Plasma fatty acids, prostaglandin $F_{2\alpha}$ metabolite, and reproductive response in postpartum heifers fed rumen bypass fat[1,2]

S. J. Filley*,†, H. A. Turner†, and F. Stormshak*,[3]

*Department of Animal Sciences, Oregon State University, Corvallis 97331 and †Eastern Oregon
Agricultural Research Center, Oregon State University, Burns 97220

ABSTRACT: An experiment was conducted to determine whether feeding rumen-protected fatty acids (FA) to postpartum heifers would increase plasma concentrations of linoleic acid and $PGF_{2\alpha}$ metabolite (PGFM), shorten the interval from calving to first increase in plasma concentrations of progesterone (P_4), and increase pregnancy rate relative to controls. Hereford × Angus heifers (346 kg) were assigned randomly to treatments containing either lipid or barley supplemented diets for the first 30 d postpartum. Lipid was .23 $kg \cdot heifer^{-1} \cdot d^{-1}$ of calcium salts of FA (CSFA; n = 20), and an isocaloric amount of barley served as the control (n = 19). Supplements, with .23 kg of barley as a vehicle, and a basal diet of meadow and alfalfa hays were pen fed to heifers (5/pen). Heifers were bled on alternate days (d 1 to 30) and twice weekly (d 30 to 2 wk after first estrus) for RIA of plasma PGFM and P_4, respectively. Weight percentage of major FA in plasma on d 1 and 7 was determined with gas chromatography. First behavioral estrus was detected by use of intact bulls and confirmed by an increase in plasma P_4. On d 7, but not d 1, plasma from heifers fed CSFA had altered proportions of major FA ($P < .01$), including an increase in linoleic acid compared with those of controls (29.1 vs 25.6% of total FA; SE = .75; $P < .01$). Analysis of variance of contrast variables revealed an effect of treatment on direction of change in PGFM from d 3 to 5 ($P < .01$). By d 7 and on d 9, plasma concentrations of PGFM were greater in heifers fed CSFA than in controls ($P = .02$ and $P = .06$, respectively). There was no difference in plasma concentration of PGFM between treatments on d 1, 3, 5, 11, 13, and 15 postpartum ($P = .80, .17, .52, .82, .46$, and $.77$, respectively). Days to first estrus with ovulation, pregnancy rate, and calving interval were not affected by treatments ($P = .58, .52$, and $.24$, respectively). Although supplemental lipid fed to primiparous beef heifers increased plasma levels of linoleic acid and production of PGFM in the early postpartum period, it did not improve the fertility of these heifers in the subsequent breeding season.

Key Words: Prostaglandins, Fatty Acids, Postpartum Period, Beef Cows, Heifers

J. Anim. Sci. 2000. 78:139–144

Introduction

It is economically important for beef cattle producers to have a well-defined breeding season whereby each cow produces one calf per year. Because the postpartum interval to first estrus with ovulation is longer in primiparous than in multiparous cows, pregnancy rates in first-calf heifers may be improved by shortening this postpartum period (Wiltbank, 1970). Fertility resumes after uterine involution and repair and the ovulations that occur result in cycles of normal duration (Kiracofe, 1980). The hormone $PGF_{2\alpha}$ is important for uterine involution and ovarian function. The duration of increased $PGF_{2\alpha}$ production in the postpartum period is negatively correlated with the number of days to complete uterine involution and the interval between parturition and resumption of normal ovarian activity (Madej et al., 1984).

Nutritional approaches have been used to improve reproductive performance in cattle, and plasma lipid profiles, hormone concentrations, and follicular dynamics can be altered by including fat in livestock diets (Dunn et al., 1969; Schneider et al., 1988; Lucy et al., 1991). Linoleic acid, an EFA, is converted to arachidonic acid, the immediate precursor of prostaglandins, and infusion of lipid into ruminants (Lucy et al., 1990; Burke et al., 1996; Filley et al., 1997) and feeding EFA to nonruminants (Mathias and DuPont, 1979; Adam et al., 1982) increases plasma prostaglandins. It is not

---

[1] Technical paper No. 11460, Oregon Agric. Exp. Sta.

[2] The authors gratefully acknowledge Dave Thomas (Dept. of Statistics, Oregon St. Univ) for statistical assistance and Rosemary Wander (Dept. of Nutrition and Home Economics, Oregon St. Univ.) for use of the gas chromatograph.

