The effects of feeding medium-chain triglycerides on the growth, insulin responsiveness, and body composition of Holstein calves from birth to 85 kg of body weight

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ABSTRACT

The objective of this study was to determine the effects of feeding calves isocaloric, isonitrogenous diets that varied in the amount and type of fatty acids on growth, response to an insulin challenge, and body composition. Thirty-six calves were assigned to a randomized block design with 3 dietary treatments, 10 calves per treatment, and a baseline group of 6 calves. Three different milk-replacer-based diets were designed to deliver less than 2% of the lipid as medium-chain triglycerides (control; diet contained no added medium-chain triglycerides), 32% medium-chain triglycerides primarily as caprylate (CAP oil), and 32% of fatty acids primarily as laurate from coconut oil (CCO). Calves were offered 0.28 Mcal of intake energy/kg of body weight (BW)^{0.75} from d 1 to 7 and 0.32 Mcal of intake energy/kg of BW^{0.75} adjusted weekly for BW from d 8 to harvest. Dry matter, intake energy, crude protein, and fat intakes were 53.7 kg, 281.8 Mcal, 14.6 kg, and 13.0 kg; 56.6 kg, 297.2 Mcal, 15.8 kg, and 14.2 kg; and 53.8 kg, 280.4 Mcal, 15.4 kg, and 13.3 kg for the control, CAP oil, and CCO treatments, respectively. Dry matter, energy, protein, and fat intakes did not differ among treatments. At approximately 65 kg of BW, 5 calves per treatment were given an insulin challenge. After the challenge the decrease in plasma glucose concentration was greater for the calves fed the CAP oil diet compared with those fed the control and CCO diets. Calves were harvested at approximately 88 kg of BW. Empty body gains were 0.92, 0.79, and 0.87 kg/d for control-, CAP oil-, and CCO-fed calves, respectively, and the gains of the CAP oil-fed calves were less than those of the control-fed calves. Empty body crude protein, ash, and water were not different among treatments. Empty body retained energy and fat tended to be 5.6 and 8.7% greater for calves consuming the CCO diet than for those fed the control diet. The livers of calves consum-

ing the CCO diet were 330 g heavier and contained 15% more fat than the livers of the control and CAP oil calves. The results of this study demonstrate that the energy demand of the calf to maintain body temperature resulted in increased oxidation of intake energy; thus, overall body composition differences could not be detected. However, the intake of CCO increased the accumulation of lipid in the liver and carcass despite the apparent cold stress conditions.

Key words: medium-chain triglyceride, lipid, body composition, calf

INTRODUCTION

Altering the relationships among the sources of calories in the diet can have a significant effect on whole body composition in calves (Roy et al., 1970; Donnelly and Hutton, 1976). Donnelly (1983) demonstrated that altering the fat-to-carbohydrate ratio and protein content of the diet had significant effects on the composition of gain. Calves fed high-fat and low-protein diets accreted a greater amount of empty body (EB) fat, whereas calves fed high-carbohydrate, high-protein diets deposited significantly more EB protein and less fat (Donnelly, 1983). Furthermore, calves consuming isocaloric and isonitrogenous diets that varied in carbohydrate:fat contained less EB fat when consuming the higher carbohydrate, lower fat diet. Empty body protein was unaffected by the treatments, indicating that above a certain energy intake level, additional fat intake was simply deposited as fat (Tikofsky et al., 2001).

Most of the fats contained in milk replacers are from tallow or lard and are composed primarily of long-chain saturated fats (Jenkins et al., 1985). However, cow's milk is composed of a wide range of fatty acids that include both medium- (8 to 12%) and short-chain (4%) fatty acids as a percentage of the total fat content (Jensen, 2002).

The hepatic oxidation of medium-chain triglycerides (MCT) is greater compared with long-chain triglycerides (LCT) because carnitine palmitoyltransferase I, the rate-limiting enzyme of oxidation, is not required

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for medium-chain fatty acid (MCFA) transport into the mitochondria, the primary site of oxidation (Graulet et al., 2000). Moreover, the rate of C12:0 β -oxidation in peroxisomes is greater than palmitate in preruminant calves (Hocquette and Bauchart, 1999) and is insulin independent, thus offering the potential to alter glucose disposal. Odle et al. (1991) found that piglets fed C7:0, C8:0, and C9:0 fatty acids had significantly greater β -hydroxybutyrate in plasma compared with pigs fed C10:0 1 h postfeeding, indicating greater β -oxidation of the MCFA.

The objective of this study was to determine the effects of feeding various MCT to preruminant dairy calves under isocaloric and isonitrogenous conditions in which protein was not considered to be a limiting nutrient (Van Amburgh and Drackley, 2005) on body composition and insulin responsiveness. Our hypothesis was that body composition and insulin responsiveness would be altered by the feeding of MCT.

MATERIALS AND METHODS

Thirty-six Holstein bull calves were assigned to a randomized block design with 3 dietary treatments, 10 calves per treatment, and one baseline group of 6 calves. All protocols involving animals were reviewed and approved by the Cornell University Institutional Animal Care and Use Committee. This study was conducted from January to April, and the mean ambient temperature during the study was $-4 \pm 8^\circ\text{C}$; therefore, calves experienced cold stress conditions during this experiment. The 3 treatments were defined as control (diet contained no MCT), CAP oil (diet contained 32% of fatty acids as MCT with a greater proportion of C8:0), and coconut oil (diet contained 32% of fatty acids as MCT with a greater proportion of C12:0). Calves were obtained from the Cornell Teaching and Research Center and 4 local dairy farms. All calves received 4 L of colostrum within 1 h of birth. Calves were transported to and housed in a greenhouse facility at the Cornell Teaching and Research Center, Harford, NY. Serum protein concentration was measured on calves after arrival at the facility to evaluate whether successful passive transfer of colostrum antibodies occurred but was measured after assignment and not used to block the calves. Upon entry to the barn, each animal received injections of 2 mL of vitamin B complex (The Butler Co., Dublin, OH), 2.5 mL of BoSe (1 mg of selenium, 50 mg of vitamin E per mL; Schering-Plough Animal Health Corporation, Union, NJ), and 2 mL of iron dextran (100 mg of elemental iron/mL, The Butler Co.). In addition, a physical exam was administered to assess general health status. Animals were weighed

after entry into the barn, before initiation of treatment, and once a week thereafter.

