EFFECTS OF L-CARNITINE ON GROWTH PERFORMANCE, APPARENT NUTRIENT DIGESTIBILITY, AND WHOLE BODY COMPOSITION IN WEANLING PIGS

By

MICHAEL JAMES RINCKER

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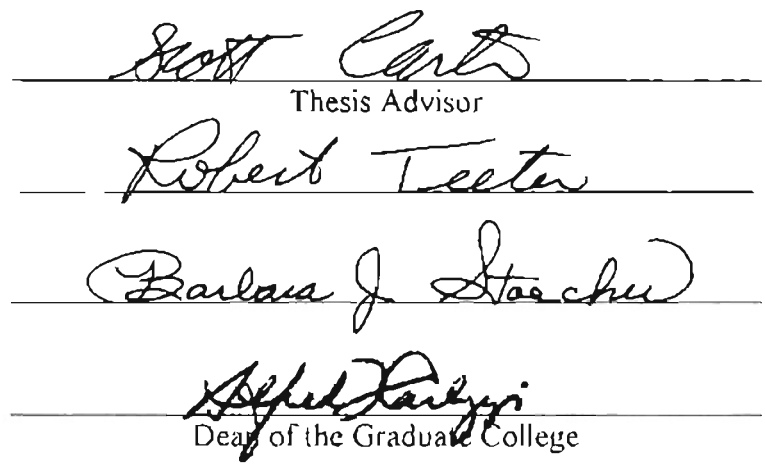

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Dean of the Graduate College

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Introduction

The swine industry, similar to many industries, has undergone a technological revolution during the last decade of the 20th century. Society has seen a change from the small, family farm, where pigs were raised to satisfy a family's need, to large, vertically-integrated swine farms that supply enough pork for the world human food demand. Because of these changes, producers are imposed with new challenges on a daily basis. These challenges range from genetically advanced breeding stock and improved management techniques and facilities, to genetically enhanced feed ingredients, new environmental regulations, and new pharmaceutical drug policies. All of these technological advancements have one common goal in the swine industry: to make swine production economically feasible, allowing the producer to manage a profitable swine operation.

Extensive research has been conducted in the area of feed ingredients and their nutrient digestibility, and subsequent effects on growth performance of swine. This is due to the fact that feed is the major economic input to the swine production system, representing over 65% of all production expenses. The stage of production where producers believe animals are hindered the most, and improvements in growth performance are lost, as a result of poor nutrient digestibility, is immediately post-weaning.

Common practice in today's swine industry is weaning pigs at 21 days of age or younger. As a result of implementing this early-weaning management strategy, a

detrimental effect on performance of weanling pigs is conceivable. This unfavorable dilemma, known as post-weaning lag, is the result of several factors. Upon weaning, the baby pig's diet is being altered from a strictly liquid milk diet to a pelleted food diet. At this early age of the animal, the gastrointestinal tract of the pig is immaturely developed and enzyme activity is limited, resulting in inefficient utilization of added dietary plant protein sources and fat sources. Another cause of post-weaning lag is environmental stress. At weaning, animals are removed from their natural surrounding and grouped in an unfamiliar nursery room with unknown animals. A third cause leading to post-weaning lag is immunological stress and the exposure of newly weaned pigs to foreign pathogens. Therefore, if the producer can make the transition into the nursery room as comfortable as possible for the weanling pig, they can minimize post-weaning lag.

The greatest challenge during the early post-weaning period is maintaining feed consumption in the young pig. The newly weaned pig, particularly during the first few days, can not consume sufficient quantities of feed to meet their energy demands for growth. Thus, complex weanling pig diets, consisting of nutrient-dense ingredients that are highly digestible and appropriate for the pig's stage of physiological development, are formulated and fed. However, at this stage of development, the weanling pig is unable to efficiently utilize and gain the beneficial effects from added dietary fat sources. The inefficient utilization of these dietary fat sources could possibly be attributed to minimal stores of L-carnitine in the newly weaned pig. Therefore, to meet the energy demands, the feeding regime of newly weaned pigs must consist of readily available carbohydrate sources that can provide a large portion of the energy supply. However, it is hypothesized that altering the metabolic processes of beta-oxidation can garner more efficient

utilization of energy found in dietary fat sources; thus, improving growth performance and body composition of the young pig by partitioning more nutrients from lipid deposition to protein accretion.

Thus, the objectives of this thesis were to evaluate the effects of supplementing L-carnitine, an intermediate in lipid metabolism, to the diet of weanling pigs and its subsequent effects on growth, feed efficiency, nutrient digestibility, and whole body composition of weanling pigs.

CHAPTER 1

Literature Review

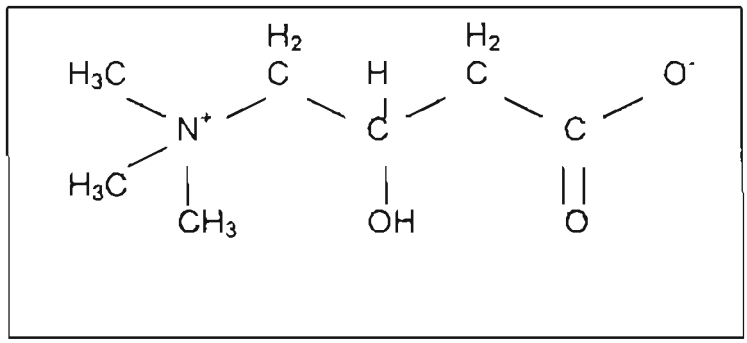
History

Carnitine, a name derived from the Latin word *caro* or *carnis*, meaning flesh, was discovered in muscle extract by two independent groups of scientists in 1905 (Gulewitsch and Krimberg, 1905; Kutscher, 1905). In the same year that Gulewitsch and Krimberg (1905) isolated carnitine from muscle extract and assigned it the empirical formula $C_7H_{15}NO_3$, another nitrogenous base was isolated from the same muscle extract by Kutscher (1905). Kutscher termed this compound "Novain" and assigned it the empirical formula $C_7H_{18}NO_2AuCl_4$. In 1908, Krimberg (1908) was able to prove, using Kutscher's isolation procedure for Novain, that a significant amount of carnitine was obtained from the original material. From this he concluded that Novain and carnitine were the same compound.

Twenty-two years would pass before scientific proof could validate that the empirical formula $C_7H_{15}NO_3$, assigned to carnitine by Gulewitsch and Krimberg, was correct. In 1927, Tomita and Sendju were successful at separating the synthetic α- and β-hydroxy isomers using brucine salts and found that the gold chloride and other derivatives of the isomers melted at the same temperature as derivatives of the natural compound. Additionally, by synthesizing natural carnitine through the methylation of β-hydroxy-γ-aminobutyric acid, Tomita and Sendju (1927) succeeded in establishing the

chemical structure (Figure 1.1) of carnitine as γ-trimethyl-β-hydroxy-butyrobetaine. Furthermore, carnitine is also known as 3-hydroxy-4-trimethyl ammonium butyrate.

Figure 1.1. Chemical structure of carnitine.



The first indication of a requirement for carnitine arose in research evaluating the nutritional requirements of the meal worm, *Tenebrio molitor* (Fraenkel and Blewett, 1947). Larvae of the meal worm failed to grow or survive on a synthetic diet consisting of casein, glucose, cholesterol, a salt mixture, and nine B vitamins; however, optimal growth ensued upon addition of small quantities of yeast or liver preparations to the medium. The factor in yeast, which aided in the survival and growth of *Tenebrio molitor*, was first named "vitamin B_T" (Fraenkel, 1948; Fraenkel et al., 1950) to indicate its place in the B-group of vitamins, and the "T" standing for *Tenebrio*. Concentrations of the vitamin B_T were found to be present in yeast, milk, and many animal tissues (Fraenkel, 1951). The identity of vitamin B_T as carnitine was established in 1951. Carter et al. (1952) reported, similar to carnitine, the pure crystalline form of vitamin B_T was highly hygroscopic, was soluble in water and the lower alcohol's, gave only end absorption in the ultraviolet, and had a specific rotation of $[\alpha]_D^{22} = -23.5°$. Furthermore, the empirical

formula of vitamin B_T was $C_7H_{15}NO_3$, and upon dehydration with sulfuric acid yields crotonobetaine, like carnitine. Also, both carnitine and vitamin B_T are present in muscle extract to the extent of 1.5-3.0% (Carter et al., 1952).

While the chemistry and physiology of carnitine had been determined, there was still uncertainty as to the exact physiological function of carnitine. In 1957, the effects of carnitine on palmitic acid oxidation by liver tissue slices, skeletal muscle particulates, and heart preparations were evaluated (Fritz, 1959). These authors proposed that the presence of carnitine in muscle and other tissues was to facilitate the transfer of long chain fatty acids to the enzymatically active intra-mitochondrial sites for fatty acid oxidation.

Later, Fritz et al. (1962) reported that altering the structure of the compound in any of a number of ways terminates the catalytic action of carnitine on long-chain fatty acid metabolism. Examples of altering the structure of carnitine include, removal of the hydroxy group attached to the β-carbon, replacement of the carboxyl group with a cyano, an alcohol, or an amide grouping, or by substitution of a primary amino grouping for the trimethyl-ammonium moiety of carnitine (Fritz et al., 1962).

With the discovery that carnitine is synthesized from the essential amino acids lysine and methionine, it was suggested that a carnitine deficiency may be a result of marginal or deficient intake of lysine (Rebouche, 1992). Subsequent studies in rats demonstrated that diets deficient in lysine resulted in the characteristic signs and symptoms of a lysine deficiency; however, no abnormalities that could be attributed to a carnitine deficiency were observed.

Scientist began to look at the role of carnitine as a dietary nutrient in human nutrition when Engel and Angelini (1973) reported the first instance of carnitine

deficiency in human skeletal muscle with associated lipid storage myopathy. Since this first report, over 20 cases with systemic carnitine deficiency have been described (Kerner and Hoppel, 1998). Because of this an abundance of research has been conducted seeking to identify the nutritional aspects of carnitine in every human physiological stage of development. The majority of research has focused on premature and full-term infants (Rebouche, 1992).

Initial studies evaluating the role of carnitine and its importance in swine nutrition were conducted in the late 1980's (Newton and Haydon, 1988, 1989; Weeden et al., 1990, 1991). Current research in this area deals with determining the effects of dietary carnitine on growth performance, nutrient digestibility, and carcass characteristics in various physiological stages of maturity of swine.

Biosynthesis of Carnitine

Two isomers of carnitine exist, the D- and L- forms; however, only the L-form is biologically active and occurs in nature (Ji, 1995). The first convincing evidence for carnitine biosynthesis in animals was obtained from chick embryos, which contained significant amounts of carnitine, whereas none was found in eggs (Fraenkel, 1953).

The key finding to establishing the pathway of carnitine biogenesis lay in discovering the origin of γ-butyrobetaine by Linneweh (1928). Two more significant clues to the mechanism of carnitine biosynthesis were discovered in 1961. It was shown that the methyl groups of carnitine are donated by the amino acid methionine (Bremer, 1961; Wolf and Berger, 1961), and that γ-butyrobetaine is converted to carnitine (Bremer, 1962; Lindstedt and Lindstedt, 1961). The next important discovery was made in 1971,

when researchers showed that labeled lysine is converted to carnitine in the fungus, *Neurospora crassa* (Horne and Broquist, 1973) and rats (Tanphaichitr and Broquist, 1973). This discovery resulted in the establishment of the origin of the carnitine four carbon chain. These key findings were the foundation for several studies (Cox and Hoppel, 1973; Hulse et al., 1978) on the biosynthesis of carnitine in both animals and microorganisms that resulted in the establishment of a metabolic pathway for the synthesis of carnitine.

The initial precursors of L-carnitine synthesis are the amino acids lysine and methionine. In *Neurospora crassa*, the methylation of free lysine with S-adenosylmethionine provides the three methyl groups (Rebouche and Broquist, 1976). However, in mammals the pathway of carnitine biosynthesis is unique in that free lysine is not subject to methylation, but rather, lysine residues contained in body proteins are the substrates for S-adenosylmethionine-dependent methyltransferases which produce the intermediate peptide-bound trimethyl-lysine (Odle et al., 2000). The primary intermediate, ∈-N-trimethyl-L-lysine, is synthesized only as a post-translational modification of protein synthesis and the intermediate is released by normal processes of protein turnover for carnitine synthesis. A number of proteins contain one or more ∈-N-trimethyl-L-lysine residues, including histones, actin, myosin, and calmodulin (Rebouche, 1992).

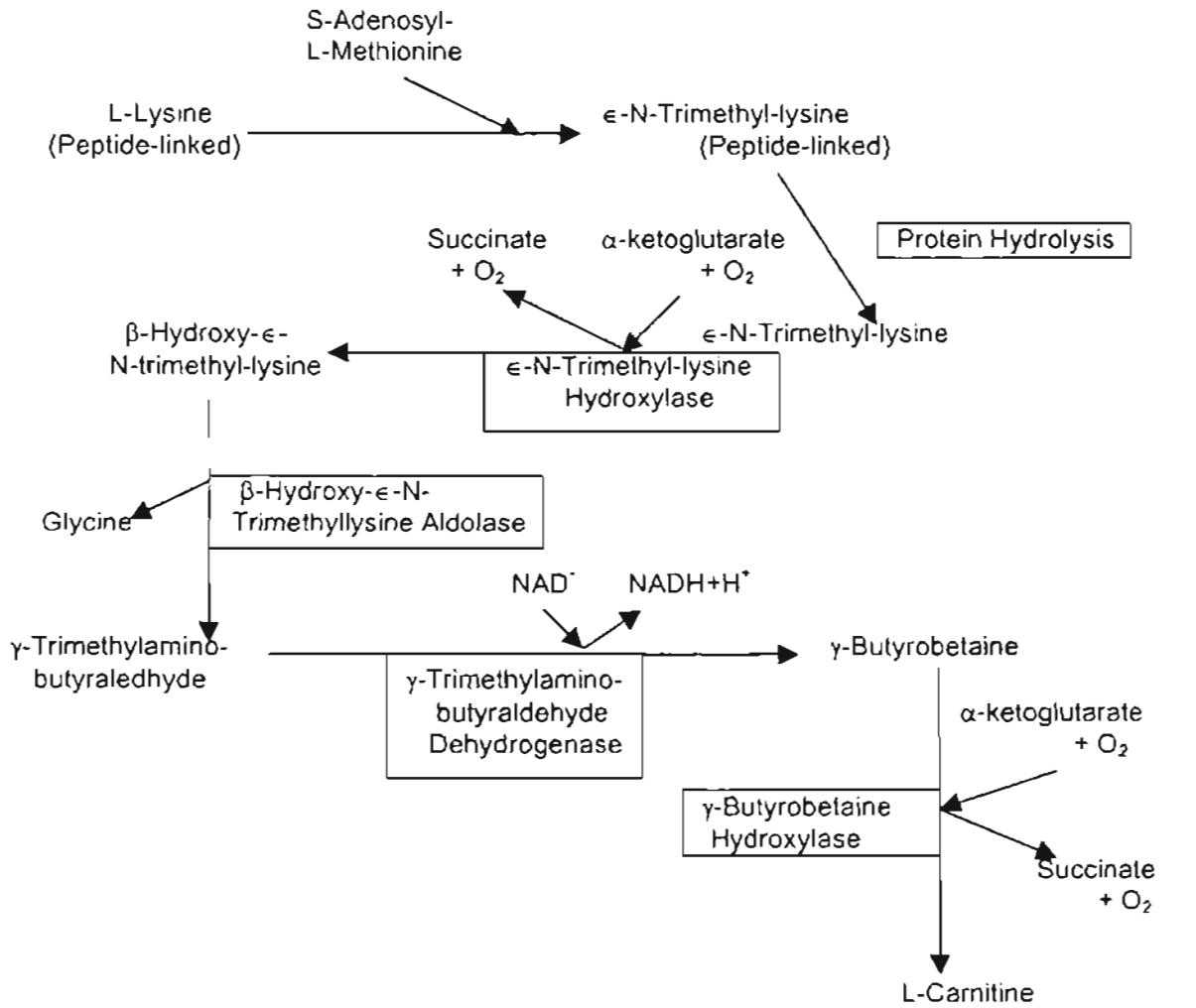
∈-N-Trimethyl-L-lysine destined for carnitine biosynthesis undergoes four enzymatic reactions. The first of these involves hydroxylation of the substrate to form β-hydroxy-∈-N-trimethyl-L-lysine. This reaction is catalyzed by ∈-N-trimethyl-L-lysine hydroxylase, an α-ketoglutarate-requiring dioxygenase found in the mitochondria of

liver, kidney, heart, skeletal muscle, and brain tissues. In the next step, β-Hydroxy-∈-N-trimethyl-L-lysine undergoes aldol cleavage to glycine and γ-trimethylaminobutyraldehyde. A pyridoxal phosphate-requiring enzyme known as serine hydroxymethyltransferase catalyzes this reaction. γ-Trimethylaminobutyraldehyde is oxidized to γ-butyrobetaine by specific and nonspecific NAD^+-requiring aldehyde dehydrogenases. The final reaction is a second hydroxylation at the β carbon of γ-butyrobetaine to form L-carnitine. The enzyme catalyzing this reaction, γ-butyrobetaine hydroxylase, also is an α-ketoglutarate-requiring dioxygenase (Rebouche, 1992). γ-Butyrobetaine hydroxylase is present only in a few tissues, and it also shows species variations in tissue distribution. In all species, this enzyme is found in the liver. However, the enzyme is also present in the kidney and brain of humans (Rebouche and Engel, 1980). As well, the enzyme is present to a small extent in the testis of rats, but is absent from all other tissues (Haigler and Broquist, 1974). The metabolic pathway of carnitine biosynthesis is detailed in Figure 1.2.

The pathway of carnitine biosynthesis contains some rate-limiting steps that can control the yield of carnitine synthesis. Olson and Rebouche (1987) reported that γ-butyrobetaine hydroxylase, a rate-limiting enzyme, varied with physiological stage of maturity in rats and humans. The hepatic γ-butyrobetaine hydroxylase activity in human infants is about 25% of that in adults. Furthermore, determinations of ∈-N-trimethyl-L-lysine content of various tissue proteins and their rates of turnover, and the rate of carnitine biosynthesis, indicated that the availability of ∈-N-trimethyl-L-lysine and the rate of carnitine biosynthesis were of the same order of magnitude (Rebouche, 1982). Because the only endogenous source of ∈-N-trimethyl-L-lysine for carnitine biosynthesis

is through protein turnover containing this amino acid, studies involving the oral supplementation of ∈-N-trimethyl-L-lysine to rats (Rebouche et al., 1986) and humans (Olson and Rebouche, 1987; Rebouche et al., 1989) elicited an increase in the rate of carnitine biosynthesis.

Figure 1.2. Mammalian pathway of L-carnitine biosynthesis[a]



[a]Adapted from Rebouche (1992).

Carnitine is not degraded systemically in humans; therefore, the rate of carnitine excretion provides a reasonable indirect estimate of carnitine synthesis. Measuring the

steady-state rate of carnitine excretion by strict vegetarian adults and children, the rate of carnitine biosynthesis in humans is estimated to be approximately 2 μmol · kg body weight^{-1} · day^{-1} (Rebouche and Seim, 1998).

In biological systems carnitine exists either as free carnitine or as esters of short-, medium-, or long-chain organic and fatty acids. Thus, when performing carnitine assays, diet, plasma, and tissue samples should be analyzed for both free carnitine and acylcarnitine concentrations (Owen, 1996). Parvin and Pande (1977) describe the preferred method for the determination of picomole amounts of total carnitine and free carnitine in the presence of short-chain acylcarnitines. The method is based on the conversion of radioactive acetyl-CoA to acetylcarnitine in the presence of carnitine acetyltransferase and oxidized glutathione or N-ethylmaleimide to pull the reaction to near completion, so that linear standard curves are obtained over a wide range. The rapid separation of acetylcarnitine from acetyl-CoA is accomplished by selective absorption of acetyl-CoA on charcoal in the presence of acid and ethanol. The charcoal separation method also allows a direct and precise assay of carnitine acetyltransferase, which is particularly useful for studies requiring low levels of acetyl-CoA (Parvin and Pande, 1977).

Metabolism of Carnitine

Carnitine transport and carnitine absorption have been evaluated in a variety of intestinal preparations ranging from rats and pigs to guinea pigs and humans. In rats (Rebouche et al., 1984) and humans (Rebouche and Chenard, 1991), approximately 54-87% of dietary carnitine is absorbed. Additionally, Gudjonsson and coworkers reported

(1985) the rapid absorption of L-carnitine by the small intestinal mucosa of rats *in vivo* with subsequent release of carnitine slowly into the circulation. By the observations that L-carnitine was absorbed twice as rapidly as the D isomer and the process was saturable, Gross and Henderson (1984) suggested the presence of a specific carrier for carnitine in the mucosal membrane. However, there is some discrepancy on the mode of transport in carnitine absorption. Gross et al. (1986) reported facilitated diffusion of carnitine into isolated guinea pig enterocytes, whereas Hamilton et al. (1986) concluded that carnitine is taken up by active transport and passive diffusion in human intestinal biopsy specimens. Using jejunal perfusions *in vivo* in human adults, Li et al (1992) concluded that carnitine is absorbed by active mechanisms during a normal meal, while pharmacological doses of carnitine are absorbed primarily by passive diffusion.

Whatever the mode of action in carnitine absorption, mechanisms are present in most tissues that establish and maintain concentration gradients between extracellular and intracellular carnitine pools. As well, carnitine concentrations typically are higher in tissue than in extracellular fluid compartments, with human skeletal and cardiac myocytes normally having over 50 times the concentration of carnitine than that of plasma (Rebouche and Seim, 1998).

The fate of absorbed carnitine in normal metabolic activities is the formation of acylcarnitine esters. Of the carnitine taken up from the lumen into the intestinal mucosa up to 50% is acetylated (Gudjonsson et al., 1985). The predominant acylcarnitine formed both intracellularly and in circulation is acetyl-L-carnitine, which participates in both anabolic pathways and catabolic pathways in cellular metabolism. Intracellular long-chain acylcarnitine esters are produced to transport the fatty acyl moieties across the

mitochondrial membrane into the mitochondrial matrix. Short- and medium-chain acylcarnitine esters are formed in mitochondria and peroxisomes, in part as a means of removing organic acids from these organelles as high-energy compounds (Borum, 1986).

Carnitine that is not absorbed in the small intestine is almost totally degraded in the large intestine by bacteria and primarily excreted in the urine as metabolites, but with traces being evident in feces. Enzymes of mammalian origin do not degrade L-carnitine, but microorganisms in the gastrointestinal tract are entirely responsible for metabolite formation (Rebouche et al., 1984; Seim et al., 1985).

Whether it is endogenous or exogenous carnitine, there are a number of different pathways of carnitine metabolism in microorganisms. During aerobic conditions, the enzyme carnitine dehydrogenase induces the reduction of L-carnitine at carbon 3, producing dehydrocarnitine (Aurich and Lorenz, 1959). Subsequently, dehydrocarnitine is cleaved to form glycine betaine by enzyme extracts and presumably acetate (Lindstedt et al., 1967). Under anaerobic conditions enterobacteria do not assimilate the carbon and nitrogen of carnitine, but they do metabolize it via crotonobetaine to γ-butyrobetaine in the presence of other carbon and nitrogen sources. *Escherichia coli* isolated from the intestinal lumen of a rat was first shown to reduce L-carnitine to γ-butyrobetaine (Seim et al., 1979). Two enzymatic reactions, a dehydration and a reduction, are involved in the transformation. Thus, the primary function of carnitine in this pathway may be as an electron acceptor during anaerobic growth of enterobacteria in the absence of preferred substrates (Seim et al., 1982).

Another metabolic pathway of carnitine degradation was first demonstrated in *Serratia marcescens* by Unemoto et al. (1966). It was shown that carnitine is

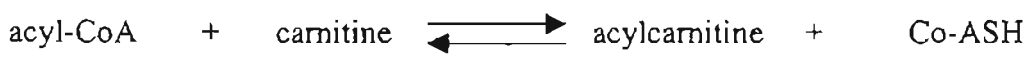
metabolized initially by cleavage of the C_4-N bond, forming trimethylamine and malate. Although it is evident that the trimethylamine- and γ-butyrobetaine forming pathways are active in intestinal flora, it has not been demonstrated that complete degradation of carnitine occurs in this pathway.

A combination of absorption of carnitine from dietary sources, rate of carnitine biosynthesis, and highly efficient re-absorption of carnitine maintain homeostasis of extracellular and intracellular carnitine concentrations in mammals.

Functions of Carnitine

Fritz (1959) first documented the idea that the function of carnitine was to facilitate the transport of long-chain fatty acids across the mitochondrial membrane. Since his discovery, it has become an established fact (Fritz and Yue, 1963; Bray and Briggs, 1980) that the primary metabolic role of carnitine is as a cofactor for enzymes that shuttle long-chain fatty acids across the otherwise impermeable inner mitochondrial membrane into the matrix of the mitochondria. Once in the mitochondrial matrix, long-chain fatty acids are utilized in the production of adenosine triphosphate (energy) via β-oxidation and oxidative phosphorylation. As well, carnitine is involved in the facilitation of activated medium- and short-chain organic acids from peroxisomes to mitochondria, referred to as the carnitine shuttle.

The functions of carnitine are mediated by one of three groups of carnitine acyltransferase (CAT) enzymes, which catalyze the reaction:



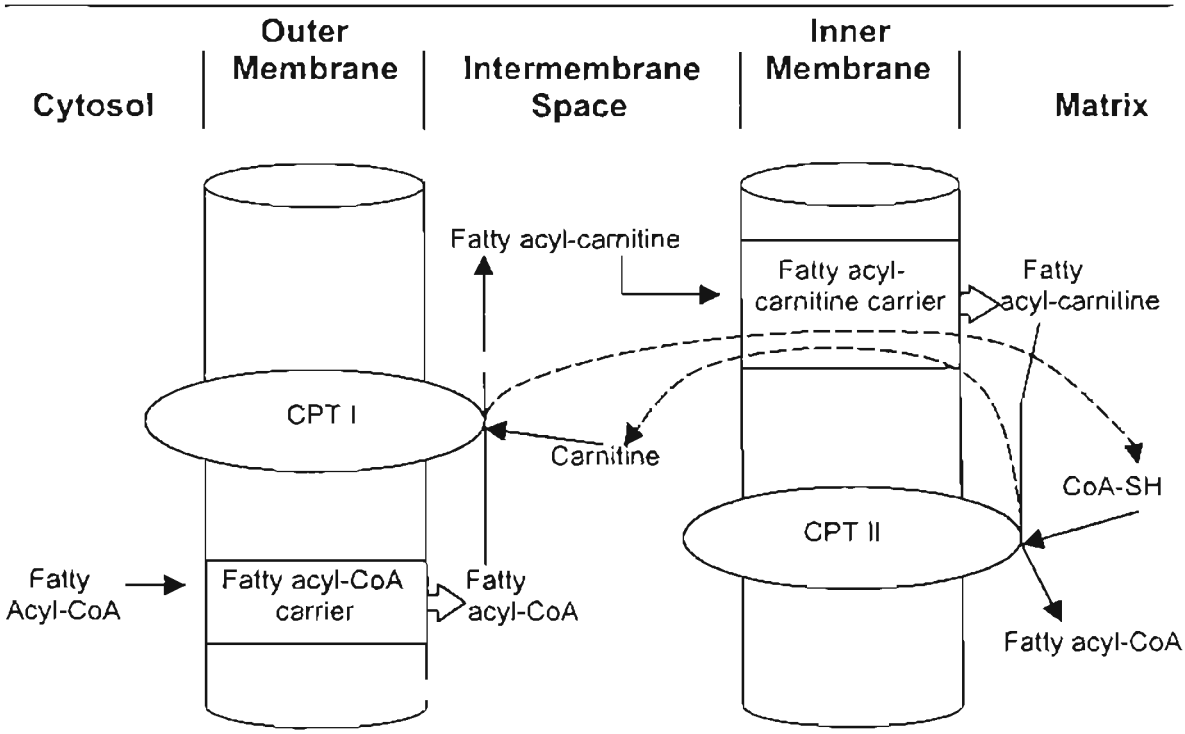
The three groups of enzymes include carnitine palmitoyltransferases, carnitine octanoyltransferases, and carnitine acetyltransferases, all of which are freely reversible enzymes (Rebouche and Seim, 1998).

The most investigated group of enzymes is carnitine acetyltransferases, which are widely distributed in nature, occurring in most if not all mammalian tissues, including brain (Choi et al., 1977) and sperm (Marquis and Fritz, 1965). Sperm are incapable of oxidizing long-chain fatty acids, and therefore have short-chain carnitine acetyltransferase activity. Carnitine octanoyltransferases and carnitine palmitoyltransferases are the enzymes that catalyze the reversible formation of medium- and long-chain acylcarnitines from medium- and long-chain acyl-CoAs and L-carnitine in mitochondria (Bieber, 1988).

Prior to the transportation of long-chain fatty acids into the mitochondrial matrix, synthetases located in the endoplasmic reticulum and in the outer membrane of the mitochondria first activate the long-chain fatty acids to their CoA thioesters. The fatty acyl-CoA may then be esterified via acyl-CoA transferases to form various triglycerides, cholesterol esters, and phospholipids that are exported from the liver as lipoproteins or stored in adipose tissue. Fatty acyl-CoA that does not undergo esterification is transported into the matrix of the mitochondria. Three carnitine dependent membrane proteins coordinate the activity of fatty acyl-CoA transport. The first reaction is catalyzed by carnitine palmitoyltransferase I (CPT I), located in the outer mitochondrial membrane, and involves the transfer of the fatty acyl moiety to join with the carnitine molecule located in the intermembrane space. Fatty acyl-carnitine is derived from this reaction. Translocase, located in the intermembrane space, catalyzes the exchange-

diffusion of fatty acyl-carnitine for free carnitine from the matrix, across the inner mitochondrial membrane. The enzyme carnitine palmitoyltransferase II (CPT II), located on the matrix side of the inner membrane, completes the transfer process by exchanging fatty acyl-carnitine for free carnitine and producing fatty acyl-CoA within the matrix. This shuttling process allows for the recycling of both carnitine and free CoA-SH between the intermembrane space and the cytosol, to begin the process anew. The carnitine acyltransferase system is detailed in Figure 1.3.

Figure 1.3. The carnitine acyltransferase system[a].



[a]Adapted from Mathews (2000).

The idea is conceivable for tissue- and organ-specific regulation of the carnitine acyltransferase system through alterations in intracellular malonyl-CoA concentrations. In the fed state, upregulation of fatty acid synthesis increases the concentration of

malonyl-CoA, the first intermediate of fatty acid synthesis. Malonyl-CoA has a profound inhibition on CPT I activity, resulting in esterification of endogenous and exogenous fatty acids rather than transportation into the mitochondrial matrix for oxidation (Sugden and Holness, 1994). Additionally, these authors suggested that the rates of oxidation of the two major oxidative energy substrates, glucose and fatty acids, i.e., the pyruvate dehydrogenase (PDH)[2] complex and the carnitine acyltransferase system (CAT), have interactive regulation. The activation of the PDH[2] system generates regulatory metabolites that suppress the activity of the CAT system, and vice versa.

In addition to facilitating the transport of long-chain fatty acids, numerous studies have established that carnitine has other roles in intermediary metabolism. Bieber et al. (1982) reported that carnitine has a more general facilitative effect on mitochondrial metabolism via its buffering of the acyl CoA/CoA-SH ratio in the matrix of the mitochondria. Utilization of all fuels by mitochondria is dependent on the availability of reduced CoA (CoA-SH). The oxidation of pyruvate, α-ketoglutarate, fatty acids, and branched-chain α-ketoacids utilize a common mitochondrial CoA-SH pool, thus, a continuous replenishment of this pool is necessary. It has been proposed that carnitine is primarily responsible for the renewal of the CoA-SH pool by removing acyl- and acetyl-CoA from the mitochondria as acetylcarnitine (Bieber et al., 1982). However, the rat is unique in that the buffering capacity of carnitine varies between the liver and heart. The buffering capacity in the liver is limited due to the low amount of carnitine acetyltransferase present in mitochondria (Lysiak et al., 1988).

Another beneficial function of carnitine and its associated acyltransferases is the elimination of toxic acyl residues (xenobiotics) arising from blockage of normal

metabolic pathways (Bieber, 1988). The elimination of these selective acyl residues is important in individuals who are deficient or seriously compromised in the enzymes needed for metabolism of the activated acyls. This is also another means to modulate the CoA-SH/acyl-CoA ratio

Carnitine has a unique function in the flight muscles of certain insects. Particular insect flight muscles are fatty acid oxidase-deficient; therefore, in the flight muscles of blowflies and bees the primary function of carnitine is not related to fatty acid oxidation. Instead, carnitine has a direct affect on carbohydrate utilization via pyruvate metabolism (Childress et al., 1966). During the initial phase of flight, pyruvate is generated at a rate faster than is utilized via the Krebs cycle. Paralleling the increase in pyruvate is a 4-fold increase in acetyl carnitine concentration. Childress et al. (1966) reported that acetyl carnitine is formed from pyruvate in mitochondria by working muscles of blowflies. By serving as an acceptor for acetyl groups from acetyl-CoA, there is a decrease in the acetyl-CoA to CoA ratio. This permits the continuous formation of acetyl-CoA from pyruvate, part of which can then condense with oxaloacetate as the Krebs cycle becomes available. Additionally, the conversion of pyruvate to acetyl carnitine provides an auxiliary store of active acetate, which can be readily transacetylated back to acetyl-CoA for subsequent oxidation.

Hahn and Skala (1975) suggested that carnitine is involved in non-shivering thermogenesis, a physiological function for which brown adipose tissue is held largely responsible. The concentration of carnitine and the activity of carnitine acetyltransferase in brown adipose tissue increase soon after birth, and in the suckling rat are higher than in any other organ. Carnitine increases fatty acid oxidation, which is closely related to heat

production in brown adipose tissue, thereby helping to maintain thermoneutrality in the suckling rat during instances of cold exposure. As well, Borum (1981) proposed that during adequate nutrition of the human neonate, brown adipose tissue is mobilized as a source of thermoregulatory heat production in response to cold environments. Whereas, during undernutrition, both in the absence and in the presence of cold-induced thermogenesis, white adipose tissue serves as a general reserve. Mobilization of either brown or white adipose tissue for thermogenesis requires adequate amounts of carnitine.

Genetic Defects of the Carnitine Acyltransferase System:

Deficiencies of specific enzymes in the carnitine acyltransferase system can have adverse affects on the oxidation of fatty acids. Although it is rare, a small number of cases have reported a hepatic deficiency of the enzyme, carnitine palmitoyltransferase I (CPT I) (Kerner and Hoppel, 1998). These authors reported that in the liver isoform, CPT I activity decreased by approximately 85-90%. Clinical symptoms of a CPT I deficiency include hypoketotic hypoglycemia, typically precipitated by fasting in infancy, but usually without myopathy or cardiomyopathy.

