# JOURNAL OF ANIMAL SCIENCE

**Effects of camelina meal supplementation on ruminal forage degradability, performance, and physiological responses of beef cattle**
B. I. Cappellozza, R. F. Cooke, D. W. Bohnert, G. Cherian and J. A. Carroll

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://www.journalofanimalscience.org/content/90/11/4042

American Society of Animal Science

www.asas.org

# Effects of camelina meal supplementation on ruminal forage degradability, performance, and physiological responses of beef cattle[1,2,3]

**B. I. Cappellozza,* R. F. Cooke,\*[4] D. W. Bohnert,* G. Cherian,† and J. A. Carroll‡**

*Oregon State University - Eastern Oregon Agricultural Research Center, Burns 97720; †Department of Animal Science, Oregon State University, Corvallis 97331; and ‡USDA – ARS, Livestock Issues Research Unit, Lubbock, TX 79403

**ABSTRACT:** Three experiments compared ruminal, physiological, and performance responses of beef steers consuming hay ad libitum and receiving grain-based supplements without (control) or with (CAM) the inclusion of camelina meal. In Exp. 1, 9 steers fitted with ruminal cannulas received CAM (2.04 kg of DM/d; n = 5) or control (2.20 kg of DM/d; n = 4). Steers receiving CAM had reduced ($P = 0.01$) total DMI and tended to have reduced ($P = 0.10$) forage DMI compared with control. No treatment effects were detected ($P \geq 0.35$) for ruminal hay degradability parameters. In Exp. 2, 14 steers fed CAM (1.52 kg of DM/d; n = 7) or control (1.65 kg of DM/d; n = 7) were assigned to a corticotropin-releasing hormone (CRH; 0.1 μg/kg of BW) and a thyrotropin-releasing hormone (TRH; 0.33 μg/kg of BW) challenge. Steers fed CAM had greater ($P < 0.05$) serum concentrations of PUFA compared with control before challenges. Upon CRH infusion, plasma haptoglobin concentrations tended ($P = 0.10$) to be reduced and ceruloplasmin concentrations increased at a lesser rate in CAM steers compared with control (treatment × time; $P < 0.01$). Upon TRH infusion, no treatment effects were detected ($P \geq 0.55$) for serum thyrotropin-stimulating hormone, triiodothyronine, and thyroxine. In Exp. 3, 60 steers were allocated to 20 pens. Pens were assigned randomly to receive CAM (2.04 kg of DM/steer daily; n = 10) or control (2.20 kg of DM/steer daily; n = 10) during preconditioning (PC; d −28 to 0). On d 0, steers were transported for 24 h. Upon arrival, pens were assigned randomly to receive CAM or control during feedlot receiving (FR; d 1 to 29). During PC, CAM steers had reduced ($P < 0.01$) forage and total DMI, and tended to have reduced ($P = 0.10$) ADG compared with control. Plasma linolenic acid concentrations increased during PC for CAM steers, but not for control (treatment × day; $P = 0.02$). During FR, steers fed CAM during PC had reduced ($P < 0.01$) forage and total DMI, but tended ($P = 0.10$) to have greater G:F compared with control. Steers fed CAM during FR had greater ($P < 0.05$) plasma concentrations of PUFA, and reduced rectal temperature and concentrations of haptoglobin and ceruloplasmin during FR compared with control. In summary, CAM supplementation to steers impaired forage and total DMI, did not alter thyroid gland function, increased circulating concentrations of PUFA, and lessened the acute-phase protein reaction elicited by neuroendocrine stress responses.

**Key words:** beef cattle, camelina meal, inflammation, performance, thyroid gland, transport

## INTRODUCTION

Stressful events, such as transportation and feedlot entry, stimulate the acute-phase response in cattle (Cooke et al., 2011). This immune response, although an important component of the innate system (Carroll and Forsberg, 2007), has been negatively associated with performance traits (Araujo et al., 2010). Hence, strategies to lessen the stress-induced acute-phase response are beneficial to cattle performance and overall efficiency of beef operations (Duff and Galyean, 2007).

Our group demonstrated that PUFA supplementation to feeder steers alleviated the acute-phase response stimulated by transport and feedlot entry (Araujo et al., 2010; Cooke et al., 2011), resulting in enhanced feedlot performance (Cooke et al., 2011). Camelina meal, a byproduct from the mechanical processing of *Camelina sativa* seeds for oil extraction, may contain up to 20% oil with the majority of the fatty acid content as PUFA (Hurtaud and Peyraud, 2007), which is substantially greater than the PUFA content in traditional feeds such as soybean meal and corn (Cooke et al., 2011). Therefore, we hypothesized that camelina meal is a nutritional alternative to alleviate the acute-phase response in cattle subjected to stress of management. However, camelina meal contains glucosinolates and erucic acid, which are compounds that impair animal health and welfare (Kramer et al., 1990; Mawson et al., 1994). In addition, feeding PUFA during preconditioning (**PC**) or feedlot receiving (**FR**) reduced DMI in feeder calves (Araujo et al., 2010; Cooke et al., 2011). To address our main hypothesis as well as potential health and DMI concerns, 3 experiments were conducted to evaluate the inclusion of camelina meal into beef cattle diets. Experiment 1 evaluated DMI and digestibility parameters in fistulated beef steers. Experiment 2 assessed acute-phase and thyroid responses in beef steers after hormonal challenges. Experiment 3 evaluated performance and physiological responses of beef steers during PC and FR.

## MATERIALS AND METHODS

All experiments were conducted at the Oregon State University Eastern Oregon Agricultural Research Center, Burns, OR. All animals used were cared for in accordance with acceptable practices and experimental protocols reviewed and approved by the Oregon State University Institutional Animal Care and Use Committee.

Supplement ingredients provided during Exp. 1, 2, and 3 were from the same batch. Similarly, hay provided during Exp. 1, 2, and 3 was harvested from the same field during June 2010. All steers were vaccinated against clostridial diseases (Clostrishield 7; Novartis Animal Health; Bucyrus, KS) and bovine virus diarrhea complex (Virashield 6 + Somnus; Novartis Animal Health) at approximately 30 d of age. At weaning, all steers were vaccinated against pasteurella (One Shot Ultra 7; Pfizer Animal Health; New York, NY) and respiratory diseases (Bovi-Shield Gold 5 and TSV-2; Pfizer Animal Health), and were also administered an anthelmintic (Dectomax; Pfizer Animal Health).

### Animals

***Exp. 1.*** Nine Angus x Hereford steers (initial BW 250 ± 9 kg; initial age 499 ± 5 d), housed in individual pens (11 × 21 m) and fitted with ruminal cannulas, were assigned on d 0 of the study to receive a grain-based supplement with (**CAM**; n = 5) or without (**control;** n = 4) the inclusion of camelina meal (Table 1) for 21 d.

***Exp. 2.*** Fourteen weaned Angus steers (initial BW 191 ± 2 kg; initial age 178 ± 4 d) were used in the study (d 0 to 36). All steers were weaned 30 d before the beginning of the study and exposed daily to halter-training techniques to become acclimated to human interaction; thus, minimizing confounding effects between human handling, weaning, and hormones challenges measured herein (Arthington et al., 2005; Curley et al., 2008; Cooke and Bohnert, 2011). Beginning 5 d before and during the study, steers were housed in individual pens (2 × 5 m) contained within an enclosed barn. On d 0, steers were ranked by initial BW in a decreasing order, and alternatingly assigned to receive control or CAM (7 steers/treatment) so both groups had similar initial BW (CAM = 191 ± 2 kg, control = 191 ± 3 kg).

***Exp. 3.*** Sixty Angus × Hereford steers (BW = 221 ± 2 kg; initial age 182 ± 1 d), weaned 35 d before the beginning of the study, were ranked by initial BW (d −28 of the study) in a decreasing order, and alternatingly allocated to 20 pens (3 steers/pen) so all pens had similar initial BW (data not shown). Pens were assigned randomly to receive CAM or control during the PC phase (d −28 to 0; Table 1). On the morning of d 0, steers were combined into 1 group, loaded into a commercial livestock trailer, transported for 24 h to simulate the stress challenge of a long-haul transport (approximately 1,500 km; Arthington et al., 2008), and returned to the research facility (d 1). Upon arrival, pens receiving CAM or control during PC were assigned randomly to receive, in a 2 × 2 factorial arrangement, CAM or control during a 28-d FR phase (10 pens/treatment). As a result, 4 treatment combinations were generated (5 pens/treatment combination): 1) control during PC and FR, 2) CAM during PC and FR, 3) CAM during PC and control during FR, and 4) control during PC and CAM during FR. The physical arrangement of pens within the research facility also was randomized upon arrival so calves were exposed, at least partially, to a new environment. Steers received a booster vaccination against pasteurella (One Shot Ultra 7; Pfizer Animal Health) and respiratory diseases (Bovi-Shield Gold 5 and TSV-2; Pfizer Animal Health) at the beginning of the study (d −28). No incidences of mortality or morbidity were observed during the entire experiment.

