Responses to Increasing Amounts of High-Oleic Sunflower Fatty Acids Infused into the Abomasum of Lactating Dairy Cows¹

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ABSTRACT

Increasing the oleic acid (18:1 *cis*-9) content of milk fat might be desirable to meet consumer concerns about dietary healthfulness and for certain manufacturing applications. The extent to which milk fat could be enriched with oleic acid is not known. Increasing the intestinal supply of polyunsaturated fatty acids decreases dry matter intake (DMI) in cows, but the effects of oleic acid have not been quantified. In a cross-over design, 4 multiparous Holstein cows were abomassally infused with increasing amounts (0, 250, 500, 750, or 1,000 g/d) of free fatty acids from high-oleic sunflower oil (HOSFA) or with carrier alone. Continuous infusions (20 to 22 h/d) were for 7 d at each amount. Infusions were homogenates of HOSFA with 240 g/d of meat solubles and 11.2 g/d of Tween 80; controls received carrier only. The HOSFA contained (by wt) 2.4% 16:0, 1.8% 18:0, 91.4% 18:1 *cis*-9, and 2.4% 18:2. The DMI decreased linearly (range 22.0 to 5.8 kg/d) as the infused amount of HOSFA increased. Apparent total tract digestibilities of dry matter, organic matter, neutral detergent fiber, and energy decreased as the infusion increased to 750 g/d and then increased when 1,000 g/d was infused. Digestibility of total fatty acids increased linearly as infused fatty acids increased. Yields of milk, fat, true protein, casein, and total solids decreased quadratically as infused amounts increased;

decreases were greatest when 750 or 1,000 g/d of HOSFA were infused. Concentrations of fat and total solids increased at the higher amounts of HOSFA. The volume mean diameter of milk fat droplets and the diameter below which 90% of the volume of milk fat is contained both increased as HOSFA infusion increased. Concentrations of short-chain fatty acids, 12:0, 14:0, and 16:0 in milk fat decreased linearly as HOSFA increased. The concentration of 18:1 *cis*-9 (19.4 to 57.4% of total fatty acids) increased linearly as HOSFA infusion increased. Concentrations of 18:1 *cis*-9 in blood triglyceride-rich lipoproteins increased linearly as infusion increased, whereas contents of 14:0, 16:0, 18:0, total 18:1 *trans*, and 18:2n-6 decreased linearly. The composition and physical characteristics of milk fat can be altered markedly by an increased intestinal supply of 18:1 *cis*-9, which could influence processing characteristics and the healthfulness of milk fat. However, an increased supply of free 18:1 *cis*-9 to the intestine decreased DMI and milk production. **Key words:** milk fat, oleic acid, fatty acid, supplemental fat

INTRODUCTION

Increasing oleic acid in milk fat at the expense of medium-chain saturated fatty acids (**FA**) such as myristic acid and palmitic acid might be a desirable outcome for some applications. Myristic and palmitic acids have been implicated as causing unfavorable blood lipid changes in humans and thus increasing risk for coronary disease, whereas oleic acid is neutral or beneficial (Woodside and Kromhout, 2005). Increased oleic acid in milk fat may improve the characteristics of some dairy products; for example, increasing the oleic acid content improves the spreadability of butter at refrigerator temperature (Enjalbert et al., 1997, 2000). Previous research has demonstrated that it is feasible to increase oleic acid in milk fat by infusion of high-oleic acid oil into the abomasum (Christensen et al., 1994; LaCount et al., 1994; Bremmer et

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al., 1998) or duodenum (Chilliard et al., 1991; Enjalbert et al., 2000) and by supplementing long-chain fatty acids (**LCFA**) as calcium soaps or amides to increase the amount of oleic acid that escapes biohydrogenation in the rumen (Ashes et al., 1992; Lin et al., 1996; Jenkins, 1998). Oleic acid content in milk fat also can be increased by supplemental fats that undergo biohydrogenation in the rumen and increase intestinal supply of stearic acid, a portion of which is then desaturated to oleic acid in the gut or mammary gland (Enjalbert et al., 1997).

The maximum extent to which milk fat can be enriched with oleic acid and the resulting effects on other characteristics of milk, milk fat, and processing qualities are not known. Earlier research from our laboratory showed that oleic acid enrichment of milk fat increased linearly to 30.4% of total milk FA as the amount of FFA from high-oleic sunflower oil (**HOSFA**) infused into the abomasum increased from 0 to 400 g/d (LaCount et al., 1994). Supplemental oleamide at 3.5% of dietary DM increased oleic acid to 48% of total milk FA (Jenkins, 1998). In a second study, Jenkins (1999) fed increasing amounts of oleamide (to 5% of total dietary DM) and reached a maximum enrichment of 43.4% oleic acid in milk fat. We are unaware of dose-response studies that have attempted to achieve a greater postruminal oleic acid supply and enrichment in milk fat.

Practical technologies to substantially increase oleic acid escape from the rumen and delivery to the small intestine for absorption might have negative effects on DMI by cows. Diets supplemented with more unsaturated LCFA supplements decreased DMI more than did supplements consisting of mostly saturated FFA (Harvatiné and Allen, 2005). Previously, we have shown that mixtures of unsaturated FFA infused into the abomasum depress DMI (Drackley et al., 1992; Christensen et al., 1994; Bremmer et al., 1998). Suppressive effects of postruminal soy LCFA on DMI were more than 2 times larger when the LCFA were supplied as FFA rather than as triglycerides (**TG**; Litherland et al., 2005), which explains the larger decreases in DMI in our experiments in which FFA were infused compared with experiments by others who infused intact TG (Gagliostro and Chilliard, 1991; Benson et al., 2001). Whether the negative effect of unsaturated LCFA on intake is attributable to specific LCFA has not been determined accurately. A mixture of FFA resembling the profile of palm oil, containing 37% oleic acid, had less effect on DMI than mixtures high in linoleic acid, but interpretation of the effects of oleic acid were confounded by the presence of appreciable quantities of *trans*-monoene in the mixture (Bremmer

Table 1. Ingredient composition of diet fed to cows

Item	% of DM
Ingredient	
Alfalfa silage	26.00
Corn silage	26.00
Ground shelled corn	24.75
Soybean meal	14.50
Soybean hulls	4.00
Mineral-vitamin premix ¹	0.15
Limestone	1.00
Sodium bicarbonate	0.75
Magnesium oxide	0.15
Dicalcium phosphate	0.50
Salt (plain)	0.20
Component	
DM, %	61.9
OM, % of DM	92.6
NDF, % of DM	30.3
ADF, % of DM	21.5
CP, % of DM	15.9
Total fatty acids, % of DM	2.2
NE _L , ² Mcal/kg DM	1.66

¹Contained 5.0% Mg, 7.5% K, 10.0% S, 0.5% Na, 3.0% Cl, 3.0% Zn, 3.0% Mn, 2.0% Fe, 0.5% Cu, 0.015% Se, 0.004% Co, 0.025% I, 1,000 IU/g of vitamin A, 300 IU/g vitamin D, and 3.5 IU/g of vitamin E.

