Effects of Rumen-Inert Fat on Lactation, Reproduction, and Health of High Producing Holstein Herds

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ABSTRACT

Two hundred twenty of 443 cows freshening between June 1989 and March 1990 in five commercial Holstein herds were fed .45 kg/d of rumen-inert fat from calving until 200 DIM. Control diets were fed as TMR and contained, on average, 3.7 to 4.8% supplemental fat (DM basis). Test herds had rolling herd averages of 9300 to 13,250 kg of milk. Production of 4% FCM and milk increased 1.01 (3.3%) and 1.50 kg/d (4.6%), respectively, for primiparous cows fed additional fat. Multiparous cows from four herds demonstrated no response; multiparous cows in one herd increased production of 4% FCM by 2.88 kg/d (8.2%), milk by 2.45 kg/d (6.4%), and milk fat by .14 kg/d (10.6%) in response to additional fat. An explanation of response differences among herds for multiparous cows was not possible. For primiparous and multiparous cows, increased genetic potential increased treatment response. Increased body condition score at calving influenced treatment response of multiparous cows. Thinner cows produced more milk and less milk fat in response to additional dietary fat than did fatter cows. Most reproductive indices were unaffected by treatment. Cows receiving additional fat had lower, but nonsignificantly lower, incidences of most health disorders. Responses to rumen-inert fat by cows receiving high concentrations of dietary fat were marginal and were affected by body condition score at calving and by genetic potential.

(**Key words**: rumen-inert fat, lactation, reproduction, health)

Abbreviation key: **BCS** = body condition score, **Ca-LCFA** = Ca salts of palm oil long-chain fatty acids, **MFP** = milk fat percentage, **MNE** = management and nutrition effect, **MPP** = milk protein percentage, **PPA** = predicted producing ability for 4% FCM.

INTRODUCTION

Efficiency of nutrient utilization theoretically is maximal when supplemental dietary fats provide 15 to 20% of dietary metabolizable energy or 7 to 8% dietary fat on a DM basis (15). Responses to supplemental fat have been highly variable, ranging from –4.4 to 9.6 kg/d of 4% FCM per kilogram of added fat (30). In most of those trials, treatment diets contained <6% supplemental fat. When fat has been supplemented at >6% of the diet, 4% FCM production was reduced (6, 25).

Effects of supplemental fat on energy balance have not been consistent. Supplemental fat has been associated with reduced DMI, resulting sometimes in no change in energy intake (13), but at other times has increased energy intake by up to 5% (19), although energy balance may not be improved, because of increased milk production (31). Early postpartum cows fed supplemental fat and experiencing rapid body fat mobilization may reduce DMI to regulate fatty acid concentrations in plasma (18). Reduced DMI from supplemental dietary fat appears to be most pronounced in the early postpartum period (13) when the correlation between reproductive performance and energy balance is the closest (12). Reduced energy intake may minimize the possible positive effects of fat supplementation on reproductive efficiency.

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Cause and effect relationships between supplemental fat and cow health have not been demonstrated. Fats that do not interfere with ruminal fermentation may help prevent spontaneous ketosis and related diseases (16). Fat supplementation has reduced NEFA and ketone concentrations in blood in several trials (3).

Supplementation of commodity fats, such as whole oilseeds, free oils, and animal fats, is limited by potentially negative effects of unsaturated long-chain fatty acids on ruminal fermentation and secretion of milk fat (19). To prevent the milk fat depression associated with commodity fats, current recommendations are to limit these fats to 3 to 5% of dietary DM (19). Calcium salts of palm oil long-chain fatty acids (**Ca-LCFA**), prilled long-chain free fatty acids, and partially hydrogenated animal fats are relatively inert within the rumen, allowing these products to be fed at rates above commonly accepted limits for commodity fats (13, 25).

The objective of this trial was to evaluate the effects of Ca-LCFA fed to lactating cows already receiving a high percentage of supplemental fat (4.2% of dietary DM, on average) from commodity fats on milk production, reproductive efficiency, cow health, and body condition score (**BCS**) of cows in five high producing commercial Holstein herds.

MATERIALS AND METHODS

Data

Five commercial Holstein herds were selected based on a rolling herd average for milk production >9000 kg/yr per cow. All herds were milked three times daily. Each herd was scheduled to be tested every 14 d by DHI. Actual test intervals varied between 12 and 31 d; the mean interval was 15.6 d. Cows calving between July 1, 1989 and March 15, 1990 were placed on trial for the first 180 to 200 d of lactation (n = 443). Rolling herd averages and average production parameters for cows on trial are presented in Tables 1 and 2, respectively. All cows were housed in confinement; herds 1, 3, 4, and 5 were housed in tie stalls, and herd 2 was housed in free stalls.

Control diets were formulated by the nutrition consultant for the herd. Control diets varied throughout the trial and were a function of forage availability and quality, commodity availability and cost, and herd production. All herds were fed TMR. Herd 2 also was fed .9 kg/d of expeller soybean meal per cow in the parlor. Forage samples were collected every 14 d from each herd. Concentrations of DM, CP, ADF, NDF, P, Ca, K, and Mg were determined by wet chemistry analysis (Wisconsin DHI Cooperative Soil and Forage Center, Bonduel, WI). Forage TDN content was calculated from

TABLE 1. Initial (July 1989) and final (August 1990) rolling herd averages.

	Herd				
Parameter	1	2	3	4	5
Milk, kg/yr	11,010[1]	10,889	12,472	11,338	9297
	10,766[2]	11,457	12,768	12,763	9625
Milk fat, %	3.58	3.78	3.57	3.50	3.80
	3.58	3.53	3.54	3.40	3.56
Milk fat, kg/yr	394	412	445	397	353
	386	405	452	434	345
Milk protein, %	3.09	3.05	3.03	3.00	3.20
	3.09	2.95	3.00	2.96	3.04
Milk protein, kg/yr	340	332	378	340	298
	332	338	384	377	293
SCC, $\times 10^3$	150	200	170	170	310
	180	207	194	139	200

[1]Initial rolling herd average.

[2]Final rolling herd average.

