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# Feeding Fatty Acids for Fertility?

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## Introduction

Just as amino acids are the individual units making up the class of nutrients called proteins, so are fatty acids the major individual units of measure of what is broadly called lipids. Just as each amino acid has a distinct structure and function in protein building, so each fatty acid has a distinct structure and possibly function in metabolism.

The essentiality of certain amino acids and certain fatty acids was established for growing rats at about the same time. W.C. Rose at the University of Illinois in the early 1930's identified 10 essential dietary amino acids for rats. Soon thereafter, these same essential dietary amino acids were confirmed experimentally in growing nonruminant livestock. The fatty acids, linoleic acid (**C18:2**) and linolenic acid (**C18:3**), were identified in 1929 and 1930 as essential fatty acids (**EFA**) for growing rats fed nearly fat-free diets (Burr and Burr). However these results did not change the way livestock were fed. In 1936, Morrison stated "Whether or not farm animals need these fatty acids is still an open question. In any event, the usual rations fed stock in all probability provide sufficient amounts of any such essential nutrient substances." In 1954, Lambert et al. reported that the preweaned baby calf required the same two essential fatty acids in their diet. Calves fed a fat-free diet developed deficiency symptoms that included retarded growth, scaly dandruff, long dry hair, excessive loss of hair, and diarrhea. These symptoms are similar to those reported for rats fed diets largely devoid of the EFA. Prolonged feeding of such diets resulted in death of the rats.

## EFA Requirement Undefined

Although the need for these specific fatty acids by livestock continues to be supported by modern nutritionists, the quantity of each fatty acid required has not been defined and appears to be of little concern. "If farm animals actually have a dietary need for EFA, it seems probable that they are adequately supplied by the commonly fed rations" (Maynard and Loosli, 1969).

As with other essential nutrients, C18:2 and C18:3 cannot be synthesized in sufficient quantity to supply the animal's requirement. The enzymes necessary to synthesize the EFA from nonessential fatty acids are present only in plants (Groff et al., 1995). Because $\Delta$12 and $\Delta$15 desaturases are absent in cows and apparently in ruminal microorganisms, the C18:1 that is found in many feedstuffs cannot be

desaturated to C18:2 or C18:3 within the animal. Therefore these two polyunsaturated fatty acids must be supplied in the diet.

Two other long chain, polyunsaturated fatty acids may have an influence on animal performance. These are fatty acids found in marine products such as algae, fish meal, fish oil, and some seafood byproducts. The two fatty acids of greatest interest are eicosapentaenoic acid (**EPA**, **C20:5**) and docosahexaenoic acid (**DHA**, **C22:6**). These fatty acids are appearing more regularly in dairy cow diets due to an increased interest in feeding fish meal as a ruminally undegradable protein source.

## Effects of Fatty Acids on Reproductive Tissues and Performance

Although the role of the EFA has been documented as a key nutrient in maintaining healthy skin and hair and good growth rates, reproductive performance has also been affected when EFA were deficient, apparently apart from the general poor health of the animal. In the early work of Burr and Burr (1930), rats were fed a fat-free diet resulting in cessation of growth and, in a majority of rats, cessation of or irregular ovulation. Rats were then supplemented with either corn oil, olive oil, linseed oil, or coconut oil at approximately 1% of dietary DM. With the exception of coconut oil, consumption of the other oils resulted in a quick resumption of ovulation (within 5 d), even before growth had hardly begun. Coconut oil contained no linoleic acid. The other oils contain between 41% (corn oil) and 7% (olive oil) linoleic acid. Their further work documented that the cessation of growth and scaly skin condition caused by feeding the fat-free diet was reversed dramatically by supplementing with linoleic acid. Stearic (**C18:0**) and oleic acid (**C18:1**) were considered ineffective.