²Calculated according to NRC (2001) guidelines.

et al., 1998). Therefore, the effects of oleic acid per se remain unclear.

Our hypothesis was that oleic acid reaching the small intestine would increase oleic acid in milk fat and decrease DMI in a dose-dependent manner. The objectives of this experiment were to determine the effects of increasing amounts of free oleic acid infused into the abomasum on DMI, total tract digestibility of nutrient fractions, milk yield and composition, and FA profiles in milk fat and blood TG.

MATERIALS AND METHODS

All procedures were conducted under protocols approved by the University of Illinois Laboratory Animal Care Advisory Committee. Four multiparous Holstein cows (mean DIM = 116) that had been fitted previously with ruminal cannulas (10-cm center diameter; Bar Diamond, Parma, ID) were housed in tie stalls fitted with rubber mats, were bedded with straw, and had continuous access to water from individual drinking cups. Cows were milked at 0630 and 1700 h and were allowed to exercise in an outside lot from 0700 to 0900 h daily. The lactation diet (Table 1) was mixed as a TMR and fed twice daily at the time of milking in amounts to ensure ad libitum intake. Orts were removed and weighed once daily.

Treatments consisted of homogenized aqueous mixtures of FFA from HOSFA or a control containing only the emulsifying ingredients. Treatments were admin-

Table 2. Schematic of experimental design and application of treatments

Cow	Preliminary	Week											
		Period 1					Washout		Period 2				
		1	2	3	4	5	6	7	8	9	10	11	12
Amount of HOSFA infused ¹ (g/d)													
6088	W	0	250	500	750	1,000	W	W	0	0	0	0	0
6097	W	0	250	500	750	1,000	W	W	0	0	0	0	0
6144	W	0	0	0	0	0	W	W	0	250	500	750	1,000
6257	W	0	0	0	0	0	W	W	0	250	500	750	1,000

¹HOSFA = high-oleic sunflower fatty acids. W = cows were infused with water only. For 0 g/d of HOSFA, cows received carrier in water.

istered by abomasal infusion to avoid ruminal biohydrogenation of unsaturated FFA. The control infusate consisted of 240 g/d of meat solubles (APC Inc., Ankeny, IA) and 11.2 g/d of Tween 80 (Sigma Chemical Co., St. Louis, MO) in 10 L of tap water. The HOSFA mixture contained the same ingredients as the control plus HOSFA FFA (Henkel Corporation, Emery Division, Cincinnati, OH) at 0, 250, 500, 750, or 1,000 g/d. As determined by GC of methyl esters (Sukhija and Palmquist, 1988), the HOSFA contained (weight basis) 2.45% 16:0, 1.80% 18:0, 91.36% 18:1 *cis*-9, 1.00% unidentified *cis*-18:1 isomers, 2.35% 18:2n-6, and 0.9% other FA.

The design of the experiment (Table 2) was essentially as used previously (LaCount et al., 1994; Graves et al., 2007). The 4 cows were administered the 2 treatments in a crossover design. After a 1-wk preliminary period in which cows were infused only with water, 2 cows received the control infusate and 2 cows received the HOSFA infusate. During this 5-wk experimental period (period 1), the 2 cows receiving the HOSFA infusate received each amount (0, 250, 500, 750, and 1,000 g/d) sequentially, with each amount being infused for 7 d before increasing to the next amount. During period 1, the 2 control cows received only the carrier infusate for the entire 5 wk. Measurements were made during the last 3 d of each infusion amount. At the end of wk 5, all cows were returned to water infusion for a 2-wk washout period before being changed to the opposite treatment for period 2. In period 2, the procedures were repeated so that the other 2 cows received the HOSFA doses in sequentially increasing amounts, and the cows that previously received HOSFA received the control infusate.

Infusates were prepared weekly at the pilot plant of the University of Illinois Food Science and Human Nutrition Department essentially as described previously (Bremmer et al., 1998). The ingredients were mixed and heated to 72°C in steam-jacketed stainless steel vats and were then homogenized into stable emulsions with a 3-piston, 2-stage homogenizer (model

M3, Gaulin Homogenizers, Everett, MA) at 17.24 MPa during the first stage and 3.45 MPa during the second stage. Homogenized mixes were cooled and stored at 4°C until use. Each day, the appropriate amount of infusate for each cow was weighed into tared buckets that were stirred continuously while being infused for 20 to 22 h daily. Abomasal infusion was accomplished by placing an apparatus into the abomasum by way of the ruminal cannula as described by Bremmer et al. (1998). Placement of the infusion apparatus was confirmed daily to ensure postruminal delivery of infusion treatments. Solutions were pumped into the abomasum by using peristaltic pumps (Harvard Apparatus, South Natick, MA). Any infusate remaining in the bucket before cows were turned out of the barn in the morning was administered via the infusion apparatus by use of 60-mL syringes. The amount of fat adhering to the buckets was measured daily and subtracted from the starting amount.

Milk was sampled from the last 6 milkings of each infusion period and stored on ice in a refrigerator until completion of the period. The samples were then composited on a daily basis according to daily milk production. Samples were composited cold after gentle inversion and pouring between containers. If fat adhered to the bottle, the bottle was swirled gently in warm water to loosen fat. Care was taken to avoid heating the samples to avoid alterations of milk fat globule size that occur upon reheating (Smith et al., 1995). An aliquot of the composite sample was used to determine the TS content in duplicate gravimetrically (AOAC, 2000; methods 33.2.26, 989.05). Another aliquot was shipped on ice by overnight courier to the Department of Food Science, Cornell University (Ithaca, NY). The milk was tested in duplicate for fat by Mojonnier ether extraction (AOAC, 2000; methods 33.2.26, 989.05). Kjeldahl N analysis was used to determine (in duplicate) CP N (AOAC, 2000; methods 33.2.11, 991.20), non-CN N (AOAC, 2000; methods 33.2.64, 998.05), and NPN (AOAC, 2000; methods 33.2.12, 991.21). All Kjeldahl N results were expressed on a protein basis (N ×

6.38). True protein was calculated as the difference between CP and NPN ($\times 6.38$), and CN was calculated as the difference between CP and non-CN N ($\times 6.38$). Milk fat globule size distribution was determined by using a laser light-scattering particle size analyzer (Mastersizer E, Malvern Instruments, Worcester, UK) capable of determining particle size distribution between 1 and 80 μm , as described by Smith et al. (1995). A reverse Fourier optical lens with a 45-mm focal length was used, and only forward light scattering was measured. Casein micelles exert minimal influence on light scattering when only forward light scattering is used to measure the particle size distribution of milk fat globules. Two parameters were used to describe milk fat globule size distribution: $d(4,3)$, the volume mean diameter (**VMD**), and $d(0.9)$, the mean fat globule diameter below which 90% of fat volume is contained.

