

DOSE EFFECTS OF L-CARNITINE ON PERFORMANCE, CARCASS
CHARACTERISTICS AND BODY FAT COMPOSITION OF
19, 35, AND 49 DAYS OLD BROILER CHICKENS

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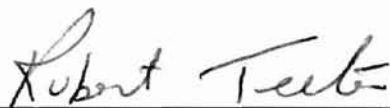
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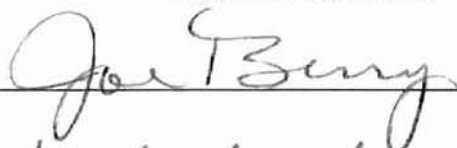
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CHAPTER I

INTRODUCTION

Nutritionally, people eat poultry meat for its high content of high-quality protein and its low fat content, and because of this reason a primary goal of the poultry industry is to maximize lean meat production in broiler chickens.

Poultry meat is slightly higher in protein and lower in fat than beef and other red meats. Additionally, the poultry protein is a rich source of all the essential amino acids (Ensminger, 1992).

The production of broiler meat that contains excess body fat is among one of the problems confronted by the white meat industry, presumably due to an increasing consumer demand for lean tissue. Research showed that high dietary fat consumption, in particular saturated fat, has been associated with the incidence of cardiovascular diseases, diabetes, colon and breast cancer (CAST, 1991).

The reasons of production of broiler meat that contains excess body fat may vary. The main reason of this problem is feeding those birds with high caloric density diets while genetics, sex, environment, protein/calorie ratio, feeding regimes, feed restriction, distress during growth period and

body weight at the processing time have also important impacts on body composition.

It has been shown that changes in energy content of feed affects the body fat content in broilers. Summers et al. (1965) found that increasing energy content of the diet increased body fat while Goodwin et al. (1969) observed that increasing feed energy level with the same crude protein level reduced the protein level in meat on dry weight basis. Petersen (1975) also found that increasing energy level from 11.3 to 13.1 MJ per kg in the diet increased the percentage of fat from 9.2 to 10.7, but an increase in crude protein level from 124 gr. to 189 gr. per 10 MJ metabolizable energy reduced fat content percentage from 13.4 to 7.9 in broilers.

Deaton et al. (1985) searched the age and dietary energy effects on abdominal fat deposition and found that abdominal fat as a percentage body weight increased with increasing age and dietary energy level during 36 to 54 day feeding period. They also noticed that although the increase in body weight were similar for both male and female broilers, abdominal fat as a percentage of body weight increased 23 for males and 38 for females. Edwards et al (1973) also found that the increase in percentage of both total and abdominal fat in females is much higher than in males.

Yu et al. (1990) found that feed restriction of broiler chickens reduced the final body weight of those birds compared to the control group while there was no significant effect of feed restriction on broiler chickens fed three feeding intervals (skip-a-day, daily, hourly). Teeter et al. (1985) force fed broiler chickens and found that live weight gain increased with feed intake up to the 140% consumption of the birds fed ad libitum. They also found that breast, thigh, and drumstick gains were not affected by increased feed intake but depressed by decreased feed intake (75%), and abdominal fat pad weight for increased feed intake level birds was up to 230% of birds fed at 75% of ad libitum level.

Goodwin et al. (1969) analyzed broilers from different strains and found that there were differences between strains in both protein and fat content of these different strains. In 1995 Hancock et al. worked on six strains of commercial broiler stocks at the same environmental and nutrient conditions and concluded that there were highly significant differences between these strains and sexes in their mature weights and nutrient and environmental requirements of those strains would differ.

Exciting multiple specie data has demonstrated L-carnitine efficacy to repartition nutrients away from fat deposition and towards muscle accretion. However, data is

needed further evaluating L-carnitine levels and their interaction with ration caloric density and broiler age.

The objective of this research was to evaluate the dose effects of L carnitine on the 19 (fed with starter ration), 35 (fed with grower ration), 49 (fed with finisher ration) day, as well as overall (from 1 to 49) broiler protein and fat accumulation.