**Table 1.** Ingredient composition, nutrient profile, and intake of grain-based supplements containing (CAM) or not containing (control) camelina meal offered during Exp. 1, 2, and 3

| | Exp. 1 and 3 | | Exp. 2 | |
|---|---|---|---|---|
| Item | Control | CAM | Control | CAM |
| Ingredients, % DM | | | | |
| Corn | 82.0 | 67.2 | 82.0 | 67.2 |
| Soybean meal | 14.6 | — | 14.6 | — |
| Camelina meal | — | 30.2 | — | 30.2 |
| Mineral and vitamin mix[1] | 3.4 | 3.4 | 3.4 | 3.4 |
| Nutrient Profile[2], DM basis | | | | |
| $NE_g$,[3] Mcal/kg | 1.46 | 1.54 | 1.46 | 1.54 |
| $NE_m$,[3] Mcal/kg | 2.01 | 2.11 | 2.01 | 2.11 |
| CP, % | 15.5 | 16.7 | 15.5 | 16.7 |
| NDF, % | 13.9 | 19.5 | 13.9 | 19.5 |
| Ether extract, % | 5.20 | 9.70 | 5.20 | 9.70 |
| Linoleic acid, % | 2.60 | 3.38 | 2.60 | 3.38 |
| Linolenic acid, % | 0.07 | 1.15 | 0.07 | 1.15 |
| PUFA, % | 2.67 | 4.53 | 2.67 | 4.53 |
| SFA + MUFA | 2.27 | 3.32 | 2.27 | 3.32 |
| Ca, % | 0.47 | 0.66 | 0.47 | 0.66 |
| P, % | 0.62 | 0.82 | 0.62 | 0.82 |
| Daily intake[4] | | | | |
| DM, kg | 2.20 | 2.04 | 1.65 | 1.52 |
| $NE_g$,[3] Mcal | 3.21 | 3.14 | 2.41 | 2.54 |
| $NE_m$,[3] Mcal | 4.42 | 4.30 | 3.32 | 3.48 |
| CP, kg | 0.34 | 0.34 | 0.26 | 0.28 |
| NDF, kg | 0.31 | 0.39 | 0.23 | 0.32 |
| Ether extract, kg | 0.11 | 0.19 | 0.09 | 0.16 |
| Linoleic acid, g | 57.2 | 68.9 | 42.9 | 55.8 |
| Linoleic acid, g | 1.54 | 23.5 | 1.16 | 19.0 |
| PUFA, g | 58.7 | 92.4 | 44.1 | 74.7 |
| SFA + MUFA, g | 49.9 | 67.7 | 37.5 | 54.8 |
| Ca, g | 10.3 | 13.4 | 7.8 | 10.9 |
| P, g | 13.6 | 16.7 | 10.2 | 13.5 |

[1]Cattleman's Choice (Performix Nutrition Systems; Nampa, ID); contained 14% Ca, 10% P, 16% NaCl, 1.5% Mg, 3.2 g/kg Cu, 0.07g/kg I, 0.9 g/kg Mn, 0.14 g/kg Se, 6.0 g/kg Zn, 136 IU/g of vitamin A, 13 IU/g of vitamin D$_3$, and 0.05 IU/g of vitamin E.

[2]Values obtained from a commercial laboratory wet chemistry analysis (Dairy One Forage Laboratory, Ithaca, NY). Fatty acid content was determined based on Table 2, and according to the procedures described by Folch et al. (1957) and Hayat et al. (2009).

[3]Calculated with the following equations (NRC 1996): $NE_g$ = 1.42 ME − 0.174 ME$^2$ + 0.0122 ME$^3$ − 0.165; $NE_m$ = 1.37 ME − 0.138 ME$^2$ + 0.0105 ME$^3$ − 1.12. Given that ME = 0.82 × DE, and 1 kg of TDN = 4.4 Mcal of DE.

[4]Estimated from the concentrate consumption of individual experimental unit.

### Diets

During all experiments, mixed alfalfa-grass hay and water were offered in amounts to ensure ad libitum consumption, and treatments and hay were not mixed. Camelina meal was included in CAM to completely substitute soybean meal, and its rate of inclusion was selected to result in similar nutritional content among

**Table 2.** Fatty acid profile of feedstuffs offered to cattle during Exp. 1, 2, and 3[1]

| Fatty acid, % | Corn | Soybean meal | Camelina meal | Hay |
|---|---|---|---|---|
| Palmitic acid (16:0) | 11.4 | 16.0 | 8.3 | 21.8 |
| Stearic acid (18:0) | 2.1 | 5.0 | 2.9 | 5.8 |
| Oleic acid (18:1) | 32.8 | 14.1 | 21.7 | 11.3 |
| Linoleic acid (18:2$n$-6) | 52.5 | 56.4 | 28.8 | 13.8 |
| Linolenic acid (18:3$n$-3) | 1.3 | 8.5 | 24.2 | 26.0 |
| Erucic acid (22:1 $n$-9) | 0.00 | 0.00 | 0.77 | 0.00 |

[1]As % of total fatty acids. All feedstuffs were analyzed for fatty acid content according to the procedures described by Folch et al. (1957) and Hayat et al. (2009).

treatments and allow iso-caloric and iso-nitrogenous intakes without major changes in treatment consumption (Table 1). Samples of treatment ingredients were collected before the beginning of all experiments, whereas samples of hay were collected weekly during each experiment. Samples were analyzed for nutritional content (Table 1) by a commercial laboratory (Dairy One Forage Laboratory, Ithaca, NY) using wet chemistry procedures for concentrations of ether extract (method 2003.05; AOAC 2006), CP (method 984.13; AOAC 2006), ADF (method 973.18 modified for use in an Ankom 200 fiber analyzer, Ankom Technology Corp., Fairport, NY; AOAC 2006), and NDF (Van Soest et al., 1991; method for use in an Ankom 200 fiber analyzer, Ankom Technology Corp.). Calculations of TDN used the equation proposed by Weiss et al. (1992), whereas $NE_m$ and $NE_g$ were calculated with the equations proposed by the NRC (1996) derived from feed TDN (Weiss et al., 1992). Further, hay and supplement samples were analyzed for fatty acid content using gas chromatography (Model HP 6890, Hewlett-Packard Co., Wilmington, DE) according to the procedures described by Folch et al. (1957) and Hayat et al. (2009). Hay quality was (DM basis; ± SE) 54 ± 0% TDN, 60.5 ± 0.6% NDF, 40.3 ± 0.8% ADF, 1.15 ± 0 Mcal/kg of $NE_m$, 0.59 ± 0 Mcal/kg of $NE_g$, 12.8 ± 0.3% CP, and 2.1 ± 0.2% ether extract. Hay fatty acid profile is described in Table 2.

*Exp. 1.* Treatments were offered daily (0700 h) at 2.20 and 2.04 kg of DM/steer for control and CAM, which corresponded to 0.89 and 0.80% of initial shrunk (after 16 h of feed and water restriction) BW, respectively. Treatments were inserted directly into the rumen of each steer via fistula to ensure complete consumption.

*Exp. 2.* Treatments were fed daily (0800 h) at 1.65 and 1.52 kg of DM/steer for control and CAM, which corresponded to 0.96 and 0.88% of initial shrunk (after 16 h of feed and water restriction) BW, respectively. Treatments were readily consumed by steers after feeding.

*Exp. 3.* Treatments were fed daily (0700 h) at the same rate as in Exp. 1, corresponding to 0.99 and 0.92% of initial shrunk (after 16 h of feed and water restric-

tion) BW for control and CAM, respectively. Treatments were readily consumed by steers after feeding.

### *Sampling*

*Exp. 1.* Steer shrunk BW was recorded 7 d before the beginning of the experiment to calculate treatment intake. Voluntary forage intake was evaluated daily from d 1 to 15, whereas intake data from d 8 to 15 were used for treatment comparison. From d 1 to 15, hay refusals were collected and weighed daily. Samples of the offered and the non-consumed hay were collected daily from the feed bunk within each pen and dried for 96 h at 50°C in forced-air ovens for DM calculation. Initial shrunk BW was used for calculation of DMI as % of BW. From d 16 to 19, steers were offered 90% of their voluntary forage DMI determined from d 1 to 15. Immediately before treatment feeding on d 16, Dacron bags (50 ± 10 μm pore size and 10 × 20 cm bag size; Ankom Technology Corp.) containing 4 g (DM basis) of ground dietary hay (2-mm screen; Wiley Mill, Model 4; Arthur H. Thomas, Philadelphia, PA) were suspended in the ruminal ventral sac of each steer, and incubated in triplicate for 0, 1, 3, 5, 8, 12, 24, 36, 48, 72, and 96 h. Before ruminal incubation, all bags were soaked in warm water (39°C) for 15 min. After ruminal incubation, bags were washed repeatedly with running water until the rinse water was colorless, and subsequently dried for 96 h at 50°C in a forced-air oven. The 0-h bags were not incubated in the rumen but were subjected to the same soaking, rinsing, and drying procedures applied to the ruminally incubated bags. Dried samples were weighed for residual DM determination, and then triplicates were combined and analyzed for NDF (Robertson and Van Soest, 1981) using procedures modified for use in an Ankom 200 Fiber Analyzer (Ankom Technology Corp.). After removal of the last Dacron bags on d 19, steers were allowed to consume hay ad libitum until the end of the study. On d 21, blood samples were collected via jugular venipuncture at 0, 1, 2, 3, 4, 5, 6, 9, and 12 h relative to treatment feeding (0 h) for determination of plasma cholecystokinin concentrations.

*Exp. 2.* From d 21 to 23 and d 31 to 33, steers were weighed daily. On d 24 and 34 of the study, all steers were fitted with an indwelling jugular vein catheter for serial blood collection and dosing of hormones according to procedures previously described (Merrill et al., 2007; Cooke et al., 2011), and an indwelling rectal temperature monitoring device (d 24 only) as described by Reuter et al. (2010). On d 25, all steers received 0.1 μg of bovine corticotropin-releasing hormone (**CRH**; #34-3-11; American Peptide Co., Inc., Sunnyvale, CA) per kilogram of BW to stimulate and evaluate treatment effects on a stress-induced acute-phase protein response (Cooke and Bohnert, 2011). On d 35, all steers received

0.33 μg of bovine thyrotropin-releasing hormone (**TRH**; #52-0-80; American Peptide Co., Inc.) per kilogram of BW to stimulate and evaluate treatment effects on thyroid function (Wichtel et al., 1996).