[3] To whom correspondence should be addressed: 112 Withycombe Hall, Corvallis, OR 97331; phone: 541/737-2325; fax: 541/737-4174; E-mail: Fred.Stormshak@orst.edu.

Received December 22, 1998.

Accepted June 25, 1999.

known whether postpartum beef heifers fed lipids containing EFA have increased plasma concentrations of linoleic acid and $PGF_{2\alpha}$ and improved reproductive performance.

Thus, our objectives were to determine whether feeding rumen-protected fatty acids to postpartum heifers would increase plasma concentrations of linoleic acid and $PGF_{2\alpha}$, shorten the interval from calving to first estrus, and increase pregnancy rate. Calf characteristics that may affect $PGF_{2\alpha}$ production and heifer performance were also examined.

Materials and Methods

Animals, Treatments, and Samples

Hereford × Angus primiparous beef heifers (346 kg) were assigned to treatments randomly as they calved (February 14 to March 28). Basal diets contained native flood meadow hay (predominantly meadow and creeping foxtail) and trace mineral salts available for ad libitum consumption, with alfalfa hay (2.3 $kg·heifer^{-1}d^{-1}$) as a source of additional protein. Treatments consisted of either fat (n = 20) or control (n = 19) supplement fed from d 1 through 30 postpartum. Fat supplement, at approximately 3% of estimated dry matter intake, was .23 $kg·heifer^{-1}·d^{-1}$ of rumen inert fat as calcium salts of fatty acids (**CSFA**; donated by Church and Dwight, Princeton, NJ; Ca salts of palm oil; 44% palmitic [C16:0], 40% oleic [C18:1], 9.5% linoleic [C18:2], and 5% stearic [C18:0] acids) mixed with .23 kg of ground barley. Control supplement was an isocaloric amount of ground barley (.72 kg). Animals were fed in groups of five (eight pens; four replicate pens per treatment), and calves remained with their dams. Crude protein and total digestible nutrients were provided at 100% of the NRC nutrient requirements for 2-yr-old heifers nursing calves (NRC, 1984). Hay was fed twice daily, with alfalfa fed in the morning and meadow hay in the afternoon. Unconsumed meadow hay was removed every morning, and daily intake was calculated as kilograms of meadow hay consumed per kilogram of metabolic body weight ($BW^{.75}$). Experimental supplements were fed once daily at noon. Feed bunks were designed to accommodate up to 20 heifers, and each of the five heifers in the pen had adequate room to receive the supplement as an individual. Heifers were eager to consume the supplement, and care was taken that each heifer ate only her portion. Heifers were scored for body condition (**BCS**; scale 1 to 9; 1 = emaciated, 9 = obese) approximately 2 wk before calving, at 30 d postpartum, and at the end of the breeding season (approximately 150 d postpartum from the date that the first heifer calved). Heifers were weighed at postpartum d 1, 30, and 150. Calving ease was scored either as unassisted, light pull (hand pull with minimal effort), or hard pull (chains with heavy effort). Only heifers scored as unassisted or light pull were included in this experiment. Calf weight at d 1 and 150 postpartum (**CWT 1** and **CWT 2**, respectively) and calf sex were recorded. To assess plasma fatty acid profiles on d 1 and 7 and PGFM production for d 1 to 15, heifers were bled by venipuncture every other morning using heparinized vacutainer tubes. The blood was immediately placed on ice and centrifuged within 1 h at 2,500 × *g* for 15 min at 4°C. The plasma was stored at –20°C. After 30 d on the supplements, heifers were released to pastures where they continued to receive meadow and alfalfa hays. At approximately 60 d postpartum, heifers were moved to summer ranges to graze high desert grasses and forbes. Behavioral activity and plasma progesterone (P_4) concentration were used to determine the number of days to first estrus. Heifers were observed twice daily for sexual behavior in the presence of bulls from 30 to 150 d postpartum, and they were bled twice weekly for plasma P_4 concentrations beginning 30 d postpartum until 12 d after first behavioral estrus. First estrus with ovulation was defined as heifers standing for the bull with subsequent plasma concentrations of P_4 being greater than 1 ng/mL for two consecutive samples. Pre-estrus luteal activity was determined by evaluating plasma P_4 concentrations in samples taken during the 2 wk prior to first estrus.

