Effects of increasing dietary L-carnitine on growth performance of weanling pigs[1,2]

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ABSTRACT: Four experiments were conducted to evaluate the effects of supplementing graded levels (0 to 100 ppm) of L-carnitine to the diet of weanling pigs on growth performance during a 34- to 38-d experimental period. A fifth experiment was conducted to determine the effects of addition of L-carnitine to diets with or without added soybean oil (SBO) on growth performance. In Exp. 1, 128 pigs (initial BW = 5.5 kg) were allotted to four dietary treatments (six pens per treatment of four to six pigs per pen). Dietary treatments were a control diet containing no added L-carnitine and the control diet with 25, 50, or 100 ppm of added L-carnitine. In Exp. 2, 3, and 4, pigs (4.8 to 5.6 kg of BW) were allotted to five dietary treatments consisting of either a control diet containing no added L-carnitine or the control diet with 25, 50, 75, or 100 ppm of added L-carnitine. All diets in Exp. 1 to 4 contained added soybean oil (4 to 6%). There were seven pens per treatment (four to five pigs per pen) in Exp. 2, whereas Exp. 3 and 4 had five and six pens/treatment (eight pigs per pen), respectively. In general, dietary carnitine additions had only minor effects on growth performance during Phases 1 and 3; however, dietary L-carnitine increased (linear [Exp. 1], quadratic [Exp. 2 to 4], $P < 0.03$) ADG and gain:feed (G:F) during Phase 2. The improvements in growth performance during Phase 2 were of great enough magnitude that carnitine addition tended to increase ADG (linear, $P < 0.10$) and improve G:F (quadratic, $P < 0.02$) for the entire 38-d period. In Exp. 5, 216 weanling pigs (5.8 kg of BW) were allotted (12 pens/treatment of four to five pigs per pen) to four dietary treatments. The four dietary treatments were arranged in a 2×2 factorial with main effects of added SBO (0 or 5%) and added L-carnitine (0 or 50 ppm). Pigs fed SBO tended ($P < 0.07$) to grow more slowly and consumed less feed compared with those not fed SBO, but G:F was improved ($P < 0.02$). The addition of L-carnitine did not affect ($P > 0.10$) ADG or ADFI; however, it improved ($P < 0.03$) G:F. Also, the increase in G:F associated with L-carnitine tended to be more pronounced for pigs fed SBO than those not fed SBO (carnitine × SBO, $P < 0.10$). These results suggest that the addition of 50 to 100 ppm of added L-carnitine to the diet improved growth performance of weanling pigs. In addition, supplemental L-carnitine tended to be more effective when SBO was provided in the diet.

Key Words: Growth, L-Carnitine, Piglets, Soybean Oil

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Introduction

Carnitine is a vitamin-like compound that is necessary for the transport of long-chain fatty acids across the inner mitochondrial membrane for β-oxidation (Groff and Gropper, 2000). Carnitine can be synthesized in the body from protein-bound lysine and methionine by most animals and is therefore not considered to be an essential nutrient for pigs. Young neonates, however, may not synthesize sufficient amounts of carnitine (Borum et al., 1983). Kerner et al. (1984) reported that the biosynthesis of carnitine is limited in pigs directly

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after weaning. Because of the current practice of weaning pigs at younger ages (<21 d), nursery pigs are started on feed at an age when carnitine biosynthesis is low.

Carnitine addition to complex nursery diets has been shown to improve growth performance of early-weaned pigs (Newton and Hayden, 1988; Li et al., 1999; Heo et al., 2000b). Weeden et al. (1990) and Owen et al. (1996) reported that adding up to 1,000 ppm of carnitine to nursery diets containing soybean oil (**SBO**) improved feed efficiency 3 to 5 wk postweaning. However, other studies have reported no effects of added L-carnitine on growth performance of weanling pigs (Hoffman et al., 1993; Owen et al., 2001b). These conflicting results appear to be characteristic of studies conducted on vitamin requirement estimates. It is likely that factors such as age, health, environment, lean growth potential, and diet influence responses to added carnitine.

Because of the relatively high cost of L-carnitine and high inclusion rates in previous research, carnitine addition to nursery diets has not been economically feasible. If improvements in growth performance were found at lower inclusion rates, carnitine addition might become economically feasible in nursery pig diets. Therefore, the objective of these studies was to determine if low levels of added L-carnitine (0 to 100 ppm) would improve growth performance of weanling pigs.

Materials and Methods

General Procedures

Experiments 1 and 5 were conducted at the Oklahoma State University Swine Teaching and Research Facility. The Oklahoma State University Animal Care and Use Committee approved the experimental protocols. The Kansas State University Animal Care and Use Committee approved the experimental protocols for Exp. 2, 3, and 4. Experiment 2 was conducted at the Kansas State University Segregated Early-Weaning Facility, and Exp. 3 and 4 were conducted at a commercial research facility in northeast Kansas.

Experiment 1. One hundred twenty-eight crossbred (Yorkshire × Hampshire) pigs were weaned at 18 ± 4 d and placed in temperature-controlled nursery rooms during 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 six replicate pens per treatment and pigs were grouped with four to six pigs per pen. Within a replicate, the number of pigs in each pen was the same. Dietary treatments were formulated by supplementing the basal diets (Table 1) with 0, 25, 50, and 100 ppm of L-carnitine (Lonza Inc., Fair Lawn, NJ). Pigs were fed in three dietary phases (Phase 1: d 0 to 10; Phase 2: d 10 to 24; and Phase 3: d 24 to 38). Complexity of the diet changed with phases to satisfy the nutrient requirements (NRC, 1998) and changes in digestive capacity 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 fishmeal, whereas Phase 3 (1.2% Lys) diets were typical corn-soybean meal based. All diets were fed in pelleted form and contained 5.0% SBO as a dietary fat source. L-Carnitine replaced cornstarch in the diet to provide the desired carnitine concentrations.