Calves were randomly assigned to blocks, 2 calves per treatment, and the seventh calf was assigned to the baseline harvest group. Seven calves were assigned to a block if they were less than 3 d of age and weighed between 47 and 53 kg. Baseline calves were harvested within 24 h of initiation of treatments to blockmates. Calves were individually housed, with blockmates placed next to each other to ensure that treatments were randomly assigned throughout different locations in the barn. Kiln-dried sawdust was used as bedding material. Treatment calves were raised to 88.1 ± 1.0 kg of live BW.

After arrival and before treatment assignment, calves were fed a 26% protein, 21% fat milk-replacer (Excelerate, MSC, Dundee, IL) diet containing an animal fat supplement [Advance Milk Energizer (7% CP, 60% fat), MSC] for a 5-d adjustment period. All diets were reconstituted to 15% solids, mixed with warm water to achieve a temperature of 43°C , and fed to calves in individual buckets. Calves were fed twice daily, at 0600 and 1800 h. Health assessment was completed during the morning feeding period, and fecal and respiration scores were taken. Fecal consistency was scored daily on a scale of 1 to 3: 1) solid, semisolid, normal; 2) runny, loose; 3) scour, watery. In addition, a respiration score was assigned daily on a scale of 1 to 4: 1) normal, 2) runny nose, 3) heavy breathing, 4) chronic coughing.

The ingredient and nutrient compositions of dietary treatments are shown in Table 1. Milk-replacer diets were designed to be isocaloric and isonitrogenous. A commercially available milk replacer (Excelerate, MSC) was used as the base formulation, and 3 sources of fat were added to achieve the fatty acid composition shown in Table 2. The control diet was supplemented with an animal fat source (Advance Milk Energizer, MSC). The CAP oil diet was supplemented with Aldo MCT Oil (Lonza Aldo MCT KFG-TAG Oil, Lonza Inc., Williamsport, PA) that was emulsified before feeding and blended with water to ensure equal mixing throughout the diet. The coconut oil diet was supplemented as a dried vegetable fat blend (Milk Specialties 7/60 Hi-Fat, MSC). Whey protein concentrate (WPC, Leprino Foods, Denver, CO) was added to all diets to ensure the diets remained isonitrogenous. There was no separation of diet ingredients, and all ingredients were readily water soluble.

The DMI for calves on all treatments was formulated so calves would consume 0.28 Mcal/kg of $\text{BW}^{0.75}$ for treatment d 1 to 3, which then increased to 0.32 Mcal/kg of $\text{BW}^{0.75}$ for the duration of the experiment. Intakes were adjusted on a weekly basis after the animals were

Table 1. Milk-replacer diet specifications and chemical composition on a DM basis¹

Item	Control	CAP oil	Coconut oil
Composition of diet, %			
Milk replacer ²	73.75	81.35	74.95
Animal fat supplement ³	19.00	5.50	0.00
Vegetable fat supplement ⁴	0.00	0.00	17.00
Whey protein concentrate	7.25	5.50	7.00
Aldo MCT Oil ⁵	0.00	7.65	1.05
Nutrient profile, DM basis			
DM, %	95.90	96.06	94.85
IE, ⁶ Mcal/kg	5.25	5.18	5.20
CP, %	28.49	28.31	28.55
Fat, %	23.16	23.54	23.40
Lactose, ⁷ %	41.83	41.56	41.53
Ash, %	6.52	6.59	6.52
Ca, %	0.95	0.99	0.96
P, %	0.61	0.62	0.61
Mg, %	0.12	0.12	0.12
K, %	1.36	1.36	1.35
Na, %	0.76	0.78	0.76
Vitamin A, ⁸ IU/kg	12,196.41	13,453.26	12,394.86
Vitamin D, ⁸ IU/kg	4,065.47	4,484.42	4,131.62
Vitamin E, ⁸ IU/kg	81.31	89.69	82.63

¹Treatments: control [diet contained no medium-chain triglycerides (MCT)], CAP oil (diet contained 32% of fatty acids as MCT with a greater proportion of C8:0), and coconut oil (diet contained 32% of fatty acids as MCT with a greater proportion of C12:0).

²Milk replacer (MSC, Dundee, IL): 28.5% protein, 15% fat, 49% lactose, 7.5% ash.

³Milk Energizer (MSC): 7% protein, 60% fat, 29.31% lactose, 3.69% ash.

⁴Milk Specialties 7/60 Hi-Fat (MSC): 7.42% protein, 62% fat, 26.89% lactose, 3.69% ash.

⁵Aldo MCT KFG-TAG Oil (Lonza Inc., Williamsport, PA): 98% fat, 2% ash.

⁶Intake energy as determined by bomb calorimetry.

⁷Lactose determined by difference.

⁸As reported by manufacturer.

weighed. Feed intake was measured at each feeding by subtracting the refusal from the amount of diet offered. Free-choice water was available at all times, and no dry feed was offered.

As calves approached 65 kg of BW, 5 calves from each treatment were randomly selected to undergo an

insulin challenge. On the day of the challenge, calves were fitted with an indwelling jugular catheter (C542, Abbott Laboratories, North Chicago, IL). Insulin (lot 123K1148, Sigma Laboratories, St. Louis, MO) was initially dissolved to a concentration of 65 µg/mL using 0.01 M HCl and then diluted to 32.5 µg/mL using

Table 2. Measured fatty acid composition of treatment diets and tabular values¹ for cow's milk for comparison²

Fatty acid, % of total fat	Control	CAP oil	Coconut oil	Whole milk ¹
C4:0 (butyric)	0.40	0.65	0.41	3.30
C6:0 (caproic)	0.20	1.49	0.92	3.00
C8:0 (caprylic)	0.36	23.30	9.18	2.00
C10:0 (capric)	0.40	9.36	5.06	3.00
C12:0 (lauric)	0.99	0.80	23.63	3.50
C14:0 (myristic)	2.12	2.01	9.91	10.20
C16:0 (palmitic)	23.31	16.23	14.51	26.30
C16:1 (palmitoleic)	2.29	1.43	0.90	2.30
C18:0 (stearic)	12.75	8.79	6.73	12.00
C18:1 (oleic)	38.35	24.76	19.15	25.10
C18:2 (linoleic)	14.07	9.41	7.30	2.40
C18:3 (linolenic)	0.63	0.53	0.36	0.80
Other	4.05	1.24	1.94	6.10

¹Jensen (2002).

