INFLUENCE OF DIETARY CARNITINE AND/OR CHROMIUM ON BLOOD PARAMETERS OF GESTATING SOWS[1]

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Summary

Gestating sows (n=44; parity=2.0; BW=458 lb) were used to determine the effects of dietary Carnitine and/or chromium picolinate on daily blood parameter profiles. Diets were formulated as a 2 × 2 factorial with carnitine (0 or 50 ppm) and chromium (0 or 200 ppb) and were fed from breeding, through gestation, lactation, and 30 d into the next gestation at which time blood was collected. Sows were fed one meal per day during gestation (2.1 kg) and ad libitum during lactation. Sows were fitted with indwelling venous catheters and blood (plasma) was 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. Chromium picolinate elicited its greatest effect immediately after feeding (0-3 h) by decreasing (P<0.05) insulin and c-peptide, whereas Carnitine decreased (P<0.05) NEFA and urea N (PUN) in the fasting state (6-24 h post-feeding). Sows fed both carnitine and chromium exhibited intermediate responses. Post-feeding glucose peak was lower (P<0.05) for diets with carnitine and/or chromium versus the control and mean glucose concentration was lower (P<0.01) for sows fed diets with chromium. Mean insulin and c-peptide concentration was lowest (P<0.01) for sows fed the diet with chromium and highest for sows fed the control, with sows fed diets with carnitine or carnitine and chromium having intermediate responses (Carnitine × chromium, P<0.01). Mean NEFA was lower (P<0.01) for sows fed diets with carnitine. Mean NEFA and glycerol were higher (P<0.03) for sows fed the diets with chromium. Sows fed the diet with only carnitine had the lowest PUN, but no differences were observed between the other three diets (carnitine × chromium, P<0.01). Dietary carnitine increased (P<0.05) the circulating leptin concentration, specifically in the fasting portion of the day. Both carnitine and chromium were observed to influence (P<0.05) the concentrations of some amino acids. No differences were observed for IGF-1, IGFBP-3, glucagon, or triglyceride (P>0.10); however, sows fed carnitine had numerically higher (P=0.11) IGF-1 and IGFBP-3 (P=0.06). In summary, the changes in metabolites and metabolic hormones indicate that both carnitine and chromium influence energy metabolism of gestating sows; however, their effects on blood parameters are different. Thus, the improvement in energy status from adding both carnitine and chromium may have an additive effect on reproductive performance of sows.

(Key Words: Sows, Carnitine, Chromium, Blood Parameters.)

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Introduction

Carnitine is a vitamin-like compound that is essential in the transport of long- and medium-chain fatty acids across the mitochondrial membrane for beta-oxidation. Carnitine also enhances pyruvate carboxylase activity and decreases the activity of branch-chain ketoacid dehydrogenase, resulting in less muscle degradation. Research has shown that dietary carnitine fed to sows will increase the number of pigs born live per litter, improve farrowing rate, and increase the muscle development of offspring. The past improvements in sow and litter performance have been attributed to improved nutrient utilization by the sow.

Chromium is a trace mineral that is essential for activating specific enzymes and stabilizing proteins and nucleic acid. Its primary role in metabolism, however, is to increase the effectiveness of insulin through its presence on a molecule known as glucose tolerance factor. Dietary chromium has been shown to improve insulin sensitivity, and consequently glucose uptake, in swine. Chromium has also been shown to increase farrowing rate and number of pigs born live per litter.

Carnitine and chromium are both essential for proper energy metabolism in swine. Researchers at Kansas State University observed that when carnitine and chromium are added to diets of gestating sows, farrowing rate improved with the greatest improvement observed from the diet containing both carnitine and chromium. However, few trials have evaluated the effects of these two dietary additives on blood parameters in the gestating sow that is fed one meal per day, similar to commercial production. The objective of this experiment was to determine the influence of dietary carnitine and(or) chromium on the daily blood parameter profiles of the limit-fed gestating sow.

Procedures

The Kansas State University Animal Care and Use Committee approved all procedures used in this experiment. Sows (n=44; parity=2.0; BW=458 lb; PIC C-22) were randomly allotted to one of four dietary treatments based on parity and weight at initial breeding. At allotment, each sow was eartagged with one of four different colors corresponding to the treatment she received so that identification throughout the experiment could easily be maintained. Sows were housed in individual gestation crates in the KSU gestation barn from breeding until approximately d 30 of gestation, at which time they were moved to outside pens and fed in individual feeding stalls. At approximately d 110 of gestation, sows were placed in the farrowing house and remained there until weaning. At weaning sows were returned to the gestation barn and placed in the individual crates and remained there until the end of the experiment.

Dietary treatments (Table 1) were corn-soybean meal-based and were formulated to meet or exceed NRC nutrient requirement estimates. Sows were fed 4.5 lb of gestation diet from breeding until d 100 of gestation, then 6.5 lb until they farrowed. Lactation diet was fed ad libitum from farrowing until weaning. Treatments were arranged in a 2 × 2 factorial design with main effects of carnitine (0 or 50 ppm) and chromium (0 or 200 ppb). Carnitine and(or) chromium replaced cornstarch in the basal diet to form the experimental treatments. Both the carnitine (Carniking) and chromium (chromium picolinate) were obtained from Lonza Inc., Fair Lawn, NJ. Sows were fed the experimental treatments starting 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)

Item, %	Gestation	Lactation
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[a]	0.50	0.50
Salt	0.50	0.50
Vitamin premix	0.25	0.25
Sow add pack	0.25	0.25
Trace mineral premix	0.15	0.15
Calculated analysis, %		
Lysine	.65	1.00
Ca	.90	.90
P	.80	.80

[a]Cornstarch was replaced with 50 ppm carnitine and(or) 200 ppb chromium to form the experimental treatments.

Approximately 5-d prior to blood collection, the sows were removed from the gestation barn and transported to a nearby surgery room. Sows were surgically fitted with indwelling cephalic vein catheters to minimize stress during blood collection. The catheters were exteriorized approximately 2 inches anterior to the point of the left shoulder and the loose end of the catheter stored in a pouch that was secured between the shoulder blades of the sow. After recovery from surgery, the sows were returned to the gestation barn and allowed four days of acclimation to the catheter prior to blood collection. Catheters were removed after blood collection on d 28 after breeding.

Blood (10 ml) from each sow was collected in tubes containing EDTA at approximately d 28 after the second breeding, or approximately 167 d after dietary treatments began. Blood was collected from each sow at feeding, once every 15 min for the first 3 h after feeding, and at 6, 9, 15, 20, and 24 h after feeding for a total of 18 collections from each sow. Samples collected from 0 to 3 h after feeding would represent the fed-state, and samples collected 6 h or later after feeding would represent the fasted state. After collection, blood samples were centrifuged and 12 separate aliquots of plasma were frozen (-40°C) for each sow at each bleeding time. Samples were then analyzed to determine insulin, connecting peptide of insulin, glucagon, glucose, IGF-1, non-esterfied fatty acids, urea nitrogen, leptin, glycerol, triglyceride, IGFBP-3, and amino acids.

Data were analyzed as a randomized complete block design with repeated measures over time with sow as the experimental unit. The experimental model included all two-way interactions and main effects of carnitine and chromium. Covariates of weight and parity at bleeding were used. Least square means was used to compare treatment means within time. Area under the response curve (AUC) was calculated using trapezoidal geometry.

Results and Discussion

Proinsulin is the peptide that is released from the beta-cells of the pancreas in response to rising concentrations of glucose and fatty acids in the blood. Proinsulin is comprised of one molecule of insulin and one molecule of the connecting peptide of insulin (c-peptide). For insulin to become active, the c-peptide must be cleaved off of the proinsulin molecule. Because the c-peptide has a greater half-life than insulin, determining the c-peptide concentration in blood will more accurately reflect the amount of activated insulin released. In our experiment, a carnitine × chromium interaction was observed ($P<0.0001$; Table 2) for mean c-peptide concentration. Sows fed the diet containing only chromium had decreased c-peptide concentrations com-

pared to sows fed the control diet; however, when carnitine was also fed in the diet the reduction was not as dramatic. A carnitine × chromium interaction was also observed ($P<0.0001$; Table 3) for the AUC of c-peptide for the first three hours after feeding (fed-state). Sows fed diets containing either carnitine or chromium had decreased c-peptide concentrations, but the decrease was not as great when both carnitine and chromium were added in the diet. The concentration of c-peptide was influenced the greatest in the first three hours after feeding (Figure 2). Sows fed diets containing neither carnitine nor chromium had greater ($P<0.05$) c-peptide concentrations compared to sows fed the other treatments at 0.5 and 0.75 hours after feeding; sows fed the diet containing chromium at 1, 1.5, 2.25, and 2.5 hours after feeding; and sows fed the diet containing carnitine at 2.75 hours after feeding. Sows fed the diet containing both carnitine and chromium had greater ($P<0.05$) c-peptide concentration compared to sows fed the diet containing chromium at one hour after feeding. Thus, diets containing chromium had the greatest influence on c-peptide concentration, primarily in the first three hours after feeding.

Insulin is the main anabolic hormone released in the body. Insulin is released in times of energy abundance. Rising concentrations of insulin in the blood would signal energy storage strategies, such as lipogenesis or muscle synthesis, whereas low concentrations of insulin are associated with energy mobilization from body tissues such as lipolysis or glycogenolysis. In our experiment, carnitine and chromium influenced insulin concentration similarly to their effects on c-peptide. A carnitine × chromium interaction was observed ($P<0.0004$) for mean insulin concentration (Table 2) and AUC for the first three hours after feeding (Table 3). Feeding diets containing either carnitine or chromium lowered insulin concentrations in the blood; however, when both carnitine and chromium were added to the diet, an intermediate response was observed (Figure 3). Area under the curve was lowest ($P<0.05$) for the total 24-hr period and the fasting period (3 to 24 hr after feeding) when sows were fed diets containing chromium. Similar to c-peptide, the greatest treatment effect on insulin concentration was observed in the first three hours after feeding (Figure 4). Sows fed the control diet had higher ($P<0.05$) insulin concentrations than sows fed diets containing chromium at .5, 1, 2.25, and 2.5 hours after feeding; sows fed all other diets at 0.75 and 1.25 hours after feeding; and sows fed the diet containing carnitine at 2.5 hours after feeding. Sows fed the diet containing carnitine and chromium had higher ($P<0.05$) insulin concentration compared to sows fed the diet containing carnitine at 0.75 hours after feeding and compared to sows fed the diet containing chromium at 1 hour after feeding. Therefore, the greatest effect on insulin concentration was observed from sows fed diets containing chromium, especially from 0 to 3 hours after the meal. Because, insulin and c-peptide were influenced similarly it would suggest that added dietary chromium resulted in less insulin secretion in response to the meal.