Carnitine palmitoyltransferase I deficiency involving the skeletal muscle isoform of the enzyme has been reported in only a few patients. In all cases there was an occurrence of rhabdomyolysis, which is the disintegration of striated muscle fibers and subsequent excretion of myoglobin in the urine (Kerner and Hoppel, 1998).

A deficiency in the acylcarnitine-carnitine translocase enzyme results in defective intramitochondrial transport of acylcarnitines formed by CPT I. Again, this deficiency is rare, but it is one of the most severe disorders of fatty acid oxidation. The disease

exhibits autosomal recessive inheritance with very early onset and lethal outcome in the perinatal and infantile period of life. Symptoms include hyperammonemia, hypoketotic hypoglycemia, and elevation of plasma long-chain acylcarnitines with very low free carnitine levels (Kerner and Hoppel, 1998).

The occurrence of a CPT II deficiency has been reported more frequently and is the most common disorder of lipid metabolism affecting skeletal muscle. The typical adult muscle form is by far the most frequent. However, the disease manifests itself in several clinical phenotypes, including affecting the hepatic enzyme isoform. The classical symptom in young adulthood is recurrent episodes of myoglobinuria induced by exercise (Kerner and Hoppel, 1998).

Role of L-Carnitine in Human Nutrition

Carnitine is a naturally occurring compound that is synthesized in the liver and kidney of humans from the essential amino acids lysine and methionine. Adult humans are capable of synthesizing adequate amounts of endogenous carnitine for normal facilitation of long chain fatty acids into the mitochondrial matrix to be used for energy production. However, carnitine is a critically important nutrient for the human neonate. Hepatic glycogen stores are rapidly depleted within the first 24 hours after birth. Consequently, lipids become an essential source of energy for newborn infants during the first few months of life. Utilization of these lipid sources requires adequate levels of carnitine. Yet, because carnitine stores are minimal and biosynthetic capabilities are reduced in the neonate compared with the adult, nutritionists have speculated that carnitine may be an essential nutrient for the human neonate (Borum, 1981).

Exogenous sources of carnitine are supplied to infants from breast milk, milk-based formulas, or carnitine-supplemented soy-based formulas. Increases are observed in the carnitine content of human breast milk during the first week postpartum from 39 to 63 nmol per milliliter, followed by decreases in the carnitine content of the milk to 45 nmol per milliliter after one month of lactation (Borum, 1981).

In 1972 the first report of a carnitine deficient patient appeared in the literature. This finding was of special interest because it provided a metabolic explanation for a muscle disease (Engel and Angelini, 1973). Human carnitine deficiency appears to be a family of syndromes ranging from a defective carnitine biosynthetic pathway to an inadequate transport of carnitine into muscle cells. Classical symptoms of a carnitine deficiency are progressive muscle weakness with lipid accumulation in Type I muscle fibers. Also, it generally reveals a biphasic progression with many patients expressing bouts of hypoglycemia or symptoms typical with hypoglycemia (Borum, 1981).

The greatest concern for a carnitine deficiency is in preterm infants. Nakano et al. (1989) reported that a positive correlation exists between concentrations of carnitine in skeletal muscle and gestational age at birth. In very premature infants the estimated level of carnitine in skeletal muscle, in relation to whole-body weight, is approximately 10 times less than that of adults (Schmidt-Sommerfeld and Penn, 1990). In an experiment to determine the effects of supplemental carnitine to preterm infants, Helms et al. (1990) observed increased nitrogen balance and weight gain for intravenously fed preterm infants (gestational age 32 ± 5 wk) supplemented with carnitine. The study was initiated 2 to 3 wk after birth when infants were administered 50 $\mu mol \cdot kg^{-1} \cdot day^{-1}$ of carnitine, after 7 days dosage levels of carnitine increased to 100 $\mu mol \cdot kg^{-1} \cdot day^{-1}$.

Contradictory to the results of Helms et al. (1990), Sulkers et al. (1990) reported that carnitine supplementation at the dosage level of 298 μmol · kg^{-1} day^{-1} to premature infants was not beneficial. Differences in opinions may be caused by two major differences in experimental designs. First, dosage levels varied between studies. Sulkers and coworkers experiment was more representative of the normal clinical situation because it is advised that fat emulsions should be infused continuously (Kao et al., 1984). Secondly, Sulkers and coworkers (1990) studied infants on d 4 to 7 after birth, in comparison Helms et al. (1990) examined infants 2 to 3 wk after birth.

As the gestational age of infants' increases, the concern for inadequate carnitine biosynthesis diminishes. However, concerns over insufficient carnitine stores still exist for infants that receive a soy-protein-based formula instead of breast milk. In a study conducted by Olson et al. (1989), the effects of an exogenous source of carnitine fed with formulas based on isolated soy protein on weight gain, serum concentrations of carnitine, and excretion of medium-chain dicarboxylic acids of human infants were determined. Normal male infants between the ages of 6 and 9 d were assigned to a diet containing either 1.2 μmol/L or 86 μmol/L of L-carnitine. Increasing levels of dietary L-carnitine did not affect growth or energy intake. However, infants fed 86 μmol/L of L-carnitine did have lower free, esterified, and total carnitine concentrations in serum samples when compared with the control. Increases in serum free fatty acids suggest an inhibition of β-oxidation due to low carnitine concentrations. Additionally, an increase in dicarboxylic acids was observed which indicates fatty acids are metabolized within the cell by the carnitine-independent pathway of microsomal-cytosolic ω-oxidation (Olson et al., 1989).

Plasma carnitine concentrations in normal omnivorous children (age 1 to 17 years) generally are in the same range as adults (total carnitine, 28 to 84 μmol/l; free carnitine 22 to 66 μmol/l) (Schmidt-Sommerfeld et al., 1988). However, concerns about adequate carnitine stores in both strict vegetarians and lactoovovegetarians, who habitually consume diets that are low in carnitine, exist. In fruits and vegetables the carnitine concentration is less than 1% that of meats while the carnitine concentration of cereal products is less than 5% that of meats. Therefore, Lombard et al. (1989) compared the plasma carnitine concentrations and urinary carnitine excretion levels of vegetarians and individuals consuming mixed diets of meat, dairy products, and cereal products. Although, the plasma carnitine concentrations were somewhat lower and the urinary carnitine excretion levels were markedly lower, carnitine levels of both groups were still within the normal range. The results of Lombard et al. (1989) suggest that carnitine biosynthesis in conjunction with renal conservation is capable of maintaining adequate carnitine stores, even when dietary carnitine intake is minimal. Similar results were published by Bowyer et al. (1989). The acute administration of carnitine to patients on long-term home parenteral nutrition with low carnitine concentrations had no affect on rates of palmitate, ketone body, glucose, or leucine metabolism, also suggesting the rate of carnitine biosynthesis is sufficient to supply adequate levels of carnitine for normal metabolic functions.

Minimal literature is available concerning the carnitine status of elderly people. No clinical conditions have been identified in aging humans that are attributable to nutritional carnitine deficiencies. Borum et al. (1987) reported that the plasma carnitine concentrations for women 40 years of age or older are higher than carnitine

concentrations for younger women. No differences were observed in circulating carnitine concentrations between older and younger males.

Some studies suggest that carnitine may have beneficial effects in the treatment of elderly humans with Alzheimer's disease. Although, the condition of patients that were treated with acetyl-L-carnitine did worsen with age, a slower rate of deterioration was apparent when compared with a placebo group (Spagnoli et al., 1991). The mechanism or mechanisms for the observed effects on patients with Alzheimer's disease are unknown, but may be associated with a decrease in cholinergic neural deterioration.

The primary role of carnitine in optimal mitochondrial fatty acid oxidation is well established in human research. Additionally, research validates that carnitine has other significant roles in intermediary metabolism, including transportation of activated medium- and short-chain organic acids from peroxisomes to mitochondria, buffering of the acetyl-CoA/CoA-SH ratio, and detoxification of poorly metabolized acyl groups. However, additional clinical trials integrating physiologic, biochemical, and pharmacological assessments are needed to definitively clarify any effects of carnitine on performance of individuals.

Role of L-Carnitine in Swine Nutrition

Dietary Sources of Carnitine:

Most animal products, including sow's colostrum and milk, are good sources of carnitine, whereas plant products are low in or devoid of carnitine. Table 1.1 lists the carnitine concentrations of animal and plant sources used in swine diets.

Table 1.1. Carnitine concentrations found in animal and plant products[a]

Product	L-Carnitine, ppm
Animal sources	
Blood meal	155
Feather meal	125
Fish bone meal	85
Fish meal	85-145
Meat bone meal (40%)	150
Skim milk powder	120-150
Whey powder	300-500
Whey powder (lactose extracted)	800-1000
Cow's milk	6-50
Goat's milk	15-20
Sheep's milk	130-320
Sow's milk	25-60
Plant Products	
Barley	10-38
Corn	5-10
Cottonseed	20-25
Milo	15
Rape seed	10
Soybean meal	0-10
Sunflower seed meal	2
Wheat	3-12

[a]Adapted from Owen (1996).

L-Carnitine promotes the mitochondrial β-oxidation of long-chain fatty acids by facilitating their transfer across the inner mitochondrial membrane. Ironically, research suggests cereal grains and their by-products have minimal concentrations of carnitine (Baumgartner and Blum, 1993). Because these feed ingredients usually constitute a major portion of swine diets, the significance of dietary L-carnitine has received intensive interest as of late. Numerous studies with neonatal pigs (Blatzell et al., 1987; Coffey et al., 1991; Hoffman et al., 1993; Kempen and Odle, 1993, 1995) and weaned pigs (Weeden et al., 1990; Newton and Burtle, 1992; Owen et al., 1996) have reported the effects of L-carnitine when supplemented to the diet. As well, the effects of L-carnitine on growth performance and carcass characteristics in growing-finishing swine have also

been observed (Owen et al., 1994; Smith et al., 1994; Newton and Haydon, 1989). Although research results are scarce, the effects of L-carnitine in sow diets have also been investigated by Musser et al. (1997a,b).

Neonatal Nutrition:

Carnitine and fatty acid metabolism are of critical importance in the neonate because hepatic glycogen stores are depleted shortly after birth, and muscle glycogen levels are minimal (Coffey et al., 1991). As a result, utilization of lipids, which constitute a majority of the energy in sow's milk, provides a large percentage of the energy to meet the energy demand. Adequate levels of carnitine are found in sow's milk; however, levels of carnitine in neonatal tissue are minimal suggesting carnitine is a critically important nutrient for the neonatal piglet (Borum, 1981).

Because of ethical issues, research in the human neonate is limited. Therefore, scientists have begun to utilize the neonatal pig as a model for human neonatal carnitine metabolism. Comparisons between the neonatal pig and the human neonate are possible because several characteristics are similar between the two groups (Blatzell et al., 1987). Similar attributes between the neonatal pig and the human neonate include anatomy and physiology, as well as degree of maturity at birth. Furthermore, the capability to investigate carnitine metabolism specifically is possible because both the animal model and the human neonate are susceptible to hypothermia and hypoglycemia, disorders associated with a carnitine deficiency. As well, both groups have the ability to adapt from a sole energy source of carbohydrates at birth to the utilization of lipids as an important energy source postpartum. Additionally, the profile of tissue carnitine concentrations of human neonates and neonatal pigs are similar during gestation (Blatzell

et al., 1987). These authors reported that the plasma and red blood cell carnitine concentrations of neonatal piglets decrease around 90 d, corresponding with 30 wk decreases observed in the plasma and red blood cell carnitine concentrations of humans. Similarly, increases in carnitine concentrations of skeletal muscle from 60 (< 26 wk human gestation) to 112-d (38-wk human gestation) observed by Blatzell et al. (1987) are comparable with those in humans.

However, the study by Blatzell et al. (1987) did not answer the question, what effects does feeding diets low in carnitine have on carnitine status or on utilization of lipid sources? Therefore, Coffey et al. (1991) conducted the first study to examine the carnitine status of piglets fed milk protein-based formulas compared with piglets reared by the sow. All piglets were allowed to nurse the sow for 48 h to develop an immune system. Pigs were then allotted to one of three diets: 1) fed by the sow; 2) fed a casein-whey (high carnitine) formula; or 3) fed an egg white protein (low carnitine) formula. Results indicate piglets fed a low carnitine diet had reduced concentrations of carnitine in plasma and liver samples on d 7 of age and throughout the 21-d study when compared with carnitine concentrations of the other two groups. However, dietary treatments resulted in minimal effects on carnitine concentrations in the heart and longissimus muscle during the experiment.

Similar to milk protein-based formulas that are low in carnitine stores, the levels of carnitine are also minimal in soy-protein based diets. Therefore, Hoffman et al. (1993) conducted an experiment to determine whether the addition of L-carnitine to a soy protein-based diet containing soybean oil would improve performance and nitrogen and energy utilization of neonatal pigs and performance of young pigs. The four dietary

treatments were obtained from combining either 0 or 800 ppm L-carnitine with either 1.18 or 12.31% soybean oil. In Phases 1 and 2 soy-protein isolate served as the only source of protein, while in Phase 3 soybean meal was the only protein source. The addition of L-carnitine did not affect any performance criteria measured in all three phases. As well, supplemental L-carnitine did not affect energy or nitrogen utilization in Phase 1. Therefore, Hoffman et al. (1993) indicated that neonatal or young pigs do not require a dietary source of L-carnitine when a soy protein-based diet containing high levels of crude soybean oil is fed. This suggests that the biosynthesis of carnitine in neonatal and growing pigs is adequate for normal nutrient utilization and growth performance, even when the diets are supplemented with high concentrations of crude soybean oil to increase caloric density.

An alternative method to improving fat utilization in neonatal pigs is by supplementing fat sources that are constituted primarily of medium-chain fatty acids. Medium-chain fatty acids are capable of passive diffusion through the inner mitochondrial membrane and subsequent intramitochondrial activation without the assistance of carnitine as a cofactor. However, *in vitro* evidence suggests carnitine stimulates the oxidation of medium-chain fatty acids in muscle (Otto, 1984). In agreement with Otto (1984), Kempen and Odle (1993) reported that carnitine plays a role in the oxidation of medium-chain fatty acids in the neonatal pig, suggesting carnitine should be included in the diet even when medium-chain fatty acids constitute a large portion of the dietary fatty acid profile.

Weanling Pig Nutrition:

Efforts to maximize farm productivity within the commercial swine industry have led to the practice of weaning pigs at 21 days of age or younger. Yet, because of this early-weaning management strategy, producers are faced with the challenge of increased post-weaning lag. In an effort to diminish post-weaning lag, complex, nutrient dense diets are being developed and fed to early-weaned pigs (Tokach et al., 1994). Milk products (20 to 40%) and supplemental fat (5 to 10%) are included within these complex weanling pig diets to increase the caloric density. However, research by Mahan (1991), Dove (1993), and Tokach et al. (1995) suggest that the addition of soybean, corn, and coconut oils or tallow as a supplemental fat source does not improve average daily gain (ADG) of pigs less than 28 d of age. Still, an improvement in ADG and feed efficiency due to added dietary fat was observed from d 14 to 35 post-weaning (Mahan, 1991: Tokach et al., 1995). The period immediately post-weaning is when L-carnitine synthesis is lowest in weanling pigs (Kerner et al., 1984). Therefore, given the role of L-carnitine in lipid metabolism, these findings would suggest inadequate amounts of carnitine are available to utilize the supplemental fat provided in the diet for energy production via β-oxidation in the mitochondrial matrix.

In early experiments dealing with L-carnitine supplementation to weanling pigs, Newton and Haydon (1988) reported that when L-carnitine was included in the diet (.60%) initially post-weaning, pigs grew faster and consumed more feed than pigs not fed L-carnitine. Weeden et al. (1990) reported improvements in ADG from 22 to 36 d of age due to added L-carnitine, however, no improvements in ADG were noted from 3 to 5 wk

post-weaning. As well, Weeden et al. (1990) suggested weanling pigs supplemented with L-carnitine accrued less carcass fat.

Because of the economic factors of supplementing maximal levels of L-carnitine, recent studies have evaluated whether smaller inclusion levels of carnitine can elicit the same beneficial improvements in growth performance and carcass characteristics. As well, current research has evaluated the effects of varying supplemental fat levels, various supplemental fat sources, and varying dietary lysine levels on performance criteria and carcass composition of swine.

Li et al. (1999) reported that the addition of L-carnitine (50 ppm) to diets with and without added fat increased average daily gain and feed intake from 15 to 28 days post-weaning. However, the increase in ADG due to L-carnitine was greater in pigs fed diets with soybean oil than in pigs fed diets containing lard. Additionally, data from the serum chemical analysis indicated lower free carnitine levels at weaning than at 14, 28 or 39 days after weaning. In both the L-carnitine supplemented and unsupplemented pigs the free carnitine levels increased with age, indicating endogenous carnitine was being synthesized after weaning (Li et al., 1999).

In comparison, Cho et al. (1999b) reported that ADG and F:G of weanling pigs responded to supplemental L-carnitine better when coconut oil, which is mainly comprised of medium-chain fatty acids, was included in the diet than when soy oil, a long-chain fatty acid source, was added to the diet. Cho et al. (1999b) suggested these improvements in growth performance were attributed to improvements in apparent nutrient digestibility that were observed in pigs fed the combination of 1000 ppm added L-carnitine and coconut oil.

In another experiment conducted by Cho et al. (1999a), the effect of dietary L-carnitine with different lysine levels on performance of weanling pigs was evaluated. Results suggested that during the first week after weaning the best performance in growth occurred when diets included 1.60% lysine and 1,000 ppm of L-carnitine. After the first week, similar performance was observed between pigs fed diets containing 1.40% lysine with 1,000 ppm L-carnitine and pigs fed diets containing 1.60% lysine with 1,000 ppm L-carnitine. In this study, a dietary lysine level of 1.80% and added L-carnitine did not show any additional effect on the performance of pigs.

These results are in contrast to Newton and Burtle (1992) who found high levels of dietary lysine (1.50% total lysine) to be detrimental to growth performance when supplemental L-carnitine was fed to nursery pigs, 28 to 42 d of age.

Not all studies have reported positive results due to the supplementation of L-carnitine, Ewan (1987) reported that the inclusion of 700 ppm L-carnitine to diets fed to pigs weaned at 22 days of age did not improve growth performance. As well, Hoffman et al. (1993) reported that ADG and energy utilization in neonatal and young pigs were not affected by added dietary L-carnitine. However, the diets formulated by Hoffman et al. (1993) contained 1.45 to 1.85% lysine from d 0 to 63 after weaning, suggesting dietary lysine levels were in excess and agreeing with the findings of Newton and Burtle (1992).

Results published by Owen et al. (1996) agree with those of Kempen and Odle (1995) that the absorption and uptake of carnitine within plasma, liver, heart, and whole carcass increased with increasing levels of dietary L-carnitine. Also, weanling pigs fed 1,000 ppm L-carnitine during Phase 1 (d 0 to 14) had less carcass lipid and daily lipid accretion to d 35 post-weaning (Owen et al., 1996).

Grower/ Finisher Pig Nutrition:

Recent marketing schemes in the commercial swine industry involve animals being sold on a carcass merit system with premiums being offered for leaner, more muscular animals. In an effort to reap larger economic rewards, producers have developed animals with a greater genetic potential for lean growth. Also, producers have begun to utilize feed additives that have a role in lean growth modulation of swine. Speculation exists that supplemental L-carnitine can improve dietary fat utilization through increased β-oxidation. Increased β-oxidation can lead to a repartitioning of nutrients for increased protein accretion and decreased lipid accretion in growing and finishing swine.

Initial studies evaluating the effects of dietary L-carnitine on growth performance and carcass characteristics of growing and finishing swine were conducted by Newton and Haydon (1989). Pigs were fed diets containing 0, 5, or 10 ppm added L-carnitine. During the first 14 days, pigs fed 5 ppm L-carnitine consumed less feed than pigs fed the control diet; however, there were no differences in weight gain or feed efficiency. During the last 14 days of the trial pigs fed L-carnitine grew faster on the same amount of feed compared with pigs fed the control diet. Unfortunately, supplemental L-carnitine had no effect on backfat measurements. This study suggested carnitine might have beneficial effects during the latter stages of the finishing period.

Owen et al. (1993) reported that the dietary inclusion of 25 ppm L-carnitine during the growing-finishing phase did not affect growth performance of swine. However, supplemental L-carnitine during the growing-finishing phase did increase longissimus muscle area and decrease fat accretion rate. A second experiment evaluating

the effects of supplementing 0, 25, 50, 75, or 125 ppm L-carnitine to the diet of growing-finishing pigs reported similar results (Owen et al., 1994). Increasing levels of dietary L-carnitine did not affect growth performance; yet, L-carnitine did elicit a response in carcass characteristics. Dietary L-carnitine reduced backfat and 10^{th} rib backfat thickness while increasing longissimus muscle area with 50 ppm providing the greatest response.

This is in agreement with data presented by Smith et al. (1994). These authors observed increases in carcass leanness due to supplemental L-carnitine. Fifty ppm L-carnitine resulted in larger longissimus muscle area, lower 10^{th} rib backfat thickness, lower average backfat thickness, and greater percent muscle.

An interesting study was designed by Heo et al. (2000) to test the hypothesis that dietary L-carnitine can alter nutrient partitioning in young growing pigs, resulting in changes in body composition. These authors formulated basal diets that were limiting in metabolizable energy (ME) so that nitrogen retention and protein accretion responded to ME. Basal diets were formulated in a 2 x 2 factorial arrangement of treatments to contain either low or high protein concentrations and either 0 or 500 ppm added L-carnitine. As well, all diets were formulated to contain 7% fat on the idea that added L-carnitine would improve ME derived from fat. Supplemental L-carnitine increased ADG by 7.3% and crude protein accretion rate by 9% in both protein levels. L-Carnitine fed pigs had a 4.5 fold greater total-body carnitine accretion rate and almost a 100% greater total body carnitine concentration than unsupplemented pigs. Additionally, diets containing L-carnitine improved the efficiency of nitrogen retention and reduced urinary nitrogen excretion by 14% in pigs. Carcass fat also was reduced in growing pigs when L-carnitine was added to their diet.

Sow Nutrition:

The physiological role of carnitine is to facilitate the transport of long-chain fatty acids across the mitochondrial membrane into the matrix of the mitochondrial for the production of energy. As well, carnitine has been shown to affect several key enzymes involved in protein and lipid metabolism, suggesting an enhancement in the productivity of gestating and lactating sows is conceivable. Therefore, Musser et al. (1997a) supplemented 50 ppm L-carnitine to gestating and lactating sows and determined the effects on sow and litter performance. Gestating sows fed 50 ppm L-carnitine had greater weight gains and last rib fat depth during gestation. At farrowing, the sows supplemented with 50 ppm L-carnitine during gestation had increased pig and litter birth weights. Subsequently, pig and litter weight gains tended to increase due to feeding L-carnitine during gestation. However, no differences were observed in sow and litter performance as a result of feeding 50 ppm L-carnitine during lactation.

In a subsequent study by Musser et al. (1997b), 50 ppm L-carnitine was supplemented to first parity gilts during lactation and the effects on sow and litter performance were determined. Supplemental L-carnitine elicited no changes in litter weaning weight or weight gain, or changes in sow weight and last rib fat depth during lactation.

Conclusions:

Literature would suggest that beneficial responses are obtainable due to the supplementation of L-carnitine in swine diets. Given the role of L-carnitine in lipid

metabolism, the greatest response attributed to L-carnitine is in dietary fat utilization. Enhancements in the utilization of dietary fat present many positive responses for swine including an increased energy supply for growth and a repartitioning of nutrients from lipid deposition towards protein accretion, resulting in improved body composition.

Chapter II

Experiment 1

Effects of L-Carnitine on Growth Performance of Weanling Pigs.

Abstract: An experiment was conducted to evaluate the effects of supplementing graded levels of L-carnitine to the diet of weanling pigs on growth performance. One-hundred twenty-eight weanling pigs (5.5 kg initial BW; 18 d) were randomly allotted by BW, sex, and litter to four dietary treatments. There were 6 pens/trt of 4 to 6 pigs/pen. Dietary treatments were the control diet with 25, 50, and 100 ppm L-carnitine. Pigs were fed in three dietary phases: (P1: d 0-10; P2: d 11-24; and P3: d 25-38 with 1.6, 1.4, and 1.2% Lys, respectively). Phase 1 and 2 diets were complex corn-soybean meal-dried whey based containing lactose, animal plasma, blood meal, and fish meal, while diets for P3 were corn-soybean meal based. Pigs were weighed and feed consumption was measured weekly for the determination of ADG, ADFI, and G:F. For the 38-d study, ADG, ADFI, and G:F were, respectively, 337, 347, 370, and 363 g; 503, 502, 516, and 523 g; and 0.669, 0.692, 0.717, and 0.693. Dietary L-carnitine increased ADG (linear, $P < 0.09$) and G:F (quadratic, $P < 0.02$) for d 0-38. However, this improvement in ADG and G:F associated with L-carnitine was greatest during Phase 2 (linear, $P < 0.03$). These results suggest that the addition of L-carnitine to the diet improved growth performance in weanling pigs. The most effective level of L-carnitine in improving growth performance of weanling pigs was 50 ppm.

Introduction

Carnitine is a naturally occurring compound that is synthesized from the essential amino acids lysine and methionine. It also became known as vitamin B_T, to indicate its place in the B-group of vitamins (Fraenkel, 1948). The presence of carnitine in muscle and other tissues is necessary to facilitate the transfer of long-chain fatty acids into the enzymatically active intra-mitochondrial matrix, resulting in the production of adenosine triphosphate (energy) via β-oxidation and oxidative phosphorylation (Fritz and Yue, 1963; Bray and Briggs, 1980).

Kerner et al. (1984) reported that the biosynthesis of carnitine is limited in pigs directly after weaning. This finding suggests that minimal carnitine stores in the weanling pig may hinder the response to supplemental fat sources. Therefore, studies to determine the effects of supplemental L-carnitine on weanling pig performance have been conducted. Newton and Haydon (1988) reported that when 0.60% L-carnitine was added to the diet initially post-weaning, pigs grew faster and consumed more feed than pigs not fed L-carnitine. Improvements in ADG and increases in feed intake from 22 to 36 days of age, due to added L-carnitine (1,000 ppm), were also reported by Weeden et al. (1990). Heo et al. (2000), Li et al. (1999), and Owen et al. (1996) also reported improvements in performance criteria of weanling pigs due to supplemental L-carnitine. In contrast, Hoffman et al. (1993) and Owen et al. (2001) did not find any improvements in growth performance of weanling pigs due to added L-carnitine.

Because of increased dietary costs due to supplementing maximal levels of L-carnitine, the addition of L-carnitine to the diet of weanling pigs may not be economically feasible for the producer. Recent studies by Real et al. (2001) have

reported that the addition of lower supplemental levels of L-carnitine enhanced growth performance of weanling pigs. Therefore, the objective of our study was to determine the effects of lower dietary concentrations of L-carnitine (0 to 100 ppm) on growth performance of weanling pigs.

Materials and Methods

One-hundred twenty-eight Yorkshire, Hampshire, and crossbred (Yorkshire x Hampshire) pigs were weaned at 18 ± 4 d and placed in temperature-controlled nursery rooms in a 38-d experiment. Initially averaging 5.5 kg, pigs were allotted randomly on the basis of weight, sex, and litter to four dietary treatments in a randomized complete block design. There were 6 pen replicates per treatment and pigs were grouped with 4 to 6 pigs per pen. Dietary treatments were formulated by supplementing the control diet (Table 2.1) with 25, 50, and 100 ppm L-carnitine. The four dietary treatments were: 1) control; 2) control + 25 ppm L-carnitine; 3) control + 50 ppm L-carnitine; and 4) control + 100 ppm L-carnitine. Pigs were fed in three dietary phases: [Phase 1 (P1): d 0-10; Phase 2 (P2): d 11-24; and Phase 3 (P3): d 25-38]. Complexity of the diet changed with phases to satisfy the nutrient requirements (NRC, 1998) of the weanling pig. Phase 1 (1.6% Lys) and Phase 2 (1.4% Lys) diets were complex corn-soybean meal-dried whey based diets containing lactose, spray-dried animal plasma, spray-dried blood meal, and fish meal, while Phase 3 (1.2% Lys) diets were typical corn-soybean meal based. All diets were fed in pelleted form and contained 5.0% soybean oil as a dietary fat source.

Pigs were housed in temperature-controlled nursery rooms and grouped in elevated pens with wire flooring. Each pen provided 1.72 square meters of space and contained a five-hole, stainless steel feeder and one nipple waterer that allowed *ad*

libitum access to feed and water throughout the experiment. Room temperature was maintained initially at 31°C, and decreased by 1.1°C weekly until the room temperature reached 25.5°C. Pig weights and feed consumption were recorded weekly for the determination of average daily gain (ADG), average daily feed intake (ADFI), and gain:feed (G:F).

Table 2.1. Composition of control diets (as-is basis).

	Diets[a]		
Ingredient, %	Phase 1	Phase 2	Phase 3
Corn	30.19	50.19	56.84
SBM, (48%)	20.75	25.00	33.75
Whey, dried	20.00	10.00	--
Lactose	10.00	--	--
Plasma, spray-dried	5.00	2.50	--
Blood meal, spray-dried	2.50	2.50	--
Fish meal, menhaden	2.50	--	--
Soybean oil	5.00	5.00	5.00
Dicalcium phosphate	1.53	2.11	2.37
Limestone	0.42	0.61	0.68
DL-Methionine	0.20	0.13	--
Ethoxyquin	0.03	0.03	0.03
Salt	0.25	0.25	0.35
Trace min/Vit premix[b]	0.30	0.30	0.30
Zinc Oxide	0.28	0.28	--
Copper sulfate	--	--	0.08
Antibiotic[c]	1.00	1.00	0.50
Cornstarch[d]	0.05	0.10	0.10

[a]Diets formulated to contain 1.6, 1.4, and 1.2% total lysine for P1, P2, and P3, respectively

[b]Provided the following per kg feed: Zn, 120 mg; Fe, 120 mg; Mn, 24 mg; Cu, 12 mg; I, .36 mg; Se, .36 mg; vitamin A, 6,615 IU; vitamin D_3, 661 IU; vitamin E, 40 IU; vitamin K (menadione activity), 4.4 mg; riboflavin, 6.6mg; d-pantothenic acid, 30 mg; niacin, 40 mg; vitamin B_{12}, 33 ug; d-biotin, 265 ug; choline, 144 mg; and folic acid, 2 mg.

[c]P1 and P2 contained Neo-terramycin® (100 g/ton oxytetracycline & 140 g/ton neomycin base) and P3 contained Lincomix® (200 g/ton lincomycin)

[d]L-carnitine (Carnikng 10, Lonza Inc., Fair Lawn, NJ) substituted at 0, 25, 50, and 100 ppm for cornstarch to obtain the four dietary treatments

Chemical analysis:

Diets were analyzed for DM according to AOAC (1998) procedures. Gross energy determinations were made by bomb calorimetry (Parr 1261 Isoperibol Calorimeter, Moline, IL), and nitrogen determinations were performed by Kjeldahl methodology (FOSS Tecator, 2400 Kjeltec Analyzer unit, 2020 Digestor, Hoganas, Sweden). As well, diets were analyzed for L-carnitine concentrations using methods described by Parvin and Pande (1977). Chemical composition of the control diets is shown in Table 2.2. The L-carnitine concentration for the four dietary treatments for each phase is detailed in Table 2.3.

Statistical analysis:

Data were analyzed as a randomized complete block design. Analysis of variance was performed using GLM procedures of SAS (SAS Inst. Inc., Cary, NC) as described by Steel et al. (1997). The model included the effects of block (rep), treatment, and block x treatment (error). The effects of increasing dietary L-carnitine concentrations were partitioned into linear and curvilinear components using orthogonal polynomial contrasts. Due to unequally spaced dietary levels of L-carnitine, coefficients were derived using the integrative matrix language (PROC IML) procedures of SAS (Version 7.11). Pen served as the experimental unit.

Table 2.2. Chemical composition of control diets (as-is basis).

Item	Phase 1	Phase 2	Phase 3
Calculated analysis			
ME, kcal/kg	3,364	3,373	3,402
Crude protein, %	22.58	21.70	21.19
Total lysine, %	1.60	1.40	1.20
Digestible lysine, %	1.36	1.18	1.00
Digestible threonine, %	0.80	0.70	0.61
Digestible Met + Cys, %	0.80	0.70	0.56
Digestible tryptophan, %	0.25	0.23	0.21
Calcium, %	0.90	0.90	0.90
Phosphorus, %	0.80	0.80	0.80
Available phosphorus, %	0.62	0.56	0.51
Analyzed values			
GE, kcal/kg	4,125	4,170	4,186
Crude protein, %	20.47	20.01	19.68

Table 2.3. L-Carnitine concentration of diets[a].

Item:	Calculated Concentration	Analyzed Concentration	Supplemented Level[b]
Phase 1		L-carnitine, ppm	
Diet 1	0	37	0
Diet 2	25	62	25
Diet 3	50	86	49
Diet 4	100	117	80
Phase 2			
Diet 1	0	19	0
Diet 2	25	41	22
Diet 3	50	71	52
Diet 4	100	106	87
Phase 3			
Diet 1	0	1	0
Diet 2	25	28	27
Diet 3	50	51	50
Diet 4	100	101	100

[a]Analysis reported on an as-is basis

[b]Supplemented level obtained by subtracting analyzed concentration from analyzed concentration of unsupplemented diet (diet 1)

Results

Supplemented levels of L-carnitine (Table 2.3) were in agreement with calculated levels, signifying proper diet mixing. Because of the inclusion of 5% soybean oil, all diets had a caloric density (Table 2.2) between 4,125 and 4,206 kcal/kg. Analyzed values of crude protein were approximately 1.75% lower than the calculated values for each phase. However, diets were formulated on a total lysine basis and were formulated to exceed NRC (1998) recommendations, thereby, limiting any affects on growth performance due to a lysine deficiency.

