Effects of L-Carnitine Fed During Gestation and Lactation on Sow and Litter Performance[1,2]

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ABSTRACT: Multiparous sows (n = 307) were used to evaluate the effects of added dietary L-carnitine, 100 mg/d during gestation and 50 ppm during lactation, on sow and litter performance. Treatments were arranged as a 2 (gestation or lactation) × 2 (with or without L-carnitine) factorial. Control sows were fed 1.81 kg/d of a gestation diet containing .65% total lysine. Treated sows were fed 1.59 kg/d of the control diet with a .23 kg/d topdressing of the control diet that provided 100 mg/d of added L-carnitine. Lactation diets were formulated to contain 1.0% total lysine with or without 50 ppm of added L-carnitine. Sows fed 100 mg/d of added L-carnitine had increased IGF-I concentration on d 60 (71.3 vs 38.0 ng/mL, $P < .01$) and 90 of gestation (33.0 vs 25.0 ng/mL, $P = .04$). Sows fed added L-carnitine had increased BW gain (55.3 vs 46.3 kg; $P < .01$) and last rib fat depth gain (2.6 vs 1.6 mm; $P = .04$) during gestation. Feeding 100 mg/d of added L-carnitine in gestation increased both total litter (15.5 vs 14.6 kg; $P = .04$) and pig (1.53 vs 1.49 kg; $P < .01$) birth weight. No differences were observed in pig birth weight variation. Added L-carnitine fed during gestation increased litter weaning weight (45.0 vs 41.3 kg, $P = .02$); however, no effect of feeding L-carnitine during lactation was observed. No differences were observed in subsequent days to estrus or farrowing rate. Compared to the control diet, feeding added L-carnitine in either gestation, lactation, or both, increased ($P < .05$) the subsequent number of pigs born alive, but not total born. In conclusion, feeding L-carnitine throughout gestation increased sow body weight and last rib fat depth gain and increased litter weights at birth and weaning.

Key Words: Sows, Carnitine, Pregnancy, Lactation

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Introduction

L-carnitine is essential in the transportation of long- and medium-chain fatty acids across the mitochondrial membrane for β-oxidation. L-carnitine is biosynthesized from trimethyl lysine. Trimethyl lysine is derived from lysine liberated during intracellular hydrolysis, then lysine is methylated by S-adenosyl methionine (Brody, 1994). In addition, several cofactors are involved such as ascorbate, niacin, vitamin B_6, and iron. Previous research (Owen et al., 1993) demonstrated that the addition of L-carnitine decreases lipid accretion in growing-finishing pigs. L-carnitine has also been shown to affect several key enzymes involved in protein and lipid metabolism (Rebouche et al., 1990; Owen et al., 1997). Because of these effects on key metabolic enzymes, we speculate that L-carnitine may enhance productivity of the gestating and lactating sow. Fremaut et al. (1993) reported that sows fed 250 mg/d of L-carnitine in lactation had decreased preweaning mortality and increased (.3 kg) pig weight at weaning (d 30). However, addition of dietary L-carnitine had mixed effects on sow and pig performance, comparing research from Europe with that from the United States. Harmeyer (1993) observed that sows fed 50 ppm of supplemental L-carnitine during lactation weaned heavier pigs than control sows. In contrast, Musser et al. (1999) observed no differences in pig weaning weights with the addition of 50, 100, or 200 ppm of L-carnitine fed during lactation. Although limited data exist on the effects of added L-carnitine during lactation, no research has been conducted to determine its possible effects during gestation. Therefore, the objective of this

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experiment was to determine whether additional dietary L-carnitine during gestation and(or) lactation would improve sow and litter performance.