Milk FA were determined by GC analysis of butyl ester derivatives (prepared by methods 963.22, 42.1.29; AOAC, 2000) on a DB-WAX column (30 m $\times$ 0.25 mm $\times$ 0.25 μm ; J & W Scientific, Folsom, CA). The gas chromatograph (model 6890, Hewlett-Packard, Avondale, CA) was equipped with a flame-ionization detector. Chromatographic conditions included a 50:1 split, with He as the carrier gas at 80 kPa gauge and a flow of 25 cm/s at 40°C. Column temperature was 45°C for 2 min, ramped to 230°C at 5°C/min, and then held constant at 230°C for 10 min. A reference standard (GLC-85, Nu Chek Prep Inc., Elysian, MN) was used for peak identification, and triheptadecanoin was used as an internal standard (10 mg/mL; Nu Chek Prep).

Samples of the TMR were obtained on the last 4 d of each sampling week and were frozen at -20°C . At the end of each sampling week, the individual samples were dried at 55°C in an oven for determination of DM content. Samples then were ground through a 2-mm screen in a Wiley mill (Arthur H. Thomas, Philadelphia, PA), composited on an equal-weight basis, re-ground through a 1-mm screen, and stored in glass bottles until analyzed. Orts from individual cows on days that feed samples were obtained were sampled, dried, and ground as described for TMR samples. The dried and ground samples were composited in proportion to the amount of Orts DM each day. Cows were dosed continuously with chromic oxide (10 g in gelatin capsules) twice daily at 0900 and 2100 h via the ruminal cannula. Fecal grab samples were collected at the time of chromium dosing on the last 4 d of each sampling week. Samples were composited immediately by placing 150-g subsamples in a covered plastic pan stored at -20°C . After the last sample was collected,

the pan was dried in an oven at 55°C . Dried samples were ground through a 1-mm screen.

Samples of TMR, Orts, and feces were analyzed for contents of DM (loss of weight after drying at 105°C), ash (600°C for 8 h), CP by Kjeldahl (method 984.13; AOAC, 2000), NDF using α -amylase (Van Soest et al., 1991), ADF (Van Soest et al., 1991), total FA (Sukhija and Palmquist, 1988), and gross energy by bomb calorimetry (1261 Isoperibol Calorimeter, Parr Instrument Co., Moline, IL). Chromium content of feces was analyzed by atomic absorption spectrophotometry (Williams et al., 1962) to calculate apparent digestibilities of nutrient fractions in the total tract.

Blood was sampled by jugular venipuncture on d 6 of each period 3 h after the a.m. feeding for determination of the FA profile of TG in TG-rich lipoproteins. Procedures for collection of blood, separation of TG-rich lipoproteins by centrifugation, extraction of lipids, methylation of FA, and GC analysis were as described by Beaulieu et al. (2002). An additional blood sample was collected before the a.m. feeding on d 5. The evacuated tubes containing sodium heparin (Vacutainer, Becton Dickinson Vacutainer Systems USA, Rutherford, NJ) were centrifuged to obtain plasma, which was stored at -20°C until analyzed for concentrations of NEFA (Johnson and Peters, 1993) and glucose by glucose oxidase (kit number 315, Sigma Chemical Co.; Trinder, 1969).

Data were analyzed statistically by using PROC MIXED of SAS (version 9.1, SAS Institute Inc., Cary, NC). The model contained the random effect of cow and fixed effects of period (i.e., the 5-wk sets of infusion amounts within each treatment type; 1 df), treatment (control or HOSFA; 1 df), amount infused (as a subplot; 4 df), and the interaction of treatment and amount (4 df). Because of concerns about how cows would tolerate sudden and large changes in amounts of HOSFA infused, infusion amounts could not be randomized within periods 1 and 2 and were thus administered sequentially. Therefore, by design, the effect of amount was confounded with time (i.e., weeks). In this analysis, the statistical parameter of interest is the interaction of treatment by amount (LaCount et al., 1994; Graves et al., 2007), which here determined whether cows receiving the HOSFA treatment responded differently with advancing amount (i.e., weeks) compared with control cows. We anticipated that measurements for control cows would be essentially constant over each 5-wk infusion period. The effects of amount and the interaction of treatment by amount were tested by using the error term of cow nested within period and treatment. To model the effects of amount and its interaction with treatment, the REPEATED statement within PROC MIXED of SAS was used. The within-

Table 3. Least squares means and SE for DMI, digestible energy (DE) intake, and intakes of long-chain fatty acids (LCFA) from diet and infusate for cows infused abomasally with increasing amounts of high-oleic sunflower fatty acids (HOSFA)¹

Variable	HOSFA (g/d)					Treatment by amount, <i>P</i>		
	0	250	500	750	1,000	Linear	Quadratic	Cubic
DMI, kg/d	22.0 ± 1.2 (17.6 ± 1.4)	21.7 ± 1.2 (20.3 ± 1.2)	18.6 ± 1.2 (21.0 ± 1.3)	13.1 ± 1.3 (22.0 ± 1.2)	5.8 ± 1.5 (20.9 ± 1.6)	0.0001	0.16	0.81
DE intake, Mcal/d	58.3 ± 4.7 (44.0 ± 4.8)	56.8 ± 4.3 (55.4 ± 4.3)	46.0 ± 4.3 (56.1 ± 4.3)	32.6 ± 4.8 (62.6 ± 4.8)	18.7 ± 5.7 (45.7 ± 5.7)	0.0001	0.28	0.24
Dietary LCFA intake, g/d	437 ± 26 (354 ± 29)	481 ± 26 (461 ± 26)	392 ± 26 (472 ± 26)	281 ± 30 (478 ± 30)	116 ± 34 (443 ± 34)	0.0001	0.24	0.81
Infused LCFA, g/d	0 (0)	224 ± 8 (0)	459 ± 8 (0)	727 ± 9 (0)	856 ± 11 (0)	0.0001	0.0007	0.0004
Total LCFA intake, ² g/d	436 ± 26 (353 ± 29)	706 ± 26 (461 ± 26)	851 ± 26 (472 ± 26)	1,013 ± 29 (477 ± 29)	966 ± 34 (447 ± 34)	0.0001	0.018	0.16

¹Values in parentheses are LSM for cows infused with control infusate for each corresponding infusion amount.