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 ad libitum access to feed and water throughout the experiment. Room temperature was maintained 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 on d 0, 10, 24, and

Table 1. Composition of basal diets for Exp. 1 and 5 (as-fed basis)

	Diets		
Ingredient, %	Phase 1 (d 0 to 10)	Phase 2 (d 10 to 24)	Phase 3 (d 24 to 38)
Corn	30.14	50.19	56.84
Soybean meal (48% CP)	20.75	25.00	33.75
Whey, dried	20.00	10.00	—
Lactose	10.00	—	—
Animal plasma, spray-dried	5.00	2.50	—
Blood meal, spray-dried	2.50	2.50	—
Fish meal, menhaden	2.50	—	—
DL-Methionine	0.20	0.13	—
Dicalcium phosphate	1.53	2.11	2.37
Limestone	0.42	0.61	0.68
Salt	0.25	0.25	0.35
Zinc oxide	0.28	0.28	—
Copper sulfate	—	—	0.08
Trace mineral/vitamin mix[a]	0.30	0.30	0.30
Ethoxyquin	0.03	0.03	0.03
Antibiotic[b]	1.00	1.00	0.50
Soybean oil[c]	5.00	5.00	5.00
Cornstarch[d,e]	0.10	0.10	0.10
Calculated analyses			
CP, %	22.6	21.7	21.2
Lysine, %	1.6	1.4	1.2
Ca, %	0.90	0.90	0.85
P, %	0.80	0.80	0.75
ME, kcal/kg	3,364	3,373	3,402

[a]Provided the following per kilogram of diet: 120 mg of Zn as $ZnSO_4$; 120 mg of Fe as $FeSO_4$, 24 mg of Mn as MnO; 12 mg of Cu as $CuSO_4$; 0.36 mg of I as CaI; 0.30 mg of Se as Na_2SeO_3; 6,615 IU of vitamin A; 661 IU of vitamin D_3; 40 IU of vitamin E; 4.4 mg of vitamin K (menadione sodium bisulfate); 6.6 mg of riboflavin; 30 mg of d-pantothenic acid; 40 mg of niacin; 33 μg of vitamin B_{12}; 265 μg of d-biotin; 144 mg of choline; and 2 mg of folic acid.

[b]Phase 1 and 2 diets contained 110 mg of oxytetracycline and 154 mg of neomycin base per kilogram, and Phase 3 diets contained 220 mg of lincomycinper kilogram of complete diet.

[c]Soybean oil was replaced by cornstarch in Exp. 5 to provide the two diets containing no added fat.

[d]Cornstarch was replaced by L-carnitine (wt/wt) to provide supplemental dietary L-carnitine levels of 0, 25, 50, and 100 ppm in Exp 1.

[e]Cornstarch was replaced by L-carnitine (wt/wt) to provide a supplemental dietary concentration of 50 ppm in Exp. 5.

Table 2. Composition of basal diets for Exp. 2 (as-fed basis)

Ingredient, %	d 0 to 7[a]	d 7 to 14[a]	d 14 to 24[b]	d 24 to 34[b]
Corn[c]	43.67	45.25	52.42	59.16
Soybean meal (46.5% CP)	17.00	24.68	26.25	32.38
Dried whey	20.00	15.00	10.00	—
Animal plasma, spray-dried	5.00	2.50	—	—
Blood cells, spray-dried	2.50	2.50	2.50	—
Fish meal	2.50	—	—	—
Soybean oil	5.00	5.00	4.00	4.00
L-lysine HCl	0.15	0.15	0.15	0.15
DL-methionine	0.15	0.15	0.13	0.05
Monocalcium phosphate (21% P)	1.26	1.70	1.68	1.56
Limestone	0.79	0.99	0.97	0.95
Salt	0.20	0.30	0.25	0.35
Zinc oxide	0.38	0.38	0.25	—
Trace mineral premix[d]	0.15	0.15	0.15	0.15
Vitamin premix[e]	0.25	0.25	0.25	0.25
Antibiotic[f]	1.00	1.00	1.00	1.00
Calculated analyses				
CP, %	21.8	21.4	20.2	20.1
Lysine, %	1.6	1.5	1.35	1.25
Ca, %	0.90	0.90	0.85	0.80
P, %	0.80	0.80	0.75	0.70
ME, kcal/kg	3,457	3,431	3,391	3,438

[a]Fed in pelleted form.
[b]Fed in meal form.
[c]Corn was replaced by L-carnitine (wt/wt) to provide supplemental dietary L-carnitine levels of 0, 25, 50, 75, and 100 ppm.
[d]Trace mineral premix provided the following per kilogram of diet: Zn, 165 mg as ZnO; Fe, 165 mg as $FeSO_4$; 40 mg as MnO; Cu, 17 mg as $CuSO_4$; I, 0.30 mg as CaI; and Se, 0.30 mg as Na_2SeO_3.
[e]Vitamin premix provided the following per kilogram of diet: vitamin A, 11,000 IU; vitamin D_3, 1,650 IU; vitamin E, 40 IU; menadione, 4.4 mg (menadione sodium bisulfate); vitamin B_{12}, 44 μg; riboflavin, 10 mg; d-pantothenic acid, 33 mg; and niacin, 55 mg.
[f]Provided 55 mg carbadox/kg of complete diet.

38 for the determination of ADG, ADFI, and gain:feed (**G:F**).

Experiment 2. One hundred eighty-five pigs (Line C22, PIC, Franklin, KY; initially 5.6 kg of BW, 16 ± 2 d of age) were housed in an environmentally regulated nursery and randomly allotted to one of five dietary treatments. Pigs were allotted by weight in a randomized complete block design with eight replicate pens per treatment, five replications had five pigs per pen and three replications contained four pigs per pen. Pigs were fed either a basal diet (no added L-carnitine) or the basal diet with 25, 50, 75, or 100 ppm of L-carnitne to achieve the five dietary treatments (Table 2). Pigs were fed corn-soybean meal-based diets in four phases with decreasing diet complexity (amounts of spray dried whey and specialty protein sources) in each successive phase. From d 0 to 7, pigs were fed a complex diet formulated to contain 1.60% Lys. From d 7 to 14, pigs were fed a less complex diet containing 1.50% Lys. These first two diets were conditioned to 60°C and then pelleted through a 4-mm die. A corn-soybean meal-based diet containing 10% dried whey and 2.5% spray dried blood cells was formulated to 1.35% Lys and fed from d 14 to 24. The final diet, fed from d 24 to 34, contained no specialty protein sources and was formulated to 1.25% lysine. The last two diets were fed in meal form. Zinc oxide was fed at 3,000 ppm in the first two phases and at 2,000 ppm from d 14 to 24. All diets contained 4 to 5% SBO, 0.15% L-Lys HCl and 55 ppm of carbadox. L-Carnitine replaced corn in the diet to provide the desired carnitine concentrations in the diet. Each pen provided 1.44 m^2 of space and contained a stainless steel, three-hole feeder and a nipple waterer that allowed ad libitum access to feed and water. Room temperature was initially 32°C and lowered by 2.5°C every week to maintain pig comfort. Pigs and feeders were weighed on d 0, 7, 14, 24, and 34 to determine ADG, ADFI, and G:F.