Materials and Methods

Animals and Housing. Sows (n = 307 PIC C15 sows bred to 326 boars) were used to determine the effects of 100 mg/d of added L-carnitine from d 5 to 112 of gestation and 50 ppm of added L-carnitine during lactation on sow and litter performance. The experiment was conducted from June to December 1996 on a 1,400-sow commercial swine farm in Northeast Kansas. Sows were housed in crates (2.13 × .61 m) in a curtain-sided breeding barn until 5 d after breeding. Sows were then placed in crates (2.13 × .61 m) on d 5 of gestation in an environmentally regulated (minimum of 15.6°C), curtain-sided gestation barn from d 5 to 112 of gestation. At breeding, sows were weighed, ultrasonically scanned (Renco, Minneapolis, MN) for last rib fat depth, and then randomly allotted to one of two dietary treatments. The gestation diet was formulated to contain .65% total lysine, .95% Ca, and .85% P; all other amino acids, vitamins, and minerals exceeded NRC (1988) requirement estimates (Table 1). Sows were fed either 1.81 kg/d of the control diet (no added L-carnitine) or 1.59 kg of the control diet and .23 kg of the same diet containing 435 ppm L-carnitine. Sows were fed at 0800 daily. Sows were allowed access to water from 2 h after feeding until 0800 the next morning. Sows were weighed, and last rib fat depth was recorded on d 112 of gestation, at which time sows were placed in crates (2.13 × .61 m) in an environmentally regulated farrowing facility and allotted to lactation treatments.

Table 1. Diet composition (as-fed basis)

Ingredient, %	Gestation[a]	Lactation[b]
Grain sorghum	79.81	62.92
Soybean meal (46.5% CP)	15.22	28.41
Soybean oil	—	4.00
Monocalcium phosphate	2.51	2.33
Limestone	1.11	1.12
Salt	.50	.50
Sow premix[c]	.25	.25
Vitamin premix[d]	.25	.25
Trace mineral premix[e]	.15	.15
Medication[f]	.20	—
Vitamin E[g]	—	.05
DL-Methionine	—	.02

[a]Gestation feeding levels of 1.81 kg/d, with or without a topdressing providing 100 mg/d added L-carnitine. Analyzed L-carnitine content was 2.5 and 478 ppm for the control diet and the L-carnitine topdressing, respectively. The gestation diet was formulated to 14.29% CP, .65% lysine, .95% Ca, and .85% P.

[b]Sows were provided ad libitum access to feed, with or without 50 ppm of added L-carnitine during lactation. Analyzed L-carnitine content was 4.6 and 48 ppm for the control and L-carnitine diets, respectively. The lactation diet was formulated to 18.84% CP, 1.0% lysine, .95% Ca, and .85% P.

[c]Supplied per kilogram of diet: 386 mg of choline, .22 mg of d-biotin, and 1.65 mg of folic acid.

[d]Supplied per kilogram of diet: 11,025 IU of vitamin A, 1,654 IU of vitamin D_3, 44.1 IU of vitamin E, 4.4 mg of menadione sodium bisulfite, 8.3 mg of riboflavin, 28.7 mg d-pantothenic acid (as d-calcium pantothenate), 49.6 mg of niacin, 165.4 mg of choline, and .03 mg of vitamin B_{12}.

[e]Supplied per kilogram of diet: 39.7 mg of Mn (oxide), 165.4 mg of Fe (sulfate), 165 mg Zn (oxide), 16.5 mg of Cu (sulfate), .30 mg of I (as Ca Iodate), and .30 mg of Se (as Na selenite).

[f]Provided 50 mg of oxytetracycline/kg of complete feed.

[g]Provided an additional 22.05 IU of vitamin E/kg of complete feed.

From d 112 of gestation to farrowing, sows were fed 1.81 kg/d of the experimental lactation diets. After farrowing, sows were offered feed three times each day to maximize feed intake. The diet was formulated to contain 1.0% total lysine, .95% Ca, and .85% P, with or without 50 ppm of added L-carnitine. All other amino acids, vitamins, and minerals were calculated to be in excess of NRC (1988) requirement estimates (Table 1).