Glucose is the main energy substrate of the body. Blood glucose concentrations will rise after a meal, then decline as insulin initiates their clearance from the blood. Blood glucose concentration is regulated by hormones such as insulin and glucagon. Mean glucose concentration was lowered ($P<0.0006$) when chromium was added to the diets; however, AUC was not influenced by carnitine, chromium, or a combination of both. Again the greatest effect of carnitine or chromium on glucose concentrations was observed in the fed state (Figure 6). Sows fed the control diet had greater ($P<0.05$) glucose concentrations compared to sows fed the other treatments at 0.5 hours after feeding; sows fed the diet con-

taining both carnitine and chromium at 0.25 and 0.75 hours after feeding; and sows fed the diet containing chromium at 0.75, 1.5, and 2.25 hours after feeding. Sows fed the diet containing carnitine and chromium had lower (P<0.05) glucose concentrations compared to the other treatments at 0.5 hours after feeding; and sows fed diets containing only carnitine at 1 and 1.25 hours after feeding. The ability of carnitine and(or) chromium to lower glucose concentrations immediately after the meal would signify more rapid clearance of glucose from the blood since all sows were fed the same amount of feed, thus dietary glucose would be constant across treatments. In agreement with other research, dietary chromium decreased glucose concentration in the presence of lower concentrations of insulin. Therefore, the action of insulin was potentiated when chromium was included in the diet. Interestingly, carnitine also improved glucose tolerance immediately after the meal.

Non-esterfied fatty acids (NEFAs) or free-fatty acids (FFAs) are fatty acids that are present in the blood in a form not bound to glycerol or other substrates. Glycerol is the carbon back bone that lipids are bound to to form triglycerides, the storage form of lipid. Blood NEFA concentrations will increase after the meal reflective of dietary NEFA supply, but are most important in the fasted state when they are the main energy source for the body. Blood NEFA and glycerol concentrations will rise in response to greater lipolytic activity, or catabolism of adipose tissue. Sows fed diets containing carnitine had lower (P<0.002) mean NEFA concentrations compared to sows fed diets without carnitine. Sows fed diets with chromium had higher (P<0.03) mean NEFA concentrations compared to sows fed diets without chromium. Sows fed diets containing carnitine had lower (P<0.0006) AUC for NEFA for the total 24-hr period as well as the fasting period (3 to 24 hours after feeding). Carnitine also tended to decrease (P<0.053) AUC for NEFA during the fed state, while sows fed diets containing chromium tended to have higher (P<0.053) NEFA AUC during the fed state compared to sows fed diets without chromium. Sows fed diets containing chromium had greater (P<0.05; Figure 7) NEFA concentrations compared to sows fed diets with carnitine at 6, 20, and 24 hours after the meal, and greater (P<0.05) NEFA concentrations compared to sows fed diets with both carnitine and chromium at 20 and 24 hours after the meal. Sows fed the control diet had elevated (P<0.05) NEFA concentrations compared to sows fed diets with carnitine or carnitine and chromium at 24 hours after the meal. Sows fed diets with carnitine or carnitine and chromium had lower (P<0.05; Figure 8) NEFA concentrations compared to sows fed diets without carnitine or chromium at feeding (0 hours after the meal), and lower (P<0.05) NEFA concentrations than sows fed the diet containing chromium at 0.25 hours after the meal. Sows fed the diet containing chromium had elevated (P<0.05) NEFA concentrations compared to sows fed diets containing carnitine at 1.5 hours after the meal. Sows fed diets containing chromium had greater (P<0.05) NEFA concentrations compared to sows fed the control diet or the diet containing carnitine at 2.5 and 2.75 hours after the meal, and greater (P<0.05) NEFA concentrations compared to sows fed the diet containing both carnitine and chromium at 2.75 hours after the meal. Sows fed the control diet had lower (P<0.05) NEFA concentrations at 2.5 hours after the meal compared to sows fed the diet containing both carnitine and chromium. Sows fed diets containing chromium had higher (P<0.05) mean glycerol concentrations and greater (P<0.05) AUC from 0 to 20, 0 to 2, and 2 to 20 h after feeding. Sows fed the diet containing chromium had greater (P<0.05; Figure 15) plasma glycerol compared to sows fed the control diet at 0.5 h after feeding. Sows fed the diets containing chromium or carnitine and chromium had greater

($P<0.05$) plasma glycerol compared to sows fed the diet containing only carnitine 6 h after the meal. These results agree with past research showing that dietary carnitine will improve utilization of fatty acids, resulting in more extraction or less breakdown (lower concentrations) of NEFA from the blood without altering the concentration of glycerol. The rise in NEFA and glycerol concentration observed from adding chromium to the diet could be a reflection of the lower insulin concentrations observed from these sows because low blood insulin would act as a signal for lipolysis.

Triglyceride is the main storage form of lipids in the body. Dietary carnitine and/or chromium had no effect ($P>0.10$) on mean triglyceride concentration or AUC. Pigs fed the diets containing either carnitine or carnitine and chromium had elevated ($P<0.05$; Figure 16) plasma triglycerides compared to sows fed the control diet or the diet containing chromium at 0.5 h after feeding. At 6 h after the meal, sows fed the diet containing chromium had greater ($P<0.05$) plasma triglyceride compared to the sows fed the diet containing carnitine.

Insulin-like growth factor 1 (IGF-1) is an important anabolic hormone. Higher concentrations of IGF-1 would be associated with protein deposition as well as initiate the release of other growth hormones important for proper fetal growth and development. Insulin-like growth factor binding protein-3 is the main carrier of IGF-1 in the blood and acts to stabilize the IGF-1 molecule and extend its half-life. Because of high variability in IGF-1, no significant treatment differences were observed for mean IGF-1 concentration, AUC, or treatment differences within time; however, the sows fed the diet containing carnitine had numerically the greatest IGF-1 concentration. Similarly, there was a tendency for carnitine to increase ($P<0.06$) the circulating concentrations of IGFBP-3, which may have indirectly increased the circulating IGF-1 by increasing its half-life. This would support past research conducted at Kansas State University showing that dietary carnitine enhanced plasma IGF-1 concentrations of gestating sows and increased the muscle development of offspring.

Glucagon is an important hormone that is released in times of greater energy demand. It acts as a signal to mobilize energy substrates from body stores. Thus, it has opposing effects to insulin. Carnitine and(or) chromium did not influence mean glucagon concentration or AUC. The only treatment difference within time occurred 1.5 hours after feeding when sows fed the diet containing chromium had greater ($P<0.05$; Figure 12) glucagon concentration compared to sows fed the diet containing carnitine. These results would suggest that carnitine and(or) chromium do not have a major effect on glucagon concentrations in the blood.

Plasma urea nitrogen (PUN) represents the nitrogenous waste present in the blood from catabolism of amino acids. A carnitine × chromium interaction was observed ($P<0.005$) for mean PUN concentration and a tendency for a carnitine × chromium interaction ($P<0.08$) was observed for PUN AUC for the total 24-hour period as well as from 3 to 24 hours after the meal. Sows fed the diet containing only carnitine had lower PUN concentration and AUC; however, there was no difference in PUN or AUC when both carnitine and chromium were added to the diet. Carnitine decreased ($P<0.05$; Figure 13) PUN concentration at 6 and 24 hours after the meal compared to the control diet, and decreased ($P<0.05$) PUN at 24 hours after the meal compared to the diet containing both carnitine and chromium. Sows fed diets containing carnitine had lower ($P<0.05$; Figure 14) PUN concentrations compared to sows fed diets containing chromium at 0.75 and 1 hours after

feeding, had lower ($P<0.05$) PUN compared to diets containing carnitine and chromium at 2 and 2.25 hours after feeding, and had lower ($P<0.05$) PUN compared to sows fed the control diet at 2.25, 2.5, 2.75, and 3 hours after feeding. Sows fed the control diet had higher ($P<0.05$) PUN compared to sows fed the diet containing chromium at 2.25 hours after feeding. Sows fed the diet containing carnitine had numerically the lowest PUN concentrations at all bleeding times, suggesting that less muscle catabolism occurred when carnitine was fed. This would agree with previous research in finishing pigs showing that carnitine decreased the activity of branch-chain ketoacid dehydrogenase, an important enzyme necessary for branch-chain amino acid catabolism.

Amino acids are the main building blocks of protein. Circulating concentrations of individual amino acids will increase after the meal but may also increase during fasting as a reflection of muscle catabolism for energy. Both carnitine and chromium influenced the circulating concentrations of some amino acids. A carnitine × chromium interaction ($P<0.05$) was observed for alanine, tyrosine, ornithine, lysine, and arginine, with all amino acids being lower when either carnitine or chromium were added to the diet. But no difference was observed when both carnitine and chromium were added to the diet compared to sows fed the control diet. Sows fed the diets containing carnitine exhibited higher ($P<0.05$) circulating concentrations of taurine, glutamine, glycine, methionine, and histidine and sows fed the diets containing chromium had higher ($P<0.05$) glutamate and lower ($P<0.05$) tryptophan concentrations. Thus, both carnitine and chromium will influence protein metabolism.

In summary, this trial illustrates that both carnitine and chromium are important modifiers of energy status of sows fed one meal per day. Carnitine's greatest effect was during the fasted state (3 h or more after the meal) when it was associated with lower PUN and NEFA concentrations, the body's main energy substrate under these conditions. However, chromium elicited its greatest effect during the fed state (0 to 3 h after the meal) by decreasing the concentrations of both plasma insulin and glucose, suggesting a greater efficiency of glucose uptake. When both carnitine and chromium were added to the diets, similar and additive responses were observed; however, the change in blood parameter profile was not as dramatic. Therefore, both carnitine and chromium may act in concert to influence carbohydrate, lipid, and protein metabolism. The additive effects on energy status that were observed in this trial may explain the additive effects on reproductive performance that were observed in a previous experiment.