The effects of graded levels of L-carnitine on pig performance are shown in Table 2.4. Increasing levels of supplemental L-carnitine improved ADG (linear, $P < 0.09$) and G:F (quadratic, $P < 0.02$) for the 38-d study. Pigs fed 50 ppm L-carnitine exhibited the greatest response to dietary L-carnitine, having the highest ADG (370 g) and best G:F (0.72) for d 0-38. However, this improvement in ADG and G:F associated with L-carnitine was greatest during Phase 2 (linear, $P < 0.03$). Again, the best response to L-carnitine was observed in pigs fed 50 ppm, as ADG and G:F tended to plateau at this concentration, while the maximum level of 100 ppm L-carnitine did not elicit further improvements in performance criteria. Responses to increasing levels of dietary L-carnitine were also observed during Phases 1 and 3. A linear increase ($P < 0.06$) in G:F was noted as the level of L-carnitine increased in the diet during Phase 1. As well, there was a numerical increase ($P = 0.17$) in ADG from d 0-10. During Phase 3, supplemental L-carnitine improved ($P < 0.08$) G:F; however, it did not affect ADG or ADFI ($P > 0.20$). The supplementation of L-carnitine had little effect on ADFI ($P > 0.20$) during any phase or for the entire experiment. These results suggest that the addition of L-carnitine

improved growth performance in weanling pigs, with the most pronounced response to supplemental L-carnitine observed in pigs fed 50 ppm.

Table 2.4. Growth performance of weanling pigs[a]

	L-carnitine, ppm					P >:[b]		
Item:	0	25	50	100	SE	Linear	Quad.	Cubic
Phase 1, d 0-10								
ADG, g/d	137	136	165	159	13.5	0.18	--	--
ADFI, g/d	173	166	185	178	10.2	--	--	--
G:F	0.79	0.80	0.89	0.89	0.04	0.06	--	--
Phase 2, d 11-24								
ADG, g/d	342	359	381	377	11.0	0.03	0.16	--
ADFI, g/d	467	468	489	477	11.2	--	--	--
G:F	0.73	0.76	0.78	0.79	0.01	0.01	0.16	--
Phase 3, d 25-38								
ADG, g/d	479	491	511	494	15.8	--	--	--
ADFI, g/d	781	782	791	815	25.1	--	--	--
G:F	0.61	0.63	0.65	0.61	0.02	--	0.08	--
Overall, d 0-38								
ADG, g/d	337	347	370	363	10.8	0.09	--	--
ADFI, g/d	503	502	516	523	14.1	--	--	--
G:F	0.67	0.69	0.72	0.69	0.01	0.15	0.02	--

[a]Least squares means for six pens/trt of four to six pigs/pen
[b]Dashes indicate P > 0.20

Discussion

Given the role of L-carnitine in fatty acid metabolism, L-carnitine may be supplemented to the diet of weanling pigs in an effort to increase a naturally low carnitine status. Increases in carnitine stores may catalyze the transport of long-chain fatty acids into the mitochondrial matrix for the production of adenosine triphosphate (energy) in an effort to improve growth performance. Although data from this study revealed no differences in feed consumption during Phase 1 (d 0-10), a slight increase in daily gain resulted in an improvement in feed efficiency due to added L-carnitine. Weeden et al. (1991) also noted an improvement in ADG with the addition of 1,000 ppm L-carnitine

during the first two weeks after weaning; however, supplemental L-carnitine did not alter feed intake or feed efficiency in their study. An immediate post-weaning response to added L-carnitine (500 ppm) was also reported by Cho et al. (1999b), as pigs fed L-carnitine grew faster and consumed more feed during the first two weeks. It is worth noting that both Weeden et al. (1991) and Cho et al. (1999b) had substantially higher addition levels of L-carnitine than those utilized in our experiment.

The responses observed during Phase 2 (d 11-24), due to dietary L-carnitine, are in agreement with results from several studies. This time frame appears to be when weanling pigs have the capability to begin utilizing the added dietary L-carnitine to increase fatty acid oxidation, resulting in increased energy production, and subsequently, improved performance criteria. Li et al. (1999) reported that the addition of 50 ppm L-carnitine increased ADG and feed consumption from 15 to 28 days post-weaning. An improvement due to the addition of 50 ppm L-carnitine was also noted by Real et al. (2001). As well, when maximum levels of L-carnitine (500 to 1,000 ppm) were added to the diet, improvements in feed efficiency during Phase 2 (wk 3 to 5) were observed by Weeden et al. (1990). Owen et al. (1996) observed a similar effect of carnitine on feed efficiency in the period of 3 to 5 weeks after weaning. In contrast to the positive responses to supplemental L-carnitine, Hoffman et al. (1993) reported that 800 ppm L-carnitine from d 0-21 after weaning did not affect ADG, G:F, and gain per megacalorie of ME. These authors also noted that the addition of 750 ppm L-carnitine did not affect any performance criteria from d 21 to 63 post-weaning.

Minimal responses in growth performance due to the addition of L-carnitine were observed during Phase 3 (d 25-38). There was a slight increase in ADG; with pigs fed 50

ppm L-carnitine having the numerically highest ADG. This minor improvement in ADG resulted in an increased G:F ratio in pigs fed 25 to 50 ppm L-carnitine. A numerical improvement in ADG and feed efficiency from d 28 to 39 after weaning, in pigs fed 50 ppm L-carnitine, was also reported by Li et al. (1999). We would hypothesize that the marginal response to added L-carnitine during Phase 3 may be due to the increased biosynthesis of carnitine as the pig matures. Adequate levels of carnitine production may result from typical endogenous and exogenous carnitine sources and mask any response to supplemental L-carnitine. Although we did not measure serum carnitine status in our study, Li et al. (1999) noted that there were no differences in total serum carnitine and free serum carnitine concentrations between pigs fed 50 ppm L-carnitine and those not fed added dietary L-carnitine. As well, on day 39 post-weaning, little variation in total serum carnitine, free serum carnitine, and short-chain acyl serum carnitine concentrations was observed between either group.

Implications

Results from the present study suggest that the addition of L-carnitine to the diet at lower concentration levels (50 ppm) can enhance growth performance of weanling pigs. Even though a slight improvement in G:F was observed immediately post-weaning (d 0-10), an adjustment period of approximately 10 days post-weaning may be required before the greatest response to supplemental L-carnitine can be observed in weanling pigs. Although the exact mechanisms are unknown, we would speculate that the supplemental L-carnitine allows for the improved utilization of the added soybean oil (energy source) in the diet; thereby, increasing energy production, and subsequently, improving growth performance in the weanling pig. Further research is needed to

determine the mode of action resulting in improved growth performance due to L-carnitine and whether the response to L-carnitine is dependent upon dietary fat content.

Chapter III

Experiment 2

Effects of L-Carnitine in the Diet of Weanling Pigs on Apparent Nutrient Digestibility, Whole Body Composition, Tissue Accretion, and Blood Metabolites.

Abstract: An experiment was conducted to evaluate the effects of supplementing L-carnitine to the diet of weanling pigs on apparent nutrient digestibility, whole body composition, tissue accretion, and blood metabolites. Six sets of four littermate barrows (4.9 kg; 18 d) were randomly allotted to four dietary treatments containing 0, 25, 50 or 100 ppm added L-carnitine. Pigs were fed in three dietary phases (P1: d 0-10; P2: d 11-24; and P3: d 25-38 with 1.6, 1.4, and 1.2% Lys, respectively). Phase 1 and 2 diets were complex corn-soybean meal-dried whey based containing lactose and additional protein sources, while diets for Phase 3 were typical corn-soybean meal based. Pigs were housed individually in metabolism chambers and a 5-d total but separate collection of urine and feces was performed during each phase (P1: d 4-9; P2: d 17-22; and P3: d 29-34). There were no treatment by period interactions; therefore, data were pooled across periods. Increasing L-carnitine resulted in a slight improvement (quadratic, $P < 0.10$) in energy digestibility and nitrogen retention with the greatest response observed in pigs fed 25 to 50 ppm L-carnitine. As well, pigs were bled by jugular venipuncture at the start of the study and then at the end of each phase. Minimal effects were noted in blood metabolites as a result of increasing levels of L-carnitine; however, responses in blood urea nitrogen,

C-reactive protein, glucose, and non-esterified fatty acids, associated with increasing L-carnitine, were observed during Phase 2. At the conclusion of the experiment, each pig was killed and ground for determination of whole body composition. Additionally, a fifth littermate from each set of pigs was killed at the beginning of the experiment for the determination of initial body composition. Added L-carnitine increased (linear, $P < 0.01$) the percentage of protein and decreased (linear, $P < .01$) the percentage of fat in the weanling pig. A quadratic increase ($P < 0.05$) in total (g) and rate (g/d) of protein and energy accretion was observed with increasing L-carnitine. Also, the ratio of protein accretion to fat accretion (1.59, 2.07, 2.08, and 2.23) improved (linear, $P < 0.01$) with supplemental L-carnitine. These results suggest the addition of L-carnitine to the diet improves whole body composition, tissue accretion, and to a lesser degree, nutrient digestibility in weanling pigs; however, the greatest response to L-carnitine was noted in pigs fed 50 ppm.

Introduction

Carnitine is a naturally occurring, vitamin B-like compound that is present in muscle and other tissues. The primary metabolic role of carnitine is to facilitate the transfer of long-chain fatty acids across the mitochondrial membrane into the matrix of the mitochondria (Fritz and Yue, 1963; Bray and Briggs, 1980). Due to the fact that carnitine is a cosubstrate of carnitine palmitoyltransferase, a vital regulatory enzyme in the pathway of fatty acid oxidation (Heo et al., 2000), the status of carnitine could conceivably affect the utilization of fatty acid stores for the production of adenosine triphosphate (energy). Up-regulation of the transport of long-chain fatty acids results in increased β-oxidation and oxidative phosphorylation in the mitochondrial matrix, in turn,

leading to increased energy production. As a consequence of the increased energy yield, a repartitioning of nutrients is possible. Interest in the role of carnitine as a feed additive to improve whole body composition arose from the desire to partition nutrients away from lipid accretion and towards protein deposition in an effort to produce higher yielding animals.

Results from Experiment 1 and Real et al. (2001) reported improvements in ADG and G:F of weanling pigs in response to 50 ppm dietary L-carnitine. However, the question still exists as to the exact mechanisms that elicit the improvements in growth performance and whether the improvements are the result of a repartitioning of nutrients. Early experiments conducted by Weeden et al. (1991) reported that pigs fed 1,000 ppm L-carnitine from d 0 to 14 post-weaning had reduced carcass fat on d 35. Owen et al. (1996) also reported similar results. These authors reported that 1,000 ppm added L-carnitine from d 0 to 14 after weaning reduced carcass lipid accretion on d 35. In a subsequent study conducted by Owen et al. (2001), the addition of L-carnitine decreased daily lipid accretion in weanling pigs. As well, Cho et al. (1999b) suggested that the supplementation of L-carnitine improved crude fat and gross energy digestibility, resulting in improved ADG and G:F during the third week after weaning. In contrast, Hoffman et al. (1993) reported that added L-carnitine did not improve the performance of young pigs nor metabolizable energy in diets that contained soybean oil. Therefore, the objective of our study was to determine the effects of graded levels of L-carnitine on nutrient digestibility, whole body composition, tissue accretion, and blood metabolites in weanling pigs.

Materials and Methods

General procedures:

Six sets of four littermate Yorkshire and crossbred (Yorkshire x Hampshire) barrows (18 ± 2 d), initially averaging 4.9 kg, were individually housed in metabolism chambers and utilized in a 38-d experiment. Pigs were allotted randomly within litter on the basis of initial body weight to the four dietary treatments used in Exp. 1 (Table 2.1) in a randomized complete block design. There were six pigs per treatment. The four dietary treatments were: 1) control; 2) control + 25 ppm L-carnitine; 3) control + 50 ppm L-carnitine; and 4) control + 100 ppm L-carnitine. Pigs were fed in three dietary phases: [Phase 1 (P1): d 0-10; Phase 2 (P2): d 11-24; and Phase 3 (P3): d 25-38].

Pigs were housed in an environmentally controlled room. Room temperature was maintained initially at 31°C and decreased by 1.1°C weekly until the room temperature reached 25.5°C. Each chamber consisted of 12.5 mm thick plexi-glass on the sides and top of chambers with an outside dimension of .80 x 1.22 m and a total pig space allowance of .75 x 1.05 m. Floors of the chambers were expanded polyurethane tenderfoot mesh design. One stainless steel self-feeder and nipple waterer per chamber were used to allow *ad libitum* access to feed and water throughout the experiment.

Growth performance:

Performance criteria were measured for the 38-d feeding experiment. Pigs were weighed and feed intake was measured at the beginning and the conclusion of each dietary phase for the determination of average daily gain (ADG), average daily feed intake (ADFI), and gain:feed (G:F).

Apparent nutrient digestibility:

The chambers were constructed to allow for the total but separate collection of urine, feces, and refused feed. A 5-d collection period of urine and feces was performed during each phase (P1: d 5-9; P2: d 17-22; and P3: d 29-34). Fecal material was collected daily during the 5-d collection period by removing a screen (1 mm) that lay directly beneath the entire floor space of the chamber. Fecal samples were collected and then placed in a plastic bag and weighed prior to storage in a -20°C freezer. Situated beneath each fecal collection screen was a stainless steel pan that was graduated toward an 8-mm hole in the center. Beneath the graduated pan, a five-quart capacity plastic container was used to collect all urine. During the collection period, 10 ml of HCL acid was added daily to each urine collection container to prevent any loss of nitrogen due to the volatilization of ammonia. Urine samples were collected daily at the same time fecal samples were collected. Total urine volume was recorded daily and a 100-ml sub-sample was collected in a plastic cup and stored in a freezer (-20°C) prior to analysis. As well, pig weights and feed consumption were recorded at the start and end of each collection period to be used in the determination of apparent nutrient digestibility.

Feed, fecal, and urine samples were assayed for DM, gross energy concentrations, and nitrogen levels and used in the determination of apparent nutrient digestibility. Chemical analysis was performed on each sample in duplicates and averages were then computed for each sample. Before any analytical procedures were performed on urine samples, the daily 100-ml sub-samples for each pig were thawed and a composite sample was gathered. A portion of each daily sub-sample in proportion to that samples percentage of the total urine output was used to obtain the composite sample. Dry matter

percentage of fecal samples was determined by initially drying the total 5-d collection of feces in a forced-air oven for 4 d at 50°C. Partially-dried fecal samples were then ground in a Wiley Mill (Standard Model No.3; Arthur H. Thomas Co., Philadelphia, PA) equipped with a 1-mm screen. Diet samples were also ground in a Wiley Mill equipped with a 1-mm screen.

Dry matter was then determined for diet and fecal samples by drying an approximate 3 g sample for 24 hr at 100°C (AOAC, 1998). Nitrogen determination of feed, fecal, and composite urine samples was performed by Kjeldahl methodology (FOSS Tecator, 2020 Digestor, 2400 Kjeltec Analyzer Unit; Hoganas, Sweden). Gross energy determinations of feed and fecal samples were made by bomb calorimetry (Parr 1261 Isoperibol Calorimeter; Moline, IL).

For urinary energy analysis, one-half gram of Solka-Floc® (cellulose) was dried for 24 hr at 100°C to determine the moisture percentage of cellulose. Two milliliters of the composite urine sample was then added to the dried Solka-Floc® sample and dried for an additional 24 hr at 100°C to calculate the percentage of dry urine. The dried urine/Solka-Floc® sample was then pelleted and bombed to determine gross energy. As well, pure Solka-Floc® pellets were bombed and used in the determination of the gross energy of the urine. The percentage of dry Solka-Floc® in the combusted pellet along with the total energy of combustion in the pure Solka-Floc® pellet were used to calculate the amount of energy that the Solka-Floc® portion provided in the urine/Solka-Floc® pellet. The amount of energy that the Solka-Floc® portion provided in the urine/Solka-Floc® pellet was subtracted from the total energy concentration of the urine/Solka-Floc®

pellet. The remaining value was then divided by the percentage of dry urine to obtain the amount of energy in the composite urine sample.

Blood metabolites:

Blood samples were collected from each pig on d 0, 3, 10, 24, and 38. On d 38 blood samples were initially taken, then pigs were fasted for 2 hours and a subsequent blood sample was drawn. Pigs were bled using 22 x 1" gauge needles (Sherwood Medicals, St. Louis, MO) by jugular venipuncture and a serum sample was collected in a 10-ml anticoagulant free vacutainer (Becton Dickinson, Franklin Lakes, NJ). Following collection, serum samples were chilled in an ice bath for 1 hour and allowed to coagulate. Next, samples were placed in a centrifuge (J-6B Centrifuge; Beckman Instruments, Inc. Fullerton, CA) and spun at 2,400 g for 25 minutes at 4°C. After centrifuging, approximately 3 ml of serum was withdrawn using a pipette and divided between two 1.5-ml micro-centrifuge tubes (Fisher Scientific, Pittsburgh, PA). Micro-centrifuge tubes were stored in a -20°C freezer until analysis.

Serum samples were allowed to thaw and then analyzed for albumin, blood urea nitrogen, C-reactive protein, glucose, non-esterified fatty acids, total protein, and triglyceride concentrations using a COBAS FARA II clinical analyzer (Roche Diagnostic Systems, Indianapolis, IN). Colorimetric procedures were used to determine the concentration of blood metabolites. Roche diagnostic kits and reagents were used for all clinical assays except C-reactive protein. Antibody Reagent Set II was used for C-reactive protein (Daisorin, Stillwater, MN).

Whole body composition:

At the conclusion of the 38-d feeding experiment, each pig was euthanized. As well, a fifth littermate from each set was euthanized at the onset of the experiment for the determination of initial body composition. After euthanization, whole pigs were placed in boxes and stored in a -20°C freezer for grinding and analysis at a latter date.

Whole pigs were ground for composition analysis. Initially, frozen pigs were cut into smaller sections with a band saw and ground three times in a commercial meat grinder (Autio Grinder, Model 801GHP; Astoria, OR) equipped with a 0.64 cm screen. Dry ice was added during the last two grindings to reduce moisture loss. Following grinding, samples were thoroughly mixed and a sub-sample of approximately 500 g was collected. Whole body sub-samples were then freeze-dried (Virtis Freezemobile 12SL; Gardiner, NY) and further ground in a Wiley Mill (Standard Model No.3; Arthur H. Thomas Co., Philadelphia, PA) equipped with a 2-mm screen.

Whole body samples were analyzed for DM, protein content, lipid concentration, ash levels, and gross energy values. Proximate analysis of whole body samples was performed in triplicate and averaged for each analytical procedure. Dry matter for whole body samples was determined by standard AOAC procedures (1998). Protein content of whole body samples was determined by Kjeldahl methodology (FOSS Tecator, 2020 Digestor, 2400 Kjeltec Analyzer Unit; Hoganas, Sweden). Lipid content of whole body samples was determined by standard extract procedures. Dried samples were placed in a soxhlet containing petroleum ether for 48 h to allow for lipid extraction. Upon removal from the soxhlet, samples were air dried for 1 h and placed in a drying oven for 18 h at 100°C. Lipid content of the samples was then calculated using the percent moisture and

the amount of lipid extraction. Ash levels were determined by heating a sample for 5.5 hr at 500°C in a muffle furnace (Sybron, Dubuque, IA). Additionally, whole body samples were assayed for gross energy by bomb calorimetry (Parr 1261 Isoperibol Calorimeter; Moline, IL).

Littermates from each block of pigs were killed at the start of the experiment for the determination of initial body composition. Pigs were handled in a similar manner as those used in the study and whole body composition was determined by the same analytical procedures. Regression equations were generated, by regressing live body weights against whole body characteristics, for whole body protein (R^2=.97), whole body fat (R^2=.51), whole body ash (R^2=.98), whole body water (R^2=.95), and whole body energy (R^2=.74). These regression equations were used to estimate initial body composition of the pigs killed at the end of the feeding study. The amount of protein, lipid, ash, moisture, and energy accrued during the 38-d study were calculated by subtracting the final concentration determined for each variable from the estimated initial concentration of each variable for each pig. In calculating energy accretion, we assumed one gram of protein contained 5.6 kcal of energy and one gram of lipid contained 9.4 kcal of energy.

Statistical analysis:

Nutrient digestibility data were analyzed as a randomized complete block design within each period. There were no treatment by period interactions as trends were similar within periods. Thus, digestibility data were pooled across periods. All data (growth performance, digestibility, composition, tissue accretion, and blood metabolites) were then analyzed in a randomized complete block design using analysis of variance as

described by Steel et al. (1997). The model included the effects of block (rep), treatment, and block x treatment (error). The effects of increasing dietary L-carnitine concentration were partitioned into linear and curvilinear components using orthogonal polynomial contrasts. Due to unequally spaced dietary levels of L-carnitine, coefficients were derived using the integrative matrix language (PROC IML) procedure of SAS (Version 7.11). Pen served as the experimental unit.

Results

The chemical composition of the four dietary treatments is reported in Table 2.2. The supplemented levels of L-carnitine concentration (Table 2.3) averaged across periods, were 0, 25, 50, and 89 ppm, respectively. Supplemented levels of carnitine were in agreement with calculated levels. Because of the inclusion of 5% soybean oil, all diets had a caloric density between 4,125 and 4,206 kcal/kg.

Growth performance trends (Table 3.1) were similar to those previously reported in Exp. 1. Pigs fed dietary L-carnitine had improved ADG (quadratic, $P < 0.03$) from d 0-38, with the greatest response being observed in pigs fed 50 ppm L-carnitine. Additionally, increasing trends were noted in ADFI (quadratic, $P < 0.08$) and G:F (linear, $P < 0.08$) in response to supplemental L-carnitine. Furthermore, during Phase 2, ADG (274, 319, 337, and 368 g) increased (linear, $P < 0.02$) due to added L-carnitine.

Table 3.1. Growth performance of weanling pigs[a]

	L-Carnitine, ppm					P >:[b]		
Item:	0	25	50	100	SE	Linear	Quad.	Cubic
Overall, d 0-38								
ADG, g	255	304	347	323	15.6	0.03	0.03	--
ADFI, g	340	379	424	393	19.7	0.13	0.08	--
G:F	0.751	0.802	0.826	0.819	0.02	0.08	0.15	--

[a]Least squares means for six pigs/trt
[b]Dashes indicate P > 0.20

Apparent energy digestibility of weanling pigs is reported in Table 3.2. An increase (linear, $P < 0.04$) in GE intake was noted in pigs fed increasing levels of dietary L-carnitine; however, this increase in GE intake was associated with a linear increase ($P < 0.01$) in daily feed consumption. Although fecal GE excretion and urinary GE loss increased (linear, $P < 0.01$), increasing trends (linear, $P = 0.13$) in DE and ME (kcal/d), respectively, were still observed due to larger increases in GE intake. However, when DE and ME were converted to a concentration basis (kcal/kg), little difference was observed between pigs fed 0, 25, and 50 ppm L-carnitine, while pigs fed 100 ppm L-carnitine had lower DE and ME (kcal/kg) values (linear, $P < 0.01$). Additionally, the inclusion of 25 and 50 ppm L-carnitine had little affect on DE:GE and ME:GE when compared with the control, while 100 ppm L-carnitine decreased (quadratic, $P < 0.09$) DE:GE and ME:GE in weanling pigs.

Table 3.2. Apparent energy digestibility of weanling pigs[a,b]

	L-Carnitine, ppm					P >:[c]		
Item:	0	25	50	100	SE	Linear	Quad.	Cubic
ADFI, g/d	415	430	476	479	14	0.01	--	--
GE Intake, kcal/d	1,944	2,001	2,147	2,166	77	0.04	--	--
Fecal GE, kcal/d	217	214	239	280	8.3	0.01	0.17	--
DE, kcal/d	1,727	1,788	1,907	1,886	76	0.13	--	--
DE, kcal/kg	4,159	4,149	4,162	4,059	24	0.01	0.13	--
Urine GE, kcal/d	10.2	12.7	10.9	13.9	0.7	0.01	--	0.02
ME, kcal/d	1,717	1,775	1,896	1,872	76	0.13	--	--
ME, kcal/kg	4,136	4,121	4,140	4,030	25	0.01	0.12	--
DE:GE, %	88.8	89.2	88.8	86.8	0.5	0.01	0.09	--
ME:DE, %	99.5	99.3	99.5	99.3	0.1	0.01	--	0.01
ME:GE, %	88.3	88.6	88.3	86.2	0.5	0.01	0.09	--

[a]Least squares means for six pigs/trt
[b]Data reported on a dry matter basis
[c]Dashes indicate $P > 0.20$

Trends for nitrogen balance (Table 3.3) were similar to those reported for energy digestibility. An increase (linear, $P < 0.08$) in N intake was observed in pigs fed added L-

carnitine; yet, this increase was associated with an increase in ADFI. Although an increase in fecal N excretion (linear, $P < 0.01$) was observed, a greater increase in N intake was noted in pigs fed 25 and 50 ppm L-carnitine, resulting in the percentage of N absorbed being improved (quadratic, $P < 0.06$) in these two groups. A quadratic response ($P < 0.06$) was also observed in the percentage of N retained, as pigs fed 25 and 50 ppm L-carnitine retained more N, while the inclusion of 100 ppm L-carnitine decreased the percentage of N retained when compared with the control.

Table 3.3. Nitrogen balance of weanling pigs[a,b]

	L-Carnitine, ppm					P >:[c]		
Item:	0	25	50	100	SE	Linear	Quad.	Cubic
N Intake, g/d	14.8	15.7	17.5	17.8	1.5	0.08	--	--
N Fecal exc., g/d	2.1	2.0	2.3	2.7	0.22	0.01	--	--
N Abs., g/d	12.7	13.7	15.2	15.1	1.4	0.13	--	--
N Abs., %	86.0	87.2	87.0	84.5	1.0	0.12	0.06	--
Urine N loss, g/d	1.2	1.3	1.2	1.6	0.36	--	--	--
N Ret., g/d	11.5	12.4	14.1	13.5	1.1	0.12	--	--
N Ret., %	78.6	79.0	80.7	76.2	1.5	0.18	0.06	--
Ret:Abs, %	91.4	90.6	92.8	90.2	1.7	--	--	--

[a]Least squares means for six pigs/trt
[b]Data reported on a dry matter basis
[c]Dashes indicate $P > 0.20$

Whole body percentages of protein and lipid are shown in Table 3.4. An improvement in whole body composition was observed as the percentage of protein increased (linear, $P < 0.01$) and the percentage of lipid decreased (linear, $P < 0.01$) in pigs fed increasing concentrations of L-carnitine. Changes in body composition were also observed in the percentage of ash (quadratic, $P < 0.01$) and the percentage of water (linear, $P < 0.01$), both of which increased with added L-carnitine. Tissue accretion rates are also shown in Table 3.4. A quadratic increase ($P < 0.05$) in the rate of protein (g/d) and energy (kcal/d) accretion was observed with increasing L-carnitine. Although the

rate of lipid accretion was unaffected (P > 0.20) by added L-carnitine, the ratio of protein accretion to lipid accretion improved (linear, P < 0.01) with increasing L-carnitine. The increase in the ratio of protein accretion to lipid accretion indicates a repartitioning of nutrients away from lipid deposition and towards the accretion of protein. In general, the response to L-carnitine tended to plateau at 50 ppm.

Table 3.4. Whole body composition and tissue accretion of weanling pigs[ab]

	L-Carnitine					P >:[c]		
Item	0	25	50	100	SE	Linear	Quad.	Cubic
Protein, %	13.4	14.0	14.0	14.1	0.14	0.01	0.05	--
Lipid, %	8.35	7.42	7.17	6.89	0.17	0.01	0.01	--
Ash, %	2.71	2.88	2.79	2.80	0.03	--	0.01	0.01
Water, %	76.4	77.1	77.4	78.1	0.32	0.01	--	--
Protein Acc., g/d	33.6	42.6	48.5	45.4	2.3	0.01	0.01	--
Lipid Acc., g/d	21.2	21.1	23.7	20.4	1.4	--	0.20	--
Protein:Lipid, g:g	1.59	2.07	2.08	2.23	0.07	0.01	0.01	0.03
Ash Acc., g/d	7.22	9.12	9.83	9.51	0.53	0.01	0.01	--
Water gain, g/d	197	238	271	256	9.5	0.01	0.01	--
Energy gain, kcal/d	379	425	483	426	29.0	--	0.05	--

[a]Least squares means for six pigs/trt
[b]Data reported on an as-is basis
[c]Dashes indicate P > 0.20

The effects of increasing levels of supplemental L-carnitine on blood metabolites of weanling pigs are presented in Table 3.5. Although, serum samples were analyzed for albumin, blood urea nitrogen (BUN), C-reactive protein (CRP), glucose, non-esterified fatty acids (NEFA), protein, and triglycerides, the majority of the changes were observed in CRP, BUN, glucose, and NEFA. Most of the response associated with increasing levels of L-carnitine was observed at the end of Phases 1 (d 10) and 2 (d 24). On d 10, decreases in CRP (quadratic, P < 0.01), BUN (quadratic, P < 0.06), and NEFA (linear, P < 0.05) were noted, while an increase in glucose (quadratic, P < 0.03) was observed due

to added L-carnitine. As well, supplemental L-carnitine increased CRP (linear, $P < 0.02$), glucose (quadratic, $P < 0.05$), and NEFA (quadratic, $P < 0.09$) in weanling pigs on d 24.

Table 3.5. Blood metabolites of weanling pigs[a]

	L-Carnitine					P >:[b]		
Item	0	25	50	100	SE	Linear	Quad.	Cubic
CRP, mg/L[c]								
D 0	1.28	1.29	1.62	1.33	0.97	--	--	--
D 3	1.67	1.84	1.53	2.22	0.37	--	--	--
D 10	2.55	1.62	1.32	2.72	0.29	--	0.01	--
D 24	1.19	1.42	1.74	2.41	0.29	0.02	--	--
D 38	2.77	3.02	2.80	3.66	1.03	--	--	--
BUN, mg/dL[c]								
D 0	7.57	8.25	4.77	5.59	0.98	0.16	--	0.13
D 3	23.12	15.40	12.34	19.17	2.79	--	0.05	--
D 10	17.93	6.97	5.32	8.16	4.39	0.14	0.06	--
D 24	9.33	10.22	8.79	7.89	0.94	--	--	--
D 38	12.20	13.63	10.04	12.11	0.85	--	--	0.03
Glucose, mg/dL								
D 0	142.3	135.3	113.2	139.0	9.34	--	0.11	--
D 3	91.3	99.0	105.6	93.4	5.34	--	0.13	--
D 10	105.2	125.3	125.3	118.5	10.83	--	0.03	--
D 24	126.3	136.2	146.2	118.0	7.29	--	0.05	--
D 38	131.0	121.7	128.6	119.0	4.5	--	--	0.19
NEFA, mmol/L[c]								
D 0	0.47	0.40	0.40	0.41	0.07	--	--	--
D 3	2.43	1.47	1.04	1.56	0.34	--	0.08	--
D 10	0.26	0.13	0.11	0.11	0.37	0.04	0.11	--
D 24	0.09	0.10	0.13	0.07	0.02	--	0.09	--
D 38	0.07	0.07	0.08	0.08	0.01	--	--	--
Albumin, g/dL								
D 0	2.78	2.53	2.53	2.42	0.14	0.11	--	--
D 3	2.98	2.82	2.52	2.57	0.14	0.03	0.19	--
D 10	2.63	2.35	2.40	2.42	0.12	--	--	--
D 24	2.28	2.30	2.46	2.29	0.05	--	0.07	0.14
D 38	2.48	2.40	2.59	2.46	0.09	--	--	--
Protein, g/dL								
D 0	5.07	4.77	4.88	4.87	0.13	--	--	--
D 3	5.33	5.13	4.90	5.17	0.13	--	0.05	--
D 10	4.77	4.32	4.37	4.65	0.12	--	0.01	--
D 24	4.32	4.28	4.47	4.47	0.10	--	--	--
D 38	4.80	4.73	4.88	5.04	0.15	--	--	--

Table 3.5. Continued. Blood metabolites of weanling pigs[a]

Item	L-Carnitine				SE	P >:[b]		
	0	25	50	100		Linear	Quad.	Cubic
TRIG, mg/dL[f]								
D 0	86.33	76.17	96.70	57.82	19.77	--	--	--
D 3	92.83	80.33	63.72	75.39	11.35	--	--	--
D 10	48.00	49.83	52.68	39.58	8.99	--	--	--
D 24	45.00	47.17	58.38	48.08	7.20	--	--	--
D 38	34.50	49.33	38.45	45.95	7.56	--	--	--

[a]Least squares means for six pigs/trt
[b]Dashes indicate P > 0.20
[c]CRP = C-reactive protein
[d]BUN = Blood urea nitrogen
[e]NEFA = Non-esterified fatty acids
[f]TRIG = Triglycerides

Discussion

Results from the apparent nutrient digestibility data combined with findings from the whole body composition data lead one to believe that a repartitioning of nutrients occurred in the weanling pig due to the addition of L-carnitine. We would hypothesize that the availability of L-carnitine was increased in pigs fed diets containing added L-carnitine, resulting in the increased transport of long-chain fatty acids into the mitochondrial matrix. The addition of soybean oil, of which long-chain fatty acids are the major constituent, is an excellent source of energy in the diet. Upon entering the matrix of the mitochondria, fatty acids are oxidized for the production of energy (adenosine triphosphate). A decrease in the percentage of lipid in the weanling pig supports the idea that an increase in the utilization of fatty acids as an energy source occurred. Additionally, a decrease in NEFA levels on d 10 supports the idea of increased fatty acid utilization, while an increase in glucose concentrations on d 10 and 24 suggests a sparing effect on carbohydrates as an energy source.

Upon review of the apparent nutrient digestibility data, the greatest response associated with supplemental L-carnitine was noted in pigs fed 25 and 50 ppm, while the addition of 100 ppm L-carnitine tended to reduce DE:GE and ME:GE when compared with the control. These results answer questions for data reported in Exp. 1 and data presented by Real et al. (2001). Both of these authors reported improvements in ADG and G:F due to the addition of 25 and 50 ppm L-carnitine in the diets of weanling pigs. These findings suggest weanling pigs fed 25 and 50 ppm L-carnitine improved the utilization of dietary energy, resulting in improved growth performance. Uncertainties still exist as to why 100 ppm L-carnitine did not elicit further improvements in energy utilization and growth performance.

Results published by Cho et al. (1999a) noted improvements in proximate nutrient digestibility when 1,000 ppm L-carnitine was supplemented to the diet. Of interest is that Cho and coworkers observed the greatest improvement in nutrient digestibility when diets contained 1.60% total lysine, while diets for our experiment were formulated to contain 1.60% total lysine only during Phase 1 (d 0-10). In a subsequent study by Cho et al. (1999b), improvements in nutrient digestibility due to the addition of 500 ppm L-carnitine were again reported. Not all studies have reported positive nutrient digestibility results. Hoffman et al. (1993) did not improve the utilization of metabolizable energy in diets that contained soybean oil with the supplementation of 800 ppm L-carnitine.