Although current wisdom in the dairy industry is that the intakes of the EFA are sufficient for meeting the lactating cow's requirements, the supplementing of some sources of fat to lactating dairy cows has improved reproductive performance. In several studies, lactating cows fed a basal diet containing whole cottonseed (~10% linoleic acid) and further supplemented with Megalac® (calcium salt of palm oil; Church and Dwight, Princeton, NJ) (~8% linoleic acid) experienced a better conception rate than cows fed the basal diet alone (Staples et al., 1998). Lactating cows fed tallow (4.3% linoleic acid) at 3% of dietary DM tended to have a better conception rate by 98 days in milk than cows not fed tallow (Son et al., 1996). Supplementing diets of lactating dairy cows with fish meal also has improved conception rates (Staples et al., 1998). In some of these studies (Armstrong et al., 1990; Bruckental et al., 1989; Carroll et al., 1994), fish meal partially replaced soybean meal resulting in a reduction of an excessive intake of ruminally degradable protein. Therefore the improved conception rates may have been due to the elimination of the negative effect of excessive intake of ruminally degradable protein on conception. However in a field study in which the concentration of ruminally undegradable protein was kept constant between dietary treatments, cows fed fish meal had a better conception rate (Burke et al., 1996) suggesting that the positive response was due to something other than a reduction in intake of ruminally degradable protein. The unique polyunsaturated fatty acids in fish (EPA and DHA) may have been responsible for the improvement in fertility.

The physiological basis by which linoleic acid, EPA, and DHA may improve reproductive performance may lie with their influence on the conversion of arachidonic acid (**C20:4**) to prostaglandin $F_{2\alpha}$ (**$PGF_{2a}$**). A quick review of the role of $PGF_{2a}$ during the postpartum period of resumption and reoccurrence of estrous cycles is in order here. Within the reproductive tract of cows, uterine tissue is a primary source of the F series prostaglandins (e.g., $PGF_{2a}$) during the early postpartum period. Concentration of 13, 14-dihydro-15-keto-$PGF_{2a}$ metabolite (**PGFM**) in plasma rose dramatically to a peak of ~2200 pg/ml by 1 d postpartum (Mattos, 2001). This rise is associated with regression of the corpus luteum (**CL**) of pregnancy and postpartum regression of the uterus. (The PGFM is produced as the uterus and lung metabolize $PGF_{2a}$.) Over the next 2 wk, PGFM gradually returned to baseline concentrations. The uterus then releases $PGF_{2a}$ regularly over the following weeks to regress each newly formed CL in order to initiate a new estrous cycle if the cow is not pregnant. If the cow does conceive, $PGF_{2a}$ release from the uterus is inhibited in order to preserve the CL on the ovary and maintain pregnancy. Because $PGF_{2a}$ has an effect on the regression of the CL, concentrations of plasma progesterone are related inversely to $PGF_{2a}$ concentrations during the period of CL regression in late diestrus. However, progesterone priming of the uterus is essential for induction of uterine lipids in order to subsequently synthesize $PGF_{2a}$.

In summary, prostaglandins play an important role in reestablishing estrous cycles both immediately after parturition and thereafter until conception occurs. Upon conception, $PGF_{2a}$ must be prevented from regressing the CL in order to maintain pregnancy (e.g., prevent early embryonic death).

## Eicosapentaenoic and Docosahexaenoic Acids

The feeding of EPA may aid in the suppression of synthesis of $PGF_{2a}$ by the uterus by competing for the key enzyme, prostaglandin endoperoxide synthase (**PGHS**), required for the conversion of arachidonic acid to $PGF_{2a}$. Although DHA is not a substrate for PGHS, it is a strong inhibitor of PGHS activity. Therefore when intake of EPA and DHA increases, conversion of arachidonic acid to $PGF_{2a}$ can be reduced, thus increasing the chances of preserving the life of the newly formed embryo. In addition, the increased presence of EPA and DHA can inhibit the synthesis of arachidonic acid from linoleic acid by inhibiting the desaturation and elongation enzymes required for that conversion. Linolenic acid (C18:3), another omega 3 fatty acid, also can compete with linoleic acid for the desaturase enzymes so that more EPA and less arachidonic are synthesized.