Prostaglandin $F_{2\alpha}$ Metabolite Analysis

A subset of heifers (12 per treatment) was selected randomly for measurement of plasma prostaglandin $F_{2\alpha}$ metabolite (**PGFM**; 13,14-dihydro-15-keto-$PGF_{2\alpha}$). Concentration of PGFM in plasma samples (100 μL) from d 1 to 15 (every other day) was measured with a RIA described by Guilbault et al. (1984) and established in our laboratory by Burke et al. (1996). Intra- and interassay CV were 7.7 and 11.7%, respectively, and the sensitivity of the assay was 50 pg/mL. The PGFM antibody (# J-53 anti-PGFM) was a gift from William Thatcher (University of Florida), and PGFM standards and competitor ([5,6,8,9,11,12,14(n)-^{3}H]13,14-dihydro-15-keto-$PGF_{2\alpha}$; 177 Ci/mmol) were purchased from Cayman Chemical Company (Ann Arbor, MI) and Amersham (Arlington Heights, IL), respectively. Plasma without detectable amounts of PGFM for use in standards was harvested from a cow treated twice (16 h apart) with an intramuscular injection of a prostaglandin synthesis inhibitor: 20 mL of 50 mg/mL (1 g) flunixin meglumine (Banamine; Schering-Plough Animal Health Corp, Kenilworth, NJ). Blood was collected into evacuated heparinized flasks 4 h after the second injection, placed on ice, and centrifuged 2,500 × *g* for 15 min at 4°C. Plasma was removed and stored at –20°C.

Fatty Acid Analysis

Fatty acids (**FA**) were extracted from plasma for d 1 and 7 with methanol:chloroform (2:1 vol/vol) (Bligh and Dyer, 1959), methylated with boron trichloride and benzene, and then subjected to gas chromatography (Song and Wander, 1991) for identification. Samples selected for FA analysis were chosen in order to compare them with samples in which plasma concentrations of PGFM were found to be altered. Values for selected FA are reported as weight percentage of total (unfractionated) plasma FA.

Progesterone Analysis

Plasma P_4 was measured in duplicate (100 μL) for all heifers with the RIA described by Koligian and Stormshak (1976) using benzene:hexane (1:2 vol/vol) to extract P_4 from plasma samples taken before and during the subsequent breeding season. Recovered quantities of P_4 were corrected for a extraction efficiency of 58.5%. Intra- and interassay CV were 6.6 and 7.8%, respectively, and sensitivity of the assay was .05 ng/mL. The antibody (#337 anti-progesterone-11-BSA) was a gift from Gordon Niswender (Colorado State University). The P_4 tracer ([1,2,6,7-^{3}H] P_4; 12×10^3 dpm; 115 Ci/mmol) was purchased from New England Nuclear (Boston, MA), and P_4 standards were purchased from Cayman Chemical Company (Ann Arbor, MI).

Statistical Analysis

All data were analyzed using SAS (1994). Data on changes in body weights and body condition scores on postpartum d 1, 30, and 150, as well as feed intake, calf weights, and number of days to first estrus with ovulation were analyzed with one-way analyses of variance. Effect of treatments on PGFM concentration and FA profiles were analyzed with repeated measures ANOVA. Sources of variation in these latter analyses included treatment (T), day postpartum (D), and T × D, with animal within treatment as the error term for T, and residual as the error term for D and T × D. Chi-squared analysis was used on data for calving assistance (no assistance or light pull), calf sex, reproductive status (estrus or anestrus) at the end of the breeding season (approximately 150 d postpartum), and pregnancy rate during the subsequent fall (pregnant or nonpregnant). Simple correlation analysis was used to evaluate relationships between weight percentage of FA and plasma concentrations of PGFM.

Results

Plasma concentrations of PGFM decreased ($P < .01$) from d 1 to basal levels on d 15 postpartum reflecting the natural decline of PGFM during the postpartum period (Figure 1). Also, there was a significant treatment × day interaction ($P < .01$), which is attributed to the increase in plasma PGFM from d 3 to 5 postpartum ($P < .01$) and greater plasma concentrations of PGFM on d 7 and 9 postpartum ($P = .02$ and $P = .06$, respectively) for CSFA-fed heifers than for controls. There was no difference in plasma concentration of PGFM between treatments on postpartum d 1, 3, 5, 11, 13, and 15 ($P = .80$, .17, .52, .82, .46, and .77, respectively).