After CRH (0 h; d 25) and TRH infusions (0 h; d 35), blood samples were collected hourly from −2 to 0 h and 4 to 8 h, and every 30 min from 0 to 4 h, via jugular catheters. Catheters were removed immediately after the collection at 8 h after infusions. Blood samples were collected via jugular venipuncture every 6 h from 12 to 72 h and every 24 h from 96 to 168 h after CRH infusion, and every 4 h from 12 to 24 h after TRH infusion. All samples collected during the CRH challenge were analyzed for plasma concentrations of ceruloplasmin and haptoglobin. Samples collected from -2 to 8 h relative to CRH challenge were also analyzed for plasma concentrations of cortisol. All samples collected during the TRH challenge were analyzed for serum concentrations of thyrotropin-stimulating hormone (**TSH**), triiodothyronine ($T_3$), and thyroxine ($T_4$). Samples collected at −2 h before CRH and TRH challenges were analyzed for plasma fatty acid content. Rectal temperatures were recorded by the indwelling device (Reuter et al., 2010) every 30 min from −2 to 8 h relative to CRH infusion, and rectal probes were removed immediately after the collection at 8 h after infusion. Rectal temperatures were assessed using a digital thermometer (GLA M750 digital thermometer; GLA Agricultural Electronics, San Luis Obispo, CA) every 6 h from 12 to 72 h, and every 24 h from 96 to 168 h after CRH infusion.

*Exp. 3.* Steer shrunk BW was collected before the beginning (d −31) and at the end of the study (d 30) after 16 h of feed and water restriction. Shrunk BW also was recorded on d 1 immediately after unloading. Preconditioning ADG was determined using values obtained on d −31 and 1, whereas ADG during FR was determined using values obtained on d 1 and 30 of the study.

Throughout the experimental period, pen voluntary forage and total DMI were recorded daily. Hay and treatment orts were collected from each pen daily, weighed, and dried for 96 h at 50°C in forced-air ovens for DM calculation. Estimated duodenal flow of linoleic and linolenic acids during the study were calculated based on treatment and hay intake of each pen, feed nutritional analysis, and the CPM-Dairy model (Cornell-Penn-Miner Dairy; Version 3.08.01; Univ. of Pennsylvania, Kennett Square; Cornell Univ., Ithaca, NY; and William H. Miner Agric. Res. Inst., Chazy, NY), which has been shown to adequately estimate intestinal fatty acid flow in cattle (Moate et al., 2004). Average shrunk BW during PC (values obtained on d −31 and d 1) and FR phase (values obtained on d 1 and d 30) were used for calculation of DMI as % of BW during each respective period. Total BW gain achieved during PC and FR phases was divid-

ed by total DMI consumed during each phase for G:F calculation. Blood samples were collected via jugular venipuncture on d −28, −15, 0, 1, 4, 7, 10, 14, 21, and 29 of the study. Samples collected from d 0 to 29 were analyzed for plasma concentrations of cortisol, haptoglobin and ceruloplasmin. Blood samples collected on d −28, −15, 0, 4, 14, and 29 also were analyzed for plasma concentrations of fatty acids. Steer rectal temperature was measured at 30-min interval with an automatic recording device (Reuter et al., 2010) during transport, whereas on d 4 and 7 rectal temperature was measured with a digital thermometer (GLA M750 Digital Thermometer) concurrently with blood collection.

### Blood Analysis

Blood samples obtained via jugular venipuncture in all experiments were collected into commercial blood collection tubes (Vacutainer, 10 mL; Becton Dickinson, Franklin Lakes, NJ) containing sodium heparin, and were placed on ice immediately. Blood samples that were obtained via jugular catheters in Exp. 2 were immediately transferred into commercial blood collection tubes (Vacutainer, 10 mL; Becton Dickinson) that did not contain additives for serum collection, or contained sodium heparin for plasma collection. All blood samples were placed immediately on ice, centrifuged at $2,500 \times g$ at 4°C for 30 min for plasma or serum harvest, and stored at −80°C on the same day of collection.

*Exp. 1.* Plasma concentrations of cholecystokinin were determined using a bovine-specific commercial ELISA kit (KT-10170; Kamyia Biomedical Company, Seattle, WA). The intraassay CV was 4.2%.

*Exp. 2.* Plasma concentrations of cortisol were determined using a bovine-specific commercial ELISA kit (Endocrine Technologies Inc., Newark, CA). Plasma concentrations of ceruloplasmin and haptoglobin were determined according to procedures previously described (Demetriou et al., 1974; Makimura and Suzuki, 1982) and used by our research group (Cooke and Bohnert, 2011; Cooke et al., 2011). Serum concentrations of TSH, $T_3$, and $T_4$ were determined using a bovine-specific commercial ELISA kit (Endocrine Technologies Inc., Newark, CA). The intra- and interassay CV were, respectively, 9.5 and 8.4% for $T_3$, 11.2 and 8.1% for $T_4$, 4.5 and 8.12% for TSH, 0.1 and 10.6% for haptoglobin, 4.5 and 7.5% for ceruloplasmin, and 3.4 and 9.2% for cortisol. Serum samples also were analyzed for fatty acid content according to the procedures described by Folch et al. (1957) and Hayat et al. (2009).

*Exp. 3.* Plasma concentrations of cortisol, haptoglobin, ceruloplasmin, and fatty acids were determined using the same procedures as described in Exp. 2. The intra- and interassay CV were, respectively, 1.8 and 9.2%

for haptoglobin, 3.7 and 8.4% for ceruloplasmin, and 4.3 and 15.4% for cortisol.

### Statistical Analysis

Data collected in all experiments were analyzed using the MIXED procedure (SAS Inst., Inc., Cary, NC) and Satterthwaite approximation to determine the denominator degrees of freedom for the tests of fixed effects, unless stated otherwise. Results are reported as least square means and separated using LSD. Results are reported according to treatment effects if the significance level of interactions containing the effects of treatments and time variables were $P > 0.10$.

*Exp. 1.* All data were analyzed using steer as the experimental unit. Kinetic parameters of hay DM and NDF disappearance were estimated using nonlinear regression procedures of SAS proposed by Ørskov and McDonald (1979) and described by Vendramini et al. (2008). The model statements used for plasma cholecystokinin concentrations, forage DMI, and total DMI contained the effects of treatment, time, and the resultant interaction. Data were analyzed using steer(treatment) as the random variable. The specified term for the repeated statement was the time variable, the subject was steer(treatment), and the covariance structure used was first-order autoregressive, which provided the best fit for these analyses according to the Akaike Information Criterion. The model statement used for ruminal in situ forage degradability parameters contained the effect of treatment. Data were analyzed using steer(treatment) as random variable.

*Exp. 2.* All data were analyzed using steer as experimental unit. Serum fatty acid profile from samples collected at −2 h before the CRH (d 25) and TRH (d 35) challenges were analyzed jointly given that steers received the same treatments throughout the experiment (d 0 to 36). This model statement contained the effects of treatment, day, and the interaction. The model statement for all other physiological data contained the effects of treatment, time, and the interaction. All data were analyzed using steer(treatment) as the random variable. The specified term for the repeated statement was the time variable, steer(treatment) was included as subject, and covariance structure used was first-order autoregressive based on the Akaike information criterion.

*Exp. 3.* All data were analyzed using pen as the experimental unit. The model statement used for ADG and G:F during PC contained the effects of PC treatment. Data were analyzed using pen(PC treatment) as the random variable. The model statement for ADG and G:F during FR contained the effects of PC treatment, FR treatment, and the resultant interaction. Data were analyzed using pen(PC × FR treatment) as the

random variable. The model statements used for DMI analyses during PC and FR were similar to models used for ADG, but also contained the effects of day, all resultant interactions, in addition to day as specified term for repeated statement, pen(treatment) as subject, and first-order autoregressive covariance structure based on the Akaike information criterion. The model statement used for fatty acid data obtained on d −28, −15, and 0, as well as rectal temperature, cortisol, ceruloplasmin, and haptoglobin data obtained on d 0 and 1 of the study contained the effects of PC treatment, day, and the resultant interaction because steers were assigned to their FR treatment after sampling on d 1. Data were analyzed using pen(PC treatment) and steer(pen) as the random variables. The model statement used for rectal temperature, cortisol, ceruloplasmin, fatty acids, and haptoglobin data obtained from d 4 to 29 contained the effects of PC treatment, FR treatment, day, and all the resultant the interactions. Data were analyzed using pen(PC × FR treatment) and steer(pen) as random variables. The specified term for the repeated statement in the analysis of variables collected from steers during PC and FR was day, the subject was steer(pen), and the covariance structure used was first-order autoregressive based on the Akaike Information Criterion.

## RESULTS AND DISCUSSION

### Experiment 1

Steers receiving CAM had reduced ($P$ = 0.01; data not shown) total DMI and tended to have reduced ($P$ = 0.10; data not shown) voluntary forage DMI compared with control cohorts (2.91 vs. 3.14 and 3.72 vs. 4.04% of BW, respectively; SEM = 0.08), concurring with previous research (Araujo et al., 2010; Cooke et al., 2011) reporting that PUFA supplementation impairs DMI in forage-fed cattle. It is also important to note that, based on DMI and treatment composition, camelina meal was included at 6.3% of diet DM for CAM steers, which is below the inclusion limit established by the FDA (10%; Benz, 2009). Lardy and Kerley (1994) reported that substitution of soybean meal for rapeseed meal, a member of the *Brassicaceae* family as *C. sativa*, reduced DMI in beef steers and attributed these outcomes to reduced palatability of diets containing rapeseed meal. In the present study, treatments were included directly into the rumen of each steer, which eliminated potential concerns associated with diet palatability. One could speculate that reduced DMI in CAM steers was due to impaired forage digestibility (Schauff and Clark, 1989; Drackley et al., 1992) as well as increased cholecystokinin synthesis and release (Allen, 2000). However, no treatment effects were detected ($P \geq 0.35$) for ruminal disappearance rate or effective ruminal degradability of hay DM and NDF

**Table 3.** Plasma concentration of cholecystokinin, and ruminal in situ disappearance parameters of mixed alfalfa-grass hay incubated in forage-fed steers offered grain-based supplements containing (CAM; n = 5) or not containing (control; n = 4) camelina meal in Exp. 1

| Item | Control | CAM | SEM | P-value |
|---|---|---|---|---|
| Plasma cholecystokinin,[1] pg/mL | 26.78 | 22.72 | 2.88 | 0.35 |
| Ruminal degradation rate, %/h | | | | |
| DM | 8.58 | 7.91 | 0.47 | 0.35 |
| NDF | 7.39 | 7.49 | 0.63 | 0.91 |
| Effective degradability,[2] % | | | | |
| DM | 64.9 | 64.3 | 0.8 | 0.57 |
| NDF | 70.9 | 70.1 | 0.9 | 0.55 |

[1]Blood samples were collected at 0, 1, 2, 3, 4, 5, 6, 9, and 12 h relative to treatment feeding (0 h). No treatment × time interaction was detected ($P$ = 0.61).