Table 2. Influence of Carnitine and(or) Chromium on Mean Blood Parameter Concentration[a]

	Carnitine, ppm	0	50	0	50		Probability, *P*<		
Item	Chromium, ppb	0	0	200	200	SEM	Carn.	Chrom.	C × C
C-peptide of insulin, nmol/L[b]		0.485	0.417	0.391	0.430	0.018	0.31	0.004	0.0001
Insulin, pmol/L[b]		190.5	148.3	135.0	158.5	15.6	0.32	0.02	0.0004
Glucose, mmol/L[b]		4.42	4.41	4.30	4.22	0.07	0.25	0.0006	0.42
NEFA, mmol/L[b]		0.145	0.135	0.167	0.138	0.008	0.002	0.03	0.10
IGF-1, nmol/L[b]		14.34	17.91	14.08	15.12	1.97	0.11	0.28	0.37
Glucagon, pmol/L[b]		30.95	30.84	32.85	30.81	1.24	0.33	0.39	0.37
Urea nitrogen, mmol/L[b]		4.61	3.64	4.32	4.46	0.21	0.04	0.18	0.005
Glycerol, mmol/L[c]		0.043	0.042	0.051	0.049	0.005	0.73	0.008	0.70
Triglyceride, mmol/L[c]		0.263	0.277	0.276	0.276	0.026	0.60	0.69	0.61
IGFBP-3, nmol/L[c]		4.70	4.86	4.72	5.40	0.23	0.06	0.19	0.22
Leptin, μg/L[b]		0.80	1.84	1.12	1.22	0.38	0.02	0.56	0.06

[a]Values represent a total of 44 sows (BW = 458 lb; parity = 2.0) with 10 or 12 sows per treatment.

[b]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.

[c]Values represent the mean of samples collected at feeding, 30 min, 1, 2, 6, and 20 h after feeding.

Table 3. Influence of Carnitine and(or) Chromium on AUC of Blood Parameters[a]

	Carnitine, ppm	0	50	0	50		Probability, *P*<		
Item	Chromium, ppb	0	0	200	200	SEM	Carn.	Chrom.	C × C
C-peptide of Insulin, min•nmol/L[b]									
0 to 24 hr after feeding		367.4	383.8	337.5	359.5	19.6	0.28	0.12	0.87
0 to 3 hr after feeding		108.5	82.6	83.9	89.9	6.0	0.10	0.14	0.008
3 to 24 hr after feeding		259.3	301.6	255.1	268.6	16.7	0.08	0.23	0.35
Insulin, min•pmol/L[b]									
0 to 24 hr after feeding		130,010	125,437	101,449	113,510	13,590	0.70	0.04	0.38
0 to 3 hr after feeding		44,557	30,321	30,900	35,290	4,892	0.22	0.27	0.02
3 to 24 hr after feeding		85.594	95,325	71,107	77,717	10,538	0.32	0.05	0.84
Glucose, min•mmol.L[b]									
0 to 24 hr after feeding		5,154	5,143	5,154	4,954	117	0.23	0.27	0.26
0 to 3 hr after feeding		778	733	740	723	35	0.16	0.25	0.50
3 to 24 hr after feeding		4,376	4,411	4,413	4,229	97	0.31	0.31	0.13
NEFA, min•mmol/L[b]									
0 to 24 hr after feeding		175.2	148.9	187.8	142.0	10.8	0.0006	0.76	0.30
0 to 3 hr after feeding		24.3	23.6	29.5	24.3	3.4	0.053	0.053	0.13
3 to 24 hr after feeding		151.2	126.0	158.3	116.5	9.7	0.0006	0.89	0.34
IGF-1, min•nmol/L[b]									
0 to 24 hr after feeding		14,578	18,757	13,433	14,324	2,745	0.35	0.29	0.53
0 to 3 hr after feeding		2,744	3,092	2,666	2,727	496	0.68	0.64	0.76
3 to 24 hr after feeding		11,817	15,625	10,783	11,658	2,287	0.29	0.25	0.49
Glucagon, min•pmol/L[b]									
0 to 24 hr after feeding		34,678	33,738	35,081	34,044	2,293	0.74	0.90	0.99
0 to 3 hr after feeding		5,501	5,100	5,953	5,451	393	0.25	0.28	0.89
3 to 24 hr after feeding		29,176	28,638	29,128	28,593	1,927	0.82	0.98	0.99
Urea nitrogen, min•mmol/L[b]									
0 to 24 hr after feeding		5,345	4,185	5,027	5,204	400	0.22	0.36	0.08
0 to 3 hr after feeding		814	611	766	786	76	0.23	0.38	0.13
3 to 24 hr after feeding		4,532	3,574	4,262	4,419	327	0.22	0.36	0.08
Glycerol, min•mmol/L[c]									
0 to 20 hr after feeding		54.69	48.38	63.55	62.61	7.33	0.45	0.02	0.57
0 to 2 hr after feeding		4.56	5.07	6.08	5.38	0.58	0.81	0.02	0.12
2 to 20 hr after feeding		50.14	43.32	57.48	57.21	6.96	0.44	0.03	0.46

Table 3. Continued

Triglyceride, min•mmol/L[c]								
0 to 20 hr after feeding	346.2	340.5	362.8	332.6	29.3	0.41	0.84	0.56
0 to 2 hr after feeding	28.4	31.6	30.8	32.1	3.3	0.27	0.48	0.63
2 to 20 hr after feeding	317.8	332.2	308.8	300.6	26.4	0.33	0.87	0.57
IGFBP-3, min•nmol/L[c]								
0 to 20 hr after feeding	3862.8	3885.5	4010.9	4582.6	344.9	0.34	0.16	0.36
0 to 2 hr after feeding	636.3	686.6	657.1	748.1	43.8	0.11	0.33	0.62
2 to 20 hr after feeding	3227.4	3200.8	3354.6	3831.1	324.3	0.43	0.18	0.37

[a]Values represent the mean of 44 sows (BW = 208; parity = 2.0), with 10 or 12 sows per treatment. AUC = area under the curve.
[b]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.
[c]Values represent the mean of samples collected at feeding, 30 min, 1, 2, 6, and 20 h after feeding.

Table 4. Influence of Carnitine and/or Chromium on Circulating Amino Acid Concentrations[a]

Item	Hours after feeding	Carnitine, ppm Chromium, ppb	0 0	50 0	0 200	50 200	Time	Carn	Chrom.	C × C
Taurine							0.0001	0.024	0.14	0.72
	0		70.0	70.1	63.2	72.0				
	0.5		67.7	73.8	80.2	81.8				
	1.0		75.3[a]	113.1[b]	81.7[a]	81.4[a]				
	2.0		111.6[a]	102.2[ab]	102.5[ab]	85.0[b]				
	6.0		114.9[a]	143.7[b]	93.4[c]	146.5[b]				
	20.0		87.1	89.9	74.0	79.1				
Aspartate							0.0001	0.42	0.68	0.10
	0		22.6	25.2	20.8	22.9				
	0.5		41.7	35.1	33.2	32.9				
	1.0		41.0[ca]	48.8[ab]	37.6[c]	56.7[b]				
	2.0		44.6	37.7	40.3	46.3				
	6.0		37.4	31.8	31.6	33.7				
	20.0		17.5	16.9	17.1	17.5				
Threonine							0.0001	0.40	0.83	0.57
	0		128.5	134.0	138.5	133.6				
	0.5		163.7	149.3	160.2	157.6				
	1.0		186.9	187.8	181.1	203.0				
	2.0		218.4[a]	168.8[b]	208.8[c]	187.5[cb]				
	6.0		188.0	188.4	165.5	175.2				
	20.0		120.3	122.5	138.1	123.7				
Serine							0.0001	0.43	0.71	0.12
	0		127.3	125.8	122.1	131.8				
	0.5		150.7	138.8	141.6	154.0				
	1.0		165.7[ab]	166.9[ab]	150.4[a]	182.3[b]				
	2.0		174.8[a]	151.8[b]	166.3[ab]	159.1[ab]				
	6.0		150.4[ab]	158.9[a]	134.3[b]	145.2[ab]				
	20.0		110.5	115.7	109.9	119.3				

Table 4. Continued

Asparagine						0.0001	0.41	0.68	0.10
	0	22.6	25.2	20.8	22.9				
	0.5	41.7	35.1	33.2	32.9				
	1.0	41.0^{ca}	48.8^{ab}	37.6^{c}	56.7^{b}				
	2.0	44.6	37.7	40.3	46.3				
	6.0	37.4	31.8	31.6	33.7				
	20.0	17.5	16.9	17.1	17.5				
Glutamate						0.0001	0.24	0.03	0.39
	0	297.7^{a}	284.9^{ab}	283.0^{ab}	231.0^{b}				
	0.5	350.5^{a}	285.9^{b}	336.3^{ab}	375.2^{a}				
	1.0	419.2^{a}	386.6^{ab}	356.5^{b}	348.2^{b}				
	2.0	475.9^{a}	364.4^{b}	420.5^{ab}	377.5^{b}				
	6.0	335.9^{a}	412.9^{b}	276.1^{c}	306.1^{c}				
	20.0	290.1	286.3	273.8	282.6				
Glutamine						0.0001	0.0001	0.66	0.12
	0	255.6^{a}	332.1^{bc}	294.2^{ab}	357.7^{c}				
	0.5	241.1^{a}	336.6^{b}	243.3^{a}	249.1^{a}				
	1.0	174.0^{a}	272.6^{b}	202.4^{a}	307.5^{b}				
	2.0	121.8^{a}	170.6^{b}	154.8^{ab}	153.9^{ab}				
	6.0	178.1	166.3	170.9	159.6				
	20.0	194.4	196.6	205.4	187.6				
Glycine						0.21	0.02	0.10	0.20
	0	847.9^{a}	816.8^{a}	894.3^{a}	1083.5^{b}				
	0.5	840.0^{a}	869.8^{a}	918.8^{a}	1101.5^{b}				
	1.0	846.9^{a}	1088.3^{b}	861.5^{a}	1125.2^{b}				
	2.0	965.7	840.2	939.9	955.0				
	6.0	854.6^{ab}	1031.4^{b}	828.3^{a}	1010.8^{ab}				
	20.0	901.7	941.6	948.3	1043.3				
Alanine						0.0001	0.46	0.11	0.05
	0	347.2	335.4	323.0	317.3				
	0.5	441.4	373.6	383.0	384.5				
	1.0	531.2^{a}	467.0^{ab}	456.5^{b}	529.4^{a}				
	2.0	532.2^{a}	453.6^{b}	495.7^{ab}	458.9^{b}				
	6.0	405.6^{a}	401.3^{a}	301.9^{b}	359.7^{ab}				
	20.0	330.1	294.3	281.3	310.8				