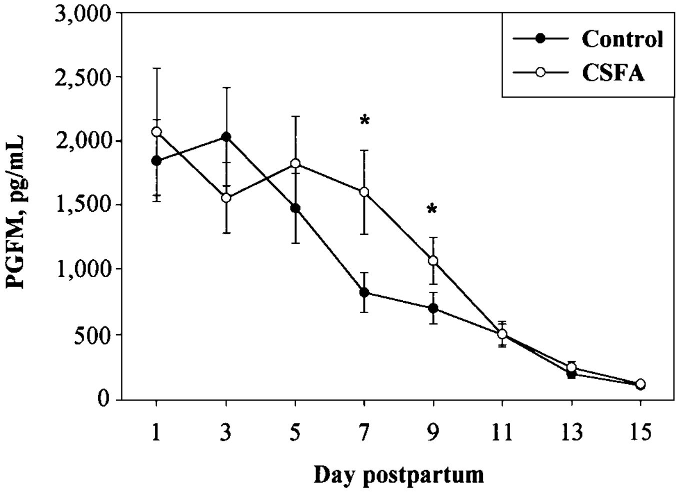

Figure 1. Mean (± SE) plasma concentration of prostaglandin $F_{2\alpha}$ metabolite (PGFM) on d 1 to 15 postpartum for CSFA (calcium salts of FA; n = 12) and control (barley; n = 12) supplemented heifers. *$P = .02$ on d 7 and *$P = .06$ on d 9 postpartum.

On postpartum d 1, the plasma FA profiles of heifers were not different between treatments. However, by d 7, proportions of major FA in plasma were different ($P < .01$) in heifers fed CSFA compared with that of controls, with some FA decreasing and some increasing (Table 1). Weight percentage of C18:2 on d 7 was correlated with plasma concentration of PGFM on d 5 (r = .46; $P = .02$) and d 9 (r = .39; $P = .06$), but not highly correlated with PGFM on d 7 (r = .31; $P = .14$).

Reproductive performance was not different between treatments ($P = .24$ to $P = .58$; Table 2). The experiment was terminated at the end of the breeding season, at which time heifers averaged 130 d postpartum, and 73.6% had exhibited estrus with no differences ($P = .99$) between treatments. Days to first estrus with ovulation,

Table 1. Means (± SE) for weight percentage of total plasma fatty acids for d 1 and 7 postpartum

Fatty acid	Day	CSFA[a]	Control[a]
Palmitic (C16:0)	1	15.7 ± .3	15.2 ± .3
	7	16.4 ± .6[b]	14.1 ± .6[c]
Stearic (C18:0)	1	13.1 ± .2	12.6 ± .2
	7	12.7 ± .4[b]	14.1 ± .4[c]
Oleic (C18:1)	1	14.1 ± .5	12.8 ± .5
	7	14.1 ± .7[b]	11.6 ± .7[c]
Linoleic (C18:2)	1	24.7 ± .7	25.0 ± .7
	7	29.1 ± .8[b]	25.6 ± .8[c]
Linolenic (C18:3)	1	10.8 ± .4	11.5 ± .4
	7	7.4 ± .4[b]	10.8 ± .4[c]
Arachidonic (C20:4)	1	2.6 ± .1	2.6 ± .1
	7	2.1 ± .1[b]	2.5 ± .1[c]

[a] CSFA (calcium salts of fatty acid; n = 20) and control (barley; n = 19).

[b,c] Means with different superscripts within the same row differ ($P < .01$).

Table 2. Mean calf weights, calving characteristics, and mean days to first estrus, pregnancy rate, and subsequent calving interval

Item	CSFA[a]	Control[a]	Common SE[b]
Calf wt on d 1 postpartum, kg	32.8[c]	35.0[d]	1.2
Calf wt on d 150 postpartum, kg	122.7	125.2	3.4
Calving assistance, %	30.0	15.8	ND
Calf sex, % male	55.0	52.9	ND
Days to first estrus	110.6	114.8	3.3
Pregnancy rate, %	72.2	68.4	ND
Calving interval, d	389.9	401.2	6.3

[a] CSFA, calcium salts of fatty acid (n = 20); control, barley (n = 19).

[b] Common estimate of the standard error; ND, not determined.