Experiments 3 and 4. Two hundred pigs (initially 4.8 kg of BW, 12 ± 2 d of age) were used in a 38-d growth study in Exp. 3, and 240 pigs (initially 4.9 kg of BW, 12 ± 2 d of age) were used in a similar study in Exp. 4. In both experiments, pigs (L337 × C22; PIC) were allotted by weight in a randomized complete block design to each of five dietary treatments with five and six replicate pens per treatment in Exp. 3 and 4, respectively. In both studies, there were eight pigs per pen. The five dietary treatments (0, 25, 50, 75, and 100 ppm of L-carnitine; Table 3) were similar to those used in Exp. 2. Pigs were fed in four dietary phases. From d 0 to 4, pigs were fed a complex corn-soybean meal diet formulated to contain 1.70% Lys. From d 4 to 10, pigs were fed a transition diet formulated to 1.60% Lys. These first two diets contained 3,000 ppm of zinc oxide,

Table 3. Composition of basal diets for Experiments 3 and 4 (as-fed basis)

Ingredient, %	d 0 to 4[a]	d 4 to 10[a]	d 10 to 24[b]	d 24 to 38[b]
Corn	33.06	39.70	48.66	56.46
Soybean meal (46.5% CP)	12.71	23.01	27.33	34.29
Whey, dried	25.00	20.00	10.00	—
Lactose	5.00	—	—	—
Animal plasma, spray-dried	6.70	2.50	—	—
Blood meal, spray-dried	1.65	2.50	—	—
Fish meal	6.00	2.50	5.00	—
Soybean oil	6.00	5.00	5.00	5.00
Lysine HCl	0.15	0.15	0.15	0.15
DL-methionine	0.15	0.13	0.01	—
Monocalcium phosphate (21% P)	0.75	1.30	1.00	1.50
Limestone	0.45	0.73	0.55	0.95
Salt	0.20	0.30	0.25	0.35
Zinc oxide	0.38	0.38	0.25	—
Trace mineral premix[c]	0.15	0.15	0.15	0.15
Vitamin premix[d]	0.25	0.25	0.25	0.25
Antibiotic[e]	1.00	1.00	1.00	0.50
Cornstarch[f]	0.40	0.40	0.40	0.40
Calculated analyses				
CP, %	22.3	22.3	21.2	20.7
Lysine, %	1.7	1.6	1.4	1.35
Ca, %	0.90	0.90	0.85	0.80
P, %	0.80	0.80	0.75	0.70
ME, kcal/kg	3,535	3,444	3,491	3,504

[a]Fed in pelleted form.
[b]Fed in meal form.
[c]Trace mineral premix provided the following per kilogram of diet: 165 mg of Zn as ZnO; 165 mg of Fe as $FeSO_4$; 40 mg as MnO; 17 mg of Cu as $CuSO_4$; 0.30 mg of I as CaI; and 0.30 mg of Se as Na_2SeO_3.
[d]Vitamin premix provided the following per kilogram of diet: vitamin A, 11,000 IU; vitamin D_3, 1,650 IU; vitamin E, 40 IU; menadione, 4.4 mg (menadione sodium bisulfate); vitamin B_{12}, 44 μg; riboflavin, 10 mg; d-pantothenic acid, 33 mg; and niacin, 55 mg.
[e]Provided 55 ppm of carbadox from d 0 to 24 and 28 ppm from d 24 to 38.
[f]Cornstarch was replaced by L-carnitine (wt/wt) to provide supplemental dietary L-carnitine levels of 0, 25, 50, 75, and 100 ppm.

and were conditioned at 60°C before pelleting through a 4-mm die. From d 10 to 24, pigs were fed a less complex corn-soybean meal formulated to 1.40% Lys. For the remaining 14 d, pigs were fed a simple corn-soybean meal-based diet formulated to 1.35% Lys. The last two diets were fed in meal form. The first three diets contained 55 ppm of carbadox and the last diet contained 28 ppm carbadox. All diets contained 5 to 6% SBO and 0.15% L-lysine HCl. L-Carnitine replaced cornstarch in the diets to provide the L-carnitine levels in the diet.

Pigs in Exp. 3 and 4 were housed in an environmentally controlled nursery in 2.25-m^2 pens. The initial room temperature was approximately 32°C and was lowered by 2.5°C weekly. Each pen contained a stainless steel (six-hole divided trough) feeder and a nipple waterer to allow ad libitum access to feed and water. Pigs were weighed and feed disappearance was determined on d 0, 4, 10, 24, and 38 to calculate ADG, ADFI, and G:F.

Experiment 5. Two hundred sixteen crossbred (Yorkshire × Hampshire) pigs were weaned at 20 ± 2 d and utilized in a 38-d experiment. Pigs (5.8 kg initial BW) were used in a 2 × 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 of L-carnitine with either 0 or 5% SBO. The four dietary treatments were: 1) 0% SBO and 0 ppm of L-carnitine; 2) 0% SBO and 50 ppm of L-carnitine; 3) 5% SBO and 0 ppm of L-carnitine; and 4) 5% SBO and 50 ppm of L-carnitine. The composition of the basal diets was similar to those used in Exp. 1 (Table 1). There were 12 pens per treatment of four to five pigs per pen, and within a replicate, the number of pigs in each pen was the same. Pigs were fed in three dietary phases: Phase 1, d 0 to 10; Phase 2, d 10 to 24; and Phase 3, d 24 to 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, containing lactose, spray-dried animal plasma, spray-dried blood meal, and fishmeal, whereas Phase 3 (1.2% Lys) diets were typical corn-soybean meal based. All diets were fed in pelleted form. Additionally, pigs were housed in the temperature-controlled nursery rooms described for Exp. 1.