²Treatments: control [diet contained no medium-chain triglycerides (MCT)], CAP oil (diet contained 32% of fatty acids as MCT with a greater proportion of C8:0), and coconut oil (diet contained 32% of fatty acids as MCT with a greater proportion of C12:0).

sterile saline. A bolus dose of 1 $\mu\text{g/kg}$ of BW insulin was administered through the catheter at the challenge, immediately followed by 3 mL of sterile saline to ensure the insulin was flushed out of the catheters. Blood samples (5 mL) were drawn from the catheters at -30, -15, -5, 0, 2.5, 5, 10, 15, 30, 45, 60, and 120 min relative to the insulin infusion using a syringe and then transferred into a Vacutainer (BD, Franklin Lakes, NJ). Catheters were flushed with sterile saline and heparin between samplings. Plasma was harvested, and samples were analyzed for concentrations of glucose, NEFA, and insulin.

Metabolite assays were conducted in 96-well microplates (NUNC, Rochester, NY) and read using a microplate reader spectrophotometer (Molecular Devices, Sunnyvale, CA). Plasma glucose was analyzed by enzymatic (glucose oxidase) determination using kit 510 (Sigma Co., St. Louis, MO). Nonesterified fatty acids were determined using the Wako NEFA-C Kit, (Wako Chemicals USA Inc., Dallas, TX) as modified by Sechen et al. (1990).

Insulin was measured by a double-antibody RIA as described by McGuire et al. (1995). Pancreatic bovine insulin (lot 615-70N-80, Lilly Research Laboratories, Greenfield, IN) was used for iodination. Commercial primary antibody (guinea pig anti-porcine insulin, Sigma #I8510; Sigma Co.), secondary antibody (goat anti-guinea pig IgG serum, Linco #2020, Linco Research, St. Charles, MO), and carrier (normal guinea pig IgG carrier, Linco #3020, Linco Research) were used. Intraassay and interassay coefficients of variation for insulin from the insulin challenge were 7.0 to 9.22% and 16.50%, respectively.

On the morning of harvest, calves were fed and then transported to the abattoir in Morrison Hall, weighed, and harvested with captive bolt and exsanguination. Body tissues were divided into 4 components: carcass; blood and organs; head, hide, feet, tail; and liver. The carcass was weighed and split in half, and the right half was reweighed and used for composition analysis. All organs were removed and weighed. The gastrointestinal tract was stripped clean of digesta, washed, and reweighed. The blood from exsanguination was weighed and added to the organs. The liver was weighed and retained as a single fraction. All fractions were bagged and frozen (-20°C) within 1 h of harvest pending further analysis.

The frozen carcass; blood and organs; and head, hide, feet, and tail tissue components were ground once through a grinding plate with 20-mm openings and 4 times through a grind plate with 10-mm openings (Model 8016, Autio Co., Astoria, OR). The liver was ground using a food processor (Model 8181D, Hobart Inc., Troy, OH). Grab samples of each com-

ponent were collected, bulked, and subsampled. Each subsample was frozen at -20°C and weighed. Samples were freeze-dried and reweighed for component DM determination. Freeze-dried samples were reground with dry ice through a Wiley mill (2-mm screen, Arthur H. Thomas, Philadelphia, PA). Ground samples and feed ingredients were further analyzed for DM by weighing samples into beakers and placing them into a 105°C oven for 16 h. Dried samples were then placed into a 550°C oven for 16 h for determination of ash. Crude protein was determined using Kjeldahl digestion (AOAC, 1990) modified to include the use of boric acid and steam distillation (Pierce and Haenisch, 1940). Crude fat was measured by ether extract (AOAC, 1981). Tissue energy (**TE**) was determined in a bomb calorimeter (Parr model 1281, Parr Inc., Moline, IL). Fatty acid analysis was performed on the dietary ingredients using a hexane:isopropanol extraction (Hara and Radin, 1978), and fatty acids were transmethyated using base-catalyzed methylation (Christie, 1982) with modifications by Chouinard et al. (1999). Fatty acid methyl esters were quantified on a gas chromatograph (HP6890, Hewlett Packard, Wilmington, DE). Samples of all feed ingredients were taken weekly, bulked, and subsampled for analyses. Proximate analyses were conducted as described for tissue samples analysis. Mineral concentrations for all ingredients were analyzed by inductively coupled plasma-mass spectrometry following acid hydrolysis (Thermo Jarrell method, Dairy One, Ithaca, NY).

Total CP, fat, ash, and intake energy (**IE**) intakes were calculated for each calf by taking the total DMI for the treatment period $\times$ nutrient content of the diet on a DM basis. Total CP, fat, ash, and TE values for fractions were calculated as follows: component weight $\times$ % nutrient $\times$ DM value from freeze-drying $\times$ DM value from oven. The final EB weight of the animal was calculated by summing the carcass, blood, organs cleaned of digesta, head, hide, feet, tail, and the liver. Initial EB weight was determined by taking the average ratio of empty BW to live BW for the baseline calves and applying it to the weight of each animal at the initiation of treatment. Because baseline calves were harvested within 24 h of blockmates going on treatment, all initial compositional data were derived from baseline calves.

An ANOVA for days on treatment, initial EB weight, final EB weight, and block was conducted using JMP software (JMP version 5, SAS Institute Inc., Cary, NC), and no significant differences were detected. The study was analyzed as a randomized block design, no covariates or additional interactions were included in the model, and the experimental unit was calf. All fecal and respiration scores from calves were tabulated and then

analyzed with chi-square analysis to determine if differences in frequencies of fecal and respiratory scores occurred. Hormones and metabolites at each time interval were analyzed as repeated measures in JMP. The final model included treatment, time, and treatment-by-time interaction, and the effect of block was tested, found to be nonsignificant, and dropped from the model. Calf was the subject specified in the repeated statement. Unadjusted least squares means among treatments and corresponding standard errors are reported. Because the diet effects would not necessarily be linear among treatments, further analysis to assess differences among treatment means was conducted with single-degree-of-freedom orthogonal contrasts.