ADF (21). The NE_L was calculated from TDN for all forages (17). Manufacturer's guaranteed analyses were used for feed additives, minerals, and vitamins. National Research Council (17) values were used to estimate nutrient contents of concentrate, protein, and fat sources, but not NE_L for Ca-LCFA (1). Amounts of RUP were calculated from feedstuff degradabilities of Satter (24) and NRC (17). Nonstructural carbohydrates were calculated by difference (100 – percentage of CP – percentage of NDF – percentage of ether extract – percentage of ash) (35). Total amounts of feed offered and orts were recorded daily for each herd. The DMI for treatment and control groups could not be determined independently because only whole herd estimates of DMI were obtained. Ingredient and nutrient compositions (DM basis) of the control TMR were calculated daily and averaged for the 7 d immediately prior to and including the day of each milk test (Tables 3 and 4). On average, control TMR contained 3.7 to 4.8% supplemental fat. Herds 2, 3, and 5 were fed Ca-LCFA in the control TMR. The TMR ingredient composition and nutrient profiles were relatively consistent throughout the trial, except those for herd 5.

TABLE 2. Mean production for all cows on trial (both control and treatment) during the first 200 DIM.

	Herd				
Parameter and parity	1	2	3	4	5
4% FCM, kg/d[1]					
Primiparous	29.3	32.5	32.4	32.1	26.6
Multiparous	38.3	40.2	45.4	42.3	35.2
Milk, kg/d					
Primiparous	32.5	34.2	35.1	34.4	28.4
Multiparous	41.3	43.1	49.6	46.9	38.5
Milk fat, %					
Primiparous	3.37	3.73	3.51	3.64	3.59
Multiparous	3.56	3.59	3.47	3.37	3.47
Milk fat, kg/d					
Primiparous	1.09	1.25	1.22	1.22	1.01
Multiparous	1.45	1.53	1.70	1.57	1.32
Milk protein, %					
Primiparous	3.02	2.85	2.97	2.88	2.98
Multiparous	3.02	2.87	2.89	2.85	2.93
Milk protein, kg/d					
Primiparous	.98	.97	1.04	.98	.84
Multiparous	1.24	1.23	1.42	1.33	1.12
SCC, linear score					
Primiparous	2.3	2.2	2.3	1.4	2.8
Multiparous	3.3	2.6	2.0	1.8	3.3
4% FCM Peak production, kg/d					
Primiparous	34.2	37.6	34.8	32.5	32.2
Multiparous	47.4	48.4	51.6	47.3	42.8
Days to 4% FCM peak					
Primiparous	57.4	68.8	89.6	145.0	76.2
Multiparous	60.6	55.6	57.8	83.5	48.9
4% FCM Cumulative production to 200 DIM, kg					
Primiparous	5893	6388	6544	6049	5504
Multiparous	7641	7891	8866	8445	7028
Cows, no.					
Primiparous	26	31	35	31	37
Multiparous	48	42	65	32	53

[1]4% FCM = .4 (milk) + 15 (milk fat).

TABLE 3. Control diet ingredient composition.

Ingredient and ration content	Herd 1	2	3	4	5
	(% of dietary DM)				
Alfalfa haylage					
Mean	39.6[1]	25.3	41.4	37.0	30.0
Minimum	29.7[2]	18.4	32.7	32.0	2.9
Maximum	50.4[3]	33.1	45.4	45.3	46.6
Alfalfa hay					
Mean	0	5.9	0	3.4	4.3
Minimum		3.6		0	0
Maximum		9.7		8.3	10.7
Corn silage					
Mean	5.5	6.6	0	4.0	9.3
Minimum	0	0		0	0
Maximum	10.6	14.4		10.0	21.1
Other forages					
Mean	0	4.9[1]	0	0	13.6[2]
Minimum		0			0
Maximum		15.5			51.3
High moisture corn, shelled or ear					
Mean	33.8	32.2[3]	30.0	30.6	23.5
Minimum	28.0	18.7	27.4	26.4	18.2
Maximum	39.2	38.5	35.0	34.9	34.9
Soybean meal					
Mean	3.4	4.1	5.1	2.9	2.5
Minimum	0	0	4.3	0	0
Maximum	9.6	8.1	6.9	5.1	11.0
Soybeans, raw and heat processed					
Mean	0	3.5	0	.8	3.5
Minimum		3.2		0	0
Maximum		4.0		4.7	10.2
Cottonseed					
Mean	10.4	8.3	10.0	12.0	1.5
Minimum	7.2	6.8	9.5	8.1	0
Maximum	13.0	14.1	11.3	14.2	3.7
Supplemental RUP[4]					
Mean	4.3	5.3	7.1	5.1	2.0
Minimum	3.2	3.8	5.8	4.5	0
Maximum	5.0	6.3	8.7	5.9	5.3
Minerals, vitamins, buffers, and feed additives					
Mean	1.1	2.0	3.5	2.1	7.8[5]
Minimum	.4	1.8	3.4	1.8	1.1
Maximum	2.4	2.7	4.0	2.4	21.2
Tallow					
Mean	1.9	.8	2.1	2.1	1.6
Minimum	1.2	0	2.0	1.8	0
Maximum	2.3	2.2	2.4	2.6	3.0
Ca-LCFA[6]					
Mean	0	1.1	.8	0	.4
Minimum		.9	.8		0
Maximum		1.7	1.0		1.1

[1]Oatlage.

[2]Mixture of sudan and alfalfa silage, barley silage, and beet pulp.

[3]Includes wet brewers grains (8.1, 6.3, and 9.5% for mean, minimum, and maximum percentage of diet DM, respectively).

[4]Meat and bone meal and blood meal (also linseed and fish meals in herd 3).

[5]Includes noncommodity fat and RUP sources, yeast, and isoplus (5.6, 0, and 19.2% for mean, minimum, and maximum percentage of dietary DM, respectively).

[6]The Ca salts of long-chain fatty acids.

TABLE 4. Control diet nutrient densities.