A similar response was observed in nitrogen balance. The supplementation of 25 and 50 ppm L-carnitine to the diet of weanling pigs improved the percentage of nitrogen absorption and the percentage of nitrogen retention. However, the addition of 100 ppm L-carnitine did not elicit further improvements in the percentage of nitrogen absorption

and the percentage of nitrogen retention when compared with the control. Results from the serum analysis are in agreement with these findings. Pigs fed 25 and 50 ppm L-carnitine had lower BUN levels on d 3 and 10. Conclusions from our study are in contrast to those reported by Heo et al. (2000). These authors conducted an experiment to evaluate nitrogen balance in weanling pigs. Low energy, fat-supplemented basal diets containing low or high protein were formulated so that protein accretion would be limited by metabolizable energy. Each basal diet was supplemented with 0 or 500 ppm L-carnitine. The addition of L-carnitine to the diet reduced urinary nitrogen excretion by 14% and improved the percentage of absorbed nitrogen retained in the body, however it did not alter daily fecal nitrogen excretion.

Transformations in whole body composition of pigs fed increasing levels of L-carnitine suggest a repartitioning of nutrients occurred and resulted in improvements in whole body composition and tissue accretion rates. Many studies have reported improvements in body composition and tissue accretion rates as a result of supplemental L-carnitine, although, only a few have also evaluated nutrient digestibility at the same time. Heo et al. (2000) reported that the addition of 500 ppm L-carnitine to the diet decreased the percentage of fat in the carcass and increased the crude protein accretion rate. Heo and coworkers suggested that the improvements in nutrient digestibility they observed explained the improvements in carcass characteristics. Findings from our experiment are consistent with those of Heo et al. in that supplemental L-carnitine had a greater affect on protein accretion rates than lipid accretion rates. However, in contrast to our results, studies by Weeden et al. (1991) and Owen et al. (1996) reported that pigs fed 1,000 ppm L-carnitine had reduced daily fat accretion and, less carcass lipid and daily

lipid accretion, respectively, while supplemental L-carnitine did not alter protein accretion rates.

Implications

This study indicates that the supplementation of L-carnitine improved nitrogen balance and utilization of gross energy provided in the diet of weanling pigs. However, the greatest improvement in nitrogen balance and energy utilization was observed in pigs fed 25 and 50 ppm L-carnitine. As well, pigs fed increasing levels of L-carnitine had improved whole body composition. Supplemental L-carnitine resulted in an increased percentage of protein and a decreased percentage of lipids in weanling pigs. Tissue accretion was also improved due to added L-carnitine, indicated by the increased protein accretion to lipid accretion ratio. We would hypothesize that the supplemental L-carnitine increased the utilization of the soybean oil provided in the diet. The increased utilization of the soybean oil resulted in improved energy utilization, which in turn led to a repartitioning of nutrients away from lipid deposition toward an increase in protein accretion as evident by the improvements in whole body composition.

Chapter IV

Experiment 3

Effects of L-Carnitine and Soybean Oil on Growth Performance in Weanling Pigs.

Abstract: Two-hundred sixteen weanling pigs were used in a 2 x 2 factorial arrangement of treatments in two separate experiments to evaluate the effects of L-carnitine (0 vs 50 ppm) and soybean oil (SBO; 0 vs 5) on growth performance. In Exp. 1, 96 weanling pigs (6.0 kg; 18 d) were randomly allotted based on BW, sex, and litter to four dietary treatments (6 pens/trt of 4 pigs/pen). In Exp. 2, 120 pigs (5.6 kg; 18 d) were randomly allotted to the same treatments as in Exp. 1 (6 pens/trt of 5 pigs/pen). The four dietary treatments were: 1) 0% SBO and 0 ppm L-carnitine; 2) 0% SBO and 50 ppm L-carnitine; 3) 5% SBO and 0 ppm L-carnitine; and 4) 5% SBO and 50 ppm L-carnitine. Pigs were fed in three dietary phases (P1: d 0-10; P2: d 11-24; and P3 d 25-38 with 1.6, 1.4, and 1.2% Lys, respectively). Phase 1 and 2 diets were complex corn-soybean meal-dried whey based containing lactose and additional protein sources, while diets for P3 were corn-soybean meal based. Pigs were weighed and feed consumption recorded weekly for the determination of ADG, ADFI, and G:F. Additionally, in Exp. 2, two pigs per pen were bled by jugular venipuncture at the initiation of the experiment and subsequently, at the conclusion of each dietary phase. There were no treatment by experiment interactions; therefore, data were pooled across experiments (12 pens/trt). For the 38-d study, ADG, ADFI, and G:F were: 394, 398, 370, and 391 g; 556, 567, 536, and 540 g;

and 0.696, 0.703, 0.690, and 0.725, respectively. Pigs fed SBO tended ($P < 0.10$) to grow slower and consume less feed compared to those not fed SBO, but G:F was not affected ($P > 0.10$). The addition of L-carnitine did not affect ($P > 0.10$) ADG or ADFI; however, it did improve ($P < 0.01$) G:F. Also, the increase in G:F associated with L-carnitine was more pronounced in pigs fed SBO than those not fed SBO (carnitine x SBO, $P < 0.08$). The greatest response to L-carnitine occurred in P2 with an increase in ADG ($P < 0.05$) and G:F ($P < 0.01$). In contrast, the response (G:F) to SBO was greatest during P3. Furthermore, a marked response to L-carnitine and soybean oil was noted in serum stores of albumin, blood urea nitrogen, non-esterified fatty acids, protein, and triglycerides. These results suggest that the addition of 50 ppm L-carnitine improved growth performance in weanling pigs; however, supplemental L-carnitine was more effective when SBO was provided in the diet. As well, the addition of L-carnitine and SBO altered blood metabolites in weanling pigs.

Introduction

Carnitine is a naturally occurring vitamin B-like compound that is present in muscle and other tissues. The primary role of carnitine in intermediary metabolism is as a cofactor for enzymes that shuttle long-chain fatty acids across the otherwise impermeable inner mitochondrial membrane into the matrix of the mitochondria. Once in the mitochondrial matrix, long-chain fatty acids are utilized in the production of energy (adenosine triphosphate) via β-oxidation and oxidative phosphorylation (Fritz and Yue, 1963; Bray and Briggs, 1980).

In an effort to diminish post-weaning lag, complex, nutrient dense diets have been developed to be fed to early-weaned pigs (Tokach et al., 1994). The increase in caloric

density within these complex diets is typically obtained from high inclusion levels of milk products (20 to 40%) and supplemental fat (5 to 10%). However, in research conducted by Mahan (1991) and Tokach et al. (1995), fat addition to the diet did not elicit an improvement in ADG of pigs less than 28 d of age. Yet, when fat was supplemented to the diet an improvement in ADG and feed efficiency was observed from d 14 to 35 post-weaning (Mahan, 1991; Tokach et al., 1995). Ironically, the period immediately post-weaning is when L-carnitine synthesis is lowest in weanling pigs (Kerner et al., 1984). Another set back is that plant products, which are a major constituent of weanling pig diets, are low in or devoid of carnitine. Therefore, studies have been conducted to evaluate the effects of supplementing L-carnitine to the diet of weanling pigs on growth performance. Results from Exp. 1 and Real et al. (2001) indicated that supplementing 50 ppm L-carnitine to diets containing added fat, improved growth performance in weanling pigs. Therefore, we speculated that immediately post-weaning, when carnitine stores are minimal, supplemental L-carnitine may be required before an improvement in growth performance due to added fat is observed. Thus, the objective of our study was to evaluate the effects of supplementing L-carnitine and soybean oil to the diet on growth performance and blood metabolites of weanling pigs.

Materials and Methods

Two-hundred sixteen Yorkshire, Hampshire, and crossbred (Yorkshire x Hampshire) pigs were weaned at 20 ± 2 d and utilized in two separate 38-d experiments. In each experiment, pigs were used in a 2 x 2 factorial arrangement of treatments and allotted randomly by initial BW, while equalizing ancestry and gender across treatments, to four dietary treatments in a randomized complete block design. The four dietary

treatments were obtained from combining either 0 or 50 ppm L-carnitine with either 0 or 5% soybean oil (SBO). The four dietary treatments were: 1) 0% SBO and 0 ppm L-carnitine; 2) 0% SBO and 50 ppm L-carnitine; 3) 5% SBO and 0 ppm L-carnitine; and 4) 5% SBO and 50 ppm L-carnitine. The composition of the basal diet for the three dietary phases is shown in Table 4.1.

Table 4.1. Composition of basal diets (as-is basis).

	Diets[a]		
Ingredient, %	Phase 1	Phase 2	Phase 3
Corn	30.19	50.19	56.84
SBM (48%)	20.75	25.00	33.75
Whey, dried	20.00	10.00	--
Lactose	10.00	--	--
Plasma, spray-dried	5.00	2.50	--
Blood meal, spray-dried	2.50	2.50	--
Fish meal, menhaden	2.50	--	--
Dicalcium phosphate	1.53	2.11	2.37
Limestone	0.42	0.61	0.68
DL-methionine	0.20	0.13	--
Ethoxyquin	0.03	0.03	0.03
Salt	0.25	0.25	0.35
Trace min/Vit premix[b]	0.30	0.30	0.30
Zinc Oxide	0.28	0.28	--
Copper sulfate	--	--	0.08
Antibiotic[c]	1.00	1.00	0.50
Cornstarch[d]	5.05	5.10	5.10

[a]Diets formulated to contain 1.6, 1.4, and 1.2% total lysine for P1, P2, and P3, respectively

[b]Provided the following per kg feed: Zn, 120 mg; Fe, 120 mg; Mn, 24 mg; Cu, 12 mg; I, .36 mg; Se, .36 mg; vitamin A, 6,615 IU; vitamin D_3, 661 IU; vitamin E, 40 IU; vitamin K (menadione activity), 4.4 mg; riboflavin, 6.6mg; d-pantothenic acid, 30 mg; niacin, 40 mg; vitamin B_{12}, 33 ug; d-biotin, 265 ug; choline, 144 mg; and folic acid, 2 mg.

[c]P1 and P2 contained Neo-terramycin® (100 g/ton oxytetracycline & 140 g/ton neomycin base) and P3 contained Lincomix® (200 g/ton lincomycin)

[d]L-carnitine (Carniking 10, Lonza Inc., Fair Lawn, NJ) substituted at 0.05% and SBO substituted at 5.0% for cornstarch to obtain the four dietary treatments

In Exp. 1, 96 weanling pigs (6.0 kg initial BW) were randomly allotted to the four dietary treatments with 6 pens per treatment of 4 pigs per pen. In Exp. 2, 120 weanling

pigs, initially averaging 5.6 kg, were randomly allotted to the four dietary treatments with 6 pens per treatments of 5 pigs per pen. Pigs in both experiments were fed in three dietary phases: [Phase 1 (P1), d 0-10; Phase 2 (P2), d 11-24; and Phase 3 (P3), d 25-38]. Complexity of the diet changed with phases to satisfy the nutrient requirements (NRC, 1998) of the weanling pig. As well, total lysine concentration of the diets was formulated to exceed NRC (1998) recommendations, thereby preventing any lysine deficiency effects on growth performance. Phase 1 (1.6% Lys) and Phase 2 (1.4% Lys) diets were complex corn-soybean meal-dried whey based containing lactose, spray-dried animal plasma, spray-dried blood meal, and fish meal, while Phase 3 (1.2% Lys) diets were typical corn-soybean meal based. All diets were fed in pelleted form.

Pigs were housed in temperature-controlled nursery rooms and grouped in elevated pens with wire flooring. Each pen provided 1.72 square meters of space and contained a five-hole, stainless steel feeder and one nipple waterer that allowed for the ad libitum access to feed and water throughout the experiment. Room temperature was maintained initially at 31°C, and decreased by 1.1°C weekly until the room temperature reached 25.5°C. Pigs were weighed and feed consumption was measured weekly for the determination of average daily gain (ADG), average daily feed intake (ADFI) and gain:feed (G:F).

At the start of Exp. 2, blood samples were drawn from the two pigs closest to the mean pen weight. Subsequent blood samples were taken at the end of each dietary phase, d 10, 24, and 38, respectively. Blood samples were drawn using a 22 x 1" gauge needle (Sherwood Medicals; St. Louis, MO) by jugular venipuncture into a 10-ml anticoagulant free vacutainer (Becton Dickinson; Franklin Lakes, NJ). After collection, samples were

placed in an ice bath for 2 h and allowed to coagulate. Following coagulation, vacutainers were centrifuged at 2,400 g for 25 min at 4°C (J-6B Centrifuge; Beckman Instruments, Inc., Fullerton, CA). Next, approximately 3 ml of a serum sample was pipetted from the vacutainers into two 1.5-ml micro-centrifuge tubes. Micro-centrifuge tubes were then stored in a -20°C freezer until analysis.

Chemical analysis:

Diets were analyzed for DM according to AOAC (1998) procedures. Gross energy determinations were made by bomb calorimetry (Parr 1261 Isoperibol Calorimeter, Moline, IL), and nitrogen determinations were performed by Kjeldahl methodology (FOSS Tecator, 2400 Kjeltec Analyzer unit, 2020 Digestor, Hoganas, Sweden). As well, diets were analyzed for L-carnitine concentrations using methods described by Parvin and Pande (1977). Chemical composition of the control diets is shown in Table 4.2. The L-carnitine concentration for the four dietary treatments for each phase is detailed in Table 4.3.

Table 4.2. Chemical composition of basal diets (as-is basis).

Item	Phase 1	Phase 2	Phase 3
Calculated analysis			
ME, kcal/kg	3,155	3,164	3,193
Crude protein, %	22.58	21.70	21.19
Total lysine, %	1.60	1.40	1.20
Digestible lysine, %	1.36	1.18	1.00
Digestible threonine, %	0.80	0.70	0.61
Digestible Met + Cys, %	0.80	0.69	0.56
Digestible tryptophan, %	0.25	0.23	0.21
Calcium, %	0.90	0.90	0.90
Phosphorus, %	0.80	0.80	0.80
Available phosphorus, %	0.62	0.56	0.51
Analyzed values			
GE, kcal/kg	3,938	4,025	3,901
Crude protein, %	22.49	20.82	20.41

Table 4.3. L-Carnitine concentration of diets[a].

Item:	Calculated Concentration	Analyzed Concentration	Supplemented Level[b]
		L-carnitine, ppm	
Phase 1			
Diet 1	0	37	0
Diet 2	50	92	55
Diet 3	0	37	0
Diet 4	50	75	38
Phase 2			
Diet 1	0	19	0
Diet 2	50	70	51
Diet 3	0	19	0
Diet 4	50	74	55
Phase 3			
Diet 1	0	1	0
Diet 2	50	50	49
Diet 3	0	1	0
Diet 4	50	48	47

[a]Analysis reported on an as-is basis
[b]Supplemented level obtained by subtracting analyzed concentration from analyzed concentration of unsupplemented diets (diets 1 & 3)

Serum samples were allowed to thaw and then analyzed for albumin, blood urea nitrogen (BUN), C-reactive protein (CRP), glucose, non-esterified fatty acids (NEFA), total protein, and triglycerides (TRIG) (COBAS FARA II clinical analyzer; Roche Diagnostic Systems, Indianapolis, IN). Determination of blood metabolite levels was made by colorimetric procedures. Roche diagnostic kits and reagents were used for all clinical chemistries except C-reactive protein. For C-reactive protein an antibody Reagent Set II was used (Daisorin; Stillwater, MN).

Statistical analysis:

The data were analyzed as a randomized complete block design within each experiment using analysis of variance procedures (Steel et al., 1997). There were no treatment by experiment interactions as trends were similar within experiments. Thus,

data were pooled across experiments with 12 pens per treatment and analyzed as a 2 x 2 factorial in a randomized complete block design. The model included the effects of block (rep), treatment, and block x treatment (error). Orthogonal contrasts were used to test the effects of L-carnitine level (0 vs 50 ppm), SBO level (0 vs 5%), and the L-carnitine level x SBO level interaction. Pen served as the experimental unit.

Results

The chemical analyses of the four dietary treatments are presented in Tables 4.2 and 4.3. Supplemented levels of L-carnitine were consistent with calculated levels, signifying proper diet mixing. Five percent added soybean oil increased the caloric density of the diet by approximately 200-225 kcal/kg.

The effects of L-carnitine and soybean oil on pig performance are shown in Table 4.4. For the 38-d study, pigs fed SBO tended ($P < 0.10$) to grow slower and consume less feed compared with those not fed SBO, but G:F was not affected ($P > 0.10$). The addition of L-carnitine did improve ($P < 0.01$) G:F; however, it did not affect ($P > 0.10$) ADG or ADFI. Also, the increase in G:F associated with L-carnitine was more pronounced in pigs fed SBO than those not fed SBO (L-carnitine x SBO, $P < 0.08$). The greatest response to L-carnitine occurred in P2 with an increase in ADG ($P < 0.05$) and G:F ($P < 0.01$). As well, the addition of SBO to the diet decreased ADG ($P < 0.04$) and ADFI ($P < 0.02$) during P2. During P3, the inclusion of SBO had little affect on ADG ($P > 0.20$); however, a decrease in ADFI ($P < 0.02$) was observed, resulting in an increase in G:F ($P < 0.01$). These results suggest that the addition of 50 ppm L-carnitine improved growth performance in weanling pigs; however, supplemental L-carnitine was more effective when SBO was included in the diet.

Table 4.4. Growth performance of weanling pigs[a]

SBO, %	0	0	5	5		P >:[b]		
Carnitine, ppm	0	50	0	50	SE	SBO	Carnitine	Int[c]
Phase 1, d 0-10								
ADG, g/d	186	189	177	179	8.9	--	--	--
ADFI, g/d	213	211	208	208	7.7	--	--	--
G:F	0.87	0.90	0.85	0.86	0.03	0.19	--	--
Phase 2, d 11-24								
ADG, g/d	420	428	386	418	9.8	0.03	0.05	--
ADFI, g/d	565	564	534	537	11.5	0.02	--	--
G:F	0.74	0.76	0.72	0.78	0.01	--	0.01	0.14
Phase 3, d 25-38								
ADG, g/d	517	518	493	516	12.7	--	--	--
ADFI, g/d	819	824	773	780	18.0	0.02	--	--
G:F	0.63	0.63	0.64	0.66	0.01	0.01	--	0.14
Overall, d 0-38								
ADG, g/d	394	398	370	392	8.7	0.09	0.16	--
ADFI, g/d	566	567	536	540	11.4	0.02	--	--
G:F	0.70	0.70	0.69	0.73	0.01	0.18	0.01	0.08

[a]Least squares means for six pens/trt of four to six pigs/pen
[b]Dashes indicate P > 0.20
[c]Int = SBO level x L-carnitine level interaction

The effects of dietary L-carnitine and soybean oil on blood metabolites of weanling pigs are presented in Table 4.5. The greatest response associated with L-carnitine and soybean oil occurred at the end of Phases 1 (d 10) and 2 (d 24). On d 10, pigs fed SBO had higher TRIG ($P < 0.08$) and NEFA ($P < 0.04$) levels than those not fed SBO. As well, the addition of L-carnitine tended ($P < 0.07$) to decrease NEFA levels on d 10; however, the decrease in NEFA levels was more obvious in pigs fed SBO than those not fed SBO (L-carnitine x SBO, $P = 0.11$). The supplementation of L-carnitine also increased ($P < 0.06$) albumin status on d 10. However, on d 24 albumin levels decreased in pigs fed SBO due to the addition of L-carnitine, while the addition of L-carnitine increased albumin levels in pigs not fed SBO (L-carnitine x SBO, $P < 0.09$).

Table 4.5. Blood metabolites of weanling pigs[a]

SBO, %	0	0	5	5		P >:[b]		
Carnitine, ppm	0	50	0	50	SE	SBO	Carnitine	Int[c]
Albumin, g/dL								
D 0	2.82	2.80	2.86	2.77	0.09	--	--	--
D 10	2.32	2.48	2.44	2.49	0.05	--	0.06	--
D 24	2.37	2.52	2.52	2.42	0.07	--	--	0.09
D 38	2.70	2.73	2.65	2.59	0.08	--	--	--
BUN, mg/dL[d]								
D 0	6.30	7.24	6 94	7.58	0.72	--	--	--
D 10	6.13	7.43	7.24	6.14	1.07	--	--	--
D 24	7.55	8.64	7.82	6.31	0.47	0.04	--	0.01
D 38	12.62	12.66	11.98	11.09	0.63	0.10	--	--
Protein, g/dL								
D 0	5.08	5.41	5.43	5.21	0.12	--	--	0.04
D 10	4.48	4.66	4.68	4.66	0.08	--	--	--
D 24	4.28	4.73	4.39	4.38	0.15	--	0.17	0.14
D 38	4.73	1.90	4.65	4.53	0.13	0.09	--	--
NEFA, mmol/L[e]								
D 0	0.363	0.318	0.361	0.308	0.05	--	--	--
D 10	0.097	0.093	0.171	0.103	0.02	0.04	0.07	0.11
D 24	0.089	0.121	0.107	0.113	0.02	--	--	--
D 38	0.059	0.057	0.104	0.093	0.01	0.01	--	--
TRIG, mg/dL[f]								
D 0	98.17	76.75	92.92	112.08	13.88	--	--	0.16
D10	33.17	32.33	36.50	38.75	2.64	0.08	--	--
D 24	38.25	38.83	39.08	39.58	3.54	--	--	--
D 38	43.33	36.08	41.75	49.08	5.03	--	--	0.17
CRP, mg/L[g]								
D 0	2.19	1.33	1.78	3.75	0.70	--	0.20	0.08
D10	2.76	3.67	2.04	3.73	0.59	0.04	--	--
D 24	4.31	4.16	4.42	6.23	1.00	--	--	--
D 38	5.01	5.54	4.30	5.20	0.76	--	--	--
Glucose, mg/dL								
D 0	128.92	131.92	152.00	132.67	9.61	--	--	--
D10	96.92	103.83	106.42	103.83	5.02	--	--	--
D 24	110.92	107.08	107.83	104.92	4.52	--	--	--
D 38	121.83	118.67	123.33	125.67	5.19	--	--	--

[a]Least squares means for six pigs/trt
[b]Dashes indicate P > 0.20
[c]Int = SBO level x L-carnitine level interaction
[d]BUN = Blood urea nitrogen
[e]NEFA = Non-esterified fatty acids
[f]TRIG = Triglycerides
[g]CRP = C-reactive protein

A similar tendency was also noted in BUN levels on d 24, as pigs fed SBO had lower ($P < 0.04$) BUN levels than those not fed SBO. However, when L-carnitine was added to the diet, a decrease in BUN levels was noted in pigs fed SBO, while an increase in BUN levels was observed in pigs not fed SBO (L-carnitine x SBO, $P < 0.01$). Responses to added soybean oil were also noted in BUN, protein, and NEFA levels at the end of Phase 3 (d 38). The supplementation of SBO decreased BUN ($P < 0.10$) and protein ($P < 0.09$) levels, while SBO increased NEFA ($P < 0.01$) levels of weanling pigs on d 38.

Discussion

Results from this study suggest that the addition of 50 ppm L-carnitine does not improve the performance of weanling pigs fed nutrient dense diets containing soybean oil during Phase 1 (d 0-10). These findings are in agreement with data reported by Owen et al. (1996) and Weeden et al. (1990). These authors reported that L-carnitine and SBO had no affect on pig performance immediately post-weaning (d 0-14). However, results from our study indicate that after approximately 10 d, weanling pigs have the capabilities to improve performance criteria due to supplemental L-carnitine and soybean oil. Although SBO decreased ADG and ADFI during Phase 2, the inclusion of L-carnitine elicited an improvement in ADG and G:F, with the greatest response being observed in pigs fed SBO. Owen et al. (1996) reported that from d 14-35 after weaning, increasing dietary L-carnitine improved G:F, while SBO improved ADG and G:F during this period. Weeden et al. (1990) also reported improvements in feed efficiency due to supplemental L-carnitine during 3 to 5 wk post-weaning. It is worth noting that Owen et al. (1996) and Weeden et al. (1990) supplemented up to 1,000 ppm L-carnitine and 10% soybean oil.

Given the metabolic role of carnitine in fatty acid oxidation, we would hypothesize that an increased carnitine status enhanced the utilization of dietary SBO, resulting in improved growth performance during P2. Additionally, we would hypothesize that increased fatty acid oxidation would decrease triglyceride and non-esterified fatty acid levels during this period. However, neither triglycerides nor non-esterified fatty acids were affected by carnitine and soybean oil supplementation on d 24. Li et al. (1999) also reported no differences in triglyceride levels on d 14 and 28 of pigs weaned at 35 days of age and fed 50 ppm L-carnitine.

For the overall study (d 0-38), improvements in growth performance were observed due to the supplementation of L-carnitine only when the diet contained soybean oil. Given the increased caloric density of diets containing added SBO, as would be expected, the weanling pig consumed less feed to meet its energy requirement. A decrease in daily gain was also observed in pigs fed SBO when compared with pigs fed diets without added SBO. However, when L-carnitine was added to the diet, an increase in daily gain was noted in pigs fed SBO, resulting in an improved feed efficiency. Yet, performance criteria of weanling pigs, fed diets without SBO, were not affected by the addition of L-carnitine. This would suggest sufficient carnitine biosynthesis for energy production in pigs fed diets without added fat sources. In contrast to results from our study, Hoffman et al. (1993) reported that the addition of L-carnitine did not affect any performance criteria, including ADG, G:F, and gain per megacalorie of ME, in pigs fed diets with and without high levels of soybean oil.

Implications

Soybean oil and other fat sources are supplemented to the diet of weanling pigs to increase the energy density of the diet, in an effort to improve growth performance, thereby diminishing the effects of post-weaning lag. However, the biosynthesis of carnitine is minimal immediately after weaning in pigs, possibly hindering the utilization of the increased caloric density of the diet. Results from the present study suggest that supplemental L-carnitine to the diet of weanling pigs does not improve the response to added soybean oil immediately post-weaning (d 0-10). However, after an adaptation period, added L-carnitine enhances growth performance when soybean oil is provided in the diet of weanling pigs.

Chapter V

Experiment 4

Effects of L-Carnitine and Source of Dietary Fat on Growth Performance of Weanling Pigs.

Abstract: Two-hundred thirty-six weanling pigs were used in two separate experiments to evaluate the effects of L-carnitine (0 vs 50 ppm) and source of dietary fat [soybean oil (SBO) vs coconut oil (CO)] on growth performance. Pigs were randomly allotted to one of five dietary treatments, in a 2 x 2 factorial arrangement of treatments, with a negative control, in a randomized complete block design. The five dietary treatments were obtained from combining the control with either 0 or 50 ppm L-carnitine and either 5% SBO or 5% CO. The five dietary treatments were: 1) control; 2) control + 0 ppm L-carnitine and 5% SBO; 3) control + 50 ppm L-carnitine and 5% SBO; 4) control + 0 ppm L-carnitine and 5% CO; and 5) control + 50 ppm L-carnitine and 5% CO. In Exp. 1, 116 weanling pigs (5.2 kg; 21 ± 1 d) were randomly allotted based on BW, sex, and litter to the five dietary treatments (Trt 1 = 4 pens of 4 to 5 pigs/pen; Trt 2-5 = 5 pens/trt of 4 to 5 pigs/pen). Pigs were fed in three dietary phases (P1: d 0-13; P2: d 14-27; and P3 d 28-41). In Exp. 2, 120 weanling pigs (5.3 kg) were randomly allotted to the same treatments as in Exp. 1 (Trt 1 = 4 pens of 5 pigs/pen; Trt 2-5 = 5 pens/trt of 5 pigs/pen). Pigs were fed in three dietary phases (P1: d 0-10; P2: d 11-24; and P3 d 25-38). In both experiments, diets were formulated to contain 1.6, 1.4, and 1.2% Lys for P1, P2, and P3, respectively. Pigs were weighed and feed intake recorded at the start and end of each

dietary phase for the determination of ADG, ADFI, and G:F. There were no treatment by experiment interactions; therefore, data were pooled across experiments. Overall performance data for ADG, ADFI, and G:F were: 334, 330, 335, 337, and 338 g; 479, 460, 471, 471, and 452 g; and 0.699, 0.719, 0.712, 0.718, and 0.750, respectively. The addition of a fat source to the diet of weanling pigs improved overall G:F ($P < 0.04$) when compared with the control. However, when L-carnitine was supplemented to the diet, an improvement in G:F was noted only in pigs fed CO (L-carnitine x Fat source, $P < 0.06$). Added L-carnitine and fat did not affect overall ADG and ADFI ($P > 0.20$). The greatest response associated with supplemental fat sources was observed in Phase 1. Pigs fed a diet containing an additional fat source consumed more feed ($P < 0.01$) and grew faster ($P < 0.08$) than control pigs. As well, the addition of L-carnitine, during Phase 1, only elicited an improvement in ADG and ADFI in pigs fed SBO (L-carnitine x Fat source, $P < 0.06$). Minimal responses were observed in growth performance, during Phase 2 and 3, due to the addition of L-carnitine or a dietary fat source. Results from this study suggest that the addition of a dietary fat source can enhance growth performance in weanling pigs. However, the improvements in performance criteria were greater in pigs fed coconut oil than pigs fed soybean oil. Additionally, the response in growth performance that was attributed to the supplementation of L-carnitine varied between pigs fed soybean oil and pigs fed coconut oil. Also, the response associated with L-carnitine varied among phases.

Introduction

Carnitine is a naturally occurring compound that is also known as vitamin B_T, to indicate its place in the B-group of vitamins (Fraenkel, 1948). The presence of carnitine

in muscle and other tissues is necessary to facilitate the transfer of long-chain fatty acids into the enzymatically active intra-mitochondrial matrix, resulting in the production of adenosine triphosphate (energy) via β-oxidation and oxidative phosphorylation (Fritz and Yue, 1963; Bray and Briggs, 1980). However, other metabolic roles of carnitine have been proposed. Markwell et al. (1973) reported that in pig liver both short- and medium-chain carnitine acyltransferase activity is present in microsomes, peroxisomes, and mitochondria. The hepatic role of these short- and medium-chain carnitine acyltransferases is to shuttle chain-shortened products, due to β-oxidation, out of peroxisomes (Bieber et al., 1982).

Typically, soybean oil is added as a fat source, to increase the caloric density of the diet. The fatty acid profile of soybean oil reveals that is comprised of approximately 95% long-chain fatty acids (≥ 16 C). However, the weanling pig is able to more efficiently utilize dietary fat sources that are constituted primarily of medium-chain fatty acids. Friedman and Nylund (1980) demonstrated through *in vitro* studies that medium-chain fatty acids are easily solubilized by bile salts. Consequently, medium-chain fatty acids have a greater potential to enter the micellar phase of the lipid-bile interface than do long-chain fatty acids (Hofman, 1963). Given that coconut oil contains a high percentage (>80%) of medium-chain fatty acids, it may be more effective as a source of dietary fat for weanling pigs than other fat sources containing high concentrations of long-chain fatty acids.

Results from Experiment 3 suggest that the supplementation of 50 ppm L-carnitine was more effective in enhancing growth performance when soybean oil was provided in the diet. Therefore, the objective of our study was to evaluate the effects of

supplementing L-carnitine with either soybean oil (long-chain fatty acids) or coconut oil (medium-chain fatty acids) to the diet on growth performance of weanling pigs.

Materials and Methods

Two-hundred thirty-six Yorkshire, Hampshire, and crossbred (Yorkshire x Hampshire) pigs were weaned at 21 ± 1 d and utilized in two separate experiments. The experimental design for each experiment was a 2 x 2 factorial arrangement of treatments, with a negative control. Pigs were allotted randomly by initial BW, stratifying pigs by litter and gender across treatments, to five dietary treatments in a randomized complete block design. The five dietary treatments were obtained from combining the control diet with either 0 or 50 ppm L-carnitine and either 5% soybean oil (SBO) or 5% coconut oil (CO). The five dietary treatments were: 1) control; 2) control + 0 ppm L-carnitine and 5% SBO; 3) control + 50 ppm L-carnitine and 5% SBO; 4) control + 0 ppm L-carnitine and 5% CO; and 5) control + 50 ppm L-carnitine and 5% CO. The composition of the control diet for the three dietary phases is shown in Table 5.1.

In Exp. 1, 116 weanling pigs (5.2 kg) were randomly allotted to the five dietary treatments. Four pens, with 4 to 5 pigs per pen, were allotted to Treatment 1, while five pens, with 4 to 5 pigs per pen, were assigned to Treatments 2 through 5. Pigs were fed in three dietary phases [Phase 1 (P1): d 0-13; Phase 2 (P2): d 14-27; and Phase 3 (P3) d 28-41). In Exp. 2, 120 weanling pigs (5.3 kg) were randomly allotted to the same treatments as in Exp. 1. Pigs were allotted with 5 pigs per pen. Four pens were allotted to Treatment 1, while five pens were allotted to Treatments 2 through 5. Pigs were fed in three dietary phases (P1: d 0-10; P2: d 11-24; and P3 d 25-38). In both experiments, diets were formulated to contain 1.6, 1.4, and 1.2% Lys for Phase 1, Phase 2, and Phase 3,

respectively. As well, complexity of the diet changed with phases to satisfy the nutrient requirements (NRC, 1998) of the weanling pig. Phase 1 and Phase 2 diets were complex corn-soybean meal-dried whey based containing lactose, spray-dried animal plasma, spray-dried blood meal, and fish meal, while Phase 3 diets were typical corn-soybean meal based. All diets were fed in pelleted form.

Table 5.1. Composition of control diets (as-is basis).

	Diets[a]		
Ingredient, %	Phase 1	Phase 2	Phase 3
Corn	30.19	50.19	56.84
SBM (48%)	20.75	25.00	33.75
Whey, dried	20.00	10.00	--
Lactose	10.00	--	--
Plasma, spray-dried	5.00	2.50	--
Blood meal, spray-dried	2.50	2.50	--
Fish meal, menhaden	2.50	--	--
Dicalcium phosphate	1.53	2.11	2.37
Limestone	0.42	0.61	0.68
DL-methionine	0.20	0.13	--
Ethoxyquin	0.03	0.03	0.03
Salt	0.25	0.25	0.35
Trace min/Vit premix[b]	0.30	0.30	0.30
Zinc Oxide	0.28	0.28	--
Copper sulfate	--	--	0.08
Antibiotic[c]	1.00	1.00	0.50
Cornstarch[d]	5.05	5.10	5.10

[a]Diets formulated to contain 1.6, 1.4, and 1.2% total lysine for P1, P2, and P3, respectively

[b]Provided the following per kg feed: Zn, 120 mg; Fe, 120 mg; Mn, 24 mg; Cu, 12 mg; I, .36 mg; Se, .36 mg; vitamin A, 6,615 IU; vitamin D_3, 661 IU; vitamin E, 40 IU; vitamin K (menadione activity), 4.4 mg; riboflavin, 6.6mg; d-pantothenic acid, 30 mg; niacin, 40 mg; vitamin B_{12}, 33 ug; d-biotin, 265 ug; choline, 144 mg; and folic acid, 2 mg.