Diet samples were collected and analyzed for free carnitine concentration (Parvin and Pandle, 1977). The analysis of the feed samples measured free carnitine from a neutralized perchloric acid extract from finely ground feed samples. Each sample was extracted twice, and each extract was analyzed for free carnitine three times and averaged. The allowed variation for this procedure was 10%. Chemical analysis indicated that analyzed L-carnitine concentrations were similar to calculated values (Table 1).

At farrowing, the number of pigs born alive, born stillborn, and mummified were recorded. Pigs were ear-notched for identification, and individual pig birth weights were recorded. All litters were equalized to approximately 10 pigs/litter within gestation treatment by d 2 of lactation. Individual pig weight and number of pigs per litter were recorded at weaning (d 15), at which time sow weight and last rib fat depth were recorded. Sow feed intake was measured daily then averaged to determine daily feed intake for 7-d intervals throughout lactation. After weaning, and throughout the subsequent gestation, sows were fed a diet with no added L-carnitine. Sows were monitored once daily with a boar for estrus detection. If a sow did not return to estrus within 35 d, she was culled. Sows were also culled for reasons of injury, age, and genetic turnover. Subsequent farrowing rate (not including culled sows), total number of pigs born, and number born alive were also determined.

Blood Samples and Analysis. Sows were bled via vena cava puncture 6 h after feeding on d 10, 60, 90, and 110 of gestation and at weaning (d 15). In addition, 15 pigs per treatment (no more than one pig per litter, and each pig was of average weight within the litter) were selected from random litters and bled at weaning. Blood samples were collected in heparinized tubes and were placed on ice until they were centrifuged at 4,000 × *g* for 30 min at 5°C. Plasma was harvested and stored at –15°C. Plasma concentrations of insulin, total IGF-I, free carnitine, and total carnitine were determined.

The analysis of free and total plasma carnitine used a modified technique of Minkler and Hoppel (1993). Briefly, the analysis used 100 μL of plasma from which carnitine was extracted with acetonitrile/methanol.

Carnitine was then isolated using a silica gel column. Samples then were derivatized with 4′-bromophenacyl trifluoromethanesulfonate and quantified using HPLC. The HPLC relied on detection of the compounds using an ultraviolet detector set to measure light absorbency at 260 nm.

Plasma IGF-I concentrations were determined using a two-sided immunoradiometric assay (**IRMA**) provided in a coated-tube kit (ActiveTM IGF-I with Extraction, DSL-5600, Webster, TX). The kit uses a modified version of the standard acid-ethanol procedure. In the assay, IGF-I is separated from its binding protein for determination of total IGF-I. The IRMA is a noncompetitive assay in which the unknown is held between two antibodies. The first antibody is immobilized to the inside of the wall of the tube, and the second antibody is radiolabeled with ^{125}I for detection. Results are calculated using a log-log curve fit, and net counts per minute (**cpm**) for the control and standards are used to calculate the IGF-I concentration of the unknown samples from their respective cpm. Assay sensitivity was .02408 ng/mL, and the intraassay CV was 3.01%.

Insulin plasma concentrations were determined using a coated-tube kit (Coat-A-Count Insulin, DPC, Los Angeles, CA). In the Coat-A-Count Insulin procedure, [^{125}I]insulin competes with insulin in the sample for sites on insulin-specific antibody immobilized to the wall of a polypropylene tube. After incubation, isolation of the antibody-bound fraction is achieved simply by decanting the supernatant. The radioactivity in this tube is measured in a gamma counter; the cpm are inversely related to the amount of insulin present in the sample. The quantity of insulin in the sample is then determined by comparing the cpm to a standard curve. When .104, .312, 1.04, 2.08, and 4.16 insulin ng/mL was added to 100 μL of zero calibrator, recovery averaged 107%. To determine parallelism of the analysis, various volumes of pig serum (100, 150, and 200 μL) were analyzed and reported levels of insulin of 1.93, 1.98, and 1.05 ng/mL, respectively. Sensitivity of the assay was 2 μIU/mL, and intraassay CV was 4.9%.