For the insulin challenge, the plasma glucose area under the curve during the response period (0 to 60 min postinfusion) was determined. The rate of decline for glucose was determined by calculating the slope of the line from 0 to 30 min postinfusion. The total decline in plasma glucose was calculated by taking the difference in plasma glucose at 0 min postinfusion and 30 min postinfusion. The area under the curve analysis for the glucose data for the insulin challenge was carried out using the fit model procedure of JMP. An overall level of statistical significance was established at $P < 0.05$. Statistical trends were defined as $0.05 < P < 0.15$.

RESULTS

Body Composition

Calves remained generally healthy and vigorous throughout the experiment. All calves had successful passive transfer of antibodies, defined as a serum protein refractometer reading greater than 5.5 g/dL (HR-200ATC Refractometer, AFAB Enterprises, Eustis, FL). A respiratory virus affected all calves during wk 6 through 8 of the experiment. Calves generally recovered within 3 d, and overall growth and feed intakes were not affected by the virus among treatments. There were no differences among treatments in the frequency of scour and respiratory scores assessed daily of individual calves; thus, no health data will be presented.

The protein and fat content of the treatment diets did not vary (Table 1). The MCT content, defined as the sum of C6:0, C8:0, C10:0, and C12:0, as a percent of total fatty acids varied among treatment diets (Table 2). The control diet contained 1.95% of the total fat as MCT, whereas the CAP oil diet contained 34.95% MCT, of which 66.6% was in the form of C8:0, and the coconut oil diet contained 38.79% MCT, of which 60.92% was in the form of C12:0. The measured IE values of the treatments were 5.25, 5.18, and 5.20 Mcal/kg for the control, CAP oil, and coconut oil diets, respectively.

Intake energy, protein, fat, and selected fatty acid intakes are presented in Table 3. No differences were detected for energy ($P = 0.45$) and protein ($P = 0.29$) intakes; thus, the experiment was conducted under isocaloric and isonitrogenous conditions. Whereas there was no difference in the total amount of fat consumed ($P = 0.22$), there were differences ($P < 0.01$) in the intake of fatty acids of different chain length as designed (Table 3).

Days on treatment, initial EB weight, final EB weight, and ADG are presented in Table 4. Mean days on treatment and initial and final BW did not differ among treatments. However, the ADG of calves fed the CAP oil treatment was less compared with calves fed the control diet ($P = 0.04$). There were no differences in ADG between the control and coconut oil treatments ($P = 0.32$) or between the coconut oil and CAP oil treatments ($P = 0.28$).

The EB chemical composition and EB gains are presented in Table 5. Empty body protein, ash, and water were similar among treatments. However, we observed a trend ($P = 0.11$) for greater fat content of the EB in the calves fed the coconut oil diet. A similar trend ($P = 0.06$) for EB TE content was observed, with TE increasing from control to CAP oil to coconut oil diets. In addition, calves fed the coconut oil diet had a greater ($P < 0.05$) mean TE content compared with the control-fed and CAP oil-fed calves. There were no differences in EB protein, fat, ash, and water gain, but a trend ($P = 0.13$) for increasing TE gain among treatments occurred.

Total tissue weight and protein, fat, ash, water, and TE content of the carcass; blood and organs; head, hide, feet and tail; and the liver are shown in Table 6. Calves fed the coconut oil diet tended to have a greater fat content in the carcass ($P = 0.07$) compared with control calves. In addition, calves fed coconut oil had significantly heavier livers ($P = 0.04$) that contained more fat ($P = 0.002$) compared with calves fed the control and CAP oil diets. Accordingly, an increase in TE content of the liver was observed ($P = 0.02$).

The blood and organs fraction of control calves weighed significantly more compared with that of the calves fed the coconut oil diet, and the blood and organs fraction of the calves fed the coconut oil diet weighed significantly more compared with that of the calves fed the CAP oil diet ($P = 0.03$). This difference in tissue weight resulted in the trend of decreased protein content ($P = 0.13$) of calves fed the CAP oil treatment compared with control calves because protein percentage of the fraction was not different. A similar trend for total water content in the blood and organs ($P = 0.13$) occurred. The TE content of the blood and organs fraction was significantly less in the CAP oil-fed calves

Table 3. Calculated DMI and intakes of intake energy (IE), protein, fat, and selected fatty acids¹

Item	Control	CAP oil	Coconut oil	SEM	P-value
DMI, kg	53.68	56.63	53.83	1.97	0.50
IE, Mcal	281.83	297.17	280.35	10.33	0.45
IE from MCT, ² Mcal	2.10 ^a	41.10 ^b	42.76 ^b	1.37	<0.01
Calories from MCT, %	0.74 ^a	13.82 ^b	15.25 ^c	0.02	<0.01
Protein intake, kg	14.58	15.81	15.37	0.55	0.29
Fat intake, kg	12.97	14.17	13.28	0.49	0.22
C6:0 intake, kg	0.03 ^a	0.21 ^b	0.12 ^c	0.01	<0.01
C8:0 intake, kg	0.04 ^a	3.30 ^b	1.22 ^c	0.07	<0.01
C10:0 intake, kg	0.05 ^a	1.33 ^b	0.67 ^c	0.03	<0.01
C12:0 intake, kg	0.13 ^a	0.11 ^a	3.14 ^b	0.08	<0.01
C14:0 intake, kg	0.27 ^a	0.28 ^a	1.32 ^b	0.04	<0.01
C16:0 intake, kg	3.02 ^a	2.30 ^b	1.93 ^c	0.08	<0.01
C18:1 intake, kg	4.97 ^a	3.51 ^b	2.54 ^c	0.12	<0.01
C18:2 intake, kg	1.82 ^a	1.33 ^b	0.97 ^c	0.05	<0.01
C18:3 intake, kg	0.08 ^a	0.08 ^a	0.05 ^b	0.01	<0.01

^{a-c}Within a row, values with different superscripts are significantly different as determined by orthogonal contrast ($P < 0.05$).