[c,d] Means with different superscripts within the same row differ (P = .06).

pregnancy rate, and calving interval were not affected by treatments (P = .58, .52, and .24, respectively), and 92% of the pregnancies were from first postpartum estrus with ovulation. Before first estrus with ovulation, 74% of the heifers exhibiting estrus had only transient (5.7 ± .2 d) increases in plasma P_4 (.9 ± .1 ng/mL), and the remaining heifers (26%) had no detected pre-estrus rise in plasma concentrations of P_4.

Heifer body weights (BW) and BCS were not different between treatments on postpartum d 1 and 30 (P = .63 to P = .85; Table 3). Kilograms of meadow hay consumed per day (as a percentage of metabolic body weight) and BW at 150 d were not different (P = .27 and P = .56, respectively) between treatments. However, BCS at 150 d was greater (P = .05) for heifers fed CSFA than for heifers fed control diets their first 30 d.

Calving ease and calf sex were not different (P = .12 and P = .90, respectively) between treatments (Table 2). Although heifers were assigned to treatment randomly, initial calf weight (CWT 1) was greater (P = .06) for heifers fed CSFA (n = 20) than controls (n = 19). However, when only the subset of heifers with PGFM measurements (n = 12/treatment) were included in the statistical analysis, there was no difference (P = .19) for CWT 1, and no data were adjusted for CWT 1. Even though this subset of heifers was chosen randomly, four of the 12 heifers fed CSFA had assistance in calving, and all 12 heifers from the control group calved unassisted. Using chi-squared analysis, no statistical difference between treatments could be found (quasi-complete separation) for the occurrence of calving assistance. The assistance given was recorded as easy hand pulls, and the biological significance of this event on PGFM concentration is uncertain. Plasma concentrations of PGFM were not different between heifers fed CSFA that calved assisted and unassisted (P = .30). There was no difference (P = .75) between treatments for CWT 2 (d 150).

Table 3. Mean body weights, body condition scores, and feed intake

Item[a]	CSFA[b]	Control[b]	Common SE[c]
Body wt on d 1, kg	347.5	344.9	4.5
Body wt on d 30, kg	328.7	335.3	4.4
Body wt on d 150, kg	341.9	333.1	4.6
Condition score, d 1 (1–9)	5.13	5.09	.52
Condition score, d 30 (1–9)	4.10	4.15	.49
Condition score, d 150 (1–9)	3.88[d]	3.65[e]	.41
Feed intake, % $BW^{.75}$	9.6	10.3	.88

[a] Body wt and condition score (scale 1 to 9) for various days postpartum. Feed intake was in kilograms of meadow hay consumed per day as a percentage of metabolic body wt.

[b] Calcium salts of fatty acid (CSFA; n = 20) and control (barley; n = 19).

[c] Common estimate of the standard error.

[d,e] Means with different superscripts within the same row differ (P = .05).

Discussion

Feeding CSFA to postpartum heifers resulted in a greater percentage of total plasma FA as linoleic acid and greater plasma concentrations of PGFM. Feeding rumen-protected FA changes plasma FA profile and increases systemic PGFM probably as a consequence of the increased systemic levels of linoleic acid. To our knowledge, this is the first study to report increased PGFM by feeding of lipid to ruminants. Moore et al. (1969) demonstrated that intraabomasal infusion of seed oils increased essential FA in plasma lipids of sheep, and Lucy et al. (1990) and Burke et al. (1996) reported increases in systemic concentrations of PGFM with intravenous infusion of lipid into ruminants. It has also been reported that in vitro treatment of bovine blastocysts, endometrium (Lewis et al., 1982), and ovarian tissue (Shemesh and Hansel, 1975) with arachidonic acid (an elongation product of linoleic acid) increases PGFM production by these tissues.

The greater plasma PGFM on d 7 and 9 for heifers fed CSFA is consistent with data demonstrating that consumption of essential FA by humans and rats (Mathias and Dupont, 1979) alters prostaglandin concentrations. In humans, plasma $PGF_{2\alpha}$ increases after 4 to 5 d of increased dietary linoleic acid (Adam et al., 1982). From data of our study, it is apparent that this lag time applies to ruminants as well; this is supported by the observed change in plasma PGFM concentration from d 3 to 5 of the trial for the heifers fed CSFA. The decrease in plasma PGFM over postpartum d 1 through 15 is consistent with published data and reflects the natural decline in $PGF_{2\alpha}$ during this period (Madej et al., 1984).