Table 4. Continued

Valine						0.0001	0.40	0.17	0.82
	0	272.8	280.9	280.7	272.6				
	0.5	310.0	306.2	317.1	299.4				
	1.0	362.7	335.0	333.2	349.6				
	2.0	377.3^{aa}	323.9^{b}	362.9^{a}	328.2^{b}				
	6.0	327.0^{ac}	352.4^{c}	283.2^{b}	312.6^{ab}				
	20.0	249.3^{ab}	271.4^{a}	270.7^{a}	235.9^{b}				
Methionine						0.0001	0.018	0.29	0.77
	0	39.9	44.2	42.8	46.2				
	0.5	46.8	46.9	47.7	53.8				
	1.0	53.4^{a}	53.4^{a}	52.1^{a}	63.6^{b}				
	2.0	50.6	50.5	53.2	56.0				
	6.0	46.5^{a}	56.5^{b}	45.6^{a}	49.7^{ab}				
	20.0	38.5	46.5	42.1	42.3				
Isoleucine						0.0001	0.35	0.15	0.78
	0	108.6^{ab}	120.4^{a}	111.5^{ab}	100.3^{b}				
	0.5	141.5	126.8	135.7	131.9				
	1.0	168.5	153.1	153.6	159.8				
	2.0	166.6	151.9	158.3	155.0				
	6.0	147.4^{a}	142.8^{ab}	126.4^{b}	136.5^{ab}				
	20.0	103.0	111.7	110.1	96.5				
Leucine						0.0001	0.75	0.08	0.29
	0	213.8	237.1	222.9	214.7				
	0.5	266.9	250.4	257.3	251.7				
	1.0	313.9^{a}	281.7^{b}	279.9^{b}	297.7^{ab}				
	2.0	316.9	297.9	300.4	297.4				
	6.0	328.4^{a}	294.9^{b}	262.2^{c}	291.9^{b}				
	20.0	208.7	233.4	216.6	214.8				
Tyrosine						0.0001	0.80	0.26	0.002
	0	72.4	73.6	70.5	74.5				
	0.5	90.1^{a}	74.8^{b}	84.2^{ab}	94.5^{a}				
	1.0	109.6^{ac}	94.6^{bc}	100.9^{c}	115.4^{a}				
	2.0	116.3	111.0	117.7	117.0				
	6.0	112.7^{a}	94.3^{b}	93.1^{b}	110.8^{a}				
	20.0	72.4	81.6	76.4	82.1				

Table 4. Continued

Phenylalanine						0.0001	0.21	0.17	0.65
	0	79.1[a]	92.2[b]	81.0[ab]	69.7[a]				
	0.5	91.1	80.8	86.3	84.1				
	1.0	111.8	100.7	110.1	106.3				
	2.0	113.5	113.7	118.1	110.0				
	6.0	109.5[a]	99.1[ab]	92.3[b]	98.5[ab]				
	20.0	72.3	79.0	76.1	70.0				
Tryptophan						0.0001	0.59	0.02	0.64
	0	33.3[a]	43.6[ab]	48.1[b]	46.0[b]				
	0.5	53.3[ab]	48.2[a]	59.2[b]	52.5[ab]				
	1.0	55.5	63.1	56.8	65.8				
	2.0	56.8[a]	44.4[b]	57.7[a]	60.9[a]				
	6.0	44.8[a]	53.5[ab]	46.4[a]	62.4[b]				
	20.0	43.3	35.2	41.7	34.5				
Ornithine						0.0001	0.37	0.87	0.006
	0	83.2	72.3	77.5	95.2				
	0.5	91.8	76.3	85.6	100.4				
	1.0	104.8[bc]	130.9[a]	90.4[c]	128.7[ab]				
	2.0	148.0[a]	112.0[b]	126.1[ab]	125.9[ab]				
	6.0	140.0[a]	132.3[ab]	114.6[b]	135.8[ab]				
	20.0	81.9	75.6	75.9	84.3				
Lysine						0.0001	0.43	0.88	0.03
	0	246.5[ab]	209.6[a]	237.1[ab]	261.5[b]				
	0.5	295.5[a]	234.7[b]	276.3[ab]	298.2[a]				
	1.0	321.4[ab]	363.7[b]	288.7[a]	362.6[b]				
	2.0	343.1[a]	254.3[c]	306.1[ab]	299.3[bc]				
	6.0	247.9	234.2	212.9	211.0				
	20.0	224.1	212.5	224.3	193.5				
Histidine						0.0001	0.02	0.47	0.20
	0	74.1[a]	80.2[ab]	78.9[ab]	88.0[b]				
	0.5	86.9[a]	83.4[b]	85.2[b]	101.7[b]				
	1.0	94.0[a]	109.4[b]	94.1[a]	114.6[b]				
	2.0	98.8	92.3	99.6	98.8				
	6.0	90.7	91.7	80.8	84.5				
	20.0	76.2	81.6	74.3	79.9				

Table 4. Continued

Arginine					0.0001	0.87	0.13	0.002
	0	124.6[a]	114.7[b]	131.5[b]	197.8[b]			
	0.5	179.2[ab]	146.1[a]	163.1[ab]	183.2[b]			
	1.0	201.8[a]	220.8[ab]	191.4[a]	237.3[b]			
	2.0	227.0[a]	170.4[b]	208.5[a]	217.1[a]			
	6.0	198.7[a]	160.7[b]	166.0[ab]	162.4[b]			
	20.0	123.7	124.9	129.3	122.2			

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

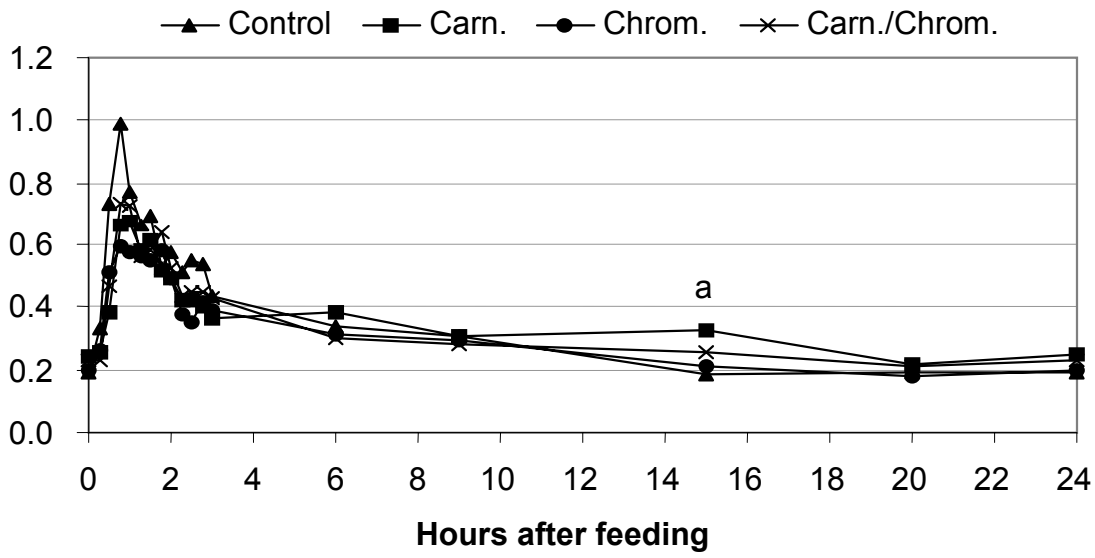

Figure 1. Influence of Carnitine and(or) Chromium on the Connecting-Peptide of Insulin (nmol/L).

[a]Carn. > Control; P<0.05.

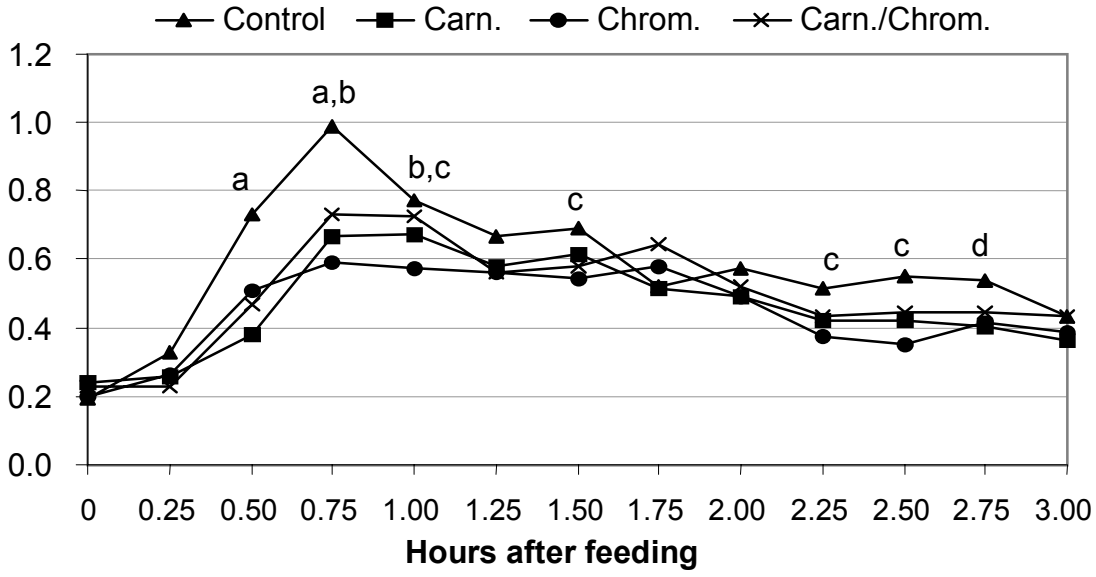

Figure 2. Influence of Carnitine and(or) Chromium on the Connecting-Peptide of Insulin (nmol/L).