[2]Calculated by fixing ruminal passage rate at 0.046/h (Poore et al., 1990) and using the model proposed by Ørskov and McDonald (1979).

(Table 3). Further, no treatment effects were detected ($P$ = 0.35) on plasma cholecystokinin concentration (Table 3).

These results support previous research from our group reporting that PUFA supplementation did not impact ruminal in situ forage degradability in beef cattle (Cooke et al., 2011). Hess et al. (2008) indicated that inclusion of supplemental fat up to 3% of diet DM is recommended to maximize the use of forage-based diets and prevent impaired forage digestibility and intake. Moreover, Leupp et al. (2006) reported that ruminal forage digestibility was not affected in forage-fed steers offered supplemental fat at 4% of diet DM. In the present study, according to DMI evaluation and feed nutritional analysis, supplemental fat was included at 2.1 and 1.1% of diet DM for CAM and control, respectively, which supports the lack of treatment effects on ruminal in situ forage degradability parameters. Diet fatty acid profile also modulates circulating concentrations of gut-peptide hormones that mediate DMI in cattle, such as cholecystokinin (Choi and Palmquist, 1996). In fact, diets rich in PUFA appear to influence cholecystokinin concentrations to a greater extent compared with diets rich in SFA (Relling and Reynolds, 2007; Bradford et al., 2008). However, Benson and Reynolds (2001) and Litherland et al. (2005) reported that abomasal infusion of PUFA reduced DMI but did not impact plasma cholecystokinin concentrations in dairy cows compared with SFA or water infusions. Similarly to these latter authors, no treatment effects on plasma cholecystokinin concentrations were detected herein, suggesting that cholecystokinin is not the sole mediator of the decreased DMI in PUFA-supplemented cattle. Nevertheless, camelina meal contains several other anti-nutritional compounds that may impair DMI and were not evaluated herein, such as sinapines, tannins, and saponins (Mawson et al., 1993; Ahlin et al., 1994).

**Table 4.** Serum fatty acid concentrations (mg/mL of plasma) of steers offered grain-based supplements containing (CAM; n = 7) or not containing (control; n = 7) camelina meal during Exp. 2[1]

| Item | Treatments | | | |
| | Control | CAM | SEM | P-value |
| --- | --- | --- | --- | --- |
| Stearic (18:0) | 0.196 | 0.210 | 0.006 | 0.14 |
| Oleic (18:1) | 0.184 | 0.229 | 0.006 | < 0.01 |
| Linoleic (18:2 *n-6*) | 0.196 | 0.230 | 0.009 | 0.02 |
| Linolenic (18:3 *n-3*) | 0.079 | 0.097 | 0.006 | 0.05 |
| Arachidonic (20:4 *n-6*) | 0.008 | 0.009 | 0.001 | 0.78 |
| Eicosapentaenoic (20:5 *n-3*) | 0.007 | 0.008 | 0.001 | 0.30 |
| Erucic acid (22:1 *n-9*) | 0.015 | 0.017 | 0.001 | 0.30 |
| Docosahexaenoic (22:6 *n-3*) | 0.007 | 0.008 | 0.006 | 0.28 |
| Total SFA | 0.455 | 0.471 | 0.013 | 0.41 |
| Total MUFA | 0.368 | 0.397 | 0.020 | 0.31 |
| Total n-3 | 0.094 | 0.114 | 0.006 | 0.05 |
| Total n-6 | 0.207 | 0.243 | 0.009 | 0.01 |
| PUFA | 0.301 | 0.359 | 0.013 | 0.01 |
| Total fatty acids | 1.135 | 1.274 | 0.041 | 0.02 |

[1]Blood samples were collected once on d 25 and 35 after beginning of treatments. No treatment × day interactions were detected ($P \geq 0.11$).

In summary, inclusion of camelina meal into forage-based diets reduced total DMI by beef steers. However, ruminal in situ forage degradability parameters and plasma cholecystokinin concentrations were not affected. Therefore, additional research is needed to understand the mechanisms by which supplementation with camelina meal, and PUFA in general, reduces DMI in forage-fed cattle.

*Experiment 2*

Steers receiving CAM had greater plasma concentrations of oleic ($P < 0.01$), linoleic ($P = 0.02$), linolenic ($P = 0.05$), n-3 ($P = 0.05$), n-6 ($P = 0.01$), PUFA ($P = 0.01$), and total ($P = 0.02$) fatty acids compared with control steers (Table 4). No treatment effects were detected ($P = 0.30$) on plasma concentrations of erucic acid, which can be present at significant amounts in camelina meal (2 to 5% of total fatty acids; Putnam et al., 1993) and has been shown to induce myocardial lipidosis in non-ruminants (Kramer et al., 1990). Supporting our findings, previous research reported that PUFA supplementation increased total fatty acid and PUFA concentrations in plasma of forage-fed beef cattle (Lake et al., 2007; Scholljegerdes et al., 2007; Cooke et al., 2011). In addition, Moriel et al. (2011) reported that beef heifers supplemented with camelina meal had greater plasma concentrations of oleic, linoleic, linolenic, PUFA, and total fatty acids compared with non-supplemented cohorts. Moriel et al. (2011) also reported that plasma concentration of erucic acid in heifers receiving camelina meal were greater, although detected in trace amounts,

**Table 5.** Physiological responses to corticotropin-releasing hormone (CRH) and thyrotropin-releasing hormone (TRH) challenges in steers offered grain-based supplements containing (CAM; n = 7) or not containing (control; n = 7) camelina meal during Exp. 2

| Item | Treatments | | | |
| | Control | CAM | SEM | P-value |
| --- | --- | --- | --- | --- |
| CRH challenge[1] | | | | |
| Plasma cortisol, ng/mL | 20.4 | 25.8 | 3.40 | 0.28 |
| Rectal temperature,ºC | 39.1 | 39.1 | 0.05 | 0.61 |
| Plasma haptoglobin, absorbance at 450 nm × 100 | 1.73 | 1.54 | 0.07 | 0.10 |
| TRH challenge[2] | | | | |
| Plasma triiodothyronine, ng/mL | 3.23 | 4.11 | 1.17 | 0.61 |
| Plasma thyroxine, ng/mL | 98.1 | 115.8 | 20.6 | 0.55 |
| Plasma thyrotropin-stimulating hormone, ng/mL | 2.13 | 2.14 | 0.11 | 0.95 |

[1]Blood samples were collected hourly from −2 to 0 h and 4 to 8 h, and every 30 min from 0 to 4 h samples relative to CRH challenge (0.1 µg/kg of BW). Rectal temperatures were recorded by every 30 min from −2 to 8 h relative to CRH challenge. No treatment × time interactions were detected ($P \geq 0.16$).

[2]Blood samples were collected hourly from -2 to 0 h and 4 to 8 h, and every 30 min from 0 to 4 h samples relative to TRH challenge (0.33 µg/kg of BW). No treatment × time interactions were detected ($P \geq 0.51$).

compared with non-supplemented cohorts. The camelina meal used by Moriel et al. (2011) contained 2.6% of erucic acid (total fatty acid basis), whereas the camelina meal used herein only contained 0.77% of erucic acid (total fatty acid basis; Table 2), which may explain the similar plasma concentrations of erucic acid among CAM and control steers.

Our group recently demonstrated that steers receiving CRH at 0.1 µg/kg of BW to induce a neuroendocrine stress response experienced an acute-phase reaction including increased rectal temperature and plasma concentrations of haptoglobin and ceruloplasmin (Cooke and Bohnert, 2011). Therefore, this research model was used herein to evaluate the potential nutraceutical effects of camelina meal. No treatment differences were observed for plasma cortisol concentrations ($P = 0.28$; Table 5). In both treatments, plasma cortisol peaked (time effect, $P < 0.01$; data not shown) at 0.5 h after the CRH infusion, indicating that CAM and control steers experienced a similar neuroendocrine stress response (Cooke and Bohnert, 2011). No treatment effects were detected ($P = 0.60$) for rectal temperature (Table 5), although rectal temperatures peaked (time effect, $P < 0.01$; data not shown), similarly to Cooke and Bohnert (2011), for both treatments at 8 h after the CRH infusion. Steers receiving CAM tended ($P = 0.10$; Table 5) to have reduced plasma haptoglobin concentrations compared with control cohorts (1.54 vs. 1.73 absorbance at 450 nm × 100, respectively; SEM = 0.07). No time effect ($P \geq 0.57$) was detected for plasma haptoglobin in CAM and control steers, indicating that
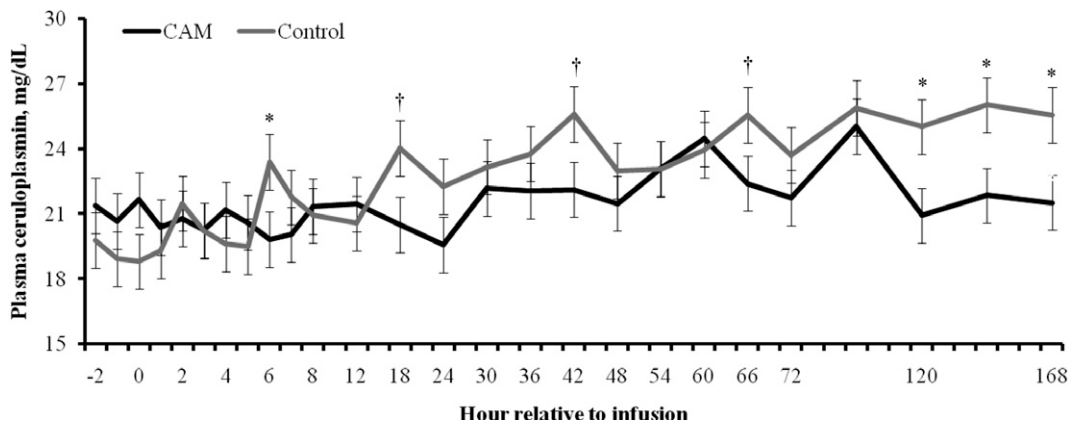
**Figure 1.** Plasma ceruloplasmin concentrations (± SEM) of beef steers offered supplements containing (CAM; n = 5) or not containing (control; n = 4) camelina meal and receiving an intravenous corticotropin-releasing hormone (CRH) infusion (0.1 μg/kg of BW) at h 0 in Exp 2. A treatment × time interaction (P < 0.01) was detected. Treatment comparison within hour: † = P < 0.10, * = P < 0.05.