²Dietary plus infused LCFA.

subjects variation was examined by using several covariance structures, and the one yielding the lowest Akaike's information criterion was used in the analysis (Littell et al., 1998, 2000). For all variables, this was the AR(1) option. Polynomial contrasts were constructed to partition the treatment by amount interaction into single degree of freedom interactions of the linear, quadratic, and cubic effects of amount by treatment, and the *P*-values associated with these contrasts are tabulated. Degrees of freedom were determined by using the Kenward-Roger method (Littell et al., 1996). Model residuals were examined and for all variables were normally distributed. Least squares means were calculated and are presented with their standard errors throughout. Significance was declared at *P* < 0.05.

RESULTS AND DISCUSSION

Actual amounts of LCFA infused (Table 3) were slightly less than target values because of small amounts of the HOSFA adhering to the sides of the container and variations in pump flow rate. For the 1,000 g/d infusion, 2 cows stopped eating and milk yield dropped to less than 4 kg/d within 2 d of infusion at that amount; consequently, infusion of the 2 cows was terminated and data were not included for this amount. Actual LCFA infused for the 2 cows at the 1,000 g/d target dosage was 856 g/d. Cows returned to preinfusion milk and DMI within 10 d after the infusion ended. Because we allowed a 14-d washout period between infusion types, we are confident that carryover effects were minimal and did not confound our results. Cows also have returned to preinfusion values within 7 d after abomasal LCFA infusions ceased in our previous experiments (Drackley et al., 1992; Christensen et al., 1994; LaCount et al., 1994; Bremmer et al., 1998; Litherland et al., 2005). Infusion of HOSFA

did not cause diarrhea at any infusion amount. In contrast, abomasal infusion of more than 450 g/d of soy FFA (containing large amounts of 18:2 and 18:3) resulted in diarrhea in an earlier experiment (Drackley et al., 1992).

The DMI decreased linearly as the amount of HOSFA infusion increased (Table 3). The quadratic effect approached significance (*P* = 0.16), indicating that the magnitude of decrease tended to increase when 750 or 1,000 g/d of LCFA was infused. Infusion of LCFA did not compensate for the decreased digestible energy intake from the diet, resulting in decreased total intake of digestible energy as the infusion amount increased. Previously, we have demonstrated that abomasally infused unsaturated FFA, but not saturated FFA, are potent inhibitors of DMI (Drackley et al., 1992; Christensen et al., 1994; Bremmer et al., 1998). The current data extend these observations by demonstrating that monounsaturated FFA also decrease DMI in a dose-dependent manner. The decrease in DMI when 500 g/d HOSFA was infused was greater than that observed previously when 400 g/d of a similar LCFA mixture was infused (LaCount et al., 1994), although doses were administered for only 3 d in that study. Decreases in DMI were also greater than when other unsaturated LCFA mixtures were infused continuously at 450 g/d for 10 to 14 d (Drackley et al., 1992; Christensen et al., 1994; Bremmer et al., 1998). From this comparison we suggest that the hypophagic effects of oleic acid are similar to those of linoleic acid, but this remains to be demonstrated conclusively. The decrease in DMI was less than observed when 400 g/d of soy FFA was infused into the abomasum in pulse doses 4 times daily (Litherland et al., 2005). The suppressive effects of unsaturated LCFA may be mediated by release of gut peptides, in particular glucagon-like peptide 1, in response to increased quantities of unsat-

Table 4. Least squares means and SE for apparent total tract digestibilities (percentage of intake) for cows infused abomasally with increasing amounts of high-oleic sunflower fatty acids (HOSFA)¹

Component	HOSFA (g/d)					Treatment by amount, <i>P</i>		
	0	250	500	750	1,000	Linear	Quadratic	Cubic
DM ²	66.0 ± 1.8 (63.5 ± 2.2)	65.5 ± 1.8 (66.4 ± 1.8)	62.2 ± 1.8 (64.3 ± 1.8)	62.8 ± 2.2 (68.1 ± 2.2)	92.3 ± 2.8 (59.4 ± 2.8)	0.0001	0.0001	0.0007
OM ²	68.0 ± 1.7 (65.8 ± 2.0)	67.2 ± 1.7 (68.3 ± 1.7)	64.5 ± 1.7 (66.2 ± 1.7)	66.0 ± 2.1 (70.0 ± 2.1)	92.8 ± 2.7 (60.9 ± 2.7)	0.0001	0.0001	0.001
CP ²	62.3 ± 2.2 (61.6 ± 2.5)	55.6 ± 2.2 (57.6 ± 2.2)	54.0 ± 2.2 (58.4 ± 2.2)	59.7 ± 2.5 (63.2 ± 2.5)	87.4 ± 3.2 (51.3 ± 3.2)	0.0001	0.0001	0.004
NDF	43.3 ± 3.6 (39.9 ± 4.2)	41.6 ± 3.6 (44.8 ± 3.6)	36.3 ± 3.6 (37.7 ± 3.6)	33.9 ± 4.2 (44.7 ± 4.3)	87.8 ± 5.2 (32.4 ± 5.3)	0.0001	0.0001	0.002
Total LCFA ²	77.1 ± 2.9 (77.0 ± 3.4)	87.9 ± 2.9 (78.8 ± 2.9)	84.2 ± 2.9 (80.5 ± 2.9)	90.0 ± 3.4 (80.1 ± 3.4)	99.0 ± 4.4 (79.5 ± 4.4)	0.033	0.50	0.22
Energy ²	66.1 ± 2.1 (62.9 ± 2.5)	64.6 ± 2.1 (65.7 ± 2.2)	61.6 ± 2.1 (63.9 ± 2.2)	62.4 ± 2.6 (67.3 ± 2.6)	91.7 ± 3.2 (58.3 ± 3.2)	0.0002	0.0001	0.003

¹Values in parentheses are LSM for cows infused with control infusate for each corresponding infusion amount.