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TABLE 1. L-carnitine content of the animal and vegetable origin feed (Data received from LONZA®)

Animal origin feed sources	L-carnitine (ppm)	Vegetable origin feed sources	L-carnitine (ppm)
Fish meal	85-145	Corn	5-10
Fish bone meal	85	Soybean meal	0-10
Feather meal	125	Barley	0-38
Blood meal	155	Wheat	3-12
Meat bone meal (40%)	150	Milo	15
Whey powder	300-500	Sunflower seed meal	2
Goat milk	15-20	Rape seed	10
Cow milk	6-50	Cottonseed	20-25
Sheep milk	130-320		
Sow milk	120-150		
Skimmed milk products	120-150		
Whey powder (lactose extracted)	800-1000		

TABLE 2. Tissue Carnitine levels of animal tissues (ppm)
(Adapted from Bach, 1982)

Beef	Heart	193
	Liver	26
	Kidney	18
	Muscle	598-885
Pork	Liver	49
Lamb	Muscle	780
	Liver	26
Sheep	Muscle	2090
	Heart	590
	Liver	22
Ram	Muscle	1620-1680
	Heart	595
	Liver	26
Rabbit	Muscle	210
	Liver	110
Chicken	Muscle	45-95
	Liver	6
Hen's egg		0-8

CHAPTER II

REVIEW OF LITERATURE

Introduction

Carnitine is described as a quaternary amine (beta-hydroxy gamma-trimethylaminobutyrate) which is easily soluble in water and has a 161.2 molecular weight (McDowell 1989). Two stereoisomeric forms of carnitine have been found and these are L-carnitine and D-carnitine.

Carnitine was first found by Russian researchers Gulewitch and Krimberg as a component of muscle meat in 1905 (1). Its structural formula was first described in 1927 by Tomita et al. ;



In 1952, Fraenkel et al. found that fat yellow mealworms (*Tenebrio molitor*) required liver extract or brewers yeast to grow. Since this new substrate was essential for this mealworm, they named the new substrate as Vitamin B₇. They isolated carnitine from liver extract and described carnitine as hygroscopic, extremely water soluble, colorless substrate.

Carnitine sources

Carnitine is widely distributed in the nature and present in all animal species, and many microorganisms and plants. Carnitine occurs naturally in the L-carnitine form and this form is the physiologically active form which has important functions in animal tissues.

Mostly food sources of plant origin are low in l-carnitine, while animal-derived foods are high. L-carnitine concentrations of animal and plant origin feedstuff are listed in Table 1. Tissue carnitine concentrations may vary. Carnitine is especially located in skeletal and heart muscle which has about 40 times the concentration of carnitine in blood (McDowell, 1989), and epididymis (Brooks, 1979). Table 2 shows some of animal tissue carnitine concentrations.

Carnitine biosynthesis

Carnitine is synthesized in the animals from two essential amino acids, lysine and methionine (Borum, 1983; LaVell et al., 1979; Feller et al, 1988), and besides these precursors, vitamin C, vitamin B6, niacin, and iron are required by the enzymes as cofactors for the biosynthesis of carnitine (LaVell et al., 1979; Feller et al, 1988). Deficiencies of lysine, methionine, vitamin C, vitamin B6,

and iron were shown to reduce the plasma or tissue carnitine levels presumably due to impaired biosynthesis (Borum, 1983)

During the biosynthesis process, methionine acts as donor of three methyl group, and lysine acts as donor of four-carbon group. LaBadie et al. (1967, In:Borum, 1983) found that protein-bound trimethyllysine is a good source of carnitine in mammalian systems.

Carnitine regulation in the body

There are two main entrances of carnitine into the body, endogenous synthesis, and dietary or exogenous carnitine. Urinary excretion of the carnitine is the only known route in the body to remove carnitine (Feller et al., 1988; Borum, 1983). Over 90% of the filtered carnitine is reabsorbed in the renal tubules (Rebouche et al., 1986 In:Feller et al., 1988) and daily urinary carnitine excretion found high with increasing age (Borum, 1983).

Feller and Rudman (1988) suggested some reasons of cellular deficiency of carnitine or impairment of carnitine function. The first one is reduced capacity for biosynthesis in the animal body due to the deficient intake of lysine, or methionine, or other cofactors such as vitamin C, vitamin B6, niacin and iron. Subnormal acyltransferase I concentration, and subnormal transport of carnitine across the cell membrane is also important factors. Subnormal

transport of carnitine across the mitochondrial membrane, excess carnitine losses due to renal diseases and some drugs and raised tissue requirements for carnitine in rapid growth of the young animals, undernourished animals and high productive animals are also important factors affecting carnitine status of the animal.