the CRH infusion did not stimulate an increase in plasma concentrations of this acute-phase protein. Nevertheless, the aforesaid tendency for a treatment effect suggests that baseline plasma haptoglobin concentrations were reduced in CAM compared with control steers. A treatment x time interaction (P < 0.01; Figure 1) was detected for plasma ceruloplasmin. Similar to Cooke and Bohnert (2011), ceruloplasmin concentrations increased after CRH infusion and peaked, as expected, for both treatments (time effects; P < 0.01) several hours after this neuroendocrine stress challenge (Gabay and Kushner, 1999; Arthington et al., 2008). However, CAM steers had reduced ceruloplasmin concentrations compared with control cohorts at 6, 18, 42, 120, 144, and 168 h.

Previous research from our group demonstrated that PUFA supplementation reduced the acute-phase response induced by stressful procedures such as transport and feedlot entry (Araujo et al., 2010; Cooke et al., 2011). These outcomes were attributed to immunomodulatory effects of PUFA, more specifically n-3 and n-6 fatty acids. Linolenic acid and its n-3 derivates promote synthesis of eicosanoids that do not elicit the acute-phase protein response, whereas linoleic acid and its n-6 derivates promote the synthesis of $PGE_2$, a potent stimulator of pro-inflammatory and acute-phase responses (Yaqoob and Calder, 1993; Carroll and Forsberg, 2007; Schmitz and Ecker, 2008). In the present experiment, CAM steers had increased plasma concentrations of linoleic, linolenic, n-3, and n-6 fatty acids compared with control steers. Cattle requirements for linoleic, linolenic, and their respective fatty acid derivates are still unknown. Therefore, it cannot be concluded if CAM steers had reduced baseline haptoglobin concentrations, as well as lessened CRH-induced increase in plasma ceruloplasmin, compared with control steers because of the additional supply of n-3, n-6, or both fatty acid groups.

Thyroid hormones are essential for control of basal metabolic rate and normal development of animals

(Paxton, 1986). Camelina meal contains glucosinolates, which are compounds that can impair function of the thyroid gland in cattle (Lardy and Kerley, 1994). Mawson et al. (1994) reported that glucosinolates reduce circulating concentrations of $T_3$ and $T_4$ and may overstimulate TSH secretion, causing hypertrophy of thyroid tissues. However, in the present study, no treatment effects were detected for serum concentrations of $T_3$, $T_4$, and TSH (P ≥ 0.55; Table 5). Lardy and Kerley (1994) reported that beef steers supplemented with rapeseed meal, which contained increased amounts of glucosinolates, had decreased serum concentrations of $T_4$ compared with non-supplemented cohorts. Moriel et al. (2011) reported that serum concentrations of $T_3$ and $T_4$ were not impaired in heifers supplemented with camelina meal. Concentration of glucosinolates in camelina are relatively low (22 μmol/g; Lange et al., 1995) compared with rapeseed meal (90 to 140 μmol/g; Lardy and Kerley, 1994), which explains the lack of detrimental effects of camelina meal on bovine thyroid function reported herein and by Moriel et al. (2011).

In summary, camelina meal supplementation to beef steers increased plasma concentrations of PUFA but not erucic acid, did not impair thyroid gland function stimulated by a TRH challenge, and reduced plasma concentrations of ceruloplasmin after a CRH challenge.

## Experiment 3

During the PC phase, CAM steers had reduced (P < 0.01; data not shown) forage and total DMI compared with control cohorts (2.23 vs. 2.46 and 3.07 vs. 3.35% of BW, respectively; SEM = 0.04). Hence, ADG during PC tended (P = 0.10; Table 6) to be reduced for CAM steers compared with control cohorts. However, no treatment effects were detected (P = 0.24; Table 6) on G:F during PC. As calculated in Exp. 1, CAM steers consumed camelina meal at 8.5% of diet DM during PC, which is below

**Table 6.** Performance and physiological responses during preconditioning (d −28 to 0) of steers offered grain-based supplements containing (CAM; n = 10) or not containing (control = 10) camelina meal in Exp. 3

| Item | Control | CAM | SEM | *P*-value |
|---|---|---|---|---|
| Performance traits | | | | |
| Shrunk BW (d -31), kg | 220 | 221 | 2 | 0.61 |
| Shrunk BW (d 1), kg | 231 | 229 | 2 | 0.56 |
| ADG,[1] kg/d | 0.37 | 0.26 | 0.04 | 0.10 |
| G:F,[2] g/kg | 49.3 | 38.8 | 6.1 | 0.24 |
| Physiological responses[3] | | | | |
| Rectal temperature, ºC | 39.16 | 39.19 | 0.03 | 0.56 |
| Haptoglobin, absorbance at 450 nm × 100 | 1.80 | 1.65 | 0.05 | 0.04 |
| Ceruloplasmin, mg/dL | 21.9 | 22.3 | 0.7 | 0.66 |
| Cortisol, ng/mL | 39.4 | 41.8 | 5.2 | 0.74 |

[1]Calculated using shrunk BW (after 16 h of feed and water restriction) obtained before beginning (d −31) and after the end (d 1) of the preconditioning phase.

[2]Calculated for dividing the total DM consumed from d −28 to 0 into the total shrunk BW gain (d −31 to 1) achieved over this time period.

[3]All steers were transported for 24 h on d 0. Physiological measurements were obtained on d 0 (before loading into the truck) and d 1 (immediately after unloading) relative to transport. No treatment × day interactions were detected ($P \geq 0.52$).

the limit established by the FDA (Benz, 2009). These findings support previous studies from our research group indicating that PUFA supplementation reduced DMI in feeder cattle, but did not impair feed efficiency parameters (Araujo et al., 2010; Cooke et al., 2011). In the present experiment, hay and treatments were not mixed. As expected, treatment intake during PC was reduced ($P = 0.01$; data not shown) in CAM compared with control steers, but similar to the offered amount (1.90 vs. 2.01 kg, respectively; SEM = 0.03). Therefore, treatment effects detected for forage intake during PC should not be attributed to reduced palatability of the CAM treatment (Lardy and Kerley, 1994). Similarly to Exp. 1, supplemental fat was included at 2.6 and 1.4% of diet DM for CAM and control during PC, respectively, which is less than the concentration suggested by Hess et al. (2008) to prevent impaired forage intake. Therefore, based on the results reported in Exp. 1, reduced forage and total DMI during PC should not be attributed to altered forage digestibility and cholecystokinin concentrations in CAM steers.

During the PC phase, a treatment × day interaction was detected ($P = 0.02$; Figure 2) for plasma linolenic acid (samples collected on d −28, −15, and 0). Similarly, Moriel et al. (2011) observed an increase in plasma linolenic acid concentrations of beef heifers fed camelina meal for 60 d. No additional treatment differences were detected ($P \geq 0.18$; Table 7) for plasma fatty acids during PC, including erucic acid concentrations ($P = 0.21$).
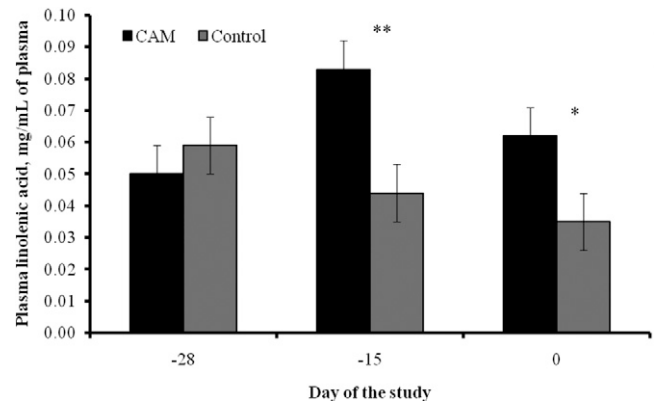


**Figure 2.** Plasma linolenic acid concentrations (± SEM) during preconditioning (d −28 to 0) of steers offered supplements containing (CAM; n = 10) or not containing (control; n = 10) camelina meal in Exp 3. A treatment × day interaction was detected ($P = 0.02$). Treatment comparison within day: * = $P < 0.05$, ** = $P < 0.01$.