Which fatty acids are the most potent when it comes to suppression of synthesis of $PGF_{2a}$? A series of in vitro experiments was performed at the University of Florida (Mattos, 2001) using bovine endometrial (**BEND**) cells from the uterus. The BEND cells were incubated with no fatty acid (control) and a variety of fatty acids that included oleic acid (OA), linoleic acid (LA), conjugated linoleic acid (CLA), linolenic acid (LNA), arachidonic acid (AA), EPA, and DHA at a concentration of 100 uM. Compared to the

control, cells incubated with AA tended to stimulate synthesis of $PGF_{2a}$. This positive response was expected since AA is the fatty acid precursor of $PGF_{2a}$. Only the omega three fatty acids (LNA, EPA, and DHA) suppressed synthesis of $PGF_{2a}$ with EPA and DHA the most repressive (Figure 1). The fact that linoleic acid did not affect secretion of $PGF_{2\alpha}$ somewhat contradicts previous reports indicating inhibitory activity of this fatty acid (Elattar and Lin, 1989; Pace-Asciak and Wolfe, 1968). Moreover, linoleic acid has been considered a potential mediator of reduced endometrial $PGF_{2\alpha}$ secretion in the pregnant cow (Thatcher et al., 1995). Conversely, because linoleic acid is the most abundant precursor for synthesis of arachidonic acid and $PGF_{2\alpha}$, it could be hypothesized that linoleic acid would increase secretion of $PGF_{2\alpha}$ through increased precursor availability. This did not occur in the BEND cell system. One possible reason could involve lack of an efficient system for conversion of linoleic acid to arachidonic acid, which involves two steps of desaturation and one step of elongation.

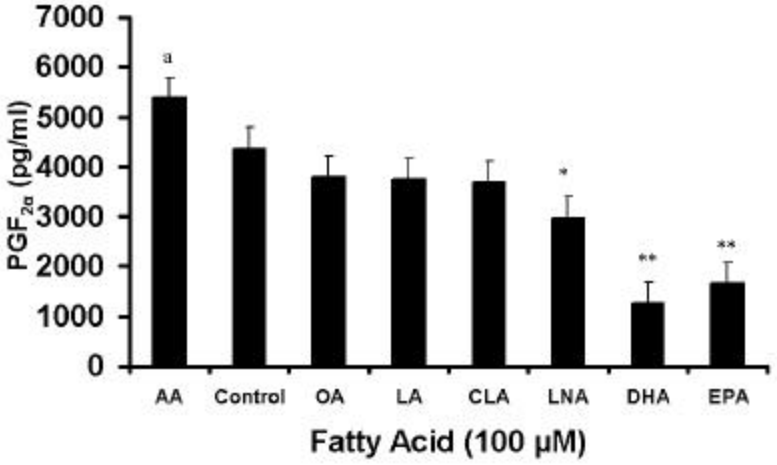


Figure 1. Effect of a variety of fatty acids on synthesis of $PGF_{2a}$ by bovine endometrial cells. Differences between each fatty acid and control: $^{a}P < 0.10$; *P < 0.05; **P < 0.01.

In a second study, BEND cells were coincubated with arachidonic acid and EPA for 24 h. Figure 2 illustrates the competing effects of the two fatty acids. Arachidonic acid increased ($P < 0.01$) secretion of $PGF_{2a}$ whereas EPA was inhibitory ($P < 0.01$). This illustrates the competition of precursors for processing by the PGHS enzymes involved in prostanoid synthesis. The reduced secretion of $PGF_{2a}$ observed in cells incubated with EPA is likely a result of a shift of the PGHS pathway from synthesis of prostanoids from the 2 series to synthesis of prostanoids of the 3 series. In the presence of EPA, less of the arachidonic acid present will be converted to $PGF_{2a.}$