At the start of the experiment, blood samples (n = 48) were drawn via vena cava puncture from the two pigs closest to the mean pen weight from six pens per

treatment. Subsequent blood samples were taken at the end of each dietary phase and centrifuged to harvest the serum. Serum samples were then stored in a –20°C freezer until analysis.

Serum samples were allowed to thaw and then analyzed for serum urea N, glucose, NEFA, and triglycerides (COBAS FARA II clinical analyzer; Roche Diagnostic Systems, Indianapolis, IN). Determination of blood metabolite levels was made by colorimetric procedures (Roche Diagnostic Systems).

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 N determinations were performed by Kjeldahl methodology (FOSS Tecator, 2400 Kjeltec Analyzer unit, 2020 Digestor, Hoganas, Sweden). Also, diets were analyzed for L-carnitine concentrations (Metabolic Analysis Labs, Inc., Madison, WI) using methods described by Parvin and Pande (1977). Basal diets in Exp. 1, 2, and 5 were analyzed separately for L-carnitine, whereas basal diets from Exp. 3 and 4 were pooled before analysis.

Statistical Analysis

In Exp. 1, data were analyzed as a randomized complete block design. Due to unequally spaced dietary levels of L-carnitine for Exp. 1, coefficients were derived using the integrative matrix language (PROC IML) procedures of SAS (version 7.11; SAS Inst., Inc., Cary, NC). Data were analyzed as a randomized complete block design using ANOVA procedures (Steel et al., 1997). The model included the effects of block (rep), treatment, and block × treatment (error). The effects of increasing dietary L-carnitine concentrations were partitioned into linear and curvilinear components using orthogonal polynomial contrasts. In Exp. 2, 3, and 4, the data were analyzed as a randomized complete block design within each experiment using ANOVA procedures (Steel et al., 1997). The data from Exp. 2, 3, and 4 were pooled and analyzed using the PROC MIXED procedures of SAS using experiment and block as random effects (Littell et. al., 1996). The effects of increasing dietary L-carnitine concentrations were partitioned into linear and curvilinear components using orthogonal polynomial contrasts. Because performance data were collected at slightly different intervals for the three experiments, the response criteria were averaged into three time intervals (Phase 1, 2, and 3). In Exp. 2, Phase 1, 2, and 3 represent performance data from d 0 to 14, 14 to 24, and 24 to 34, respectively. In Exp. 3 and 4, Phase 1, 2, and 3 were from d 0 to 10, 10 to 24, and 24 to 38, respectively. In Exp. 5, data were analyzed as a 2 × 2 factorial in a randomized complete block design. 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 × SBO level interaction. In each experiment, pen served as the experimental unit.

The overall effects of carnitine were assessed by statistically analyzing the percentage change in G:F from the control diet. The percentage change in G:F was calculated for each block within each experiment by comparing the response of pigs fed 25, 50, and 100 ppm carnitine to pigs fed the control diet (no added carnitine) in Exp. 1 to 4, as these dietary treatments were common across these experiments. The means for percentage change from the control diet for G:F are based on 6, 8, 5, and 6 pen replicates per treatment in Exp. 1, 2, 3, and 4, respectively, for a total of 753 pigs. The percentage change was analyzed using the PROC MIXED procedure of SAS using experiment and block as random effects (Littell et. al., 1996). The effects of increasing dietary L-carnitine concentrations were partitioned into linear and curvilinear components using orthogonal polynomial contrasts. Pen served as the experimental unit.

Results

Experiment 1

The basal diets contained 38, 19, and <5 ppm of L-carnitine for Phases 1, 2, and 3, respectively. The analyzed concentrations of supplemented L-carnitine for the four dietary treatments were within 10% of their targeted levels. Analyzed values of CP were approximately 1.75% lower than the targeted values for each phase.

The effects of graded levels of L-carnitine on pig performance are shown in Table 4. For Phase 1, increasing concentrations of dietary carnitine did not affect ($P > 0.10$) ADG or ADFI, but carnitine tended to increase (linear, $P < 0.06$) G:F. During Phase 2, increasing dietary carnitine increased (linear, $P < 0.03$) ADG and G:F, but did not affect ($P > 0.10$) ADFI. Average daily gain and ADFI were not affected ($P > 0.10$) by supplemental carnitine during Phase 3; however, G:F tended to increase (quadratic, $P < 0.08$) with increasing carnitine concentrations during Phase 3. Overall, increasing levels of supplemental L-carnitine tended to improve (linear, $P < 0.09$) ADG for the 38-d experiment. Furthermore, for the entire 38-d period, G:F of pigs fed increasing carnitine concentrations plateaued (quadratic, $P < 0.02$) at 50 ppm, with no further increase in G:F noted with 100 ppm. The supplementation of L-carnitine had little affect ($P > 0.10$) on ADFI for the 38-d period.

Experiments 2, 3, and 4

In Exp. 2, the basal diets (no added carnitine) fed from d 0 to 7, 7 to 14, 14 to 24, and 24 to 34 contained 64.2, 38.8, 19.8, and 6.0 ppm of carnitine, respectively. In Exp. 3 and 4, the basal diets fed from d 0 to 4, 4 to 10, 10 to 24, and 24 to 38 contained 65.7, 38.7, 17.3, and <5 ppm of free L-carnitine, respectively. Analyzed

Table 4. Growth performance of weanling pigs in Experiment 1[a]