Carnitine functions

The most important and well known function of carnitine is the transport of long chain fatty acids into the mitochondrial matrix (Borum, 1983) for β oxidation by the fatty acid oxidation complex. Carnitine acyltransferase is the enzyme responsible for this transportation. Carnitine acyltransferase I converts acyl-CoA to acylcarnitine and acylcarnitine crosses to the mitochondrial matrix. Then carnitine acyltransferase II releases carnitine and Acyl-CoA into the mitochondrial matrix. Acyl-CoA is then catabolized via β oxidation and carnitine is recycled (McDowell, 1989; Lehninger, 1993).

Carnitine also has an important role in thermogenesis in brown adipose tissue. Brown adipose tissue plays an important role in heat production of infant mammals. Fatty acid oxidation in brown adipose tissue is almost completely dependent upon carnitine and ATP. Hahn et al. (1972, In: Borum, 1981) found that carnitine content and

acyltransferase of rat brown adipose tissue increased rapidly immediately after birth and reached the peak level in 10 days and exposure to the cold increases both carnitine and enzyme activity. Hahn et al. (1975, In:Borum, 1983) showed that accumulation of subcutaneously injected [14C]-DL carnitine in the brown adipose tissue of suckling rats was more rapid than skeletal or heart muscle.

Research showed that the control of initiation of ketogenesis is another function of the carnitine. McGarry and Foster (1976) showed that more endogenous carnitine is converted into acylcarnitine when fatty acids (acetate, butyrate, β -hydroxy butyrate) are perfused into rat heart and acetylcarnitine drops to a very low level when glucose is perfused with insulin. Broekhuysen et al. (1965) showed that carnitine can prevent acidosis and ketosis generated during lipid perfusion in the starved dogs.

Carnitine In Animal Nutrition

Carnitine in swine nutrition: Due to the similarities between human and pig in dental characteristics, renal morphology and physiology, skin morphology and physiology, and digestive morphology and physiology (Pond et al, 1978 In:Miller et al., 1987), the pig is the most widely used animal in nutritional research. Miller et al (1987)

compared the digestive systems of human and pig and found that the morphology and physiology of the digestive systems were alike. Most of the carnitine research, especially parenteral nutrition research, was performed on pigs due to this reason. Bohles et al (1983) studied the affect of intravenously administered carnitine effect on parenteral feeding piglets, and they found that carnitine supplementation during parenteral feeding of piglets increased lipolysis and increased the oxidation fatty acids and as a result of better energy supply, nitrogen retention was increased. Bohles et al. (1984) worked on miniature piglets with and without parenteral nutrition. They fed a group of piglets orally and fed the other group parenterally with and without carnitine supplementation and compared the tissue (liver, brain, heart and muscle) amino acid concentration of these two orally and parenterally fed group of animals. They found that tissue amino acid levels of parenterally fed animal are reduced and with carnitine supplementation of these pigs, amino acid levels returned to the normal levels similar to the orally fed group. Theo et al. (1993) also worked on 16 newborn pigs to investigate the carnitine effects on the medium-chain fatty acid metabolism. They infused [14C]medium-chain fatty acids via umbilical arterial catheters providing energy equivalent to 50-175% of the animal's metabolic rate and placed those pigs into the

respiration chambers. They also infused 3 levels of carnitine during medium-chain fatty acids and calculated the fatty acid oxidation rate based on the specific radioactivity of the expired CO_2 . Regardless of the carnitine infused level, they found that carnitine increased the fatty acid oxidation rate up to 20% if the energy provided as medium-chain fatty acids exceeded 50% of the metabolic rate. Bohles et al. (1983) determined the tissue (skeletal muscle, heart, liver, kidney, and brain) total, free, and acyl carnitine levels of mini pigs after seven days of total parenteral nutrition. They also administered carnitine supplementantation to a group of pigs while the other group was not. They noticed that the concentration in liver and kidney were not affected with carnitine supplementation and brain and heart free carnitine levels remained unchanged without carnitine supplementantation but showed an increase with carnitine supplementation.

