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Influence of dietary L-carnitine and chromium picolinate on blood hormones and metabolites of gestating sows fed one meal per day[1,2]

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ABSTRACT: Gestating sows (n = 44; parity = 2.0; BW = 208 kg) were used to determine the effects of dietary L-carnitine and Cr picolinate (CrP) on daily blood hormone and metabolite profiles. Diets were formulated as a 2 × 2 factorial with L-carnitine (0 or 50 ppm) and CrP (0 or 200 ppb) and were fed from breeding through gestation, lactation, and 28 d into the subsequent gestation, at which time blood collection occurred. Sows were fed 1 meal per day during gestation (2.04 kg from breeding until d 100 and 2.95 kg from d 100 until farrowing) and ad libitum during lactation. Sows were fitted with indwelling venous catheters, and blood (plasma) was collected at feeding, then once every 15 min for the first 3 h after feeding, and at 6, 9, 15, 20, and 24 h after feeding. Postfeeding and overall insulin and connecting peptide of insulin (c-peptide) was decreased for sows fed diets with CrP or L-carnitine and was greatest for sows fed the control diet; however, sows fed both L-carnitine and CrP had an intermediate response (L-carnitine × CrP, $P < 0.01$). Postfeeding glucose peak was decreased ($P < 0.05$) in sows fed diets with L-carnitine, CrP, or both, vs. the control, and mean glucose concentration was decreased ($P < 0.01$) for sows fed diets with CrP. L-Carnitine decreased ($P < 0.04$) the NEFA concentration. Sows fed diets with CrP exhibited increased ($P < 0.03$) postfeeding and overall NEFA and greater ($P < 0.02$) fasting and overall glycerol. Overall plasma urea N was lowest for sows fed the diet with L-carnitine; however, diets containing CrP had intermediate responses compared with the control (L-carnitine × CrP, $P < 0.005$). Sows fed diets with L-carnitine had greater ($P < 0.008$) IGF-I from 3 to 24 h after feeding and tended to exhibit greater ($P < 0.06$) overall IGFBP-3. Sows fed the diets with CrP had greater ($P < 0.05$) IGFBP-3 from 2 to 20 h after feeding. No differences were observed for glucagon or triacylglycerol ($P > 0.10$). The changes in metabolites and metabolic hormones indicate that both L-carnitine and CrP influence energy metabolism of gestating sows; however, their effects on blood hormones and metabolites differ. Thus, the improvement in energy status from adding both L-carnitine and CrP may have an additive effect on reproductive performance of sows.

Key words: sow, carnitine, chromium, hormonal regulation, metabolite

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INTRODUCTION

Recently, L-carnitine and Cr picolinate (**CrP**) have been added to diets fed to sows to improve reproductive and litter performance. Dietary L-carnitine fed during gestation and lactation increased the number of pigs born live (Musser et al., 1999), number of pigs weaned (Eder et al., 2001), and litter weight gain (Ramanau et al., 2004). Similarly, Lindemann et al. (1995) observed that CrP increased number of pigs born alive and weaned, litter birth weights, and litter weights 21 d after farrowing. The exact modes of action behind these responses have not been elucidated. However, we speculate that these feed additives might enhance the energy status of sows, because L-carnitine has been shown to improve fatty acid utilization (Owen et al., 2001), and CrP has been shown to increase insulin sensitivity (Matthews et al., 2001) in growing-finishing pigs.

Little research has been conducted to determine the effects of L-carnitine and CrP on blood hormones and metabolites as indicators of energy status. Musser et al. (1999) observed that L-carnitine increased plasma

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IGF-I concentration at 60 and 90 d after breeding, and it tended to increase plasma insulin 10 and 60 d after breeding in sows bled 6 h after feeding. Lindemann et al. (1995) observed that CrP decreased insulin and insulin:glucose postfeeding (2 h after feeding), but it did not have any effect immediately before feeding. No research has evaluated the combination of both L-carnitine and CrP on blood hormones and metabolites.

Therefore, the objective of this experiment was to determine the effects of L-carnitine and CrP (individually or in combination) on diurnal changes in blood hormones and metabolites from gestating sows fed 1 meal per day.

MATERIALS AND METHODS

Animals and Housing

The Kansas State University Institutional Animal Care and Use Committee approved all procedures used in this experiment.

Gestating sows (n = 44; BW = 208 kg; parity = 2.0) were used to determine the influence of L-carnitine and CrP on blood profiles of metabolites and metabolic hormones. Sows (PIC C-22; Franklin, KY) were randomly allotted to 1 of 4 dietary treatments based on parity and BW at initial breeding. Sows (approximately 32, 43, and 25% at parity 1, 2, and 3, respectively) averaged 185, 215, and 223 kg for parity 1, 2, and 3, respectively, when the blood samples were collected. At allotment, each sow was ear-tagged with 1 of 4 colors corresponding to the treatment received to maintain identification throughout the experiment. Sows were housed in individual crates (1.83 × 0.55 m) in an environmentally controlled gestation barn from breeding until approximately d 30 of gestation and then were moved to outside group pens and fed in individual feeding stalls. At approximately d 110 of gestation, sows were moved to an environmentally controlled farrowing house and placed in farrowing crates (2.1 × 0.6 m) until weaning. At weaning (21 ± 3 d), sows were returned to the gestation barn and placed in the gestating crates where estrous detection and breeding occurred. Sows remained there until the end of the experiment. At the end of the gestation period, after blood sample collection, sows were again moved to the farrowing facility, and litter characteristics were measured. Number of pigs born alive was 12.76, 13.15, 12.88, and 13.05 (SE = 0.64) for sows fed the control, L-carnitine, CrP, and the combination of L-carnitine and CrP, respectively. Statistical significance was not expected based on the small sample size.

Diets

Diets (Table 1) were corn-soybean meal-based and formulated to meet or exceed the NRC (1998) nutrient requirement estimates for gestating and lactating sows. Diets were fed to sows in gestation once per day, similar to commercial production. Sows were fed 2.04 kg/d of gestation diet from breeding until d 100 of gestation, then 2.95 kg/d until they farrowed. Lactation diet was increased from the day of farrowing until d 3 and offered ad libitum from d 3 until weaning. Sows were always allowed ad libitum access to water. Treatments were arranged in a 2 × 2 factorial, with main effects of L-carnitine (0 or 50 ppm) and CrP (0 or 200 ppb). L-Carnitine and CrP replaced cornstarch in the basal diet to form the experimental treatments. Both the L-carnitine (Carniking; 50% L-carnitine) and CrP (12% Cr) were obtained from Lonza Inc. (Allendale, NJ). Sows were fed the experimental treatments beginning at the initial breeding, through gestation, the following lactation and wean-to-breeding interval, and approximately 28 d into the subsequent gestation, at which time blood was collected.

Table 1. Diet composition (as-fed basis)[1]

Item	Gestation	Lactation
Ingredient, %		
Corn	79.47	64.60
Soybean meal, 46.5%	14.50	27.60
Monocalcium phosphate	2.28	2.05
Limestone	1.10	1.10
Soy oil	1.00	3.00
Cornstarch[2]	0.50	0.50
Salt	0.50	0.50
Vitamin premix[3]	0.25	0.25
Sow add pack[4]	0.25	0.25
Trace mineral premix[5]	0.15	0.15
Calculated analysis, %		
Lys	0.65	1.00
Ca	0.90	0.90
P	0.80	0.80

[1]Diets were formulated to meet or exceed the NRC (1998) estimated requirements for gestating and lactating sows.
[2]Cornstarch was replaced with 50 ppm L-carnitine or 200 ppb Cr, or both, from Cr picolinate to form the experimental treatments.
[3]Provided the following per kilogram of complete feed: 11,023 IU of vitamin A, 1,653.45 IU of vitamin D_3, 44.09 IU of vitamin E, 4.41 mg of menadione, 0.04 mg of vitamin B_{12}, 9.92 mg of riboflavin, 33.07 mg of pantothenic acid, and 55.12 mg of niacin.
[4]Provided the following per kilogram of complete feed: 551 mg of choline, 0.22 mg of biotin, 1.65 mg of folic acid, and 15.16 mg of pyridoxine.
[5]Provided the following per kilogram of complete feed: 165.3 mg of Zn (ZnO), 39.7 mg of Mn (MnO), 165.3 mg of Fe ($FeSO_4$), 16.5 mg of Cu ($CuSO_4$), 0.30 mg of I (ethylenediamine dihydroiodide), and 0.30 mg of Se (Na_2SeO_3).

Catheterization

Approximately 5 d before blood collection, sows were removed from the gestation barn and transported to a nearby surgery room. Sows were anesthetized i.v. with sodium thiopental (25 mg/kg) before surgery, and the surgical plane of anesthesia was maintained by inhalation of halothane (2 to 5%). Indwelling cephalic vein catheters (100-cm long; 1.02-mm i.d. and 2.16-mm o.d.; Helix Medical Inc., Carpinteria, CA) were inserted using techniques modified for sows from those described by Takken and Williams (1981). Sows were transported

back to the gestation barn once they regained locomotor capacity. Patency of the catheters was maintained by flushing the catheters 2 times per day with a 10% heparin solution. Catheters were removed after blood collection.