As a percentage of the total FA, greater d-7 plasma palmitic (C16:0) and oleic (C18:1) acids in heifers fed CSFA compared with controls is not surprising given that CSFA is 44% C16:0 and 40% C18:1. However, although CSFA is only 9.5% linoleic acid (C18:2), a 17.8% increase in plasma linoleate from d 1 to 7 is remarkable when compared with a 4.4% increase for C16:0. Ruminants conserve essential FA for essential functions (Moore et al., 1969), and increased plasma retention may be a part of that process. Lower plasma percentages of stearic (C18:0) and linolenic acid (C18:3) in lipid-fed heifers were not surprising due to the low and negligible levels of these FA in CSFA. In sheep, abomasal infusion of linoleic acid increased plasma C18:2 and decreased concentrations of C16:0 and C18:0 (Moore et al., 1969). From plasma FA profiles determined here, it can be seen that some changes do not directly reflect FA profile of the diet. As exemplified above by the increase in plasma C18:2, essential FA are conserved in ruminants, whereas other FA tend to be in equilibrium with tissue FA (Moore et al., 1969). The latter is evident in plasma C18:1, because the high content in CSFA did not lead to a large increase in plasma C18:1. Proper fluidity of membranes is maintained by controlling lipid content, with melting point of FA increasing with increasing chain length and degree of saturation (Singer and Nicolson, 1972). Although both C16:0 and C18:1 were major FA in the lipid supplement, the disproportionate change in plasma levels of these FA may have been due to adjustments for maintaining plasma membrane integrity and other cellular functions. Lower plasma arachidonic acid (C20:4) was unexpected for CSFA-fed heifers because, in nonruminants, supplemental C18:2 induces increased C20:4 in plasma phospholipids (Sanders and Younger, 1981). More information should be gathered by examining changes in specific lipid fractions (e.g., phospholipid or cholesterol esters of plasma in ruminants fed lipids).

Although plasma PGFM for CSFA-fed heifers was significantly greater than for controls, reproductive performance was not improved. Duration of elevated PGFM is negatively correlated with days to complete uterine involution and days to first estrus (Lindell et al., 1982; Madej et al., 1984); however, many other factors also influence the interval from calving to resumption of estrus (Malven, 1984). Uterine involution was not determined in this experiment, because manipulation of the uterus shortens involution time, increases plasma PGFM (Tolleson and Randel, 1987), and could have led to confounding of dietary influence on PGFM concentrations. Overall mean days to first estrus (112.7 ± 8.7 d) was greater than published observations (60 to 80 d) for primiparous beef heifers (Kiracofe, 1980; Randel, 1990), and uterine environment is not considered to influence fertility after the early postpartum period unless infection or trauma to the uterus is present (Kiracofe, 1980). Because of the extended anestrous period, it is concluded that, in this instance, return to estrus was not dependent on or influenced by early postpartum uterine environmental conditions imposed by the preceding pregnancy.

Preceding estrous cycles of normal length, the first postpartum estrus is frequently associated with a cycle of short duration, generally 8 to 10 d in length, with P_4 $\geq$ 1 ng/mL (Odde et al., 1980; Peters and Lamming, 1984; Wright et al., 1988). An improperly steroid-conditioned uterus is thought to be the cause of premature release of $PGF_{2\alpha}$ and an early demise of the corpus luteum. Preexposure of the uterus to P_4 improves fertility (Lishman and Inskeep, 1991); however, short cycles are not a prerequisite for fertility (Peters and Lamming, 1984). Indeed, no short cycles were observed in the heifers of our study. It is theoretically possible that the uterus had ample time to recover through long-term low ovarian or adrenal gland steroid production during the extended postpartum anestrous period. Feeding of CSFA in this experiment may have been too soon postpartum to positively affect ovarian follicular dynamics as reported for the feeding of CSFA to postpartum dairy cows (Lucy et al., 1991). A longer period or altered delivery scheme of lipid supplementation may be required to maximally affect reproduction, particularly with respect to ovarian function.