In a third study, BEND cells were incubated with 0, 20, 40, 60, 80, and 100 uM of either linolenic acid (LNA), DHA, or EPA. Incubation with increasing concentrations of omega 3 fatty acids resulted in a dose-responsive reduction in secretion of $PGF_{2a}$ (P <

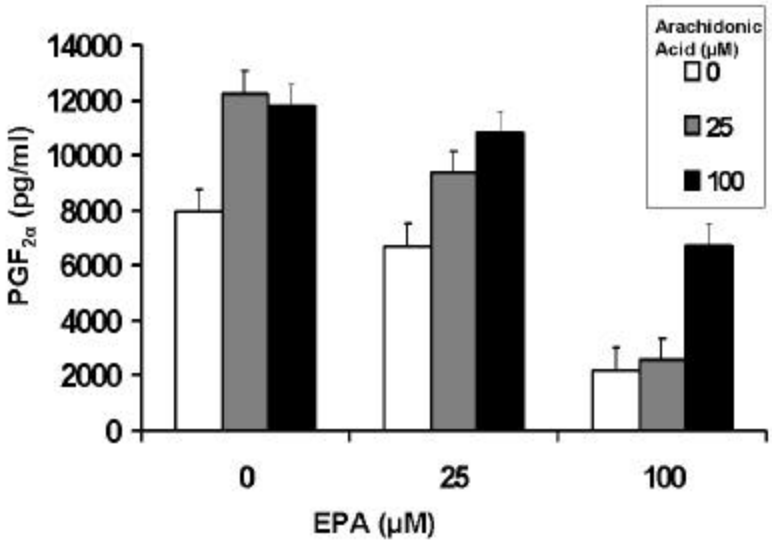


Figure 2. Effects of EPA and arachidonic acid on in vitro synthesis of $PGF_{2a}$ by bovine endometrial cells of the uterus.

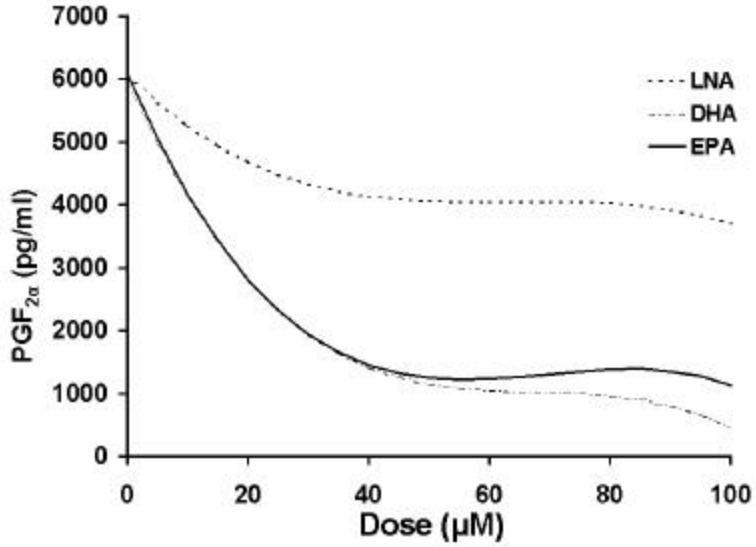


Figure 3. Effect of increasing concentration of linolenic acid (LNA), DHA, and EPA on synthesis of $PGF_{2a}$ by bovine endometrial cells.

0.001). Treatment with 20 uM of any of the three fatty acids resulted in lower secretion of $PGF_{2a}$ in comparison with the control without fatty acid present (Figure 3). Inhibition

of $PGF_{2a}$ was 22, 60, and 61% using LNA, DHA, or EPA respectively ($P < 0.02$). Other studies have demonstrated reduced prostanoid synthesis when fatty acids of the omega 3 and 6 families were fed. For instance, dietary supplementation with gamma-linolenic (C18:3, ω-6) or EPA reduced the synthesis of $PGE_{2a}$ and $PGF_{2a}$ from human endometrial explants (Graham et al., 1994). It is not clear why LNA was less inhibitory than DHA and EPA. Linolenic acid is the precursor for synthesis of DHA and EPA, and can be converted to them in a process that relies on activities of desaturase and elongase enzymes.