¹Treatments: control [diet contained no medium-chain triglycerides (MCT)], CAP oil (diet contained 32% of fatty acids as MCT with a greater proportion of C8:0), and coconut oil (diet contained 32% of fatty acids as MCT with a greater proportion of C12:0).

²Intake energy from MCT is the total intake from C6:0, C8:0, C10:0, and C12:0 $\times$ 8.3 cal/g (Aurousseau et al., 1984).

compared with the control- and coconut oil-fed calves ($P = 0.04$). There were no treatment differences for the head, hide, feet, and tail fraction.

Composition of the EB on a percentage basis is shown in Table 7. Whole EB protein, ash, and water were not different among treatment groups. Similar to the individual tissue components, there was a trend for the coconut oil-fed calves to have a greater percent fat ($P = 0.11$), which was significantly greater than that of the control calves ($P = 0.04$). On a dry EB basis, there was a trend for less protein content and greater fat content in the coconut oil treatment compared with the control and CAP oil treatments ($P = 0.14$ and $P = 0.15$, respectively).

Insulin Challenge

Figure 1 shows that all calves realized an increase in plasma insulin during the insulin challenge by 2.5 min

postinfusion; thus, the time portion of the model was different ($P < 0.01$) for all treatments. There was no difference in plasma insulin among treatments ($P = 0.14$) and no interaction between treatment and time ($P = 0.79$).

The effects of the insulin challenge on plasma glucose concentrations are shown in Figure 2. No treatment, time, or treatment-by-time interactions were significant ($P = 0.23$, 0.46, and 0.94 for each of the respective model statements). Plasma glucose started to decline between 0 and 2.5 min postinfusion, and the decline continued for 30 min postinfusion before returning to preinfusion levels by 60 min postinfusion.

Total glucose response to the insulin challenge was determined using 3 different methods (Table 8). The area under the curve represents the total effect of insulin on glucose concentrations. A treatment difference ($P = 0.001$) was detected, with calves fed the CAP oil diet having a total response area greater than that

Table 4. Days on treatment, initial and final full BW, and ADG for all treatments¹

Item	Control	CAP oil	Coconut oil	SEM	P-value
n	10	10	10		
Days on treatment	42.70	45.70	44.00	1.94	0.56
Initial BW, kg	46.58	48.03	46.98	1.50	0.78
Final BW, kg	88.48	88.12	87.71	1.01	0.87
Average gain, kg/d	0.99 ^a	0.88 ^b	0.94 ^a	0.03	0.13
Gain-to-feed ratio	0.78 ^a	0.71 ^b	0.76 ^{ab}	0.02	0.11

^{a,b}Within a row, values with different superscripts are different by contrast, $P < 0.05$.

¹Treatments: control [diet contained no medium-chain triglycerides (MCT)], CAP oil (diet contained 32% of fatty acids as MCT with a greater proportion of C8:0), and coconut oil (diet contained 32% of fatty acids as MCT with a greater proportion of C12:0).

Table 5. Whole empty body (EB) weight and gain composition for tissue energy, protein, fat, ash, and water for 3 diets differing in fatty acid profile¹

Item	Control	CAP oil	Coconut oil	SEM	<i>P</i> -value
Initial EB weight, ² kg	44.11	45.49	44.50	1.42	0.78
Final EB weight, kg	83.06	81.27	82.53	1.03	0.46
Empty to live BW, %	93.91 ^{ab}	92.20 ^a	94.11 ^b	0.60	0.06
EB gain, kg/d	0.92 ^a	0.79 ^b	0.87 ^{ab}	0.03	0.04
Whole EB					
Tissue energy, Mcal	133.20 ^a	133.35 ^a	140.33 ^b	2.34	0.06
Protein, kg	14.39	14.00	14.20	0.20	0.40
Fat, kg	7.57	7.62	8.23	0.24	0.11
Ash, kg	3.21	3.17	3.15	0.07	0.88
Water, kg	57.90	56.48	56.95	0.80	0.45
EB gain					
Tissue energy, Mcal	79.00	77.46	85.67	2.95	0.13
Protein, kg	6.52	5.88	6.26	0.26	0.22
Fat, kg	5.56	5.55	6.20	0.26	0.16
Ash, kg	1.33	1.23	1.26	0.07	0.62
Water, kg	25.55	23.13	24.32	1.22	0.39

^{a,b}Values within a row with different superscripts are statistically different. Orthogonal contrasts used to determine differences among treatment means ($P < 0.05$).

¹Treatments: control [diet contained no medium-chain triglycerides (MCT)], CAP oil (diet contained 32% of fatty acids as MCT with a greater proportion of C8:0), and coconut oil (diet contained 32% of fatty acids as MCT with a greater proportion of C12:0).

²Initial EB weight was calculated as 94.7% of initial BW, derived from EB weight data of baseline calves.

of the calves fed the coconut oil and control diets. However, the rate of decline and the total decline were not different among treatments ($P = 0.66$ and 0.46 , respectively), but the total area was different due to the recovery time.

The plasma NEFA concentrations of calves during the insulin challenge are shown in Figure 3, and a treatment effect ($P = 0.02$) was detected. The calves fed coconut oil had the highest NEFA concentrations throughout the insulin challenge. In addition, a time effect ($P < 0.01$) was observed, with the greatest NEFA concentrations occurring from -5 to 2.5 min relative to insulin infusion. No treatment-by-time interactions were observed ($P = 0.67$).

DISCUSSION

Body Composition

The body composition of preruminant calves can be altered by varying the fat and carbohydrate ratio of the diet, as evidenced by Donnelly (1983) and Tikofsky et al. (2001). Tikofsky et al. (2001) found that calves fed a diet high in fat (30.62%) deposited more fat on an EB basis compared with calves fed a diet low in fat (14.79%) that contained the same IE. Our objective was to determine if the source of calories from different fatty acids also affected body composition. Vegetable oils, rich in MCT, have been identified as potential replacements of animal lipid

sources due to growing concerns over bovine spongiform encephalopathy in countries that ban the use of animal products.