²Dietary plus infused long-chain fatty acids (LCFA) and carrier.

urated FFA reaching the proximal small intestine (Benson et al., 2001; Litherland et al., 2005). Others also have noted decreased DMI when fat supplements that delivered increasing quantities of oleic acid to the postruminal tract were fed to Holstein cows (Jenkins, 1999).

Apparent total tract digestibilities of DM, OM, NDF, and energy (Table 4) were affected by interactions of treatment with the cubic effects of increasing infusion amount. Digestibilities for these fractions trended downward as HOSFA infusion increased to 750 g/d, and then increased sharply when 1,000 g/d was infused. Measurements made at the highest infusion amount may be less accurate because data were available for only 2 cows and DMI and fecal output were very low. In contrast, digestibility of total LCFA increased linearly as infusion increased. These data confirm the high intestinal digestibility of oleic acid reaching the small intestine in dairy cows (Klasmeyer and Clark, 1991; Enjalbert et al., 1997; Jenkins, 1998, 1999). Amounts of oleic acid delivered and absorbed postruminally were substantially greater than observed under typical dietary scenarios.

Milk yield decreased markedly as HOSFA infusion increased, which was likely attributable at least in part to the decreased DMI and intakes of digestible nutrients (Table 5). Milk yield remained stable in control cows. Milk yield also decreased in previous studies when unsaturated FFA were infused postruminally (Drackley et al., 1992; Christensen et al., 1994). Milk fat concentration increased as infusion increased, with increases being greater as larger doses were infused (quadratic effect, $P = 0.002$). Milk fat yield was unaffected when up to 500 g/d was infused, but then decreased sharply as milk yield declined (treatment by

quadratic effect of amount, $P = 0.002$). Contents of total protein, true protein, and CN were not affected significantly by infusion, but yields decreased in a quadratic fashion similar to milk yield, with the largest decreases when 750 or 1,000 g/d was infused. The content of NPN in milk responded quadratically to infusion, with the largest concentration occurring when 1,000 g/d was infused; NPN yield in milk followed a pattern similar to other milk components. The content of TS in milk increased as the amount of HOSFA increased, largely as a result of the changes in fat content. In contrast, yield of TS decreased as infusion increased.

Milk fat globule size increases as milk fat yield increases (Wiking et al., 2004) and can affect milk processing characteristics (Fauquant et al., 2005). Because we reasoned that abomasal infusion of HOSFA could affect milk fat yield, we measured changes in the size and distribution of milk fat globules in response to abomasal infusions of HOSFA or the control (Table 5). Both VMD and d(0.9) increased linearly as HOSFA infusion increased, whereas values for control cows did not vary appreciably. Values for VMD and d(0.9) were highly correlated with each other ($r = 0.969$, $P < 0.001$), and both VMD ($r = 0.746$, $P < 0.001$) and d(0.9) ($r = 0.685$, $P < 0.001$) were correlated with milk fat concentration. In contrast to our data, Fauquant et al. (2005) determined that large fat globules (6- μ m diameter) had similar amounts of oleic acid, less medium-chain FA, and more stearic acid than small (3- μ m) milk fat globules. The difference in globule size as HOSFA infusion increased likely was not attributable only to alterations in milk FA composition. Whether the increased milk fat globule size reflected the increased concentration of milk fat in the decreased vol-

Table 5. Least squares means and SE for yield and composition of milk from cows infused abomasally with increasing amounts of high-oleic sunflower fatty acids (HOSFA)¹

Variable	HOSFA (g/d)					Treatment by amount, <i>P</i>		
	0	250	500	750	1,000	Linear	Quadratic	Cubic
Milk, kg/d	27.1 ± 3.0 (23.1 ± 3.1)	26.4 ± 3.0 (22.4 ± 3.0)	23.8 ± 3.0 (24.0 ± 3.0)	14.0 ± 3.0 (22.6 ± 3.0)	6.8 ± 3.2 (22.1 ± 3.0)	0.0001	0.003	0.14
Fat, %	3.29 ± 0.22 (3.12 ± 0.22)	3.49 ± 0.22 (3.20 ± 0.22)	3.71 ± 0.22 (3.17 ± 0.22)	4.46 ± 0.22 (3.27 ± 0.22)	5.93 ± 0.28 (3.18 ± 0.22)	0.0001	0.002	0.18
Fat, kg/d	0.90 ± 0.14 (0.74 ± 0.14)	0.93 ± 0.14 (0.72 ± 0.14)	0.89 ± 0.14 (0.78 ± 0.14)	0.63 ± 0.14 (0.76 ± 0.14)	0.37 ± 0.14 (0.72 ± 0.14)	0.0001	0.002	0.25
Total protein, %	3.14 ± 0.08 (3.11 ± 0.12)	3.17 ± 0.08 (3.12 ± 0.11)	3.12 ± 0.08 (3.15 ± 0.11)	3.28 ± 0.08 (3.19 ± 0.11)	3.52 ± 0.12 (3.19 ± 0.11)	0.13	0.18	0.56
Total protein, kg/d	0.85 ± 0.08 (0.70 ± 0.08)	0.83 ± 0.08 (0.68 ± 0.08)	0.74 ± 0.08 (0.74 ± 0.08)	0.46 ± 0.08 (0.71 ± 0.08)	0.26 ± 0.09 (0.70 ± 0.08)	0.0001	0.013	0.11
True protein, %	2.93 ± 0.10 (2.91 ± 0.11)	2.96 ± 0.10 (2.92 ± 0.10)	2.92 ± 0.10 (2.93 ± 0.10)	3.08 ± 0.10 (2.98 ± 0.10)	3.28 ± 0.13 (2.98 ± 0.10)	0.15	0.26	0.65
True protein, kg/d	0.80 ± 0.07 (0.66 ± 0.08)	0.78 ± 0.07 (0.64 ± 0.07)	0.70 ± 0.07 (0.69 ± 0.07)	0.43 ± 0.07 (0.66 ± 0.07)	0.24 ± 0.08 (0.65 ± 0.07)	0.0001	0.010	0.12
CN, %	2.41 ± 0.08 (2.38 ± 0.09)	2.40 ± 0.08 (2.36 ± 0.08)	2.34 ± 0.08 (2.36 ± 0.08)	2.34 ± 0.08 (2.41 ± 0.08)	2.35 ± 0.11 (2.40 ± 0.08)	0.46	0.94	0.68
CN, kg/d	0.65 ± 0.06 (0.54 ± 0.06)	0.63 ± 0.06 (0.52 ± 0.06)	0.56 ± 0.06 (0.56 ± 0.06)	0.33 ± 0.06 (0.54 ± 0.06)	0.17 ± 0.07 (0.53 ± 0.06)	0.0001	0.022	0.11
NPN, %	0.20 ± 0.01 (0.20 ± 0.01)	0.21 ± 0.01 (0.20 ± 0.01)	0.20 ± 0.01 (0.22 ± 0.01)	0.20 ± 0.01 (0.20 ± 0.01)	0.24 ± 0.02 (0.21 ± 0.01)	0.35	0.04	0.20
NPN, kg/d	0.055 ± 0.005 (0.046 ± 0.005)	0.055 ± 0.005 (0.044 ± 0.005)	0.048 ± 0.005 (0.049 ± 0.005)	0.029 ± 0.005 (0.044 ± 0.005)	0.015 ± 0.005 (0.046 ± 0.005)	0.0001	0.005	0.18
TS, %	12.02 ± 0.19 (11.73 ± 0.21)	12.32 ± 0.19 (11.85 ± 0.19)	12.34 ± 0.19 (11.72 ± 0.19)	12.90 ± 0.19 (11.86 ± 0.19)	14.20 ± 0.23 (11.86 ± 0.19)	0.0001	0.001	0.08
TS, kg/d	3.28 ± 0.41 (2.71 ± 0.41)	3.27 ± 0.41 (2.66 ± 0.41)	2.96 ± 0.41 (2.83 ± 0.41)	1.82 ± 0.41 (2.70 ± 0.41)	0.97 ± 0.42 (2.64 ± 0.41)	0.0001	0.003	0.11
VMD, ² μm	2.98 ± 0.11 (3.02 ± 0.12)	3.19 ± 0.11 (3.00 ± 0.11)	3.32 ± 0.11 (2.98 ± 0.11)	3.60 ± 0.11 (2.94 ± 0.11)	4.02 ± 0.13 (2.90 ± 0.11)	0.0001	0.09	0.46
d(0.9), ³ μm	5.16 ± 0.23 (5.22 ± 0.24)	5.52 ± 0.23 (5.27 ± 0.23)	5.97 ± 0.23 (5.18 ± 0.23)	6.35 ± 0.23 (5.10 ± 0.23)	6.96 ± 0.27 (5.02 ± 0.23)	0.0001	0.33	0.99

