 150 or 300 times the NRC requirement for the vitamin had no significant effect on rate of gain and, consequently, body weights at either 2 or 4 weeks of age. Mean body weights at 2 weeks ranged from 319 g to 335 g and at 4 weeks ranged from 837 g to 904 g. These weights were typical of commercial broilers raised in battery brooders.

B. Feed:gain ratios

No treatment effects were observed for any of the calculated periods. From 0-2 weeks, feed:gain ratios ranged from 1.29 to 1.34; from 2-4 weeks values ranged from 2.06 to 2.28 while 0-4 week ratios ranged from 1.82 to 1.92.

C. Total serum lipids, triglycerides and cholesterol

Mean total serum lipids, triglycerides and cholesterol values are listed in Table 19. Mean squares from analysis of variance tables are reported in Appendix 10.

Average total lipid concentrations ranged from 435 mg/dl to 477

Table 18. Mean body weights, weight gains and feed:gain ratios of male broilers - Experiment 4

Parameters	Periods (weeks)	Dietary treatment[1,2] T_1	T_2	T_3	T_4
Mean body weight (g)	0	47.0	47.0	47.0	47.0
	2	335±6.9	319±12.8	319±8.5	325±5.8
	4	903±19.9	837±16.7	853±22.5	864±10.6
Mean weight gain (g)	0-2	288±6.9	272±12.8	272±8.5	278±5.8
	2-4	568±16.5	518±22.4	534±19.5	539±7.7
	0-4	856±19.9	790±16.7	806±22.5	817±10.6
Feed:gain	0-2	1.34±.05	1.31±.03	1.29±.02	1.30±.03
	2-4	2.06±.06	2.28±.13	2.16±.07	2.09±.09
	0-4	1.80±.05	1.92±.07	1.86±.04	1.82±.06

[1]Treatment groups were as follows: T_1 - control, T_2 - 50x niacin, T_3 - 150x niacin, T_4 - 300x niacin.

[2]There were no significant differences among treatments.

Table 19. Mean 4 week total serum lipids, triglycerides and cholesterol of male broilers - Experiment 4

Parameters	Dietary treatment			
	T_1	T_2	T_3	T_4
Total lipids (mg/dl)	$477\pm29.6_A$[1]	$468\pm21.7_A$	$435\pm14.5_A$	$457\pm11.4_A$
Triglycerides (mg/dl)	$194\pm17.5_A$	$166\pm9.7_A$	$122\pm7.7_B$	$122\pm5.2_B$
Cholesterol (mg/dl)	$174\pm12.8_A$	$171\pm9.1_A$	$135\pm7.4_B$	$145\pm3.6_{AB}$

[1]Means within a row not followed by the same subscript are significantly different at (A,B) $P<0.01$.

mg/dl, however these differences were not significant ($P<0.05$).

The addition of niacin to the diet at either 150 or 300 times the NRC requirement resulted in a significant reduction in serum triglycerides. However at 50 times NRC requirement levels no effect on serum triglycerides ($P<0.05$) concentration was observed. Values for the control treatment and the group that received 50 times the requirement levels were 194 mg/dl and 166 mg/dl respectively. A mean value of 122 mg/dl was obtained for both groups fed the higher levels of nicotinic acid.

Addition of niacin at 150 times the NRC requirement significantly ($P<0.01$) reduced mean serum cholesterol concentrations, yielding a value of 135 mg/dl. Niacin added at 300 times the requirement level also produced a value lower than that of either the control treatment or the group fed niacin at 50 times requirement levels but the difference was not significant. There was also no significant difference between cholesterol concentrations for 150 and 300 times requirement levels. Values for the control group, 50 and 300 times niacin requirement were 174 mg/dl, 171 mg/dl and 145 mg/dl respectively.

DISCUSSION

The effects of pharmacological doses of nicotinic acid have not been studied in the chick. Much research, however, has been conducted on the effects of mega doses of niacin in man. The purpose of this experiment was to determine if the effects of niacin observed in humans were similar in the chick.

A. Mean body weights, weight gains and feed:gain ratios

In humans, niacin has a limited toxicity of 1-4 g/kg of body weight with considerable individual variations, (Kutsky 1973). Indications of an overdose include burning and itching skin caused by vasodilation, increased pulse and respiratory rate and fatty liver. Scott et al. (1982) suggested that feeding excess nicotinic acid to chicks would produce similar reactions to those seen in man as well as possibly reduced growth rates.

The results of Experiment 4, however, indicated that side effects present in man did not occur in the chick and large doses of niacin are well tolerated. The behaviour of the niacin treated birds was normal in that they did not appear hot or excitable. The presence of niacin in the feed did not depress intake by the birds as feed was readily accepted and at no time were the chicks off feed. Feed:gain ratios were not significantly different from the control birds. Water consumption was also not affected.

Niacin at high levels did not have any growth depressing effect as suggested by Scott et al. (1982). Rate of gain and final body weights were not significantly different among treatments. It is possible that there may be a threshold level that would reduce growth and cause other side effects but this limit was not exceeded by dose levels used in this experiment.

B. Blood lipid parameters

In man, pharmacological doses of nicotinic acid have been shown to affect various serum lipid concentrations. Altschul et al. (1955) first discovered that oral doses of between 3 to 9 grams/day of niacin could

lower plasma cholesterol concentrations in man. Since that time several clinical studies have shown that plasma cholesterol and triglyceride values can be lowered by using nicotinic acid given in large doses (Altschul 1958; Carlson et al. 1969; Schlierf and Hess 1977). Reductions in cholesterol of 10-25% and reductions in triglycerides of 23-46% have been recorded (Wahlquist 1981).

To the knowledge of this author, similar research on the effect of nicotinic acid on blood lipid parameters in the chick has not been reported. The results of Experiment 4 indicate, however, that nicotinic acid is capable of reducing serum cholesterol and triglycerides in the chick. The reductions in these parameters were not reflected in total lipid values however although total serum lipids tended to be less in the 150 and 300 times niacin requirement groups.

The dose required to lower triglyceride and cholesterol concentrations in this experiment fell somewhere between 50 and 150 times the requirement level. The larger dose of 300 times the requirement was no more effective than 150 times indicating that an optimum level exists above which no further cholesterol or triglyceride reduction occurs.

The values obtained for total lipids (average values ranging from 435 mg/dl to 477 mg/dl) were similar to values reported in the literature. Rudas et al. (1972) listed total serum lipid values obtained from unsexed white Leghorn chicks ranging in age from 1-15 weeks as being between 420 mg/dl and 480 mg/dl. Leclerq et al. (1974) sampled broiler type pullets and found that total serum lipids averaged 572 mg/dl.

Cholesterol levels determined in Experiment 4 ranged from an average of 135 mg/dl to 174 mg/dl (treatment 3 and 1 respectively). Similarly,

a wide range of values has been reported in the literature. Estep et al. (1969) sampled cockerels ranging in age from 1-15 weeks and reported cholesterol concentrations in plasma ranging from 83 mg/dl to 132 mg/dl. Marion et al. (1961) found that, depending on diet, cholesterol levels obtained from 4 week old chicks raised in battery brooders ranged from 121 mg/dl to 531 mg/dl.

Triglyceride values determined in Experiment 4 were considerably higher (ranging from an average of 122 mg/dl in treatments 3 and 4 to 194 mg/dl in treatment 1) than values presented by Nir et al. (1973) which were obtained from cockerels and averaged slightly less than 100 mg/dl. That plasma triglyceride concentrations can be higher and exhibit large variations was demonstrated by Bartov et al. (1974b). These authors sampled broilers raised in cages to 8 weeks and found triglyceride levels to range from 112±22 mg/dl to 139±54 mg/dl.

Comparing plasma cholesterol and triglyceride levels with various reports is difficult as these parameters can be influenced by age, strain and environment (Sturkie 1976). Other factors that account for variation include sex and diet.

C. Possible mode of action of nicotinic acid

The results of Experiment 4 indicated that nicotinic acid can have the same effect on serum (plasma) cholesterol and triglycerides in the chick as it does in man. It therefore seems likely that the mode of action is similar in both species.

The reduction of plasma cholesterol and triglyceride concentrations resulting from niacin therapy appears to be due to an antilipolytic

effect. That nicotinic acid can be antilipolytic was demonstrated by Carlson (1971) and Carlstrom and Lavrell (1968). Although the mechanism is not completely understood, the fact that nicotinic acid inhibits adenyl cyclase activity and thus cyclic AMP formation in adipose tissue may explain the observed antilipolytic effect (Anderson et al. 1973). Because of reduced lipolysis, the flux of free fatty acids (FFA) to the liver is reduced and consequently hepatic production of very low density lipoprotein (VLDL) is diminished, triglycerides being a major component of VLDL. The reduction in plasma triglyceride caused by nicotinic acid, therefore, may be due to the effect of niacin on adipose tissue (Carlson 1977).

Furthermore, very low density lipoprotein is a precursor of low density lipoprotein (LDL). Studies by Levy and Langer (1972) showed that nicotinic acid did lower LDL by reducing VLDL synthesis and that reduced LDL levels were not a consequence of increased catabolism. As LDL is the major cholesterol bearing lipoprotein (Jackson et al. 1976), reduction in LDL synthesis would lower plasma cholesterol. Nicotinic acid may inhibit cholesterolgenesis (Walquist 1981) although this point is disputed (Kritchevsky 1971; Miettinen 1971). Kritchevsky and Tepper (1962) reported that nicotinic acid may increase cholesterol oxidation resulting in decreased cholesterol levels.

Nicotinic acid in pharmacological doses can also affect plasma levels of chylomicrons and high density lipoprotein (HDL). Niacin has been found to raise the activity of lipoprotein lipase (LL), an enzyme that splits fatty acids off triglyceride molecules, and to increase the rate of incorporation of fatty acids into adipose tissue (Otway et al. 1971;

Nikkila 1971). The increased activity of LL and incorporation of FFA into adipose tissue may then result in a stimulation of the catabolism of chylomicrons, thus explaining the lowered plasma chylomicron concentrations associated with nicotinic acid therapy (Carlson 1977). With increased LL activity and enhanced triglyceride clearance (via catabolism of chylomicrons and VLDL), HDL cholesterol concentration has been found to rise and may be a secondary occurrence (Wahlquist 1981).

In summary, nicotinic acid could effect plasma lipids in the following manner:

1) Inhibition of lipolysis results in a reduced flux of FFA to the liver reducing the synthesis of VLDL.
2) A reduction in hepatic VLDL production is reflected in lowered plasma triglycerides as VLDL is a main source of plasma triglyceride.
3) A decrease in VLDL synthesis reduces LDL synthesis as VLDL is a precursor of LDL.
4) Lowered LDL production reduces plasma cholesterol as LDL is the principal cholesterol bearing lipoprotein.
5) Nicotinic acid may also reduce plasma triglycerides by increasing LL activity and the rate of incorporation of fatty acids into adipose tissue thus enhancing the catabolism of chylomicrons and VLDL.
6) Cholesterol levels may also be reduced by niacin inducing oxidation of cholesterol and/or reducing its synthesis.

V. The effects of niacin and calorie:protein ratios on the incidence of Sudden Death Syndrome (SDS). (Experiment 5).

RESULTS

Mean body weights, weight gains and feed:gain ratios for the 7 week experimental period are presented in Table 20. Mean squares from analysis of variance tables are reported in Appendix 11.