	Added L-carnitine, ppm					Probability, $P <$	
Item	0	25	50	100	SE	Linear	Quadratic
Phase 1, d 0 to 10							
ADG, g	137	136	165	159	13.5	0.18	0.51
ADFI, g[b]	173	166	185	178	10.2	0.55	0.77
Gain:feed	0.79	0.80	0.89	0.89	0.04	0.06	0.45
Phase 2, d 10 to 24							
ADG, g	342	359	381	377	11.0	0.03	0.16
ADFI, g[b]	467	468	489	477	11.2	0.44	0.35
Gain:feed	0.73	0.76	0.78	0.79	0.01	0.01	0.16
Phase 3, d 24 to 38							
ADG, g	479	491	511	494	15.8	0.49	0.27
ADFI, g[b]	781	782	791	815	25.1	0.31	0.79
Gain:feed	0.61	0.63	0.65	0.61	0.02	0.68	0.08
Overall, d 0 to 38							
ADG, g	337	347	370	363	10.8	0.09	0.22
ADFI, g[b]	503	502	516	523	14.1	0.26	0.99
Gain:feed	0.67	0.69	0.72	0.69	0.01	0.15	0.02

[a]Least squares means for six pens per treatment of four to six pigs per pen. There were 128 pigs initially 18 ± 4 d of age and 5.5 kg of BW.
[b]ADFI is reported on an as-fed basis.

L-carnitine values of the various treatment diets were within 10% of their expected calculated values for the amount of added carnitine. Crude protein values were also within 1.5 percentage units of their expected calculated values, although almost all values were slightly lower than their calculated values. Growth performance for Exp. 2, 3, and 4 are presented in Table 5. There were no experiment × treatment interactions ($P > 0.10$) observed with the exception of G:F during Phase 2. This interaction appeared to be the result of improved G:F with increasing added dietary L-carnitine in Exp. 2 and 3, whereas added L-carnitine did not improve G:F to the same magnitude in Exp. 4. Average daily gain, ADFI, and G:F were not affected ($P > 0.10$) by increasing L-carnitine during Phase 1. For Phase 2, pigs fed increasing L-carnitine had improved ADG (linear, $P < 0.04$) and G:F (quadratic, $P < 0.01$); however, ADFI was unaffected ($P > 0.10$). There were no effects ($P > 0.10$) of increasing L-carnitine on growth performance during Phase 3. Overall, pigs fed increasing L-carnitine tended

Table 5. Growth performance of weanling pigs in Experiments 2, 3, and 4[a]

	Added L-carnitine, ppm						Probability, $P <$	
Item	0	25	50	75	100	SE	Linear	Quadratic
Phase 1[b]								
ADG, g	205	213	210	212	201	9.4	0.68	0.19
ADFI, g[c]	226	239	234	236	227	9.7	0.95	0.16
Gain:feed	0.91	0.89	0.90	0.90	0.89	0.02	0.59	0.69
Phase 2[d]								
ADG, g	320	349	356	361	354	16.8	0.03	0.10
ADFI, g[c]	552	542	532	543	531	18.5	0.31	0.73
Gain:feed	0.59	0.66	0.68	0.68	0.68	0.02	0.001	0.001
Phase 3[e]								
ADG, g	559	566	548	592	578	22.7	0.19	0.79
ADFI, g[c]	821	811	833	875	841	31.5	0.14	0.74
Gain:feed	0.68	0.70	0.66	0.68	0.69	0.02	0.93	0.24
Overall								
ADG, g	364	382	375	393	383	12.3	0.08	0.32
ADFI, g[c]	537	534	537	554	540	17.5	0.50	0.83
Gain:feed	0.71	0.74	0.73	0.74	0.75	0.01	0.001	0.29

[a]Values represent means of 19 observations per treatment over three experiments. There was a total of 625 pigs, initially averaging 16 to 12 ± 2 d of age and 5.6 to 4.8 kg of BW.
[b]Phase 1 is from d 0 to 14 in Exp. 2 and d 0 to 10 in Exp. 3 and 4.
[c]ADFI is reported on an as-fed basis.
[d]Phase 2 is from d 14 to 24 in Exp. 2 and from d 10 to 24 in Exp. 3 and 4.
[e]Phase 3 is from d 24 to 34 in Exp. 2 and from d 24 to 38 in Exp. 3 and 4.

Table 6. Growth performance of weanling pigs in Experiment 5[a]

	Soybean oil, %							
	0		5					
	Carnitine, ppm					Probability, $P <$		
Item	0	50	0	50	SE	SBO	Carnitine	SBO × Carnitine[b]
Phase 1, d 0 to 10								
ADG, g	191	189	175	179	9.8	0.21	0.95	0.79
ADFI, g[c]	220	210	208	208	8.7	0.41	0.59	0.61
Gain:feed	0.86	0.89	0.84	0.86	0.02	0.28	0.32	0.79
Phase 2, d 10 to 24								
ADG, g	429	428	386	418	10.1	0.02	0.15	0.12
ADFI, g[c]	582	568	534	537	12.0	0.01	0.65	0.47
Gain:feed	0.74	0.75	0.75	0.78	0.01	0.07	0.03	0.50
Phase 3, d 24 to 38								
ADG, g	532	528	499	520	12.8	0.12	0.51	0.34
ADFI, g[c]	840	836	781	785	20.0	0.02	0.99	0.87
Gain:feed	0.64	0.62	0.64	0.66	0.01	0.01	0.42	0.15
Overall, d 0 to 38								
ADG, g	403	400	370	392	9.5	0.07	0.33	0.22
ADFI, g[c]	579	570	535	539	13.6	0.01	0.87	0.62
Gain:feed	0.69	0.70	0.70	0.72	0.01	0.02	0.03	0.10

[a]Least squares means for six pens per treatment of four to six pigs per pen. There were 216 pigs initially 20 ± 2 d of age and 5.8 kg of BW.
[b]Soybean oil level × L-carnitine level interaction.
[c]ADFI is reported on an as-fed basis.

to have increased (linear, $P < 0.07$) ADG. Gain:feed was also improved (linear, $P < 0.01$) when pigs were fed increasing levels of L-carnitine. Average daily feed intake was not affected ($P > 0.10$) by carnitine supplementation for the overall period.

Experiment 5

The analyzed carnitine concentrations for the basal diets were 37, 19, and 1 ppm, and carnitine concentrations for diets formulated to contain 50 ppm of added L-carnitine were 89, 72, and 49 ppm for Phases 1, 2, and 3, respectively. These analyses confirmed that the added levels of L-carnitine were close to the targeted increase in added L-carnitine (50 ppm). The GE of the diets was increased by approximately 200 to 225 kcal/kg, which is indicative of the addition of 5% SBO to the diet. Crude protein concentration of the diets was close to targeted levels.