¹Values in parentheses are LSM for cows infused with control infusate for each corresponding infusion amount.

²Volume mean diameter of milk fat globules.

³Diameter of milk fat globules below which 90% of the volume of milk fat is contained.

ume of milk or possible alterations in milk fat globule membrane composition (Fauquant et al., 2005), or were a consequence of the decreased DMI and nutrient supply cannot be determined from our data but would be of interest to clarify in subsequent experiments.

Changes in milk fat composition (Table 6) generally were as predicted based on established knowledge of factors affecting FA composition when cows are fed diets supplemented with fats and oils (Palmquist et al., 1993). Increasing the amount of HOSFA infused into the abomasum tended ($P < 0.06$) to linearly decrease the concentration of 4:0. Increasing HOSFA resulted in significant linear decreases in percentages of 6:0, 8:0, 10:0, 12:0, 14:0, 14:1, 15:0, 16:0, 18:0, and 18:3n-3 (Table 6). The content of 18:1 *cis*-9 increased linearly as infusion increased, whereas the percentages of 16:1 *cis*-7, 18:1 *trans*, and 18:2n-6 were not affected.

The percentage of oleic acid in milk fat (57.4%) for cows infused with the largest amount of HOSFA is, to our knowledge, the greatest reported enrichment of oleic acid in milk fat. Enrichment of oleic acid in milk fat during 750 g/d of HOSFA infusion was similar to values reported by Jenkins (1998) for cows fed 3.5% oleamide. From a quantitative standpoint, the increased incorporation of oleic acid primarily came at the expense of 14:0 and 16:0 (Table 6), although all of the shorter-chain FA were decreased as well. Changes were qualitatively similar to those seen previously when up to 400 g/d of HOSFA was infused into the abomasum (LaCount et al., 1994) or when 400 g/d of oleic acid was infused into the duodenum (Enjalbert et al., 2000), except that contents of 4:0 and 6:0 in milk fat were not decreased in the earlier studies. The FA content of milk fat was also altered in a generally similar manner when up to 5% oleamide was fed to

Table 6. Least squares means and SE for fatty acids in milk fat from cows infused abomasally with increasing amounts of high-oleic sunflower fatty acids (HOSFA)¹