Based on results from Exp. 2 and the fatty acid profile of treatments (Tables 1 and 2), it was expected that CAM steers would have greater plasma concentrations of linoleic acid, n-3, n-6, PUFA, and total fatty acids compared with control cohorts. Based on treatment and hay intake of each pen during PC, feed nutritional analysis, and the CPM-Dairy model (Version 3.08.01), average daily duodenal flow of linoleic and linolenic acid per steer was greater ($P < 0.01$; data not shown) for CAM compared with control cohorts (14.2 vs. 10.3 g/d of linoleic acid, SEM = 0.2; and 4.21 vs. 1.23 g/d of linolenic acid, SEM = 0.06; respectively). Plasma fatty acid profile typically reflects the duodenal fatty acid flow (Archibeque et al., 2005; Scholljegerdes et al., 2007; Hess et al., 2008). However, other physiological mechanisms that may alter

**Table 7.** Plasma fatty acid concentrations (mg/mL of plasma) during preconditioning (d −28 to 0) of steers offered grain-based supplements containing (CAM; n = 10) or not containing (control; n = 10) camelina meal in Exp. 3[1]

| Item | Control | CAM | SEM | *P*-value |
|---|---|---|---|---|
| Stearic (18:0) | 0.259 | 0.247 | 0.012 | 0.47 |
| Oleic (18:1) | 0.268 | 0.287 | 0.016 | 0.40 |
| Linoleic (18:2 *n-6*) | 0.199 | 0.215 | 0.008 | 0.18 |
| Arachdonic (20:4 *n-6*) | 0.009 | 0.009 | 0.001 | 0.92 |
| Eicosapentaenoic (20:5 *n-3*) | 0.029 | 0.022 | 0.005 | 0.40 |
| Erucic acid (22:1 n-9) | 0.018 | 0.019 | 0.001 | 0.30 |
| Docosahexaenoic (22:6 *n-3*) | 0.013 | 0.010 | 0.001 | 0.26 |
| Total SFA | 0.628 | 0.596 | 0.029 | 0.44 |
| Total MUFA | 0.460 | 0.487 | 0.020 | 0.36 |
| Total n-3 | 0.088 | 0.097 | 0.008 | 0.45 |
| Total n-6 | 0.225 | 0.232 | 0.007 | 0.54 |
| PUFA | 0.342 | 0.356 | 0.014 | 0.47 |
| Total fatty acids | 1.430 | 1.434 | 0.060 | 0.96 |

[1]Blood samples were collected on d -28, d -15, and d 0 of preconditioning. No treatment × day interactions were detected ($P \geq 0.14$).

plasma fatty acid profile and explain the lack of treatment effects on linoleic acid, n-3, n-6, and PUFA, such as tissue incorporation and enzymatic activity (Archibeque et al., 2005; Scholljegerdes et al., 2007), were not evaluated in the present experiment.

No treatment effects were detected for rectal temperature ($P = 0.56$) and plasma concentrations of cortisol ($P = 0.74$) and ceruloplasmin ($P = 0.66$) immediately before (d 0) and after transportation (d 1; Table 6). However, CAM steers had reduced ($P = 0.04$) plasma haptoglobin concentrations during the same period compared with control cohorts (Table 6). Similar cortisol concentrations between treatments suggest that CAM and control steers experienced a similar neuroendocrine stress response before and after transport (Crookshank et al., 1979; Sapolsky et al., 2000). Circulating haptoglobin typically increases 3 d after transport and feedlot entry in feeder steers (Arthington et al., 2008; Araujo et al., 2010). Therefore, as in Exp. 2, treatment effects detected for plasma haptoglobin suggest that CAM steers had reduced baseline haptoglobin concentrations compared with control steers. This outcome can be attributed, at least partially, to the greater concentrations of plasma linolenic acid in CAM steers during PC, given that this fatty acid favors anti-inflammatory reactions (Yaqoob and Calder, 1993; Schmitz and Ecker, 2008).

No PC treatment × FR treatment interactions ($P \geq 0.20$) were detected for any of the performance and physiological variables evaluated during FR. A PC treatment effect was detected ($P < 0.01$) on forage and total DMI during FR phase because steers that were fed CAM during PC, independent of treatment during FR, had reduced forage and total DMI compared with steers that were fed control (Table 8). The reasons for this continued decrease in forage and total DMI in steers receiving CAM during PC is unknown and deserves further investigation. However, steers receiving CAM during PC tended (PC treatment effect; $P = 0.10$) to have improved G:F compared with control cohorts during FR (Table 8). Supporting this outcome, previous research from our group reported that PUFA supplementation during PC enhanced performance of feeder steers (Cooke et al., 2011). No additional PC treatment effects were detected ($P \geq 0.22$) on FR performance, physiological responses (Table 8), and plasma fatty acid concentrations (data not shown). In addition, no FR treatment effects were detected ($P \geq 0.21$) on FR performance (Table 9), concurring with previous research (Araujo et al., 2010) reporting similar ADG and DMI in newly-received cattle supplemented or not with PUFA. Similarly to Exp. 1 and PC, CAM steers consumed camelina meal at 7.1% of diet DM during FR, which is below the limit established by the FDA (Benz, 2009).

**Table 8.** Performance and physiological responses during feedlot receiving (d 1 to 29) of steers offered preconditioning grain-based supplements (from d −28 to 0) containing (CAM; n = 10) or not containing (control; n = 10) camelina meal in Exp. 3

| | Treatments | | | |
|---|---|---|---|---|
| Item | Control | CAM | SEM | P-value |
| Performance traits | | | | |
| Shrunk BW (d 30), kg | 281 | 279 | 3 | 0.69 |
| Forage DMI, % of BW | 2.61 | 2.45 | 0.03 | < 0.01 |
| Total DMI, % of BW | 3.35 | 3.19 | 0.03 | < 0.01 |
| ADG,[1] kg/d | 1.71 | 1.77 | 0.05 | 0.43 |
| G:F,[2] g/kg | 215 | 231 | 7 | 0.10 |
| Physiological responses[3] | | | | |
| Rectal temperature, ºC | 39.14 | 39.05 | 0.05 | 0.23 |
| Plasma cortisol, ng/mL | 30.9 | 27.6 | 3.3 | 0.48 |
| Plasma haptoglobin, absorbance at 450 nm × 100 | 1.85 | 1.86 | 0.10 | 0.96 |
| Plasma ceruloplasmin, mg/dL | 20.6 | 20.8 | 0.5 | 0.83 |

[1] Calculated using shrunk BW (after 16 h of feed and water restriction) on at feedlot entry (d 1) and after the end of feedlot receiving (d 30).

[2] Calculated for dividing the total DM consumed from d 1 to 29 into the total shrunk BW gain (d 1 to 30) achieved over this time period.

[3] All steers were transported for 24 h on d 0. Rectal temperature was assessed on d 4 and 7 after transport. Blood samples were collected on d 4, 7, 10, 14, 21, and 29 after transport. No treatment × day interactions were detected ($P \geq 0.27$).

A FR treatment effect was detected for plasma oleic acid ($P < 0.01$), linoleic acid ($P < 0.05$), linolenic acid ($P < 0.01$), and PUFA ($P < 0.05$) because CAM steers had greater concentrations of these fatty acids compared with control steers during FR (Table 10). Similar tendencies for FR treatment effects were detected for plasma concentrations of n-3 ($P = 0.06$) and n-6 ($P = 0.07$; Table 10). No treatment effects were detected for plasma concentrations of erucic acid during FR ($P = 0.26$; Table 10), supporting the results detected during the PC phase and Exp. 2. According to treatment and hay intake of each pen during FR, feed nutritional analysis, and the CPM-Dairy model (Version 3.08.01), average daily duodenal flow of linoleic and linolenic acid per steer during FR was greater ($P < 0.01$; data not shown) for CAM steers compared with control cohorts (18.09 vs. 12.44 g/d of linoleic acid, SEM = 0.22; and 5.45 vs. 1.56g/d of linolenic acid, SEM = 0.06; respectively). Hence, FR treatment effects detected on plasma fatty acid profile during FR reflect the greater PUFA content of the CAM treatment, supporting the outcomes from Exp. 2 and results reported by our (Cooke et al., 2011) and other research groups (Lake et al., 2007; Scholljegerdes et al., 2007).

Day effects ($P < 0.01$; Table 11) were detected for plasma haptoglobin and ceruloplasmin in samples collected from d 0 to 29 of the study. Similar to our previous efforts (Araujo et al., 2010; Cooke et al., 2011), hap-

**Table 9.** Performance and physiological responses during feedlot receiving (d 1 to 29) of steers offered receiving grain-based supplements containing (CAM; n = 10) or not containing (control; n = 10) camelina meal in Exp. 3

| | Treatments | | | |
|---|---|---|---|---|
| Item | Control | CAM | SEM | *P*-value |
| Performance traits | | | | |
| Shrunk BW (d 30), kg | 281 | 279 | 3 | 0.59 |
| Forage DMI, % of BW | 2.52 | 2.55 | 0.03 | 0.67 |
| Total DMI, % of BW | 3.29 | 3.26 | 0.32 | 0.52 |
| ADG,[1] kg/d | 1.79 | 1.70 | 0.05 | 0.21 |
| G:F,[2] g/kg | 229 | 217 | 6 | 0.22 |
| Physiological responses[3] | | | | |
| Rectal temperature, ºC | 39.19 | 39.01 | 0.05 | 0.02 |
| Plasma cortisol, ng/mL | 29.2 | 29.3 | 3.31 | 0.98 |
| Plasma haptoglobin, 450 nm × 100 | 2.02 | 1.69 | 0.09 | 0.02 |
| Plasma ceruloplasmin, mg/dL | 21.5 | 19.9 | 0.5 | 0.05 |

[1]Calculated using shrunk BW (after 16 h of feed and water restriction) on at feedlot entry (d 1) and after the end of feedlot receiving (d 30).

[2]Calculated for dividing the total DM consumed from d 1 to 29 into the total shrunk BW gain (d 1 to 30) achieved over this time period.

[3]All steers were transported for 24 h on d 0. Rectal temperature was assessed on d 4 and 7 after transport. Blood samples were collected on d 4, 7, 10, 14, 21, and 29 after transport. No treatment × day interactions were detected ($P \geq 0.16$).