Blood Collection and Analysis

At d 28 ± 2 after the second breeding or approximately 167 d after dietary treatments began, blood was collected from each sow at feeding (0 h), once every 15 min for the first 3 h after feeding, and at 6, 9, 15, 20, and 24 h after feeding. Patency was maintained between each collection by flushing the catheters with a 10% heparin solution. Before each collection, 3 mL of blood was collected and discarded, and then 10 mL of blood was collected in a sterile12-mL syringe, placed into a glass tube containing 0.1 mL of 15% EDTA (Monoject; Sherwood Medical, St. Louis, MO), and immediately placed on ice. The blood was centrifuged (2,500 × *g* for 15 min), and then plasma was collected, separated into 12 aliquots, and frozen (–40°C) until analysis.

Plasma was analyzed at each of the 18 bleeding times to determine concentrations of insulin (Insulin RIA, DSL-1600; Diagnostic Systems Laboratories Inc., Webster, TX), connecting peptide of insulin (c-peptide; Porcine C-Peptide RIA, PCP-22K, Linco Research Inc., St. Charles, MO), IGF-I (Active IGF-I with Extraction, DSL-5600; Diagnostic Systems Laboratories Inc), glucagon (Double Antibody Glucagon, KGND1; Diagnostic Products Corporation, Los Angeles, CA), glucose (Glucose, 510-A; Sigma Diagnostics, St. Louis, MO), plasma urea N (**PUN;** Blood Urea Nitrogen, 535-B, Sigma Diagnostics), and NEFA (NEFA-C Kit, ACS-ACOD Method; Wako Chemicals US Inc., Richmond, VA.). At feeding and 0.5, 1, 2, 6, and 20 h after feeding (due to limited sample amounts), plasma was also analyzed to determine concentrations of IGFBP-3 (Active IGFBP-3 IRMA, DSL-6600; Diagnostic Systems Laboratories Inc.), triacylglycerol and glycerol (Triglyceride GPO-Trinder; 337-B, Sigma Diagnostics), and AA (AOAC, 1995).

The immunoassays used for determination of plasma hormones were validated for use in porcine plasma, with the exception of connecting peptide of insulin, which was a porcine-specific assay. Details of the validation of IGF-I have been reported previously (Balaji et al., 2000). For the insulin, glucagon, and IGFBP-3 assays, quantitative recovery of added mass and linearity of dilution were determined in porcine plasma. When insulin, glucagon, and IGFBP-3 standards were added into samples of porcine plasma and measured in the respective assays, the ratio of concentration measured to that expected averaged 117.4, 90.1, and 93.2%, respectively. Similarly, when varying volumes of porcine plasma were assayed in the insulin, glucagon, and IGFBP-3 assays, and the concentrations measured in the assay were corrected for dilution, the ratio of concentration measured to that expected averaged 88.3, 105.0, and 98.0%. For all immunoassays used to evaluate plasma hormones, the intra- and interassay CV averaged less than 15%.

Statistics

Data were analyzed as a randomized complete block design with repeated measures over time using the MIXED procedure (SAS Inst. Inc., Cary, NC). Sow was the experimental unit. The experimental model included all 2-way interactions and the main effects of L-carnitine and CrP. Covariates of sow BW and parity at bleeding were used. Initially, treatment × covariate interactions were tested, but they were not significant ($P > 0.10$). Therefore, the interaction terms for the covariates were deleted from the model, and only sow BW and parity were used. These covariates were significant ($P < 0.10$) for all blood criteria. Least squares means were used to compare treatment means within time. Degree of significance was defined as follows: $P > 0.10$, not significant; $P = 0.10$ to > 0.05, trends; $P \leq 0.05$, significant. Area under the response curve (**AUC**) was calculated using trapezoidal geometry.

RESULTS

Connecting Peptide of Insulin

An L-carnitine × CrP interaction was observed ($P < 0.001$; Table 2, Figure 1a and 1b) for mean c-peptide concentration from 0 to 3 h after feeding and overall (pooled 0 to 24 h). Sows fed the diet containing only CrP had lower c-peptide concentrations compared with sows fed the control diet; however, when L-carnitine was also fed, the reduction was not as dramatic. An L-carnitine × CrP interaction was also observed ($P < 0.008$; Table 3) for the AUC of c-peptide for the first 3 h after feeding (fed state). Sows fed diets containing either L-carnitine or CrP had decreased c-peptide concentrations, but the decrease was not as great when both L-carnitine and CrP were added in the diet. The concentration of c-peptide was affected the greatest in the first 3 h after feeding (Figure 1b). Sows fed the control diet had greater ($P < 0.05$) c-peptide concentrations compared with sows fed all other treatments at 0.5 and 0.75 h after feeding. Sows fed the control diet also had greater ($P < 0.05$) c-peptide concentrations compared with sows fed the diet containing CrP at 1, 1.5, 2.25, and 2.5 h after feeding and sows fed the diet containing L-carnitine at 2.75 h after feeding. Sows fed the diet containing both L-carnitine and CrP had greater ($P < 0.05$) c-peptide concentration compared with sows fed the diet containing CrP at 1 h after feeding.

Insulin

The L-carnitine and CrP treatments had a similar effect on insulin concentration as observed for c-pep-

Table 2. Influence of L-carnitine and Cr picolinate on mean blood parameter concentration[1]

	Carnitine, ppm/Cr, ppb					*P*-value		
Hours after feeding	0/0	50/0	0/200	50/200	SEM	Carnitine	Cr	Carnitine × Cr
C-peptide of insulin,[2] nmol/L								
0 to 3 h	0.580	0.462	0.450	0.495	0.026	0.09	0.02	0.0001
3 to 24 h	0.271	0.310	0.262	0.286	0.015	0.009	0.16	0.51
0 to 24 h	0.485	0.417	0.391	0.430	0.018	0.31	0.004	0.0001
Insulin,[2] pmol/L								
0 to 3 h	234.6	168.7	162.8	192.9	23.4	0.23	0.10	0.001
3 to 24 h	84.3	92.1	69.2	79.7	9.3	0.09	0.01	0.79
0 to 24 h	190.5	148.3	135.0	158.5	15.6	0.32	0.02	0.0004
Glucose,[2] mmol/L								
0 to 3 h	4.43	4.39	4.24	4.20	0.09	0.62	0.01	0.93
3 to 24 h	4.51	4.57	4.53	4.39	0.08	0.45	0.11	0.07
0 to 24 h	4.42	4.41	4.30	4.22	0.07	0.25	0.0006	0.42
Glucagon,[2] pmol/L								
0 to 3 h	31.15	31.21	33.64	31.01	1.66	0.40	0.44	0.36
3 to 24 h	30.57	29.76	30.85	30.19	1.28	0.41	0.68	0.93
0 to 24 h	30.95	30.84	32.85	30.81	1.24	0.33	0.39	0.37
Urea N,[2] mmol/L								
0 to 3 h	4.60	3.64	4.30	4.44	0.32	0.20	0.41	0.07
3 to 24 h	4.66	3.65	4.33	4.57	0.35	0.26	0.37	0.06
0 to 24 h	4.61	3.64	4.32	4.46	0.21	0.04	0.18	0.005
NEFA,[2] mmol/L								
0 to 3 h	0.139	0.138	0.166	0.140	0.014	0.04	0.03	0.05
3 to 24 h	0.163	0.132	0.172	0.126	0.008	0.0001	0.87	0.25
0 to 24 h	0.145	0.135	0.167	0.138	0.008	0.002	0.03	0.10
Triacylglycerol,[2] mmol/L								
0 to 2 h	0.247	0.272	0.263	0.276	0.028	0.26	0.56	0.72
2 to 20 h	0.283	0.279	0.296	0.273	0.022	0.25	0.75	0.42
0 to 20 h	0.263	0.277	0.276	0.276	0.026	0.60	0.69	0.61
Glycerol,[3] mmol/L								
0 to 2 h	0.041	0.044	0.052	0.048	0.005	0.88	0.06	0.31
2 to 20 h	0.046	0.042	0.053	0.054	0.006	0.63	0.02	0.56
0 to 20 h	0.043	0.042	0.051	0.049	0.005	0.73	0.008	0.70
IGF-I,[2] nmol/L								
0 to 3 h	15.26	19.12	15.02	15.52	2.54	0.34	0.39	0.45
3 to 24 h	12.11	15.70	11.49	12.90	1.58	0.008	0.06	0.23
0 to 24	14.34	17.91	14.08	15.12	1.97	0.11	0.28	0.37
IGFBP-3,[3] nmol/L								
0 to 2 h	5.49	5.77	5.53	6.32	0.33	0.10	0.34	0.42
2 to 20 h	2.79	2.73	3.04	3.28	0.23	0.66	0.05	0.44
0 to 20 h	4.70	4.86	4.72	5.40	0.23	0.06	0.19	0.22