[a]Control > others; P<0.05. [b]Carn./Chro. > Chrom.; P<0.05. [c]Control > Chrom.; P<0.05. [d]Control > Carn.; P<0.05.

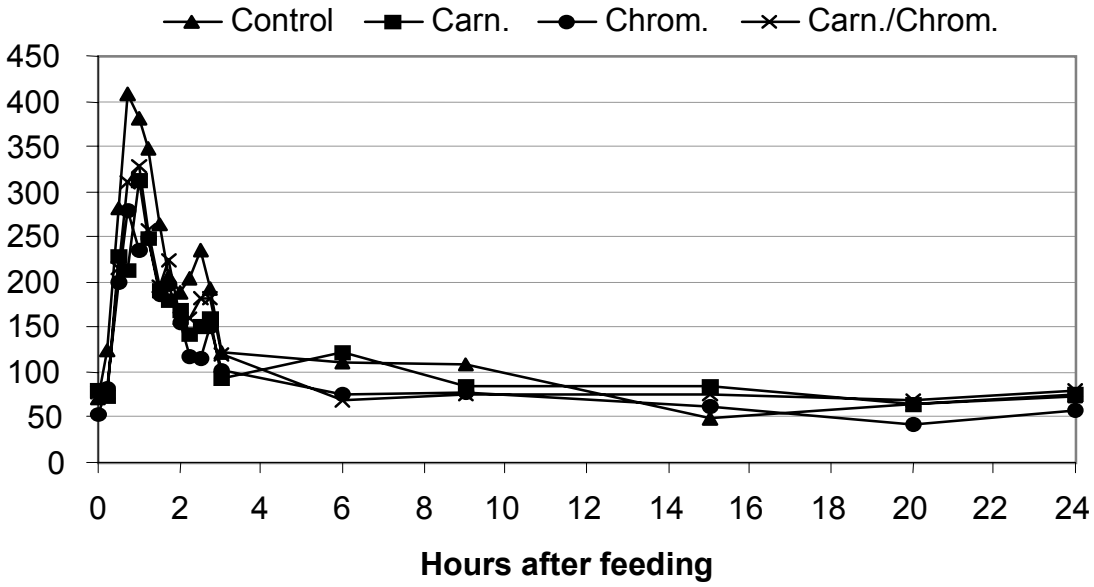

Figure 3. Influence of Carnitine and(or) Chromium on Insulin (pmol/L).

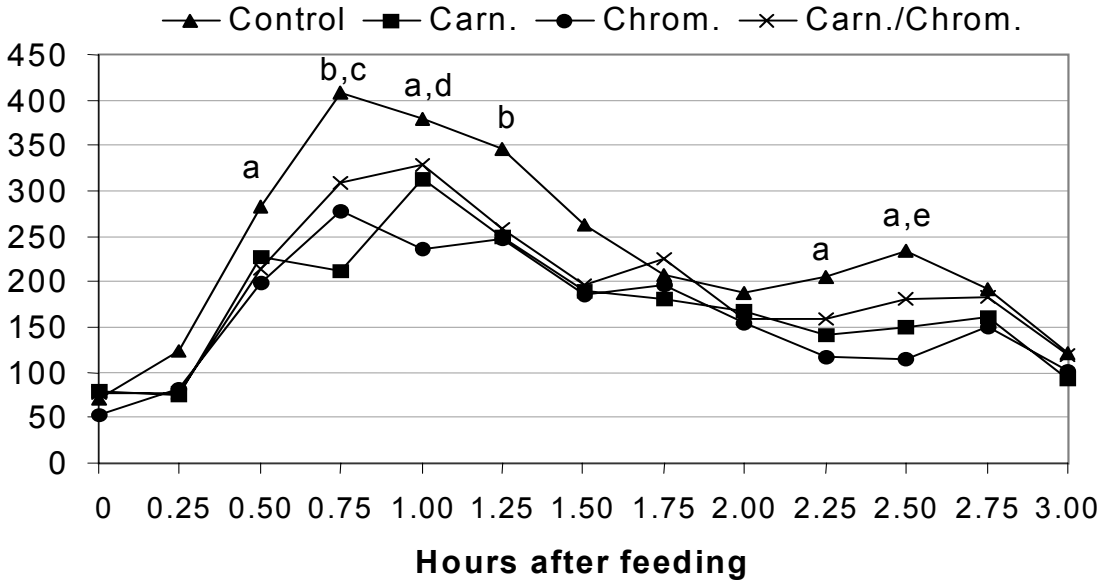

Figure 4. Influence of Carnitine and(or) Chromium on Insulin (pmol/L).

[a]Control > Chrom.; P<0.05. [b]Control > others; P<0.05. [c]Carn./Chrom. > Carn.; P<0.05. [d]Carn./Chrom > Chrom.; P<0.05. [e]Control > Carn.; P<0.05.

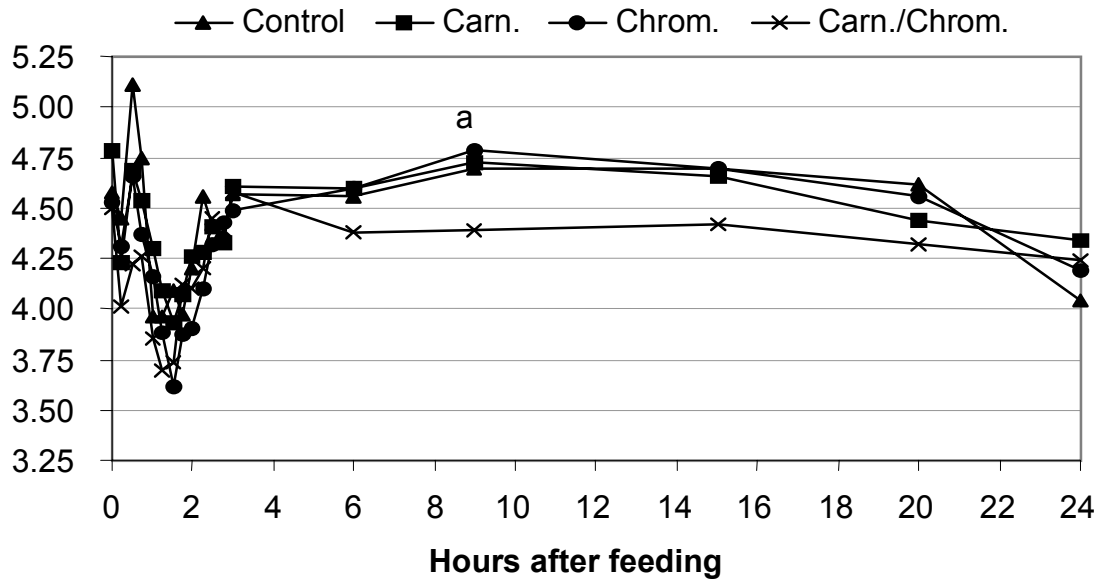

Figure 5. Influence of Carnitine and(or) Chromium on Glucose (mmol/L).

[a]Chrom. > Carn./Chrom.; P<0.05.

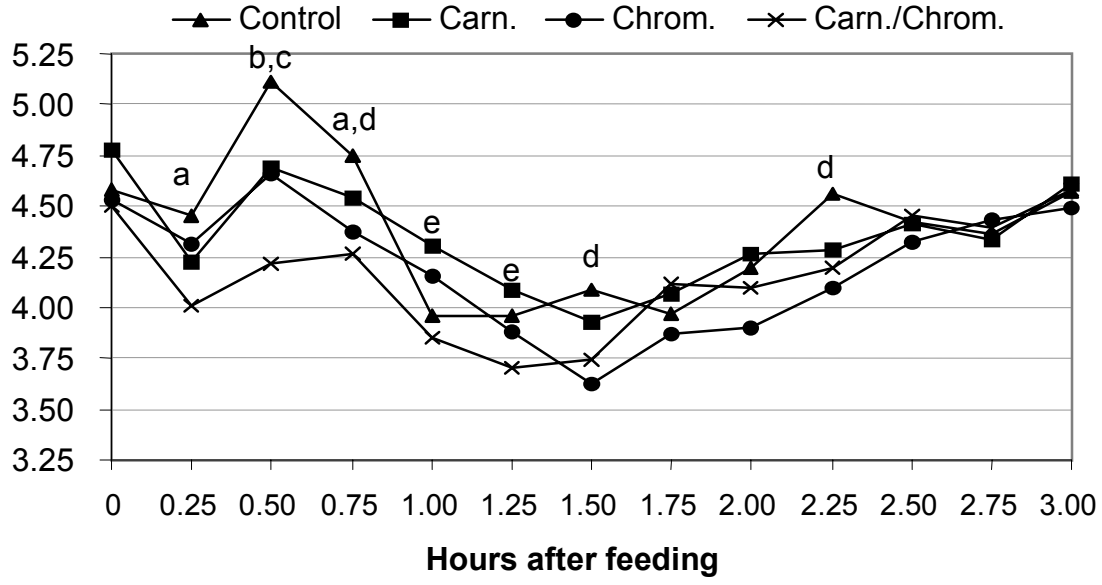

Figure 6. Influence of Carnitine and(or) Chromium on Glucose (mmol/L).

[a]Control > Carn./Chrom.; P<0.05. [b]Control > others; P<0.05. [c]Carn./Chrom. < others; P<0.05.
[d]Control > Chrom.; P<0.05. [e]Carn. > Carn./Chrom.; P<0.05.