Statistical Analysis. Data were analyzed with ANOVA using the GLM procedure of SAS (1988). Treatments were arranged in a split-plot design; the whole plot included added L-carnitine fed during gestation, and the subplot included added L-carnitine fed during lactation. Sow body weight at breeding was used as a covariate for the analysis of sow weight on d 112 and gestation BW gain. Last rib fat depth at breeding and sow weight on d 112 were used as covariates to determine the effects of added L-carnitine fed during gestation on last rib fat depth on d 112 and last rib fat depth change during gestation. Variation in pig birth weight within treatment was analyzed using Levene's test (Milliken and Johnson, 1984). Briefly, this calculated the residual for each observation (absolute value of the differences between the actual pig birth weight and the litter mean birth weight). Smaller residual means would indicate less variation of pig birth weights within litter. Values reported are least squares means with or without the specified covariates.

Results

Gestation. Sows fed 100 mg/d of added L-carnitine had greater BW gain ($P < .01$) and last rib fat depth ($P < .04$) gain during gestation (Table 2). At farrowing, sows fed L-carnitine had increased pig ($P < .01$) and litter ($P < .04$) birth weights. No differences between dietary treatments were observed in the variation of pig birth weights within litters (residual birth weight, $P > .10$).

There were no differences between treatments in total number of pigs born, number born alive, or mummies; however, sows fed L-carnitine had decreased numbers of stillborn pigs per litter ($P < .02$). Nonetheless, the differences in number of stillborn pigs did not affect the total number of pigs born alive.

Lactation. No gestation × lactation interactions were observed ($P > .10$), except for pig survivability ($P < .10$) and last rib fat depth of sows at weaning ($P = .05$; Table 3). No differences were observed in sow or litter criteria for sows fed added L-carnitine during lactation. Sows fed added L-carnitine during gestation had increased pig ($P < .01$) and litter ($P < .07$) weaning weight compared with sows fed the control diet. Both pig and litter weight gain throughout lactation tended ($P = .03$ and $P = .12$, respectively) to be increased for pigs from sows fed added L-carnitine during gestation. Sows fed L-carnitine in gestation were heavier at weaning than control sows ($P < .01$; Table 3).

Feeding added L-carnitine during gestation and(or) lactation had no effect on the subsequent days to estrus or farrowing rate ($P > .10$; Table 4). No differences were observed in subsequent total number of pigs born, but added L-carnitine in gestation and(or) lactation increased the number of pigs born alive ($P < .05$).

Laboratory Analysis. Plasma insulin concentrations were increased on d 10 and 60 of gestation in sows fed added L-carnitine compared with sows fed the control diet ($P = .07$; Table 5). The IGF-I concentrations were increased on d 60 and 90 in sows fed added L-carnitine ($P < .05$). No differences were observed in plasma insulin or IGF-I from blood samples collected from pigs at weaning ($P > .10$).

Plasma free carnitine concentrations were increased ($P < .01$; Table 5) on d 60 and 90 of gestation for sows fed additional L-carnitine. Similarly, total plasma carnitine concentrations on d 60, 90, and 110 were numerically higher for sows fed L-carnitine, but only the difference found on d 90 reached significance ($P < .02$).

Discussion

The addition of L-carnitine to the gestation diet seemed to improve the utilization of dietary nutrients by the sows resulting in increased sow weight, last rib fat depth, and litter birth weight. The increase in sow