Carnitine in ruminant nutrition: Snoswell and Henderson (1979) reviewed the role of carnitine on the liver and skeletal muscle of the ruminant animals. They found that muscle tissues of sheep, cattle, and goat contain large amount of free carnitine under normal conditions and carnitine concentration of the same muscle varies with age and is considerably less in young animals. Like the other animals, ewes' milk is a rich source of carnitine and

provides carnitine to the young lamb. Carnitine concentration also increases with diabetes in adult sheep. They noticed that acetate, main energy source for ruminant animal, is readily penetrates into the inner mitochondria and therefore ruminant muscle does not require high amount of carnitine. They noticed that carnitine equiement of muscle tissue is inceased with metabolism of branched-chain amino aids. Liver carnitine concentrations were affected by season and type of feed. Lactation also reduced the liver carnitine concentration. Pregnancy toxemia and diabetes in sheep increased the liver carnitine level dramatically. LaCount et al (1995) searched the response of dairy cows to carnitine administration into rumen and abomasum. They found that apparent digestibility of lipid, energy, and total fatty acids increased with carnitine administration while milk production and dry matter intake remained unaffected. Carnitine concentrations in plasma and liver increased with carnitine administration in both rumen and abomasum. Hill et al. (1995) searched the affect of carnitine supplementation on both heifers and steers separately. They found that average daily gain was higher for carnitine supplemented beef heifers than control group while dry matter intake was similar for both control and carnitine supplemented beef heifers. In contrast to heifers, control group of beef steers had higher average

daily gain at the same dry matter intake. Plasma urea concentrations and volatile fatty acids in rumen were not affected by dietary carnitine treatment.

Carnitine in fish nutrition: Santulli et al. (1986, 1986b) searched the effect of carnitine on growth and lipid metabolism of the sea bass (*Dicentrarchus labrax*). They found that supplementation of carnitine to the diet of cultured sea bass increased the growth rate and decreased the lipid content of the tissues. They also compared the effect of dextrorotatory isomer of L-carnitine (D-carnitine) and found the opposite effect on growth and carcass parameters. Bilinski et al. (1970) searched the effect of coenzyme A and carnitine on muscle, heart, liver, and kidney mitochondrial fractions of Rainbow trout and found that fatty acid oxidation of these fractions were increased by addition of the carnitine.

Carnitine in poultry nutrition: Effects of carnitine supplementation were studied on trained pigeons, turkeys, broilers, and layers. Leibetseder (1995) determined the effects of supplemental carnitine on carcass composition, performance on chickens fed with high-low lysine and methionine and without or 5% fat supplement. He also investigated the effect of niacin and carnitine on yolk

cholesterol level, and effect of carnitine supplementation on hatchability. He found that performance and abdominal fat were affected by dietary fat and amino acids but were not influenced by carnitine. As expected fat supplementation reduced and carnitine addition significantly increased the tissue carnitine levels. In the second trial, he observed that carnitine-nicotinic acid supplementation significantly increased the yolk carnitine levels. Hatchability of the birds fed with 50 or 100 mg/kg carnitine was increased from 83% to 87% and 82.4% to 85.3% in groups and carnitine level of these eggs were higher than control group. Barker et al. (1994) searched the effect of dietary carnitine affects on performance and carcass composition of turkeys and chickens fed low or high fat diets. They found that carnitine supplementation did not affect the performance and body composition of turkeys and broilers in both experiments. Cartwright (1986) reported that .5% carnitine failed to reduce excessive fat accretion in 6 week old broilers fed high energy rations.

Application and benefits of carnitine in poultry

nutrition: The aim of today's broiler market is to produce a lean animal in as few days to market as possible with highest benefits to both consumers and producers. Increased genetic capacity of the meat animals made it necessary to

feed those animals differently from their ancestors to reach the highest genetic potential and feeding these animals differently requires some changes on the diet formulation.

For years, carnitine requirement of the animals was not considered due to endogenous carnitine biosynthesis, but research shows that carnitine become an essential nutrient under certain circumstances, such as limited carnitine biosynthesis in young animals, diets high in fat content, diets low in carnitine and high productive young animals.

The results found in swine, fish, and ruminant nutrition area encouraged us to evaluate the carnitine requirements of broiler chicken. The range of the carnitine supplementation of poultry used in the past research is between 20 (Leibetseder, 1995) to 500 (Cartwright, 1986) ppm and the intent of our research was to evaluate the best carnitine dose applicable to the broiler industry.