Nutrient and ration content	Herd 1	2	3	4	5
	(% of dietary DM)				
DM					
Mean	59.0	53.3	61.3	63.9	56.6
Minimum	54.1	47.8	56.1	54.2	48.5
Maximum	65.4	59.2	65.8	70.0	68.0
TDN					
Mean	75.0	72.7	72.2	76.0	71.8
Minimum	70.5	68.4	70.0	73.9	69.9
Maximum	80.2	76.0	76.5	77.4	74.4
NE_L, Mcal/kg					
Mean	1.75	1.70	1.70	1.78	1.68
Minimum	1.63	1.58	1.64	1.73	1.62
Maximum	1.88	1.79	1.80	1.82	1.77
CP					
Mean	18.3	19.7	19.1	18.7	17.6
Minimum	16.8	17.9	18.2	18.1	16.0
Maximum	20.1	21.3	19.9	20.1	18.8
RUP (calculated), % of CP					
Mean	35.3	40.9	38.3	37.9	37.0
Minimum	31.9	38.5	37.0	35.7	30.3
Maximum	38.9	43.1	43.7	41.4	41.6
NDF					
Mean	33.5	35.0	34.6	30.1	32.2
Minimum	29.6	30.2	28.4	28.0	26.8
Maximum	37.9	41.2	38.0	33.5	36.8
NDF from forage					
Mean	22.2	22.3	20.7	22.6	27.2
Minimum	17.3	18.4	16.9	19.5	21.6
Maximum	27.6	28.2	24.2	26.4	32.2
ADF					
Mean	23.6	22.3	23.6	20.3	21.9
Minimum	21.2	20.8	21.0	18.9	17.7
Maximum	25.5	25.8	26.4	22.5	25.1
Nonstructural carbohydrates (calculated)					
Mean	37.0	32.3	32.2	37.6	37.6
Minimum	30.3	27.6	28.5	33.7	29.8
Maximum	43.6	37.4	38.4	39.9	44.8
Added dietary fat					
Mean	3.9	3.8	4.8	4.6	3.7
Minimum	2.6	2.8	4.5	4.0	1.4
Maximum	4.9	5.9	5.4	5.4	6.4
Estimated DMI, kg/d					
Mean	22.0	23.3	26.3	22.9	21.7
Minimum	18.7	20.0	23.6	18.7	18.8
Maximum	26.3	25.5	28.2	27.1	27.0
Ca					
Mean	1.14	1.33	1.24	1.18	1.14
Minimum	1.00	1.15	1.18	1.10	.83
Maximum	1.43	1.55	1.40	1.28	1.58
P					
Mean	.60	.60	.65	.59	.59
Minimum	.49	.56	.61	.53	.46
Maximum	.70	.63	.72	.65	.77
Mg					
Mean	.36	.38	.39	.41	.32
Minimum	.28	.34	.37	.36	.23
Maximum	.43	.43	.42	.45	.39
K					
Mean	1.53	1.34	1.24	1.60	1.65
Minimum	1.16	1.01	1.18	1.26	1.39
Maximum	1.84	1.52	1.40	1.76	2.01

Within each herd, primiparous and multiparous cows were grouped separately into blocks of 4 cows based on calving date, and 2 cows were assigned at random to receive either the control or the treatment diet. Originally designed as a 2 × 2 factorial experiment to evaluate Ca-LCFA and bST, this trial was subsequently limited to evaluation of the effects of Ca-LCFA because of milk marketing issues related to the use of bST.

One-half of the cows within each herd (n = 220) were fed the control diet plus .45 kg/d per cow of Ca-LCFA (Megalac®; 82% fatty acids; Church and Dwight Co., Inc., Princeton, NJ) from calving through 180 to 200 DIM. For four of the five herds, Ca-LCFA was added directly to the mixer or mixed with the TMR after it was delivered to the cow, depending on the number of cows receiving the treatment diet. For herd 2, Ca-LCFA were fed in the parlor. Control cows received .9 kg/d of expeller soybean meal per cow, and treatment cows received 1.35 kg/d per cow of expeller soybean meal and Ca-LCFA mixed in a 2:1 (wt:wt) ratio. Based upon observation of the cows, consumption of Ca-LCFA was not a problem in any herd. The DMI was assumed to be consistent between control and treatment groups; .45 kg/d of Ca-LCFA increased dietary TDN by 1.0 to 1.8 percentage units and dietary NE_L by .05 to .07 Mcal/kg.

On average, all TMR met or exceeded minimum NRC (17) guidelines for CP, fiber, minerals, and vitamins. Although NDF exceeded suggested NRC (17) minima, amounts of NDF from forage were frequently <21% in herd 3. Herds 1, 2, and 4 also occasionally were fed TMR that were low in NDF from forage. Based on production (Table 2), energy intake was most limiting for milk production in all five herds, assuming that estimated DMI were accurate.

Herds were also visited every 14 d for collection of non-DHI data. The BCS [scale from 1 to 5, to the nearest .25, where 1 = emaciated and 5 = excessively conditioned (9)] and BW (estimated by tape measure; US Feeds, Eldora, IA) were measured for each cow on the first visit after freshening and every 28 d thereafter. Health disorders were recorded by herd managers and included mastitis, ketosis, displaced abomasum, off feed (unknown etiology), diarrhea, milk fever, respiratory disorders, foot problems (cracks, ulcers, or abscesses), foot rot, lameness (unknown etiology), and disease (no diagnosis). All diseases were recorded as present without indication of severity or duration. Multiple reports of a single disease within one lactation were treated as separate occurrences only if the occurrences were at least 14 d apart.

Rectal palpation of each herd was completed every 2 to 4 wk by the herd veterinarian. Reproductive disorders included retained placenta, uterine infection, abortion, and static (noncycling) and cystic ovaries. Other reproductive parameters recorded included estruses, breedings, and the use of prostaglandin. Intensity of estrus was subjectively described by herd managers as standing, weak, none (determined to be in estrus by rectal palpation or timed breeding without signs of estrus), or bled off without detection of estrus. Voluntary waiting periods before first breeding were determined by each herd manager and ranged from breeding at the first observed estrus (earliest was 38 DIM) to 90 DIM. Prostaglandin $F_{2\alpha}$ was used at the discretion of the herd manager to induce estrus either at the time of herd health examinations or 7 to 10 d after expected estrus in selected cows that did not demonstrate physical signs of estrus. Records for embryo donors and culled cows (22% of population) were not included in the analysis of reproductive indices.