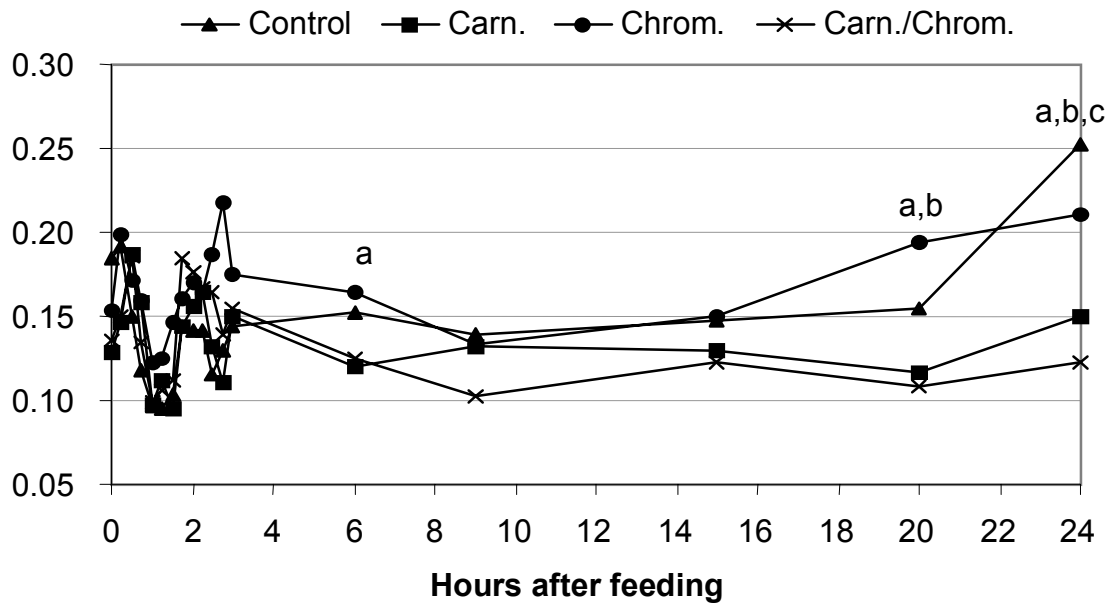

Figure 7. Influence of Carnitine and(or) Chromium on NEFA (mmol/L).

[a]Chrom. > Carn.; P<0.05. [b]Chrom. > Carn./Chrom.; P<0.05. [c]Control > Carn. and Carn./Chro.; P<0.05.

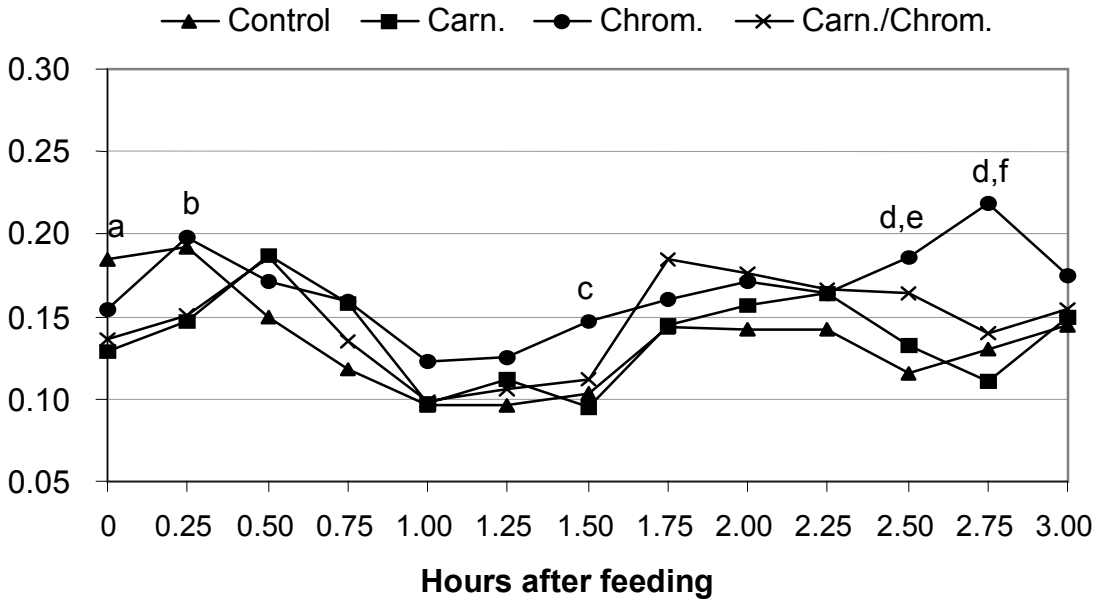

Figure 8. Influence of Carnitine and(or) Chromium on NEFA (mmol/L).

[a]Control > Carn. and Carn./Chrom.; P<0.05. [b]Chrom. > Carn. and Carn./Chrom.; P<0.05. [c]Chrom. > Carn.; P<0.05. [d]Chrom. > Control and Carn.; P<0.05. [e]Carn./Chrom. > Control.; P<0.05. [f]Chrom. > Carn./Chro.; P<0.05.

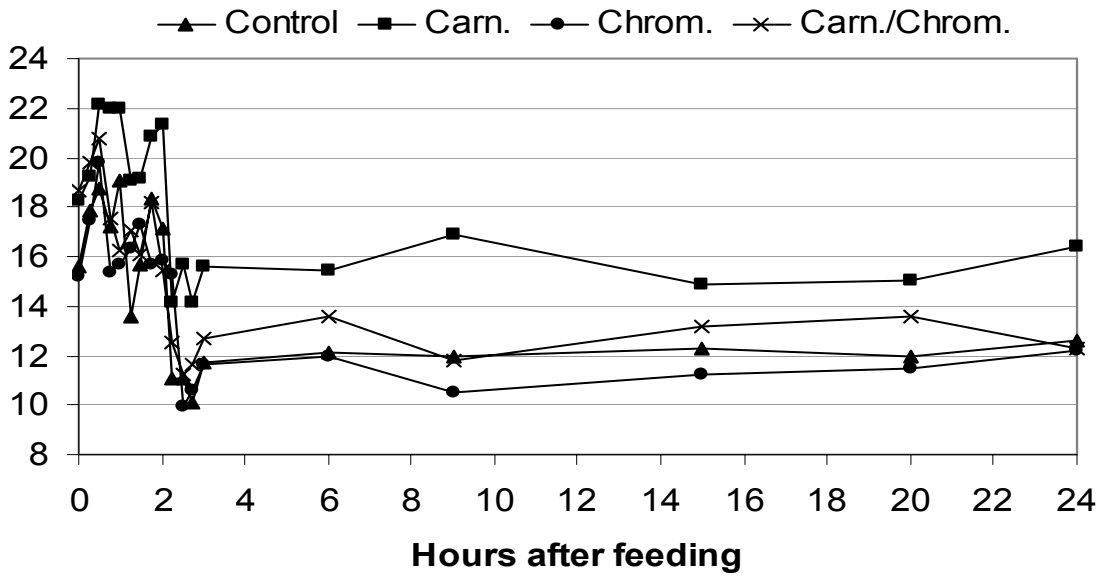

Figure 9. Influence of Carnitine and(or) Chromium on IGF-1 (nmol/L).

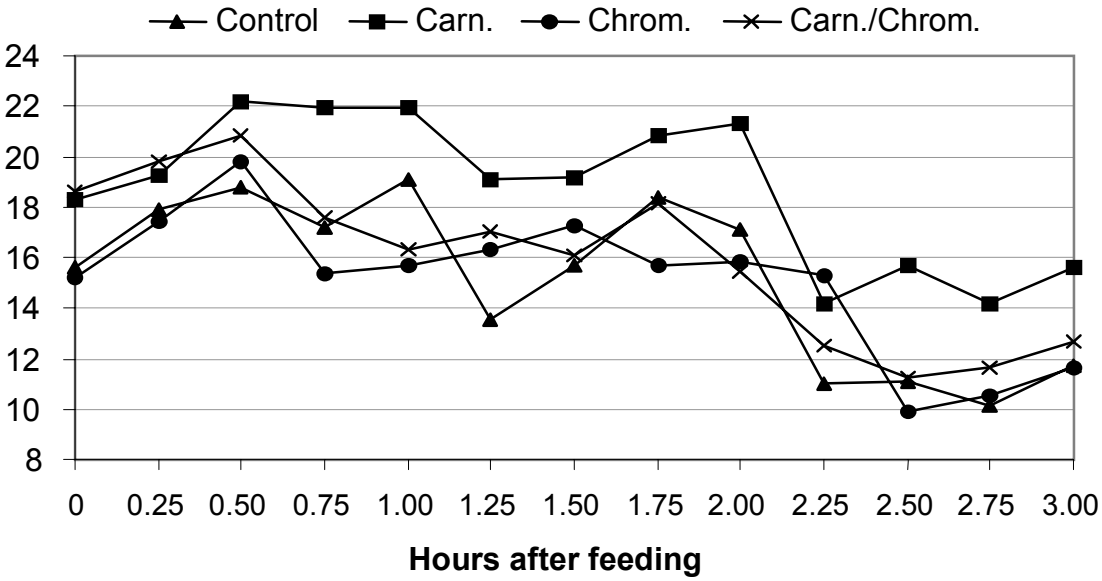

Figure 10. Influence of Carnitine and(or) Chromium on IGF-1 (nmol/L).

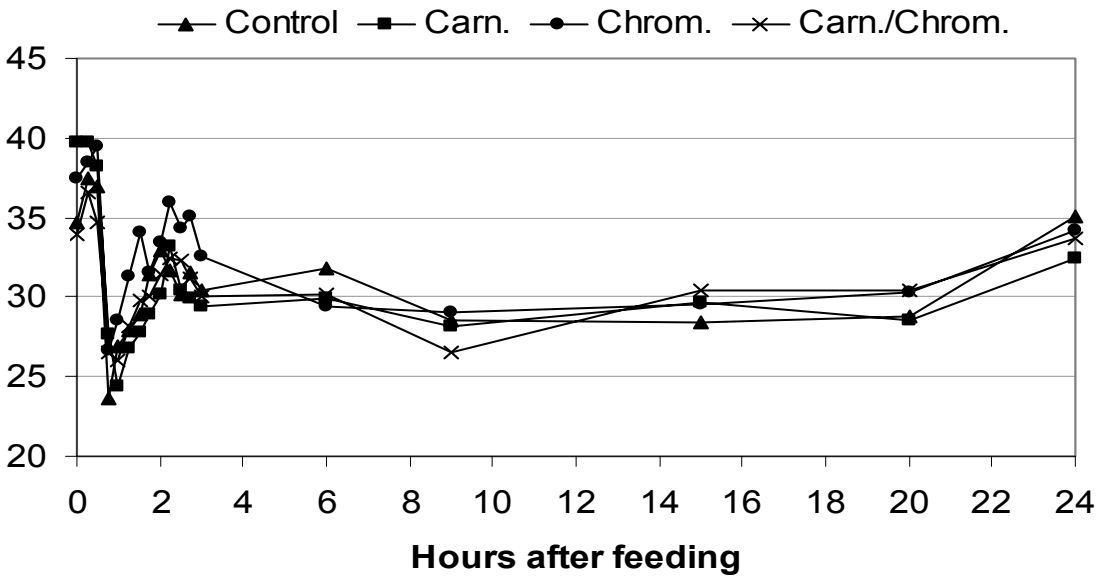

Figure 11. Influence of Carnitine and(or) Chromium on Glucagon (pmol/L).