A. Mean body weights and weight gains

The addition of niacin to the starter ration at 150x the NRC requirement had no significant effect on body weight measured at 4 weeks of age with average weights ranging from 895 g to 906 g. Protein level and niacin level of the finisher diet also did not affect weight gains from 4-7 weeks and consequently there were no significant differences in 7 week body weights among the 4 treatment groups. Seven week body weights ranged from 2,237 g to 2,269 g.

B. Feed:gain ratio

The addition of niacin to the starter ration at 150x the NRC requirement for niacin had no significant effect on feed conversion from 0-4 weeks. Calculated values ranged from 1.61 to 1.65. Similarly, the addition of niacin to the finisher ration at 150x requirement had no effect on feed:gain ratios from 4-7 weeks. Protein level of the finisher diet tended to influence feed conversion from 4-7 weeks of age in that the treatment groups fed the higher protein ratio (24% vs 19%) were more feed efficient. The effect of protein level on feed:gain ratios was approaching significance at $P<0.05$. Feed:gain ratios from 4-7 weeks were 2.10 and 2.11 for treatments 4 and 2 (24% protein finisher) respectively and 2.15

Table 20. Mean body weights, weight gains and feed:gain ratios of male broilers - Experiment 5

Parameters	Periods (weeks)	Dietary treatment[1,2]			
		T_1	T_2	T_3	T_4
Mean body weight (g)	0	39.0	39.0	39.0	39.0
	4	914.0±7.3	895.0±7.7	908.0±7.0	896.0±6.8
	7	2269±19.3	2237±13.1	2244±10.2	2244±11.4
Mean weight gain (g)	0-4	875.0±7.3	856.0±7.7	869.0±7.0	857.0±6.8
	4-7	1355.0±13.3	1342.0±14.3	1336.0±9.4	1348.0±8.4
	0-7	2230.0±19.3	2198.0±13.1	2205±10.2	2205±11.4
Average feed:gain	0-4	1.62±.01	1.64±.01	1.61±.01	1.63±.01
	4-7	2.14±.01	2.10±.02	2.15±.03	2.10±.02
	0-7	1.93±.01	1.91±.01	1.93±.01	1.91±.01

[1]Dietary treatments were as follows: T_1 - control - standard starter/finisher; T_2 - standard starter/high protein finisher; T_3 - standard starter plus niacin/finisher plus niacin; T_4 - standard starter plus niacin/high protein finisher plus niacin.

[2]There were no significant differences among treatments.

for both treatments 1 and 3 (19% protein finisher). Overall (0-7 week) feed:gain ratios were not significantly affected by niacin and protein levels and ranged from 1.91 to 1.93.

C. Mortality

Total and SDS mortality from the 7 week experimental period are presented in Table 21. Mean squares from analysis of variance tables are reported in Appendix 12.

At the end of the 4 week starting period, there was a significant ($P<0.05$) affect of niacin on total mortality. The niacin treated group had 41 (4.18%) birds culled or die as compared to a total mortality of 24 birds (2.45%) in the control groups. SDS mortality during this period tended to be greater in the niacin fed birds with the difference approaching significance at $P<0.05$. SDS mortality in this group totalled 18 individuals (1.84%) as compared to 8 birds (0.82%) in the control group.

From 4-7 weeks there were no significant differences among treatments for either total or SDS mortality. Total mortality ranged from 2.07% to 2.78% and SDS mortality ranged from 1.27% to 1.49%.

Although there were no significant differences among treatments with regard to overall (0-7 week) total mortality, there was a significant ($P<0.05$) affect of niacin, in that total mortality was increased in the niacin treated birds. There were no treatment effects with regard to overall SDS mortality however. Total mortality ranged from 3.47% to 7.14% with SDS mortality ranging from 2.04% to 3.67%.

Table 21. Mortality data, male broilers - Experiment 5

Periods (weeks)	Total no. mortality[2] (%)			
	Dietary treatment			
	$T_1 + T_2$		$T_3 + T_4$	
0-4	$24(2.45)_a$[1]		$41(4.18)_b$	
	T_1	T_2	T_3	T_4
4-7[3]	$10(2.07)_A$	$10(2.11)_A$	$12(2.55)_A$	$13(2.78)_A$
0-7	$17(3.47)_A$	$27(5.51)_A$	$31(6.33)_A$	$35(7.14)_A$

Periods (weeks)	Total no. SDS mortality (%)			
	Dietary treatment			
	$T_1 + T_2$		$T_3 + T_4$	
0-4	$8(0.82)_A$		$18(1.84)_A$	
	T_1	T_2	T_3	T_4
4-7[3]	$7(1.45)_A$	$6(1.27)_A$	$7(1.49)_A$	$6(1.28)_A$
0-7	$10(2.04)_A$	$11(2.24)_A$	$18(3.67)_A$	$13(2.65)_A$

[1]Means within a row not followed by the same subscript are significantly different at (A,B) $P<0.01$, (a,b) $P<0.05$.

[2]Includes culled birds.

[3]Percents are calculated by dividing the no. of mortalities by the no. of live birds at 4 weeks of age.

D. Blood lipid parameters

Mean total plasma lipids, triglycerides and cholesterol values measured at 4 and 7 weeks of age are reported in Table 22. Mean squares from analysis of variance tables are presented in Appendix 13.

The addition of niacin at 150x NRC requirement to the starter ration had no significant affect on total plasma lipids when measured at 4 weeks of age. Mean total lipids for the niacin treated birds were 395 mg/dl versus 401 mg/dl for the birds fed the control ration. Similarly, no significant affect of protein or niacin level of the finisher ration was observed for total lipids measured at 7 weeks of age. Values ranged from 415 mg/dl to 475 mg/dl.

The addition of niacin to the starter ration had no significant affect on plasma triglycerides at 4 weeks of age with concentrations ranging from 101 mg/dl to 123 mg/dl. Measured at 7 weeks of age, no significant treatment differences were observed although some trends were apparent in that both protein level and niacin seemed to reduce plasma triglycerides. The highest concentration was found to be in the control group, fed a lower protein diet (92 mg/dl) followed by the low protein plus niacin group (T_3) with an average value of 76 mg/dl. Treatment 2, fed a high protein finisher, produced a mean triglyceride concentration of 68 mg/dl while treatment 4, fed a high protein plus niacin diet, had mean levels of 58 mg/dl.

Measured at 4 weeks of age, no significant differences in cholesterol levels between niacin treated and control birds were observed. Similarly, there were no significant affects of protein level or niacin level of the finisher diet on cholesterol concentrations measured at 7 weeks of

Table 22. Mean total plasma lipids, triglycerides and cholesterol values of male broilers - Experiment 5

Parameters	Week 4[1] Dietary treatment	
	$T_1 + T_2$	$T_3 + T_4$
Total lipids (mg/dl)	402±21.7	395±18.8
Triglycerides (mg/dl)	108±10.8	123±8.9
Cholesterol (mg/dl)	138±7.7	125±8.5

Parameters	Week 7 Dietary treatment[1]			
	T_1	T_2	T_3	T_4
Total lipids (mg/dl)	464±25.1	475±60.3	415±18.7	421±23.2
Triglycerides (mg/dl)	92±20.7	68±8.1	76±12.6	58±2.9
Cholesterol (mg/dl)	140±13.1	124±8.9	133±9.1	126±13.0

[1]There were no significant differences among treatments.

age although the high protein treatments tended to have slightly reduced plasma cholesterol. The values for these groups (T_2 and T_4) were 124 mg/dl and 126 mg/dl respectively. The lower protein treatments (1 and 3) were found to have mean cholesterol concentrations of 140 mg/dl and 133 mg/dl respectively.

DISCUSSION

A. Mean body weights and weight gains

The addition of 150x NRC requirement for niacin to the starter and finisher ration was shown to have no significant affect on weight gain and consequently body weights at either 4 or 7 weeks of age. These results are in agreement with those of experiment 4 where no growth effects at 4 weeks were observed when niacin was added at 50x, 150x and 300x requirement levels. Feeding niacin for an additional 3 weeks indicated that the chick can tolerate a high level of the vitamin for an extended period of time. A further discussion of possible growth effects of niacin was presented in Experiment 4.

Growth rate from 4-7 weeks was not significantly affected by protein level (C:P ratio) suggesting that the lower level fed to treatments 1 and 3 was adequate to sustain maximum growth rates. Scott et al. (1955) and Donaldson et al. (1956) found that as long as dietary protein level was adequate in terms of the energy content of the ration, growth rates were not reduced. Thus the additional protein added to the finishing diets of treatments 2 and 4 was in excess of that required to achieve good growth rates although the high protein groups likely gained more lean

tissue than the birds offered the lower protein ration.

B. Feed:gain ratios

Niacin was found to have no significant effect on feed conversion for any of the calculated periods. Similar results were observed in Experiment 4 where niacin was found to not affect feed efficiency.

Protein content of the finishing diet was found to have a significant effect on feed:gain ratios in that increasing the protein content of the diet resulted in improved feed efficiency. Similar results were observed in Experiment 3 and confirmed by the reports of Scott et al. (1955) and Waldroup et al. (1976).

The improved feed efficiency due to increased protein may have been due to the higher protein diets containing excess amino acids that could have provided additional energy in the form of carbon skeletons. More likely, however, is that the higher protein diets contained a better balance of amino acids; Scott et al. (1982) stated that as dietary amino acid balance is improved, efficiency of utilization also improves.

C. Mortality

The effects of mega doses of nicotinic acid on total and SDS mortality in the chick have not been previously reported. The purpose of Experiment 5 was to determine if pharmacological doses of nicotinic acid could reduce the incidence of SDS by lowering plasma cholesterol, triglycerides and possibly free fatty acids (FFA) concentrations and also to attempt to replicate the results of Experiment 3 in which protein level of the finisher diet was found to affect SDS mortality.

i) Significance of reduced plasma lipids

In human medicine, the rationale for lowering various plasma lipid parameters such as cholesterol, triglycerides and FFA is the possible relationship between these factors and cardiovascular diseases. Studies by Carlson and Bottiger (1972) and Kannel et al. (1971) indicated a relationship between increased serum cholesterol and increased incidence of coronary heart disease. Similarly, studies by Carlson and Bottiger (1972) and Pelkonen et al. (1977) implicated serum triglycerides as a factor associated with cardiovascular disease. Carlson (1977) stated that elevated plasma FFA levels have been shown to induce cardiac arrythmias and increase the severity of acute myocardial infarction.

The rationale, therefore, for attempting to lower plasma lipid levels of the broiler chicken was: a) as chickens are prone to similar cardiovascular diseases as humans with similar relationships between arteriosclerosis and blood lipid levels (Sturkie 1976), reductions in plasma cholesterol, triglycerides and/or FFA may influence the occurrence of cardiovascular disease in the chicken, b) SDS appears to be a cardiovascular disease with the immediate cause of death being acute heart failure. Therefore, it was hypothesized that a reduction in vascular and heart damage, mediated by reduced plasma lipid levels, might lower the incidence of SDS.