[c]P1 and P2 contained Neo-terramycin® (100 g/ton oxytetracycline & 140 g/ton neomycin base) and P3 contained Lincomix® (200 g/ton lincomycin)

[d]L-carnitine (Carniking 10, Lonza Inc., Fair Lawn, NJ) substituted at 0.05%, and SBO and CO substituted at 5.0% for cornstarch to obtain the five dietary treatments

Pigs were housed in temperature-controlled nursery rooms and grouped in elevated pens with wire flooring. Each pen provided 1.72 m^2 of space and contained a

five-hole, stainless steel feeder and one nipple waterer that allowed for the *ad libitum* access to feed and water throughout the experiment. Room temperature was maintained initially at 31°C, and decreased by 1.1°C weekly until the room temperature reached 25.5°C. Pigs were weighed at the initiation of the experiment and then pigs weights and feed consumption were measured at the end of each dietary phase for the determination of average daily gain (ADG), average daily feed intake (ADFI), and gain:feed (G:F).

Chemical analysis:

Diets were analyzed for DM according to AOAC (1998) procedures. Gross energy determinations were made by bomb calorimetry (Parr 1261 Isoperibol Calorimeter, Moline, IL), and nitrogen determinations were performed by Kjeldahl methodology (FOSS Tecator, 2400 Kjeltec Analyzer unit, 2020 Digestor, Hoganas, Sweden). As well, diets were analyzed for L-carnitine concentrations using methods described by Parvin and Pande (1977). Chemical composition of the control diets is shown in Table 5.2. The L-carnitine concentration for the five dietary treatments for each phase is detailed in Table 5.3.

Table 5.2. Chemical composition of control diets (as-is basis).

Item	Phase 1	Phase 2	Phase 3
Calculated analysis			
ME, kcal/kg	3,155	3,164	3,193
Crude protein, %	22.58	21.70	21.19
Total lysine, %	1.60	1.40	1.20
Digestible lysine, %	1.36	1.18	1.00
Digestible threonine, %	0.80	0.70	0.61
Digestible Met + Cys, %	0.80	0.69	0.56
Digestible tryptophan, %	0.25	0.23	0.21
Calcium, %	0.90	0.90	0.90
Phosphorus, %	0.80	0.80	0.80
Available phosphorus, %	0.62	0.56	0.51
Analyzed values			
GE, kcal/kg	3,979	3,972	4,003
Crude protein, %	21.09	20.57	20.70

Table 5.3. L-Carnitine concentration of diets[a].

Item:	Calculated Concentration	Analyzed Concentration	Supplemented Level[b]
Phase 1		L-carnitine, ppm	
Diet 1	0	37	0
Diet 2	0	37	0
Diet 3	50	83	46
Diet 4	0	37	0
Diet 5	50	83	46
Phase 2			
Diet 1	0	19	0
Diet 2	0	19	0
Diet 3	50	56	37
Diet 4	0	19	0
Diet 5	50	63	44
Phase 3			
Diet 1	0	1	0
Diet 2	0	1	0
Diet 3	50	29	28
Diet 4	0	1	0
Diet 5	50	28	27

[a]Analysis reported on an as-is basis
[b]Supplemented level obtained by subtracting analyzed concentration from analyzed concentration of unsupplemented diets (diet 1)

Statistical analysis:

The data were analyzed as a randomized complete block design within each experiment using analysis of variance procedures (Steel et al., 1997). There were no treatment by experiment interactions as trends were similar within experiments. Thus, data were pooled across experiments and analyzed as a 2 x 2 factorial, with a negative control, in a randomized complete block design. Treatment 1 contained 8 reps/trt and Treatments 2 through 5 contained 10 reps/trt. The model included the effects of block (rep), treatment, and block x treatment (error). Orthogonal contrasts were used to test the effects of control vs addition of a dietary fat source, L-carnitine level (0 vs 50 ppm),

source of dietary fat (SBO vs CO), and the L-carnitine level x added dietary fat source interaction. Pen served as the experimental unit.

Results

The chemical analyses of the five dietary treatments are presented in Tables 5.2 and 5.3. For Phase 1, supplemented levels of L-carnitine were consistent with calculated levels, signifying proper diet mixing. However, supplemented levels of L-carnitine for Phase 2 and 3 diets were considerably lower than calculated levels. Reasons for the discrepancy between the calculated and supplemented L-carnitine levels are unknown. The addition of five-percent soybean oil and coconut oil to the diet increased the caloric density to approximately 4,157-4,240 kcal/kg.

The effects of L-carnitine and source of dietary fat on growth performance are presented in Table 5.4. The addition of L-carnitine and either soybean oil or coconut oil had little affect on overall performance. Neither ADG nor ADFI ($P > 0.20$) were affected by L-carnitine or source of dietary fat; however, pigs fed a diet containing added fat had greater G:F ($P < 0.04$) when compared with pigs fed the control diet. Additionally, when L-carnitine was supplemented to the diet an improvement in G:F was noted in pigs fed CO, while pigs fed SBO tended to have decreased G:F ratios (L-carnitine x fat source, $P < 0.06$).

The greatest response attributed to the supplementation of a fat source to the diet was observed in Phase 1. The inclusion of a dietary fat source increased ADG ($P < 0.08$) and ADFI ($P < 0.01$) when compared with performance criteria of pigs fed the control diet. However, the addition of L-carnitine to the diet increased ADG and ADFI in pigs fed diets containing SBO, while pigs fed diets containing CO had decreased ADG and

ADFI due to the addition of L-carnitine (L-carnitine x fat source, $P < 0.03$). Additionally, pigs fed SBO consumed more feed than pigs fed CO (SBO vs CO effect, $P < 0.04$).

The addition of a dietary fat source also elicited a response in growth performance during Phase 3. Pigs fed a diet containing added fat tended to eat less ($P = 0.14$) feed, resulting in improved feed efficiency ($P < 0.01$) when compared with pigs fed the control diet. As well, when L-carnitine was added to the diet, pigs fed CO had increased G:F, while pigs fed SBO had decreased G:F (L-carnitine x SBO & CO interaction, $P < 0.01$). Feed efficiency was also affected by source of dietary fat, as pigs fed diets containing CO had greater G:F ($P < 0.04$) than pigs fed diets containing SBO.

Neither the addition of L-carnitine nor the supplementation of a fat source enhanced performance criteria during Phase 2. No differences in ADG, ADFI, and G:F ($P > 0.18$) were noted due to alterations in dietary treatments.

Results from this study suggest that the addition of soybean oil or coconut oil, to increase caloric density of the diet, can enhance growth performance of weanling pigs. The greatest response in growth performance, attributed to the inclusion of soybean oil or coconut oil in the diet, was observed immediately post-weaning (Phase 1). However, minimal responses associated with the supplementation of L-carnitine were observed, with the responses being varied among phases and source of dietary fat.

Table 5.4. Growth performance of weanling pigs.

Carnitine, ppm	0	0	50	0	50				
Fat Source, %	0	5 SBO	5 SBO	5 CO	5 CO		P <:[c]		
Treatment	1[a]	2[b]	3[b]	4[b]	5[b]	SE	L-carnitine	Fat Source[d]	Interaction[e]
Phase 1[g]									
ADG, g[f]	105	117	146	128	115	13.92	--	--	0.04
ADFI, g[f]	135	159	179	164	142	11.58	--	0.06	0.01
G:F	0.78	0.73	0.82	0.78	0.80	0.05	0.18	--	--
Phase 2[h]									
ADG, g	366	347	346	356	361	15.20	--	--	--
ADFI, g	467	441	443	453	444	17.91	--	--	--
G:F	0.79	0.79	0.78	0.79	0.82	0.02	--	--	0.20
Phase 3[j]									
ADG, g	489	495	480	487	501	22.19	--	--	--
ADFI, g	774	738	738	739	718	29.18	--	--	--
G:F[f]	0.63	0.67	0.65	0.66	0.70	0.02	--	0.05	0.01
Overall[k]									
ADG, g	334	330	335	337	338	12.77	--	--	--
ADFI, g	479	460	471	471	452	17.15	--	--	--
G:F[f]	0.70	0.72	0.71	0.72	0.75	0.01	--	0.09	0.08

[a]Least squares means for eight pens/trt of four to five pigs/pen
[b]Least squares means for ten pens/trt of four to five pigs/pen
[c]Dashes indicate P > 0.20
[d]SBO vs CO effect
[e]L-carnitine x fat source interaction
[f]SBO and CO vs negative control (P < 0.10)
[g]Phase 1 = d 0-13 in Exp. 1; d 0-10 in Exp. 2
[h]Phase 2 = d 14-27 in Exp. 1; d 11-24 in Exp. 2
[j]Phase 3 = d 28-41 in Exp. 1; d 25-38 in Exp. 2
[k]Overall = d 0-41 in Exp. 1; d 0-38 in Exp. 2

Discussion

The addition of either soybean oil or coconut oil increased ADG and ADFI during Phase 1. In contrast to the responses reported during Phase 1, results from Experiment 3 suggest that performance criteria are not affected by the addition of a fat source during Phase 1. Cho et al. (1999b) also reported that ADG and ADFI were not affected by the addition of either soybean oil or coconut oil to the diet. The results from Exp. 3 and Cho et al. (1999b) are in agreement with findings by Tokach et al. (1995). These authors reported that weanling pigs require an adjustment period to utilize fat, with no improvements in growth performance, due to added fat, being observed from d 0 to 14 post-weaning. However, we would assume that the increases in ADG and ADFI of pigs fed diets containing coconut oil, observed during Phase 1 of this study, could be attributed to the improved utilization of the medium-chain fatty acids provided in the coconut oil.

The addition of L-carnitine to the diet improved ADG and ADFI in pigs fed SBO during Phase 1. Given the role of L-carnitine in fatty acid metabolism, we would hypothesize that an increase in the oxidation of long-chain fatty acids found in the soybean oil occurred, resulting in the improved performance criteria. Increases in feed intake and daily gain during Phase 1 (0 to 2 wk after weaning), due to the addition of 1,000 ppm L-carnitine in diets containing 5% SBO, were also reported by Weeden et al. (1991). However, studies by Owen et al. (1996; 2001) and Weeden et al. (1990) did not report any improvements in performance criteria that were attributed to the supplementation of L-carnitine to diets containing added fat sources immediately post-weaning.

Results from Experiment 1 and Experiment 3 suggest that the greatest response associated with L-carnitine was observed during Phase 2 (d 10-24). The fact that minimal responses were observed during Phase 2 and 3 in this study could be attributed to the insufficient levels of supplemented L-carnitine in the diets. Supplemented levels of L-carnitine were 37 and 44 ppm for Diets 3 and 5, respectively, in Phase 2. In Exp. 1, slight numeric increases in performance criteria were noted in pigs fed 25 ppm L-carnitine during Phase 2. Additionally, in Exp. 4, supplemented levels of L-carnitine were substantially lower than calculated levels, as Diets 3 and 5 contained 28 and 27 ppm, respectively. These findings would suggest that marginal levels of L-carnitine were available for the utilization of the long-chain fatty acids provided in the form of soybean oil.

Overall performance indicates that pigs fed diets containing added fat sources have improved feed efficiency. Although not significant, numeric increases in ADG and decreases in ADFI were observed resulting in the improved feed efficiency. Lawrence and Maxwell (1983) also reported that added dietary fat improved feed efficiency, while not affecting ADG of weaned pigs.

Minimal differences in performance criteria were observed between pigs fed coconut oil and soybean oil. Pigs fed coconut oil consumed less feed during Phase 1 and had greater feed efficiency during Phase 3. As well, overall feed efficiency was improved in pigs fed coconut oil when compared with pigs fed soybean oil. These results are in agreement with findings by Cera et al. (1989). These authors reported that pigs fed coconut oil (medium-chain fatty acids) had improved feed efficiency when compared with pigs fed diets containing added fat sources that were comprised primarily of long-

chain fatty acids. However, Cho et al. (1999b) reported that the supplementation of coconut oil to the diet improved ADG, while having no affect on ADFI or feed efficiency of weanling pigs.

Results from this study suggest that the addition of soybean oil or coconut oil can enhance growth performance of weanling pigs. The greatest response in performance criteria, associated with a supplemental fat source, was observed immediately post-weaning (Phase 1). However, minimal responses associated with the addition of L-carnitine to the diet were observed, with the responses being varied among phases and source of dietary fat.

Implications

Weanling pig diets are supplemented with fat sources, to increase the caloric density of the diet, in an effort to improve growth performance, thereby diminishing the effects of post-weaning lag. However, the efficiency with which weanling pigs utilize supplemental fat varies between source of dietary fat. Research has shown that weanling pigs are able to more efficiently utilize sources of dietary fat that are comprised primarily of medium-chain fatty acids. Results from this study suggest that diets containing added fat sources enhanced performance criteria of weanling pigs. As well, slight improvements in growth performance of weanling pigs fed coconut oil (medium-chain fatty acids) when compared with pigs fed diets containing soybean oil (long-chain fatty acids) were observed. However, because of questionable supplemented levels of L-carnitine, the effects of L-carnitine on different fat sources are uncertain.

Chapter VI

Summary and Conclusions

A common practice in today's swine industry is weaning pigs at 21 days of age or younger. Ironically, the gastrointestinal tract is immaturely developed and enzyme activity is limited at this early age of the pig, resulting in inefficient utilization of added dietary plant protein sources and fat sources. The inefficient utilization of these dietary ingredients is one of the major causes of a dilemma known as post-weaning lag. Another factor contributing to the effects of post-weaning lag is that during the first few days after weaning, appreciable quantities of feed can not be consumed to meet the young pig's energy demand for growth.

In an effort to diminish the negative effects attributed to post-weaning lag, nutritionists are developing complex, nutrient-dense diets, containing ingredients that are highly digestible and appropriate for the pig's stage of physiological development. One area that nutritionists are evaluating in the feeding regime of weanling pigs is the improved utilization of supplemental fat sources. On average, fat sources provide 2.25 times more energy than protein or carbohydrate sources. Therefore, during periods of minimal feed consumption, if an improvement in the utilization of dietary fat sources can be obtained, an enhancement in the performance of weanling pigs is conceivable.

One possible method to improving dietary fat utilization is by supplementing L-carnitine, a cosubstrate in lipid metabolism, to the diet of weanling pigs. The primary metabolic role of L-carnitine is to facilitate the transfer of long-chain fatty acids across

the otherwise impermeable mitochondrial membrane into the matrix of the mitochondria for the production of adenosine triphosphate (energy) via beta-oxidation and oxidative phosphorylation.

Thus, the objectives of this thesis were to evaluate the effects of supplementing L-carnitine to the diet of weanling pigs and its subsequent effects on growth performance, nutrient digestibility, and whole body composition.

In Exp. 1, improvements in growth performance were observed in pigs fed increasing levels of L-carnitine. Although no changes were noted in daily feed intake for the 38-d study, increases in daily gain were observed due to the addition of L-carnitine, resulting in improved feed efficiencies in weanling pigs. Pigs fed 50 ppm L-carnitine had the highest ADG and best G:F for d 0-38. The improvement in ADG and G:F, associated with L-carnitine, was greatest during Phase 2. Again, the best response to supplemental L-carnitine was noted in pigs fed 50 ppm. Results from Exp. 1 indicate that the addition of L-carnitine improved growth performance in weanling pigs, with the most pronounced response to supplemental L-carnitine observed in pigs fed 50 ppm.

Growth performance trends observed in Exp. 2 were similar to those reported in Exp. 1. Weanling pigs fed dietary L-carnitine had improved ADG from d 0-38, with the greatest response being observed in pigs fed 50 ppm L-carnitine. Additionally, in Exp. 2, pigs fed increasing levels of L-carnitine had increased fecal GE and urine GE excretion; however, increasing trends in DE and ME (kcal/d) were still observed, due to greater increases in GE intake. We would assume that the slight improvements in nutrient digestibility observed in pigs fed supplemental levels of L-carnitine resulted in the improvements in growth performance of weanling pigs.

Trends for nitrogen balance were similar to those reported for energy digestibility. The addition of increasing levels of L-carnitine to the diet increased fecal nitrogen excretion of weanling pigs. However, larger increases in nitrogen intake were noted in pigs fed 25 and 50 ppm L-carnitine resulting in the percentages of nitrogen absorbed and nitrogen retained being greater in these two groups when compared with pigs fed diets containing 0 and 100 ppm added L-carnitine.

Improvements in whole body composition were also observed in Exp. 2. In general, the response to L-carnitine tended to plateau at 50 ppm. Increasing concentrations of L-carnitine resulted in increases in the percentage of protein and decreases in the percentage of lipid in weanling pigs. Additionally, increasing levels of L-carnitine enhanced the daily rate of protein accretion. Although, no affects on the daily rate of lipid accretion were observed, the ratio of protein accretion to lipid accretion improved with increasing levels of L-carnitine. The increase in the ratio of protein accretion to lipid accretion indicates a repartitioning of nutrients away from lipid deposition and towards the accretion of protein in weanling pigs.

Results from Exp. 1 and Exp. 2 indicate that supplementing 50 ppm L-carnitine to diets containing added fat improves growth performance in weanling pigs. The next question we tried to answer was whether the addition of fat sources to the diet of weanling pigs might be required before an improvement in growth performance attributed to supplemental L-carnitine is observed.

Results from Exp. 3 suggest that the addition of 50 ppm L-carnitine improved growth performance in weanling pigs; however, supplemental L-carnitine was more effective in improving performance criteria when soybean oil was included in the diet.

The addition of L-carnitine did improve G:F for the 38-d study. However, the increase in G:F associated with L-carnitine was more pronounced in pigs fed soybean oil than those not fed soybean oil. Similar to Exp. 1, the greatest response to L-carnitine occurred in Phase 2 with an increase in ADG and G:F. Given the role of L-carnitine in lipid metabolism, we would assume that in pigs fed diets without soybean oil, adequate levels of L-carnitine were synthesized for long-chain fatty acid oxidation and supplemental L-carnitine was not required. However, when soybean oil was added to the diet and long-chain fatty acid concentrations were increased, supplemental levels of L-carnitine were needed to utilize the additional long-chain fatty acids.

The addition of L-carnitine and either soybean oil or coconut oil to the diet of weanling pigs had little affect on overall performance in Exp. 4. However, we would attribute this to insufficient levels of L-carnitine supplementation in the diets. The reasons for the discrepancy between supplemented levels and formulated levels of L-carnitine in Exp. 4 are unknown. In Exp. 4, the greatest response associated with the supplementation of a fat source to the diet was observed in Phase 1. The inclusion of a dietary fat source increased ADG and ADFI when compared with performance criteria of pigs fed the control diet. Additionally, as would be expected, the addition of L-carnitine to the diet increased ADG and ADFI in pigs fed diets containing soybean oil (long-chain fatty acids) while pigs fed diets containing coconut oil (medium-chain fatty acids) had decreased ADG and ADFI due to the addition of L-carnitine. The response in performance criteria observed during Phase 1 is in contrast to results from Exp. 1 and Exp. 3, in which both experiments reported the greatest improvements, associated with the addition of L-carnitine during Phase 2.

The topic of this thesis focused on the supplementation of L-carnitine to the diet of weanling pigs and its subsequent effects on growth performance, nutrient digestibility, and whole body composition. In summarizing the data from all four experiments, we would believe that beneficial effects are conceivable due to the addition of L-carnitine to the diet of weanling pigs. Data indicates positive responses attributed to the supplementation of L-carnitine when soybean oil or other fat sources are provided in the diet as a means to increase caloric density; thereby, meeting the energy requirements of weanling pigs and improving growth performance. Additionally, data indicates improvements in body composition as a repartitioning of nutrients from lipid deposition towards protein accretion occurred. The improvements in body composition offer an incentive to producers, by allowing the producer to merit greater rewards in today's industry in which the majority of the animals are marketed on a carcass based system. However, more research is needed to better understand the mode of action of L-carnitine in lipid metabolism and its subsequent effects on performance criteria.

Chapter VII

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APPENDIX

Pen Means and Analysis of Variance Tables

Appendix Table 1

Pen means for average daily gain, average daily feed intake, and gain:feed for Phases 1 and 2 – Experiment 1.

			Phase 1			Phase 2		
Pen	Trt	Rep	ADG (g)	ADFI (g)	G:F	ADG (g)	ADFI (g)	G:F
2	4	1	116	163	.715	409	498	.822
3	2	1	80	153	.524	372	492	.756
4	1	2	139	176	.789	320	439	.728
5	4	2	142	159	.891	343	449	.764
6	1	3	125	174	.718	266	373	.714
7	2	3	115	136	.843	261	350	.747
9	1	1	117	184	.635	360	483	.746
10	3	1	191	210	.909	429	520	.825
11	2	2	169	188	.899	373	477	.783
12	3	2	173	179	.964	344	445	.772
13	4	3	222	225	.986	293	408	.719
14	3	3	133	154	.862	346	434	.799
22	1	4	135	160	.844	366	506	.724
23	2	4	188	206	.912	441	558	.789
24	4	5	159	179	.886	418	510	.819
25	3	5	194	197	.984	371	494	.752
26	4	6	151	174	.867	367	482	.761
27	1	6	159	187	.850	357	504	.708
29	4	4	163	168	.968	434	513	.847
30	3	4	183	194	.941	455	585	.777
31	1	5	144	159	.906	380	495	.767
32	2	5	----	----	----	----	----	----
33	3	6	117	174	.674	340	457	.744
34	2	6	114	148	.775	324	442	.732

Trt 1: Control diet
Trt 2: Control diet + 25 ppm L-carnitine
Trt 3: Control diet + 50 ppm L-carnitine
Trt 4: Control diet + 100 ppm L-carnitine

Dashes indicate data for this variable not included in analysis due to poor pen performance.

Appendix Table 2

Analysis of variance for average daily gain, average daily feed intake, and gain:feed for Phases 1 and 2 – Experiment 1.

		Mean Squares					
		Phase 1			Phase 2		
Source	d.f.	ADG	ADFI	G:F	ADG	ADFI	G:F
Total	22						
Error	14	1099.9	621.3	.009	729.0	757.7	.001
Repetition	5	949.8	63.3	.028	8581.6	10245.5	.002
Treatment	3	1291.5	331.0	.016	1972.6	611.0	.004
Linear	1	2231.8	229.8	.035	4053.2	179.0	.009
Quadratic	1	493.1	54.8	.005	1588.9	711.1	.002
Cubic	1	1155.8	713.4	.007	179.4	586.4	.000
C.V., %		22.26	14.2	11.05	7.4	5.8	3.81

Appendix Table 3

Pen means for average daily gain, average daily feed intake, and gain:feed for Phase 3 and the entire 38-d period – Experiment 1.

			Phase 3			Overall		
Pen	Trt	Rep	ADG (g)	ADFI (g)	G:F	ADG (g)	ADFI (g)	G:F
2	4	1	557	868	.642	386	546	.708
3	2	1	479	793	.604	335	513	.652
4	1	2	457	795	.575	323	501	.645
5	4	2	529	821	.645	359	510	.703
6	1	3	456	758	.602	291	448	.649
7	2	3	441	630	.700	289	397	.728
9	1	1	479	785	.610	340	516	.659
10	3	1	595	900	.661	428	578	.739
11	2	2	538	822	.654	380	528	.719
12	3	2	470	715	.658	345	474	.728
13	4	3	483	768	.629	345	492	.700
14	3	3	480	749	.641	325	449	.725
22	1	4	493	773	.638	352	513	.686
23	2	4	524	871	.601	405	581	.697
24	4	5	496	858	.578	378	551	.686
25	3	5	528	849	.621	382	547	.699
26	4	6	412	775	.532	326	509	.641
27	1	6	504	780	.646	359	522	.688
29	4	4	489	802	.610	383	528	.725
30	3	4	547	900	.608	417	598	.697
31	1	5	484	795	.609	356	517	.688
32	2	5	----	----	----	----	----	----
33	3	6	447	632	.707	321	447	.717
34	2	6	468	757	.618	311	463	.671

Trt 1: Control diet
Trt 2: Control diet + 25 ppm L-carnitine
Trt 3: Control diet + 50 ppm L-carnitine
Trt 4: Control diet + 100 ppm L-carnitine

Dashes indicate data for this variable not included in analysis due to poor pen performance.

Appendix Table 4

Analysis of variance for average daily gain, average daily feed intake, and gain:feed for Phase 3 and the entire 38-d period – Experiment 1.

		Mean Squares					
		Phase 3			Overall		
Source	d.f.	ADG	ADFI	G:F	ADG	ADFI	G:F
Total	22						
Error	14	1487.9	3768.7	.002	703.7	1186.4	.001
Repetition	5	2969.9	10011.0	.001	3285.0	6405.8	.001
Treatment	3	1050.7	1465.0	.002	1312.4	585.5	.002
Linear	1	744.2	4166.0	.001	2356.5	1598.0	.002
Quadratic	1	2004.5	271.9	.006	1140.0	.08	.005
Cubic	1	298.2	11.2	.001	376.7	207.5	.001
C.V., %		7.81	7.76	6.45	7.50	6.75	3.90

Appendix Table 5

Pen means for initial and final body weight, average daily gain, average daily feed intake, and gain:feed for the entire 38-d period – Experiment 2.

					Overall		
Pen	Trt	Rep	Initial Wt. (kg)	Final Wt. (kg)	ADG (g)	ADFI (g)	G:F
1	2	1	6.67	17.28	279	352	.793
2	3	1	7.26	--	--	--	--
3	4	1	5.44	16.69	296	383	.772
4	1	1	5.31	15.51	269	350	.766
5	4	2	5.12	16.83	308	411	.749
6	2	2	4.49	18.10	358	438	.818
7	1	2	5.22	17.41	321	436	.737
8	3	2	3.76	--	--	--	--
9	1	3	4.22	12.52	218	275	.796
10	4	3	4.31	17.82	356	415	.857
11	3	3	3.76	17.46	360	449	.803
12	2	3	4.63	17.01	326	413	.788
13	2	4	5.26	17.10	312	369	.843
14	4	4	5.44	--	--	--	--
15	3	4	5.76	17.87	319	396	.804
16	1	4	5.35	14.10	230	308	.749
17	2	5	5.49	16.19	282	366	.770
18	1	5	4.26	13.24	236	327	.724
19	4	5	3.90	16.78	339	374	.906
20	3	5	3.13	13.83	282	305	.925
21	4	6	4.72	--	--	--	--
22	2	6	3.17	13.42	270	338	.799
23	1	6	4.67	14.33	254	347	.732
24	3	6	4.94	20.27	403	499	.808

Trt 1: Control diet
Trt 2: Control diet + 25 ppm L-carnitine
Trt 3: Control diet + 50 ppm L-carnitine
Trt 4: Control diet + 100 ppm L-carnitine

Dashes indicate data for this variable not included in analysis due to poor pig performance.

Appendix Table 6

Analysis of variance for average daily gain, average daily feed intake, and gain:feed for the entire 38-d period – Experiment 2.

		Mean Squares		
		Overall		
Source	d.f.	ADG	ADFI	G:F
Total	19			
Error	11	1454.9	2323.3	0.00243
Repetition	5	1428.8	3707.2	0.00139
Treatment	3	7493.3	5767.2	0.00598
Linear	1	9480.8	6064.9	0.00896
Quadratic	1	9433.1	8455.5	0.00578
Cubic	1	331.4	838.9	0.00003
C.V., %		12.68	12.77	6.19

Appendix Table 7

Pen means for average daily feed intake, gross energy intake, fecal energy excretion, and digestible energy (kcal/d, kcal/kg) – Experiment 2 (Period 1).

			Energy Balance				
Pen	Trt	Rep	ADFI (g)	GE (kcal/d)	FE (kcal/d)	DE (kcal/d)	DE (kcal/kg)
1	2	1	61.99	281.66	20.73	260.93	4209.3
2	3	1	--	--	--	--	--
3	4	1	111.68	503.35	52.86	450.49	4033.6
4	1	1	137.34	620.70	52.90	567.81	4134.4
5	4	2	196.91	887.43	70.38	817.05	4143.5
6	2	2	235.82	1071.51	116.62	954.89	4049.3
7	1	2	197.45	892.40	57.97	834.43	4226.0
8	3	2	--	--	--	--	--
9	1	3	--	--	--	--	--
10	4	3	176.66	796.20	63.99	732.21	4144.7
11	3	3	244.75	1101.02	118.83	982.19	4013.0
12	2	3	204.07	927.23	64.38	862.85	4228.3
13	2	4	206.48	938.21	95.44	842.77	4081.6
14	4	4	--	--	--	--	--
15	3	4	185.58	834.85	43.40	791.44	4264.7
16	1	4	--	--	--	--	--
17	2	5	--	--	--	--	--
18	1	5	19.19	86.72	6.82	79.90	4164.2
19	4	5	145.40	655.29	58.77	596.51	4102.7
20	3	5	54.13	243.49	44.97	198.52	3667.7
21	4	6	--	--	--	--	--
22	2	6	91.91	417.63	32.20	385.43	4193.4
23	1	6	83.04	375.33	18.52	356.81	4296.6
24	3	6	243.08	1093.49	105.37	988.12	4065.0

Trt 1: Control diet
Trt 2: Control diet + 25 ppm L-carnitine
Trt 3: Control diet + 50 ppm L-carnitine
Trt 4: Control diet + 100 ppm L-carnitine

Dashes indicate data for this variable not included in analysis due to poor pig performance.

Appendix Table 8

Analysis of variance for average daily feed intake, gross energy intake, fecal energy excretion, and digestible energy (kcal/d, kcal/kg) – Experiment 2 (Period 1).

		Mean Squares				
Source	d.f.	ADFI	GE	FE	DE (kcal/d)	DE (kcal/kg)
Total	16					
Error	8	3017.2	61699.4	1044.3	48966.9	16393.9
Repetition	5	9103.8	185902.9	1010.1	160950.2	20826.3
Treatment	3	2344.3	45682.3	1133.2	33576.6	37951.0
Linear	1	2734.9	53135.7	747.4	41276.6	13507.5
Quadratic	1	2823.6	57354.7	2582.5	35596.0	94323.2
Cubic	1	1626.0	29559.6	130.7	25759.8	9412.3
C.V., %		35.98	36.01	53.64	35.15	3.11

Appendix Table 9

Pen means for fecal excretion and urine excretion, urinary energy and metabolizable energy (kcal/d, kcal/kg) – Experiment 2 (Period 1).

			Energy Balance				
Pen	Trt	Rep	Feces (g)	Urine (g)	UE (kcal/d)	ME (kcal/d)	ME (kcal/kg)
1	2	1	4.07	0.81	2.71	258.22	4165.6
2	3	1	--	--	--	--	--
3	4	1	11.20	1.15	3.32	447.17	4003.9
4	1	1	12.79	1.05	2.13	565.68	4118.9
5	4	2	16.37	1.52	3.89	813.16	4129.7
6	2	2	28.22	1.43	1.88	953.01	4041.3
7	1	2	13.86	1.63	4.26	830.17	4204.4
8	3	2	--	--	--	--	--
9	1	3	--	--	--	--	--
10	4	3	14.15	1.13	2.36	729.85	4131.3
11	3	3	28.93	1.74	2.76	979.43	4001.7
12	2	3	14.77	2.74	6.69	856.16	4195.5
13	2	4	23.00	1.25	3.18	839.59	4066.2
14	4	4	--	--	--	--	--
15	3	4	10.50	2.22	5.51	785.93	4234.9
16	1	4	--	--	--	--	--
17	2	5	--	--	--	--	--
18	1	5	0.97	0.85	1.76	78.14	4072.6
19	4	5	12.81	2.27	5.45	591.07	4065.2
20	3	5	10.40	0.61	1.52	197.00	3639.6
21	4	6	--	--	--	--	--
22	2	6	6.50	0.89	2.13	383.30	4170.2
23	1	6	4.05	1.17	3.21	353.60	4257.9
24	3	6	23.72	2.65	5.34	982.78	4043.1

Trt 1: Control diet
Trt 2: Control diet + 25 ppm L-carnitine
Trt 3: Control diet + 50 ppm L-carnitine
Trt 4: Control diet + 100 ppm L-carnitine

Dashes indicate data for this variable not included in analysis due to poor pig performance.

Appendix Table 10

Analysis of variance for fecal excretion and urine excretion, urinary energy and metabolizable energy (kcal/d, kcal/kg) – Experiment 2 (Period 1).

		Mean Squares				
Source	d.f.	Feces	Urine	UE (kcal/d)	ME (kcal/d)	ME (kcal/kg)
Total	16					
Error	8	62.101	0.6052	3.8969	48549.9	13766.0
Repetition	5	66.919	0.2162	0.8976	160429.2	25247.8
Treatment	3	63.169	0.1434	0.6739	33379.9	32348.2
Linear	1	26.377	0.2165	1.8754	40723.4	8955.2
Quadratic	1	156.150	0.1021	0.0320	35662.8	82778.0
Cubic	1	11.756	0.1150	0.0703	25674.1	8377.6
C.V., %		56.68	52.67	57.76	35.19	2.87

Appendix Table 11

Pen means for energy ratios – Experiment 2 (Period 1).

			Energy Balance		
Pen	Trt	Rep	DE:GE (%)	ME:DE (%)	ME:GE (%)
1	2	1	92.64	98.96	91.68
2	3	1	--	--	--
3	4	1	89.50	99.26	88.84
4	1	1	91.48	99.63	91.13
5	4	2	92.07	99.52	91.63
6	2	2	89.12	99.80	88.94
7	1	2	93.50	99.49	93.03
8	3	2	--	--	--
9	1	3	--	--	--
10	4	3	91.96	99.68	91.67
11	3	3	89.21	99.72	88.96
12	2	3	93.06	99.22	92.34
13	2	4	89.83	99.62	89.49
14	4	4	--	--	--
15	3	4	94.80	99.30	94.14
16	1	4	--	--	--
17	2	5	--	--	--
18	1	5	92.14	97.80	90.11
19	4	5	91.03	99.09	90.20
20	3	5	81.53	99.23	80.91
21	4	6	--	--	--
22	2	6	92.29	99.45	91.78
23	1	6	95.07	99.10	94.21
24	3	6	90.36	99.46	89.88

Trt 1: Control diet
Trt 2: Control diet + 25 ppm L-carnitine
Trt 3: Control diet + 50 ppm L-carnitine
Trt 4: Control diet + 100 ppm L-carnitine

Dashes indicate data for this variable not included in analysis due to poor pig performance.

Appendix Table 12

Analysis of variance for energy ratios – Experiment 2 (Period 1).

		Mean Squares		
Source	d.f.	DE:GE	ME:DE	ME:GE
Total	16			
Error	8	8.0633	0.17391	6.7778
Repetition	5	10.2777	0.29636	12.4517
Treatment	3	16.6529	0.14167	14.1929
Linear	1	3.5725	0.24286	1.9950
Quadratic	1	46.1710	0.17198	40.4497
Cubic	1	0.9222	0.01352	0.7098
C.V., %		3.12	0.42	2.88

Appendix Table 13

Pen means for average daily feed intake, gross energy intake, fecal energy excretion, and digestible energy (kcal/d, kcal/kg) – Experiment 2 (Period 2).