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CHAPTER III

DOSE EFFECTS OF L-CARNITINE ON PERFORMANCE, CARCASS CHARACTERISTICS AND BODY FAT COMPOSITION OF 19, 35, AND 49 DAY OLD BROILER CHICKENS

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ABSTRACT

An experiment was conducted utilizing 2100 day old CobbxCobb male broilers to evaluate the dose effect of L carnitine on broiler performance and carcass characteristics and composition. A Completely Randomized Block Design was used to search the L carnitine dose effect (6 treatments, 10 blocks, 10 replicants per treatment). L carnitine levels used in this experiment were 0 (control), 40, 80, 120, 160 and 200 ppm throughout the starter, grower, and finisher periods. No significant interaction of carnitine was noted for performance (feed efficiency, weight gain) or carcass composition ($p>.05$) of birds fed six different carnitine levels. Overall performance and carcass composition data did not differ from the findings of Barker et al. (1994) and Cartwright (1986) and carnitine had no positive effect on carcass or performance data. However the most significant

effect of different carnitine levels was observed on survivability. On overall basis, the survivability rate for broilers were significantly increased by increasing carnitine concentration in the diet.

In summary, carnitine had no significant effect on broiler performance and carcass composition, however significantly increased the survivability.

Introduction

The production of broiler meat that contains excess body fat is among one of the problems for the white meat industry due to an increasing consumer demand for the lean tissue. Saturated fat has been implicated in serious health problems and as if particular cancer. Research showed that high dietary fat consumption, in particular saturated fat, has been associated with the incidence of cardiovascular diseases, diabetes, colon and breast cancer (CAST, 1991).

The causes of broiler meat production that contains excess body fat may vary. The main reason is presumably the feeding of high caloric density diets while genetics, sex, environment, protein/calorie ratio, feeding regimes, feed restriction, distress during growth period and body weight at the processing time also have important impacts on body composition.

Recent studies have shown that L-carnitine has efficacy to repartition nutrients away from fat deposition and towards muscle accretion. The objective of this research was to evaluate the dose effects of L carnitine on the 19 (fed with starter ration), 35 (fed with grower ration), 49 (fed with finisher ration) day, as well as overall (from 1 to 49) broiler protein and fat accumulation.

Materials and Methods

This experiment was conducted utilizing 2100 day old CobbxCobb male broilers to evaluate the dose effect of L carnitine on broiler performance and carcass composition. The reason of using CobbxCobb broilers was their high growth rate, sensitivity to the treatments, and being one of the widely used birds in the poultry industry.

One day old broiler chicks were distributed randomly into 60 floor pens (35 birds per pen), and raised on wood shavings litter. Feed and water were available for ad libitum consumption throughout the experimental period. Mortality and weight of dead birds were recorded daily. Lighting was set at 24 hour daily.

Completely Randomized Block Design was used to examine the L carnitine dose effect. The 60 pen house was divided into 5 blocks and 6 dietary carnitine treatments were randomly assigned into these blocks (10 pens per treatment).

L carnitine levels used in this experiment were 0 (control), 40, 80, 120, 160 and 200 ppm and these levels were the same for the starter, grower and finisher periods.

The birds were fed ad libitum with starter ration until 19 days of age, grower ration until 35 days of age, and finishing ration until the processing (49 day). Corn-Soybean based rations were used since it is most commonly employed by the poultry industry. Energy and Crude protein values of starter, grower, and finisher rations were arranged to satisfy NRC requirements of broilers and essential amino acid concentrations in the diet were slightly higher than NRC requirements. Energy and Crude protein levels of starter, grower, and finisher diets used in the experiment are shown below:

	Energy (Kcal ME/Kg)	Crude Protein (%)
Starter Ration	3185	24
Grower Ration	3200	21
Finisher Ration	3200	19

On day 19, 35, and 49, group weight and feed consumption of birds were recorded. At the same time on day 19 6 birds, on day 35 3 birds, and on day 49 4 birds were selected randomly. These birds were wing banded, weighed individually, and sent to the OSU's processing unit. In the processing unit birds were weighed, electrically stunned,

bled for 15 minutes and passed through a scalding vat, and plucking machine. Then the birds were decapitated, eviscerated, and legs were removed (Belay, 1991). Carcasses were weighed in air and water and recorded to calculate the specific gravity (Teeter and Smith, 1985). Hot carcasses were placed into chilled water for 2-3 hours and weighed. Breasts removed from the carcass and breast yield was recorded. Abdominal fat pad was removed from the ischium, cloaca, and adjacent abdominal muscles (Belay, 1991), weighed, and recorded. Carcasses were then frozen (including breast and abdominal fat pad) for dry matter analysis.