The close resemblance of avian arteriosclerosis to that found in humans has been realized since the early part of this century (Pick and Katz 1965). In addition, that the causal factors of avian arteriosclerosis may be similar to those implicated in human studies of the disease was shown by Weiss et al. (1968) who found a degree of involve-

ment of blood lipid parameters in the development of spontaneous avian arteriosclerosis and by Kakita et al. (1972) who found that the elevation of plasma cholesterol to high levels induced arteriosclerosis in short term experiments. Physiological fatty streaking in vessels of young chicks (1-6 weeks of age) is not uncommon, although it is usually not apparent after 6 weeks of age (Petrak 1969).

There is some evidence to indicate that SDS is a cardiovascular disease. A number of researchers have reported or suggested circulatory and heart tissue damage in SDS mortalities. Volk et al. (1974) reported unspecified circulatory disorders with resulting regressive changes in the myocardium. Ononiwu et al. (1979a) and Steele et al. (1982) also observed degeneration of myofibers in a high percentage of the hearts of SDS birds. In addition, Riddell and Orr (1980) suggested that ultrastructural lesions of the heart could be a contributing factor in SDS. Page[1] (1982) found that in many SDS birds sampled, there was an abnormally large deposition of cholesterol and other fat in the coronary arteries. It was suggested that a reduced blood supply to the heart could be the cause of mortality and that diet was involved.

Death as a result of SDS very closely resembles the acute cardiac arrest that can occur in man and other animals. Observed events and postmortem examination support the hypothesis that acute cardiac arrest is the immediate cause of death. Birds succumbing to SDS were observed to run and twitch spasmodically while continuously gasping for breath.

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[1]Personal communication, Dr. K. Page is a field veterinarian in Georgia.

Death occurred within a minute, the affected individual would be off its feet, lying in the characteristic position associated with the syndrome. This behaviour is characteristic of acute cardiac arrest where an animal falls down and may thrash about for a brief period. Death is accompanied by deep gasps with exaggerated inspiratory efforts (The Merck Veterinary Manual 1979). Upon postmortem examination massive pulmonary congestion and edema are observed. Lung edema in the broiler then is likely as a result of heart failure and not related to the cause of the disease. Further evidence that lung edema is a postmortem artifact was provided by Riddell and Orr (1980) who found that recent SDS mortalities (post-mortemed immediately after death) did not exhibit pulmonary congestion and edema.

In summary, as chickens can be prone to cardiovascular disease similar to that of humans with similar relationships between arterio-sclerosis and lipid parameters such as cholesterol and because SDS birds show some indications of cardiovascular damage and appear to die of heart failure, it was hypothesized that lowering plasma levels of choles-terol, triglycerides and/or FFA by feeding large doses of nicotinic acid might reduce the incidence of SDS in the following manner.

A certain percentage of individuals in a flock may have high levels of cholesterol and triglycerides which could result in a degree of arteriosclerosis. The development of this condition can result in damage to the myocardium (myocardial infarction). Thus the heart may be predisposed to acute heart failure, especially during time of stress. Myocardial lesions can result in various types of arrythmias including ventricular fibrillation that occurs in acute heart failure, (The Merck

Veterinary Manual 1979). Plasma FFA levels may also contribute to SDS. As a result of stress, FFA levels increase and may contribute to a fatal reinfarction and/or arrythmias leading to acute heart failure. Carlson (1977) stated that elevated plasma FFA levels have been shown to increase the severity of acute myocardial infarction and also result in cardiac arrythmia.

ii) Calorie:protein ratio and nicotinic acid

The results of Experiment 5 were disappointing in that the reduced SDS mortality as a result of a high protein diet observed in Experiment 3 was not replicated in this trial. The effectiveness of increased protein in reducing SDS mortality is thus questionable. Similarly, nicotinic acid supplementation failed to significantly reduce plasma levels of cholesterol and triglycerides. Consequently a relationship between these parameters and SDS could not be determined. The fact that nicotinic acid significantly reduced plasma cholesterol and triglyceride levels in Experiment 4 but did not cause a reduction in these parameters in Experiment 5 may have been due to environmental conditions as the former trial tested caged birds and the latter sampled birds raised on the floor. Sturkie (1976) stated that blood lipid levels could be affected by numerous factors including environment. Other factors such as ration, age, strain and sex were similar between trials so were likely not a factor. The number of samples analyzed in both trials (7 per treatment) may have been insufficient to give a true indication of the effectiveness of nicotinic acid in reducing cholesterol and triglyceride especially as both parameters show great individual variation.

Niacin was found to significantly ($P<0.05$) increase total mortality

from 0-4 and 0-7 weeks. This may suggest that the level fed produced a toxic reaction, however, this seems unlikely. The higher mortality in the niacin treated birds from 0-4 weeks was due to a greater number of birds culled due to leg problems and other miscellaneous conditions that would not likely be affected by niacin. The difference in 0-4 week total mortality was great enough to influence 0-7 week total mortality as from 4-7 weeks there were no significant differences among treatments. Therefore, although there was a significant ($P<0.05$) effect of niacin on total mortality, it may be misleading to attribute this to high levels of the vitamin.

VI. The effects of sodium and potassium levels of broiler diets on the incidence of SDS. (Experiment 6).

RESULTS

Mean body weights, weight gains and feed:gain ratios for the 7 week experimental period are presented in Table 23. Mean squares from analysis of variance tables are reported in Appendix 14.

A. Mean body weights and weight gains

The addition of Na or K (as NaCl or KCl) to the diet at levels 1.3x the calculated amount in the control ration did not significantly affect body weights measured at 4 and 7 weeks of age. Final body weights for the control, high Na and high K groups were 2,079 g, 2,115 g and 2,072 g respectively.

B. Feed:gain ratios

The addition of NaCl or KCl to the diet had no significant affect on feed conversion for any of the periods tested. Overall feed:gain ratios (0-7 weeks) were 1.93, 1.96 and 1.92 for the controls, high Na group and high K group respectively.

C. Mortality

Total and SDS mortality are presented in Table 24. Mean squares from analysis of variance tables are reported in Appendix 15. By 4 weeks of age there were no significant treatment differences with regards to either total or SDS mortality.

From 4-7 weeks of age, however, the percent mortality of the high Na group was significantly ($P<0.01$) greater than the control group

Table 23. Mean body weights, weight gains and feed:gain ratios of male broilers - Experiment 6

Parameters	Periods (weeks)	Dietary treatment[1,2] T_1 (control)	T_2	T_3
Mean body weight (g)	0	45.0	45.0	44.0
	4	780±10.1	798±12.4	781±5.6
	7	2079±13.9	2115±13.4	2072±11.8
Mean weight gain (g)	0-4	735±10.1	753±12.4	737±5.6
	4-7	1299±12.9	1317±18.0	1291±12.9
	0-7	2034±13.9	2070±13.4	2028±11.8
Feed:gain	0-4	1.73±.01	1.73±.01	1.74±.01
	4-7	2.06±.03	2.08±.01	2.02±.03
	0-7	1.93±.02	1.96±.01	1.92±.02

[1]Treatment groups were as follows: T_1 - control, standard starter finisher; T_2 - standard starter plus NaCl/standard finisher plus NaCl; T_3 - standard starter plus KCl/standard finisher plus KCl.

[2]There were no significant differences among treatments.

Table 24. Mortality data, male broilers - Experiment 6

Parameters	Periods (weeks)	Dietary treatment[1] T_1 (control)	T_2	T_3
Total[2] no. of mortalities (%)	0-4	$18(4.28)_A$[1]	$19(4.52)_A$	$13(3.09)_A$
	4-7[3]	$7(1.74)_A$	$9(2.24)_A$	$5(1.47)_A$
	0-7	$25(5.95)_A$	$28(6.66)_A$	$19(4.74)_A$
Total no. of SDS mortalities (%)	0-4	$9(2.14)_A$	$6(1.43)_A$	$5(1.19)_A$
	4-7[3]	$2(0.50)_A$	$7(1.74)_B$	$3(0.74)_{AB}$
	0-7	$11(2.62)_A$	$13(3.10)_A$	$8(1.91)_A$

[1]Means within a row not followed by the same subscript are significantly different at (A,B) $P<0.01$.

[2]Includes culled birds.

[3]Percents were calculated by dividing the no. of mortalities by the no. of live birds at 4 weeks of age.

(1.74% SDS as compared to 0.50%). The high Na group also tended to have a greater number of SDS mortalities than the high K group with the difference approaching significance. SDS mortality in the K group (0.74%) was not significantly different from that of the control treatment.

From 0-7 weeks, SDS mortality was not significantly different among treatments. The highest percent of SDS was in the increased Na group with 3.10% of the original population being affected. Percent SDS mortality for the control and high K groups were 2.62% and 1.91% respectively.

Total mortality from 4-7 and 0-7 weeks was not significantly affected by treatment with percent total mortality ranging from 4.74% to 6.66%

D. Heart analysis

Mean copper, potassium, sodium and calcium levels (dry matter basis) of the sampled hearts are presented in Table 25.

i) Potassium

No significant difference in potassium content was observed between the hearts of SDS birds and the hearts of culled birds, yielding mean values of 1.00% and 1.07% respectively.

ii) Sodium

No significant difference in sodium levels of the hearts was observed with mean values being 0.61% and 0.64% for the SDS birds and the culled birds respectively.

iii) Calcium

As with potassium and sodium, no significant difference in heart levels of calcium were found between SDS birds and the culls. The mean values for the former group was 400 µg/g and 441 µg/g for the latter

Table 25. Mean potassium, sodium, calcium and copper content of heart tissue (dry matter basis) - Experiment 6

Parameters	SDS birds	Culled birds
Potassium (%)	$1.00\pm0.03_a$	$1.07\pm0.06_a$
Sodium (%)	$0.61\pm0.01_a$	$0.64\pm0.04_a$
Calcium (µg/g)	$400\pm37_a$	$441\pm51_a$
Copper (µg/g)	$4.97\pm0.90_a$	$7.35\pm0.83_b$

Means within a row not followed by the same subscript are significantly different at $P<0.05$.

group.

iv) Copper

A significant difference ($P<0.05$) in the copper content of the hearts was observed. The SDS birds were found to have a mean heart copper content of 4.97 µg/g as compared to 7.35 µg/g in the culled individuals. This difference was calculated to be approximately 32%.

DISCUSSION

A. Mean body weights and weight gains

Mongin and Sauveur (1977) found that maximum growth response with respect to acid base balance occurred when the equation $(Na_{meq} + K_{meq}) - Cl_{meq}$ equalled 250 $_{meq}$ with a range of approximately 200-300$_{meq/kg}$ of diet. The values determined from the feed analysis for the diets fed in this experiment ranged from 233$_{meq}$ to 258$_{meq/kg}$ of diet (starter) and 181 to 252$_{meq/kg}$ of diet in the finisher and thus were generally within the range suggested. The fact that the levels of Na, K and Cl were found to be higher than calculated amounts should not have been of consequence as these amounts were well within recommended levels suggested by Scott (1982). Differences between calculated and actual Na, K and Cl content were likely due to the calculations being based on values taken from NRC tables which represent average levels, whereas local ingredients were used in the formulation.

Although the increase in Na content of the starter ration was found to be greater than intended (2 times as compared to 1.3 times control levels), this observation is not necessarily critical as the purpose of

the trial was to investigate the affect of additional Na on SDS mortality. Furthermore, the increase in Na of the finisher diet fed to the high Na group was close to the calculated increase.