Statistical Analysis

Observations on production data for treatment and control populations were not uniformly distributed across DIM. Observations on the treatment population were clustered around peak milk production (45 to 104 DIM). Independent of this clustering effect, treatment observations were, on average, .1 d later in early lactation and .4 d earlier following peak production than were control observations. For these reasons and to help fulfill the statistical assumptions inherent to general linear modeling procedures, analysis of 4% FCM, milk, and milk fat percentage (**MFP**) was completed using statistical techniques described by Scott et al. (28, 29). Raw data were initially fit to a nonlinear model (modified lactation curve). A unique nonlinear model was fitted to each parity (1, 2, and >2) within herd subclass. The

fitting of a unique nonlinear model to each parity within herd subclass (15 subclasses) accounted for herd, parity, herd by parity interactions, and DIM effects. The residuals from these nonlinear models were then used in conventional split-plot analysis. Fifteen degrees of freedom were removed from the whole plot error sum of squares to account for fitting 15 separate models to the data before analysis. However, fitting 15 separate nonlinear models to the data before split-plot analysis eliminated the need to include herd (4 df), parity (2 df), and herd by parity interactions (8 df) in the whole-plot portion of the split-plot model. Remaining production variables were not modeled in this manner because of the lack of nonlinear equations capable of modeling these production variables over DIM.

Residuals obtained from fitting lactation curves, or the raw data when curves were not fitted, were then modeled using the general linear models procedure of SAS (23). Split-plot model structure was utilized. Because numbers of observations were unequal within blocking variables, Type III sums of squares were utilized to determine significance (23) at $P < .10$ for whole-plot effects and at $P < .05$ for split-plot effects. Lactation curves for 4% FCM were also fitted separately for each individual cow with production records from ≥7 test dates (383 cows; 86% of cows on trial). Time to peak 4% FCM production, peak 4% FCM production, and cumulative 4% FCM production for the first 200 DIM were predicted from these curves.

General linear models were specified separately for 4% FCM, milk, MFP, and milk protein percentage (**MPP**). All models were determined by forward selection and backward elimination stepwise procedures. Interaction terms were limited to two-way interactions until shown to be significant, at which time all three-way interactions involving the two-way interaction were tested. No three-way interactions were significant. Models developed for primiparous cows were different from those developed for multiparous cows. No parity effects were detected in multiparous cows, so data for multiparous cows were combined for analysis. If an independent effect or interaction was significant in two of the four models solved, it was included in the final model.

General linear models developed were

$$\begin{aligned} y_{abcdjkl} = {} & cov_{abcd} + cm_a + f_b + h_c(f_b) \\ & + w_{abcd} + c_{abcd} + t_d + (cov_{abcd} \\ & \times t_d) + (c_{abcd} \times f_b) + id_{abcd} + d_j \\ & + (d_j \times h_c) + tm_k + (tm_k \times h_c) \\ & + (tm_k \times t_d) + (tm_k \times cm_a) \\ & + (tm_k \times w_{abcd}) + e_{abcdjkl} \end{aligned}$$

for primiparous cows and

$$\begin{aligned} y_{abcdejkl} = {} & cov_{abcde} + cm_a + f_b + h_c(f_b) \\ & w_{abcde} + c_{abcde} + t_d + (cov_{abcde} \\ & \times c_{abcde}) + (cov_{abcde} \times t_d) \\ & + (f_b \times t_d) + (c_{abcde} \times t_d) \\ & + (f_b \times cm_a) + id_{abcde} + d_j \\ & + (d_j \times h_c) + tm_k + l_e + (tm_k \\ & \times cov_{abcde}) + (tm_k \times h_c) + (tm_k \\ & \times cm_a) + (tm_k \times w_{abcde}) + (tm_k \\ & \times l_e) + e_{abcdejkl} \end{aligned}$$

for multiparous cows

where

$y_{abcd(e)jkl}$ = production measurement (4% FCM, milk, MFP, and MPP);
$cov_{abcd(e)}$ = genetic covariant for 4% FCM (.4 * predicted producing ability $(PPA)_{milk}$ + $15PPA_{fat}$),
cm_a = month of calving,
f_b = management and nutrition effect (**MNE**),
$h_c(f_b)$ = herd within MNE,
$w_{abcd(e)}$ = BW at calving,
$c_{abcd(e)}$ = BCS at calving,
t_d = treatment group,
l_e = lactation,
$id_{abcd(e)}$ = whole-plot error (cow),
d_j = DIM in groups of 10-d intervals (e.g., 5 to 14 DIM, 15 to 24 DIM, used only for production measurements that were not modeled with lactation curves),
tm_k = calendar month in which production was observed, and
$e_{abcd(e)jkl}$ = error.

Both month of calving and calendar month in which production was observed were classi-

fied by 2-mo intervals. For multiparous cows, herd within MNE was included in the model for all production variables not modeled with lactation curves. The PPA is an index combining the estimated genetic merit of an individual cow and the expected contribution of permanent environment to the phenotype of the individual cow (26). This index represents the best estimate of expected future production. For primiparous cows, PPA was calculated as the average genetic merit of both parents because no prior production records existed. The MNE blocked herds into two populations based on the magnitude of response to treatment. All effects were treated as fixed effects except whole-plot error and split-plot error, which were random effects. Both error terms were assumed to be normally distributed with constant variance and a mean of zero.

Month of calving, MNE, herd within MNE, treatment group, lactation, DIM, and calendar month in which production was observed are discrete or class variables (except DIM when lactation curves were used), and PPA, BW at calving, and BCS at calving are continuous variables. Both BW and BCS at calving exhibited linear relationships with production traits; only extreme values (BCS at calving ≤ 1.5 or ≥ 4.75) became nonlinear. Observations at these extremes were limited (n = 5), and models fitted with BCS at calving that were defined as a class or a continuous independent effect resulted in similar model coefficients of multiple determination with the same independent effects and interactions as in the final model. One reason for this relationship may have been that the majority of cows with BCS at calving ≥ 4 were in herd 4, which maintained a high rolling herd average (12,275 kg/yr per cow) throughout the trial.

When other production data were evaluated, the random addition of other main effects and interactions to the full model did not reveal additional significant terms.

Categorical data (health and most reproductive indices) were analyzed as a prospective cohort study using a categorical modeling procedure (23) for logit-linear regression. Logit analysis permits analysis of discrete dependent variables such that the predicted values of the model are probabilities of the occurrence of the class variables. These probabilities necessarily sum to 1. Because of the relatively small number of cows on trial, models included only treatment, the variable of interest, and the interaction of treatment with the variable of interest. Statistical comparison of incidence rates of most of the variables of interest between different herds and different lactations could not be completed because individual cell values were too low (<5 observations per cell); however, no consistent patterns were detected between indices for different herds. Continuous data on health and reproduction were analyzed using general linear models (23); models included treatment, herd, lactation, BCS at calving, PPA, season of calving, and all two-way interactions of treatment, PPA, and BCS at calving.