The effects of L-carnitine and SBO on pig performance are shown in Table 6. During Phase 1, there were no effects ($P > 0.10$) of SBO or carnitine on growth performance. However, in Phase 2, SBO decreased ($P < 0.02$) ADG and ADFI, but it tended to improve ($P < 0.07$) G:F compared with pigs not fed SBO. Also, carnitine addition improved ($P < 0.03$) G:F. The only responses observed during Phase 3 were a reduction ($P < 0.02$) in ADFI and an increase ($P < 0.01$) in G:F associated with SBO addition. There were no SBO × carnitine interactions during any individual phase. For the entire 38-d study, pigs fed SBO tended to grow slower ($P < 0.07$) and consume less ($P < 0.01$) feed compared with those not fed SBO, but G:F was increased ($P < 0.02$) with SBO addition. The addition of L-carnitine improved ($P < 0.03$) G:F; however, it did not affect ($P > 0.10$) ADG or ADFI. Also, the increase in G:F associated with L-carnitine tended to be more pronounced in pigs fed SBO than those not fed SBO (L-carnitine × SBO, $P < 0.10$).

The effects of dietary L-carnitine and SBO on blood metabolites of weanling pigs are presented in Table 7. There were no differences ($P > 0.10$) in blood metabolites at the start of the experiment. The greatest response associated with L-carnitine and SBO occurred at the end of Phases 1 (d 10) and 2 (d 24). On d 10, pigs fed SBO tended to have higher triglyceride ($P < 0.08$) and NEFA ($P < 0.04$) levels than those not fed SBO. Also, the addition of L-carnitine tended to decrease ($P < 0.07$) NEFA levels on d 10; however, the decrease in NEFA levels tended be more pronounced in pigs fed SBO compared to those not fed SBO (L-carnitine × SBO, $P < 0.10$). On d 24, pigs fed SBO had lower ($P < 0.04$) serum urea N levels than those not fed SBO. However, when L-carnitine was added to the diet, a decrease in serum urea N levels was noted in pigs fed SBO, whereas an increase was observed in pigs not fed SBO (L-carnitine × SBO, $P < 0.01$). Responses to added SBO were also noted in serum urea N and NEFA levels at the end of Phase 3 (d 38). The supplementation of SBO tended to decrease ($P < 0.10$) serum urea N levels, whereas SBO increased ($P < 0.01$) NEFA levels of weanling pigs on d 38. There was no effect ($P > 0.10$) of SBO or L-carnitine on glucose concentrations.

Table 7. Blood metabolites of weanling pigs in Experiment 5[a]

	Soybean oil, %							
	0		5					
	Carnitine, ppm					Probability, $P <$		
Item	0	50	0	50	SE	SBO	Carnitine	SBO × Carnitine[b]
Serum urea N, mg/dL								
Day 10	6.13	7.43	7.24	6.14	1.07	0.51	0.29	0.83
Day 24	7.55	8.64	7.82	6.31	0.47	0.04	0.66	0.01
Day 38	12.62	12.66	11.98	11.09	0.63	0.10	0.51	0.47
NEFA, mmol/L								
Day 10	0.097	0.093	0.171	0.103	0.02	0.04	0.07	0.10
Day 24	0.089	0.121	0.107	0.113	0.02	0.77	0.26	0.46
Day 38	0.059	0.057	0.104	0.093	0.01	0.01	0.58	0.72
Triglycerides, mg/dL								
Day 10	33.2	32.3	36.5	38.8	2.6	0.08	0.79	0.56
Day 24	38.3	38.8	39.1	39.6	3.5	0.82	0.88	0.99
Day 38	43.3	36.1	41.8	49.1	5.0	0.27	0.99	0.17
Glucose, mg/dL								
Day 10	96.9	103.8	106.4	103.8	5.02	0.67	0.36	0.36
Day 24	110.9	107.1	107.8	104.9	4.52	0.47	0.57	0.92
Day 38	121.8	118.7	123.3	125.7	5.19	0.94	0.43	0.60

[a]Least square means for six pens per treatment of two pigs per pen. There were 216 pigs initially 20 ± 2 d of age and 5.8 kg of BW.
[b]Soybean oil level × L-carnitine level interaction.

Discussion

De novo synthesis of carnitine from protein-bound lysine and methionine appears to be sufficient to meet the metabolic needs of adult mammals (Rebouche and Seim, 1998). However, Borum et al. (1983) suggested that young neonates might not synthesize sufficient amounts of carnitine to meet metabolic needs. Although colostrum and sow's milk are good sources of carnitine for neonatal pigs, following weaning, the biosynthesis of carnitine is limited (Kerner et al., 1984). Given the role of carnitine in fatty acid metabolism, addition of carnitine to the diets of weanling pigs may affect the utilization of dietary fat via β-oxidation. An improvement in the carnitine status of weanling pigs via dietary addition, at a time when de novo synthesis of carnitine is limited, could possibly translate to an increase in growth performance during the initial nursery phase(s).

However, in our experiments, we observed no response to added carnitine in the period immediately after weaning, but rather in the period from approximately 10 to 24 d later. Because of limited carnitine synthesis shortly after weaning (Kerner et al., 1984), we would expect a greater response to dietary carnitine immediately after weaning compared with 2 to 4 wk later. On the other hand, this period happens to coincide with relatively high dietary carnitine concentrations from the added animal protein sources in the diet (Owen et al., 1996) and the relatively poor dietary fat absorption (Cera et al., 1988). Thus, the levels of carnitine in the basal diets for Phase 1 may have been great enough to meet the needs of the pig during this period of limited carnitine synthesis and relatively poor dietary fat absorption. In contrast to our results, others have reported a response to dietary L-carnitine supplementation immediately after weaning. Weeden et al. (1991) noted an improvement in ADG with the addition of 1,000 ppm of L-carnitine during the first 2 wk after weaning; however, supplemental L-carnitine did not alter feed intake or feed efficiency in their study. Also, an immediate postweaning response to added L-carnitine (500 ppm) was reported by Cho et al. (1999) since pigs fed L-carnitine grew faster and consumed more feed during the first 2 wk. It is worth noting that both Weeden et al. (1991) and Cho et al. (1999) had substantially greater levels of L-carnitine supplementation than those utilized in our experiments.