Based on the measurements taken in this study, we reject the hypothesis that feeding MCT altered the body composition of calves, with the exception of an increase in the lipid content of the liver of coconut oil-fed calves. There are 2 potential reasons why MCT did not alter body composition: the amount of MCT fed relative to other studies and the cold environment in which the study was conducted.

Whole bovine milk fat consists of triglycerides containing 8 to 12% MCFA (Jensen, 2002). Although milk and milk replacer contain similar amounts of saturated fat, the fatty acid profile of whole milk differs significantly from commercial milk replacers, in which the fat source is mainly edible lard or tallow that lacks short- and medium-chain fatty acids. To elucidate and evaluate potential effects on growth and body composition, milk-replacer diets were designed to contain 32% of the total fatty acids as MCT, about 2.7 times the amount of MCT found in whole milk. The theoretical total intake of MCT from whole milk using the DMI from this study was 1.88 kg. In this study, calves consumed 4.95 and 5.15 kg of MCT from the CAP oil and coconut oil diets, respectively.

Jenkins et al. (1985) studied the effects of feeding over 60% of the total lipid as MCT in preruminant calves. Calves fed MCT had a similar ADG compared with control calves; however, calves had significantly

Table 6. The total weight and protein, fat, ash, water, and tissue energy (TE) content of the empty body components for baseline and treatment animals¹

Item	Baseline ²	Control	CAP oil	Coconut oil	SEM ³	P-value
n	6	10	10	10		
Carcass, kg	25.86	50.94	50.41	50.38	0.79	0.86
TE, Mcal	30.77	80.97 ^a	83.48 ^{ab}	86.37 ^b	1.68	0.09
Protein, kg	4.42	8.87	8.72	8.63	0.16	0.56
Fat, kg	1.11	4.55 ^a	4.70 ^{ab}	5.09 ^b	0.16	0.07
Ash, kg	1.30	2.33	2.31	2.31	0.057	0.97
Water, kg	19.03	35.20	34.69	34.36	0.59	0.60
Blood and organs, kg	7.24	15.82 ^a	14.68 ^b	15.40 ^c	0.28	0.03
TE, Mcal	7.83	25.32 ^a	22.51 ^b	25.29 ^a	0.86	0.04
Protein, kg	0.98	2.09 ^a	1.90 ^b	2.00 ^{ab}	0.05	0.04
Fat, kg	0.37	1.68	1.50	1.72	0.08	0.13
Ash, kg	0.08	0.14	0.13	0.13	0.003	0.27
Water, kg	5.82	11.91 ^a	11.15 ^b	11.54 ^{ab}	0.26	0.13
HHFT, ⁴ kg	9.62	13.88	13.74	13.99	0.26	0.80
TE, Mcal	13.76	23.63	24.06	24.83	0.46	0.20
Protein, g	2.16	2.94	2.88	3.03	0.062	0.25
Fat, g	0.47	1.20	1.29	1.24	0.04	0.26
Ash, g	0.48	0.70	0.69	0.66	0.02	0.50
Water, kg	6.51	9.04	8.87	9.06	0.20	0.77
Liver, kg	1.14	2.43 ^a	2.43 ^a	2.76 ^b	0.10	0.04
TE, Mcal	1.46	3.27 ^a	3.30 ^a	3.84 ^b	0.14	0.02
Protein, kg	0.25	0.49	0.49	0.53	0.02	0.24
Fat, kg	0.05	0.13 ^a	0.14 ^a	0.18 ^b	0.008	0.002
Ash, kg	0.02	0.04 ^a	0.04 ^a	0.05 ^b	0.002	0.08
Water, kg	0.81	1.76 ^a	1.76 ^a	2.00 ^b	0.07	0.04

^{a-c}Values within a row with different superscripts are statistically different. Linear contrasts used to determine differences among treatment means ($P < 0.05$).

¹Treatments: control [diet contained no medium-chain triglycerides (MCT)], CAP oil (diet contained 32% of fatty acids as MCT with a greater proportion of C8:0), and coconut oil (diet contained 32% of fatty acids as MCT with a greater proportion of C12:0).

²Baseline data are provided as reference for initial composition.

³SEM for treatment animals, $n = 30$.

⁴Body components consisting of head, hide, feet, and tail.

lesser gain-to-feed ratios (0.62 and 0.67 for the MCT and control treatments, respectively). Our calves fed the control and coconut oil treatments had similar gain-to-feed ratios (0.78 and 0.76 for the 2 diets, respectively), a result of feeding less of the total lipid as MCT

compared with Jenkins et al. (1985), who suggested minimizing the MCT content of the diet. Differences observed between our study and that of Jenkins et al. (1985) could be due to the temperature effects and the level of nutrient intake.

Table 7. Whole empty body and dry empty body composition of calves fed isocaloric and isonitrogenous diets composed of different fatty acid profiles¹

Item	Control	CAP oil	Coconut oil	SEM	P-value
Whole empty body composition					
Protein, %	17.32	17.23	17.21	0.15	0.86
Fat, %	9.11 ^a	9.41 ^{ab}	9.96 ^b	0.28	0.11
Ash, %	3.86	3.90	3.82	0.07	0.75
Water, %	69.71	69.47	69.01	0.27	0.19
Dry empty body composition					
Protein, %	57.20 ^a	56.45 ^{ab}	55.56 ^b	0.56	0.14
Fat, %	30.04	30.78	32.08	0.73	0.15
Ash, %	12.75	12.77	12.36	0.27	0.49

^{a,b}Values within a row with different superscripts are statistically different. Orthogonal contrasts used to determine differences among treatment means ($P < 0.05$).

¹Treatments: control [diet contained no medium-chain triglycerides (MCT)], CAP oil (diet contained 32% of fatty acids as MCT with a greater proportion of C8:0), and coconut oil (diet contained 32% of fatty acids as MCT with a greater proportion of C12:0).

Figure 1. Concentrations of plasma insulin presented by treatment relative to time of insulin infusion. Data shown are means $\pm$ SE. TRT = treatment. Treatments: control [diet contained no medium-chain triglycerides (MCT)], CAP oil (diet contained 32% of fatty acids as MCT with a greater proportion of C8:0), and coconut oil (diet contained 32% of fatty acids as MCT with a greater proportion of C12:0).