			Energy Balance				
Pen	Trt	Rep	ADFI (g)	GE (kcal/d)	FE (kcal/d)	DE (kcal/d)	DE (kcal/kg)
1	2	1	347.4	1607	159.0	1448	4167
2	3	1	--	--	--	--	--
3	4	1	393.5	1824	214.8	1609	4089
4	1	1	315.5	1473	147.1	1326	4202
5	4	2	329.1	1525	236.5	1289	3916
6	2	2	396.3	1833	194.9	1638	4133
7	1	2	394.1	1840	124.7	1715	4352
8	3	2	--	--	--	--	--
9	1	3	209.0	976	117.6	858	4106
10	4	3	374.7	1737	216.6	1520	4057
11	3	3	397.9	1861	249.2	1611	4049
12	2	3	273.7	1266	139.4	1126	4115
13	2	4	346.7	1603	165.6	1438	4147
14	4	4	--	--	--	--	--
15	3	4	348.5	1629	141.1	1488	4270
16	1	4	327.4	1528	184.3	1344	4106
17	2	5	366.8	1696	201.5	1495	4075
18	1	5	368.6	1721	202.3	1519	4120
19	4	5	451.5	2092	293.2	1799	3985
20	3	5	362.3	1694	241.1	1453	4010
21	4	6	--	--	--	--	--
22	2	6	345.9	1600	184.9	1415	4090
23	1	6	354.0	1652	224.5	1428	4034
24	3	6	494.9	2314	259.4	2054	4151

Trt 1: Control diet
Trt 2: Control diet + 25 ppm L-carnitine
Trt 3: Control diet + 50 ppm L-carnitine
Trt 4: Control diet + 100 ppm L-carnitine

Dashes indicate data for this variable not included in analysis due to poor pig performance.

Appendix Table 14

Analysis of variance for average daily feed intake, gross energy intake, fecal energy excretion, and digestible energy (kcal/d, kcal/kg) – Experiment 2 (Period 2).

		Mean Squares				
Source	d.f.	ADFI	GE	FE	DE (kcal/d)	DE (kcal/kg)
Total	19					
Error	11	2770.29	60157.2	1099.02	53685.5	7878.96
Repetition	5	3903.41	84501.9	2652.62	64663.7	7910.08
Treatment	3	6240.12	137041.6	5970.20	91189.0	17064.63
Linear	1	11092.94	229697.2	16175.87	123970.3	45732.42
Quadratic	1	3521.85	76511.1	20.23	76653.3	7366.45
Cubic	1	2741.42	76271.5	1009.65	59491.4	5338.57
C.V., %		14.62	14.66	17.01	15.67	2.16

Appendix Table 15

Pen means for fecal excretion and urine excretion, urinary energy and metabolizable energy (kcal/d, kcal/kg) – Experiment 2 (Period 2).

			Energy Balance				
Pen	Trt	Rep	Feces (g)	Urine (kg)	UE (kcal/d)	ME (kcal/d)	ME (kcal/kg)
1	2	1	36.01	0.0027	7.76	1440	4145
2	3	1	--	--	--	--	--
3	4	1	45.26	0.0024	7.66	1601	4069
4	1	1	32.92	0.0016	5.61	1320	4185
5	4	2	48.21	0.0021	6.31	1283	3897
6	2	2	45.30	0.0024	5.92	1632	4118
7	1	2	26.15	0.0020	5.86	1709	4337
8	3	2	--	--	--	--	--
9	1	3	24.80	0.0014	4.20	854	4086
10	4	3	49.06	0.0029	7.58	1512	4036
11	3	3	55.09	0.0014	4.85	1606	4037
12	2	3	28.96	0.0018	5.28	1121	4096
13	2	4	36.53	0.0014	3.50	1434	4137
14	4	4	--	--	--	--	--
15	3	4	30.97	0.0018	4.44	1484	4258
16	1	4	41.43	0.0023	5.39	1339	4086
17	2	5	42.61	0.0029	7.23	1487	4055
18	1	5	45.89	0.0019	5.31	1513	4105
19	4	5	66.40	0.0031	7.29	1792	3969
20	3	5	55.32	0.0011	2.68	1450	4002
21	4	6	--	--	--	--	--
22	2	6	40.83	0.0019	5.09	1410	4075
23	1	6	48.75	0.0019	4.66	1423	4021
24	3	6	58.24	0.0021	4.91	2049	4141

Trt 1: Control diet
Trt 2: Control diet + 25 ppm L-carnitine
Trt 3: Control diet + 50 ppm L-carnitine
Trt 4: Control diet + 100 ppm L-carnitine

Dashes indicate data for this variable not included in analysis due to poor pig performance.

Appendix Table 16

Analysis of variance for fecal and urine excretion, urinary energy, and metabolizable energy (kcal/d, kcal/kg) – Experiment 2 (Period 2).

		Mean Squares				
Source	d.f.	Feces	Urine	UE (kcal/d)	ME (kcal/d)	ME (kcal/kg)
Total	19					
Error	11	66.615	2.77e-7	1.2627	53327.7	7850.3
Repetition	5	143.162	6.42e-8	0.8926	64540.5	7776.1
Treatment	3	282.665	7.38e-7	3.8866	91332.9	17843.2
Linear	1	736.420	1.05e-6	5.7094	122532.6	46494.2
Quadratic	1	6.501	5.92e-7	3.8011	77718.6	8787.6
Cubic	1	63.604	1.11e-6	5.1927	60693.9	6576.6
C.V., %		19.01	25.62	20.15	15.68	2.16

Appendix Table 17

Pen means for energy ratios – Experiment 2 (Period 2).

			Energy Balance		
Pen	Trt	Rep	DE:GE (%)	ME:DE (%)	ME:GE (%)
1	2	1	90.1	99.5	89.6
2	3	1	--	--	--
3	4	1	88.2	99.5	87.8
4	1	1	90.0	99.6	89.6
5	4	2	84.5	99.5	84.1
6	2	2	89.4	99.6	89.0
7	1	2	93.2	99.7	92.9
8	3	2	--	--	--
9	1	3	87.9	99.5	87.5
10	4	3	87.5	99.5	87.1
11	3	3	86.6	99.7	86.3
12	2	3	89.0	99.5	88.6
13	2	4	89.7	99.8	89.5
14	4	4	--	--	--
15	3	4	91.3	99.7	91.1
16	1	4	87.9	99.6	87.6
17	2	5	88.1	99.5	87.7
18	1	5	88.2	99.7	87.9
19	4	5	86.0	99.6	85.6
20	3	5	85.8	99.8	85.6
21	4	6	--	--	--
22	2	6	88.4	99.6	88.1
23	1	6	86.4	99.7	86.1
24	3	6	88.8	99.8	88.6

Trt 1: Control diet
Trt 2: Control diet + 25 ppm L-carnitine
Trt 3: Control diet + 50 ppm L-carnitine
Trt 4: Control diet + 100 ppm L-carnitine

Dashes indicate data for this variable not included in analysis due to poor pig performance.

Appendix Table 18

Analysis of variance for energy ratios – Experiment 2 (Period 2).

		Mean Squares		
Source	d.f.	DE:GE	ME:DE	ME:GE
Total	19			
Error	11	3.59141	0.00664	3.63896
Repetition	5	3.65474	0.00922	3.60330
Treatment	3	5.99290	0.02454	6.08855
Linear	1	16.29025	0.00781	16.45825
Quadratic	1	3.11969	0.03048	3.55290
Cubic	1	0.02040	0.05490	0.00080
C.V., %		2.14	0.08	2.17

Appendix Table 19

Pen means for average daily feed intake, gross energy intake, fecal energy excretion, and digestible energy (kcal/d, kcal/kg) – Experiment 2 (Period 3).

			Energy Balance				
Pen	Trt	Rep	ADFI (g)	GE (kcal/d)	FE (kcal/d)	DE (kcal/d)	DE (kcal/kg)
1	2	1	516.6	2415	200.4	2215	4287
2	3	1	--	--	--	--	--
3	4	1	596.5	2813	240.3	2572	4312
4	1	1	480.8	2260	194.1	2066	4297
5	4	2	640.7	3021	385.0	2636	4114
6	2	2	594.6	2780	258.6	2521	4240
7	1	2	663.7	3119	343.1	2776	4183
8	3	2	--	--	--	--	--
9	1	3	401.3	1886	257.7	1629	4058
10	4	3	474.8	2239	290.2	1948	4104
11	3	3	544.1	2557	267.9	2289	4207
12	2	3	528.2	2469	238.4	2231	4224
13	2	4	443.3	2073	295.0	1778	4010
14	4	4	--	--	--	--	--
15	3	4	496.1	2332	231.8	2100	4232
16	1	4	489.1	2299	278.6	2020	4131
17	2	5	464.3	2171	237.6	1933	4163
18	1	5	467.6	2198	202.8	1995	4267
19	4	5	589.5	2779	282.4	2497	4236
20	3	5	491.9	2312	235.3	2076	4221
21	4	6	--	--	--	--	--
22	2	6	535.3	2502	290.8	2212	4132
23	1	6	505.9	2378	327.1	2051	4054
24	3	6	545.9	2565	337.4	2228	4081

Trt 1: Control diet
Trt 2: Control diet + 25 ppm L-carnitine
Trt 3: Control diet + 50 ppm L-carnitine
Trt 4: Control diet + 100 ppm L-carnitine

Dashes indicate data for this variable not included in analysis due to poor pig performance.

Appendix Table 20

Analysis of variance for average daily feed intake, gross energy intake, fecal energy excretion, and digestible energy (kcal/d, kcal/kg) – Experiment 2 (Period 3).

		Mean Squares				
Source	d.f.	ADFI	GE	FE	DE (kcal/d)	DE (kcal/kg)
Total	19					
Error	11	2043.20	45047.9	772.01	44447.7	4629.8
Repetition	5	9578.18	211169.1	7079.55	175489.8	19099.9
Treatment	3	4109.76	98266.1	3002.77	74576.1	658.3
Linear	1	10864.27	260634.4	6877.80	182380.2	323.7
Quadratic	1	214.27	2787.5	2792.58	11142.1	8881.8
Cubic	1	532.43	16336.1	76.44	14088.9	3251.7
C.V., %		8.63	8.63	10.30	9.63	1.63

Appendix Table 21

Pen means for fecal and urine excretion, urinary energy and metabolizable energy (kcal/d, kcal/kg) – Experiment 2 (Period 3).

			Energy Balance				
Pen	Trt	Rep	Feces (g)	Urine (kg)	UE (kcal/d)	ME (kcal/d)	ME (kcal/kg)
1	2	1	46.45	0.0060	18.52	2196	4251
2	3	1	--	--	--	--	--
3	4	1	55.10	0.0083	24.42	2548	4271
4	1	1	45.11	0.0054	15.82	2050	4264
5	4	2	86.72	0.0114	28.72	2607	4069
6	2	2	60.14	0.0080	21.94	2499	4203
7	1	2	75.17	0.0101	26.01	2750	4144
8	3	2	--	--	--	--	--
9	1	3	55.72	0.0051	11.28	1617	4030
10	4	3	63.20	0.0084	21.44	1927	4059
11	3	3	57.49	0.0060	14.76	2275	4180
12	2	3	51.54	0.0102	24.26	2207	4178
13	2	4	62.98	0.0078	21.51	1756	3961
14	4	4	--	--	--	--	--
15	3	4	51.64	0.0048	11.74	2088	4209
16	1	4	61.55	0.0045	11.66	2009	4107
17	2	5	51.72	0.0070	17.93	1915	4125
18	1	5	44.68	0.0051	13.88	1981	4237
19	4	5	61.45	0.0055	14.09	2483	4212
20	3	5	53.12	0.0063	16.90	2060	4187
21	4	6	--	--	--	--	--
22	2	6	63.36	0.0050	12.95	2199	4108
23	1	6	71.82	0.0058	12.86	2038	4028
24	3	6	74.02	0.0074	18.10	2210	4048

Trt 1: Control diet
Trt 2: Control diet + 25 ppm L-carnitine
Trt 3: Control diet + 50 ppm L-carnitine
Trt 4: Control diet + 100 ppm L-carnitine

Dashes indicate data for this variable not included in analysis due to poor pig performance.

Appendix Table 22

Analysis of variance for fecal and urine excretion, urinary energy and metabolizable energy (kcal/d, kcal/kg) – Experiment 2 (Period 3).

		Mean Squares				
Source	d.f.	Feces	Urine	UE (kcal/d)	ME (kcal/d)	ME (kcal/kg)
Total	19					
Error	11	29.9113	0.000003	15.8151	44469.1	5253.3
Repetition	5	330.6480	0.000006	38.8956	170933.9	18579.1
Treatment	3	145.6074	0.000003	29.7156	72979.0	3807.0
Linear	1	343.1027	6.715e-6	49.3362	176808.8	626.4
Quadratic	1	115.1079	1.088e-8	0.9703	11005.7	8725.1
Cubic	1	7.4505	3.295e-6	31.8676	15610.8	5086.5
C.V., %		9.17	23.12	22.17	9.71	1.75

Appendix Table 23

Pen means for energy ratios – Experiment 2 (Period 3).

			Energy Balance		
			DE:GE	ME:DE	ME:GE
Pen	Trt	Rep	(%)	(%)	(%)
1	2	1	91.7	99.2	90.9
2	3	1	--	--	--
3	4	1	91.5	99.1	90.6
4	1	1	91.4	99.2	90.7
5	4	2	87.3	98.9	86.3
6	2	2	90.7	99.1	89.9
7	1	2	89.0	99.1	88.2
8	3	2	--	--	--
9	1	3	86.3	99.3	85.7
10	4	3	87.0	98.9	86.1
11	3	3	89.5	99.4	88.9
12	2	3	90.3	98.9	89.4
13	2	4	85.8	98.9	84.7
14	4	4	--	--	--
15	3	4	90.1	99.4	89.6
16	1	4	87.9	99.4	87.4
17	2	5	89.1	99.1	88.2
18	1	5	90.8	99.3	90.1
19	4	5	89.8	99.4	89.3
20	3	5	89.8	99.2	89.1
21	4	6	--	--	--
22	2	6	88.4	99.4	87.9
23	1	6	86.2	99.4	85.7
24	3	6	86.8	99.2	86.1

Trt 1: Control diet
Trt 2: Control diet + 25 ppm L-carnitine
Trt 3: Control diet + 50 ppm L-carnitine
Trt 4: Control diet + 100 ppm L-carnitine

Dashes indicate data for this variable not included in analysis due to poor pig performance.

Appendix Table 24

Analysis of variance for energy ratios – Experiment 2 (Period 3).

		Mean Squares		
Source	d.f.	DE:GE	ME:DE	ME:GE
Total	19			
Error	11	2.10171	0.03538	2.43588
Repetition	5	8.74890	0.02298	8.33872
Treatment	3	2.37871	0.05608	2.37565
Linear	1	1.12268	0.03370	1.44409
Quadratic	1	6.68087	0.00001	6.43074
Cubic	1	0.21811	0.12873	0.62869
C.V., %		1.6295	0.1897	1.7687

Appendix Table 25

Analysis of variance for average daily feed intake, gross energy intake, fecal energy excretion, and digestible energy (kcal/d, kcal/kg) – Experiment 2 (Periods 2 & 3 pooled).

		Mean Squares				
Source	d.f.	ADFI	GE	FE	DE (kcal/d)	DE (kcal/kg)
Total	47					
Error	20	1981.4	42971.18	1591.93	36786.04	5560.71
Repetition	5	12539.3	224933.95	7831.72	186928.2	24899.28
Treatment	3	12658.9	142095.39	11112.58	85683.5	29292.17
Linear	1	30459.0	352640.24	30616.75	175277.2	64926.86
Quadratic	1	3981.7	42372.83	1699.12	60892.76	18398.54
Cubic	1	3535.8	31273.11	1021.85	20880.64	4551.09
Rep x Trt	15	2466.1	72525.67	825.29	68826.57	7046.65
Period	1	324262.6	7188912.0	67432.52	5863212	60634.08
Trt x Period	3	663.0	20715.17	1360.01	13153.06	8737.25
C.V., %		9.89	10.04	16.80	10.50	1.80

Appendix Table 26

Analysis of variance for urinary energy and metabolizable energy (kcal/d, kcal/kg) – Experiment 2 (Periods 2 & 3 pooled).

		Mean Squares		
Source	d.f.	UE (kcal/d)	ME (kcal/d)	ME (kcal/kg)
Total	47			
Error	20	11.61916	36080.592	5846.7250
Repetition	5	31.83295	183299.200	23219.1833
Treatment	3	33.73480	84434.944	32231.7222
Linear	1	61.46394	169121.877	70150.4341
Quadratic	1	0.68271	61341.325	19662.7236
Cubic	1	39.05782	22841.687	6882.0015
Rep x Trt	15	5.58420	68645.278	7327.8389
Period	1	1920.77603	5655387.000	32448.0000
Trt x Period	3	9.40098	13105.056	8123.8333
C.V., %		28.5674	10.4655	1.8619

Appendix Table 27

Analysis of variance for energy ratios – Experiment 2 (Periods 2 & 3 pooled).

		Mean Squares		
Source	d.f.	DE:GE	ME:DE	ME:GE
Total	47			
Error	20	2.5141250	0.01920833	2.6894167
Repetition	5	11.41770833	0.03120833	10.49483333
Treatment	3	14.00743056	0.08743056	14.94500000
Linear	1	30.86147444	0.09300599	33.37751656
Quadratic	1	10.89587595	0.01000537	11.44000327
Cubic	1	0.26491198	0.15928048	0.01745330
Rep x Trt	15	3.20759722	0.01343056	3.38116667
Period	1	5.13520833	2.47520833	0.65333333
Trt x Period	3	2.24909722	0.01520833	1.96944444
C.V., %		1.793707	0.139445	1.866578

Appendix Table 28

Pen means for average daily feed intake, nitrogen intake, nitrogen excretion, and nitrogen absorbed (g/d, %) – Experiment 2 (Period 1).

			Energy Balance				
Pen	Trt	Rep	ADFI (g)	N Intake (g/d)	N Exc. (g/d)	N Abs. (g/d)	N Abs. (%)
1	2	1	61.99	2.37	0.196	2.17	91.74
2	3	1	--	--	--	--	--
3	4	1	111.68	4.30	0.650	3.65	84.88
4	1	1	137.34	4.93	0.674	4.25	86.33
5	4	2	196.91	7.59	0.989	6.60	86.96
6	2	2	235.82	9.00	1.595	7.41	82.28
7	1	2	197.45	7.08	0.793	6.29	88.80
8	3	2	--	--	--	--	--
9	1	3	--	--	--	--	--
10	4	3	176.66	6.81	0.862	5.94	87.33
11	3	3	244.75	9.28	1.544	7.74	83.37
12	2	3	204.07	7.79	0.895	6.89	88.51
13	2	4	206.48	7.88	1.147	6.73	85.45
14	4	4	--	--	--	--	--
15	3	4	185.58	7.04	0.526	6.51	92.53
16	1	4	--	--	--	--	--
17	2	5	--	--	--	--	--
18	1	5	--	--	--	--	--
19	4	5	145.40	5.60	0.732	4.87	86.93
20	3	5	54.13	2.05	0.558	1.49	72.80
21	4	6	--	--	--	--	--
22	2	6	91.91	3.51	0.303	3.21	91.37
23	1	6	83.04	2.98	0.226	2.75	92.41
24	3	6	243.08	9.22	1.370	7.85	85.14

Trt 1: Control diet
Trt 2: Control diet + 25 ppm L-carnitine
Trt 3: Control diet + 50 ppm L-carnitine
Trt 4: Control diet + 100 ppm L-carnitine

Dashes indicate data for this variable not included in analysis due to poor pig performance.

Appendix Table 29

Analysis of variance for average daily feed intake, nitrogen intake, fecal nitrogen excretion, and nitrogen absorbed (g/d, %) – Experiment 2 (Period 1).

		Mean Squares				
Source	d.f.	ADFI	N Intake	N Exc.	N Abs. (g/d)	N Abs. (%)
Total	15					
Error	7	3299.171	4.71755	0.206194	3.200873	22.4702
Repetition	5	7149.507	10.24887	0.204860	7.821428	25.7174
Treatment	3	1069.822	2.06750	0.124266	1.276718	20.5855
Linear	1	1017.240	2.61205	0.066354	1.848954	0.1387
Quadratic	1	1484.278	2.74735	0.314450	1.214448	51.7959
Cubic	1	1665.147	1.66155	0.039620	1.187149	12.7071
C.V., %		35.67	35.67	55.63	33.94	5.47

Appendix Table 30

Pen means for urinary nitrogen excretion, nitrogen retention (g/d, %), and nitrogen retention:nitrogen absorption – Experiment 2 (Period 1).

			Energy Balance			
Pen	Trt	Rep	Urine N Exc (g/d)	N Ret. (g/d)	N Ret. (%)	N Ret:N Abs (%)
1	2	1	0.184	1.99	83.95	91.51
2	3	1	--	--	--	--
3	4	1	0.260	3.39	78.85	92.89
4	1	1	0.232	4.02	81.61	94.54
5	4	2	0.438	6.16	81.19	93.36
6	2	2	0.330	7.08	78.62	95.54
7	1	2	0.328	5.96	84.17	94.78
8	3	2	--	--	--	--
9	1	3	--	--	--	--
10	4	3	0.276	5.67	83.28	95.36
11	3	3	0.384	7.36	79.24	95.04
12	2	3	0.414	6.48	83.20	93.99
13	2	4	0.248	6.49	82.31	96.32
14	4	4	--	--	--	--
15	3	4	0.383	6.13	87.09	94.12
16	1	4	--	--	--	--
17	2	5	--	--	--	--
18	1	5	--	--	--	--
19	4	5	0.410	4.46	79.60	91.57
20	3	5	0.183	1.31	63.88	87.74
21	4	6	--	--	--	--
22	2	6	0.277	2.93	83.47	91.35
23	1	6	0.243	2.51	84.26	91.17
24	3	6	0.338	7.51	81.48	95.70

Trt 1: Control diet
Trt 2: Control diet + 25 ppm L-carnitine
Trt 3: Control diet + 50 ppm L-carnitine
Trt 4: Control diet + 100 ppm L-carnitine

Dashes indicate data for this variable not included in analysis due to poor pig performance.

Appendix Table 31

Analysis of variance for urinary nitrogen excretion, nitrogen retention (g/d, %), and nitrogen retention:nitrogen absorption – Experiment 2 (Period 1).

		Mean Squares			
Source	d.f.	Urine N Exc	N Ret. (g/d)	N Ret. (%)	N Ret:N Abs
Total	19				
Error	7	0.0071557	3.0274998	17.813187	4.2828844
Repetition	5	0.0075144	7.3905126	41.086207	9.3008511
Treatment	3	0.0036114	1.1731837	17.842942	0.3371752
Linear	1	0.0098518	1.5814456	0.004768	0.3318555
Quadratic	1	0.0000111	1.2038421	47.654266	0.1939701
Cubic	1	0.0005421	1.1341880	6.896344	0.3089093
C.V., %		27.4648	35.0403	5.2098	2.2149

Appendix Table 32

Pen means for average daily feed intake, nitrogen intake, nitrogen excretion, and nitrogen absorbed (g/d, %) – Experiment 2 (Period 2).

			Energy Balance				
Pen	Trt	Rep	ADFI (g)	N Intake (g/d)	N Exc. (g/d)	N Abs. (g/d)	N Abs. (%)
1	2	1	347.44	12.70	1.302	11.40	89.75
2	3	1	--	--	--	--	--
3	4	1	393.48	14.73	1.816	12.92	87.67
4	1	1	315.55	11.31	1.543	9.77	86.36
5	4	2	329.13	12.32	2.413	9.91	80.42
6	2	2	396.31	14.49	2.092	12.40	85.56
7	1	2	394.06	14.13	1.211	12.92	91.43
8	3	2	--	--	--	--	--
9	1	3	209.00	7.49	1.024	6.47	86.33
10	4	3	374.72	14.03	2.090	11.94	85.10
11	3	3	397.94	14.98	2.678	12.30	82.12
12	2	3	273.70	10.1	1.268	8.74	87.33
13	2	4	346.65	12.67	1.744	10.93	86.24
14	4	4	--	--	--	--	--
15	3	4	348.48	13.12	1.363	11.75	89.61
16	1	4	327.39	11.74	1.855	9.88	84.20
17	2	5	366.76	13.41	1.799	11.61	86.58
18	1	5	368.64	13.22	1.947	11.27	85.27
19	4	5	451.46	16.90	3.036	13.87	82.04
20	3	5	362.31	13.64	2.296	11.34	83.16
21	4	6	--	--	--	--	--
22	2	6	345.89	12.65	1.801	10.85	85.76
23	1	6	353.96	12.69	2.181	10.51	82.81
24	3	6	494.88	18.63	2.527	16.10	86.43

Trt 1: Control diet
Trt 2: Control diet + 25 ppm L-carnitine
Trt 3: Control diet + 50 ppm L-carnitine
Trt 4: Control diet + 100 ppm L-carnitine

Dashes indicate data for this variable not included in analysis due to poor pig performance.

Appendix Table 33

Analysis of variance for average daily feed intake, nitrogen intake, fecal nitrogen excretion, and nitrogen absorbed (g/d, %) – Experiment 2 (Period 2).

		Mean Squares				
Source	d.f.	ADFI	N Intake	N Exc.	N Abs. (g/d)	N Abs. (%)
Total	19					
Error	11	2769.61	3.75588	0.188740	3.17451	8.34016
Repetition	5	3901.30	5.19663	0.262846	6.39758	5.91384
Treatment	3	6240.67	12.62648	0.625533	8.08727	8.33295
Linear	1	11089.10	22.58692	1.625442	12.12182	19.20842
Quadratic	1	3524.64	7.37502	0.004268	6.98922	6.22649
Cubic	1	2744.85	4.99054	0.166693	3.30950	0.79715
C.V., %		14.62	14.63	22.87	15.71	3.37

Appendix Table 34

Pen means for urinary nitrogen excretion, nitrogen retention (g/d, %), and nitrogen retention:nitrogen absorption – Experiment 2 (Period 2).

			Energy Balance			
Pen	Trt	Rep	Urine N Exc (g/d)	N Ret. (g/d)	N Ret. (%)	N Ret:N Abs (%)
1	2	1	0.869	10.53	82.91	92.38
2	3	1	--	--	--	--
3	4	1	1.012	11.90	80.80	92.16
4	1	1	0.562	9.21	81.39	94.25
5	4	2	0.553	9.36	75.93	94.42
6	2	2	0.449	11.95	82.46	96.37
7	1	2	0.803	12.11	85.74	93.78
8	3	2	--	--	--	--
9	1	3	0.619	5.85	78.06	90.43
10	4	3	0.814	11.13	79.30	93.18
11	3	3	0.604	11.70	78.08	95.09
12	2	3	1.163	7.58	75.71	86.70
13	2	4	0.556	10.37	81.85	94.92
14	4	4	--	--	--	--
15	3	4	1.029	10.72	81.76	91.25
16	1	4	0.513	9.37	79.83	94.81
17	2	5	0.867	10.74	80.12	92.53
18	1	5	0.630	10.64	80.50	94.41
19	4	5	0.672	13.19	78.06	95.15
20	3	5	0.249	11.09	81.34	97.81
21	4	6	--	--	--	--
22	2	6	0.468	10.38	82.06	95.69
23	1	6	0.454	10.05	79.23	95.68
24	3	6	0.626	15.47	83.07	96.11

Trt 1: Control diet
Trt 2: Control diet + 25 ppm L-carnitine
Trt 3: Control diet + 50 ppm L-carnitine
Trt 4: Control diet + 100 ppm L-carnitine

Dashes indicate data for this variable not included in analysis due to poor pig performance.

Appendix Table 35

Analysis of variance for urinary nitrogen excretion, nitrogen retention (g/d, %), and nitrogen retention:nitrogen absorption – Experiment 2 (Period 2).

		Mean Squares			
Source	d.f.	Urine N Exc	N Ret. (g/d)	N Ret. (%)	N Ret:N Abs
Total	19				
Error	11	0.06227587	2.9512749	5.1412910	5.4085301
Repetition	5	0.04263087	4.3557886	8.3336231	9.5797521
Treatment	3	0.02424547	7.8091254	6.2702579	3.2644008
Linear	1	0.02665422	10.9979684	11.0434841	0.8591274
Quadratic	1	0.00047930	6.8868228	9.8595261	1.0017741
Cubic	1	0.04021843	4.0842080	2.6070408	8.3881556
C.V., %		36.9377	16.1051	2.8199	2.4779

Appendix Table 36

Pen means for average daily feed intake, nitrogen intake, nitrogen excretion, and nitrogen absorbed (g/d, %) – Experiment 2 (Period 3).

			Energy Balance				
Pen	Trt	Rep	ADFI (g)	N Intake (g/d)	N Exc. (g/d)	N Abs. (g/d)	N Abs. (%)
1	2	1	516.63	18.87	1.626	17.24	91.38
2	3	1	--	--	--	--	--
3	4	1	596.53	21.93	2.146	19.78	90.21
4	1	1	480.79	17.00	1.689	15.31	90.06
5	4	2	640.70	23.55	3.975	19.58	83.13
6	2	2	594.56	21.71	2.477	19.24	88.59
7	1	2	663.68	23.47	3.211	20.26	86.32
8	3	2	--	--	--	--	--
9	1	3	401.34	14.19	2.649	11.54	81.33
10	4	3	474.77	17.45	2.623	14.83	84.97
11	3	3	544.15	19.72	2.505	17.21	87.29
12	2	3	528.19	19.29	2.070	17.22	89.27
13	2	4	443.35	16.19	2.604	13.59	83.91
14	4	4	--	--	--	--	--
15	3	4	496.12	17.98	1.899	16.08	89.44
16	1	4	489.11	17.29	2.662	14.63	84.61
17	2	5	464.32	16.96	2.170	14.79	87.20
18	1	5	467.55	16.53	1.722	14.81	89.58
19	4	5	589.47	21.67	2.669	19.00	87.68
20	3	5	491.91	17.83	2.197	15.63	87.67
21	4	6	--	--	--	--	--
22	2	6	535.27	19.55	2.883	16.66	85.25
23	1	6	505.93	17.89	2.979	14.91	83.34
24	3	6	545.88	19.78	3.205	16.58	83.80

Trt 1: Control diet
Trt 2: Control diet + 25 ppm L-carnitine
Trt 3: Control diet + 50 ppm L-carnitine
Trt 4: Control diet + 100 ppm L-carnitine

Dashes indicate data for this variable not included in analysis due to poor pig performance.

Appendix Table 37

Analysis of variance for average daily feed intake, nitrogen intake, fecal nitrogen excretion, and nitrogen absorbed (g/d, %) – Experiment 2 (Period 3).

		Mean Squares				
Source	d.f.	ADFI	N Intake	N Exc.	N Abs. (g/d)	N Abs. (%)
Total	19					
Error	11	2042.86	2.61872	0.111001	2.74060	4.93388
Repetition	5	9574.68	12.41514	0.919561	9.42713	17.75539
Treatment	3	4109.85	8.32781	0.381012	6.39429	6.72829
Linear	1	10862.63	22.24671	0.819288	14.53537	1.47225
Quadratic	1	215.02	0.77975	0.407980	2.30856	18.63566
Cubic	1	533.07	0.06195	0.010355	0.02132	0.00671
C.V., %		8.63	8.54	13.33	10.07	2.56

Appendix Table 38

Pen means for urinary nitrogen excretion, nitrogen retention (g/d, %), and nitrogen retention:nitrogen absorption – Experiment 2 (Period 3).

			Energy Balance			
Pen	Trt	Rep	Urine N Exc (g/d)	N Ret. (g/d)	N Ret. (%)	N Ret:N Abs (%)
1	2	1	2.024	15.22	80.65	88.26
2	3	1	--	--	--	--
3	4	1	3.092	16.69	76.11	84.37
4	1	1	2.169	13.14	77.30	85.83
5	4	2	3.646	15.93	67.65	81.38
6	2	2	1.472	17.76	81.81	92.35
7	1	2	3.474	16.78	71.51	82.85
8	3	2	--	--	--	--
9	1	3	1.402	10.14	71.45	87.85
10	4	3	2.081	12.75	73.05	85.97
11	3	3	1.170	16.04	81.36	93.20
12	2	3	2.888	14.33	74.30	83.23
13	2	4	2.220	11.37	70.20	83.66
14	4	4	--	--	--	--
15	3	4	1.999	14.08	78.32	87.57
16	1	4	1.007	13.63	78.79	93.12
17	2	5	1.493	13.29	78.39	89.90
18	1	5	1.555	13.25	80.18	89.50
19	4	5	1.693	17.31	79.87	91.09
20	3	5	1.363	14.27	80.03	91.28
21	4	6	--	--	--	--
22	2	6	1.538	15.13	77.39	90.77
23	1	6	0.819	14.09	78.77	94.51
24	3	6	1.023	15.55	78.63	93.83

Trt 1: Control diet
Trt 2: Control diet + 25 ppm L-carnitine
Trt 3: Control diet + 50 ppm L-carnitine
Trt 4: Control diet + 100 ppm L-carnitine

Dashes indicate data for this variable not included in analysis due to poor pig performance.

Appendix Table 39

Analysis of variance for urinary nitrogen excretion, nitrogen retention (g/d, %), and nitrogen retention:nitrogen absorption – Experiment 2 (Period 3).

		Mean Squares			
Source	d.f.	Urine N Exc	N Ret. (g/d)	N Ret. (%)	N Ret:N Abs
Total	19				
Error	11	0.4737388	2.6384712	15.2436638	14.2726274
Repetition	5	0.8115952	6.2998800	16.9991731	16.9963580
Treatment	3	0.4453463	4.7309944	19.2550357	10.0823716
Linear	1	1.0269068	7.8546035	14.4098922	10.5466547
Quadratic	1	0.2630902	4.1132153	47.1762759	12.2372729
Cubic	1	0.2982970	0.4759013	11.1181538	16.0821840
C.V., %		36.1040	11.1734	5.0845	4.2676

Appendix Table 40

Analysis of variance for average daily feed intake, nitrogen intake, fecal nitrogen excretion, and nitrogen absorbed (g/d, %) – Experiment 2 (Periods 2 & 3 pooled).