The responses observed during Phase 2 due to dietary L-carnitine are in agreement with results from several studies (Weeden et al., 1990; Owen et al., 1996; and Li et al., 1999). Li et al. (1999) reported that the addition of 50 ppm of L-carnitine increased ADG and feed consumption from 15 to 28 d postweaning. Also, when higher 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 L-carnitine on feed efficiency 3 to 5 wk after weaning.

As observed in Phase 1, minimal responses in growth performance due to the addition of L-carnitine were observed during Phase 3. We would hypothesize that the marginal response to added L-carnitine during Phase 3 is due to the increased biosynthesis of L-carnitine as the pig matures. By this age, L-carnitine synthe-

sis may be adequate and mask any response to supplemental dietary L-carnitine. It could also be that by this time, supplemental carnitine may not be affecting growth rate, but rather lipid and protein deposition. For example, Owen et al. (2001) observed that added L-carnitine did not affect finishing pig growth performance, but did decrease backfat and increased loin depth. Their hypothesis was that the finishing pig either synthesized enough carnitine or that the dietary concentrations were adequate for normal growth, but supplemental levels were necessary to affect carcass characteristics and intermediary metabolism.

The improvements in growth performance during Phase 2 resulted in improvements in ADG and G:F for the entire experiment. Our results are similar to several other experiments evaluating supplemental L-carnitine in nursery diets, which reported improvements in ADG and/or G:F (Newton and Haydon, 1988; Weeden et al., 1990; Owen et al., 1996; Li et al., 1999). Whereas most of the beneficial responses to carnitine appear to occur during the period 2 to 5 wk postweaning, it is not known, based on the present data, whether the positive response to carnitine for the overall nursery period would have been observed if carnitine had been supplemented to the diet only during Phase 2. Tissue carnitine concentrations in pigs increase in response to carnitine supplementation (Owen et al., 1996; Heo et al., 2000b) and liver carnitine levels of pigs fed vegetable-based diets have been reported to decrease to levels of that for neonatal pigs (Heo et al., 2000a). Thus, the increase in tissue carnitine concentrations associated with carnitine supplementation may have elicited changes in body composition during the entire experimental period, while only affecting growth performance during Phase 2. This assertion (that carnitine affects nutrient partitioning) is supported by the data of Heo et al. (2001), Owen et al. (2001a), and Rincker et al. (2001).

Heo et al. (2000a) reported that carnitine supplementation increased the activity of carnitine palmitoyltransferase-I in muscle and liver. This may imply an increase in available energy to the pig through an improvement in fatty acid oxidation. Considering that energy availability will limit protein accretion rate in young pigs, the improvements in G:F observed in the present experiments could be accounted for by this increase in available energy for protein accretion and/or growth. Recently, Owen et al. (2001a) found L-carnitine to increase palmitate oxidation and pyruvate carboxylase flux, while at the same time decreasing flux through branched-chain keto acid dehydrogenase in liver mitochondria. This would result in better lipid utilization for energy, as well as diverting carbon and branched-chain AA toward AA synthesis.

In a recent experiment conducted at our station (Rincker et al., 2001) using the same dietary treatments employed in Exp. 1, we reported a quadratic improvement in the rate (g/d) of protein accretion and an increase in the rate of protein:fat accretion with increasing (0 to 100 ppm) dietary carnitine concentrations. Also, Owen et al. (1996; 2001b) reported a decrease in daily fat accretion rates when pigs were fed increasing dietary carnitine during the nursery phase. Heo et al. (2000b) observed an improvement in daily N retention, an increase in daily protein accretion, and a decrease in carcass fat concentration when pigs were fed carnitine. These authors speculated that endogenous carnitine biosynthesis is sufficient to maintain tissue levels for growth, but that supplementing carnitine may alter nutrient partitioning, and thus, body composition. This hypothesis would help to explain the changes in body composition of pigs fed carnitine, as reported by Owen et al. (1996, 2001b) and Rincker et al. (2001). These reported changes in body composition (increased protein, decreased fat) might explain the improvements in G:F ratio observed in the present experiments.

Given the role of carnitine in fatty acid oxidation, the efficacy of carnitine supplementation may be enhanced in diets containing added fat. In Exp. 1 to 4, all diets contained 4 to 6% SBO; thus, the response to carnitine may have been dependent on the fat content of the diet. Owen at al. (1996) and Li et al. (1999) reported an improvement in growth performance due to carnitine, but there was no interaction between dietary carnitine level and SBO supplementation. Our results from Exp. 5 would tend to agree in that we observed an improvement in G:F due to the addition of 50 ppm of L-carnitine, and as observed in Exp. 1 to 4, the greatest response to L-carnitine was during Phase 2. Also, we did not observe an interaction between carnitine and SBO, with the exception that for the entire 38-d period, G:F was marginally improved to a greater extent when pigs were fed diets containing 5% SBO. Although not significant, we did observe greater numerical improvements in ADG and G:F when carnitine was added to diets containing 5% SBO vs. those not containing added fat. These subtle effects on growth performance may be an indication of an effect of carnitine on composition of body weight gain or nutrient utilization as reported by others (Owen et al., 1996; Heo et al., 2000b). In contrast to results from Exp. 5, Hoffman et al. (1993) reported that the addition of L-carnitine did not affect any performance criteria irrespective of dietary fat inclusion.