Estimated carcass fat values were obtained by the predictive equation developed in Oklahoma State University's Avian Climatological Center (Wiernusz et al., 1994) from carcass specific gravity of broiler chickens.

Data for all response variables were subjected to analysis of variance using the General Linear Models procedure of SAS (SAS, 1985), and treatment means were compared using least square analysis of variance in case of significant F statistic (Steel and Torrie, 1960).

Results and Discussion

Different dose effects of carnitine on starter, grower, and finisher broilers' weight gain, feed efficiency,

survivability and carcass characteristics are presented in tables 4, through Table 11. No significant interaction of carnitine was noted for performance (feed efficiency, weight gain) or carcass composition ($p > .05$) of these birds fed six different carnitine levels. Even though there were very slight numerical increase in hot carcass and chilled carcass weight with carnitine addition, overall performance and carcass composition data did not differ from the findings of Barker et al. (1994) and Cartwright (1986). In both of these studies, carnitine failed to reduce excessive fat accretion in broilers fed high energy rations. However the most significant effect of different carnitine levels was on survivability. On overall basis, the survivability rate for broilers were significantly increased by increasing carnitine concentration in the diet.

Utilization of carbohydrate and lipid in heart muscle is closely coupled to the energy needs of the heart and when carbohydrate and lipid sources are present together, fatty acid is used in preference to glucose in heart tissue (Neely and Morgan, 1974).

The rate of fatty acid β oxidation in heart is dependent upon the available exogenous fatty acids, the rate of acetyl-CoA oxidation by the citric acid cycle, and utilization of energy by the tissue (Neely and Morgan, 1974). It is also proposed that the flux of fatty acids via

β oxidation in heart is controlled by the mitochondrial Acetyl CoA:Free CoA ratio since they are product and substrate and their concentrations is the regulatory signal for β oxidation (Schulz, 1994). Wang et al. (1991) demonstrated that carnitine addition to the respiring heart mitochondria decreased Acetyl-CoA:Free CoA ratio and resulted in stimulation of β oxidation. This regulatory mechanism is not effective in liver due to other pathways involved in the regeneration of Free CoA from Acetyl CoA such as ketone body synthesis (Schulz, 1994).

Research showed that mitochondria from heart muscle have the highest capacity to oxidize acyl-carnitine of any tissue studied (Neely and Morgan, 1974). When oxidative phosphorylation and fatty acid oxidation were stimulated by increased cardiac work, the tissue levels of fatty acyl-carnitine increased in association with decreased levels of fatty acyl-CoA and Acetyl-CoA and this indicates that oxidation of acyl-carnitine limited maximal rates of fatty acid utilization at fast rates of ATP consumption.

As a result of increasing survivability overall live yield data per 100 broiler chickens started (adjusted for survivability) increased. Overall feed efficiency (feed/gain ratio) increased from 2.46 (control) to 2.29 (40

ppm), 2.24 (80 and 120 ppm), 2.18 (160 ppm), and 2.20 (200 ppm) respectively by addition of carnitine.

In summary, carnitine had no significant effect on broiler performance and carcass composition, however significantly increased the survivability and overall live yield data. Previous multiple species data also showed that carnitine is an important factor under certain circumstances especially in the young animals, and it is proved that carnitine addition to the diet increases the tissue carnitine concentration and may have beneficial effect on the animal's nutrition status presumably due to sparing effect of carnitine on its precursors (Lysine, methionine).

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TABLE 1. Composition of the starter diet

Ingredients	%
Corn, ground	49.629
Soy meal (47.5 %)	41.369
Soybean oil	5.335
Dicalcium phosphate (CDP)	1.799
Limestone	0.979
Salt	0.250
Vitamin mix ^a	0.200
Methionine (99 %)	0.220
Trace mineral mix ^b	0.100
Coban	0.050
Selenium mix	0.050
Ethoxyquin	0.020
Energy (Kcal ME/Kg)	3185
Crude Protein (%)	24

^aVitamin mix is supplied in the following per kilogram of diet: Vit. A, 17500 IU; Cholecalciferol, 5000 IU; Vitamin E, 25 IU; Vitamin B₁₂, 0.03 mg; Riboflavin, 15 mg; Niacin, 75 mg; D panthothenic acid, 25 mg; Cholin, 705.5 mg; Menadione, 5 mg; Folic acid, 1.5 mg; Pyridoxine, 6.25 mg; Thiamine 3.03 mg; D-biotin, 0.127 mg.