		Mean Squares				
Source	d.f.	ADFI	N Intake	N Exc.	N Abs. (g/d)	N Abs. (%)
Total	39					
Error	11	1451.03	1.97984	0.125901	1.73588	4.86464
Repetition	5	9992.95	13.02800	0.803465	9.98655	18.27750
Treatment	3	9942.29	19.94557	0.880406	13.91662	12.43114
Rep x Trt	11	3361.44	4.39476	0.173840	4.17923	8.40940
Period	1	252215.4	303.6708	3.333320	243.3554	11.47248
Trt x Period	3	408.24	1.00872	0.126138	0.56494	2.63001
Rep x Period	5	3483.03	4.58378	0.378942	3.13815	5.39173
C.V., %		8.62	8.74	16.14	9.48	2.56

Appendix Table 41

Analysis of variance for urinary nitrogen excretion, nitrogen retention (g/d, %), and nitrogen retention:nitrogen absorption – Experiment 2 (Periods 2 & 3 pooled).

		Mean Squares			
Source	d.f.	Urine N Exc	N Ret. (g/d)	N Ret. (%)	N Ret:N Abs
Total	39				
Error	11	0.17425809	1.5050746	10.3229579	5.3383311
Repetition	5	0.47893303	9.1737610	15.8339978	20.1162786
Treatment	3	0.31390648	12.1636433	23.0772463	9.8247394
Rep x Trt	11	0.36175662	4.0846715	10.0619669	14.3428264
Period	1	15.26884413	136.782096	133.417206	291.499885
Trt x Period	3	0.15568530	0.3764766	2.4480472	3.5220330
Rep x Period	5	0.37529299	1.4819076	9.4987983	6.4598315
C.V., %		32.3348	9.7349	4.0878	2.5337

Appendix Table 42

Pen means for initial and final body weight, and average daily gain – Experiment 2.

Pen	Trt	Rep	Initial BW (g)	Final BW (g)	Overall ADG (g/d)
1	2	1	6674	17297	279.6
2	3	1	--	--	--
3	4	1	5448	16707	296.3
4	1	1	5312	15527	268.8
5	4	2	5130	16843	308.2
6	2	2	4495	18115	358.4
7	1	2	5221	17434	321.4
8	3	2	--	--	--
9	1	3	4222	12530	218.6
10	4	3	4313	17842	356.0
11	3	3	3768	17479	360.8
12	2	3	4631	17025	326.2
13	2	4	5266	17116	311.8
14	4	4	--	--	--
15	3	4	5766	17888	319.0
16	1	4	5357	14119	230.6
17	2	5	5493	16208	282.0
18	1	5	4268	13257	236.6
19	4	5	3904	16798	339.3
20	3	5	3133	13847	282.0
21	4	6	--	--	--
22	2	6	3178	13438	270.0
23	1	6	4676	14346	254.5
24	3	6	4949	20294	403.8
Initial Pigs					
1		1		6219.8	
2		2		4630.8	
3		3		3995.2	
4		4		5084.8	
5		5		4403.8	
6		6		4040.6	

Trt 1: Control diet
Trt 2: Control diet + 25 ppm L-carnitine
Trt 3: Control diet + 50 ppm L-carnitine
Trt 4: Control diet + 100 ppm L-carnitine

Dashes indicate data for this variable not included in analysis due to poor pig performance.

Appendix Table 43

Analysis of variance for initial and final body weight, and average daily gain – Experiment 2.

Source	d.f.	Mean Squares		
		Initial BW	Final BW	ADG
Total	19			
Error	11	586242.24	3245427.23	1457.19941
Repetition	5	1487548.456	2999245.56	1429.33564
Treatment	3	77879.787	9978482.94	7504.09357
Linear	1	81078.153	11686237.94	9487.767060
Quadratic	1	2031.917	13313927.10	9453.303860
Cubic	1	141315.036	100112.76	331.999049
C.V., %		16.085	11.116	12.674

Appendix Table 44

Pen means for percentage of protein, lipid, ash, and water – Experiment 2.

Pen	Trt	Rep	Protein (%)	Lipid (%)	Ash (%)	Water (%)
1	2	1	13.73	7.03	2.70	77.72
2	3	1	--	--	--	--
3	4	1	13.85	6.77	2.75	78.79
4	1	1	13.73	8.59	2.59	76.48
5	4	2	13.92	7.18	2.59	78.19
6	2	2	14.24	7.71	2.83	77.13
7	1	2	13.51	8.47	2.62	76.14
8	3	2	--	--	--	--
9	1	3	12.65	8.66	2.79	76.71
10	4	3	14.05	6.99	2.91	77.75
11	3	3	13.88	7.39	2.80	77.09
12	2	3	14.34	8.13	3.08	75.64
13	2	4	13.58	7.38	2.77	77.47
14	4	4	--	--	--	--
15	3	4	14.24	7.84	2.72	76.31
16	1	4	13.38	8.85	2.54	75.98
17	2	5	14.27	7.50	3.01	76.14
18	1	5	13.28	8.11	2.83	76.33
19	4	5	14.23	6.66	2.96	77.83
20	3	5	13.44	5.85	2.94	79.08
21	4	6	--	--	--	--
22	2	6	13.91	6.79	2.89	78.22
23	1	6	13.99	7.39	2.86	76.89
24	3	6	14.41	7.44	2.93	76.47
Initial Pigs						
1		1	13.76	11.45	2.91	68.73
2		2	14.42	6.78	2.47	73.46
3		3	14.11	6.75	2.80	74.22
4		4	13.22	13.12	2.60	68.68
5		5	14.44	8.42	2.69	71.86
6		6	13.83	13.82	2.57	67.13

Trt 1: Control diet
Trt 2: Control diet + 25 ppm L-carnitine
Trt 3: Control diet + 50 ppm L-carnitine
Trt 4: Control diet + 100 ppm L-carnitine

Dashes indicate data for this variable not included in analysis due to poor pig performance.

Appendix Table 45

Analysis of variance for percentage of protein, lipid, ash, and water – Experiment 2.

Source	d.f.	Mean Squares			
		Protein	Lipid	Ash	Water
Total	19				
Error	11	0.1631758	0.2402041	0.0053729	0.8142193
Repetition	5	0.0776066	0.4734476	0.0515196	0.3976160
Treatment	3	0.5002998	2.0393655	0.0306771	2.2602100
Linear	1	0.6535578	4.1246908	0.0051294	6.2916513
Quadratic	1	0.3604240	0.9037437	0.0221852	0.0548495
Cubic	1	0.1665366	0.1367051	0.0424234	0.0650133
C.V., %		2.9205	6.5031	2.6127	1.1701

Appendix Table 46

Pen means for rates of protein, lipid, ash, water, and energy accretion – Experiment 2.

Pen	Trt	Rep	Protein Acc., g/d	Lipid Acc., g/d	Ash Acc., g/d	Water Gain, g/d	Energy Gain, kcal/d
1	2	1	38.12	14.73	7.24	227.29	314.68
2	3	1	--	--	--	--	--
3	4	1	40.96	17.14	8.57	239.94	355.28
4	1	1	36.66	22.95	7.19	208.39	399.37
5	4	2	42.88	20.26	8.26	245.70	420.20
6	2	2	51.20	27.17	10.65	278.43	534.52
7	1	2	42.86	26.99	8.69	246.82	498.51
8	3	2	--	--	--	--	--
9	1	3	25.93	19.78	6.45	168.89	326.41
10	4	3	49.91	23.75	10.90	279.24	495.99
11	3	3	49.53	26.44	10.19	279.42	537.17
12	2	3	47.13	26.45	10.89	247.10	504.15
13	2	4	41.87	21.22	9.13	245.63	416.32
14	4	4	--	--	--	--	--
15	3	4	45.99	23.13	8.96	247.34	438.57
16	1	4	30.11	20.56	6.02	177.43	329.04
17	2	5	40.78	19.21	9.30	217.50	399.70
18	1	5	30.40	19.37	7.10	181.35	363.92
19	4	5	48.12	21.52	10.41	266.18	481.84
20	3	5	36.59	15.26	7.99	226.01	368.33
21	4	6	--	--	--	--	--
22	2	6	36.69	17.86	7.49	213.50	382.90
23	1	6	35.52	17.77	7.88	197.64	359.63
24	3	6	58.76	28.77	12.54	310.75	612.97

Trt 1: Control diet
Trt 2: Control diet + 25 ppm L-carnitine
Trt 3: Control diet + 50 ppm L-carnitine
Trt 4: Control diet + 100 ppm L-carnitine

Dashes indicate data for this variable not included in analysis due to poor pig performance.

Appendix Table 47

Analysis of variance for rates of protein, lipid, ash, water, and energy accretion – Experiment 2.

Source	d.f.	Mean Squares				
		Protein	Lipid	Ash	Water	Energy
Total	19					
Error	11	42.87573	16.25834	2.26458	742.5279	5698.210
Repetition	5	33.50832	24.53071	1.64028	779.5655	7606.781
Treatment	3	209.2437	7.22267	7.14625	5164.634	9840.869
Linear	1	275.3044	0.58610	9.47025	7238.268	6630.070
Quadratic	1	248.4180	14.46934	7.73646	5793.975	18062.44
Cubic	1	1.2167	12.18360	0.14284	136.2903	2969.365
C.V., %		15.7780	18.7399	17.1152	11.5843	17.679

Pen means for albumin levels – Experiment 2.

Pen	Trt	Rep	Albumin levels, g/dL				
			D 0	D 3	D 10	D 24	D 38
1	2	1	2.7	3.2	2.3	2.1	2.2
2	3	1	2.9	2.8	2.8	--	--
3	4	1	2.9	3.0	2.3	2.2	2.3
4	1	1	2.8	2.9	2.5	2.1	2.4
5	4	2	3.2	3.1	2.7	2.7	3.0
6	2	2	3.0	3.2	2.7	2.7	2.8
7	1	2	3.1	3.1	2.6	2.6	2.8
8	3	2	3.0	2.9	2.8	--	--
9	1	3	2.4	2.7	2.6	2.3	2.2
10	4	3	2.0	2.3	2.1	2.1	2.4
11	3	3	2.4	2.2	2.3	2.6	2.5
12	2	3	2.6	2.9	2.3	2.3	2.6
13	2	4	2.1	2.4	2.2	2.3	2.4
14	4	4	2.0	2.5	2.6	--	--
15	3	4	2.3	2.6	2.2	2.4	2.6
16	1	4	2.5	3.1	2.8	2.2	2.4
17	2	5	2.9	3.0	2.6	2.1	2.2
18	1	5	2.8	3.2	2.7	2.4	2.9
19	4	5	1.7	1.8	1.9	2.3	2.3
20	3	5	1.9	2.0	1.9	2.3	2.3
21	4	6	2.7	2.7	2.9	--	--
22	2	6	1.9	2.2	2.0	2.3	2.2
23	1	6	3.1	2.9	2.6	2.1	2.2
24	3	6	2.7	2.6	2.4	2.3	2.7

Trt 1: Control diet
Trt 2: Control diet + 25 ppm L-carnitine
Trt 3: Control diet + 50 ppm L-carnitine
Trt 4: Control diet + 100 ppm L-carnitine

Dashes indicate data for this variable not included in analysis due to poor pig performance.

Appendix Table 49

Analysis of variance for albumin levels – Experiment 2.

Source	d.f.	Mean Squares					
		D 0	D 3	D 10	d.f.	D 24	D 38
Total	23				19		
Error	15	0.1233	0.1088	0.0864	8	0.0143	0.0502
Repetition	5	0.4387	0.2374	0.0880	5	0.1077	0.1288
Treatment	3	0.1433	0.2871	0.0944	3	0.0259	0.0278
Linear	1	0.3360	0.5921	0.0762	1	0.0006	0.0009
Quadratic	1	0.0503	0.2091	0.1431	1	0.0549	0.0111
Cubic	1	0.0410	0.0601	0.6402	1	0.0352	0.0790
C.V., %		13.6827	12.1203	12.0006		5.1526	9.0670

Appendix Table 50

Pen means for blood urea nitrogen levels – Experiment 2.

			Blood urea nitrogen levels, mg/dL				
Pen	Trt	Rep	D 0	D 3	D 10	D 24	D 38
1	2	1	3.8	18.1	9.4	8.6	12.7
2	3	1	--	--	--	--	--
3	4	1	3.7	30.4	10.7	7.3	14.3
4	1	1	4.8	13.4	8.6	7.8	12.6
5	4	2	12.6	12.1	7.2	7.6	9.2
6	2	2	15.3	12.3	5.3	6.9	12.9
7	1	2	10.0	18.1	7.5	10.5	12.2
8	3	2	--	--	--	--	--
9	1	3	5.2	21.5	41.1	14.0	12.1
10	4	3	5.1	23.2	8.1	11.4	12.9
11	3	3	4.6	11.9	5.6	7.7	8.7
12	2	3	7.0	21.2	6.7	12.7	14.0
13	2	4	3.5	17.8	6.9	10.8	16.7
14	4	4	--	--	--	--	--
15	3	4	5.3	17.8	8.0	9.1	7.6
16	1	4	8.4	42.0	19.9	8.9	10.7
17	2	5	13.0	13.3	4.1	10.5	13.2
18	1	5	11.2	26.6	20.4	8.9	15.0
19	4	5	3.5	8.7	7.1	5.9	12.4
20	3	5	3.1	19.2	8.7	12.2	11.8
21	4	6	--	--	--	--	--
22	2	6	6.9	9.7	9.4	11.8	12.3
23	1	6	5.8	17.1	10.1	5.9	10.6
24	3	6	3.6	4.1	4.8	8.2	12.7

Trt 1: Control diet
Trt 2: Control diet + 25 ppm L-carnitine
Trt 3: Control diet + 50 ppm L-carnitine
Trt 4: Control diet + 100 ppm L-carnitine

Dashes indicate data for this variable not included in analysis due to poor pig performance.

Appendix Table 51

Analysis of variance for blood urea nitrogen levels – Experiment 2.

Source	d.f.	Mean Squares				
		D 0	D 3	D 10	D 24	D 38
Total	19					
Error	8	7.07374	54.2664	57.9929	5.30298	4.28296
Repetition	5	27.00693	93.1375	41.5799	6.17177	2.07615
Treatment	3	12.45211	104.5964	175.4154	4.45268	9.73830
Linear	1	15.88734	16.6101	141.3952	8.13577	1.65455
Quadratic	1	2.55383	253.6970	246.1452	1.09304	3.30426
Cubic	1	18.06215	0.0440	20.9310	3.47929	26.03862
C.V., %		38.9978	41.0967	72.6652	24.6687	16.9218

Appendix Table 52

Pen means for C-reactive protein levels – Experiment 2.

			C-reactive protein levels, mg/L				
Pen	Trt	Rep	D 0	D 3	D 10	D 24	D 38
1	2	1	1.10	--	1.78	1.10	4.37
2	3	1	--	--	--	--	--
3	4	1	1.10	1.10	2.87	2.72	2.02
4	1	1	1.76	1.10	1.88	1.62	1.75
5	4	2	1.10	2.10	2.63	1.59	6.71
6	2	2	1.78	4.07	2.52	1.10	3.13
7	1	2	1.50	2.17	3.35	1.10	8.25
8	3	2	--	--	--	--	--
9	1	3	1.10	1.10	2.14	1.10	2.11
10	4	3	2.04	2.22	3.37	3.55	4.07
11	3	3	1.10	1.56	1.58	1.10	3.97
12	2	3	1.56	2.32	1.41	1.62	6.23
13	2	4	1.10	1.46	1.82	2.51	2.17
14	4	4	--	--	--	--	--
15	3	4	2.66	1.58	1.10	--	--
16	1	4	1.10	3.47	4.36	1.10	1.50
17	2	5	1.10	1.10	1.10	1.10	1.10
18	1	5	1.10	1.10	1.10	1.10	1.88
19	4	5	--	3.31	1.70	1.62	4.39
20	3	5	--	1.10	1.10	2.78	1.10
21	4	6	--	--	--	--	--
22	2	6	1.10	1.10	1.10	1.10	1.10
23	1	6	1.10	1.10	2.46	1.10	1.10
24	3	6	1.10	1.84	1.10	1.10	1.93

Trt 1: Control diet
Trt 2: Control diet + 25 ppm L-carnitine
Trt 3: Control diet + 50 ppm L-carnitine
Trt 4: Control diet + 100 ppm L-carnitine

Dashes indicate data for this variable not included in analysis due to poor pig performance.

Appendix Table 53

Analysis of variance for C-reactive protein – Experiment 2.

Source	DF	Mean Squares							
		D 0	DF	D 3	DF	D 10	DF	D 24	D 38
Total	17		18		19		18		
Error	9	0.2798	10	0.9115	11	0.455	10	0.493	3.247
Repetition	5	0.1118	5	0.9417	5	0.948	5	0.301	7.978
Treatment	3	0.0778	3	0.3162	3	1.950	3	1.182	0.657
Linear	1	0.0140	1	0.5488	1	0.300	1	3.544	1.633
Quadratic	1	0.1324	1	0.2609	1	5.537	1	0.013	0.240
Cubic	1	0.1319	1	0.3161	1	0.001	1	0.002	0.276
C.V., %		38.86		51.98		33.35		44.29	58.15

Appendix Table 54

Pen means for glucose levels – Experiment 2.

Pen	Trt	Rep	Glucose levels, mg/dL				
			D 0	D 3	D 10	D 24	D 38
1	2	1	126	103	126	128	125
2	3	1	--	--	--	--	--
3	4	1	187	92	101	98	118
4	1	1	123	87	107	134	131
5	4	2	129	98	131	121	126
6	2	2	112	122	139	168	128
7	1	2	146	94	124	124	128
8	3	2	--	--	--	--	--
9	1	3	178	94	74	127	122
10	4	3	136	73	111	127	133
11	3	3	105	116	125	141	137
12	2	3	161	93	111	151	119
13	2	4	135	82	138	127	126
14	4	4	--	--	--	--	--
15	3	4	132	89	124	184	137
16	1	4	135	94	111	119	133
17	2	5	130	81	117	119	119
18	1	5	142	80	111	123	154
19	4	5	101	106	123	126	104
20	3	5	109	79	105	123	116
21	4	6	--	--	--	--	--
22	2	6	148	113	121	124	113
23	1	6	130	99	104	131	118
24	3	6	110	129	137	133	120

Trt 1: Control diet
Trt 2: Control diet + 25 ppm L-carnitine
Trt 3: Control diet + 50 ppm L-carnitine
Trt 4: Control diet + 100 ppm L-carnitine

Dashes indicate data for this variable not included in analysis due to poor pig performance.

Appendix Table 55

Analysis of variance for glucose levels – Experiment 2.

Source	d.f.	Mean Squares				
		D 0	D 3	D 10	D 24	D 38
Total	19					
Error	8	544.277	185.273	134.050	318.413	121.731
Repetition	5	331.673	357.766	322.674	238.874	89.208
Treatment	3	673.094	170.998	500.290	539.734	148.597
Linear	1	46.177	3.155	193.851	219.098	211.752
Quadratic	1	1600.781	484.464	819.106	1542.472	0.301
Cubic	1	587.663	21.857	128.332	105.700	227.945
C.V., %		17.44	14.15	9.90	13.58	8.80

Appendix Table 56

Pen means for non-esterified fatty acid levels – Experiment 2.

			Non-esterified fatty acid levels, mmol/L				
Pen	Trt	Rep	D 0	D 3	D 10	D 24	D 38
1	2	1	0.33	2.58	0.31	0.07	0.07
2	3	1	--	--	--	--	--
3	4	1	0.51	2.58	0.14	0.08	0.06
4	1	1	0.56	2.86	0.17	0.07	0.05
5	4	2	0.43	2.16	0.09	0.12	0.11
6	2	2	0.43	0.32	0.10	0.09	0.06
7	1	2	0.39	1.89	0.15	0.16	0.06
8	3	2	--	--	--	--	--
9	1	3	0.32	2.02	--	0.11	0.08
10	4	3	0.27	1.78	0.09	0.08	0.06
11	3	3	0.31	0.16	0.07	0.09	0.08
12	2	3	0.25	2.39	0.09	0.13	0.07
13	2	4	0.34	1.40	0.07	0.05	0.05
14	4	4	--	--	--	--	--
15	3	4	0.40	1.91	0.08	0.11	0.06
16	1	4	0.79	1.76	0.38	0.06	0.06
17	2	5	0.51	1.81	0.13	0.17	0.07
18	1	5	0.31	3.11	0.28	0.08	0.08
19	4	5	0.26	0.16	0.08	0.07	0.08
20	3	5	0.32	1.40	0.19	0.24	0.09
21	4	6	--	--	--	--	--
22	2	6	0.51	0.31	0.10	0.09	0.07
23	1	6	0.46	2.93	0.34	0.07	0.06
24	3	6	0.54	0.21	0.09	0.07	0.08

Trt 1: Control diet
Trt 2: Control diet + 25 ppm L-carnitine
Trt 3: Control diet + 50 ppm L-carnitine
Trt 4: Control diet + 100 ppm L-carnitine

Dashes indicate data for this variable not included in analysis due to poor pig performance.

Appendix Table 57

Analysis of variance for non-esterified fatty acid levels – Experiment 2.

Source	d.f.	Mean Squares				
		D 0	D 3	D 10	D 24	D 38
Total	19					
Error	11	0.01554	0.82723	0.00740	0.00172	0.00018
Repetition	5	0.02584	0.64520	0.00363	0.00334	0.00025
Treatment	3	0.00716	1.72142	0.02305	0.00226	0.00020
Linear	1	0.00493	1.24604	0.03706	0.00090	0.00030
Quadratic	1	0.00854	2.98421	0.02321	0.00583	0.00007
Cubic	1	0.00272	0.00260	0.00228	0.00178	0.00019
C.V., %		30.26	53.91	55.40	41.30	19.37

Appendix Table 58

Pen means for protein levels – Experiment 2.

Pen	Trt	Rep	Protein levels, g/dL				
			D 0	D 3	D 10	D 24	D 38
1	2	1	4.7	5.6	4.7	4.0	4.5
2	3	1	5.0	5.0	4.5	--	--
3	4	1	4.8	5.4	4.3	4.3	4.7
4	1	1	4.9	4.9	4.5	4.0	4.8
5	4	2	5.9	6.0	5.1	4.8	5.7
6	2	2	5.6	5.8	5.0	4.7	5.0
7	1	2	5.9	6.1	5.0	4.9	5.4
8	3	2	5.8	5.8	5.3	--	--
9	1	3	4.4	4.7	4.8	4.2	4.3
10	4	3	4.3	4.6	4.5	4.4	5.1
11	3	3	4.6	4.5	4.1	4.6	4.9
12	2	3	4.8	5.2	4.0	4.5	5.2
13	2	4	4.8	5.1	4.5	4.6	5.0
14	4	4	4.2	5.3	5.0	--	--
15	3	4	4.9	5.3	4.2	4.4	5.2
16	1	4	4.7	5.8	4.9	4.1	4.7
17	2	5	4.6	4.6	4.0	4.0	4.5
18	1	5	5.1	5.5	4.8	4.5	5.2
19	4	5	4.8	4.6	4.2	4.5	4.8
20	3	5	4.3	4.4	3.9	4.1	4.2
21	4	6	5.2	5.1	4.8	--	--
22	2	6	4.1	4.5	3.7	3.9	4.2
23	1	6	5.4	5.0	4.6	4.2	4.4
24	3	6	4.7	4.4	4.2	4.6	4.9

Trt 1: Control diet
Trt 2: Control diet + 25 ppm L-carnitine
Trt 3: Control diet + 50 ppm L-carnitine
Trt 4: Control diet + 100 ppm L-carnitine

Dashes indicate data for this variable not included in analysis due to poor pig performance.

Appendix Table 59

Analysis of variance for protein levels – Experiment 2.

Source	d.f.	Mean Squares					
		D 0	D 3	D 10	d.f.	D 24	D 38
Total	23				19		
Error	15	0.10775	0.09844	0.08667	8	0.05526	0.13504
Repetition	5	0.84642	0.89267	0.40600	5	0.17526	0.27108
Treatment	3	0.09375	0.19111	0.28500	3	0.04683	0.07652
Linear	1	0.05030	0.07619	0.00043	1	0.08388	0.18325
Quadratic	1	0.10823	0.45926	0.76001	1	0.00293	0.01835
Cubic	1	0.12273	0.03788	0.09456	1	0.04750	0.02608
C.V., %		6.705	6.112	6.506		5.385	7.600

Appendix Table 60

Pen means for triglyceride levels – Experiment 2.

			Triglyceride levels, mg/dL				
Pen	Trt	Rep	D 0	D 3	D 10	D 24	D 38
1	2	1	124	166	45	52	47
2	3	1	--	--	--	--	--
3	4	1	--	--	38	55	57
4	1	1	35	110	40	30	32
5	4	2	133	82	39	65	77
6	2	2	73	36	57	47	47
7	1	2	142	90	46	42	29
8	3	2	--	--	--	--	--
9	1	3	109	63	90	75	46
10	4	3	46	74	44	35	39
11	3	3	100	56	61	70	46
12	2	3	75	70	46	81	43
13	2	4	44	91	51	38	29
14	4	4	--	--	--	--	--
15	3	4	43	55	34	88	35
16	1	4	120	141	42	40	26
17	2	5	68	79	62	35	89
18	1	5	57	72	25	38	37
19	4	5	36	32	40	42	23
20	3	5	131	80	36	26	23
21	4	6	--	--	--	--	--
22	2	6	73	40	38	30	41
23	1	6	55	81	45	45	37
24	3	6	73	26	83	46	40

Trt 1: Control diet
Trt 2: Control diet + 25 ppm L-carnitine
Trt 3: Control diet + 50 ppm L-carnitine
Trt 4: Control diet + 100 ppm L-carnitine

Dashes indicate data for this variable not included in analysis due to poor pig performance.

Appendix Table 61

Analysis of variance for triglyceride levels – Experiment 2.

Source	d.f.	Mean Squares D 0	D 3	d.f	D 10	D 24	D 38
Total	18			19			
Error	10	1566.890	841.191	11	295.562	310.833	343.102
Repetition	5	1306.537	1947.134	5	233.479	446.282	99.343
Treatment	3	846.201	654.973	3	110.771	142.998	251.405
Linear	1	1103.371	523.546	1	181.266	36.911	120.797
Quadratic	1	879.399	984.659	1	202.010	269.235	30.380
Cubic	1	1299.086	143.573	1	20.926	157.159	496.785
C.V., %		49.03	38.16		35.74	35.98	43.95

Appendix Table 62

Pen means for average daily gain, average daily feed intake, and gain:feed for Phases 1 and 2 – Experiment 3 (Room A).

			Phase 1			Phase 2		
Pen	Trt	Rep	ADG (g)	ADFI (g)	G:F	ADG (g)	ADFI (g)	G:F
2A	4	1	249	302	.824	537	679	.790
3A	2	1	215	225	.956	528	668	.791
4A	4	2	224	224	1.000	441	554	.796
5A	3	2	168	183	.918	415	592	.700
6A	2	3	197	210	.935	387	536	.721
7A	1	3	177	188	.944	399	588	.679
9A	3	1	258	290	.890	457	695	.658
10A	1	1	242	256	.947	555	717	.775
11A	2	2	178	199	.897	490	610	.804
12A	1	2	232	228	1.014	487	671	.726
13A	4	3	145	174	.836	395	496	.796
14A	3	3	203	207	.985	414	548	.755
22A	1	4	226	257	.881	410	543	.754
23A	2	4	250	269	.927	471	641	.736
24A	2	5	204	232	.882	485	602	.806
25A	4	5	199	213	.932	405	528	.768
26A	4	6	228	229	.996	475	580	.819
27A	1	6	173	179	.962	392	501	.783
29A	4	4	195	225	.869	449	557	.806
30A	3	4	152	181	.842	365	479	.763
31A	1	5	266	274	.970	486	642	.757
32A	3	5	145	182	.800	396	508	.779
33A	2	6	212	217	.979	410	550	.745
34A	3	6	222	259	.856	349	551	.633

Trt 1: 0% SBO and 0 ppm L-carnitine
Trt 2: 0% SBO and 50 ppm L-carnitine
Trt 3: 5% SBO and 0 ppm L-carnitine
Trt 4: 5% SBO and 50 ppm L-carnitine

Appendix Table 63

Analysis of variance for average daily gain, average daily feed intake, and gain:feed for Phases 1 and 2 – Experiment 3 (Room A).

		Mean Squares					
		Phase 1			Phase 2		
Source	d.f.	ADG	ADFI	G:F	ADG	ADFI	G:F
Total	23						
Error	15	1214.58	1174.65	0.0033	1148.86	2011.79	0.0020
Repetition	5	1522.08	2498.25	0.0037	7777.55	12761.2	0.0008
Treatment	3	793.86	207.27	0.0055	4913.53	3587.51	0.0070
SBO	1	1387.91	178.05	0.0124	6772.42	10467.1	0.0001
L-carnitine	1	47.01	57.75	0.0001	5039.04	47.12	0.0158
Interaction	1	946.65	386.00	0.0040	2929.13	248.26	0.0053
C.V., %		16.87	15.23	6.21	7.68	7.67	5.91

Appendix Table 64

Pen means for average daily gain, average daily feed intake, and gain:feed for Phase 3 and overall – Experiment 3 (Room A).

			Phase 3			Overall		
Pen	Trt	Rep	ADG (g)	ADFI (g)	G:F	ADG (g)	ADFI (g)	G:F
2A	4	1	621	911	.682	492	666	.739
3A	2	1	594	896	.663	470	635	.740
4A	4	2	589	878	.670	439	587	.748
5A	3	2	556	841	.661	402	576	.698
6A	2	3	477	751	.635	370	529	.698
7A	1	3	540	795	.679	392	558	.702
9A	3	1	556	902	.617	441	664	.664
10A	1	1	615	947	.650	495	680	.728
11A	2	2	599	907	.661	449	611	.734
12A	1	2	571	910	.627	451	642	.702
13A	4	3	447	678	.659	348	478	.728
14A	3	3	455	747	.609	373	532	.702
22A	1	4	528	762	.693	405	548	.738
23A	2	4	637	975	.654	474	666	.712
24A	2	5	550	866	.635	435	602	.723
25A	4	5	520	762	.682	393	531	.740
26A	4	6	558	818	.682	440	575	.766
27A	1	6	477	691	.690	365	486	.752
29A	4	4	664	949	.700	461	614	.751
30A	3	4	505	790	.639	360	515	.700
31A	1	5	628	987	.637	481	672	.716
32A	3	5	465	704	.661	356	494	.720
33A	2	6	538	838	.643	405	568	.713
34A	3	6	470	711	.661	360	533	.716

Trt 1: 0% SBO and 0 ppm L-carnitine
Trt 2: 0% SBO and 50 ppm L-carnitine
Trt 3: 5% SBO and 0 ppm L-carnitine
Trt 4: 5% SBO and 50 ppm L-carnitine

Appendix Table 65

Analysis of variance for average daily gain, average daily feed intake, and gain:feed for Phase 3 and Overall – Experiment 3 (Room A).

		Mean Squares					
		Phase 3			Overall		
Source	d.f.	ADG	ADFI	G:F	ADG	ADFI	G:F
Total	23						
Error	15	1961.44	5596.47	0.0004	974.55	2316.41	0.0003
Repetition	5	8570.81	18761.8	0.0004	5117.75	9487.45	0.0002
Treatment	3	5958.44	8596.88	0.0017	3657.78	3148.96	0.0027
SBO	1	5026.30	16522.3	0.0001	4378.32	7832.26	0.0001
L-carnitine	1	7636.95	8198.10	0.0008	3605.91	1082.19	0.0036
Interaction	1	5212.06	1070.27	0.0041	2989.09	532.42	0.0046
C.V., %		8.08	8.97	3.17	7.45	8.27	2.56

Appendix Table 66

Pen means for average daily gain, average daily feed intake, and gain:feed for Phases 1 and 2 – Experiment 3 (Room B).

			Phase 1			Phase 2		
Pen	Trt	Rep	ADG (g)	ADFI (g)	G:F	ADG (g)	ADFI (g)	G:F
2B	1	7	170	213	.796	402	547	.736
3B	2	7	119	186	.639	381	551	.692
4B	2	8	217	222	.980	440	595	.740
5B	4	8	109	182	.600	378	507	.746
6B	1	9	143	196	.731	425	563	.756
7B	2	9	175	187	.937	459	563	.816
9B	4	7	118	184	.640	431	547	.788
10B	3	7	131	190	.689	415	523	.794
11B	1	8	168	215	.784	440	590	.747
12B	3	8	153	199	.767	388	521	.745
13B	4	9	153	202	.753	442	577	.766
14B	3	9	149	188	.792	385	526	.733
22B	2	10	192	215	.890	336	563	.725
23B	4	10	186	208	.893	413	549	.752
24B	1	11	161	204	.787	370	509	.728
25B	4	11	169	173	.974	348	459	.759
26B	3	12	150	194	.771	303	453	.670
27B	4	12	173	175	.990	301	415	.725
29B	1	10	162	201	.805	315	455	.691
30B	3	10	201	225	.891	389	535	.726
31B	2	11	143	161	.893	380	478	.795
32B	3	11	177	200	.886	360	477	.755
33B	2	12	165	204	.804	369	511	.722
34B	1	12	111	144	.767	350	457	.768

Trt 1: 0% SBO and 0 ppm L-carnitine
Trt 2: 0% SBO and 50 ppm L-carnitine
Trt 3: 5% SBO and 0 ppm L-carnitine
Trt 4: 5% SBO and 50 ppm L-carnitine

Appendix Table 67

Analysis of variance for average daily gain, average daily feed intake, and gain:feed for Phases 1 and 2 – Experiment 3 (Room B).

		Mean Squares					
		Phase 1			Phase 2		
Source	d.f.	ADG	ADFI	G:F	ADG	ADFI	G:F
Total	23						
Error	15	729.29	328.73	0.0101	1108.26	1297.71	0.0012
Repetition	5	1120.02	588.24	0.0197	5494.62	6765.81	0.0014
Treatment	3	377.13	147.63	0.0067	437.31	572.39	0.0005
SBO	1	136.37	29.95	0.0017	562.60	1558.32	0.0001
L-carnitine	1	82.47	195.80	0.0116	742.59	140.70	0.0013
Interaction	1	912.54	217.14	0.0073	6.72	18.15	0.0001
C.V., %		17.09	9.32	12.39	8.66	6.99	4.67

Appendix Table 68

Pen means for average daily gain, average daily feed intake, and gain:feed for Phase 3 and overall – Experiment 3 (Room B).