In this study, calves fed the coconut oil diet had heavier livers that contained 38% more lipid compared with calves fed the control and CAP oil diets. In a previous study, feeding 50% of the total lipid as MCT versus feeding a diet containing all LCT resulted in an increased rate and extent of hepatic oxidation of MCFA compared with long-chain fatty acids, and no differences in ADG or organ weights between the diets were observed (Piot et al., 1999). In addition, an 18-fold increase in hepatic accumulation of triglycerides occurred in the calves fed the diet high in MCT compared with calves fed LCT (Piot et al., 1999). The researchers suggested that this increased lipid accumulation results from a higher rate of hepatic de novo lipogenesis or fatty acid elongation. In their study, the peroxisomal oxidation of C12:0 was increased, which suggests that after an initial peroxisomal β -oxidation to shorter fatty acids, acyl-CoA or acetyl-CoA, de novo synthesis or chain elongation occurred. Thus, Piot et al. (1999) suggested that C12:0 can be retailored into long-chain fatty acids and incorporated into liver triglycerides. We had thought this observed increase in liver lipid content might be an artifact of lesser demand for energy. This does not appear to hold true because energy required for maintenance during the entire treatment period in our trial was high due to cold stress conditions.

The coconut oil diet contained significantly more C12:0 and C14:0 than the control and CAP oil diets. The fatty acid C14:0 has been associated with greater atherosclerotic capacity compared with other fatty acids in humans (Tholstrup et al., 2003), which may result

in greater fat deposition in the liver because C14:0 is difficult to metabolize. Graulet et al. (2004) observed a 12-fold increase in liver lipid accumulation in calves fed coconut oil-based diets; however, it is still unknown as to why this accumulation occurs.

Another explanation for why MCT diets did not alter body composition was the cold stress conditions the calves were exposed to for the duration of the trial. The thermoneutral range for calves less than 3 weeks of age is 15 to 25°C (Schrama et al., 1993; Arieli et al., 1995). The lower critical temperature of newborn calves is approximately 13°C and decreases with increasing age of the animal to 6°C at 30 d of age (Gonzalez-Jimenez and Blaxter, 1962). In this experiment, all mean weekly temperatures were below 6°C. Scibilia et al. (1987) studied maintenance energy requirements for milk-fed calves at -4°C and 10°C using 3 fat levels and found that the NE_M requirement for the 2 groups was 134 $\text{kcal/kg}^{0.75}$ and 100 $\text{kcal/kg}^{0.75}$, an increase of 33%. In this experiment, calves had an average TE gain of 1.85, 1.69, and 1.95 Mcal/d and an average IE of 6.6, 6.5, and 6.4 Mcal/d for the control, CAP oil, and coconut oil treatments, respectively. Using a conversion value of 0.912 for IE to ME and an efficiency of use of energy for growth of 0.60 (Van Amburgh and Drackley, 2005), the calculated maintenance energy cost for these calves was 127.6 $\text{kcal/kg}^{0.75}$, 136.3 $\text{kcal/kg}^{0.75}$, and 113.2 $\text{kcal/kg}^{0.75}$ for the control, CAP oil, and coconut oil treatments, respectively. These values are similar to the calculated

Figure 2. Concentrations of plasma glucose presented by treatment relative to time of insulin infusion. Data shown are means $\pm$ SE. TRT = treatment. Treatments: control [diet contained no medium-chain triglycerides (MCT)], CAP oil (diet contained 32% of fatty acids as MCT with a greater proportion of C8:0), and coconut oil (diet contained 32% of fatty acids as MCT with a greater proportion of C12:0).

Figure 3. Concentrations of plasma NEFA presented by treatment relative to time of insulin infusion. Data shown are means $\pm$ SE. TRT = treatment. Treatments: control [diet contained no medium-chain triglycerides (MCT)], CAP oil (diet contained 32% of fatty acids as MCT with a greater proportion of C8:0), and coconut oil (diet contained 32% of fatty acids as MCT with a greater proportion of C12:0).

maintenance requirement of 124 kcal/kg^{0.75} for the average calf with a lower critical temperature of 6°C, using the value of an additional maintenance requirement of 2.15 kcal/kg^{0.75} for every degree Celsius below the lower critical temperature (NRC, 2001). We believe that the increase in energy demand due to cold stress conditions resulted in the increased oxidation of fatty acids for use in maintaining body temperature; thus, effects of the levels of MCT fed during the trial on body composition could not be elucidated.

The ADG during the treatment period were 0.99, 0.88, and 0.94 kg/d for the control, CAP oil, and coconut oil treatments, respectively. The rate of gain for all treatments exceeded other studies performed in pre-ruminant calves, in both cold stress and thermoneutral conditions. Jaster et al. (1992) investigated effects of increasing total caloric intake as extra fat or increasing milk-replacer solids during periods of cold stress and

found that health and growth performance of calves was improved when additional fat was supplemented. Average daily gains for calves ranged from 0.33 to 0.41 kg/d, significantly less than the ADG observed in our trial. However, the IE of calves in Jaster et al. (1992) ranged from 114 to 148 Mcal of IE for calves 7 to 42 d of age, whereas calves in our trial consumed 280 to 297 Mcal of IE during the same period. Thus, increasing the total caloric intake will increase ADG in cold stressed calves. Jaster et al. (1992) also found that supplementing fat also increased ADG; however, as fat supplementation increased, megacalories of IE per kilogram of gain increased, suggesting a decrease in efficiency of use of energy as the calories from fat intake increased. This decrease in efficiency is most likely a function of lower total energy intake and protein-deficient diets and might also indicate that calories from fat are not efficient at providing energy for protein synthesis in the preruminant calf (Tikofsky et al., 2001).