Potassium increases were fairly consistent among treatments and rations although the chlorine concentration of the finishing diet fed to treatment 3 was unexpectedly low. However, the increased chlorine did not alter the acid-base balance to the extent that the calculated value was outside the acceptable range proposed by Mongin and Sauveur (1977) and growth was not affected.

Thus, increasing the Na content of a standard broiler diet by up to a factor of 2 and increasing the K content of the diet by 30% does not affect growth rates if the elements are added as chlorides and net acid base balance maintained within suggested ranges.

B. Feed:gain ratios

Feed conversion was not affected by additional NaCl or KCl. This is in agreement with the results of Mongin and Sauveur (1977) who found that feed efficiency was not affected if acid base balance was maintained.

Hurwitz et al. (1973) found that, with constant K levels, an excess of chlorine reduced body weights by decreasing feed intake but did not affect feed efficiency in young birds. However, Hurwitz et al. (1974) found that excess chlorine in older birds reduced body weight and feed efficiency. Feed conversion was not affected by increasing chlorine levels (as a result of adding NaCl or KCl) indicating that increased chlorine concentrations were offset by the additional Na or K and that the depressing effect of chlorine on feed efficiency depends on the cation content (either Na or K) of the diet.

C. Mortality

Total mortality was not significantly affected by dietary treatment. Although mortality has been found to increase when NaCl intake is high, levels of over 5.0% of the diet are required (Scott et al. 1982). The maximum NaCl content fed (treatment 2) was 0.23% added NaCl and thus well under the percent required to affect mortality.

The effects of additional NaCl or KCl in broiler diets on the incidence of SDS have not been reported although Hulan et al. (1980) suggested that electrolyte levels could be involved with the syndrome.

Sodium was investigated due to its affect on blood pressure (Ganong 1975) and because of the sequence of events in SDS proposed by Ononiwu et al. (1979a) who suggested that SDS could be a result of circulatory lesions increasing the permeability of the circulatory system. This permeability, caused by increases in blood pressure, was suggested to be usually reversible but when the stimulus surpasses the tolerance level, vessel permeability is irreversible and the tissue they supply (i.e. the heart) is damaged. The authors further proposed that the cause of death was heart damage leading to lung edema which rendered the bird unable to breathe. Thus, if increased permeability of blood vessels (and eventual death) is related to blood pressure, NaCl could be of concern. If blood pressure is related to SDS, sex differences in SDS could be partially explained as males have been found to have higher blood pressure than females (Sturkie 1976).

SDS mortality was found to be significantly ($P<0.05$) increased from 4-7 weeks in the group fed the higher levels of NaCl (treatment 2). As SDS mortality from 0-4 weeks was slightly less in this group as compared

to the controls, the relative increase in SDS from 4-7 weeks may suggest that prolonged feeding of a high NaCl diet affects the incidence of SDS possibly by affecting blood pressure as suggested by Ononiwu et al. (1979a). From 0-7 weeks, SDS was not significantly greater in the high NaCl fed birds although this group had the greatest percent SDS mortality.

The effects of potassium on SDS mortality have similarly not been reported. Potassium was studied due to its involvement in heart (muscle) function. Potassium is required for normal heart activity, reducing the contractility and favouring relaxation (Scott et al. 1982). As SDS hearts are often tightly contracted, the possible involvement of K was suggested. In addition, low levels of serum K have been found to increase cardiac arrythmia (Walquist 1981). Furthermore, K is usually considered to be adequate in broiler rations and therefore not added (Hurwitz et al. 1973).

There was a slight, although non significant reduction in SDS in the group fed supplemental KCl. However as SDS was not significantly reduced, increasing the K level of the diet by 30% did not seem to be an effective measure. Either this amount of K in the ration was not high enough or this element is not involved in the occurrence of SDS.

D. Heart analysis

Further evidence that neither Na or K is involved in SDS was provided by the heart analysis for Ca, Na, K and Cu as there were no significant differences in the heart content of either sodium or potassium. Similar conclusions can be drawn with regard to calcium as there was no difference in Ca content of SDS and cull hearts.

There was, however, a significant difference in copper levels of the hearts of SDS and culled birds. Copper levels were approximately 32% less in the SDS hearts.

The possible involvement of copper in SDS is interesting in light of the similarities between SDS and a condition known as "bovine falling disease" first reported by Bennetts and Hill (1939). This disease is similar to SDS in that degeneration of the myocardium is evident and the sudden deaths are believed to be as a result of heart failure, usually after excitement or exercise (Underwood 1981). Falling disease is preventable by the treatment of the animal or pasture with copper.

Furthermore, copper has been linked with cardiovascular disease in other animals. In reviewing the literature, Klevay (1980) stated the hypothesis that an absolute or relative copper deficiency (which can be mediated by zinc) may be a primary factor in heart disease. Copper deficiency has been found to result in hypercholestrolemia, decreased levels of plasma HDL, myocardial and arterial damage, sudden death and increased mortality. These effects of low copper levels have been found in mice, rats and swine and the author suggested that copper is also likely involved in human cardiovascular disease.

Although copper was supplemented in the diet as part of the mineral premix at a rate of 25 mg/kg, it may be that either this level is too low to prevent SDS in susceptible individuals or that the zinc added to the premix or in the feed ingredients resulted in a relative copper deficiency in some birds.

Although hardly conclusive, because of the observed low copper content of SDS hearts and the similarities between "bovine falling disease" and other possible relationships between copper and risk factors associated with cardiovascular disease, a possible relationship between copper and SDS may exist and should be further investigated.

GENERAL DISCUSSION

In reviewing the literature, it was apparent that little information as to the cause and/or prevention of SDS existed. Basically, only sex differences, growth rates and possible cardiovascular disorders had been implicated. Due to the lack of information, a consistent approach to reduce the incidence of the syndrome was difficult and therefore a number of different factors such as vitamins, growth rate, fat deposition and electrolyte levels were investigated.

Carcass and abdominal fat deposition has received considerably more attention than SDS and was investigated in conjunction with SDS as both areas have been associated with growth rate. Furthermore, as both problems are of relatively recent concern, it was hypothesized that the occurrence of SDS may have been related to an increase in the fat content of the modern broiler.

A. Sudden Death Syndrome

i) Sex differences

Hemsley (1965), Jackson et al. (1972) and Hulan et al. (1980) reported that SDS most often affected male birds. The results of Experiment 1 confirms this observation as more males were found to be affected than females. As the incidence of SDS is low, experiments were designed to maximize SDS mortality and thus make possible treatment differences more apparent. Consequently, all trials following Experiment 1 used male birds. Furthermore, birds fed a wheat based ration tended to have greater SDS mortality than corn fed birds (Experiment 1) so all further trials employed wheat diets.

ii) Growth rates

A relationship between growth rate and SDS has been suggested by numerous authors including Hulan et al. (1980) and Gasperdone (1981). Small reductions in growth rate (Experiment 2) were found to not reduce SDS incidence. However, large differences in growth rate may result in an increase in affected individuals. The greatest incidence of SDS was observed in the control group of Experiment 3 in which 6.53% of the initial population died from SDS. The birds in this trial exhibited exceptional growth rates resulting in 49 day weights of approximately 2500 g. In other experiments, SDS mortality and growth rates were considerably lower. For example, SDS mortality for the control group in Experiment 6 (same ration formulation as the control group in Experiment 1) was 2.62% with average 49 day body weights of 2079 g, 17% less than 49 day weights in Experiment 3. Thus, it appears that a dramatic difference in growth rate may affect SDS mortality.

iii) Stress

Ononiwu et al. (1979b) found that SDS incidence was greater in birds exposed to continuous light than in birds reared under intermittent light and suggested that increased mortality due to SDS was because of the greater stress of the continuous light regimes.

Indications that stress can affect SDS were observed in the experiments conducted. SDS mortality was found to be greatest after stresses such as handling the bird during individual weighing. Over a 2 hour period of weighing (Experiment 3), 5 birds were found to have died from SDS. Also, the increased 4-7 week mortality of birds fed a high NaCl

ration (Experiment 6) may have been due to physiological stresses (such as increased blood pressure) placed on the bird. Although SDS is likely not totally preventable by reducing stress (such as handling or sudden noises), reductions in stress would probably lower SDS mortality.

iv) Cause of death

Ononiwu et al. (1979a) indicated that a loss of fluids from the circulatory system into the lung tissue resulted in the bird being unable to breathe. The loss of fluids was also the explanation given as to why the heart was damaged and failed, that is enough fluid was lost from the circulatory system to cause peripheral circulatory failure and shock.

From observations of birds that died from SDS and because of the report of Riddell and Orr (1980) where fresh SDS mortalities were found to not exhibit pulmonary congestion and edema, it was hypothesized that lung edema and congestion were not the cause of death but rather death was a direct result of acute heart failure. Birds affected were found to exhibit classical signs of acute heart failure such as spasmodic movements, falling and exaggerated inspiration. Post-mortem pulmonary congestion and edema are also evidence of acute heart failure and occur after death rather than before, as suggested by Ononiwu et al. (1979a). Therefore, lung edema may be useful in determining the cause of death but does not seem to be responsible for the birds dying.

v) Vitamins

As biotin had been suggested to reduce the incidence of SDS, additional vitamin supplementation was investigated. Increasing the levels of vitamins added to the diet did not reduce SDS and thus the

hypothesis that biotin or other vitamins are involved can be questioned.

vi) Lipid metabolism

Lipid metabolism was thought to be involved for a number of reasons. Firstly, SDS and increased fatness of broilers are chronologically linked (both being relatively recent problems) and both are associated with growth rate. Riddell and Orr (1980) had reported increased total serum lipids in some SDS affected birds and suggested further research in this area. In addition, differences in fat metabolism, could also play a role in male-female SDS incidence being different.

The results of Experiment 3 indicated that fatness of the birds may be involved as the groups fed a high protein diet tended to be leaner and concurrently had a significantly smaller number of birds succumb to SDS from 4-7 weeks of age.

It was also speculated that plasma cholesterol concentrations could be involved due to the implied relationship of cholesterol and cardiovascular disease. Cholesterol was considered since Yeh and Leveille (1973) showed that increased dietary protein could lower plasma cholesterol. Therefore, the low SDS mortality observed in Experiment 3 may have been related to high protein levels reducing cholesterol in the plasma.

Attempts to lower SDS by reducing blood levels of triglycerides and cholesterol of birds in Experiment 5 was disappointing as the addition of nicotinic acid to the diet did not significantly reduce these blood parameters and therefore definite relationships to SDS could not be established.

There was, however, an indication that plasma FFA may be involved in SDS. This was evidenced by the observation that SDS mortality from

0-4 weeks (Experiment 5) tended to be greater in the niacin treated birds. A total of 18 individuals in this group died of SDS as compared to 8 individuals in the control group. This tendency for niacin treated birds to have a greater SDS incidence was opposite to the desired affect but does not necessarily refute the hypothesis that blood lipid parameters, specifically FFA, may be involved in SDS.