^bTrace mineral mix is supplied in the following per kilogram of diet: Manganese, 120 mg; Zinc, 100 mg; Copper, 10 mg; Iodine, 2.5 mg; Calcium, 135 mg; Iron, 75 mg; Selenium, 0.15 mg.

TABLE 2. Composition of the grower diet

Ingredients	%
Corn, ground	58.842
Soy meal (47.5 %)	33.563
Soybean oil	4.624
Dicalcium phosphate (CDP)	1.267
Limestone	0.933
Salt	0.243
Vitamin mix ^a	0.203
Methionine (99 %)	0.101
Trace mineral mix ^b	0.101
Coban	0.051
Selenium mix	0.051
Ethoxyquin	0.020
Energy (Kcal ME/Kg)	3200
Crude Protein (%)	21

^aVitamin mix is supplied in the following per kilogram of diet: Vit. A, 17500 IU; Cholecalciferol, 5000 IU; Vitamin E, 25 IU; Vitamine B₁₂, 0.03 mg; Riboflavin, 15 mg; Niacin, 75 mg; D panthothenic acid, 25 mg; Cholin, 705.5 mg; Menadione, 5 mg; Folic acid, 1.5 mg; Pyridoxine, 6.25 mg; Thiamine 3.03 mg; D-biotin, 0.127 mg.

^bTrace mineral mix is supplied in the following per kilogram of diet: Manganese, 120 mg; Zinc, 100 mg; Copper, 10 mg; Iodine, 2.5 mg; Calcium, 135 mg; Iron, 75 mg; Selenium, 0.15 mg.

TABLE 3. Composition of the finisher diet

Ingredients	%
Corn, ground	65.450
Soy meal (47.5 %)	28.212
Soybean oil	3.619
Dicalcium phosphate (CDP)	1.022
Limestone	0.920
Salt	0.286
Vitamin mix ^a	0.204
Methionine (99 %)	0.061
Trace mineral mix ^b	0.102
Coban	0.051
Selenium mix	0.051
Ethoxyquin	0.020
Energy (Kcal ME/Kg)	3200
Crude Protein (%)	19

^aVitamin mix is supplied in the following per kilogram of diet: Vit. A, 17500 IU; Cholecalciferol, 5000 IU; Vitamin E, 25 IU; Vitamine B₁₂, 0.03 mg; Riboflavin, 15 mg; Niacin, 75 mg; D panthothenic acid, 25 mg; Cholin, 705.5 mg; Menadione, 5 mg; Folic acid, 1.5 mg; Pyridoxine, 6.25 mg; Thiamine 3.03 mg; D-biotin, 0.127 mg.

^bTrace mineral mix is supplied in the following per kilogram of diet: Manganese, 120 mg; Zinc, 100 mg; Copper, 10 mg; Iodine, 2.5 mg; Calcium, 135 mg; Iron, 75 mg; Selenium, 0.15 mg.

TABLE 4. Effects of various levels of L-carnitine on body weight gain of starter , grower, and finisher broiler chickens.

Carnitine (ppm)	<u>0</u>	<u>40</u>	<u>80</u>	<u>120</u>	<u>160</u>	<u>200</u>		
	Body Weight Gain (gr)						Pooled SEM	Probability
starter	592	588	598	590	596	597	4.45	0.4595
grower	1657	1632	1647	1663	1629	1659	14.8	0.4997
finisher	2625	2613	2645	2584	2580	2633	24.2	0.3329

TABLE 5. Effects of various levels of L-carnitine on feed efficiency of 19 , ,
35, and 49 day old broiler chickens.

Carnitine (ppm)	<u>0</u>	<u>40</u>	<u>80</u>	<u>120</u>	<u>160</u>	<u>200</u>		
	Feed Efficiency (F/G)						Pooled SEM	Probability
Starter (1-19)	1.26	1.24	1.21	1.22	1.23	1.24	0.016	0.2599
Grower (1-35)	1.71	1.70	1.69	1.67	1.69	1.68	0.012	0.5384
Finisher (1-49)	2.06	2.138	2.122	2.132	2.10	2.11	0.030	0.1663

TABLE 6. Effects of various levels of L-carnitine on survivability of starter (1-19 day), grower (19-35), finisher (35-49), and overall (1-49) broiler chickens.