Fatty acid	HOSFA (g/d)					Treatment by amount, <i>P</i>		
	0	250	500	750	1,000	Linear	Quadratic	Cubic
	(g/100 g of fatty acid butyl esters)							
4:0	4.54 ± 0.23 (4.18 ± 0.23)	3.99 ± 0.23 (4.00 ± 0.23)	4.10 ± 0.23 (3.95 ± 0.23)	3.67 ± 0.23 (4.27 ± 0.23)	3.34 ± 0.33 (3.84 ± 0.23)	0.056	0.98	0.68
6:0	2.86 ± 0.12 (2.53 ± 0.12)	2.48 ± 0.12 (2.52 ± 0.12)	2.49 ± 0.12 (2.57 ± 0.12)	2.13 ± 0.12 (2.75 ± 0.12)	1.48 ± 0.17 (2.47 ± 0.12)	0.0001	0.45	0.77
8:0	1.71 ± 0.06 (1.51 ± 0.06)	1.48 ± 0.06 (1.53 ± 0.06)	1.49 ± 0.06 (1.60 ± 0.06)	1.22 ± 0.06 (1.67 ± 0.06)	0.75 ± 0.09 (1.52 ± 0.06)	0.0001	0.27	0.50
10:0	3.96 ± 0.17 (3.46 ± 0.17)	3.34 ± 0.17 (3.63 ± 0.17)	3.24 ± 0.17 (3.83 ± 0.17)	2.60 ± 0.18 (3.92 ± 0.17)	1.37 ± 0.26 (3.64 ± 0.17)	0.0001	0.45	0.32
12:0	4.41 ± 0.21 (3.84 ± 0.2)	3.72 ± 0.21 (4.10 ± 0.21)	3.43 ± 0.21 (4.47 ± 0.21)	2.79 ± 0.21 (4.48 ± 0.21)	1.19 ± 0.28 (4.37 ± 0.21)	0.0001	0.29	0.18
14:0	12.42 ± 0.41 (11.01 ± 0.41)	11.12 ± 0.41 (12.15 ± 0.41)	9.83 ± 0.41 (12.91 ± 0.41)	7.96 ± 0.43 (12.65 ± 0.41)	4.32 ± 0.62 (12.58 ± 0.41)	0.0001	0.46	0.23
14:1	1.02 ± 0.15 (0.98 ± 0.14)	0.86 ± 0.15 (0.96 ± 0.14)	0.66 ± 0.20 (1.01 ± 0.17)	0.70 ± 0.16 (1.05 ± 0.14)	0.48 ± 0.18 (1.05 ± 0.14)	0.0001	0.43	0.39
15:0	1.00 ± 0.10 (0.98 ± 0.10)	0.89 ± 0.10 (1.03 ± 0.10)	0.68 ± 0.10 (0.87 ± 0.10)	0.56 ± 0.10 (1.12 ± 0.10)	0.40 ± 0.19 (1.06 ± 0.10)	0.002	0.71	0.76
16:0	28.23 ± 0.99 (26.80 ± 0.99)	25.21 ± 0.99 (27.98 ± 0.99)	20.36 ± 0.98 (29.26 ± 0.99)	19.53 ± 0.99 (29.48 ± 0.99)	15.82 ± 1.30 (29.76 ± 0.99)	0.0001	0.20	0.72
16:1 <i>cis</i> -7	1.32 ± 0.10 (1.62 ± 0.10)	1.01 ± 0.10 (1.30 ± 0.10)	0.77 ± 0.10 (1.37 ± 0.10)	1.03 ± 0.10 (1.32 ± 0.10)	1.08 ± 0.14 (1.36 ± 0.10)	0.91	0.27	0.95
18:0	9.93 ± 0.56 (10.29 ± 0.56)	9.43 ± 0.56 (10.46 ± 0.56)	8.04 ± 0.56 (9.58 ± 0.56)	5.40 ± 0.56 (9.60 ± 0.56)	7.26 ± 0.82 (9.95 ± 0.56)	0.014	0.46	0.08
18:1 <i>cis</i> -9	19.44 ± 1.43 (24.45 ± 1.43)	28.48 ± 1.44 (20.33 ± 1.43)	39.23 ± 1.46 (19.64 ± 1.43)	46.47 ± 1.49 (19.34 ± 1.43)	57.37 ± 2.12 (20.12 ± 1.43)	0.0001	0.20	0.47
18:1 <i>trans</i>	1.72 ± 0.10 (1.92 ± 0.10)	1.45 ± 0.10 (1.58 ± 0.10)	1.29 ± 0.10 (1.60 ± 0.10)	1.12 ± 0.10 (1.54 ± 0.10)	1.23 ± 0.14 (1.66 ± 0.10)	0.17	0.87	0.32
18:2n-6	3.75 ± 0.26 (3.84 ± 0.26)	3.61 ± 0.26 (3.58 ± 0.26)	2.67 ± 0.26 (3.62 ± 0.26)	3.40 ± 0.27 (3.68 ± 0.26)	3.35 ± 0.39 (3.73 ± 0.26)	0.52	0.23	0.54
18:3n-3	0.72 ± 0.05 (0.71 ± 0.04)	0.68 ± 0.04 (0.73 ± 0.04)	0.55 ± 0.05 (0.79 ± 0.04)	0.44 ± 0.06 (0.80 ± 0.04)	0.40 ± 0.10 (0.78 ± 0.05)	0.042	0.43	0.77
18:1 <i>cis</i> -9/18:0	1.97 ± 0.36 (2.38 ± 0.36)	3.07 ± 0.36 (1.98 ± 0.36)	4.92 ± 0.36 (2.09 ± 0.36)	8.66 ± 0.37 (2.02 ± 0.36)	8.08 ± 0.49 (2.05 ± 0.36)	0.0001	0.19	0.001
16:1 <i>cis</i> -7/16:0	0.047 ± 0.0045 (0.061 ± 0.0045)	0.040 ± 0.0045 (0.046 ± 0.0045)	0.037 ± 0.0044 (0.047 ± 0.0045)	0.053 ± 0.0044 (0.045 ± 0.0045)	0.064 ± 0.0061 (0.046 ± 0.0045)	0.003	0.23	0.85
14:1/14:0	0.085 ± 0.014 (0.087 ± 0.013)	0.080 ± 0.013 (0.078 ± 0.013)	0.069 ± 0.017 (0.079 ± 0.016)	0.085 ± 0.015 (0.084 ± 0.013)	0.102 ± 0.023 (0.084 ± 0.013)	0.14	0.28	0.85
Spreadability index ²	1.49 ± 0.11 (1.12 ± 0.11)	0.92 ± 0.11 (1.38 ± 0.11)	0.54 ± 0.11 (1.50 ± 0.11)	0.43 ± 0.11 (1.54 ± 0.11)	0.28 ± 0.16 (1.51 ± 0.11)	0.0001	0.003	0.41
Health index ³	0.31 ± 0.044 (0.42 ± 0.038)	0.47 ± 0.038 (0.32 ± 0.038)	0.69 ± 0.044 (0.30 ± 0.038)	0.97 ± 0.054 (0.30 ± 0.038)	1.65 ± 0.061 (0.32 ± 0.044)	0.0001	0.023	0.43

¹Values in parentheses are LSM for cows infused with control infusate for each corresponding infusion amount.²Calculated as 16:0/18:1 *cis*-9 (Couvreux et al., 2006).³Calculated as (16:1 *cis*-7 + 18:1 *cis*-9 + 18:2n-6 + 18:3n-3)/[12:0 + (4 × 14:0) + 16:0], based on Chen et al. (2004).

lactating cows (Jenkins, 1999), with the exception that 4:0 was not decreased and the content of 18:0 was increased rather than decreased as in the present study. Possible reasons for these differences in responses among studies may be related to the severity of DMI depression caused by the supplemental LCFA, with a corresponding reduction of substrate supply to the mammary gland.