The tendency for increased SDS mortality in the niacin treated birds may have been related to the observation that nicotinic acid has been shown to cause a rebound rise in plasma FFA levels in man (Rowe et al. 1973) and sheep (Nye and Buchanan 1969). These studies found that after a single oral dose of niacin, plasma FFA levels dropped. However, as niacin was excreted and blood levels of the vitamin dropped, FFA levels in the plasma began to rise and eventually overshot the initial baseline levels. After a brief period of elevated plasma FFA (approximately 2 hours), concentrations of FFA returned to baseline levels. This rebound effect was suggested to account for an excess of atrial fibrillation and other cardiac arrythmias observed in some individuals that received nicotinic acid therapy (Walquist 1981). Although it was believed that continuous feeding would nullify any possible rebound rise in plasma FFA, this rebound effect may have occurred in Experiment 5 and resulted in cardiac arrythmias that could have affected the incidence of SDS.

Furthermore, the effect of protein levels on SDS observed in Experiment 3 was not replicated in Experiment 5 and the effect of C:P on SDS therefore remains in doubt.

vii) Sodium, potassium and copper

The addition of NaCl to the diet tended to increase the number of birds affected by SDS. However, as overall (0-7 weeks, Experiment 6) SDS mortality was not significantly increased, it appears likely that NaCl is not a contributing factor in the syndrome although NaCl cannot be positively ruled out. Similarly, increasing the potassium level of the diet did not significantly reduce SDS and therefore it is doubtful that this element is involved in the disease. As copper levels in heart were found to be lower in SDS birds, further research in this area is warranted.

B. Abdominal fat

i) Restricted intake

Nutrient restriction was found to be an effective method for reducing abdominal fat deposition and was also shown to be economical. The use of restricted feeding would require further research to determine estimates of feed required by different strains of birds.

ii) Fat restriction

The elimination of added dietary fat may not be effective in reducing abdominal fat in all strains of commercial broilers. In slower growing birds that show little compensatory ability compared to faster growing strains, fat restriction from 0-7 days has been shown to significantly reduce abdominal fat deposition (Hargis and Creger 1980). However, in fast growing birds that can compensate for early growth restriction, not adding fat to the diet from 0-7 days may actually increase abdominal fat (Experiment 3). Further research in the severity

of the restriction (time and fat level) and the strain of bird is required before recommendations can be made.

SUMMARY AND CONCLUSIONS

Six experiments were designed to examine several possible nutritional aspects of Sudden Death Syndrome. Two of the 6 experiments were concerned with nutritional aspects of abdominal fat deposition in the broiler. Examination of the data collected from these experiments has led to the following conclusions.

A. Sudden Death Syndrome

1. Sudden death syndrome may affect up to 6.5% of the total population and account for over 60% of total mortality. Males tend to be more susceptible to the disease.
2. SDS mortality can begin as early as 7 days of life and continues until birds reach market weight.
3. A very rapid growth rate tends to result in a greater number of birds dying from SDS although the condition still occurs in relatively slower growing flocks.
4. Stress placed on the birds, such as handling, does seem to increase the incidence of SDS.
5. From observations of birds succumbing to SDS, acute heart failure appears to be the immediate cause of death although other predisposing factors are likely involved. Lung congestion and edema is likely a post-mortem condition and not a cause of death.
6. Supplementation of the ration with additional water or fat soluble vitamins does not reduce the incidence of SDS. Similarly, supplemental potassium or sodium does not appear

to affect the incidence of the syndrome.

7. Fat content of the bird and/or blood lipid parameters may or may not be involved in the development and occurrence of SDS. Although there were indications that decreasing the C:P ratio of the finishing diet could reduce the number of SDS mortalities, the failure to repeat these results indicated that C:P ratios do not affect SDS mortality.
8. The hearts of SDS birds were found to be low in copper. Copper levels of the diet or copper metabolism could be involved with the disease.

B. Abdominal fat

1. Restricting caloric intake by feeding 90% of ad libitum intake is an effective and economically feasible method of reducing abdominal fat deposition in the broiler, although it does result in lowered final body weights.
2. The elimination of added dietary fat from the diet from 0-7 days of age may not in all cases reduce abdominal fat deposition and can actually cause an increased production. In rapidly growing birds that demonstrate compensatory growth ability, this method is not effective in reducing the amount of abdominal fat.

Several aspects of SDS and fat deposition warrant further investigation:

1. Differences in SDS mortality between males and females (indicating that genetic or metabolic factors may be involved)

should be further researched.

2. As an involvement of blood lipid parameters in SDS was not shown, additional studies in the area of fat metabolism may be of interest.
3. Further histological examinations of SDS birds are required especially regarding the hearts and associated blood vessels to determine if cardiovascular disorders (such as cholesterol deposition in coronary arteries) are present.
4. Studies are required to determine if and how copper metabolism is involved in SDS.
5. As there are indications that SDS may be congenital, nutrition of the broiler breeders should be investigated as well as other genetic factors such as strain differences.
6. Generally, in the study of SDS it may be beneficial to use male birds to produce the highest possible number of mortalities and thus increase the likelihood of showing treatment differences. Furthermore, as the percent of the population affected is low, as large a population as possible should be employed in studies of SDS. In addition, as SDS mortality is low, the cost of possible preventative measures should be taken into account so that decreases in income due to SDS losses are not offset by increases in feed or other costs.
7. As restricted feed intake was shown to be effective in reducing abdominal fat deposition, more studies are required to determine the amount of feed required for a particular strain of bird.

8. Few studies have been reported regarding removal of added fat from the ration. Further research on the length of the period of fat restriction, C:P ratios and how these parameters affect different strains of birds is required.

BIBLIOGRAPHY

Adams, R.L., F.N. Andrews, J.C. Rogler and C.W. Carrick. 1962. The protein requirement of 4 week old chicks as affected by temperature. J. Nutr. 77:121-126.

Almquist, H.J. 1954. Utilization of amino acids by chicks. Arch. Biochem. Biophys. 52:197-202.

Altschul, R., A. Hoffer and J.D. Stephen. 1955. Influence of nicotinic acid on serum cholesterol in man. Arch. Biochem. Biophys. 54:558-559.

Altschul, R. 1958. Niacin (nicotinic acid) and serum cholesterol. JAMA 166:822.

Anderson, R., L. Harthon, M. Hedstrom and L. Lundholm. 1973. Inhibition of cyclic AMP formation and lipolysis in rat adipose tissue by nicotinic acid. Atherosclerosis 18:399.

Association of Official Analytical Chemists. 1980. Official methods of analysis, (13th edition). Washington, D.C. U.S.A.

Baldini, J.T. and H.R. Rosenberg. 1955. The effect of productive energy level of the diet on the methionine requirement of the chick. Poult. Sci. 34:1301-1307.

Bartov, I. 1979. Nutritional factors affecting quantity and quality of carcass fat in chickens. Federation Proceedings 38:2627-2630.

Bartov, I. 1982. Corticosterone and fat deposition in broiler chicks: Effect of injection time, breed, sex and age. Br. Poult. Sci. 23:161-170.

Bartov, I. and S. Bornstein. 1977. Stability of abdominal fat and meat of broilers: relative effects of Vitamin E, butylated hydroxytoluene and ethoxyquin. Br. Poult. Sci. 18:59-68.

Bartov, I., B. Lipstein and S. Bornstein. 1974a. Differential effects of dietary acidulated soybean oil soapstock, cottonseed oil soapstock and tallow on broiler carcass fat characteristics. Poult. Sci. 53:115-124.

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

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

Becker, W.A., J.V. Spencer, L.W. Mirosh and J.A. Verstrate. 1981a. Specific gravity, carcass fat, abdominal fat and yield data in broiler chickens. Poult. Sci. 60:2045-2052.

Becker, W.A., J.V. Spencer, L.W. Mirosh and J.A. Verstrate. 1981b. Abdominal and carcass fat in five broiler strains. Poult. Sci. 60:693-697.

Bennets, H.W. and H.T.B. Hill. 1939. "Falling disease" of cattle in the south-west of Western Australia. Aust. Vet. J. 35:163-167.

Brigden, J.L. and C. Riddell. 1975. A survey of mortality in four broiler flocks in western Canada. 1975. Can. Vet. J. 16:194-200.

Brody, S. 1935. Nutrition. Ann. Rev. Biochem. 4:383-412.

Carew, L.B. and F.W. Hill. 1958. Studies on the effect of fat on metabolic efficiency of energy utilization. Poult. Sci. 37:1191.

Carew, L.B. and F.W. Hill. 1961. Effect of methionine deficiency on the utilization of energy by the chick. J. Nutr. 74:185-190.

Carlson, L.A., C. Hedbom, E. Helgstrand, B. Sjoberg and N.E. Stjernstrom. 1969. In: Drugs Affecting Lipid Metabolism. L.A. Carlson and R. Paolett, Editors. Plenum, New York, N.Y. p. 85-92.

Carlson, L.A. and L.E. Bottiger. 1972. Ischaemic heart disease in relation to fasting values of plasma triglycerides and cholesterol. Stockholm prospective study. Lancet 1:865-871.

Carlson, L.A. 1977. Nicotinic acid and inhibition of fat mobilizing lipolysis. Present status of effects on lipid metabolism. Adv. Exp. Biol. Med. 85:225-238.

Carlson, N.A. 1971. Nicotinic acid: its metabolism and its effects on free fatty acids. In: Metabolic Effects of Nicotinic Acid and its Derivatives. K.F. Gey and L.A. Carlson, Editors. Hans Huber, Publishing Co., Bern, Switzerland. p. 157.

Carlstrom, S. and S. Lavrell. 1968. The effect of nicotinic acid on the diurnal variation of the free fatty acids of plasma. Acta Med. Scand. 184:121.

Cassidy, D.M., M.A. Gibson and F.G. Proudfoot. 1975. The histology of cardiac blood clots in chicks exhibiting the "flip-over" syndrome. Poult. Sci. 54:1882-1886.

Cave, N.A. 1980. Effect of intermittent lighting on feed efficiency and broiler carcass fat. Poult. Sci. 59:1590 (abstract).

Cherry, J.A., P.B. Siegel and W.L. Beane. 1978. Genetic-nutritional relationships in growth and carcass characteristics of broiler chickens. Poult. Sci. 57:1482-1487.

Coombs, G.F. 1967. Amino acid allowances for growing chicks including broilers. In: Protein Utilization by Poultry. Oliver and Boyd, Edinburgh and London. p. 119-136.

Coombs, G.F. 1968. Amino acid requirement of broilers and laying hens. Proceedings of the Maryland Nutrition Conference. p. 87-90.

Dale, N.M. and H.L. Fuller. 1980. Effect of diet composition on feed intake and growth of chicks under heat stress. II. Constant vs cycling temperatures. Poult. Sci. 59:1434-1441.

Dam, R.R., M. Leach, Jr., T.S. Nelson, L.C. Norris and F.W. Hill. 1959. Studies on the effect of quality and type of fat on chick growth. J. Nutr. 68:615-632.

Dawson, N.J., S.K. Stephenson and D.K. Fredline. 1972. Body composition of mice subjected to genetic selection for different body proportions. Comp. Biochem. Physiol. 42:679-691.

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

Deaton, J.W., L.F. Kubena, T.C. Chen and F.N. Reece. 1974. Factors influencing the quantity of abdominal fat in broilers. 2. Cage vs floor rearing. Poult. Sci. 53:574-576.

Deaton, J.W., F.N. Reece, L.F. Kubena and J.D. May. 1976. Effect of varying light intensity on broiler performance. Poult. Sci. 55:515-519.

Deaton, J.W., F.N. Reece and J.L. McNaughton. 1978a. The effect of temperature during the growing period on broiler performance. Poult. Sci. 57:1070-1074.