RESULTS AND DISCUSSION

Production Effects

Effects of Ca-LCFA on milk production of primiparous and multiparous cows are presented in Table 5. No interactions of treatment by herd were evident for primiparous cows, so estimates of treatment effects were weighted equally across all five herds. However, when data for multiparous cows were evaluated, herd 5 demonstrated considerably higher responses to treatment than the other four herds. Statistical models predicting 4% FCM, milk, MFP, and MPP for multiparous cows were determined separately for each herd. Each model contained the same significant independent effects and interactions as the model solved for all multiparous cows. Coefficients for each independent effect except treatment and slopes for each interaction were similar (within 5%) among the models generated for each herd. When data from the four low treatment response herds were pooled and MNE was used as a two-level blocking factor, estimates of treatment effects were within 1% of the estimated treatment responses predicted by independent models for each herd. Based on these results, all data from multiparous cows were pooled, and MNE was added to the model as a blocking factor.

As a group, multiparous cows in the low MNE herds did not respond to Ca-LCFA (.11 kg/d 4% FCM, −.09 kg/d of milk, and .028 kg/d of milk fat; data not provided); however, multiparous cows in the high response MNE

herd produced more 4% FCM, milk, and milk fat (2.88, 2.45, and .14 kg/d, respectively; data not provided) when fed Ca-LCFA. The MFP was unaffected by treatment. Cumulative 4% FCM production, as determined from individual lactation curves, showed a response to Ca-LCFA similar to individual test weights (3.46 kg/d of 4% FCM; data not provided) for multiparous cows in the high response herd. Estimates of treatment effects based on analysis of test day data only (2.88 kg/d of 4% FCM) should be more accurate because cumulative 4% FCM estimates were based on nonlinear extrapolation; however, the similarities in estimates between test day and projected cumulative 4% FCM production supported similar conclusions regarding the effects of Ca-LCFA. Peak 4% FCM production was delayed 17.6 d for cows fed Ca-LCFA. Delayed peak milk production has been associated with supplemental dietary fat in early lactation (13). Primiparous cows responded to Ca-LCFA by increasing production of 4% FCM (1.01 kg/d) and milk (1.50 kg/d) without affecting milk fat production and MFP.

These responses to supplemental fat are similar to those reported in a review by Shaver (30). Primiparous cows appear to respond less to supplemental fat than do multiparous cows. Addition of .45 kg/d of Ca-LCFA to diets

TABLE 5. Estimated treatment response for production parameters.

Parameter and parity	Estimated treatment response	SEE[1]	*P*
4% FCM, kg/d[2]			
Primiparous	1.01	.53	.06
Multiparous	1.50	.43	.001
Milk, kg/d			
Primiparous	1.50	.61	.02
Multiparous	1.18	.58	.05
Milk fat, %			
Primiparous	−.09	.07	.13
Multiparous	.06	.05	.22
Milk fat, kg/d			
Primiparous	.028	.022	.22
Multiparous	.078	.021	.001
Milk protein, %			
Primiparous	−.08	.02	.002
Multiparous	−.04	.02	.11
Milk protein, kg/d			
Primiparous	.021	.017	.24
Multiparous	.023	.018	.19
SCC, linear score			
Primiparous	−.08	.2	.72
Multiparous	−.30	.2	.16
4% FCM Peak production, kg/d			
Primiparous	1.3	1.3	.32
Multiparous	1.5	1.8	.42
Days to 4% FCM peak			
Primiparous	−9.1	10.7	.40
Multiparous	14.1	6.6	.04
4% FCM Cumulative production to 200 DIM, kg			
Primiparous	290.4	69.9	.09
Multiparous	339.2	141.6	.02

[1]Standard error of the estimate.

[2]4% FCM = .4 (milk) + 15 (milk fat).

containing no supplemental fat for the first 100 to 150 DIM to primiparous cows increased 4% FCM production only 25 to 59% of the increase observed for multiparous cows (10, 27). Addition of Ca-LCFA to diets already containing fat elicited variable responses. During early lactation, addition of .3 kg/d of Ca-LCFA to diets containing 2.3% fat from roasted soybeans or 2.7% fat from whole cottonseed had minor negative effects (–.7 kg/d) on 4% FCM (8, 14), although addition of .4 to .45 kg/d of Ca-LCFA to diets containing approximately 2.3% fat from whole cottonseed increased 4% FCM production by 1.6 to 4.6 kg/d (2, 7).

Although milk protein production was not significantly affected for primiparous cows, MPP declined (–.08%; $P < .01$). Negative effects of Ca-LCFA on MPP on multiparous cows were restricted to the high MNE herd (–.07%; $P < .1$; data not provided). Because the decline in MPP was associated with increased milk volume within these groups, decreased MPP likely represented a dilution effect rather than a negative effect of Ca-LCFA on milk protein synthesis. These results were similar to those reported (2, 7, 8, 14) in a number of trials examining the addition of Ca-LCFA to diets already containing supplemental fat.

Determination of the cause of MNE was impossible because only one herd (experimental unit) was in the high response MNE group. When only one experimental unit is included within a single classification of a predictor variable, the predictor may not represent a true cause and effect relationship (34). The TMR fat content, source, or both may have contributed to the increased response to treatment observed for the high response MNE herd. However, other factors, such as RUP content, forage feeding rates, rolling herd average, or management strategies may have contributed to the difference in treatment response.

In addition to MNE, several important linear interactions were detected that affected the magnitude of treatment response. Figure 1 stratifies the effects of these interactions on expected treatment response. Quadratic and cubic terms were tested in both models and were nonsignificant for both interactions. Interactions between PPA and treatment response were present in both primiparous and multiparous cows. Estimated treatment responses for multiparous cows increased .30 kg/d of 4% FCM per kg of PPA per d (Figure 1) and .015 kg/d of MF per kg of PPA per d (Figure 1). Coefficients for all other production parameters were not significantly different from 0; however, the coefficient for milk production was similar to that for 4% FCM. For primiparous cows, the only significant interactions between PPA and treatment response affected MFP (.12%/kg of PPA per d; data not provided) and days to peak production (20.8 d/kg of PPA per d; data not provided); however, milk production may also have been negatively correlated with PPA (–.84 kg/d of milk per kg PPA per d; $P < .11$; data not provided). In response to Ca-LCFA, multiparous cows with higher genetic merit appeared to allocate additional nutrients for the production of milk and milk fat than did multiparous cows with lower genetic merits. Results were similar when cows with different genetic merits were fed diets of variable energy densities (4). Cows with higher genetic merit demonstrated greater increases in milk production than did cows of lower genetic merit when fed the same diets.