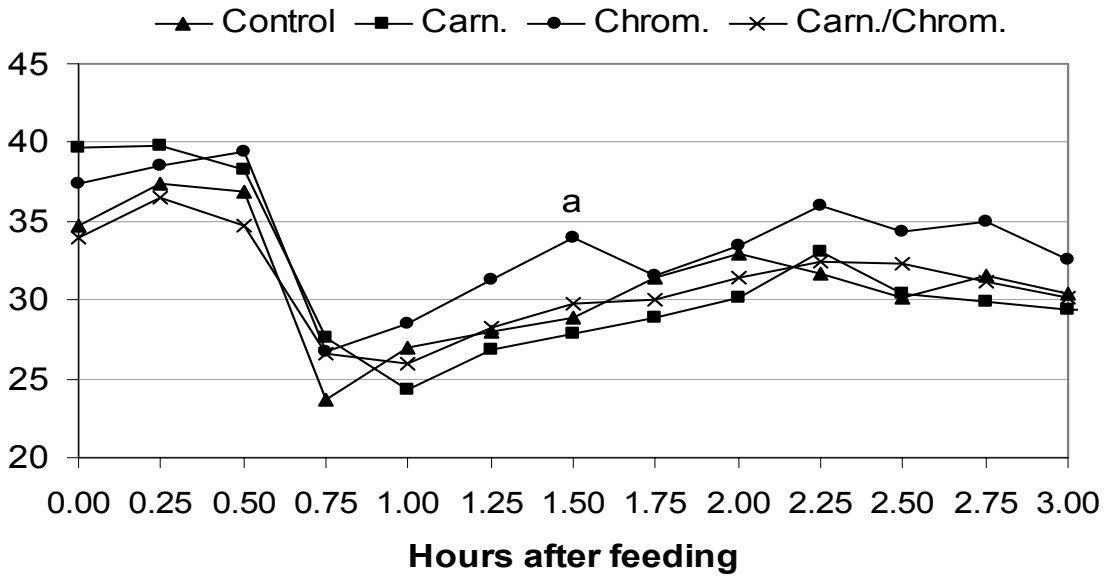

Figure 12. Influence of Carnitine and(or) Chromium on Glucagon (pmol/L).

[a]Chrom. > Carn.; P<0.05.

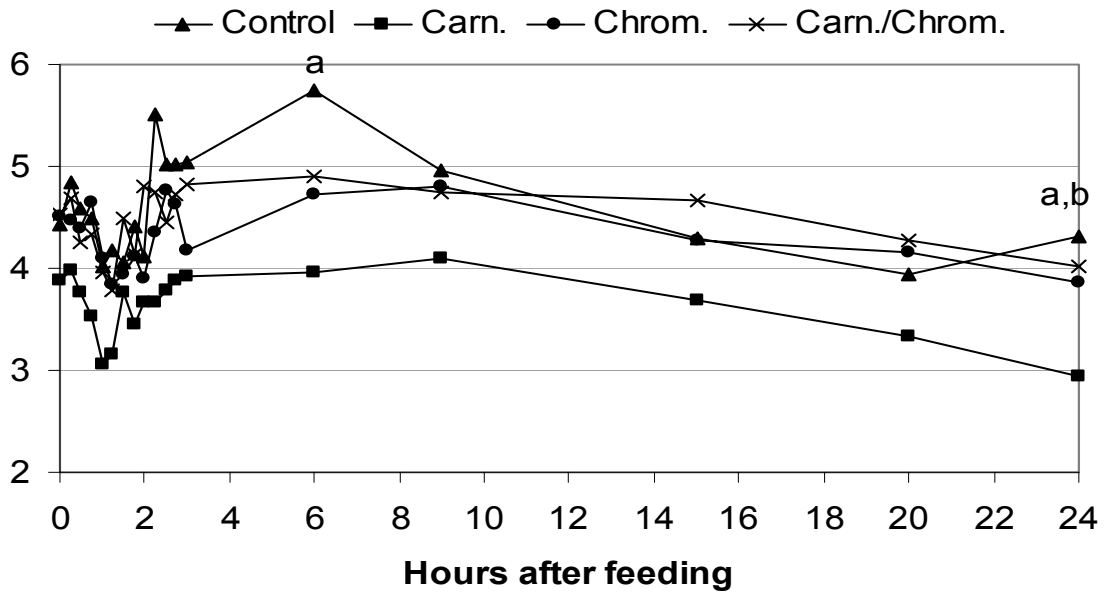

Figure 13. Influence of Carnitine and(or) Chromium on Plasma Urea Nitrogen (mmol/L).

[a]Control > Carn.; P<0.05. [b]Carn./Chrom. > Carn.; P<0.05.

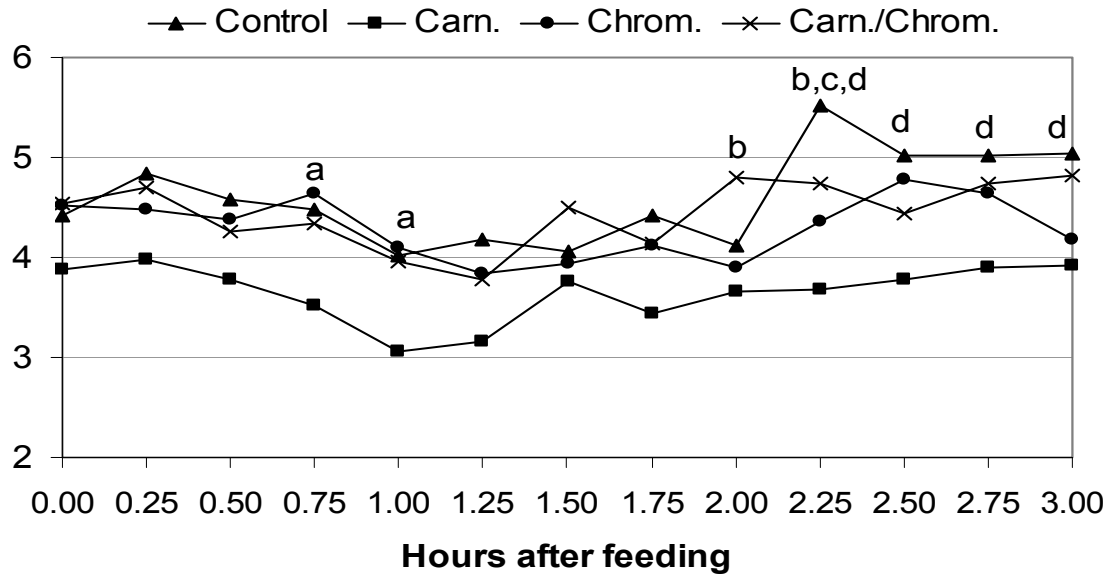

Figure 14. Influence of Carnitine and(or) Chromium on Plasma Urea Nitrogen (mmol/L).

[a]Chrom. > Carn.; P<0.05. [b]Carn./Chrom. > Carn.; P<0.05. [c]Control > Chrom.; P<0.05. [d]Control > Carn.; P<0.05.

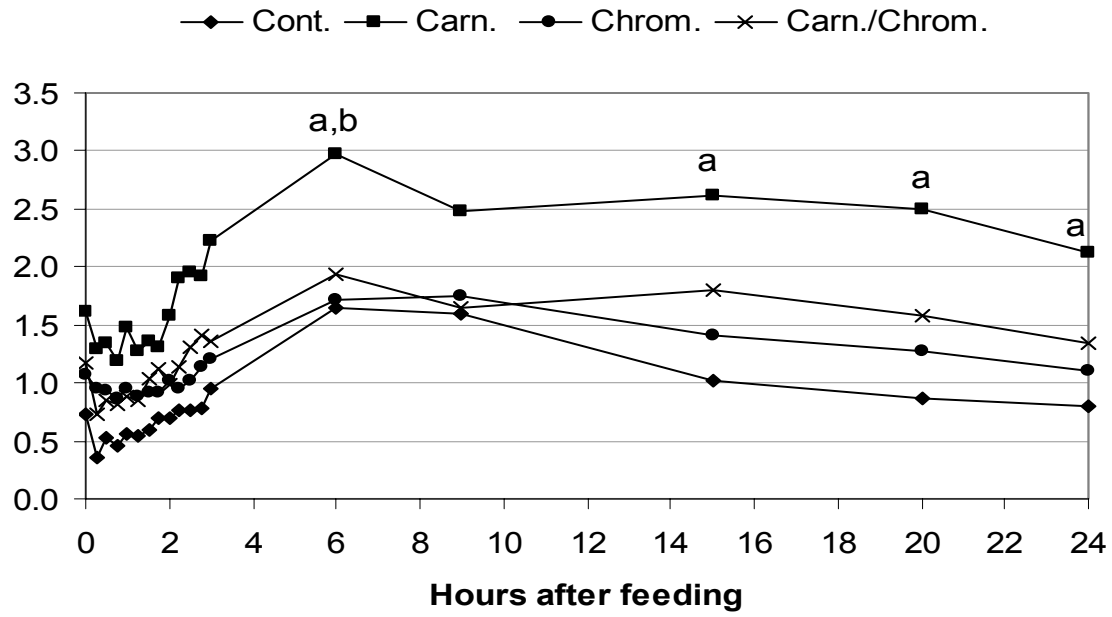

Figure 15. Influence of Carnitine and(or) Chromium on Plasma Leptin (μg/L).