Application of these results to the dairy cow was tested. Our hypothesis was that feeding EPA and DHA through fish oil during the periparturient period would increase the proportion of these fatty acids in uterine tissue and reduce the spontaneous secretion of uterine $PGF_{2a}$ of dairy cows at parturition. Pregnant Holstein cows (n = 17) and heifers (n = 9) were assigned randomly to diets containing fish oil (n = 13) (Arista Industries, Wilton, CT) or olive oil (n = 13) (Classico, Bertolli, Italy). A ration containing either fish oil or olive oil was supplied from 21 days before the expected calving date until parturition, when it was replaced by greater nutrient density rations also containing either fish oil or olive oil that were fed until cows reached 21 days postpartum. Cows (n = 6) and heifers (n = 6) that had moderate to severe dystocia, or that were diagnosed with displaced abomasum, retained fetal membranes, or toxic metritis within 10 days after parturition were removed from the analysis.

Rations were formulated to provide approximately 2% oil prepartum and 1.8% oil postpartum. The fatty acid profile of each oil is shown in Table 1. The fish oil used contained 36% EPA and 28% DHA. Therefore the combined intake of EPA (68 g/d) and DHA (53 g/d) was 121 g/d pre and postpartum. Cows were milked three times daily. Blood was obtained once daily at 1730 h from 14 days prior to calving until parturition and from 14 to 21 d postpartum. Between the day of parturition and day 14 postpartum, blood samples were collected twice daily at 0800 and 1730 h. Blood was analyzed for PGFM, a product of $PGF_{2a}$ metabolism. Caruncles were collected by manual extraction through the vagina within 12 h of parturition, frozen in liquid nitrogen, freeze-dried, and analyzed for fatty acid composition.

Concentrations of EPA and DHA in caruncular tissue were increased 5 to 6 fold in cows fed fish oil ($P < 0.01$) (Table 2). The combined concentration of caruncular EPA and DHA were correlated positively with the number of days that cows were supplemented with fish oil ($r^2 = 0.64$), suggesting that introduction of fish oil before 21 d prepartum could have increased the concentrations of EPA and DHA in the uterus.

Cows fed fish oil had reduced concentrations of plasma PGFM during the period of maximum secretion in the early postpartum period compared to cows fed olive oil. Differences were significant ($P < 0.05$) at 0, 0.5, 2, and 2.5 days postpartum (Figure 4). The pattern of postpartum concentrations of plasma PGFM was similar to what was previously reported. Cows fed fish meal at 2.7, 5.2, and 7.8% of dietary DM attenuated the plasma PGFM response to injections of estradiol-17β and oxytocin given on day 15 of the estrus cycle compared to cows not fed fish meal (Mattos, 2001). The increased

Table 1. Fatty acid profile of olive oil and fish oil fed to periparturient dairy cows.

| | Oil Source | |
|---|---|---|
| Fatty Acid | Olive Oil | Fish Oil |
| | ----------(g/100 g of fatty acids)--------- | |
| C14 | 0.00 | 0.41 |
| C14:1 | 0.00 | 0.09 |
| C15:0 | 0.00 | 0.26 |
| C16:0 | 16.62 | 7.03 |
| C16:1 | 1.86 | 6.33 |
| C18:0 | 2.70 | 1.00 |
| tC18:1 | 0.00 | 1.44 |
| C18:1, n9 | 61.28 | 4.46 |
| C18:2, n6 | 16.47 | 2.48 |
| CLA t9t11 | 0.00 | 0.71 |
| CLA c9t11 | 0.00 | 0.00 |
| C18:3, n3 | 0.61 | 1.95 |
| C20:0 | 0.46 | 0.56 |
| C21:0 | 0.00 | 6.36 |
| C20:5, n3 | 0.00 | 35.81 |
| C22:0 | 0.00 | 0.24 |
| C22:6, n3 | 0.00 | 28.42 |
| C24:0 | 0.00 | 0.09 |