Similar to other reports (Mahan, 1991; Tokach et al., 1995), addition of SBO did not affect growth performance during Phase 1. However, during Phase 2 and for the overall experimental period, added SBO tended to decrease ADG, but it improved G:F in Phases 2 and 3 and during the overall period. In a review by Pettigrew and Moser (1991), these authors reported that fat addition to diets for weanling pigs resulted in a slight decrease in ADG, a decrease in ADFI, and an improvement in G:F. The reduction in ADG due to fat addition was more severe when diets were not adjusted to a constant protein:energy ratio. Our results from Exp. 5 agree with these results in that a slight reduction in ADG was observed with an improvement in G:F. The reduction in ADG in the current experiment may have been due to the diet containing fat having a lower

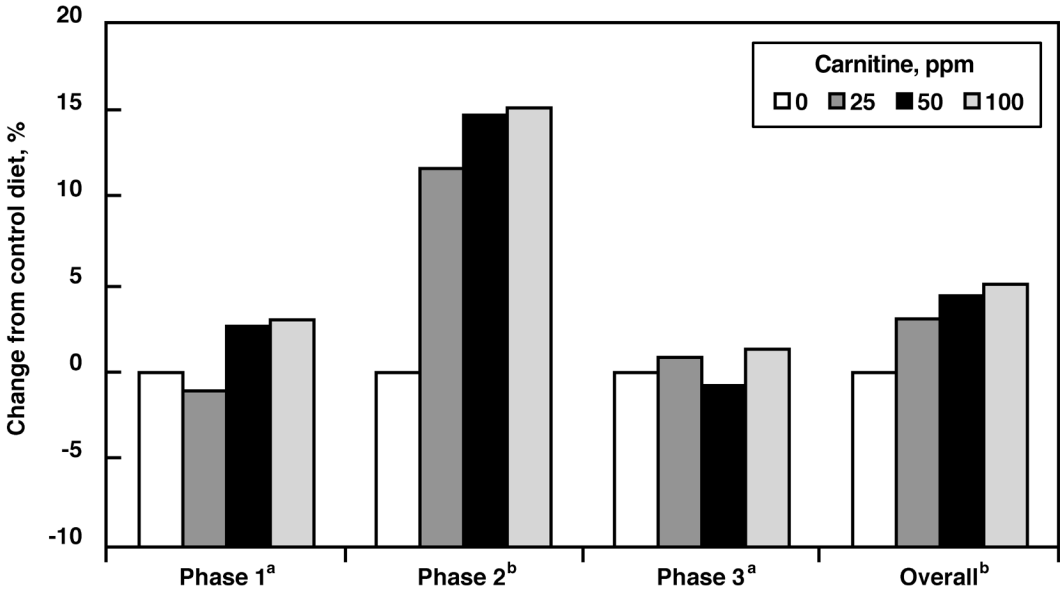

Figure 1. Percentage change in gain:feed from the pigs fed the basal diet of weanling pigs fed varying dietary concentrations of added L-carnitine based on 25 replicate pens per treatment (753 total pigs). SE = 2.05, 1.93, 1.93, and 0.90 for Phases 1, 2, 3, and overall, respectively. [a]No carnitine effects ($P > 0.10$). [b]Carnitine effect (quadratic, $P < 0.01$).

Lys:ME ratio, which, due to the reduction in feed intake, decreased Lys (and other AA) intake in pigs fed supplemental soybean oil. Although fat supplementation does not appear to improve growth performance of pigs from d 0 to 14 postweaning, added fat is necessary to pellet diets containing high quantities of milk products (Leaver, 1988).

The effects of carnitine on blood metabolites of weanling pigs were marginal. However, SBO tended to reduce serum urea N on d 24 and 38, which is similar to results reported by Owen et al. (1996). This response may indicate that energy for protein synthesis was limiting in the control diet and that SBO addition provided additional energy such that AA were utilized for protein synthesis. Furthermore, the decrease in serum urea N associated with SBO addition was more profound for pigs fed carnitine. Additionally, NEFA concentrations on d 10 were decreased to a greater extent when carnitine was added to diets containing SBO. Similar results (data not shown) due to carnitine were observed in Exp. 1 with diets containing SBO. The further decrease in fatty acids due to carnitine addition to diets containing SBO may be indicative of an increase in the catabolism of fatty acids via β-oxidation. The increase in available energy could then be used for protein synthesis, resulting in decreased urea N levels. These responses would correspond with Phase 2, where the effects of carnitine on growth performance were observed. Although speculative, the decrease in circulating fatty acid levels with a concurrent decrease in serum urea N would tend to agree with the model proposed for carnitine palmitoyltransferase-I activity by Heo et al. (2000b). These authors proposed that an increase in carnitine palmitoyltransferase-I activity, associated with carnitine supplementation, would result in an increase in β-oxidation of circulating fatty acids, resulting in an increase in metabolic energy available for protein synthesis.

As stated previously, other reports (Hoffman et al., 1993; Owen et al., 2001b) have found no effect of carnitine supplementation on the performance of weanling pigs regardless of whether the diet contained added fat. These conflicting results may be due to many factors, such as energy status of the pig (Heo et al., 2000b), age, health, environment, lean growth potential, diet composition, and nutrient concentration. However, in the current studies, which utilized similar diets conducted with differing genotypes and health statuses of pigs and differing environments at three distinct production sites, we consistently observed an improvement in G:F during Phase 2 with supplemental carnitine, which was of a large enough magnitude to improve overall G:F. Figure 1 shows the percentage change in G:F associated with carnitine supplementation compared with pigs fed no added carnitine. Supplementation of carnitine, averaged across experiments, resulted in a 4 to 5% improvement in G:F for the overall period. However, the greatest response occurred during Phase 2, where a 10 to 15% improvement in G:F was observed. During Phase 2 and overall, 50 ppm of added carnitine resulted in an improvement in G:F similar to that found with 100 ppm.

Implications

Results from the present studies utilizing pigs at three different locations suggest that the addition of L-carnitine to nutrient-dense diets enhances gain:feed of

weanling pigs by 10 to 15% approximately 2 to 4 wk after weaning. These improvements in growth performance carried over to result in an overall improvement in the efficiency of gain for the entire experiment. Although not conclusive, our results suggest that the response to added L-carnitine may depend on dietary fat inclusion level. These results suggest that relatively low concentrations (50 to 100 ppm) of L-carnitine elicit an improvement in gain:feed of weanling pigs consuming nutrient-dense diets containing soybean oil. However, because the response to L-carnitine supplementation preferentially occurred approximately 2 to 4 wk after weaning, our results indicate a greater need for L-carnitine during this period compared with the preceding and subsequent phases of the nursery period.

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