Deaton, J.W., F.N. Reece and J.L. McNaughton. 1978b. Effect of intermittent light on broilers reared under moderate temperature conditions. Poult. Sci. 57:785-788.

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

Donaldson, W.E., G.F. Combs and G.L. Romoser. 1956. Studies on energy levels in poultry rations. 1. The effect of calorie:protein ratio of the ration on growth, nutrient utilization and body composition of chicks. Poult. Sci. 35:1100-1105.

Edwards, H.M. 1971. Effect of type of fat supplementation on the body composition of broilers. Feedstuffs 43(16):66-70.

Edwards, H.M. and P. Hart. 1971. Carcass composition of chickens fed carbohydrate-free diets containing various lipid energy sources. J. Nutr. 101:989-996.

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

Eisen, E.J., H. Bakker and J. Nagal. 1977. Body composition and energetic efficiency in two lines of mice selected for rapid growth rate and their F_1 crosses. Theoret. Appl. Gen. 49:21-34.

Estep, G.D., R.C. Fanguy and T.M. Ferguson. 1969. The effect of age and heredity upon serum cholesterol levels in chickens. Poult. Sci. 48:1908-1911.

Evans, D.G., T.C. Goodwin and L.D. Andrews. 1976. Chemical composition, carcass yield and tenderness of broilers as influenced by rearing methods and genetic strains. Poult. Sci. 55:748-755.

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

Fisher, H. and R. Shapiro. 1961. Amino acid imbalance: Rations low in tryptophan, methionine and lysine and the efficiency of utilization of nitrogen in imbalanced rations. J. Nutr. 75:395-401.

Forbes, R.M. and M. Yohe. 1955. Effect of energy intake on the biological value of protein fed to rats. J. Nutr. 55:499-506.

Fowler, R.F. 1958. The growth and carcass composition of strains of mice selected for large and small body size. J. Agric. Sci. 51:137-148.

Fraps, G.S. 1943. Relation of the protein, fat and energy of the rations to the composition of chickens. Poult. Sci. 22:421-424.

French, H. and P. Hunton. 1979. Abdominal fat in broilers. Shaver Focus 8(1):6-7.

Frings, C.S. and R.T. Dunn. 1970. A colorimetric method for determination of total serum lipids based on the sulfo-phospho-vanillin reaction. Am. J. Clin. Path. 53:89-91.

Fuller, H.L. and M. Rendon. 1977. Energetic efficiency at different dietary fats for growth of young chicks. Poult. Sci. 56:549-557.

Ganong, W.F. 1975. Review of Medical Physiology, (7th edition). Lange Medical Publications, Los Altos, California. 587 p.

Gasperdone, H. 1981. Feeding schedules and "flippers". Canada Poultryman 68(12):34-36.

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

Griffiths, L., S. Leeson and J.D. Summers. 1977b. Influence of energy system and level of various fat sources on performance and carcass composition of broilers. Poult. Sci. 56:1018-1026.

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

Hargis, P.H. and C.R. Creger. 1980. Effects of varying dietary protein and energy levels on growth rate and body fat of broilers. Poult. Sci. 59:1499-1504.

Heath, J.L., R.C. Covey and S.L. Owens. 1980. Abdominal leaf fat separation as a result of evisceration of broiler carcasses. Poult. Sci. 59:2456-2461.

Hemsley, L.A. 1965. The causes of mortality in fourteen flocks of broiler chickens. Vet. Rec. 77:467-472.

Hill, D.C. and E.M. Olsen. 1963. Effect of the addition of imbalanced amino acid mixtures to a low protein diet on weight gains and plasma amino acids of chicks. J. Nutr. 79:296-302.

Hill, F.W. and L.M. Dansky. 1950. Studies on the energy requirements of chickens. Poult. Sci. 34:1206.

Hill, F.W. and L.M. Dansky. 1954. Studies of the energy requirements of chickens. 1. The effect of dietary energy level on growth and feed consumption. Poult. Sci. 33:112-119.

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

Hood, R.L. and R.A.E. Pym. 1982. Correlated response for lipogenesis and adipose tissue cellularity in chickens selected for body weight gain, food consumption and food conversion efficiency. Poult. Sci. 61:122-127.

Hulan, W.H., F.G. Proudfoot and K.B. McRae. 1980. Effect of vitamins on the incidence of mortality and acute death syndrome ("flip-over") in broiler chickens. Poult. Sci. 59:927-931.

Hurwitz, S., I. Cohen, A. Bar and S. Bornstein. 1973. The sodium and chloride requirements of the chick: relationship to acid-base balance. Poult. Sci. 52:903-909.

Hurwitz, S., I. Cohen, A. Bar and V. Minkov. 1974. Sodium and chloride requirements of the 7-9 week-old broiler. Poult. Sci. 53:326-331.

Jackson, C.A.W., D.J. Kingston and L.A. Hemsley. 1972. A total mortality survey of nine batches of broiler chickens. Aust. Vet. J. 48:481-487.

Jackson, R.L., J.D. Morrison and A.M. Gotto, Jr. 1976. Lipoprotein structure and metabolism. Physiol. Rev. 56:260-316.

Kakita, C., P.J. Johnson, R. Pick and L.N. Katz. 1972. Relationship between plasma cholesterol level and coronary atherosclerosis in cholesterol-oil fed cockerels. Atherosclerosis 15:17-25.

Kannel, W.B., W.P. Castelli and T. Gordon. 1971. Serum cholesterol lipoproteins and risk of coronary heart disease. The Framingham study. Ann. Int. Med. 74:1-12.

Kleiber, M. and J.E. Dougherty. 1934. The influence of environmental temperature on the utilization of food energy in baby chicks. J. Gen. Physiol. 17:701-726.

Klevay, L.M. 1980. Interactions of copper and zinc in cardiovascular disease. In: Micronutrient Interactions: Vitamins, Minerals and Hazardous Elements. Annals of the New York Academy of Sciences, Volume 355. O.A. Levander and L. Cheng, Editors. The New York Academy of Sciences, New York, N.Y.

Kondra, P.A., J.F. Richards and G.C. Hodgson. 1962. The effect of sex, ration and strain on meat yield and its determination in chicken broilers. Poult. Sci. 41:922-927.

Kritchevsky, D. 1971. Effect of nicotinic acid and its derivatives on cholesterol metabolism: a review. In: Metabolic Effects of Nicotinic Acid and its Derivatives. K.F. Gey and L.A. Carlson, Editors. Hans Huber Publishing Co., Bern, Switzerland. p. 541.

Kritchevsky, D. and S.A. Tepper. 1962. Influence of nicotinic acid homology on oxidation of cholesterol-26-C^{14} by rat liver mitochondria. Arch. Int. Pharmacodyn 138:349-353.

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

Kubena, L.F., T.C. Chen, J.W. Deaton and F.N. Reece. 1974a. Factors influencing the quantity of abdominal fat in broilers. 3. Dietary energy levels. Poult. Sci. 53:974-978.

Kubena, L.F., J.W. Deaton, T.C. Chen and F.N. Reece. 1974b. Factors influencing the quantity of abdominal fat in broilers. 1. Rearing temperature, sex, age or weight and dietary choline chloride and inositol supplementation. Poult. Sci. 53:211-214.

Kutsky, R.J. 1973. Handbook of vitamins and hormones. Van Nostrand Reinhold Company, New York, Cincinnati, Toronto, London, Melbourne p. 96-103.

Lang, B.J. and J.E. Legates. 1969. Rate, composition and efficiency of growth in mice selected for large and small body weight. Theoret. Appl. Gen. 39:306-314.

Leclerq. B., I. Hassan and J.C. Blum. 1974. The influence of force-feeding on the transport of plasma lipids in the chicken (Gallus gallus L.). Comp. Biochem. Physiol. 47:289.

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

Leveille, G.A., D.R. Romsos, Y.Y. Yeh and E.K. O'Hea. 1975. Lipid biosynthesis in the chick. A consideration of site of synthesis, influence of diet and possible regulatory mechanisms. Poult. Sci. 54:1075-1093.

Levy, R.I. and T. Langer. 1972. Hypolipidemic drugs and lipoprotein metabolism. Adv. Exp. Biol. Med. 26:155-171.

Littlefield, L.H. 1972. Strain differences in quantity of abdominal fat in broilers. Poult. Sci. 51:1829 (abstract).

Malone, G.W., G.W. Chaloupka, E.W. Walpole and L.H. Littlefield. 1980a. The effect of dietary energy and light treatment on broiler performance. Poult. Sci. 59:576-581.

Malone, G.W., G.W. Chaloupka, J.W. Merkley and L.H. Littlefield. 1980b. The effect of feeder space and light treatment on broiler performance. Poult. Sci. 59:2697-2702.

March, B. and J. Biely. 1957. Fat studies in poultry. 6. Utilization of fats of different melting points. Poult. Sci. 36:71-75.

March, B.E. and G. Hanson. 1977. Lipid accumulation and cell multiplication in adipose bodies in White Leghorn and broiler-type chicks. Poult. Sci. 56:886-894.

Marion, J.E., H.M. Edwards, Jr. and J.C. Driggers. 1961. Influence of diet on serum cholesterol in the chick. J. Nutr. 74:171-175.

Marks, H.L. 1979. Compensatory growth of selected and nonselected broilers following early protein restriction. Poult. Sci. 58:1409-1414.

Matterson, L.D., L.M. Potter, L.D. Stinson and E.P. Singsen. 1955. Studies on the effect of varying protein and energy levels in poultry rations on growth and feed efficiency. Poult. Sci. 34:1210.

Maurice, D.V. and A.P. Deodato. 1982. Sodium chloride-induced reduction of abdominal fat in broilers. Poult. Sci. 61:1508 (abstract).

McCartney, M.G. and H.B. Brown. 1977. The effects of feed restriction time on the growth and feed conversion of broiler males. Poult. Sci. 56:713-715.

Merkley, J.W., B.T. Weinland, G.W. Malone and G.W. Chaloupka. 1980. Evaluation of five commercial broiler crosses. 2. Eviscerated yield and component parts. Poult. Sci. 59:1755-1760.

Mickleberry, W.C., J.C. Rogler and W.J. Stadelmon. 1966. The influence of dietary fat and environmental temperature upon chick growth and carcass composition. Poult. Sci. 45:313-321.

Miettinen, T.A. 1971. Influence of nicotinic acid in the treatment of hypercholesterolemia. In: Metabolic Effects of Nicotinic Acid and its Derivatives. K.F. Gey and L.A. Carlson, Editors. Hans Huber Publishing Co., Bern, Switzerland. p. 649.

Mirosh, L.W. and W.A. Becker. 1983. Testing a caliper for measuring thickness of the abdominal region in live broiler chickens. Poult. Sci. 62:1-5.

Mongin, P. and B. Sauveur. 1977. Interrelationships between mineral nutrition, acid-base balance, growth and cartilage abnormalities. In: Growth and Poultry Meat Production. K.N. Bourman and B.S. Wilson (editors). British Poultry Science Ltd. Edinburgh, Scotland.

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

Moran, Jr., E.T. 1980. Early protein restriction of the broiler chicken and carcass quality upon later marketing. Poult. Sci. 59:378-382.

Moses, G., E. Ollvero and T.F. Draisey. 1975. Simultaneous determination of serum cholesterol and triglycerides after preliminary column chromatography. Clin. Chem. 21:428-431.