Table 2. Fatty acid profile of caruncles of Holstein cows fed diets containing olive or fish oil (% of total fatty acid in tissue).

| | Diet | | | |
|---|---|---|---|---|
| Fatty Acid | Olive Oil | Fish Oil | SE | TRT, P= |
| | ----(g/100 g of fatty acids)---- | | | |
| C14 | 1.30 | 1.24 | 0.10 | 0.69 |
| C14:1 | 0.27 | 0.18 | 0.05 | 0.25 |
| C15:0 | 0.41 | 0.47 | 0.06 | 0.50 |
| C16:0 | 19.84 | 21.79 | 0.74 | 0.09 |
| C16:1 | 1.11 | 1.10 | 0.11 | 0.91 |
| C18:0 | 24.97 | 25.53 | 0.47 | 0.04 |
| tC18:1 | 1.09 | 2.44 | 0.21 | <0.01 |
| C18:1, n9 | 19.92 | 20.18 | 0.77 | 0.81 |
| C18:2, n6 | 12.22 | 9.30 | 0.46 | <0.01 |
| CLA t9t11 | 0.35 | 0.27 | 0.07 | 0.40 |
| CLA c9t11 | 0.36 | 0.35 | 0.05 | 0.88 |
| C18:3, n3 | 0.51 | 0.44 | 0.08 | 0.52 |
| C20:0 | 1.10 | 0.89 | 0.05 | <0.01 |
| C20:5, n3 | 0.23 | 1.66 | 0.02 | <0.01 |
| C22:0 | 5.09 | 4.09 | 0.20 | <0.01 |
| C22:6, n3 | 0.59 | 3.11 | 0.37 | <0.01 |
| C24:0 | 0.70 | 0.63 | 0.08 | 0.53 |

concentrations of EPA and DHA in caruncular tissue of cows fed fish oil suggest that these fatty acids may be the active compounds reducing secretion of $PGF_{2a}$. However a consistent difference in plasma PGFM concentrations between cows fed olive oil and fish oil was not observed throughout the experimental period. Plasma PGFM concentrations of cows fed olive oil and fish oil converged at about day 5 postpartum and remained similar until the end of the experiment. The reduction in plasma PGFM concentrations could be explained by the detachment and shedding of caruncular tissue with high $PGF_{2a}$ synthetic activity that normally takes place in the postpartum period. Preferential shedding of caruncular tissue in cows fed olive oil could have reduced the apparent difference between plasma PGFM concentrations of cows fed olive oil and fish oil.

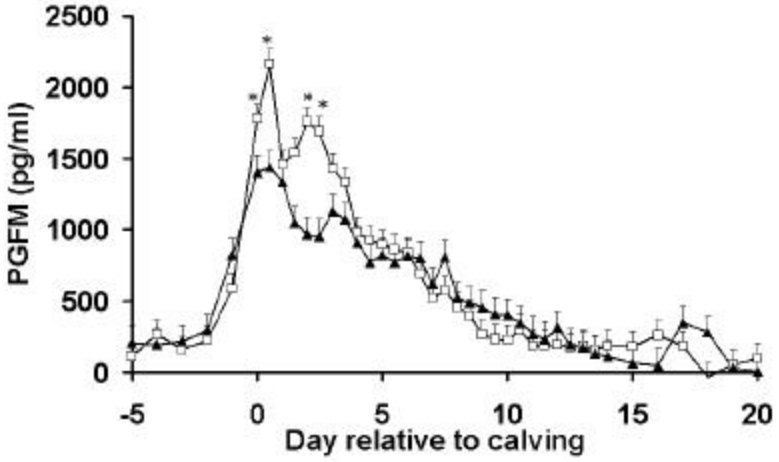


Figure 4. Pre- and postpartum plasma concentrations of prostaglandin $F_{2a}$ metabolite (PGFM) of cows fed fish oil (▲) or olive oil (□) (LSM + SE). The PGFM concentrations were lower in cows fed fish oil at 0, 0.5, 2, and 2.5 days after parturition (*, $P < 0.05$).