			Phase 3			Overall		
Pen	Trt	Rep	ADG (g)	ADFI (g)	G:F	ADG (g)	ADFI (g)	G:F
2B	1	7	490	781	.627	373	545	.684
3B	2	7	418	700	.597	326	510	.638
4B	2	8	520	872	.597	411	599	.686
5B	4	8	503	776	.649	353	520	.679
6B	1	9	431	782	.551	353	547	.646
7B	2	9	491	782	.627	396	544	.728
9B	4	7	395	661	.598	336	494	.680
10B	3	7	500	800	.625	371	537	.691
11B	1	8	558	890	.628	412	602	.685
12B	3	8	498	789	.632	366	535	.685
13B	4	9	523	765	.683	396	548	.722
14B	3	9	499	734	.680	365	513	.711
22B	2	10	468	783	.598	346	516	.672
23B	4	10	518	841	.616	392	567	.692
24B	1	11	494	807	.612	360	538	.669
25B	4	11	477	720	.663	349	480	.727
26B	3	12	437	712	.614	312	480	.650
27B	4	12	378	598	.632	296	420	.706
29B	1	10	435	769	.566	319	503	.633
30B	3	10	476	791	.602	371	548	.678
31B	2	11	475	780	.608	353	506	.697
32B	3	11	494	750	.660	361	505	.716
33B	2	12	451	744	.606	345	516	.669
34B	1	12	442	711	.622	321	469	.686

Trt 1: 0% SBO and 0 ppm L-carnitine
Trt 2: 0% SBO and 50 ppm L-carnitine
Trt 3: 5% SBO and 0 ppm L-carnitine
Trt 4: 5% SBO and 50 ppm L-carnitine

Appendix Table 69

Analysis of variance for average daily gain, average daily feed intake, and gain:feed for Phase 3 and Overall – Experiment 3 (Room B).

		Mean Squares					
		Phase 3			Overall		
Source	d.f.	ADG	ADFI	G:F	ADG	ADFI	G:F
Total	23						
Error	15	1393.09	2058.08	0.0007	674.31	914.10	0.0005
Repetition	5	4089.74	9348.06	0.0010	2187.49	3960.04	0.0008
Treatment	3	365.72	4442.94	0.0024	91.87	1093.00	0.0012
SBO	1	28.86	8988.46	0.0072	94.68	2569.70	0.0025
L-carnitine	1	783.70	3572.65	0.0001	5.37	458.15	0.0011
Interaction	1	284.42	767.72	0.0001	175.55	251.17	0.0001
C.V., %		7.88	5.93	4.31	7.26	5.79	3.38

Appendix Table 70

Analysis of variance for average daily gain, average daily feed intake, and gain:feed for Phases 1 and 2 – Experiment 3 (Rooms A and B pooled).

		Mean Squares					
		Phase 1			Phase 2		
Source	d.f.	ADG	ADFI	G:F	ADG	ADFI	G:F
Total	47						
Error	33	949.90	709.27	0.0067	1147.16	1575.52	0.0016
Repetition	11	3779.23	2413.50	0.0233	9611.97	14128.0	0.0012
Treatment	3	441.46	69.84	0.0057	4017.65	3376.67	0.0055
SBO	1	1197.20	177.02	0.0106	5619.48	10051.4	0.0001
L-carnitine	1	127.01	20.44	0.0063	4825.23	12.48	0.0131
Interaction	1	0.16	12.06	0.0003	1608.23	66.08	0.0035
C.V., %		16.90	12.70	9.43	8.20	7.22	5.40

Appendix Table 71

Analysis of variance for average daily gain, average daily feed intake, and gain:feed for Phase 3 and Overall – Experiment 3 (Rooms A and B pooled).

		Mean Squares					
		Phase 3			Overall		
Source	d.f.	ADG	ADFI	G:F	ADG	ADFI	G:F
Total	47						
Error	33	1934.83	3894.24	0.0007	918.15	1557.70	0.0005
Repetition	11	11817.9	18074.7	0.0021	7421.14	9936.82	0.0019
Treatment	3	1813.70	8475.94	0.0025	1894.35	3259.79	0.0025
SBO	1	2146.69	24941.8	0.0046	2880.37	9687.24	0.0010
L-carnitine	1	176.70	473.45	0.0008	1944.76	66.04	0.0044
Interaction	1	1530.70	12.54	0.0020	857.93	26.11	0.0021
C.V., %		8.61	7.81	4.05	7.80	7.15	3.28

Appendix Table 72

Pen means for albumin levels – Experiment 3.

Pen	Trt	Rep	Albumin levels, g/dL			
			D 0	D 10	D 24	D 38
2	1	1	3.00	2.45	2.35	2.60
3	2	1	3.30	2.70	2.15	2.50
4	2	2	3.05	2.65	2.55	2.95
5	4	2	3.05	2.75	2.60	2.70
6	1	3	3.10	2.45	2.60	2.80
7	2	3	2.85	2.35	2.70	2.95
9	4	1	2.70	2.65	2.15	2.25
10	3	1	2.90	2.70	2.45	2.85
11	1	2	2.60	2.20	2.35	2.70
12	3	2	2.70	2.25	2.40	2.35
13	4	3	2.90	2.45	2.60	2.50
14	3	3	2.95	2.55	2.65	2.80
22	2	4	2.80	2.45	2.50	2.50
23	4	4	2.60	2.25	2.40	2.90
24	1	5	2.80	2.25	2.05	2.85
25	4	5	2.65	2.55	2.45	2.70
26	3	6	2.75	2.30	2.65	2.60
27	4	6	2.70	2.30	2.30	2.50
29	1	4	2.70	2.30	2.70	2.65
30	3	4	3.15	2.35	2.50	2.55
31	2	5	2.50	2.45	2.45	2.75
32	3	5	2.70	2.50	2.45	2.75
33	2	6	2.30	2.30	2.75	2.70
34	1	6	2.70	2.25	2.15	2.60

Trt 1: 0% SBO and 0 ppm L-carnitine
Trt 2: 0% SBO and 50 ppm L-carnitine
Trt 3: 5% SBO and 0 ppm L-carnitine
Trt 4: 5% SBO and 50 ppm L-carnitine

Appendix Table 73

Analysis of variance for albumin levels – Experiment 3.

Source	d.f.	Mean Squares			
		D 0	D 10	D 24	D 38
Total	23				
Error	15	0.0474653	0.0168333	0.0283333	0.0359167
Repetition	5	0.0873542	0.0546667	0.0656667	0.0294167
Treatment	3	0.0087153	0.0391667	0.0337500	0.0208333
SBO	1	0.0001042	0.0266667	0.0037500	0.0504167
L-carnitine	1	0.0176042	0.0704167	0.0037500	0.0016667
Interaction	1	0.0084375	0.0204167	0.0937500	0.0104167
C.V., %		7.7521	5.3319	6.8587	7.1069

Appendix Table 74

Pen means for blood urea nitrogen levels – Experiment 3.

Pen	Trt	Rep	Blood urea nitrogen levels, mg/dL			
			D 0	D 10	D 24	D 38
2	1	1	6.35	5.60	6.50	12.50
3	2	1	5.50	9.15	8.90	13.55
4	2	2	6.45	6.10	8.75	11.70
5	4	2	7.65	9.60	5.50	10.55
6	1	3	7.40	6.05	8.30	12.30
7	2	3	11.90	6.65	7.20	11.95
9	4	1	6.35	7.80	7.95	13.60
10	3	1	6.30	6.35	7.15	12.35
11	1	2	4.30	6.95	7.20	10.75
12	3	2	6.85	8.30	8.10	11.90
13	4	3	7.00	2.80	5.20	9.95
14	3	3	5.65	13.00	9.35	13.30
22	2	4	5.30	7.30	7.60	13.90
23	4	4	9.80	5.10	6.70	11.50
24	1	5	9.30	5.60	6.65	13.20
25	4	5	9.80	6.80	7.05	10.05
26	3	6	5.55	6.60	8.45	11.95
27	4	6	4.85	4.75	5.45	10.90
29	1	4	4.90	2.80	7.05	11.95
30	3	4	8.50	4.35	6.25	9.60
31	2	5	7.40	10.15	9.40	15.05
32	3	5	8.80	4.85	7.60	12.80
33	2	6	6.90	5.25	10.00	9.80
34	1	6	5.55	9.80	9.60	15.00

Trt 1: 0% SBO and 0 ppm L-carnitine
Trt 2: 0% SBO and 50 ppm L-carnitine
Trt 3: 5% SBO and 0 ppm L-carnitine
Trt 4: 5% SBO and 50 ppm L-carnitine

Appendix Table 75

Analysis of variance for blood urea nitrogen levels – Experiment 3.

Source	d.f.	Mean Squares			
		D 0	D 10	D 24	D 38
Total	23				
Error	15	3.0809097	6.8555556	1.3279444	2.4080556
Repetition	5	5.7728512	3.8735000	0.9176667	1.7980000
Treatment	3	1.7631597	2.9168056	5.6023611	3.2168056
SBO	1	3.7209375	0.0600000	0.2604167	1.0837500
L-carnitine	1	1.4259375	0.0504167	6.4066667	7.2600000
Interaction	1	0.1426042	8.6400000	10.1400000	1.3066667
C.V., %		25.0229	38.8618	15.2044	12.8380

Appendix Table 76

Pen means for C-reactive protein levels – Experiment 3.

Pen	Trt	Rep	C-reactive protein levels, mg/L			
			D 0	D 10	D 24	D 38
2	1	1	1.10	4.36	7.02	6.44
3	2	1	1.10	3.53	4.19	4.63
4	2	2	1.85	2.18	4.29	3.54
5	4	2	1.10	1.74	3.67	5.11
6	1	3	1.10	2.30	4.67	2.47
7	2	3	1.50	4.58	11.36	4.83
9	4	1	1.10	2.94	3.76	4.20
10	3	1	4.26	5.61	11.38	7.66
11	1	2	1.10	2.41	4.57	3.62
12	3	2	1.10	2.39	2.97	4.50
13	4	3	1.10	1.66	5.82	3.53
14	3	3	2.46	4.37	3.36	3.80
22	2	4	7.63	3.08	5.73	6.59
23	4	4	1.83	1.85	5.68	4.14
24	1	5	1.10	5.14	4.67	4.80
25	4	5	2.60	1.92	4.55	6.39
26	3	6	1.25	1.10	3.28	2.86
27	4	6	2.96	2.11	3.02	2.42
29	1	4	--	1.59	1.31	11.46
30	3	4	1.78	1.39	1.89	8.79
31	2	5	3.48	2.61	4.87	6.70
32	3	5	--	1.72	3.00	2.43
33	2	6	6.92	6.42	6.95	4.89
34	1	6	1.10	6.19	2.71	4.43

Trt 1: 0% SBO and 0 ppm L-carnitine
Trt 2: 0% SBO and 50 ppm L-carnitine
Trt 3: 5% SBO and 0 ppm L-carnitine
Trt 4: 5% SBO and 50 ppm L-carnitine

Dashes indicate data for this variable not included in analysis due to poor pig performance.

Appendix Table 77

Analysis of variance for C-reactive protein levels – Experiment 3.

Source	d.f.	Mean Squares D 0	d.f.	D 10	D 24	D 38
Total	21		23			
Error	13	2.92690	15	2.05548	5.97092	3.48845
Repetition	5	2.57430	5	3.13783	6.84560	9.88343
Treatment	3	6.21393	3	3.90838	5.68719	1.63740
SBO	1	1.61297	1	10.12700	4.13340	3.06020
L-carnitine	1	5.34668	1	0.65010	7.10682	1.64850
Interaction	1	10.44810	1	0.94804	5.82135	0.20350
C.V., %		76.0057		47.0128	51.1202	37.2833

Appendix Table 78

Pen means for glucose levels – Experiment 3.

Pen	Trt	Rep	Glucose levels, mg/dL			
			D 0	D 10	D 24	D 38
2	1	1	132.0	95.0	106.5	120.0
3	2	1	131.5	103.5	93.0	123.0
4	2	2	123.0	106.0	107.5	129.0
5	4	2	151.5	98.0	135.5	113.0
6	1	3	127.0	120.0	113.5	128.5
7	2	3	136.0	115.5	108.0	107.5
9	4	1	142.5	88.0	83.0	119.5
10	3	1	120.5	93.5	112.0	114.5
11	1	2	129.0	108.5	115.0	118.5
12	3	2	120.5	96.0	126.0	131.0
13	4	3	234.5	128.0	108.5	136.5
14	3	3	128.5	93.5	104.0	113.5
22	2	4	151.0	96.5	113.0	118.5
23	4	4	124.0	108.5	95.0	121.5
24	1	5	131.0	108.0	88.5	107.5
25	4	5	110.5	102.5	116.5	115.5
26	3	6	123.5	101.5	106.5	137.5
27	4	6	149.0	113.5	108.5	134.0
29	1	4	149.0	111.0	119.0	110.5
30	3	4	157.5	81.0	100.5	108.0
31	2	5	124.5	88.5	104.5	102.0
32	3	5	123.0	116.0	116.5	126.5
33	2	6	130.0	113.0	103.5	174.0
34	1	6	123.5	80.5	100.0	127.0

Trt 1: 0% SBO and 0 ppm L-carnitine
Trt 2: 0% SBO and 50 ppm L-carnitine
Trt 3: 5% SBO and 0 ppm L-carnitine
Trt 4: 5% SBO and 50 ppm L-carnitine

Appendix Table 79

Analysis of variance for glucose levels – Experiment 3.

Source	d.f.	Mean Squares			
		D 0	D 10	D 24	D 38
Total	23				
Error	15	553.61667	151.41389	122.67431	161.84722
Repetition	5	608.60000	165.07500	217.16875	473.32500
Treatment	3	666.79167	99.63889	36.98264	51.59722
SBO	1	400.16667	28.16667	68.34375	1.04167
L-carnitine	1	852.04167	135.37500	41.34375	108.37500
Interaction	1	748.16667	135.37500	1.26042	45.37500
C.V., %		17.2535	11.9757	10.2852	10.3959

Appendix Table 80

Pen means for non-esterified fatty acid levels – Experiment 3.

Pen	Trt	Rep	Non-esterified fatty acid levels, mmol/L			
			D 0	D 10	D 24	D 38
2	1	1	0.405	0.085	0.140	0.070
3	2	1	0.315	0.070	0.090	0.055
4	2	2	0.260	0.110	0.100	0.055
5	4	2	0.380	0.080	0.190	0.085
6	1	3	0.355	0.045	0.105	0.065
7	2	3	0.455	0.050	0.165	0.065
9	4	1	0.310	0.170	0.150	0.080
10	3	1	0.525	0.175	0.125	0.160
11	1	2	0.235	0.125	0.045	0.040
12	3	2	0.485	0.120	0.095	0.065
13	4	3	0.160	0.080	0.070	0.045
14	3	3	0.125	0.220	0.100	0.070
22	2	4	0.360	0.075	0.120	0.040
23	4	4	0.240	0.065	0.090	0.105
24	1	5	0.385	0.050	0.085	0.090
25	4	5	0.405	0.095	0.095	0.170
26	3	6	0.285	0.160	0.105	0.080
27	4	6	0.355	0.130	0.085	0.070
29	1	4	0.170	0.085	0.055	0.030
30	3	4	0.275	0.130	0.125	0.155
31	2	5	0.260	0.160	0.180	0.070
32	3	5	0.470	0.220	0.090	0.095
33	2	6	0.260	0.090	0.070	0.055
34	1	6	0.625	0.190	0.105	0.060

Trt 1: 0% SBO and 0 ppm L-carnitine
Trt 2: 0% SBO and 50 ppm L-carnitine
Trt 3: 5% SBO and 0 ppm L-carnitine
Trt 4: 5% SBO and 50 ppm L-carnitine

Appendix Table 81

Analysis of variance for non-esterified fatty acid levels – Experiment 3.

Source	d.f.	Mean Squares			
		D 0	D 10	D 24	D 38
Total	23				
Error	15	0.017110	0.002046	0.001638	0.000953
Repetition	5	0.012985	0.001687	0.000598	0.001354
Treatment	3	0.004775	0.008186	0.001097	0.003409
SBO	1	0.000204	0.010838	0.000150	0.009801
L-carnitine	1	0.014017	0.007704	0.002204	0.000301
Interaction	1	0.000104	0.006017	0.000938	0.000126
C.V., %		38.7571	39.0509	37.6492	39.5085

Appendix Table 82

Pen means for protein levels – Experiment 3.

Pen	Trt	Rep	Protein levels, g/dL			
			D 0	D 10	D 24	D 38
2	1	1	5.40	4.80	5.05	4.70
3	2	1	5.40	5.00	4.60	4.50
4	2	2	5.35	4.80	4.65	4.70
5	4	2	5.70	5.10	4.95	4.80
6	1	3	5.30	4.70	4.95	4.85
7	2	3	5.85	4.55	5.05	5.65
9	4	1	4.75	4.80	4.50	4.45
10	3	1	5.75	5.00	4.80	4.90
11	1	2	5.20	4.45	4.65	4.25
12	3	2	5.15	4.70	4.45	4.35
13	4	3	5.30	4.65	4.80	4.60
14	3	3	5.90	5.15	5.05	4.60
22	2	4	5.50	4.45	5.00	4.60
23	4	4	5.00	4.35	4.30	4.60
24	1	5	5.00	4.20	3.35	4.60
25	4	5	5.20	4.75	4.20	4.55
26	3	6	5.20	4.40	4.15	4.70
27	4	6	5.30	4.30	3.50	4.20
29	1	4	4.60	4.25	4.25	5.20
30	3	4	5.60	4.30	3.90	4.60
31	2	5	4.85	4.50	4.30	4.80
32	3	5	5.00	4.50	4.00	4.75
33	2	6	5.50	4.65	4.80	5.15
34	1	6	5.00	4.45	3.40	4.80

Trt 1: 0% SBO and 0 ppm L-carnitine
Trt 2: 0% SBO and 50 ppm L-carnitine
Trt 3: 5% SBO and 0 ppm L-carnitine
Trt 4: 5% SBO and 50 ppm L-carnitine

Appendix Table 83

Analysis of variance for protein levels – Experiment 3.

Source	d.f.	Mean Squares			
		D 0	D 10	D 24	D 38
Total	23				
Error	15	0.0917500	0.0363889	0.1385486	0.0952222
Repetition	5	0.1479167	0.1961667	0.7029375	0.0706667
Treatment	3	0.1675000	0.0538889	0.2395486	0.1426389
SBO	1	0.0337500	0.0600000	0.0876042	0.3037500
L-carnitine	1	0.0150000	0.0416667	0.2926042	0.0037500
Interaction	1	0.4537500	0.0600000	0.3384375	0.1204167
C.V., %		5.7332	4.1320	8.3763	6.5597

Appendix Table 84

Pen means for triglyceride levels – Experiment 3.

Pen	Trt	Rep	Triglyceride levels, mg/dL			
			D 0	D 10	D 24	D 38
2	1	1	154.5	38.0	40.0	44.5
3	2	1	104.0	27.0	35.0	18.0
4	2	2	88.5	28.5	34.5	27.5
5	4	2	175.0	26.5	37.5	38.5
6	1	3	121.5	31.5	32.5	34.5
7	2	3	80.5	34.0	35.0	53.0
9	4	1	139.5	36.0	55.5	55.0
10	3	1	73.5	37.0	20.5	35.0
11	1	2	60.5	29.5	29.5	26.5
12	3	2	89.0	26.0	41.5	40.5
13	4	3	84.0	57.0	35.0	32.5
14	3	3	60.5	52.5	35.5	45.5
22	2	4	50.0	31.0	44.5	45.0
23	4	4	99.5	39.5	40.0	57.0
24	1	5	103.0	34.5	40.0	71.5
25	4	5	76.5	41.0	36.0	53.5
26	3	6	90.5	38.0	44.0	46.5
27	4	6	98.0	32.5	33.5	58.0
29	1	4	45.0	39.5	57.5	24.0
30	3	4	86.0	28.0	50.0	36.5
31	2	5	65.5	38.5	36.0	32.5
32	3	5	158.0	37.5	43.0	46.5
33	2	6	72.0	35.0	48.0	40.5
34	1	6	104.5	26.0	30.0	59.0

Trt 1: 0% SBO and 0 ppm L-carnitine
Trt 2: 0% SBO and 50 ppm L-carnitine
Trt 3: 5% SBO and 0 ppm L-carnitine
Trt 4: 5% SBO and 50 ppm L-carnitine

Appendix Table 85

Analysis of variance for triglyceride levels – Experiment 3.

Source	d.f.	Mean Squares			
		D 0	D 10	D 24	D 38
Total	23				
Error	15	1155.4132	41.79653	74.96042	151.7188
Repetition	5	1061.8854	115.21875	90.74375	203.1688
Treatment	3	1278.5382	53.28819	1.84375	171.5104
SBO	1	1357.5104	142.59375	3.76042	195.5104
L-carnitine	1	7.5938	3.01042	1.76042	0.0104
Interaction	1	2470.5104	14.26042	0.01042	319.0104
C.V., %		35.7882	18.3731	22.2356	28.9396

Appendix Table 86

Pen means for average daily gain, average daily feed intake, and gain:feed for Phases 1 and 2 – Experiment 4 (Room A).

			Phase 1			Phase 2		
Pen	Trt	Rep	ADG (g)	ADFI (g)	G:F	ADG (g)	ADFI (g)	G:F
1A	2	5	61	97	.627	249	284	.878
2A	2	3	172	204	.844	347	480	.724
3A	5	3	123	149	.826	346	402	.860
4A	4	3	179	196	.915	296	397	.746
5A	3	3	203	228	.893	392	507	.773
6A	1	3	86	128	.672	343	436	.787
7A	4	5	119	130	.911	330	387	.855
8A	3	5	150	138	1.088	293	387	.757
9A	1	4	122	156	.785	307	421	.729
10A	2	4	102	119	.854	320	400	.801
11A	4	4	184	200	.923	369	476	.775
12A	5	4	57	101	.563	321	360	.892
13A	3	4	139	172	.806	261	374	.698
14A	5	5	105	104	1.011	271	321	.845
15A	5	1	75	107	.699	442	505	.876
16A	2	1	122	183	.668	429	551	.779
17A	4	1	105	167	.628	455	568	.801
18A	3	1	100	145	.693	413	483	.855
19A	1	1	75	135	.555	445	603	.737
20A	1	2	117	140	.840	414	498	.832
21A	3	2	166	196	.847	383	472	.810
22A	4	2	120	198	.609	319	441	.725
23A	5	2	177	202	.883	461	533	.867
24A	2	2	165	186	.891	415	486	.853

Trt 1: Control diet
Trt 2: Control diet + 5% SBO and 0 ppm L-carnitine
Trt 3: Control diet + 5% SBO and 50 ppm L-carnitine
Trt 4: Control diet + 5% CO and 0 ppm L-carnitine
Trt 5: Control diet + 5% CO and 50 ppm L-carnitine

Appendix Table 87

Analysis of variance for average daily gain, average daily feed intake, and gain:feed for Phases 1 and 2 – Experiment 4 (Room A).

		Mean Squares					
		Phase 1			Phase 2		
Source	d.f.	ADG	ADFI	G:F	ADG	ADFI	G:F
Total	23						
Error	15	1115.25	685.76	0.0175	1660.50	2337.15	0.0025
Repetition	4	3362.50	3868.76	0.0382	17047.8	24219.6	0.0024
Treatment	4	2513.56	2524.07	0.0097	297.88	1070.17	0.0073
Fat	1	4355.68	3367.01	0.0161	57.05	1991.21	0.0031
L-carnitine	1	56.68	953.99	0.0096	135.41	793.04	0.0044
Interaction	1	4723.20	5123.52	0.0010	413.69	1429.74	0.0168
Fat Source	1	918.69	651.74	0.0030	585.36	66.69	0.0049
C.V., %		26.49	16.64	16.66	11.34	10.77	6.24

Appendix Table 88

Pen means for average daily gain, average daily feed intake, and gain:feed for Phase 3 and overall – Experiment 4 (Room A).

			Phase 3			Overall		
Pen	Trt	Rep	ADG (g)	ADFI (g)	G:F	ADG (g)	ADFI (g)	G:F
1A	2	5	369	524	.705	230	306	.752
2A	2	3	501	782	.640	344	496	.694
3A	5	3	474	637	.745	319	402	.794
4A	4	3	476	665	.716	321	425	.755
5A	3	3	554	815	.680	387	524	.740
6A	1	3	534	807	.661	327	465	.703
7A	4	5	512	715	.716	325	417	.779
8A	3	5	462	691	.669	305	412	.742
9A	1	4	369	604	.612	270	399	.675
10A	2	4	474	688	.689	290	389	.746
11A	4	4	553	800	.691	373	499	.748
12A	5	4	486	666	.729	294	383	.767
13A	3	4	417	613	.681	276	392	.704
14A	5	5	438	585	.749	275	342	.805
15A	5	1	575	870	.661	371	503	.737
16A	2	1	549	869	.632	372	543	.686
17A	4	1	468	785	.596	348	515	.677
18A	3	1	596	919	.649	376	524	.717
19A	1	1	606	997	.608	383	589	.649
20A	1	2	580	839	.692	377	501	.752
21A	3	2	452	763	.592	338	484	.698
22A	4	2	423	723	.584	292	460	.634
23A	5	2	544	794	.684	400	517	.773
24A	2	2	612	861	.711	403	519	.777

Trt 1: Control diet
Trt 2: Control diet + 5% SBO and 0 ppm L-carnitine
Trt 3: Control diet + 5% SBO and 50 ppm L-carnitine
Trt 4: Control diet + 5% CO and 0 ppm L-carnitine
Trt 5: Control diet + 5% CO and 50 ppm L-carnitine

Appendix Table 89

Analysis of variance for average daily gain, average daily feed intake, and gain:feed for Phase 3 and overall – Experiment 4 (Room A).

		Mean Squares					
		Phase 3			Overall		
Source	d.f.	ADG	ADFI	G:F	ADG	ADFI	G:F
Total	23						
Error	15	4848.38	6709.36	0.0015	1640.98	2010.83	0.0013
Repetition	4	9556.80	45640.2	0.0042	6506.21	18795.8	0.0028
Treatment	4	346.16	3299.19	0.0031	65.56	1255.62	0.0035
Fat	1	518.21	6772.86	0.0019	81.93	737.63	0.0035
L-carnitine	1	194.50	170.47	0.0013	88.20	360.49	0.0026
Interaction	1	600.28	2248.68	0.0069	90.65	3162.36	0.0057
Fat Source	1	71.63	4004.73	0.0025	1.44	762.00	0.0023
C.V., %		13.90	10.91	5.76	12.16	9.78	5.01

Appendix Table 90

Pen means for average daily gain, average daily feed intake, and gain:feed for Phases 1 and 2 – Experiment 4 (Room B).

			Phase 1			Phase 2		
Pen	Trt	Rep	ADG (g)	ADFI (g)	G:F	ADG (g)	ADFI (g)	G:F
1B	4	10	90	109	.825	310	390	.794
2B	1	8	117	122	.955	356	453	.787
3B	4	8	116	153	.758	348	437	.796
4B	5	8	145	162	.894	356	472	.755
5B	3	8	122	168	.730	328	407	.806
6B	2	8	73	152	.476	380	477	.797
7B	3	10	152	188	.812	316	414	.764
8B	2	10	132	164	.801	259	379	.684
9B	3	9	112	141	.794	319	404	.790
10B	2	9	129	159	.812	314	396	.794
11B	4	9	122	143	.848	297	373	.798
12B	5	9	114	142	.808	333	447	.745
13B	1	9	136	153	.887	373	467	.799
14B	5	10	104	142	.733	341	427	.798
15B	3	6	145	190	.766	370	482	.767
16B	5	6	154	174	.886	402	520	.772
17B	1	6	87	131	.667	413	511	.808
18B	4	6	125	179	.700	392	540	.726
19B	2	6	115	173	.665	386	472	.818
20B	4	7	117	168	.697	445	527	.844
21B	5	7	93	141	.658	340	455	.746
22B	1	7	127	169	.752	386	487	.791
23B	2	7	103	158	.649	374	484	.772
24B	3	7	170	229	.742	383	499	.767

Trt 1: Control diet
Trt 2: Control diet + 5% SBO and 0 ppm L-carnitine
Trt 3: Control diet + 5% SBO and 50 ppm L-carnitine
Trt 4: Control diet + 5% CO and 0 ppm L-carnitine
Trt 5: Control diet + 5% CO and 50 ppm L-carnitine

Appendix Table 91

Analysis of variance for average daily gain, average daily feed intake, and gain:feed for Phases 1 and 2 – Experiment 4 (Room B).

		Mean Squares					
		Phase 1			Phase 2		
Source	d.f.	ADG	ADFI	G:F	ADG	ADFI	G:F
Total	23						
Error	15	606.52	441.71	0.0091	837.11	910.83	0.0013
Repetition	4	82.80	739.72	0.0133	5721.84	8789.13	0.0004
Treatment	4	695.70	1173.56	0.0136	609.08	701.25	0.0007
Fat	1	90.84	1362.90	0.0168	1507.80	993.35	0.0008
L-carnitine	1	1832.46	684.68	0.0175	14.78	141.09	0.0006
Interaction	1	602.58	488.86	0.0042	27.19	162.68	0.0015
Fat Source	1	256.90	2157.80	0.0157	886.58	1507.89	0.0001
C.V., %		20.39	13.24	12.54	8.15	6.63	4.69

Pen means for average daily gain, average daily feed intake, and gain:feed for Phase 3 and overall – Experiment 4 (Room B).

			Phase 3			Overall		
Pen	Trt	Rep	ADG (g)	ADFI (g)	G:F	ADG (g)	ADFI (g)	G:F
1B	4	10	426	614	.694	295	398	.739
2B	1	8	440	713	.617	324	462	.702
3B	4	8	457	677	.675	327	451	.725
4B	5	8	494	735	.671	351	488	.720
5B	3	8	451	707	.638	319	455	.702
6B	2	8	484	778	.623	339	498	.680
7B	3	10	400	654	.612	304	443	.687
8B	2	10	480	694	.691	307	439	.699
9B	3	9	422	618	.683	302	414	.731
10B	2	9	447	636	.703	314	422	.745
11B	4	9	461	662	.696	311	419	.743
12B	5	9	464	688	.675	311	436	.713
13B	1	9	469	736	.637	346	484	.716
14B	5	10	451	666	.677	319	440	.725
15B	3	6	530	810	.654	370	526	.703
16B	5	6	558	779	.716	394	525	.751
17B	1	6	468	810	.578	348	521	.667
18B	4	6	597	993	.601	397	612	.649
19B	2	6	517	810	.638	351	495	.709
20B	4	7	501	757	.662	379	517	.733
21B	5	7	528	757	.698	344	483	.712
22B	1	7	542	864	.627	375	542	.692
23B	2	7	489	741	.660	345	493	.700
24B	3	7	511	787	.649	374	534	.700

Trt 1: Control diet
Trt 2: Control diet + 5% SBO and 0 ppm L-carnitine
Trt 3: Control diet + 5% SBO and 50 ppm L-carnitine
Trt 4: Control diet + 5% CO and 0 ppm L-carnitine
Trt 5: Control diet + 5% CO and 50 ppm L-carnitine

Appendix Table 93

Analysis of variance for average daily gain, average daily feed intake, and gain:feed for Phase 3 and overall – Experiment 4 (Room B).

		Mean Squares					
		Phase 3			Overall		
Source	d.f.	ADG	ADFI	G:F	ADG	ADFI	G:F
Total	23						
Error	15	920.68	3420.60	0.0008	327.92	1044.41	0.0006
Repetition	4	8080.75	27081.8	0.0013	3749.24	10030.3	0.0008
Treatment	4	1036.39	1391.95	0.0031	147.19	287.96	0.0006
Fat	1	692.55	3852.53	0.0081	22.70	898.34	0.0012
L-carnitine	1	120.49	1288.98	0.0001	28.54	0.04	0.0001
Interaction	1	1232.29	1.43	0.0018	0.59	123.01	0.0001
Fat Source	1	2100.23	424.86	0.0023	536.96	130.46	0.0012
C.V., %		6.29	7.94	4.25	5.33	6.75	3.38

Appendix Table 94

Analysis of variance for average daily gain, average daily feed intake, and gain:feed for Phases 1 and 2 – Experiment 4 (Rooms A and B pooled).

		Mean Squares					
		Phase 1			Phase 2		
Source	d.f.	ADG	ADFI	G:F	ADG	ADFI	G:F
Total	47						
Error	34	875.45	594.29	0.0132	1129.86	1529.12	0.0024
Repetition	9	1519.73	2027.96	0.0250	10110.9	14783.9	0.0021
Treatment	4	2224.53	2874.43	0.0101	669.21	953.84	0.0020
Fat	1	2852.30	4507.13	0.0001	1075.73	2898.68	0.0004
L-carnitine	1	622.28	11.14	0.0266	30.38	132.57	0.0008
Interaction	1	4349.94	4388.82	0.0136	114.38	313.94	0.0042
Fat Source	1	1073.61	2590.65	0.0025	1456.37	470.18	0.0027
C.V., %		23.97	15.42	14.78	9.41	8.65	6.19

Appendix Table 95

Analysis of variance for average daily gain, average daily feed intake, and gain:feed for Phase 3 and overall – Experiment 4 (Rooms A and B pooled).

		Mean Squares					
		Phase 3			Overall		
Source	d.f.	ADG	ADFI	G:F	ADG	ADFI	G:F
Total	47						
Error	34	2634.73	4621.05	0.0011	881.54	1405.57	0.0010
Repetition	9	8307.91	32628.0	0.0026	4602.90	13376.2	0.0021
Treatment	4	621.28	3399.53	0.0056	103.02	1053.40	0.0032
Fat	1	6.31	10420.8	0.0089	9.19	1632.01	0.0044
L-carnitine	1	4.41	1198.48	0.0009	108.54	184.26	0.0016
Interaction	1	1776.36	1068.43	0.0078	52.92	2266.38	0.0036
Fat Source	1	698.06	910.40	0.0048	241.42	130.94	0.0034
C.V., %		10.44	9.14	4.93	8.23	8.00	4.28

VITA

Michael James Rincker

Candidate for the Degree of

Master of Science

Thesis: EFFECTS OF L-CARNITINE ON GROWTH PERFORMANCE, APPARENT NUTRIENT DIGESTIBILITY, AND WHOLE BODY COMPOSITION IN WEANLING PIGS

Major Field: Animal Science

Biographical:

Personal Data: Born in Effingham, Illinois, July 11, 1977, the son of Jim and Sharon Rincker.

Education: Graduated from Windsor High School, Windsor, Illinois in May, 1995; Associate in Science degree from Lake Land College in May, 1997; Bachelor of Science degree in Animal Science from Oklahoma State University, Stillwater, in May, 1999; Completed requirements for the Master of Science degree with a major in Animal Science at Oklahoma State University in August, 2001.

Experience: Raised on a grain and livestock farm near Windsor, Illinois, while helping in every area of production throughout high school and junior college; employed by Oklahoma State University Swine Research Facility as an undergraduate; employed by The Pork Group, a division of Tyson Foods, Summer, 1999; served as a graduate research and teaching assistant, Oklahoma State University, Fall, 1999 to present.

Professional Membership: American Society of Animal Science
American Registry of Professional Animal Scientists