**Table 10.** Plasma fatty acid concentrations (mg/ml of plasma) during feedlot receiving (d 1 to 29) of steers offered receiving grain-based supplements containing (CAM; n = 10) or not containing (control; n = 10) camelina meal in Exp. 3[1]

| | Treatments | | | |
|---|---|---|---|---|
| Item | Control | CAM | SEM | *P*-value |
| Stearic (18:0) | 0.265 | 0.254 | 0.016 | 0.61 |
| Oleic (18:1) | 0.242 | 0.289 | 0.014 | 0.03 |
| Linoleic (18:2 *n-6*) | 0.242 | 0.260 | 0.006 | 0.05 |
| Linolenic (18:3 *n-3*) | 0.086 | 0.118 | 0.006 | 0.01 |
| Arachdonic (20:4 *n-6*) | 0.008 | 0.009 | 0.001 | 0.52 |
| Eicosapentaenoic (20:5 *n-3*) | 0.016 | 0.007 | 0.004 | 0.08 |
| Erucic acid (22:1 *n-9*) | 0.017 | 0.018 | 0.001 | 0.26 |
| Docosahexaenoic (22:6 *n-3*) | 0.008 | 0.008 | 0.0005 | 0.67 |
| Total SFA | 0.606 | 0.564 | 0.037 | 0.44 |
| Total MUFA | 0.441 | 0.460 | 0.020 | 0.52 |
| Total n-3 | 0.110 | 0.134 | 0.008 | 0.06 |
| Total n-6 | 0.256 | 0.275 | 0.007 | 0.07 |
| PUFA | 0.388 | 0.429 | 0.014 | 0.05 |
| Total fatty acids | 1.435 | 1.455 | 0.066 | 0.83 |

[1]All steers were transported for 24 h on d 0. Blood samples were collected on d 4, 14, and 29 after transport. No treatment × day interactions were detected ($P \geq 0.17$).

toglobin concentrations peaked on d 4 whereas ceruloplasmin peaked on d 1 after transport (Table 11), indicating that transport and feedlot entry stimulated an acute-phase protein reaction in steers from both treatments. No FR treatment effects were detected ($P = 0.98$) for plasma cortisol concentrations (Table 9) during FR, suggesting that steers receiving CAM or control experienced a similar neuroendocrine stress response after transport and feedlot entry (Crookshank et al., 1979; Sapolsky et al., 2000). However, FR treatment effects were detected for rectal temperature ($P = 0.02$), plasma haptoglobin ($P = 0.02$) and ceruloplasmin ($P = 0.05$) during the FR phase. Steers receiving CAM had reduced rectal temperature and plasma concentrations of haptoglobin and ceruloplasmin (Table 9), concurring with previous research from our group reporting that PUFA supplementation lessened the acute-phase response induced by transport and feedlot entry (Cooke et al., 2011). Araujo et al. (2010) also reported that PUFA supplementation

during PC and FR prevented the haptoglobin response to transport and feedlot entry, but did not improve FR performance variables as in the present experiment. Still, circulating concentrations of acute-phase proteins during feedlot receiving have been negatively associated with performance (Qiu et al., 2007; Cooke et al., 2009) and positively associated with incidence of respiratory diseases (Berry et al., 2004) in overtly healthy cattle. Therefore, development of management strategies that prevent or alleviate the acute-phase response is essential for optimal performance, health, and efficiency variables in beef operations (Duff and Galyean, 2007). Similar to Exp. 2, FR treatment effects detected on rectal temperature and plasma haptoglobin and ceruloplasmin can be attributed to immunomodulatory effects of PUFA. However, CAM steers had greater plasma concentrations of linoleic acid, linolenic acid, and their respective derivates during FR (Table 10), which are known to favor or prevent, respectively, inflammatory and acute-phase reactions (Yaqoob and Calder, 1993; Schmitz and Ecker, 2008). Therefore, it cannot be concluded if steers

**Table 11.** Day effects on plasma concentrations of acute-phase proteins of transported steers (n = 60) in Exp. 3.[1]

| | Day of the study | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| Item | 0 | 1 | 4 | 7 | 10 | 14 | 21 | 29 | SEM | *P*-value |
| Haptoglobin, 450 nm × 100 | 1.79 [ac] | 1.65 [ac] | 2.22 [b] | 2.18 [b] | 1.92 [a] | 1.51 [c] | 1.88 [a] | 1.42 [c] | 0.13 | < 0.01 |
| Ceruloplasmin, mg/dL | 21.3 [ac] | 22.8 [b] | 22.2 [ab] | 22.7 [b] | 22.7 [b] | 20.3 [c] | 18.7 [d] | 17.7 [e] | 0.56 | < 0.01 |

[1]Steers loaded into a commercial livestock trailer on d 0, transported for 24 h, and unloaded on d 1. Blood samples were collected immediately before (d 0) and 1, 4, 7, 10, 14, 21, and 29 d relative to beginning of transport.

[a-e]Within rows, values bearing different superscripts differ ($P < 0.05$).

receiving CAM during FR had reduced rectal temperature and plasma concentrations of haptoglobin and ceruloplasmin compared with control cohorts because of the additional supply of n-3, n-6, or both fatty acid groups.

In summary, camelina meal supplementation during PC increased plasma linolenic acid and reduced baseline plasma haptoglobin concentrations, feed intake, and ADG during PC, but benefited feed efficiency during FR. Camelina meal supplementation during FR increased plasma concentrations of linolenic acid, linolenic acid, and their respective derivates, decreased the acute-phase protein reaction induced by transport and feedlot entry, but did not improve FR performance.

### *Overall Conclusions*

Camelina meal supplementation to beef cattle, within the limits established by the FDA, did not impair thyroid gland function nor increase plasma concentrations of erucic acid, which are 2 of the main health concerns associated with this feed ingredient. However, steers receiving camelina meal had decreased forage and total DMI, but these outcomes were not associated with altered ruminal forage disappearance rate or circulating cholecystokinin concentrations. Camelina meal supplementation lessened the acute-phase protein reaction associated with neuroendocrine stress responses induced by CRH challenge or transport and feedlot entry, and benefited feed efficiency parameters if offered during preconditioning. Therefore, camelina meal is a feasible feed ingredient to alleviate stress-induced inflammatory reactions and promote cattle welfare in beef operations.

### LITERATURE CITED

Ahlin, K. A., M. Emanuelson, and H. Wiktorsson. 1994. Rapeseed products from double-low cultivars as feed for dairy cows: Effects of long-term feeding on thyroid function, fertility and animal health. Acta. Vet. Scand. 35:37–53.

Allen, M. S. 2000. Effects of diet on short-term regulation of feed intake by lactating dairy cattle. J. Dairy Sci. 83:1598–1624.

AOAC. 2006. Official Methods of Analysis.18th ed. Association of Official Analytical Chemists, Arlington, VA.

Araujo, D. B., R. F. Cooke, G. R. Hansen, C. R. Staples, and J. D. Arthington. 2010. Effects of rumen-protected polyunsaturated fatty acid supplementation on performance and physiological responses of growing cattle following transportation and feedlot entry. J. Anim. Sci. 88:4120–4132.

Archibeque, S. L., D. K. Lunt, C. D. Gilbert, R. K. Tume, and S. B. Smith. 2005. Fatty acid indices of stearoyl-CoA desaturase do not reflect actual stearoyl-CoA desaturase enzyme activities in adipose tissues of beef steers finished with corn-, flaxseed-, or sorghum-based diets. J. Anim. Sci. 83:1153–1166.

Arthington, J. D., X. Qiu, R. F. Cooke, J. M. B. Vendramini, D. B. Araujo, C. C. Chase, Jr., and S. W. Coleman. 2008. Effects of preshipping management on measures of stress and performance of beef steers during feedlot receiving. J. Anim. Sci. 86:2016–2023.

Arthington, J. D., J. W. Spears, and D. C. Miller. 2005. The effect of early weaning on feedlot performance and measures of stress in beef calves. J. Anim. Sci. 83:933–939.

Benson, J. A., and C. K. Reynolds. 2001. Effects of abomasal infusion of long-chain fatty acids on splanchnic metabolism of pancreatic and gut hormones in lactating dairy cows. J. Dairy Sci. 84:1488–1500.

Benz, S. A. 2009. Food and Drug Administraton − Department of Health and Human Services. Accessed August 5, 2011. http://agr.mt.gov/camelina/FDAletter11-09.pdf.

Berry, B. A., A. W. Confer, C. R. Krehbiel, D. R. Gill, R. A. Smith, and M. Montelongo. 2004. Effects of dietary energy and starch concentrations for newly received feedlot calves: II. Acute-phase protein response. J. Anim. Sci. 82:845–850.

Bradford, B. J., K. J. Harvatine, and M. S. Allen. 2008. Dietary unsaturated fatty acids increase plasma glucagon-like peptide-1 and cholecystokinin and may decrease premeal ghrelin in lactating dairy cows. J. Dairy Sci. 91:1443–1450.

Carroll, J. A., and N. E. Forsberg. 2007. Influence of stress and nutrition on cattle immunity. Vet. Clin. Food. Anim. 23:105–149.

Choi, B., and D. L. Palmquist. 1996. High fat diets increase plasma cholecystokinin and pancreatic polypeptide, and decrease plasma insulin and feed intake in lactating cows. J. Nutr. 11:2913–2919.

Cooke, R. F., J. D. Arthington, B. R. Austin, and J. V. Yelich. 2009. Effects of acclimation to handling on performance, reproductive, and physiological responses of Brahman-crossbred heifers. J. Anim. Sci. 87:3403–3412.

Cooke, R. F., and D. W. Bohnert. 2011. Technical note: Bovine acute-phase response after corticotropin-release hormone challenge. J. Anim. Sci. 89:252–257.