Table 2. Effects of L-carnitine on gestation performance

Item	Control	L-carnitine	$P <$	SEM
No. of sows	155	153		
Average sow parity	3.81	3.67	.42	.13
Sow weight, kg				
Breeding	182	185	.58	2.41
d 112[a]	230	239	.01	.85
Change[a]	46.5	55.4	.01	1.03
Sow last rib fat depth, mm				
Breeding	15.9	15.8	.75	.29
d 112[b]	17.5	18.4	.02	.25
Change[c]	1.6	2.6	.04	.25
Litter weight at 24 h, kg[d]	14.65	15.45	.09	.33
Pig weight at 24 h, kg[d]	1.48	1.58	.01	.02
Residual birth weight per pig, kg[d]	.29	.30	.12	.01
No. of pigs per litter				
Total born	11.28	11.11	.62	.26
Born alive	10.33	10.43	.75	.23
Stillborn	.76	.49	.02	.09
Mummies	.17	.14	.64	.04
Pigs alive at 24 h	10.08	9.98	.78	.23

[a]Means were adjusted for sow weight at breeding by covariate analyses.
[b]Means were adjusted for sow last rib fat depth at breeding by covariate analyses.
[c]Means were adjusted for sow weight at d 112 by covariate analyses.
[d]Means were adjusted for parity by covariate analyses.

Table 3. Effects of L-carnitine on lactation performance

		Dietary treatment				Probability ($P <$)			
Item	Gestation: Lactation:	Control Control	Control Carnitine	Carnitine Control	Carnitine Carnitine	Gest.	Lact.	Gest. × Lact.	SEM
No. of sows		75	75	86	58				
Parity		3.72	3.82	3.64	3.77	.63	.43	.92	.13
Lactation length, d		15.7	15.9	15.3	15.7	.15	.30	.62	.16
Sow weight, kg									
d 112		228	230	239	243	.01	.43	.79	3.1
Weaning[c]		224	221	232	238	.01	.61	.29	3.3
Change during lactation[c]		–2.7	–3.2	–5.6	–5.4	.14	.97	.85	1.4
Sow last rib fat depth, mm									
d 112		17.54	17.55	18.36	17.31	.58	.32	.31	.48
Weaning[c]		16.25	16.44	18.36	16.12	.13	.09	.05	.51
Change during lactation[c]		–1.34	–.72	–.09	–1.24	.42	.54	.09	.48
Average daily feed intake, kg									
wk 1		5.31	5.24	5.22	5.39	.73	.64	.28	.09
wk 2		6.51	6.54	6.67	6.74	.11	.64	.84	.10
Overall		6.00	5.90	5.99	6.16	.20	.63	.12	.08
Litter weight, kg									
Birth		14.29	14.84	15.63	15.64	.03	.55	.57	.44
Weaning		41.14	41.68	44.30	44.91	.07	.75	.99	1.55
Pig weight, kg									
Birth		1.46	1.51	1.56	1.60	.01	.17	.81	.03
Weaning[a]		4.68	4.71	4.96	4.98	.01	.79	.99	.08
Litter weight gain, kg[a]		26.62	26.57	28.49	29.14	.12	.84	.81	1.23
Pig weight gain, kg[a]		3.21	3.22	3.41	3.38	.03	.91	.76	.07
Pigs per litter									
d 2		9.98	10.09	10.20	10.01	.85	.91	.67	.32
Weaned[b]		8.91	8.89	9.02	9.00	.76	.96	.99	.31
Survivability, %[b]		89.57	86.08	86.87	90.45	.69	.98	.10	1.84

[a]Means were adjusted for pig weight on d 2 by covariate analyses.
[b]Means were adjusted for pigs per litter on d 2 by covariate analyses.
[c]Means were adjusted for weight and last rib fat depth on d 112 by covariate analyses.

Table 4. Effect of L-carnitine on subsequent reproductive performance

Item	Gestation: Lactation:	Dietary treatment: Control Control	Control Carnitine	Carnitine Control	Carnitine Carnitine	Probability ($P <$): Gest.	Lact.	Gest. × Lact.	SEM
No. of sows returning to estrus		47	44	55	37				
No. of sows removed[a]		28	31	31	21				
Days to estrus		5.28	5.82	6.11	5.37	.64	.80	.12	.38
Farrowing rate of sows returning to estrus, %		96.1	96.3	86.5	93.2	.22	.51	.54	.05
Total born/litter[b]		11.24	12.26	11.97	12.85	.21	.09	.90	.40
Born alive/litter		10.15	11.22	11.17	12.03	.04	.05	.83	3.46