[1]Values represent the mean of 44 sows (BW = 208; parity = 2.0), with 10 or 12 sows per treatment.
[2]Values represent the mean of samples collected at feeding, once every 15 min for the first 3 h after feeding, and at 6, 9, 15, 20, and 24 h after feeding.
[3]Values represent the mean of samples collected at feeding, 30 min, and 1, 2, 6, and 20 h after feeding.

tide. An L-carnitine × CrP interaction was observed ($P < 0.001$) for mean insulin concentration (0 to 3 and 0 to 24 h; Table 2) and AUC for the first 3 h after feeding (Table 3). Feeding diets containing either L-carnitine or CrP decreased insulin concentrations in the blood compared with feeding the control diet; however, when both L-carnitine and CrP were added to the diet, an intermediate response was observed (Table 2 and Figure 1c and 1d). Mean and AUC of insulin was lowest ($P < 0.05$) for the total 24-h period and the fasting period (3 to 24 h after feeding) when sows were fed diets containing CrP. Similar to c-peptide, the greatest treatment effect on insulin concentration was observed in the first 3 h after feeding (Figure 1c and 1d) compared with samples collected after 3 h. Sows fed the control diet had greater ($P < 0.05$) insulin concentrations than sows fed diets containing CrP at 0.5, 1, 2.25, and 2.5 h after feeding; sows fed all other diets at 0.75 and 1.25 h after feeding; and sows fed the diet containing L-carnitine at 2.5 h after feeding. Sows fed the diet containing L-carnitine and CrP had greater ($P < 0.05$) insulin concentrations compared with sows fed the diet containing L-carnitine at 0.75 h after feeding and compared with sows fed the diet containing CrP at 1 h after feeding.

Glucose

Mean glucose concentration from 0 to 3 h after feeding and overall was decreased ($P < 0.001$) when CrP was

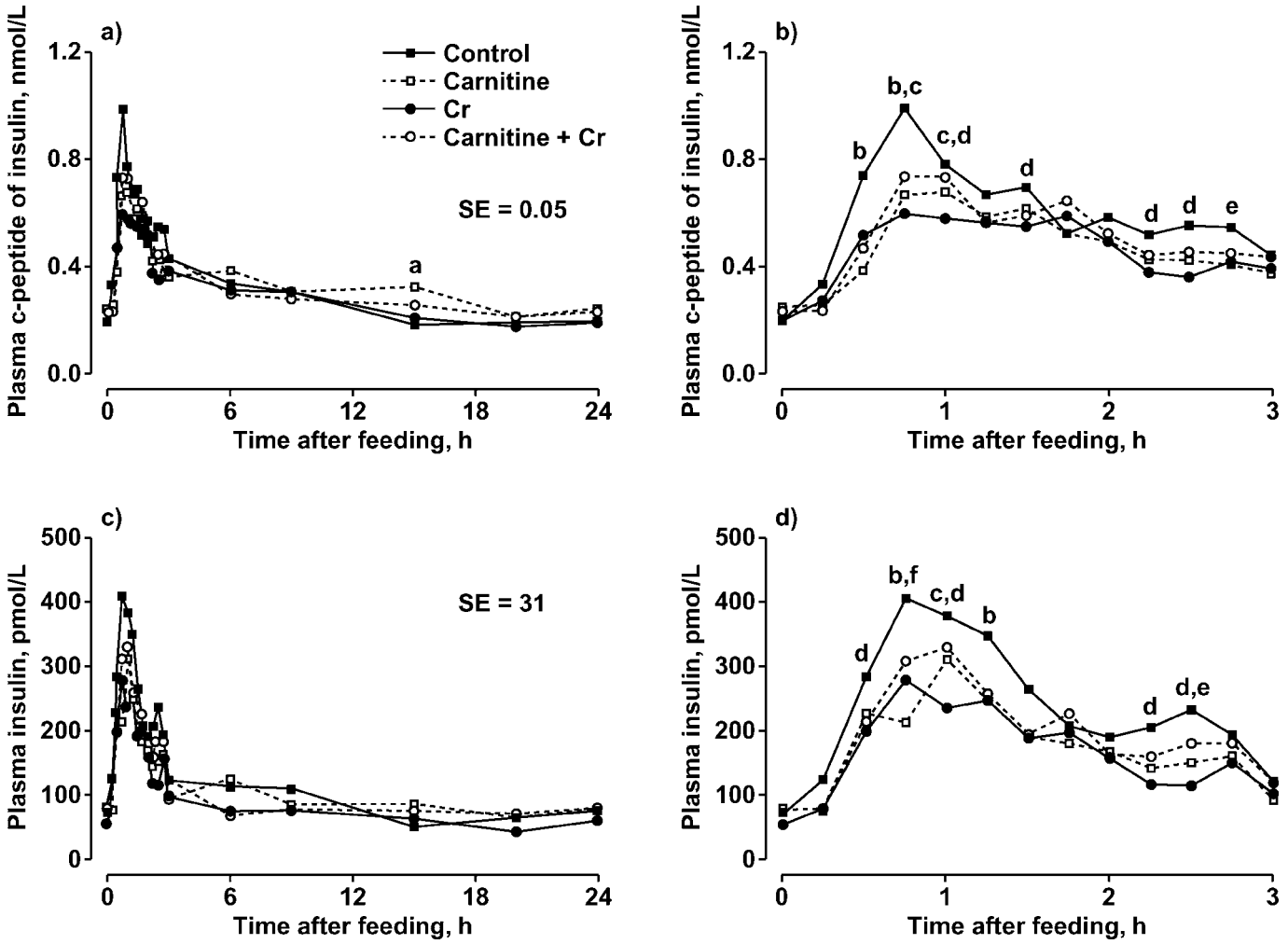

Figure 1. Influence of a control diet or a diet with L-carnitine and Cr on the (a) 24-h and (b) postfeeding connecting peptide of insulin profile and the (c) 24-h and (d) postfeeding insulin profile of gestating sows ($P < 0.05$). [a]Carnitine vs. control; [b]control vs. others; [c]carnitine + Cr vs. Cr; [d]control vs. Cr; [e]control vs. carnitine; [f]carnitine + Cr vs. Carnitine.

added to the diets (Table 2); however, AUC was not influenced ($P > 0.10$) by L-carnitine, CrP, or the combination of both (Table 3). Again the greatest effect of L-carnitine or CrP on glucose concentration was observed in the fed state (0 to 3 h) vs. the fasting state (3 to 24 h; Figure 2a and 2b). Sows fed the control diet had greater ($P < 0.05$) glucose concentrations compared with sows fed the other treatments at 0.5 h after feeding, sows fed the diet containing both L-carnitine and CrP at 0.25 and 0.75 h after feeding, and sows fed the diet containing CrP at 0.75, 1.5, and 2.25 h after feeding. Sows fed the diet containing L-carnitine and CrP had lower ($P < 0.05$) glucose concentrations compared with the other treatments at 0.5 h after feeding and sows fed diets containing only L-carnitine at 1 and 1.25 h after feeding.

Glucagon

L-carnitine, CrP, or both, did not influence ($P > 0.10$) mean glucagon concentration or AUC (Tables 2 and 3, Figure 2c). The only treatment difference within time occurred 1.5 h after feeding when sows fed the diet containing CrP had greater ($P < 0.05$; Figure 2d) glucagon concentration compared with sows fed the diet containing L-carnitine.

PUN

An L-carnitine × CrP interaction was observed ($P = 0.005$) for overall PUN concentration (Table 2), and a tendency for an L-carnitine × CrP interaction ($P < 0.08$) was observed for 0 to 3 and 3 to 24 h. An interaction for PUN AUC was also observed for the total 24-h period as well as from 3 to 24 h after the meal (Table 3). Sows fed the diet containing only L-carnitine had lower PUN concentration and AUC; however, there was no difference in PUN or AUC when both L-carnitine and CrP were added to the diet. The L-carnitine treatment decreased ($P < 0.05$; Figure 3a and 3b) PUN concentration at 6 and 24 h after the meal compared with the control diet and decreased ($P < 0.05$) PUN at 24 h after the meal

Table 3. Influence of L-carnitine, Cr picolinate, or both, on AUC of blood hormones and metabolites[1]