An interaction between BCS at calving and treatment response was detected only for multiparous cows. Compared with cows calving with lower BCS, cows with higher BCS at calving produced less milk (–1.84 kg/d per unit of BCS; Figure 1) and more milk fat (.085 kg/d per unit of BCS; data not provided) when Ca-LCFA was added to the diet. Milk protein production declined as BCS at calving increased (–.056 kg/d per unit of BCS; data not provided) when Ca-LCFA was added to the diet. Thus, increased production of milk and milk protein were accompanied by decreased milk fat production when cows with lower BCS at calving were fed Ca-LCFA. This combination led to a nonsignificant interaction between treatment response and BCS at calving on 4% FCM production. Garnsworthy and Huggett (11) evaluated the effects of a fat-supplemented diet, containing 2.7% Ca-LCFA (DM basis) that was isoenergetic and isonitrogenous, compared with a control diet that contained no fat. The BCS at calving of thin and fat cows were 3.0 and 4.0, respectively. Thinner cows produced more milk than did fatter cows in response to supplemental fat. When cows received supplemental fat, milk fat production of fatter cows increased and of thinner cows was unaffected.

When these interactions were combined with MNE, the analysis of treatment response became more meaningful. Based on a milk price of $.265/kg, the costs of $.93/kg of Ca-LCFA and $.15/kg of TMR fed prior to the addition of Ca-LCFA, no change in DMI, and no labor cost associated with the Ca-LCFA fed, the needed production response to break even was 1.34 kg/d of 4% FCM per cow when .45 kg/d of Ca-LCFA per cow was fed [cost of .45 kg of Ca-LCFA ($.422) – cost of .45 kg of control diet ($.068) = 1.34 kg of milk ($.337)]. If DMI was reduced by Ca-LCFA, the break even milk production of 1.34 kg/d would decrease by .57 kg/d per kg of reduction in DMI. With no change in DMI, in the MNE

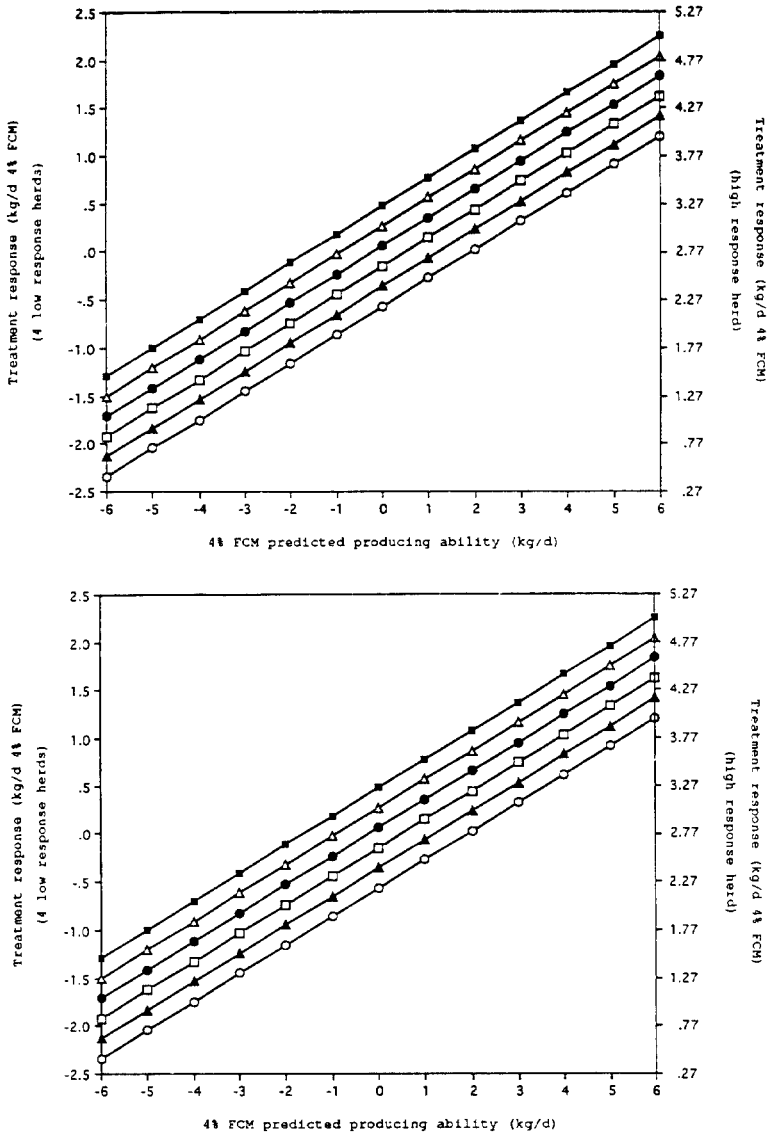

Figure 1. Estimated responses of 4% FCM (top) and milk (bottom) to supplemental fat by predicted producing ability, body condition score (BCS) at calving, and effect of management and nutrition for multiparous cows. BCS at calving 2.0 (■), 2.5 (△), 3.0 (●), 3.5 (□), 4.0 (▲), and 4.5 (○).

herds with low response, the addition of Ca-LCFA to the diet provided economical returns only from multiparous cows with very high PPA that were also thin when they calved. However, in the high response MNE herd, production responses were profitable for all except the fattest cows with extremely low PPA. The addition of Ca-LCFA to the diets of primiparous cows did not result in profitable production responses.