Hill et al. (2007) evaluated addition of fat to milk replacer in the form of coconut oil or canola oil (high in C18:3, considered to be an essential fatty acid) on the growth and efficiency of gain of calves from 0 to 56 d of age. Milk-replacer treatment diets were designed to contain 3% coconut oil, 1% canola oil, or a combination of 3% coconut oil and 1% canola oil. Fat levels in the milk-replacer diets were not different, and there were no differences in ADG between calves fed a control diet and those fed the diet containing coconut oil. However, calves demonstrated an increase ($P < 0.05$) in ADG when fed the diet containing canola oil, suggesting a limitation in C18:3; however, ADG in the Hill et al. trial ranged from 0.51 to 0.61 kg/d, significantly less than that of the calves on our trial. The inclusion rates of coconut oil were much less in their trial, which might have made it difficult to elucidate a growth response (Hill et al., 2007).

Piot et al. (1999) achieved an ADG for the diets containing LCT and MCT of 0.86 and 0.91 kg/d, which are comparable to our values. The total IE for the calves in the study conducted by Piot et al. (1999) was 5.7 Mcal of IE/d, about 0.5 Mcal of IE/d less than the calves in our trial. Thus, cold stress had an effect on

Table 8. Plasma glucose response to insulin challenge¹

Item	Control	CAP oil	Coconut oil	SEM	P-value
Area under curve, mg/dL	627.37 ^a	1,360.68 ^b	781.57 ^a	107.61	0.001
Rate of decline, mg/min	0.07	0.08	0.06	0.02	0.66
Total decline, mg/dL	15.40	24.30	18.90	4.88	0.46

^{a,b}Values within a row with different superscripts are statistically different. Orthogonal contrasts used to determine differences among treatment means ($P < 0.05$).

¹Treatments: control [diet contained no medium-chain triglycerides (MCT)], CAP oil (diet contained 32% of fatty acids as MCT with a greater proportion of C8:0), and coconut oil (diet contained 32% of fatty acids as MCT with a greater proportion of C12:0).

energy utilization, and we believe that these conditions resulted in fatty acids being oxidized for heat production. Therefore, no effects on body composition other than the liver were apparent. However, the observation of increased fat deposition in the calves fed the coconut oil diet despite the cold stress does invite more investigation.

Insulin Challenge

No difference in plasma insulin concentrations was detected at any time point of the insulin challenge; thus, conclusions made about the effects of insulin on plasma metabolites can be attributed to treatment. Plasma NEFA concentrations were greater in the calves fed the coconut oil diet compared with that of calves fed the CAP oil and control diets ($P = 0.02$). These animals also tended to have greater fat in the total EB ($P = 0.11$) and in the carcass ($P = 0.07$) compared with the MCT- and control-treatment calves, suggesting that fat metabolism is altered when calves are fed MCT in the form of C8:0 or C12:0.

There are 2 hypotheses for the increased insulin responsiveness observed in animals fed MCT. The rate of β -oxidation of MCFA in the liver and muscle has been correlated with an increase in glucose sensitivity during an insulin challenge (Han et al., 2003; Perdomo et al., 2004). To determine effects of feeding MCT on adipose mass and insulin responsiveness of rats, Han et al. (2003) fed diets that contained greater than 90% of the lipid as either MCT or LCT. Rats fed MCT had significantly less adipose tissue mass and a greater plasma glucose decrease when subjected to an insulin challenge. In addition, the rats fed MCT had a greater glucose tolerance. The hypothesis that Han et al. (2003) proposed for the lesser adipose mass and improved insulin sensitivity is a suppression of peroxisome proliferator-activated receptor gamma (PPAR γ), a major adipogenic transcription factor. Peroxisome proliferator-activated receptor gamma is expressed in white and brown adipose tissue and stimulates adipocyte differentiation (Kubota et al., 1999). The ligands for PPAR γ include long-chain fatty acids and eicosanoids (Kliwer et al., 1997). Medium-chain fatty acids have a low affinity for PPAR γ (Forman et al., 1997; Kliwer et al., 1997; Han et al., 2003) at low circulating fatty acid concentrations. Han et al. (2003) demonstrated that octanoate inhibits PPAR γ and reduces synthesis of cellular triglycerides. This decrease in adipose tissue accretion results in protection against the negative feedback of fatty acids on insulin signaling (Perdomo et al., 2004).

However, the calves in the current study did not have less fat among treatments. The calves fed the CAP oil diet demonstrated the greatest insulin responsiveness

by having a significantly larger area under the curve for plasma glucose. There was no difference in the total EB fat, fat deposited, or percent fat in any given body fraction compared with the control calves. Thus, the hypothesis of relating insulin sensitivity to body composition is not supported by the current data. Calves that received MCT primarily in the form of C8:0 and C10:0 did not have lesser body fat stores but exhibited greater insulin responsiveness, in contrast to the hypothesis of Han et al. (2003).

Perdomo et al. (2004) observed that by increasing β -oxidation in muscle cells by overexpressing carnitine palmitoyltransferase I, there was an enhancement of ability of insulin to cause phosphorylation of p38MAPK, a signaling molecule believed to be involved in insulin stimulation of glucose uptake in muscle cells, possibly through increasing the activity of plasma membrane Glut4 glucose transporters. Although the source of fatty acids was not directly studied, Perdomo et al. (2004) observed that overexpressing carnitine palmitoyltransferase I in cells had no effect on the oxidation of octanoate, whereas the oxidation of palmitate and oleate was increased 2-fold, suggesting that this rate-limiting step of oxidation is bypassed by MCFA and in vivo, MCFA are more rapidly oxidized, thus resulting in greater insulin responsiveness.

The data from the calves in the present study agree with this hypothesis for increased insulin responsiveness. Changes in body composition were not a component for the explanation of increased insulin responsiveness, whereas increased β -oxidation appears to have a large role in the regulation of glucose metabolism. However, differences in plasma β -hydroxybutyrate in calves in our study could not be detected (data not shown).

CONCLUSIONS

The inclusion of MCT in the form of CAP oil or coconut oil had significant effects on the profile of fatty acids consumed. The CAP oil calves had less ADG compared with the control and coconut oil calves, but no significant effects on the total EB composition were detected. The livers of calves fed coconut oil were larger and contained more lipid, suggesting that the hepatic metabolism of C12:0 was modified, possibly by increasing the rate of β -oxidation. To further elucidate the effects of MCT on body composition, the trial should be performed in both cold stress and thermoneutral conditions.

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