	Carnitine, ppm/Cr, ppb					*P*-value		
Hours after feeding	0/0	50/0	0/200	50/200	SEM	Carnitine	Cr	Carnitine × Cr
C-peptide of insulin,[2] min·nmol/L								
0 to 3 h	108.5	82.6	83.9	89.9	6.0	0.10	0.14	0.008
3 to 24 h	259.3	301.6	255.1	268.6	16.7	0.08	0.23	0.35
0 to 24 h	367.4	383.8	337.5	359.5	19.6	0.28	0.12	0.87
Insulin,[2] min·pmol/L								
0 to 3 h	44,557	30,321	30,900	35,290	4,892	0.22	0.27	0.02
3 to 24 h	85,594	95,325	71,107	77,717	10,538	0.32	0.05	0.84
0 to 24 hh	130,010	125,437	101,449	113,510	13,590	0.70	0.04	0.38
Glucose,[2] min·mmol/L								
0 to 3 h	778	733	740	723	35	0.16	0.25	0.50
3 to 24 h	4,376	4,411	4,413	4,229	97	0.31	0.31	0.13
0 to 24 h	5,154	5,143	5,154	4,954	117	0.23	0.27	0.26
Glucagon,[2] min·pmol/L								
0 to 3 h	5,501	5,100	5,953	5,451	393	0.25	0.28	0.89
3 to 24 h	29,176	28,638	29,128	28,593	1,927	0.82	0.98	0.99
0 to 24 h	34,678	33,738	35,081	34,044	2,293	0.74	0.90	0.99
Urea N,[2] min·mmol/L								
0 to 3 h	814	611	766	786	76	0.23	0.38	0.13
3 to 24 h	4,532	3,574	4,262	4,419	327	0.22	0.36	0.08
0 to 24 h	5,345	4,185	5,027	5,204	400	0.22	0.36	0.08
NEFA,[2] min·mmol/L								
0 to 3 h	24.3	23.6	29.5	24.3	3.4	0.053	0.053	0.13
3 to 24 h	151.2	126.0	158.3	116.5	9.7	0.0006	0.89	0.34
0 to 24 h	175.2	148.9	187.8	142.0	10.8	0.0006	0.76	0.30
Triacylglycerol,[3] min·mmol/L								
0 to 2 h	28.4	31.6	30.8	32.1	3.3	0.27	0.48	0.63
2 to 20 h	317.8	332.2	308.8	300.6	26.4	0.33	0.87	0.57
0 to 20 h	346.2	340.5	362.8	332.6	29.3	0.41	0.84	0.56
Glycerol,[3] min·mmol/L								
0 to 2 h	4.56	5.07	6.08	5.38	0.58	0.81	0.02	0.12
2 to 20 h	50.14	43.32	57.48	57.21	6.96	0.44	0.03	0.46
0 to 20 h	54.69	48.38	63.55	62.61	7.33	0.45	0.02	0.57
IGF-I,[2] min·nmol/L								
0 to 3 h	2,744	3,092	2,666	2,727	496	0.68	0.64	0.76
3 to 24 h	11,817	15,625	10,783	11,658	2,287	0.29	0.25	0.49
0 to 24 h	14,578	18,757	13,433	14,324	2,745	0.35	0.29	0.53
IGFBP-3,[3] min·nmol/L								
0 to 2 h	636.3	686.6	657.1	748.1	43.8	0.11	0.33	0.62
2 to 20 h	3,227.4	3,200.8	3,354.6	3,831.1	324.3	0.43	0.18	0.37
0 to 20 h	3,862.8	3,885.5	4,010.9	4,582.6	344.9	0.34	0.16	0.36

[1]Values represent the mean of 44 sows (BW = 208; parity = 2.0), with 10 or 12 sows per treatment. AUC = area under the curve.
[2]Values represent the mean of samples collected at feeding, once every 15 min for the first 3 h after feeding, and at 6, 9, 15, 20, and 24 h after feeding.
[3]Values represent the mean of samples collected at feeding, 30 min, and 1, 2, 6, and 20 h after feeding.

compared with the diet containing both L-carnitine and CrP. Sows fed diets containing L-carnitine had decreased ($P < 0.05$; Figure 3b) PUN concentrations compared with sows fed diets containing CrP at 0.75 and 1 h after feeding; decreased ($P < 0.05$) PUN compared with sows fed diets containing L-carnitine and CrP at 2 and 2.25 h after feeding, and decreased ($P < 0.05$) PUN compared with sows fed the control diet at 2.25, 2.5, 2.75, and 3 h after feeding. Sows fed the control diet had greater ($P < 0.05$) PUN compared with sows fed the diet containing CrP at 2.25 h after feeding.

NEFA

An L-carnitine × CrP interaction was observed for NEFA from 0 to 3 h ($P = 0.05$) and 0 to 24 h ($P = 0.10$) after feeding (Table 2). Sows fed the diet containing CrP had greater NEFA concentrations, but when fed in combination with L-carnitine, NEFA concentrations were reduced. The L-carnitine treatment also tended to decrease ($P < 0.053$) AUC for NEFA during the fed state (0 to 3 h after feeding), whereas sows fed diets containing CrP tended to have greater (L-carnitine × CrP interaction, $P < 0.053$) NEFA AUC during the fed state compared with sows fed diets without CrP. Sows fed diets containing L-carnitine had lower ($P < 0.001$) AUC for NEFA for the total 24-h period as well as the fasting period (3 to 24 h after feeding). Sows fed diets containing CrP had greater ($P < 0.05$; Figure 3c) NEFA concentrations compared with sows fed diets with L-carnitine at 6, 20, and 24 h after the meal and greater ($P < 0.05$) NEFA concentrations compared with sows

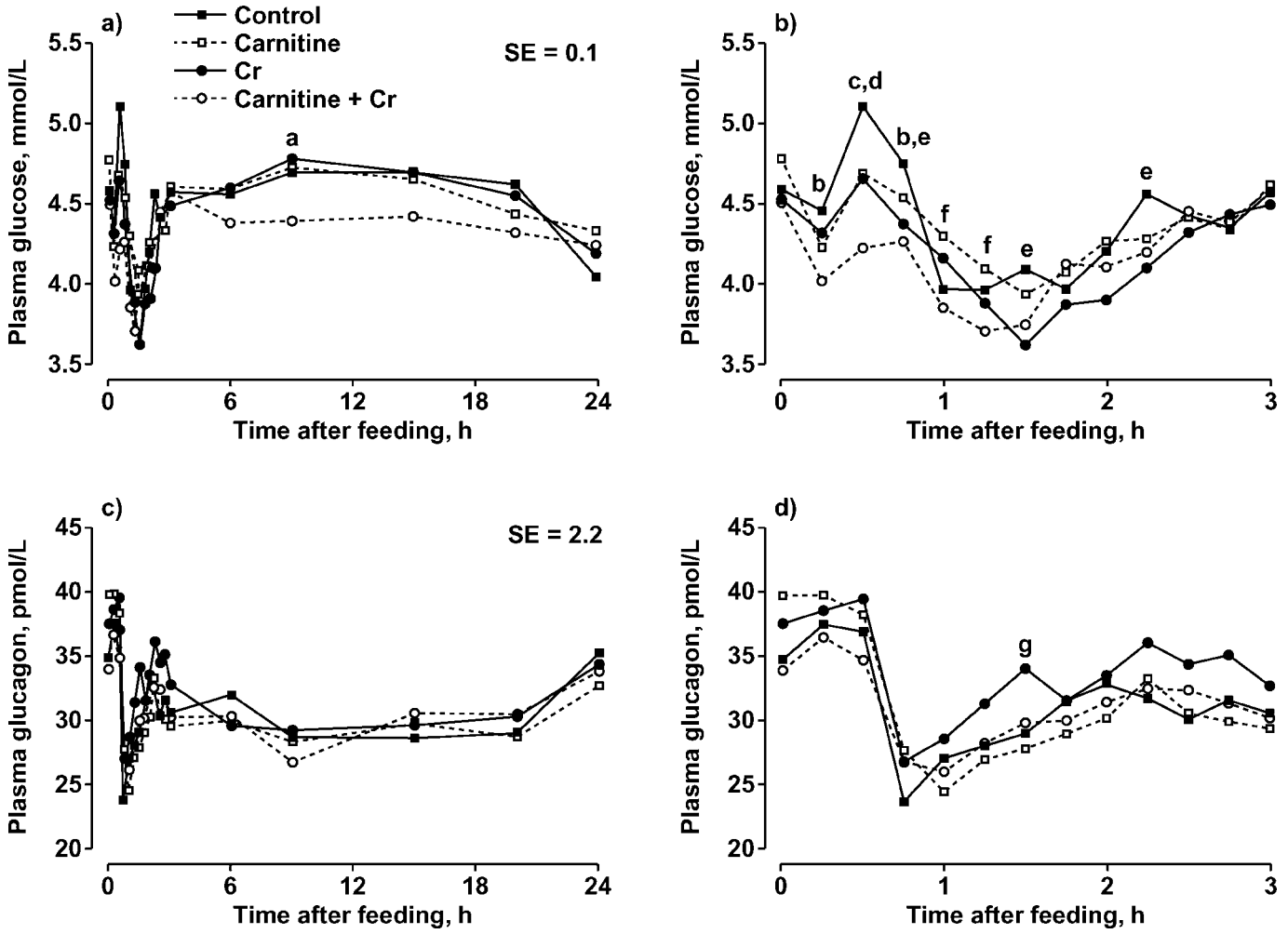

Figure 2. Influence of a control diet or a diet with L-carnitine and Cr on the (a) 24-h and (b) postfeeding glucose profile and the (c) 24-h and (d) postfeeding glucagon profile of gestating sows ($P < 0.05$). [a]Chromium vs. carnitine + Cr; [b]control vs. carnitine + Cr; [c]control vs. others; [d]carnitine + Cr vs. others; [e]control vs. Cr; [f]carnitine vs. carnitine + Cr; [g]Cr vs. carnitine.