Reproductive response to lipid supplement has been shown to be dependent on body condition score (BCS) (Ryan et al., 1994). Although heifers in this study were at an acceptable BCS at calving, their body weight was less than optimal, which can also delay the return to estrus (Wiltbank et al., 1964). Heifers fed CSFA did perform better than controls with respect to terminal heifer BCS and calf weight gain. This may have been due to positive effects of CSFA feeding, such as increased milk yield and increased efficiency of energy utilization with respect to milk fat synthesis (Schneider et al., 1988). For CSFA-treated and control heifers, BW did not decrease in a manner similar to the decrease in BCS during the last 120 postpartum days (d 30 to 150), possibly due to heifers using body reserves (condition) for lactation while continuing to grow (increasing skeletal frame) without increases in BW through ample nutrient intake.

Sex of calf has been found to influence plasma concentrations of PGFM in postpartum cows (Lammoglia et al., 1995). Also, severe dystocia causes trauma to the uterus, requires repair to the uterine lining, and delays return to a normal uterine environment (Kiracofe, 1980). Although these factors may influence $PGF_{2\alpha}$ secretion during the postpartum period, neither percentage of calves born male nor calving assistance as recorded in this experiment were different between treatments.

Implications

Although supplemental lipid fed to primiparous beef heifers can alter plasma fatty acid profiles to favor increased linoleic acid and increased plasma prostaglandin $F_{2\alpha}$ metabolite in the early postpartum period, it does not seem to improve the fertility of the heifers in the subsequent breeding season. Continued studies in nutrition and reproduction using specific nutrients to affect specific hormones and initiation factors are warranted.

Literature Cited

Adam, O., G. Wolfram, and M. Zollner. 1982. Prostaglandin formation in man during intake of different amounts of linoleic acid in formula diets. Ann. Nutr. Metab. 26:315–323.

Bligh, E. G., and W. J. Dyer. 1959. A rapid method of total lipid extraction and purification. Can. J. Biochem. Physiol. 37:911–917.

Burke, J. M., D. J. Carroll, K. E. Rowe, W. W. Thatcher, and F. Stormshak. 1996. Intravascular infusion of lipid into ewes stimulates production of progesterone and prostaglandin. Biol. Reprod. 55:169–175.

Dunn, T. G., J. E. Ingalls, D. R. Zimmerman, and J. N. Wiltbank. 1969. Reproductive performance of 2-year-old Hereford and Angus heifers as influenced by pre-and post-calving energy intake. J. Anim. Sci. 29:719–726.

Filley, S. J., H. A. Turner, and F. Stormshak. 1997. Prostaglandin concentrations in lipid-infused postpartum heifers. Biol. Reprod. 56(Suppl. 1):207 (Abstr.).

Guilbault, L. A., W. W. Thatcher, M. Drost, and S. M. Hopkins. 1984. Source of F series prostaglandins during the early postpartum period in cattle. Biol. Reprod. 31:879–887.

Kiracofe, G. H. 1980. Uterine involution: Its role in regulating postpartum intervals. J. Anim. Sci. 51(Suppl. 2):16–28.

Koligian, K. B., and F. Stormshak. 1976. Progesterone synthesis by ovine fetal cotyledons in vitro. J. Anim. Sci. 42:439–443.

Lammoglia, M. A., J. W. Holloway, A. W. Lewis, D. A. Neuendorff, and R. D. Randel. 1995. Influence of maternal and service-sire breed on serum progesterone and estrogen before calving and plasma 13,14-dihydro-15-keto-prostaglandin $F_{2\alpha}$ after calving. J. Anim. Sci. 73:1167–1173.

Lewis, G. S., W. W. Thatcher, F. W. Bazer, and J. S. Curl. 1982. Metabolism of arachidonic acid *in vitro* by bovine blastocysts and endometrium. Biol. Reprod. 27:431–439.

Lindell, J-O., H. Kindahl, L. Jansson, and L. Edqvist. 1982. Postpartum release of prostaglandin $F_{2\alpha}$ and uterine involution in the cow. Theriogenology 17:237–245.

Lishman, A. W., and E. K. Inskeep. 1991. Deficiencies in luteal function during re-initiation of cyclic breeding activity in beef cows and in ewes. S. Afr. J. Anim. Sci. 21:59–75.

Lucy, M. C., T. S. Gross, and W. W. Thatcher. 1990. Effects of intravenous infusion of a soybean oil emulsion on plasma concentration of 15-keto-13,14-dihydro-prostaglandin $F_{2\alpha}$ and ovarian function in cycling Holstein heifers. In: Livestock Reproduction in Latin America. pp 119–132. Int. Atomic Energy Agency, Vienna, Austria.