[a]Carn. > Control or Chrom.; P<0.05. [b]Carn. > Carn./Chrom.; P<0.05.

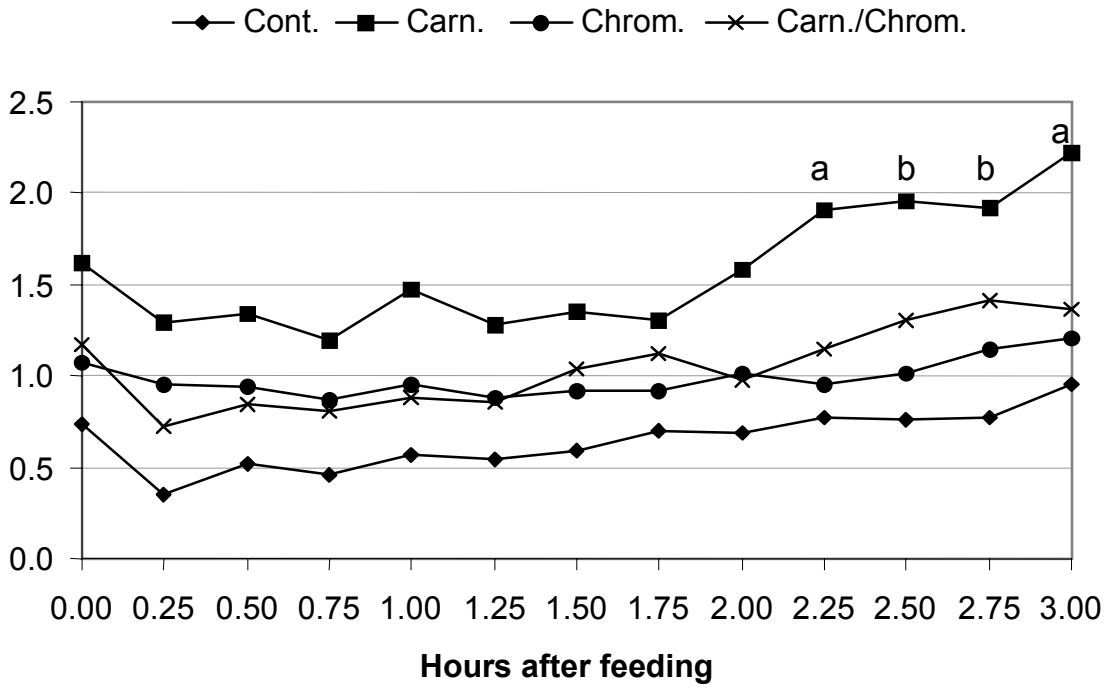

Figure 16. Influence of Carnitine and(or) Chromium on Plasma Leptin (μg/L).

[a]Carn > Control or Chrom.; P<0.05. [b]Carn. > Control; P<0.05.

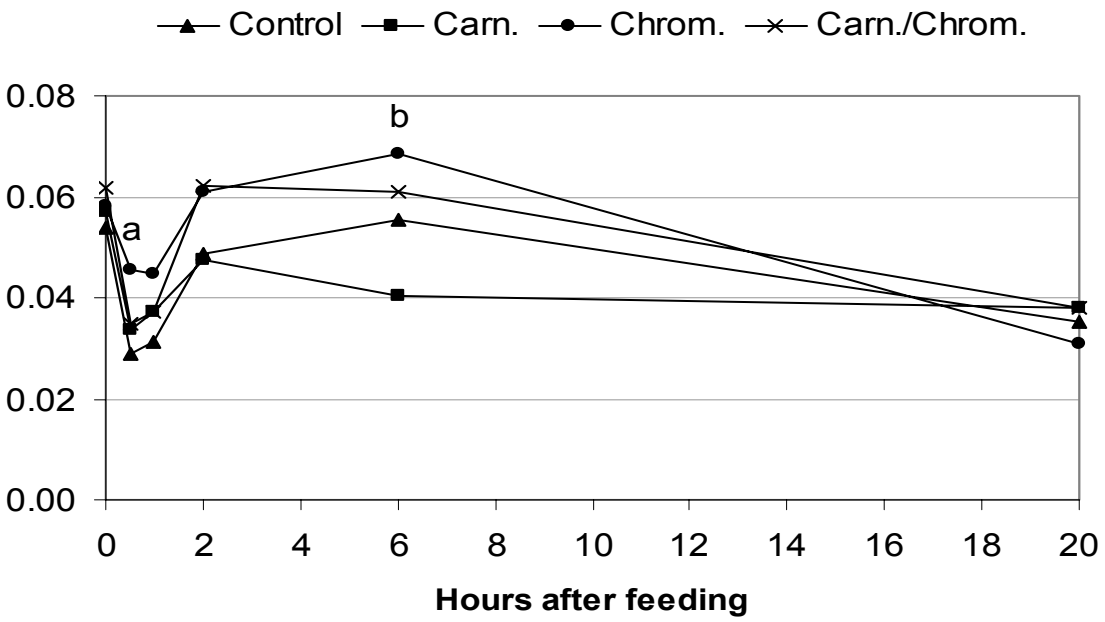

Figure 17. Influence of Carnitine and(or) Chromium on Glycerol (mmol/L).

[a]Chrom. > Control; P<0.05. [b]Chrom. and Carn./Chrom. > Carn.; P<0.05.

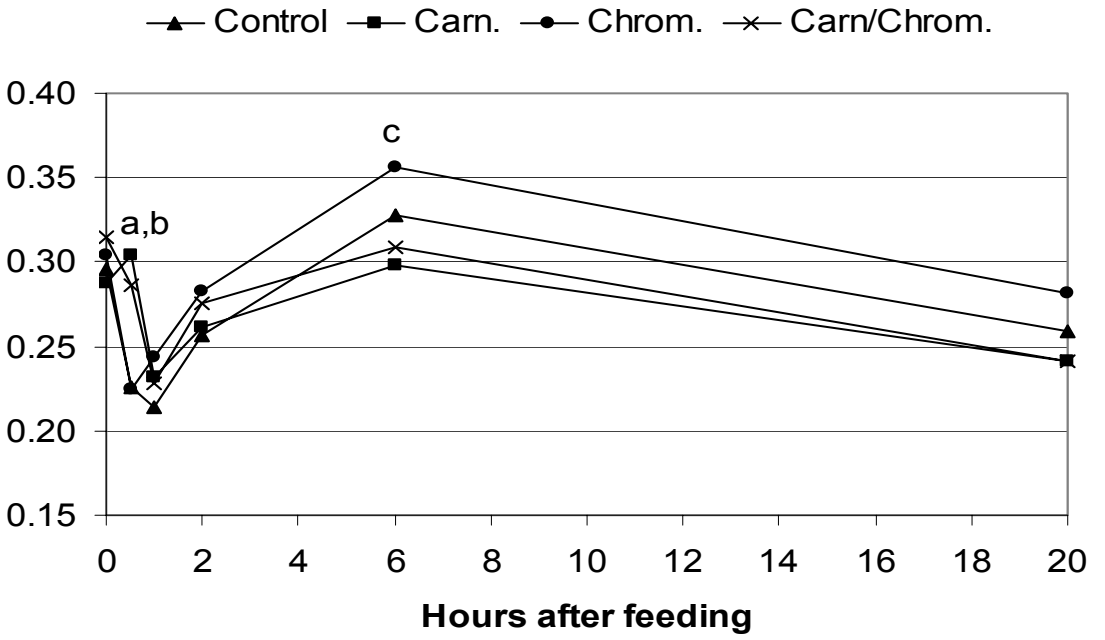

Figure 18. Influence of Carnitine and(or) Chromium on Triglyceride (mmol/L).

[a]Carn. and Carn./Chrom > Control.; P<0.05. [b]Carn. and Carn./Chrom. > Chrom.; P<0.05. [c]Chrom. > Carn.; P<0.05.

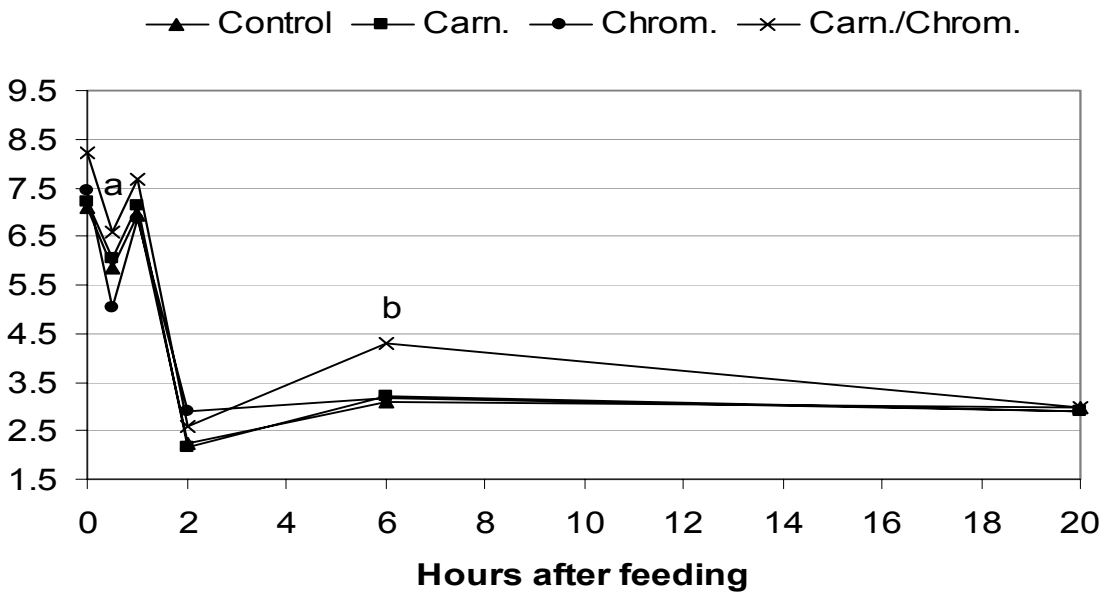

Figure 19. Influence of Carnitine and(or) Chromium on IGF Binding Protein-3 (nmol/L).

[a]Carn. and Carn./Chrom. > Chrom.; P<0.05. [b]Carn./Chrom. > Control and Chrom.; P<0.05.