fed diets with both L-carnitine and CrP at 20 and 24 h after the meal. Sows fed the control diet had elevated ($P < 0.05$) NEFA concentrations compared with sows fed diets with L-carnitine or L-carnitine and CrP at 24 h after the meal. Sows fed diets with L-carnitine or L-carnitine and CrP had decreased ($P < 0.05$; Figure 3d) NEFA concentrations compared with sows fed diets without L-carnitine or CrP at feeding (0 h after the meal) and decreased ($P < 0.05$) NEFA concentrations compared with sows fed the diet containing CrP at 0.25 h after the meal. Sows fed the diet containing CrP had elevated ($P < 0.05$) NEFA concentrations compared with sows fed diets containing L-carnitine at 1.5 h after the meal. Sows fed diets containing CrP had greater ($P < 0.05$) NEFA concentrations compared with sows fed the control diet or the diet containing L-carnitine at 2.5 and 2.75 h after the meal and greater ($P < 0.05$) NEFA concentrations compared with sows fed the diet containing both L-carnitine and CrP at 2.75 h after the meal. Sows fed the control diet had decreased ($P < 0.05$) NEFA concentrations at 2.5 h after the meal compared with sows fed the diet containing both L-carnitine and CrP.

Triacylglycerol

Dietary L-carnitine and CrP individually or in combination had no effect ($P > 0.10$) on mean triacylglycerol concentration or AUC (Tables 2 and 3). Pigs fed the diets containing either L-carnitine or L-carnitine and CrP had elevated ($P < 0.05$; Figure 4a) plasma triacylglycerols compared with sows fed the control diet or the diet containing CrP at 0.5 h after feeding. At 6 h after the meal, sows fed the diet containing CrP had greater ($P < 0.05$) plasma triacylglycerol compared with the sows fed the diet containing L-carnitine.

Glycerol

Sows fed diets containing CrP had greater ($P < 0.005$) mean glycerol concentrations from 2 to 20 and 0 to 20

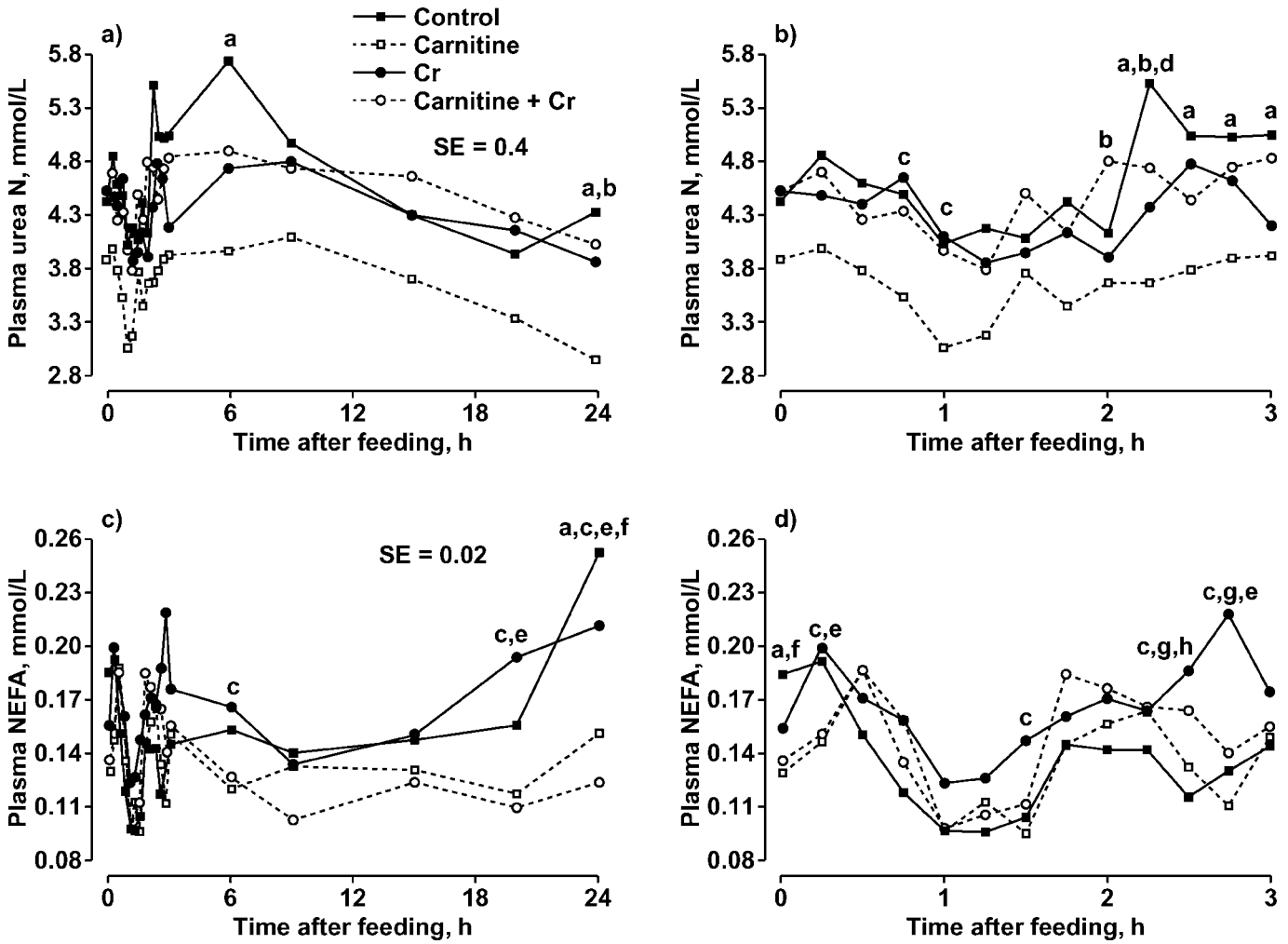

Figure 3. Influence of a control diet or a diet with L-carnitine and Cr on the (a) 24-h and (b) postfeeding plasma urea N profile and the (c) 24-h and (d) postfeeding NEFA profile of gestating sows ($P < 0.05$). [a]Control vs. carnitine; [b]carnitine + Cr vs. carnitine; [c]Cr vs. carnitine; [d]control vs. Cr; [e]Cr vs. carnitine + Cr; [f]control vs. carnitine + Cr; [g]Cr vs. control; [h]carnitine + Cr vs. control.

h after feeding and greater ($P < 0.05$) AUC from 0 to 20, 0 to 2, and 2 to 20 h after feeding (Tables 2 and 3). Sows fed the diet containing CrP had greater ($P < 0.05$; Figure 4b) plasma glycerol compared with sows fed the control diet at 0.5 h after feeding. Sows fed the diets containing CrP or L-carnitine and CrP had greater ($P < 0.05$) plasma glycerol compared with sows fed the diet containing only L-carnitine 6 h after the meal.

IGF-I

Sows fed diets with L-carnitine had greater ($P < 0.008$) IGF-I concentrations from 3 to 24 h after feeding (Table 2). No treatment differences ($P > 0.10$) were observed for AUC (Table 3) or treatment differences within time (Figure 5a and 5b).

IGFBP-3

Dietary CrP increased ($P < 0.05$) mean IGFBP-3 concentration from 2 to 20 h after feeding, and dietary L-carnitine tended to increase ($P < 0.10$) IGFBP-3 concentration from 0 to 2 and 0 to 20 h after feeding (Table 2). The L-carnitine or CrP treatments did not affect ($P > 0.11$) AUC for IGFBP-3 (Table 3). Sows fed the diets containing L-carnitine or L-carnitine and CrP had greater ($P < 0.05$; Figure 5c) plasma IGFBP-3 at 0.5 h after the meal. At 6 h after the meal, sows fed the diet containing L-carnitine and CrP had greater ($P < 0.05$) plasma IGFBP-3 compared with sows fed the control diet or the diet containing CrP.