Cooke, R. F., D. W. Bohnert, P. Moriel, B. W. Hess, and R. R. Mills. 2011. Effects of polyunsaturated fatty acid supplementation on ruminal in situ forage degradability, performance, and physiological responses of feeder cattle. J. Anim. Sci. 89:3677–3689.

Crookshank, H. R., M. H. Elissalde, R. G. White, D. C. Clanton, and H. E. Smalley. 1979. Effect of transportation and handling of calves upon blood serum composition. J. Anim. Sci. 48:430–435.

Curley, K. O., Jr., D. A. Neuendorff, A. W. Lewis, J. J. Cleere, T. H. Welsh, Jr., and R. D. Randel. 2008. Functional characteristics of the bovine hypothalamic-pituitary-adrenal axis vary with temperament. Horm. Behav. 53:20–27.

Demetriou, J. A., P. A. Drewes, and J. B. Gin, 1974. Ceruloplasmin. Pages 857–846 in Clinical Chemistry. D. C. Cannon, and J. W. Winkelman, ed. Harper and Row, Hagerstown, MD.

Drackley, J. K., T. H. Klusmeyer, A. M. Trusk, and J. H. Clark. 1992. Infusion of long-chain fatty acids varying in saturation and chain length into the abomasum of lactating dairy cows. J. Dairy Sci. 75:1517–1526.

Duff, G. C., and M. L. Galyean. 2007. Board-invited Review: Recent advances in management of highly stressed, newly received feedlot cattle. J. Anim. Sci. 85:823–840.

Folch, J., M. Less, and G. H. Sloane-Stanley. 1957. A simple method for the isolation and purification of total lipids from animal tissues. J. Biol. Chem. 226:497–509.

Gabay, C., and I. Kushner. 1999. Acute-phase proteins and other systemic responses to inflammation. N. Engl. J. Med. 340:448–454.

Hayat, Z., G. Cherian, T. Pasha, F. Khattak, and M. Jabbar. 2009. Effect of feeding flax and two types of antioxidants on egg production, egg quality, and lipid composition of eggs. J. Appl. Poult. Res. 18:541–551.

Hess, B. W., G. E. Moss, D. C. Rule. 2008. A decade of developments in the area of fat supplementation research with beef cattle and sheep. J. Anim. Sci. 86(Suppl.):E188–E204.

Hurtaud, C, and J. L. Peyraud. 2007. Effects of feeding camelina (seeds or meal) on milk fatty acid composition and butter spreadability. J. Dairy Sci. 90:5134–5145.

Kramer, J. K. G, E. R. Farnworth, K. M. Johnston, M. S. Wolynetz, H. W. Modler, and F. D. Sauer. 1990. Myocardial changes in newborn piglets fed sow milk or milk replacer diets containing different levels of erucic acid. Lipids 25:729–737.

Lake, S. L., T. R. Weston, E. J. Scholljegerdes, C. M. Murrieta, B. M. Alexander, D. C. Rule, G. E. Moss, and B. W. Hess. 2007. Effects of postpartum dietary fat and body condition score at parturition on plasma, adipose tissue, and milk fatty acid composition of lactating beef cows. J. Anim. Sci. 85:717–730.

Lange, R., W. Schumann, M. Petrzika, H. Busch, and R. Marquard. 1995. Glucosinolate in Leindottersamen. FettWiss Technol. 97:146–152.

Lardy, G. P., and M. S. Kerley. 1994. Effect of increasing the dietary level of rapeseed meal on intake by growing beef steers. J. Anim. Sci. 72:1936–1942.

Leupp, J. L., G. P. Lardy, S. A. Soto-Navarro, M. L. Bauer, and J. S. Caton. 2006. Effects of canola seed supplementation on intake, digestion, duodenal protein supply, and microbial efficiency in steers fed forage-based diets.J. Anim. Sci. 84:499–507.

Litherland, N. B., S. Thire, A. D. Beaulieu, C. K. Reynolds, J. A. Benson, and J. K. Drackley. 2005. Dry matter intake is decreased more by abomasal infusion of unsaturated free fatty acids than unsaturated triglycerides. J. Dairy Sci. 88:632–643.

Makimura, S. and N. Suzuki. 1982. Quantitative determination of bovine serum haptoglobin and its elevation in some inflammatory diseases. Jpn. J. Vet. Sci. 44:15–21.

Mawson, R., R. K. Heaney, Z. Zdunczyk, and H. Kozlowska. 1993. Rapeseed meal-glucosinolates and their nutritional effects. Part 2. Flavour and palatability. Nahrung. 37:336–344.

Mawson, R., R. K. Heaney, Z. Zdunczyk, and H. Kozlowska. 1994. Rapeseed meal-glucosinolates and their antinutritional effects. Part 4. Goitrogenicity and internal organs abnormalities in animals. Nahrung. 38:178–191.

Merrill, M. L., D. W. Bohnert, D. L. Harmon, A. M. Craig, and F. N. Schrick. 2007. The ability of a yeast-derived cell wall preparation to minimize the toxic effects of high-ergot alkaloid tall fescue straw in beef cattle. J. Anim. Sci. 85:2596–2605.

Moate, P. J., W. Chalupa, T. C. Jenkins, and R. C. Boston. 2004. A model to describe ruminal metabolism and intestinal absorption of long chain fatty acids. Anim. Feed. Tech. Sci. 112:79–105.

Moriel, P., V. Nayigihugu, B. I. Cappellozza, E. P. Gonçalves, J. M. Krall, T. Foulke, K. M. Cammack, and B. W. Hess. 2011. Camelina meal and crude glycerin as feed supplements for developing replacement beef heifers. J. Anim. Sci. 89:4314–4324.

NRC. 1996. Nutrient Requirements of Beef Cattle. 7th rev. ed. National Academy Press, Washington, D.C.

Ørskov, E. R., and I. McDonald. 1979. The estimation of protein degradability in the rumen from incubation measurements weighted according to rate of passage. J. Agric. Sci. 92:499–503.

Paxton, M. J. W. 1986. Endocrinology: Biological and medical perspectives. M. J. W. Paxton, ed. Wm. C. Brown Publishers, Dubeque, IA.

Poore, M. H., J. A. Moore, and R. S. Swingle. 1990. Differential passage rates and digestion of neutral detergent fiber from grain and forages in 30, 60 and 90% concentrate diets fed to steers. J. Anim. Sci. 68:2965–2973.

Putnam, D. H., J. T. Budin, L. A. Filled, and W. M. Breene. 1993. A promising low input oilseed. Pages 314–322 in New Crops. J. Fanick and J. E. Simon, eds. Wiley, New York, NY.

Qiu, X., J. D. Arthington, D. G. Riley, C. C. Chase, Jr., W. A. Phillips, S. W. Coleman, and T. A. Olson. 2007. Genetic effects on acute phase protein response to the stresses of weaning and transportation in beef calves. J. Anim. Sci. 85:2367–2374.

Relling, A. E., and C. K. Reynolds. 2007. Feeding rumen-inert fats differing in their degree of saturation decreases intake and increases plasma concentrations of gut peptides in lactating dairy cows. J. Dairy Sci. 90:1506–1515.

Reuter, R. R., J. A. Carroll, L. E. Hulbert, J. W. Dailey, and M. L. Galyean. 2010. Technical note: Development of a self-contained, indwelling rectal temperature probe for cattle research. J. Anim. Sci. 88:3291–3295.

Robertson, J. B., and P. J. Van Soest. 1981. The detergent system of analyses and its application to human foods. Pages 123–158 in The Analysis of Dietary Fiber. W. P. T. James and O. Theander, eds. Marcell Dekker, New York, NY.

Sapolsky, R.M., L. M. Romero, A. U. Munck. 2000. How do glucocorticoids influence stress responses? Integrating permissive, suppressive, stimulatory, and preparative actions. Endocr. Rev. 21:55–89.

Schauff, D. J., and H. Clark. 1989. Effects of prilled fatty acids and calcium salts of fatty acids on rumen fermentation, nutrient digestibilities, milk production, and milk composition. J. Dairy Sci. 72:917–927.

Schmitz, G. and J. Ecker. 2008. The opposing effects of n-3 and n-6 fatty acids. Prog. Lipid Res. 47:147–155.

Scholljegerdes, E. J., S. L. Lake, T. R. Weston, D. C. Rule, G. E. Moss, T. M. Nett, and B. W. Hess. 2007. Fatty acid composition of plasma, medial basal hypothalamus, and uterine tissue in primiparous beef cows fed high-linoleate safflower seeds. J. Anim. Sci. 85:1555–1564.

Van Soest, P. J., J. B. Robertson, and B. A. Lewis. 1991. Methods for dietary fiber, neutral detergent fiber, and nonstarch polysaccharides in relation to nutrition animal. J. Dairy Sci. 74:3583–3597.

Vendramini, J. M. B., L. E. Sollenberger, A. Adesogan, J. C. B. Dubeux, Jr., S. M. Interrante, R. L. Stewart, Jr., and J. D. Arthington. 2008. Protein fractions of Tifton 85 and rye-ryegrass due to sward management practices. Agron. J. 100:463–469.

Weiss, W. P., H. R. Conrad, and N. R. St. Pierre. 1992. A theoretically-based model for predicting total digestible nutrient values of forages and concentrates. Anim. Feed Sci. Technol. 39:95–110.

Wichtel, J. J., A. L. Craigie, D. A. Freeman, H. Varela-Alvarez, and N. B. Williamson. 1996. Effect of selenium and iodine supplementation on growth rate, thyroid and somatotropic function in dairy calves at pasture. J. Dairy Sci. 79:1865–1872.

Yaqoob, P. and P. C. Calder. 1993. The effects of fatty acids on lymphocyte functions. Int. J. Biochem. 25:1705–1714.

**References**

This article cites 48 articles, 22 of which you can access for free at:
http://www.journalofanimalscience.org/content/90/11/4042#BIBL