Diet did not affect the number of days between the expected due date and actual calving date. It was anticipated that reduced uterine $PGF_{2a}$ secretion could result in delayed parturition. Cows fed olive oil and fish oil calved $3.4 \pm 1.8$ and $3.3 \pm 2.1$ days before the due date, respectively.

Pat Burns (Burns et al., 2000) at Colorado State University has demonstrated that nonlactating beef cows will store EPA and DHA in endometrial tissue when fed fish meal. Mature Angus cows were fed a corn silage-based diet containing either corn gluten meal (n=4) or Menhaden fish meal (n=3) at 8.7 and 5% of dietary DM, respectively. Diets were isonitrogenous and isocaloric. After 25 days of supplementation, estrous cycles of cows were synchronized. Cows were slaughtered at d 18 of the second estrous cycle and uteri were collected, frozen, and analyzed for

linolenic acid, EPA, and DHA. The uterus of cows fed fish meal had greater concentrations of EPA ($P < 0.01$) and tended to have greater concentrations of DHA ($P = 0.12$). Concentrations of linolenic acid were similar between the two groups.

In a follow-up study, Bonnette et al. (2001) fed 82 lactating, primiparous beef cows a corn silage-based diet containing either 5% fish meal or 8.7% corn gluten meal (DM basis). Diets were initiated at 25 days prior to the breeding season and continued through the 90-d breeding season. Cows were artificially inseminated and pregnancy determined at 25-30 days post breeding using ultrasonography. First service conception rate tended to be greater for cows fed fish meal (75.6 vs. 61.5%; $P = 0.14$). Serum progesterone concentrations after insemination were similar between the two groups.

## Linoleic acid

Other fats beside fish oil have been shown to negatively affect $PGF_{2a}$ concentrations. Linoleic acid has demonstrated inhibitory effects both in vitro and in vivo. Supplementation of linoleic acid has reduced the production of arachidonic acid in preweaned calves (Jenkins, 1988). Using in vitro incubation techniques, additional linoleic acid reduced production of $PGF_{2?}$ by bovine pulmonary artery endothelial cells (Kaduce et al., 1982) and by oral squamous carcinoma cells (Elattar and Lin, 1989). In addition, linoleic acid can be converted to a shunt metabolite, eicosadienoic acid ($C_{20:2}$), rather than to arachidonic acid (Kaduce et al., 1982) when excess linoleic acid is present, thereby reducing synthesis of series 1 and 2 prostaglandins. Linoleic acid has been shown to be an inhibitor of prostaglandin synthesis produced by the endometrium in response to the conceptus in order to preserve its integrity (Thatcher et al., 1994). The mechanism of inhibition is thought to occur by linoleic acid competing with arachidonic acid for binding of the key enzyme, PGHS.

Concentrations of plasma PGFM post injection of oxytocin on d 15 of a synchronized estrous cycle was reduced greatly in lactating dairy cows that were abomasally infused with 0.45 kg/d of yellow grease compared with infusions of tallow, glucose, and water (Oldick et al., 1997). Yellow grease and tallow differed in proportion of many fatty acids and this suppressing effect on PGFM cannot be attributed to only one fatty acid. However, linoleic acid is a prime candidate of influence because yellow grease supplied 78 g/d of linoleic acid but tallow supplied only 9 g/d.