AA

Significant sampling time effects for all AA except Gly (Table 4) were observed. All AA other than Gly increased in the 3 h after feeding and then returned to basal concentrations after 3 h. Glycine concentration remained constant throughout the sampling period. Both L-carnitine and CrP affected circulating concentrations of some AA (Table 4). An L-carnitine × CrP interaction ($P < 0.05$) was observed for Ala, Tyr, Orn,

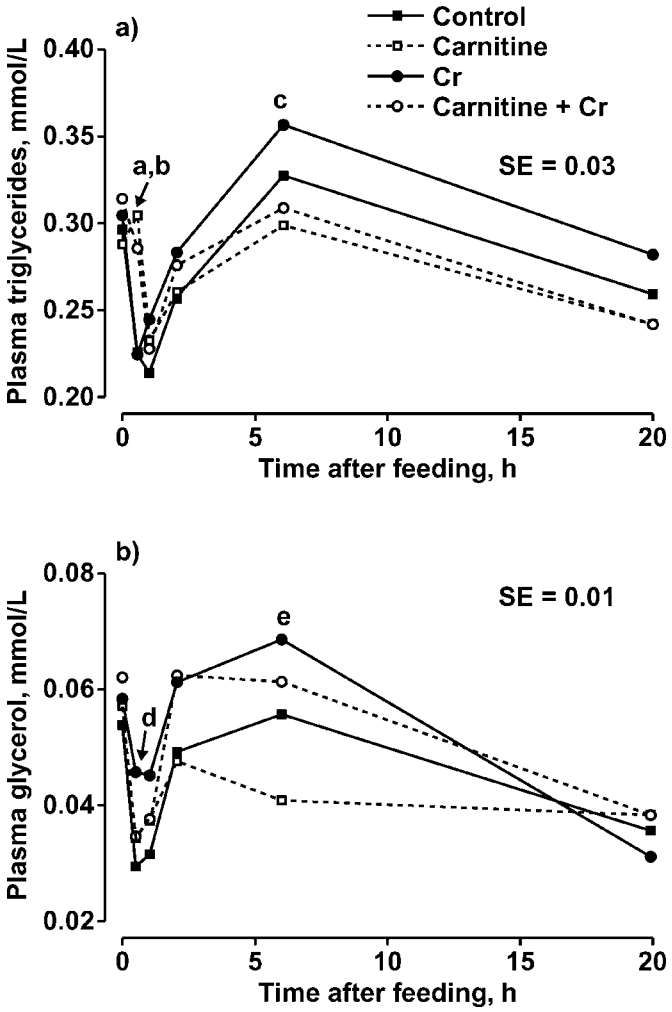

Figure 4. Influence of a control diet or a diet with L-carnitine and Cr on the (a) triacylglycerol and (b) glycerol profile of gestating sows ($P < 0.05$). [a]Carnitine and carnitine + Cr vs. control; [b]carnitine and carnitine + Cr vs. Cr; [c]Cr vs. carnitine; [d]Cr vs. control; [e]Cr and carnitine + Cr vs. carnitine.

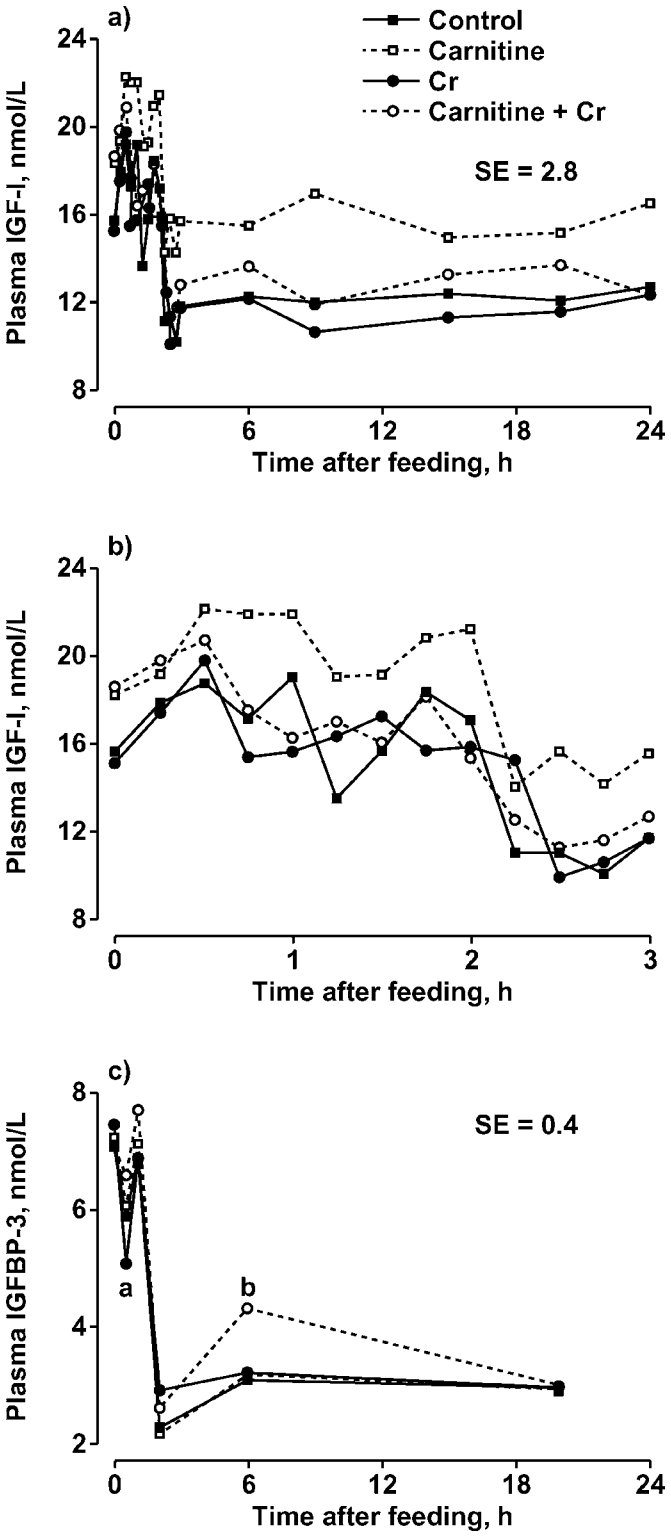

Figure 5. Influence of a control diet or a diet with L-carnitine and Cr on the (a) 24-h and (b) postfeeding IGF-I profile and (c) IGFBP-3 profile of gestating sows ($P < 0.05$). [a]Carnitine and carnitine + Cr vs. Cr; [b]carnitine + Cr vs. control and Cr.

Lys, and Arg, with all AA exhibiting decreased concentrations when either L-carnitine or CrP were added to the diet. No difference in these AA was observed when both L-carnitine and CrP were added to the diet compared with sows fed the control diet. Sows fed diets containing L-carnitine exhibited greater ($P < 0.05$) circulating concentrations of taurine, Gln, Gly, Met, and His, and sows fed diets containing CrP had greater ($P < 0.05$) glutamate and greater ($P < 0.05$) Trp concentrations.

DISCUSSION

L-Carnitine is required for the transport of medium- and long-chain fatty acids into the mitochondria for β-oxidation (Fritz, 1955). Dietary L-carnitine has been shown to enhance the utilization of fatty acids by increasing the oxidation of palmitate in isolated hepatocytes obtained from finishing pigs (Owen et al., 2001) and Atlantic salmon (Ji et al., 1996). In our experiment, sows fed diets containing L-carnitine had decreased mean NEFA concentrations compared with sows fed the

Table 4. Influence of carnitine, Cr, or both, on circulating AA concentrations[1]