Carnitine (ppm)	<u>0</u>	<u>40</u>	<u>80</u>	<u>120</u>	<u>160</u>	<u>200</u>		
	Survivability (%)						Pooled SEM	Probability
Starter	98.3	98.8	99.4	99.7	98.9	98.6	0.53	0.4409
Grower	97.9	97.6	98.6	97.9	99.0	99.7	0.77	0.4330
Finisher	93.8	97.1	96.8	97.6	98.4	98	1.13	0.0779
Overall	90.3	93.8	94.5	95.3	96.3	96.3	1.46	0.0521

TABLE 7. Effects of various levels of L-carnitine on carcass characteristics of broiler chickens (Average values of starter, grower, and finisher periods).

Carnitine (ppm)	Hot Dressing (HCW/Live W)	Chilled Dressing (CCW/Live W)	Specific Gravity (gr)	Breast Yield (% BW)	Abdominal Fat Pad (gr)
0	662	691	1049	182	14.8
40	664	694	1050	184	13.5
80	666	696	1049	181	13.3
120	664	693	1050	181	15.2
160	665	693	1050	184	15.3
200	665	695	1050	181	17.8
Pooled SEM	1.97	2.65	0.64	1.33	1.50
Probability	0.7856	0.9227	0.9297	0.2165	0.4998

TABLE 8. Effects of various levels of L-carnitine on carcass characteristics of starter broilers.

Carnitine (ppm)	Hot Dressing (HCW/Live W)	Chilled Dressing (CCW/Live W)	Specific Gravity (gr)	Breast Yield (% BW)	Abdominal FatPad (gr)
0	0.564	0.600	1052	17.8	3.01
40	0.567	0.608	1054	17.9	3.24
80	0.564	0.602	1052	17.5	3.15
120	0.563	0.600	1052	17.6	3.30
160	0.566	0.598	1052	17.8	3.17
200	0.566	0.604	1054	17.6	3.00
Pooled SEM	0.0034	0.0045	1.1	0.23	1.2
Probability	0.7856	0.9227	0.9297	0.2165	0.4998

TABLE 9. Effects of various levels of L-carnitine on carcass characteristics of grower broilers.

Carnitine (ppm)	Hot Dressing (HCW/Live W)	Chilled Dressing (CCW/Live W)	Specific Gravity (gr)	Breast Yield (% BW)	Abdominal FatPad (gr)
0	0.690	0.716	1048	16.9	13.34
40	0.691	0.714	1049	17.3	9.09
80	0.696	0.718	1050	17.1	9.62
120	0.690	0.711	1050	16.7	15.37
160	0.695	0.717	1051	17.3	16.81
200	0.688	0.713	1049	17.0	22.31
Pooled SEM	0.034	0.0045	1.1	0.23	4.1
Probability	0.7856	0.9227	0.9297	0.2165	0.4998

TABLE 10 Effects of various levels of L-carnitine on carcass characteristics of finisher broilers.

Carnitine (ppm)	Hot Dressing (HCW/Live W)	Chilled Dressing (CCW/Live W)	Specific Gravity (gr)	Breast Yield (% BW)	Abdominal FatPad (gr)
0	0.732	0.759	1048	20.1	28.09
40	0.734	0.761	1046	20.1	28.14
80	0.738	0.767	1047	19.6	27.22
120	0.741	0.770	1047	19.9	26.98
160	0.735	0.765	1046	20.1	26.14
200	0.742	0.768	1047	19.7	28.03
Pooled SEM	0.034	0.0045	1.1	0.23	1.2
Probability	0.7856	0.9227	0.9297	0.2165	0.4998

TABLE 11. Effects of various levels of L-carnitine on carcass fat composition of broiler chickens.

Carnitine (ppm)	<u>0</u>	<u>40</u>	<u>80</u>	<u>120</u>	<u>160</u>	<u>200</u>		
	(% of BW)						Pooled SEM	Probability
Starter	10.59	10.11	10.74	10.72	10.55	10.07	0.34	0.9297
Grower	11.89	11.70	11.36	11.16	10.95	11.46	0.34	0.9297
Finisher	11.97	12.35	12.19	12.06	12.36	12.07	0.34	0.9297
Overall	11.48	11.39	11.43	11.31	11.28	11.12	0.20	0.9297

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