Health Effects

Lactation incidence rates measure the percentage of cows exhibiting clinical symptoms of a disease at any time within a single lactation. Odds ratios measure the increase (odds ratio >1) or decrease (odds ratio <1), relative to the control population, in lactation incidence rates for the treatment population. Because of the relatively few cows in each treatment group (n = 223 for control, and n = 220 for Ca-LCFA populations) and low lactation incidence rates for specific diseases, direct comparison of individual health and reproductive disorders must be interpreted cautiously, especially for those disorders with lactation incidence rates <10%. For example, the odds ratio for udder edema, 1.22, and displaced abomasum, 1.16, represent a difference of one and two occurrences of each disease, respectively, between the control and Ca-LCFA populations (Table 6). These limitations also make statistical interpretation difficult, providing little power to discriminate between population means. All comparisons, unless otherwise noted, were numeric only and were not statistically different.

The likelihood that an individual fed Ca-LCFA would have at least one clinical health disorder during lactation was .95 that of the

TABLE 6. Lactational incidence rates of health and reproductive disorders.

Health or reproductive disorder	Lactational incidence rate, %		Odds ratio	90% Confidence interval
	Control	Ca-LCFA[1]		
All health	65.9	62.7	.95	.85–1.07
Gastrointestinal	20.2	16.4	.81	.58–1.13
Reproductive	66.0	67.3	1.02	.91–1.14
Metabolic	20.2	15.4	.77	.54–1.08
Locomotion	15.7	14.6	.93	.64–1.34
Mastitis[2]	33.2	27.3	.82	.65–1.04
Diarrhea[2,3]	8.5	6.8	.80	.46–1.38
Retained placenta[2,4]	9.0	12.7	1.41	.90–2.24
Uterine infection[2,4]	21.1	25.5	1.21	.91–1.61
Abortions[2,4]	3.1	.9	.29	.08–1.07
Static ovaries[4]	30.5	24.6	.80	.62–1.04
Cystic ovaries[4]	30.9	31.8	1.03	.82–1.30
Milk fever[2,5]	8.5	5.0	.59	.32–1.07
Udder edema[2,5]	2.2	2.7	1.22	.46–3.25
Displaced abomasum[2,3,5]	6.3	7.3	1.16	.65–2.07
Feet problems[2,6]	14.4	13.6	.95	.64–1.40
Lameness (not feet)[2,6]	1.8	1.8	1.00	.32–3.21
Ketosis, off feed[2,3,5,7]	8.5	5.0	.59	.32–1.07
Respiratory disease[2]	1.8	1.8	1.00	.32–3.21
Undiagnosed health[2]	7.2	10.4	1.46	.87–2.43

[1]The Ca salts of long-chain fatty acids.

[2]All disorders so labeled were included in the summary group labeled all health.

[3]All disorders so labeled were included in the summary group labeled gastrointestinal.

[4]All disorders so labeled were included in the summary group labeled reproductive.

[5]All disorders so labeled were included in the summary group labeled metabolic.

[6]All disorders so labeled were included in the summary group labeled locomotion.

[7]Limited to primary ketosis (i.e., not secondary clinical sign of another disease).

control population. In general, cows fed Ca-LCFA had insignificantly lower lactation incidence rates for each of the health disorders observed. Notable exceptions to this pattern were retained placenta, uterine infection, and undiagnosed health problems. Retained placenta occurred at calving, and differences in lactation incidence rates of retained placenta resulted from random assignment to treatment group. Retained placenta also predisposed a cow to uterine infections (20). When cows with retained placenta were removed from the population, the odds ratio for uterine infections fell from 1.21 to 1.02. The odds ratios for grouped indices that included retained placenta and uterine infection (all health disorders and reproductive disorders) were unaffected by the deletion of records for cows that were positive for retained placenta.

Trends and magnitudes of odds ratios were similar when multiple occurrences of the same disorder were compared within an individual cow. Regression analysis revealed no differences between control and Ca-LCFA populations in DIM until the first occurrence of each disorder within a lactation.

No interaction was detected between lactation incidence rates and BCS at calving for the Ca-LCFA population; however, this interaction was present in the control population ($P < .10$). Cows with BCS at calving >3.4 (population mean) had a lactation incidence rate of 73.7% for all health disorders, but cows with BCS at calving <3.4 had a lactation incidence rate of 57.1% for all health disorders (data not provided). Most of this difference was due to differences in the lactation incidence rate for metabolic disorders in the control population (27.1 and 12.4% for BCS at calving of >3.4 and <3.4, respectively; $P < .05$; data not provided). This interaction suggests that, as BCS at calving increases, so does the likelihood of metabolic diseases.

Culling

Culling rates also suggest a possible benefit of Ca-LCFA once cows have adapted to high percentages of dietary fat. Culling decisions were at the discretion of individual producers, and no differences were detected for population culling rates (20.6 and 23.6% for control cows and cows fed Ca-LCFA, respectively; data not provided), culling rates within a lactation, or cause of culling between control and Ca-LCFA populations. However, differences were detected for the timing of culling between control and Ca-LCFA populations. One-half of the cows culled in the Ca-LCFA group were culled prior to peak lactation (70 DIM). Expressed as a percentage of the population culled prior to 70 DIM, 5.4 and 11.8% of the control and Ca-LCFA populations were culled ($P < .05$; data not provided). Differences in early culling practices between the control and Ca-LCFA populations may represent those cows that had difficulty adapting to the higher percentage of dietary fat. These numbers are similar to the lactation incidence ratio for undiagnosed health disorders for each population (7.2 and 10.4%, respectively). After 70 DIM, only 2.7% of the Ca-LCFA group was culled monthly compared with 3.5% of the control population ($P < .05$; data not provided).

Reproduction

Reproductive performances of control and Ca-LCFA populations were similar (Table 7). Reported effects of supplemental fat on reproductive performance have been variable. Reproductive performance changes associated with fat supplementation appear to be related to the magnitude of the milk response to fat supplementation. Milk production increases of <2 kg/d have been associated with increased conception rates (27, 32, 33), and milk production increases of ≥2 kg/d have been associated with no change in conception rates (10, 22). Our study appears to be an exception to this pattern, because milk production gains were <2 kg/d for all cows, except multiparous cows in one herd, without any associated improvement in reproductive performance. This difference in reproductive response to fat supplementation might have been related to the higher fat content in the control TMR fed in our study. No differences were detected in reproductive indices for multiparous cows between the low and high MNE populations.