Hours after feeding	Carnitine, ppm/Cr, ppb				Pooled SEM	*P*-value			
	0/0	50/0	0/200	50/200		Time	Carnitine	Cr	Carnitine × Cr
Ala					28.41	0.0001	0.46	0.11	0.05
0 h	347.2	335.4	323.0	317.3					
0.5 h	441.4	373.6	383.0	384.5					
1.0 h	531.2^{a}	467.0^{ab}	456.5^{b}	529.4^{a}					
2.0 h	532.2^{a}	453.6^{b}	495.7^{ab}	458.9^{b}					
6.0 h	405.6^{a}	401.3^{a}	301.9^{b}	359.7^{ab}					
20.0 h	330.1	294.3	281.3	310.8					
Arg					9.86	0.0001	0.87	0.13	0.002
0 h	124.6^{a}	114.7^{b}	131.5^{b}	197.8^{b}					
0.5 h	179.2^{ab}	146.1^{a}	163.1^{ab}	183.2^{b}					
1.0 h	201.8^{a}	220.8^{ab}	191.4^{a}	237.3^{b}					
2.0 h	227.0^{a}	170.4^{b}	208.5^{a}	217.1^{a}					
6.0 h	198.7^{a}	160.7^{b}	166.0^{ab}	162.4^{b}					
20.0 h	123.7	124.9	129.3	122.2					
Asn					2.93	0.0001	0.41	0.68	0.10
0 h	22.6	25.2	20.8	22.9					
0.5 h	41.7	35.1	33.2	32.9					
1.0 h	41.0^{ac}	48.8^{ab}	37.6^{c}	56.7^{b}					
2.0 h	44.6	37.7	40.3	46.3					
6.0 h	37.4	31.8	31.6	33.7					
20.0 h	17.5	16.9	17.1	17.5					
Asp					2.93	0.0001	0.42	0.68	0.10
0 h	22.6	25.2	20.8	22.9					
0.5 h	41.7	35.1	33.2	32.9					
1.0 h	41.0^{ac}	48.8^{ab}	37.6^{c}	56.7^{b}					
2.0 h	44.6	37.7	40.3	46.3					
6.0 h	37.4	31.8	31.6	33.7					
20.0 h	17.5	16.9	17.1	17.5					
Glutamate					16.00	0.0001	0.24	0.03	0.39
0 h	297.7^{a}	284.9^{ab}	283.0^{ab}	231.0^{b}					
0.5 h	350.5^{a}	285.9^{b}	336.3^{ab}	375.2^{a}					
1.0 h	419.2^{a}	386.6^{ab}	356.5^{b}	348.2^{b}					
2.0 h	475.9^{a}	364.4^{b}	420.5^{ab}	377.5^{b}					
6.0 h	335.9^{a}	412.9^{b}	276.1^{c}	306.1^{c}					
20.0 h	290.1	286.3	273.8	282.6					
Gln					12.58	0.0001	0.0001	0.66	0.12
0 h	255.6^{a}	332.1^{bc}	294.2^{ab}	357.7^{c}					
0.5 h	241.1^{a}	336.6^{b}	243.3^{a}	249.1^{a}					
1.0 h	174.0^{a}	272.6^{b}	202.4^{a}	307.5^{b}					
2.0 h	121.8^{a}	170.6^{b}	154.8^{ab}	153.9^{ab}					
6.0 h	178.1	166.3	170.9	159.6					
20.0 h	194.4	196.6	205.4	187.6					
Gly					56.48	0.21	0.02	0.10	0.20
0 h	847.9^{a}	816.8^{a}	894.3^{a}	1,083.5^{b}					
0.5 h	840.0^{a}	869.8^{a}	918.8^{a}	1,101.5^{b}					
1.0 h	846.9^{a}	1,088.3^{b}	861.5^{a}	1,125.2^{b}					
2.0 h	965.7	840.2	939.9	955.0					
6.0 h	854.6^{ab}	1,031.4^{b}	828.3^{a}	1,010.8^{ab}					
20.0 h	901.7	941.6	948.3	1,043.3					
His					4.00	0.0001	0.02	0.47	0.20
0 h	74.1^{a}	80.2^{ab}	78.9^{ab}	88.0^{b}					
0.5 h	86.9^{a}	83.4^{b}	85.2^{b}	101.7^{b}					
1.0 h	94.0^{a}	109.4^{b}	94.1^{a}	114.6^{b}					
2.0 h	98.8	92.3	99.6	98.8					
6.0 h	90.7	91.7	80.8	84.5					
20.0 h	76.2	81.6	74.3	79.9					
Ile					3.98	0.0001	0.35	0.15	0.78
0 h	108.6^{ab}	120.4^{a}	111.5^{ab}	100.3^{b}					
0.5 h	141.5	126.8	135.7	131.9					
1.0 h	168.5	153.1	153.6	159.8					
2.0 h	166.6	151.9	158.3	155.0					
6.0 h	147.4^{a}	142.8^{ab}	126.4^{b}	136.5^{ab}					
20.0 h	103.0	111.7	110.1	96.5					

Continued

Table 4 (Continued). Influence of carnitine, Cr, or both, on circulating AA concentrations[1]

Hours after feeding	Carnitine, ppm/Cr, ppb				Pooled SEM	*P*-value			
	0/0	50/0	0/200	50/200		Time	Carnitine	Cr	Carnitine × Cr
Leu					6.42	0.0001	0.75	0.08	0.29
0 h	213.8	237.1	222.9	214.7					
0.5 h	266.9	250.4	257.3	251.7					
1.0 h	313.9[a]	281.7[b]	279.9[b]	297.7[ab]					
2.0 h	316.9	297.9	300.4	297.4					
6.0 h	328.4[a]	294.9[b]	262.2[c]	291.9[b]					
20.0 h	208.7	233.4	216.6	214.8					
Lys					9.05	0.0001	0.43	0.88	0.03
0 h	246.5[ab]	209.6[a]	237.1[ab]	261.5[b]					
0.5 h	295.5[a]	234.7[b]	276.3[ab]	298.2[a]					
1.0 h	321.4[ab]	363.7[b]	288.7[a]	362.6[b]					
2.0 h	343.1[a]	254.3[c]	306.1[ab]	299.3[bc]					
6.0 h	247.9	234.2	212.9	211.0					
20.0 h	224.1	212.5	224.3	193.5					
Met					1.79	0.0001	0.018	0.29	0.77
0 h	39.9	44.2	42.8	46.2					
0.5 h	46.8	46.9	47.7	53.8					
1.0 h	53.4[a]	53.4[a]	52.1[a]	63.6[b]					
2.0 h	50.6	50.5	53.2	56.0					
6.0 h	46.5[a]	56.5[b]	45.6[a]	49.7[ab]					
20.0 h	38.5	46.5	42.1	42.3					
Orn					7.58	0.0001	0.37	0.87	0.006
0 h	83.2	72.3	77.5	95.2					
0.5 h	91.8	76.3	85.6	100.4					
1.0 h	104.8[bc]	130.9[a]	90.4[c]	128.7[ab]					
2.0 h	148.0[a]	112.0[b]	126.1[ab]	125.9[ab]					
6.0 h	140.0[a]	132.3[ab]	114.6[b]	135.8[ab]					
20.0 h	81.9	75.6	75.9	84.3					
Phe					3.06	0.0001	0.21	0.17	0.65
0 h	79.1[a]	92.2[b]	81.0[ab]	69.7[a]					
0.5 h	91.1	80.8	86.3	84.1					
1.0 h	111.8	100.7	110.1	106.3					
2.0 h	113.5	113.7	118.1	110.0					
6.0 h	109.5[a]	99.1[ab]	92.3[b]	98.5[ab]					
20.0 h	72.3	79.0	76.1	70.0					
Ser					7.00	0.0001	0.43	0.71	0.12
0 h	127.3	125.8	122.1	131.8					
0.5 h	150.7	138.8	141.6	154.0					
1.0 h	165.7[ab]	166.9[ab]	150.4[a]	182.3[b]					
2.0 h	174.8[a]	151.8[b]	166.3[ab]	159.1[ab]					
6.0 h	150.4[ab]	158.9[a]	134.3[b]	145.2[ab]					
20.0 h	110.5	115.7	109.9	119.3					
Taurine					4.92	0.0001	0.024	0.14	0.72
0 h	70.0	70.1	63.2	72.0					
0.5 h	67.7	73.8	80.2	81.8					
1.0 h	75.3[a]	113.1[b]	81.7[a]	81.4[a]					
2.0 h	111.6[a]	102.2[ab]	102.5[ab]	85.0[b]					
6.0 h	114.9[a]	143.7[b]	93.4[c]	146.5[b]					
20.0 h	87.1	89.9	74.0	79.1					
Thr					7.69	0.0001	0.40	0.83	0.57
0 h	128.5	134.0	138.5	133.6					
0.5 h	163.7	149.3	160.2	157.6					
1.0 h	186.9	187.8	181.1	203.0					
2.0 h	218.4[a]	168.8[b]	208.8[c]	187.5[bc]					
6.0 h	188.0	188.4	165.5	175.2					
20.0 h	120.3	122.5	138.1	123.7					
Trp					1.97	0.0001	0.59	0.02	0.64
0 h	33.3[a]	43.6[ab]	48.1[b]	46.0[b]					
0.5 h	53.3[ab]	48.2[a]	59.2[b]	52.5[ab]					
1.0 h	55.5	63.1	56.8	65.8					
2.0 h	56.8[a]	44.4[b]	57.7[a]	60.9[a]					
6.0 h	44.8[a]	53.5[ab]	46.4[a]	62.4[b]					
20.0 h	43.3	35.2	41.7	34.5					

Continued

Table 4 (Continued). Influence of carnitine, Cr, or both, on circulating AA concentrations[1]

Hours after feeding	Carnitine, ppm/Cr, ppb				Pooled SEM	*P*-value			
	0/0	50/0	0/200	50/200		Time	Carnitine	Cr	Carnitine × Cr
Tyr					3.34	0.0001	0.80	0.26	0.002
0 h	72.4	73.6	70.5	74.5					
0.5 h	90.1[a]	74.8[b]	84.2[ab]	94.5[a]					
1.0 h	109.6[ac]	94.6[bc]	100.9[c]	115.4[a]					
2.0 h	116.3	111.0	117.7	117.0					
6.0 h	112.7[a]	94.3[b]	93.1[b]	110.8[a]					
20.0 h	72.4	81.6	76.4	82.1					
Val					7.50	0.0001	0.40	0.17	0.82
0 h	272.8	280.9	280.7	272.6					
0.5 h	310.0	306.2	317.1	299.4					
1.0 h	362.7	335.0	333.2	349.6					
2.0 h	377.3[a]	323.9[b]	362.9[a]	328.2[b]					
6.0 h	327.0[ac]	352.4[c]	283.2[b]	312.6[ab]					
20.0 h	249.3[ab]	271.4[a]	270.7[a]	235.9[b]					

[a–c]Means within the same row with different superscripts differ ($P < 0.05$).
[1]Values represent the mean of 44 sows (BW = 208; parity = 2.0), with 10 or 12 sows per treatment.

other treatments, which could be reflective of enhanced fatty acid utilization, such as that observed in pigs by Owen et al. (2001). In our experiment, the reduction in NEFA concentration increased as time after the meal increased, which could be reflective of the sow relying more on fatty acids as the major energy substrate during the fasted state. Today, it is common in commercial production to feed gestating sows a single daily meal that is often consumed within 15 min, similar to our experiment. Therefore, the sow relies on catabolism of body tissue (primarily adipose) to meet her energy demand during the majority of the day. Consequently, the change in circulating NEFA concentration suggests that dietary L-carnitine has a significant effect on fatty acid utilization and energy metabolism in gestating sows.