The changes in milk fat composition resulting from increased supply of oleic acid to mammary cells likely occurred through decreased *de novo* synthesis of short- and medium-chain FA (Palmquist et al., 1993) as well as mass-action effects on esterification from the increased supply of oleic acid relative to other FA. Oleic acid is found in milk TG at all 3 *sn*-positions of glycerol. Breckenridge and Kuksis (1968) reported that distri-

Table 7. Least squares means and SE for fatty acids in the triglyceride-rich fraction of plasma lipoproteins from cows infused abomasally with increasing amounts of high-oleic sunflower fatty acids (HOSFA)¹

Fatty acid	HOSFA (g/d)					Treatment by amount, <i>P</i>		
	0	250	500	750	1,000	Linear	Quadratic	Cubic
	(g/100 g of fatty acid methyl esters)							
14:0	2.78 ± 0.35 (3.04 ± 0.35)	2.30 ± 0.51 (2.84 ± 0.52)	1.45 ± 0.35 (2.66 ± 0.35)	1.42 ± 0.35 (2.33 ± 0.35)	0.76 ± 0.52 (2.94 ± 0.35)	0.032	0.62	0.52
16:0	23.44 ± 1.12 (24.58 ± 1.12)	17.89 ± 1.66 (24.13 ± 1.63)	14.03 ± 1.12 (23.02 ± 1.12)	12.45 ± 1.12 (22.05 ± 1.12)	11.62 ± 1.59 (23.02 ± 1.12)	0.0001	0.15	0.54
18:0	49.83 ± 2.25 (47.11 ± 2.25)	32.67 ± 3.16 (47.16 ± 3.16)	27.80 ± 2.25 (51.93 ± 2.25)	14.62 ± 2.25 (52.63 ± 2.25)	10.40 ± 3.27 (49.12 ± 2.25)	0.0001	0.038	0.62
18:1 <i>cis</i> -9	12.43 ± 2.70 (13.38 ± 2.70)	35.06 ± 3.94 (13.60 ± 3.94)	46.70 ± 2.70 (10.57 ± 2.70)	64.86 ± 2.70 (11.18 ± 2.70)	70.70 ± 3.97 (11.61 ± 2.70)	0.0001	0.065	0.76
18:1 <i>trans</i>	2.67 ± 0.41 (2.98 ± 0.41)	3.25 ± 0.68 (3.79 ± 0.67)	2.11 ± 0.41 (3.42 ± 0.41)	1.19 ± 0.41 (3.89 ± 0.41)	1.33 ± 0.66 (4.40 ± 0.41)	0.005	0.74	0.53
18:2n-6	8.86 ± 0.68 (8.90 ± 0.68)	7.92 ± 1.11 (9.37 ± 1.08)	7.90 ± 0.68 (8.39 ± 0.68)	5.48 ± 0.68 (7.91 ± 0.68)	5.28 ± 1.07 (8.91 ± 0.68)	0.035	0.56	0.69

¹Values in parentheses are LSM for cows infused with control infusate for each corresponding infusion amount.

bution of oleic acid was in a ratio of approximately 3:1.8:1 for the *sn*-1, *sn*-2, and *sn*-3 positions, respectively. Esterification of oleic acid at the *sn*-1 site would predominantly displace 14:0, 16:0, and 18:0, whereas oleic would primarily displace 14:0 and 16:0 at the *sn*-2 position (Breckenridge and Kuksis, 1968). The short-chain FA 4:0 and 6:0 are found almost exclusively at the *sn*-3 position, which likely explains the marked decrease of these FA by the increased oleic acid supply in our study.

Manufacturing and organoleptic qualities of the oleic acid-enriched milk fat would be expected to be quite different from those of typical milk fat. Butter produced from milk fat with increased oleic acid and decreased palmitic acid is softer at refrigerator temperature (Enjalbert et al., 1997, 2000). Thus, the ratio of 16:0 to 18:1 *cis*-9 has been used as an index of butter spreadability (Couvreux et al., 2006). This ratio (Table 6) decreased in a quadratic fashion as the amount of infused HOSFA increased. Jenkins (1999) calculated the inverse of this ratio (18:1 *cis*-9/16:0) and found that it increased linearly as oleic acid in milk fat was enriched by increasing amounts of oleamide in the diet. The changes in milk FA profile also resulted in a quadratic increase in the "health index" (Table 6), described by Chen et al. (2004) as the inverse of the atherogenic index first reported by Ulbricht and Southgate (1991).

Changes in milk fat composition were accompanied by corresponding changes in the LCFA profile of TG in the TG-rich lipoprotein fraction of blood (Table 7). The weight percentages of 14:0, 16:0, 18:1 *trans*, and 18:2 decreased linearly, whereas 18:0 decreased in a quadratic fashion as the rate of decrease slowed at the higher infusion amounts. The percentage of oleic acid

increased linearly; the quadratic effect approached significance ($P < 0.07$) as the rate of increase tended to slow at infusion of the 2 largest amounts. The changes were similar to those observed in an earlier study in which HOSFA was infused into the abomasum (LaCount et al., 1994).

Concentrations of NEFA and glucose in blood were affected by interactions of treatment and the quadratic effect of amount (data not tabulated). The concentration of NEFA in plasma averaged 109 μ Eq/L for control cows. The NEFA concentration increased sharply for cows infused with HOSFA at 1,000 g/d (726 μ Eq/L), likely reflecting the state of negative energy balance that resulted from the sharp depression in DMI. The mean concentration of glucose in plasma for control cows was 70.8 mg/dL. Cows infused with HOSFA had similar glucose concentrations until the amount of infused HOSFA reached 1,000 g/d, when glucose concentration increased sharply to 103.5 mg/dL. The elevated glucose concentration may have been a stress response to the suppressed nutrient intake, or may have resulted from the markedly decreased milk yield and resultant decreased mammary use of glucose for lactose output and de novo FA synthesis (Chilliard et al., 1991; Gagliostro et al., 1991).

CONCLUSIONS

Increasing the supply of oleic acid to the postruminal digestive tract can markedly change the FA composition of milk fat. The content of oleic acid in milk fat responds linearly to the amount of intestinally absorbable oleic acid, primarily at the expense of 14:0 and 16:0 as well as shorter chain FA. Thus, these data demonstrate that the bovine mammary gland has the

ability to produce a remarkably different milk fat when presented with a drastically altered profile of milk fat precursors. Such changes in milk fat composition would be expected to have an impact on the nutritional and processing characteristics of milk fat, which would be important to evaluate.

As the amount of oleic acid provided to the small intestine was increased by abomasal infusion, DMI decreased substantially. These data indicate that oleic acid has suppressive effects on DMI in dairy cows, as observed for linoleic and linolenic acids in previous experiments. Provision of postruminal oleic acid in amounts sufficient to result in large changes in milk fat composition may be difficult to achieve without decreasing DMI and nutrient supply to cows.

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