Lucy, M. C., C. R. Staples, F. M. Michel, W. W. Thatcher, and D. J. Bolt. 1991. Effect of feeding calcium soaps to early postpartum dairy cows on plasma prostaglandin $F_{2\alpha}$, luteinizing hormone, and follicular growth. J. Dairy Sci. 74:483–489.

Madej, A., H. Kindahl, W. Woyno, L. E. Edqvist, and R. Stupnicki. 1984. Blood levels of 15-keto-13,14-dihydro-prostaglandin $F_{2\alpha}$ during the postpartum period in primiparous cows. Theriogenology 21:279–287.

Malven, P. V. 1984. Pathophysiology of the puerperium: Definition of the problem. Proc. 10th Int. Congr. Anim. Reprod. Artif. Insem. IV:III-1.

Mathias, M. M., and J. Dupont. 1979. The relationship of dietary fats to prostaglandin biosynthesis. Lipids 14:247–252.

Moore, J. H., R. C. Noble, and W. Steele. 1969. The incorporation of linolenic and linoleic acids into plasma lipids of sheep given intra-abomasal infusions of linseed oil, maize oil or linoleic acid. Br. J. Nutr. 23:141–152.

NRC. 1984. Nutrient Requirements of Beef Cattle (6th Ed.). National Academy Press, Washington, DC.

Odde, K. G., H. S. Ward, G. H. Kiracofe, R. M. McKee, and R. J. Kittok. 1980. Short estrous cycles and associated serum progesterone levels in beef cows. Theriogenology 14:105–112.

Peters, A. R., and G. E. Lamming. 1984. Reproductive activity of the cow in the post-partum period. II. Endocrine patterns and induction of ovulation. Br. Vet. J. 140:269–279.

Randel, R. D. 1990. Nutrition and postpartum rebreeding in cattle. J. Anim. Sci. 68:853–862.

Ryan, D. P., R. A. Spoon, M. K. Griffith, and G. L. Williams. 1994. Ovarian follicular recruitment, granulosa cell steroidogenic potential and growth hormone/insulin-like growth factor-I relationships in suckled beef cows consuming high lipid diets: Effects of graded differences in body condition maintained during the puerperium. Domest. Anim. Endocrinol. 11:161–174.

Sanders, T.A.B., and K. Younger. 1981. The effect of dietary supplements of $\omega 3$ polyunsaturated fatty acids on the fatty acid composition of platelets and plasma choline phosphoglycerides. Br. J. Nutr. 45:613–616.

SAS. 1994. Version 6.10. SAS Inst. Inc., Cary, NC.

Schneider, P., D. Sklan, W. Chalupa, and D. S. Kronfeld. 1988. Feeding calcium salts of fatty acids to lactating cows. J. Dairy Sci. 71:2143–2150.

Shemesh, M., and W. Hansel. 1975. Stimulation of prostaglandin synthesis in bovine ovarian tissues by arachidonic acid and luteinizing hormone. Biol. Reprod. 13:448–452.

Singer, S. J., and G. L. Nicolson. 1972. The fluid mosaic model of the structure of cell membranes. Science 175:720–731.

Song, J., and R. C. Wander. 1991. Effects of dietary selenium and fish oil (MaxEPA) on arachidonic acid metabolism and hemostatic function in rats. J. Nutr. 121:284–292.

Tolleson, D. R., and R. D. Randel. 1987. Physical manipulation of the postpartum bovine uterus and the subsequent release of prostaglandin $F2\alpha$. J. Anim. Sci. 65(Suppl. 1):414 (Abstr).

Wiltbank, J. N. 1970. Research needs in beef cattle reproduction. J. Anim. Sci. 31:755–762.

Wiltbank, J. N., W. W. Rowden, J. E. Ingalls, and D. R. Zimmerman. 1964. Influence of post-partum energy level on reproductive performance of Hereford cows restricted in energy intake prior to calving. J. Anim. Sci. 23:1049–1053.

Wright, J. M., G. H. Kiracofe, and K. B. Beeman. 1988. Factors associated with shortened estrous cycles after abortion in beef heifers. J. Anim. Sci. 66:3185–3189.