National Research Council. 1977. Nutrient requirements of poultry, 7th revised edition. National Academy of Science, Washington, D.C.

Nelson, T.S. 1980. Feeding for changes in body composition of broilers. Publication of Arkansas Agricultural Experimental Station.

Nikkila, E.A. 1971. In: Metabolic Effects of Nicotinic Acid and its Derivatives. K.F. Gey and L.A. Carlson, Editors. Hans Huber Publishing Co., Bern, Switzerland. p. 487.

Nir, I., V. Levy and M. Perek. 1973. Response of plasma glucose, free fatty acids and triglycerides to starving and re-feeding in cockerels and geese. Br. Poult. Sci. 14:263-268.

Nordstrom, J.O., R.H. Towner, G.B. Haverstein and G.L. Walker. 1978. Influence of genetic strain, sex and dietary energy level on abdominal fat deposition in broilers. Poult. Sci. 57:1176.

Nye, E.R. and H. Buchanan. 1969. Short-term effect of nicotinic acid on plasma level and turnover of free fatty acids in sheep and man. J. Lipid Res. 10:193-196.

Ononiwu, J.C., R.G. Thomson, H.C. Carlson and R.J. Julian. 1979a. Pathological studies of "Sudden Death Syndrome" in broiler chickens. Can. Vet. J. 20:70-73.

Ononiwu, J.C., R.G. Thomson, H.C. Carlson and R.J. Julian. 1979b. Studies on effect of lighting on "Sudden Death Syndrome" in broiler chickens. Can. Vet. J. 20:74-77.

Otway, S., D.C. Robinson, M.P. Rogers and D.R. Wing. 1971. In: Metabolic Effects of Nicotinic Acid and its Derivatives. K.F. Gey and L.A. Carlson, Editors. Hans Huber, Publishing Co., Bern, Switzerland. p. 497.

Pelkonen, R., E.A. Nikkila, S. Koskinen, K. Penttinen and S. Sarna. 1977. Association of serum lipids and obesity with cardiovascular mortality. Br. Med. J. 11:1185-1187.

Persad, A. and V.R. Sedagopan. 1976. Effect of supplemental methionine in broiler diets on dressing percentage and carcass composition. Ind. J. Nutr. Dietet. 13:406.

Peterson, D.W., C.R. Grau and N.F. Peek. 1954. Growth and food consumption in relation to dietary levels of protein and fibrous bulk. J. Nutr. 52:241-257.

Petrak, M.L. (Editor). 1969. Diseases of cage and aviary birds. Lea and Febiger, Philadelphia, P.A. p. 296.

Pfaff, F.E. and R.E. Austic. 1976. Influence of diet on development of the abdominal fat pad in the pullet. J. Nutr. 106:443-450.

Pick, R. and L.N. Katz. 1965. The morphology of experimental cholesterol and oil-induced atherosclerosis in the chick. In: Comparative Atherosclerosis. J.R. Roberts, Jr., R. Strauss and M.S. Cooper, Editors. Harper and Row, New York, N.Y.

Proudfoot, F.G. and H.W. Hulan. 1982. Effects of reduced feeding time using all mash or crumble-pellet dietary regimens on chicken broiler performance, including the incidence of acute death syndrome. Poult. Sci. 61:750-754.

Proudfoot, F.G., H.W. Hulan and K.B. McRae. 1982. The effect of crumbled and pelleted feed on the incidence of sudden death syndrome among male chicken broilers. Poult. Scil 61:1766-1768.

Proudman, J.A., W.J. Mellen and D.L. Anderson. 1970. Utilization of feed in fast and slow growing lines of chickens. Poult. Sci. 49:961-972.

Pym, R.A.E. and A.J. Solvyns. 1979. Selection for food conversion in broilers: body composition of birds selected for increased body weight gain, food consumption and food conversion ratio. Br. Poult. Sci. 20:87-97.

Pym, R.A.E. and J.M. Thompson. 1980. A simple caliper technique for the estimation of abdominal fat in live broilers. Br. Poult. Sci. 21:281-286.

Recommended Dietary Allowances, 8th revised edition, Food and Nutrition Board, National Research Council - National Academy of Sciences. 1974. In: Review of Medical Physiology, 7th edition, 1975. W.F. Ganong. Lange Medical Publications, Los Altos, California.

Ricard, F.H. and R. Rouvier. 1967. Etude de la composition anatomique du poulet. I. Variabilite de la repartitiom des differentes parties corporelles chez des coquelets "Bresse Pile". Annoles de Zootechnie 16:23-29.

Ricard, F.H. and R. Rouvier. 1969. Etude de la composition anatomique du poulet. II. Variabilite de la repartition des parties corporelles dans une souche de type Cornol. Annales de Genetique et Selection animale 1:151-165.

Riddell, C. and J.P. Orr. 1980. Chemical studies of the blood, and histological studies of the heart of broiler chickens dying from acute death syndrome. Avian Dis. 24:751-757.

Ried, J.T. and O.D. White. 1978. The phenomenon of compensatory growth. Proceedings of the Maryland Nutrition Conference for Feed Manufacturers, March 16-17, Cornell University Press, Ithaca, N.Y.

Roberts, R.C. 1965. Some contributions of the laboratory mouse to animal breeding research. Anim. Br. Abstr. 33:339-353.

Rowe, N.J., M.A. Dolder, B.J. Kirby and M.F. Oliver. 1973. Effect of a nicotinic acid analogue on raised plasma free fatty acids after acute myocardial infarction. Lancet 2:814.

Rudas, B., G. Wick and R.K. Cole. 1972. Serum lipid pattern in chicks of the obese strain. J. Endocrinol. 55:609-615.

Salleh, M.S., N.L. Kamus and A.J. Farr. 1978. Ration and strain effects on broiler growth performance and processing parameters. Poult. Sci. 57:1186 (abstract).

Schlierf, G. and G. Hess. 1977. Inhibition of carbohydrate-induced hypertriglyceridemia by nicotinic acid. Artery 3:174-179.

Schwartz, H.G., M.W. Taylor and H. Fisher. 1958. The effect of dietary energy concentration and age on lysine requirements of growing chick. J. Nutr. 65:25-37.

Scott, H.M., L.C. Sims and D.L. Staheli. 1955. The effect of varying protein and energy on the performance of chicks. Poult. Sci. 34:1220 (abstract).

Scott, H.M. and R.M. Forbes. 1958. The arginine requirement of chicks in relation to diet composition. Poult. Sci. 37:1347-1349.

Scott, M.L., F.W. Hill, E.H. Parsons, Jr. and J.H. Bruchner. 1959. Studies on duck nutrition. 7. Effect of dietary energy:protein relationships upon growth, feed utilization and carcass composition. Poult. Sci. 38:497-507.

Scott, M.L., M.C. Nesheim and R.J. Young. 1982. Nutrition of the chicken. M.L. Scott & Associates, Ithaca, N.Y. 562 p.

Seaton, K.W., O.P. Thomas, R.M. Gous and H.E. Bossard. 1970. The effect of diet on liver glycogen and body composition in the chick. Poult. Sci. 57:692-698.

Sell, J.L., F. Horani and R.L. Johnson. 1976. The "extra caloric" effect of fat in laying hen rations. Feedstuffs 48(27):28-29.

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

Siegel, P.B. and E.L. Wisman. 1966. Selection for body weight at eight weeks of age. 6. Changes in appetite and feed utilization. Poult. Sci. 45:1391-1397.

Snedecor, G.W. and W.G. Cochran. 1980. Statistical Methods, 7th edition. The Iowa State University Press, Ames, Iowa.

Steele, P., J. Edgar and G. Concon. 1982. Effect of biotin supplementation on incidence of acute death syndrome in broiler chickens. Poult. Sci. 61:909-913.

Sturkie, P.D. 1976. Avian Physiology, 3rd Edition. Springer-Verlag, New York, Heidelberg, Berlin. 400 p.

Summers, J.D., S.J. Slinger and G.C. Ashton. 1965. The effect of dietary energy and protein on carcass composition with a note on a method for estimating carcass composition. Poult. Sci. 44:501-509.

Summers, J.D. and S. Leeson. 1979. Composition of poultry meat as affected by nutritional factors. Poult. Sci. 58:536-542.

Sunde, M.L. 1956. A relationship between protein level and energy level in chick rations. Poult. Sci. 35:350-354.

The Merck Veterinary Manual. 1979. (5th edition). Merck and Company, Inc., Rahway, N.J.

Thomas, O.P. and P.V. Twining. 1971. Broiler nutrition during the withdrawal period (7-8 1/2 weeks). Proceedings of the Maryland Nutrition Conference, p. 87-90.

Timon, V.M., E.J. Eisen and J.M. Leatherwood. 1970. Comparisons of ad libitum and restricted feeding of mice selected and unselected for postweaning gain. II. Carcass composition and energetic efficiency. Genetics 65:145-155.

Underwood, E.J. 1981. The Mineral Nutrition of Livestock, 2nd Edition. Commonwealth Agricultural Bureaux, Farnham Royal, England. p. 95.

Velu, J.G., D.H. Baker and H.M. Scott. 1971. Protein and energy utilization by chicks fed graded levels of a balanced mixture of crystalline amino acids. J. Nutr. 102:741-747.

Velu, J.G., H.M. Scott and D.H. Baker. 1972. Body composition and nutrient utilization of chicks fed amino acid diets containing graded amounts of either isoleucine or lysine. J. Nutr. 102:741-748.

Volk, M., M. Herceg, B. Marzan, M. Kralj, S. Meknic and V. Tadic. 1974. Investigations of fatal syncope of fowl in broilers. I. Incidence, clinical symptoms, pathomorphological findings and pathogenesis. Veterinarski Archiv. 44:23.

Wahlquist, M.L. 1981. Effects on plasma cholesterol of nicotinic acid and its analogues. In: Vitamins in Human Biology and Medicine. M.H. Briggs, Editor, CRC Press, Inc., Boca Raton, Florida. p. 82-94.

Waldroup, P.W., R.J. Mitchell, J.R. Payne and Z.B. Johnson. 1976. Characterization of the response of broiler chickens to diets varying in nutrient density content. Poult. Sci. 55:130-145.

Weiss, H.S., H. Fisher and P. Griminger. 1968. Interrelationship among plasma cholesterol, blood pressure, blood coagulation and spontaneous avian atherosclerosis. Poult. Sci. 47:137-147.

Wethli, E. and J.P.H. Wessels. 1973. The association between body fat content and thyroid activity, feed intake, mass gain, feed conversion and final body mass in growing chickens. Agroanimelia 5:83-88.

Winchester, C.F. and F. Kleiber. 1938. The effect of environmental temperature on mortality, rate of growth and utilization of food energy in White Leghorn chicks. J. Agr. Res. 57:529-544.

Yeh, Y.Y. and G.A. Leveille. 1969. Effect of dietary protein on hepatic lipogenesis in the growing chick. J. Nutr. 98:356-366.

Yeh, S.C. and G.A. Leveille. 1973. Influence of dietary protein level on plasma cholesterol turnover and fecal steroid excretion in the chick. J. Nutr. 103:407-411.

Yoshida, M., H. Hoshii and H. Morimoto. 1966. Lipolytic diet to control carcass fat deposition of growing chick. 13th World's Poultry Congress, Kiev, Ukraine. p. 200-202.