In our experiment, sows fed the diet containing only L-carnitine had lower overall PUN concentrations compared with sows fed the other treatments. In finishing pigs, Owen et al. (2001) observed that dietary L-carnitine increased the incorporation of AA into protein in isolated hepatocytes and decreased the flux through branch chain keto-acid dehydrogenase in liver and muscle mitochondria, both of which would be associated with reduced PUN. Therefore, the reduction in PUN we observed could be reflective of greater muscle anabolism but is probably more a function of less muscle catabolism, because the sow did not need to rely on branch chain AA for energy because fatty acid utilization was improved.

Musser et al. (1999) observed that sows fed dietary L-carnitine had greater plasma concentrations of IGF-I at d 60 and 90 of gestation and numerically greater concentrations at d 10 of gestation compared with control sows. Similarly, in our experiment, sows fed diets containing L-carnitine had greater IGF-I from 3 to 24 h after feeding and tended to have elevated plasma IGF-I concentrations overall when determined at approximately d 30 of gestation. Insulin-like growth factor I has been reported to increase muscle fiber differentiation and proliferation (Florini et al., 1991). Muscle development of offspring has been reported to begin from d 30 to 50 of gestation for swine (Ashmore et al., 1973). Consequently, the greater lean percentage and increased muscle fiber numbers observed from offspring obtained from sows fed diets containing L-carnitine reported by Musser et al. (2001) may have occurred because of the elevated maternal IGF-I concentration.

Dietary L-carnitine also tended to increase IGFBP-3, the major IGFBP found in serum (Jones and Clemmons, 1995). Similar to our results, Owens et al. (1999) observed that IGFBP-3 increased in conjunction with increases in IGF-I. As opposed to free IGF-I, Clapper et al. (2000) suggested that IGFBP-3 might provide a stable reservoir for IGF-I, thus increasing the amount of available circulating IGF-I. Therefore, dietary L-carnitine may not only act directly to increase IGF-I concentration but also increase IGFBP-3, which would indirectly increase the available circulating IGF-I. Alternatively, the increase in circulating IGF-I observed when carnitine is fed to sows may just be a reflection of increased IGFBP-3 acting as a plasma reservoir.

Sows that were fed diets containing L-carnitine had lower meal-induced glucose peaks compared with sows fed the control diet. This would suggest that L-carnitine might improve glucose uptake in peripheral tissue, because all sows were fed the same amount of feed. This hypothesis is supported by research reported by Giancaterini et al. (2000) that showed that acetyl-L-carnitine improved glucose disposal in human diabetic patients.

Chromium, as a component of the glucose tolerance factor, has been shown to potentiate the action of insulin and improve insulin sensitivity in swine (Steele et al., 1977). Lindemann et al. (1995) observed that postfeeding (2 h after the meal) insulin concentration and insulin:glucose were decreased; however, fasting (24 h after the meal) did not affect insulin concentration or insulin:glucose when sows were fed a diet containing

200 ppb CrP compared with control sows. Similarly, in our experiment, sows fed diets with CrP had reduced overall mean insulin concentrations. Evock-Clover et al. (1993) also showed that CrP reduced the insulin concentration in growing pigs.

We also observed that the c-peptide of insulin was reduced in a similar pattern as that of insulin when CrP was added to the diet. The c-peptide of insulin, as opposed to insulin itself, has been shown to be a better indicator of insulin secretion, because there is little hepatic extraction of c-peptide (Polonsky et al., 1983). Thus, our findings would suggest that CrP decreased the plasma insulin concentrations by reducing the amount of insulin that was released by the β-cells of the pancreas, as opposed to increased hepatic extraction of insulin from the blood.

In our experiment, sows fed the diets containing CrP had decreased mean glucose concentrations compared with other sows. Sows fed the diets containing CrP also had reduced postfeeding glucose peaks compared with the sows fed the control diet. The decreased glucose in the presence of lower plasma insulin concentrations would suggest that the glucose-clearing actions of insulin were enhanced. Other investigators have also reported that glucose half-life was reduced and insulin sensitivity was enhanced when CrP was added to the diets of grow-finish pigs (Amoikon et al., 1995; Matthews et al., 2001).

Sows fed diets containing CrP had increased NEFA concentrations in our experiment. In experiments conducted with growing pigs, Johnston et al. (1999) observed that Cr nicotinate increased plasma NEFA in 1 of 2 experiments; however, other experiments conducted with growing-finishing pigs reported that CrP decreased NEFA (Ward et al., 1997; Matthews et al., 2001). To our knowledge, ours is the only experiment that has evaluated the effects of CrP on plasma NEFA in sows. Sows fed diets containing CrP also had greater plasma glycerol compared with sows fed diets without CrP. The observed increase in glycerol concentration, in combination with the increased NEFA concentration, suggests that lipolysis was increased in sows fed diets containing CrP. No other experiments have evaluated the effects of CrP on lipolysis in swine to confirm this hypothesis. However, the improvements in lean percentage and reduction in backfat of finishing pigs fed diets containing CrP (Page et al., 1993; Lindemann et al., 1995) could be reflective of greater rates of lipolysis.

Both L-carnitine and CrP affected the circulating concentrations of some AA (Glu, Gln, Gly, Ala, Met, and Tyr), indicating that these feed additives may play a role in AA or protein metabolism. There has been little research conducted in other models to evaluate the influence of L-carnitine or CrP on AA metabolism. Owen et al. (2001) indicated that L-carnitine affected the AA concentration of muscle and liver tissue in growing pigs, and Cho et al. (1999) showed that L-carnitine fed to weanling pigs improved AA digestibility of some AA. With Cr, Roginski and Mertz (1969) observed an effect on AA metabolism by increasing their incorporation into the tissues of rats. These data suggest that L-carnitine and CrP may influence AA or protein metabolism.

This study evaluated the effects of both dietary L-carnitine and CrP on blood hormones and metabolites of gestating sows. The effect of L-carnitine on plasma NEFA and the effect of CrP on glucose and glycerol were still observed when both feed additives were fed. Intermediate effects of CrP on plasma c-peptide and insulin and intermediate effects of L-carnitine on PUN were observed when both CrP and L-carnitine were added to the diets. These results suggest that the effects of L-carnitine and CrP on blood hormones and metabolites of gestating sows are synergistic and that no detrimental effects were observed from supplementing the diet with both compounds.

Woodworth et al. (2004) observed that feeding L-carnitine, but not Cr tripicolinate, elevated the concentration of circulating leptin in gestating sows fed 1 meal per day. The elevated leptin concentrations suggest that feeding carnitine will affect the biochemical pathways involved in energy metabolism similar to the results herein. Increased leptin concentration has been shown to improve glucose utilization (Muller et al., 1997) and is consistent with our findings of decreased insulin and glucose concentrations in sows fed L-carnitine. Likewise, the improvements in reproductive performance previously observed in sows fed diets with carnitine may be reflective of elevated circulating leptin and other hormones and metabolites. Therefore, the beneficial responses to sow reproductive performance observed when L-carnitine (Musser et al., 1999; Eder et al., 2001) and CrP (Lindemann et al., 1995; Hagen et al., 2000) are added to the diets may also be additive, as suggested by Real (2001).

It was not an objective of this study to compare number of pigs born alive among dietary treatments, because the small number of observations per treatment would be too low for the possibility of statistical significance. However, from a purely observational standpoint, the observed increase ($P > 0.64$) in number of pigs born alive would agree with data reported by Real (2001), who also observed an additive increase in sow productivity when both L-carnitine and CrP are included in diets fed to sows. These data suggest that the past observations of improved reproductive performance when L-carnitine (Musser et al., 1999) or CrP (Lindemann et al., 1995) are fed to sows is a reflection of a heightened energy status.

These results suggest that L-carnitine and CrP additively influenced the blood hormones and metabolites and energy status of gestating sows fed 1 meal per day. The reductions in plasma NEFA and urea N observed from feeding L-carnitine and the reduction in plasma insulin and glucose observed from feeding CrP suggest that energy status of the sows was improved. The improvement in energy status of sows fed diets containing L-carnitine and CrP could explain the improvements in reproductive performance observed in previous experi-

ments from feeding these additives. The additive effect of L-carnitine and CrP on blood hormones and metabolites could explain the similar additive effect on reproductive performance previously observed and suggests that both L-carnitine and CrP should be included in diets fed to sows.

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