[a]Sows were removed for injury, no estrus by d 35, or age.
[b]Including pigs born alive, mummified, and stillborn per litter.

last rib fat depth gain in gestation was unpredicted because of previous research (Owen et al., 1997) with finishing pigs. Owen et al. (1997) reported that increasing dietary L-carnitine increased fatty-acid oxidation and resulted in leaner finishing pigs. However, a confounding factor between our results and those of Owen et al. (1997) is that our sows were limit-fed, whereas their finishing pigs had ad libitum access to feed. The finishing pig is in a state of decreasing protein deposition and increasing lipid deposition as BW increases (Smith et al., 1997), with a small proportion of energy and amino acids used for maintenance. However, gestating sows have a much larger maintenance requirement relative to that for growth (Close et al., 1985; Noblet and Etienne, 1987). After meeting nutrient demand for maintenance, the remaining nutrients are used for fetal growth and for replacement of tissue protein and lipid reserves lost during the previous lactation (Tokach et al., 1996). Therefore, the partitioning of nutrients is indeed different for the growing-finishing pig than it is for gestating sows, and these differences could help to explain the dissimilar effect of L-carnitine additions.

If L-carnitine increases β-oxidation in gestating sows as in newborn and finishing pigs (Owen et al., 1997), this may allow for an enhanced oxidation of dietary triglycerides (Coffey et al., 1991). However, because gestating sow diets are considerably lower in fat than finishing pig diets, other factors may influence fat deposition.

The mechanisms behind the observed increases in litter and pig birth weights are unclear. Britt (1986)

Table 5. Effects of L-carnitine during gestation on plasma IGF-I, insulin, and total and free carnitine plasma concentrations[a]

Item	Control	L-carnitine	$P <$	SEM
No. of sows	14	14		
Sow IGF-I, ng/mL				
d 10	72.06	78.36	.82	12.24
d 60	37.95	71.25	.01	8.17
d 90	24.98	33.02	.04	2.71
Pig IGF-I at weaning[b]	108.17	119.00	.69	18.92
Sow insulin, ng/mL				
d 10	.3727	.6300	.07	.053
d 60	.5138	.8167	.07	.110
d 90	.5451	.5000	.66	.072
Pig insulin at weaning[b]	.2638	.2227	.41	.031
Free carnitine from sow plasma, nmol/mL				
d 10	23.70	23.12	.84	1.15
d 60	15.30	19.16	.01	1.27
d 90	22.74	27.12	.01	1.02
d 110	29.29	30.97	.42	1.54
Total carnitine from sow plasma, nmol/mL				
d 10	27.60	26.32	.70	1.38
d 60	20.02	22.54	.11	1.63
d 90	26.63	31.29	.02	1.24
d 110	33.72	36.84	.25	1.98

[a]Analysis of sows by gestation treatment.
[b]Analysis of plasma from 15 pigs at weaning from sows fed either control or additional L-carnitine throughout gestation and lactation.

reported that increased energy intake during the last 3 wk of gestation resulted in increases in birth weight similar to those observed in the current study. If the addition of L-carnitine increases feed utilization, then the response could mimic the effect of added feed. It is possible that the increase in sow weight gain in response to the addition of L-carnitine also produced an increase in fetal weight gain. However, Harmeyer (1993) fed additional L-carnitine from 4 wk prior to farrowing until weaning and observed no differences in pig birth weight.

We observed a decrease in the number of stillborn pigs from feeding L-carnitine during gestation, with no differences in total or live pigs per litter. Fremaut et al. (1993) found no differences in the number of stillborn pigs from sows fed 250 mg/d of added dietary L-carnitine from d 104 of gestation until weaning. The percentage stillborn in control litters was lower in the study of Fremaut et al. (1993) than in the current experiment, even though lactation treatments started at a similar time (d 104 and 112 of gestation, respectively).