Conception rates were similar at 150 DIM (65.9% vs. 65.6% for control and Ca-LCFA, respectively) and at 200 DIM (85.2% vs. 79.1% for control and Ca-LCFA, respectively). A larger proportion (19.2% vs. 8.6%) of the Ca-LCFA population was not detected in es-

trus prior to 200 DIM ($P < .05$). When conception rates at 200 DIM were expressed as a percentage of cows exhibiting estrus, 97.9% of the Ca-LCFA group had conceived compared with 93.5% of controls ($P < .05$; data not provided). Most cows not exhibiting estrus were primiparous; 10.5% for control cows vs. 26.9% for cows fed Ca-LCFA ($P < .01$; data not provided). Carroll et al. (5) noted that 21.7% (5 of 23) of cows fed 5% supplemental fat failed to show signs of estrus prior to 100 DIM; however, all control cows displayed signs of estrus prior to 100 DIM. Sklan et al. (33) found that cows fed Ca-LCFA at 2.6% of ration DM required increased time to commence ovarian activity; however, once cycling had commenced, more cows fed Ca-LCFA had normal cycle lengths than did control cows. Although mean estrus interval was similar for both groups in our study (31.3 d for control cows vs. 31.1 d for cows fed Ca-LCFA), more $PGF_{2\alpha}$ was used for the control population (55.7% vs. 43.7%; $P < .05$). On average, a cow in the control group received .98 injections of $PGF_{2\alpha}$, and a cow in the Ca-LCFA group received .76 injections of $PGF_{2\alpha}$. Estrus intensity was also subjectively rated as being stronger for the Ca-LCFA population (71.4% vs. 65.6% of estruses rated as standing; $P < .10$). Incidence of noncycling ovaries, as determined by rectal palpation, also tended to be lower for the Ca-LCFA population (30.5% for control and 24.6% for cows fed Ca-LCFA; Table 6). The population of cows fed Ca-LCFA apparently cycled more consistently than did the control population; however, the distribution of interestrus intervals was similar for both populations.

BCS and BW

No treatment effects were detected for BCS or BW changes (Table 8). Lack of treatment

TABLE 7. Comparison of reproductive indices between control cows and cows fed Ca salts of long-chain fatty acid (Ca-LCFA).

	Control	Ca-LCFA
Conception rate		
At 150 DIM,[1] %	65.9	65.6
At 200 DIM,[1] %	85.2	79.1
Days open		
Pregnant cows only	108.7	111.0
All cows[2]	137.9	145.8
Times bred		
Pregnant cows only	1.61	1.63
All cows[1]	1.74	1.71
Days to first AI	94.4	95.1
Conception rate		
Per AI, %	49.0	46.3
First AI, %	49.3	45.7
Second AI, %	39.2	41.6
DIM at last reported static ovaries[3]	44.0	45.1
Mean number of observed estruses, per cow	2.1	1.9
Mean number of $PGF_{2\alpha}$ injections, per cow	.98	.76
Mean estrus interval, d	31.3	31.1
Standing estruses, %	65.6	71.4†
Cows never detected in estrus, %	8.6	19.2*
Cows receiving $PGF_{2\alpha}$, %	55.7*	43.7

[1]Not including culled cows.

[2]Assuming open cows are open only 200 d, not including culled cows.

[3]Determined by rectal palpation and only includes cows reported as noncycling.

†$P < .10$.

*$P < .05$.

differences for these variables suggests that production gains for primiparous cows and multiparous cows in the high response herd were related to increased energy intake.

CONCLUSIONS

When Ca-LCFA were added to diets containing 3.7 to 4.8% supplemental fat (DM basis) and fed to cows in high producing herds, 4% FCM production increased 1.01 kg/d of 4% FCM per cow for primiparous cows, increased 2.88 kg/d per cow for multiparous cows in one herd, and was unaffected for multiparous cows in the remaining four herds. Because in only one herd did multiparous cows demonstrate a significant response to Ca-LCFA, cause and effect relationships between Ca-LCFA and the greater treatment response for this herd could not be determined. Differences in the fat content of the control diets for the various herds may have contributed to the differences in response to Ca-LCFA.

Two interactions affected the magnitude of treatment response: estimated treatment effects for production of 4% FCM and milk fat increased as the genetic potential of multiparous cows increased, but, for primiparous cows, increased genetic potential was associated with increased MFP and decreased milk production in response to Ca-LCFA. The BCS at calving also influenced responses to Ca-LCFA of multiparous cows. Cows with low BCS at calving increased milk production but decreased MFP when cows were fed Ca-LCFA. Conversely, cows with higher BCS at calving responded to

TABLE 8. Body condition score (BCS) and BW responses to Ca salts of long-chain fatty acids (Ca-LCFA).

Response and parity	Population mean	Response[1] to Ca-LCFA	SE
Minimum			
BCS[2]			
Primiparous	2.87	−.03	.05
Multiparous	2.69	.03	.05
BW, kg			
Primiparous	482.3	−4.3	5.0
Multiparous	545.9	9.4	7.4
Lost			
BCS			
Primiparous	.55	.03	.05
Multiparous	.72	−.03	.05
BW, kg			
Primiparous	57.3	4.3	5.0
Multiparous	61.4	−2.7	5.1
DIM			
At lowest BCS			
Primiparous	54.1	9.4	6.4
Multiparous	61.0	−1.3	6.3
At lowest BW			
Primiparous	71.8	15.0†	7.9
Multiparous	75.8	4.7	7.3
Rate of gain			
In BCS,[3]			
Primiparous	.09	.03	.02
Multiparous	.13	.00	.02
In BW,[3]			
Primiparous	9.3	.8	1.6
Multiparous	11.8	1.0	2.3

[1]Treatment mean minus control mean.

[2]Scale of 1 to 5.

[3]After minimum BCS or BW attained.

†$P < .10$.

Ca-LCFA by increasing MFP and decreasing milk production.

High dietary percentages of supplemental fat do not appear to affect adversely the health status of lactating dairy cows. Although the data suggest a positive effect of Ca-LCFA on health performance, this relationship could not be statistically confirmed. No differences were detected in reproductive performance between control and Ca-LCFA populations, except that supplemental fat resulted in fewer cows expressing visible signs of estrus. However, once estrus activity was detected, estrus expression was stronger when cows were fed Ca-LCFA. Supplemental fat also had no affect on BCS or BW, suggesting little change in energy balance from diets containing >5% supplemental fat (DM basis). To elucidate further the possible roles of dietary fats on health and reproduction, studies involving larger numbers of cows are required.

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