Yoshida, M. and M. Morimoto. 1970. Periodical change in the carcass composition of chicks after switching over the diet to lipogenic or lipolytic diet. Agric. Biol. Chem. 34:423-431.

Appendix 1. Mean squares from analysis of variance tables - mean weight gains, feed consumption and feed:gain ratios - Experiment 1

Periods (weeks)	Source	df	Mean squares		
			Av. wt. gain	Av. feed consumption	Feed:gain
0-1	Treatment	3	162.17**	19.82	0.039**
	Sex	1	109.23*	92.43*	0.001
	T x S	3	5.15	1.38	0.002
	Error	16	17.46	12.80	0.001
1-5	Treatment	3	0.065**	0.120**	0.012**
	Sex	1	0.176**	0.310**	0.030**
	T x S	3	0.0002	0.003	0.001
	Error	16	0.0004	0.001	0.0007
5-7	Treatment	3	0.032**	0.006	0.202**
	Sex	1	0.109**	0.406**	0.086**
	T x S	3	0.001	0.002	0.020
	Error	16	0.020	0.002	0.010
0-7	Treatment	3	0.093**	0.168**	0.009**
	Sex	1	0.580**	1.458**	0.033**
	T x S	3	0.005	0.009	0.002
	Error	16	0.031	0.004	0.001

* Indicates significance at $P<0.05$.

** Indicates significance at $P<0.01$.

Appendix 2. Mean squares from analysis of variance tables - dressing percent, fat pad percent and mortality data - Experiment 1

Periods (weeks)	Source	df	Mean squares	
			Dressing percent	Fat pad percent
7	Treatment	3	6.869**	12.903*
	Sex	1	6.697*	35.454**
	T x S	3	0.799	1.117
	Error	88	1.518	3.451

Periods (weeks)	Source	df	Mean squares	
			Total mortality	SDS mortality
0-7	Treatment	3	14.547	22.650
	Sex	1	96.962	72.141
	T x S	3	28.883	17.387
	Error	16	30.517	20.665

* Indicates significance at $P<0.05$.

** Indicates significance at $P<0.01$.

Appendix 3. Comparison of net[1] profits between birds fed ad libitum and birds fed 90% of ad libitum intake (Experiment 1)

I. Current feed prices[2]

Starter crumbles	- 26.8¢/kg
Finisher pellets	- 25.9¢/kg

II. Price/kg of live wt. - 99¢

III. Feed consumption (from table 11)

	Ad libitum	90%
Starter	2.015 kg	1.745 kg
Finisher	1.695 kg	1.642 kg

IV. Average 7 wk wts.

Ad libitum: 1.857 kg

90%: 1.760 kg

A. Feed Cost

	Ad libitum	90%
Starter	2.015 x 26.8 = 54¢	1.745 x 26.8¢ = 47¢
Finisher	1.695 x 25.9 = 44¢	1.642 x 25.9¢ = 42¢
	$0.98	$0.89

B. Gross income/bird (Av. wt x $0.99)

Ad libitum: 1.857 x 0.99 = $1.83

90%: 1.760 x 0.99 = $1.74

C. Net income (gross income - feed cost)

Ad libitum: 1.83 - 0.98 = $0.85

90%: 1.74 - 0.89 = $0.85

[1]Other costs that would affect net profit are not considered.

[2]From the price list of Feed-Rite Mills, Winnipeg, Manitoba, January 1983.

Appendix 4. Mean squares from analysis of variance tables - mean body weights, weight gain and feed:gain - Experiment 2

Periods (weeks)	Source	df	Mean squares		
			Mean body wt.	Mean wt. gain	Feed:gain
0-2	Treatment	4	956.000**	956.000**	0.0275*
	Error	21	49.904	49.904	0.0095
0-5	Treatment	4	5279.500**	5279.500**	0.0025
	Error	21	636.386	636.286	0.0023
0-7	Treatment	4	8914.750*	8914.750*	0.0015
	Error	21	1798.142	1798.142	0.0021

* Indicates significance at $P<0.05$.

** Indicates significance at $P<0.01$.

Appendix 5. Mean squares from analysis of variance tables - mortality data - Experiment 2

Periods (weeks)	Source	df	Mean squares	
			Total mortality	SDS mortality
0-2	Treatment	4	13.672	6.225
	Error	21	14.453	11.009
0-5	Treatment	4	6.524	2.721
	Error	21	16.979	20.749
0-7	Treatment	4	4.890	1.654
	Error	21	11.642	19.829

Appendix 6. Mean squares from analysis of variance tables - mean body weights, weight gains and feed:gain ratios (0-4 weeks) - Experiment 3

Periods (weeks)	Source	df	Mean squares		
			Mean body wt.	Mean gain	Feed:gain
0-1	Treatment	3	614.00**	614.00**	0.667**
	Error	24	11.12	11.12	0.0012
1-2	Treatment	3	1060.00**	78.33	.0049*
	Error	24	55.12	181.95	.0016
2-3	Treatment	3	1327.33*	20.66	.0013
	Error	24	312.37	63.58	.0007
3-4	Treatment	3	1276.33	5.00	.0023
	Error	24	342.37	112.45	.0010
0-4	Treatment	3		1276.33*	.0005
	Error	24		342.37	.0005

* Indicates significance at $P \leq .05$.

** Indicates significance at $P \leq .01$.

Appendix 7. Mean squares from analysis of variance tables - mean body weights, weight gains and feed:gain ratios (4-7 weeks) - Experiment 3

Periods (weeks)	Source	df	Mean squares		
			Mean body wt.	Mean gain	Feed:gain
4-7	Treatment	3		1353.00	0.0090*
	Restriction	1		1290.00	0.0060*
	Protein level	1		2760.00	0.0200**
	R x P	1		9.00	0.0010
	Error	24		1222.37	0.0018
0-7	Treatment	3	914.66	914.66	.0035*
	Restriction	1	670.00	672.00	.0022
	Protein level	1	2074.00	207.00	.0082**
	R x P	1	0.00	0.00	.0002
	Error	24	1891.29	1891.29	.0008

* Indicates significance at $P \leq .05$.

** Indicates significance at $P \leq .01$.

Appendix 8. Mean squares from analysis of variance tables - mortality data - Experiment 3

Periods (weeks)	Source	df	Total mortality	SDS mortality
0-4	Treatment	1	4.263	3.1559
	Error	26	17.778	22.505
4-8	Treatment	3	60.319	80.422*
	Restriction	1	8.459	9.454
	Protein	1	143.510*	227.145**
	R x P	1	28.988	4.665
	Error	24	23.092	25.510
0-8	Treatment	3	13.269	20.887
	Restriction	1	4.781	1.745
	Protein	1	29.808	60.006
	R x P	1	5.220	0.910
	Error	24	20.705	21.318

* Indicates significance at $P<0.05$.

** Indicates significance at $P<0.01$.

Appendix 9. Mean squares from analysis of variance tables - average carcass weights, dressing percent, fat pad weight and fat pad percent - Experiment 3

Source	df	Mean squares			
		Mean carcass wt.	Dressing percent	Fat pad wt.	Fat pad percent
Treatment	3	2206.792	0.928	154.878	4.001
Restriction	1	13.062	0.669	342.834	0.487
Protein	1	0.437	2.042	17.281	8.499
R x P	1	6606.875	0.074	104.520	3.017
Error	80	28517.067	7.786	182.272	2.771

Appendix 10. Mean squares from analsis of variance tables - mean body weights, weight gains, feed:gain ratios, plasma total lipids, triglycerides and cholesterol - Experiment 4

Periods (weeks)	Source	df	Mean squares					
			Av. body wt.	Av. gains	Feed:gain	Total lipids	Triglycerides	Cholesterol
0-2	Treatment	3	425.177	215.246	0.003			
	Error	24	556.760	550.429	0.008			
2-4	Treatment	3		3670.326	0.070			
	Error	24		2127.274	0.059			
0-4	Treatment	3	5535.914	4299.781	0.019	2320.327	8021.339**	2542.134**
	Error	24	2268.086	2216.877	0.022	2511.438	711.489	479.374

* Indicates significance at P<0.05.

** Indicates significance at P<0.01.

Appendix 11. Mean squares from analysis of variance tables - mean body weights, weight gains and feed:gain ratios - Experiment 5

Periods (weeks)	Source	df	Mean squares		
			Mean body weights	Mean gains	Feed:gain
0-4	Treatment	3	620.572	620.572	0.002
	Error	24	366.237	366.237	0.0009
4-7	Treatment	3	1423.916	464.291	0.005
	Niacin	1	622.312	357.156	0.00005
	Protein level	1	1889.312	3.609	0.016*
	N x P	1	1760.125	1032.109	0.0001
	Error	24	1358.710	947.833	0.002
0-7	Treatment	3		1423.916	0.001
	Niacin	1		622.312	0.00008
	Protein level	1		1889.312	0.003
	N x P	1		1760.125	0.00003
	Error	24		1358.710	0.0008

Appendix 12. Mean squares from analysis of variance tables - mortality data - Experiment 5

Periods (weeks)	Source	df	Mean squares: Total mortality	Mean squares: SDS mortality
0-4	Treatment	1	110.167*	58.928
	Error	26	19.521	14.203
4-7	Treatment	3	7.024	3.511
	Niacin	1	10.825	6.355
	Protein level	1	0.035	3.250
	N x P	1	10.212	0.928
	Error	24	14.280	21.790
0-7	Treatment	3	30.287	16.101
	Niacin	1	75.308*	25.997
	Protein level	1	13.832	17.664
	N x P	1	1.720	4.641
	Error	24	16.856	13.716

* Indicates significance at $P<0.05$.

** Indicates significance at $P<0.01$.

Appendix 13. Mean squares from analysis of variance tables - total plasma lipids, triglycerides and cholesterol - Experiment 5

			Mean squares		
Week	Source	df	Total lipids	Triglycerides	Cholesterol
4	Treatment	1	392.050	1417.840	991.310
	Error	25	5510.00	1278.670	889.680
7	Treatment	3	5416.375	1218.944	307.000
	Niacin	1	15759.377	962.666	6.000
	Protein	1	459.376	2646.000	793.000
	N x P	1	30.373	48.166	121.499
	Error	24	7728.091	991.083	754.816

Appendix 14. Mean squares from analysis of variance tables - mean body weights, weight gains and feed:gain ratios - Experiment 6

Periods (weeks)	Source	df	Mean squares		
			Mean body wt.	Mean gain	Feed:gain
0-4	Treatment	2	635.164	607.164	0.0001
	Error	15	577.710	577.710	0.0002
4-7	Treatment	2	3165.500	1055.167	0.006
	Error	15	1023.164	1312.009	0.003
0-7	Treatment	2		3066.515	0.002
	Error	15		1023.166	0.001

Appendix 15. Mean squares from analysis of variance tables - total and SDS mortality - Experiment 6

Periods (weeks)	Source	df	Mean squares	
			Total mortality	SDS mortality
0-4	Treatment	2	11.498	23.306
	Error	15	11.668	11.325
4-7	Treatment	2	13.371	89.972**
	Error	15	17.654	11.061
0-7	Treatment	2	21.163	13.876
	Error	15	9.746	9.252

* Indicates significance at $P<0.05$.

** Indicates significance at $P<0.01$.