The ability to alter preweaning survival with the supplementation of L-carnitine has been reported by Fremaut et al. (1993). The decrease in preweaning mortality for both of experiments in Fremaut et al. (1993) was approximately 3 percentage units in sows that received added L-carnitine compared with control sows (12.2 vs 9.5%). In the current experiment, no differences were observed in preweaning mortality. However, increased birth weight might be expected to increase preweaning survivability.

Research has implicated IGF-I's importance in myogenic differentiation and proliferation (Florini et al., 1991; Magri et al., 1991). The actions of IGF-I may enhance muscle fiber development, resulting in improved postnatal growth in offspring (Wigmore and Stickland, 1983). Increases in maternal insulin and IGF-I concentrations were observed in the current experiment at a period of gestation that coincides with fetal secondary muscle fiber development (Wigmore and Stickland, 1983). Although L-carnitine fed during gestation increased insulin and IGF-I concentrations, there is no research to demonstrate the possible effects on fetal muscle development. Additional research is needed to determine the effects of various inclusion rates of L-carnitine in gestating sow diets on fetal development.

Owen et al. (1997) reported that added dietary L-carnitine fed to finishing pigs increased plasma free and total carnitine concentrations. Sows fed added L-carnitine had significantly higher plasma free and total carnitine concentrations on d 60 and 90 of gestation, although not different on d 10 and 110, compared to control sows.

Although no differences were observed in the total number of pigs born or born alive in litters farrowed during the actual treatment period, an increase ($P < .05$) in pigs born alive was observed in the subsequent litters. A possibility for the increase is the observation earlier in gestation of an increase in maternal plasma insulin and IGF-I concentrations. Cox et al. (1987) observed that administration of exogenous insulin (.1 IU/d) increased ovulation rate. Improvements in subsequent number of pigs born alive could be a reflection of improved nutritional status of the developing embryo in early gestation. Nutrient uptake and many of the metabolic activities that provide for fetal growth and development in early gestation are controlled by the placenta. Research has shown that, even though triglycerides do not cross the placenta (Knopp et al., 1986), free fatty acids are taken up and utilized (Campbell et al., 1995). If additional L-carnitine would increase β-oxidation of fatty acids in the placenta, as it does in finishing pigs (Owen et al., 1997), it might result in a high rate of gluconeogenisis from substrates and, thus, improve cellular energy charge and increase glycogen concentration (Nishida et al., 1989). An increase in L-carnitine to the placenta might improve the metabolic status of the fetus by increased glucose uptake, as it does in newborn pigs (Nishida et al., 1989), and possibly improve early embryonal survival.

If tissue reserves of carnitine were maintained during lactation, an increase in insulin and IGF-I would likely result. Cox et al. (1987) observed an increase in LH secretion with the infusion of insulin, resulting in an increased ovulation rate. Flushing is a realimentation of gilts previously restricted in feed intake for 11 to 14 d prior to estrus (Anderson and Melampy, and 1971) researchers have found that flushing improves ovulation rates of gilts. Increases in insulin and IGF-I might stimulate LH surges (Rhodes, 1986; Britt et al., 1987; Booth, 1990). Even though flushing treatments have been found to be more beneficial in gilts than in later-parity sows, our increase was observed in sows. If additional dietary L-carnitine increases insulin and IGF-I during lactation similar to the increase during gestation, then effects on LH and, therefore, ovulation rate could be expected.

Implications

Feeding 100 mg/d of L-carnitine during gestation increased sow body weight gain and backfat thickness, indicating that supplemental L-carnitine improved nutrient utilization by the sow. In contrast, 50 ppm of supplemental L-carnitine in the lactation diets did not affect either sow weight change or pig weaning weights. Further research is needed to determine the optimum level of L-carnitine needed in gestation diets and to determine the mechanism whereby added L-carnitine improves nutrient utilization in the gestating sow.

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