Feeding a diet with a fat source containing mainly linoleic acid has improved reproductive performance of lactating dairy cows (Boken, 2001). Soybean oil refining byproduct (**SORB**) (Archer-Daniels-Midland Co., Chattanooga, TN) was fed to cows in free-stall barns fed TMR and to cows grazing rye-ryegrass pasture (2 x 2 factorial design) in winter for 98 days postpartum. The SORB is composed of approximately 70% sodium salts of long chain fatty acids and 30% water having a pH of 8 to 9. It contained only a trace of lecithin. Linoleic acid made up 53% of the fatty acids. The SORB was mixed with liquid molasses (Westway Trading Corp., Tomball, TX) such that SORB made up 30% of molasses (DM basis). The SORB made up 0 or 2% of diet DM.

The liquid molasses alone or molasses with SORB were mixed with the concentrate portion of the diet before feeding. Concentrate was fed to pastured cows at a rate of 1 kg of concentrate (as-fed) to 2.5 kg of milk produced. Blood was sampled three times weekly and analyzed for progesterone in order to determine when cows first started cycling. Cows were artificially inseminated using estrus monitoring devices (HeatWatch®, DDX Inc., Denver, CO).

Cows fed SORB produced milk fat containing more linoleic acid (4.34 vs. 3.96% of fat content) ($P < 0.05$) indicating that some linoleic acid from SORB was escaping biohydrogenation in the rumen and being absorbed in the small intestine. Mean production of 3.5% fat-corrected milk was not affected by SORB but was greater for cows housed in free-stalls and fed TMR (37.4 vs. 33.8 kg) (Table 3). In addition, cows in the barn lost less body weight than cows kept on pasture (-2.35 vs. –4.5 kg/week). In agreement were the mean concentrations of plasma NEFA (262 vs. 464 mEq/L for barn and pasture cows respectively).

Using plasma progesterone concentrations, the postpartum day that ovulations started was determined. Management system had no effect on the number of days to first ovulation. However cows fed SORB commenced ovulation cycles earlier (26.8 d postpartum) ($P = 0.05$) than cows not supplemented with SORB (42.4 d postpartum) (table 4). During the first ovulatory cycle, cows managed on pasture had higher peak plasma progesterone concentration than cows managed in the barn (7.0 vs 4.6 ng/ml, respectively; $P = 0.01$). Eleven cows on pasture compared to 13 cows in the barn were inseminated; total inseminations were 15 and 16, respectively. Nine cows of the 24 inseminated cows became pregnant. Of those 9 cows, seven were managed on pasture (Table 4). Pregnancy rate of cows on pasture as a percentage of cows inseminated was greater ($P = 0.03$) than that of cows housed in the barn (63.6 (7/11) vs. 15.4% (2/13)). Pregnancy rate as a percentage of all cows in the treatment group was greater ($P = 0.03$) for cows managed on pasture (41.2% (7/17)) as compared to cows managed in the barn (11.1% (2/18)). The pasture + SORB group had the greatest pregnancy rate both on a total cow basis (62.5%) and as a percentage of cows inseminated (83.3%). This resulted in an interaction between SORB supplementation and management ($P < 0.05$) as no cows from the barn + SORB group became pregnant.

Other fat supplements containing linoleic acid have improved fertility. Lactating dairy cows fed a calcium salt of palm oil, Megalac® (Church and Dwight, Princeton, NJ), that contains ~8% linoleic acid have shown improvements in conception/pregnancy rates (Ferguson et al., 1990; Garcia-Bojalil et al., 1998; Scott et al., 1995; Sklan et al., 1991). Protection of dehulled cottonseeds (~9% linoleic acid) with protein-aldehyde complexes (Protected Lipid, Rumentek Industries, Australia) delivered approximately 175 g/d of linoleic acid to the lower gut of lactating Hereford cows. Overall pregnancy rates were improved from 63 to 79% (Wilkins et al., 1996).

Table 3. Effect of housing management and supplemental fat (soybean oil refining byproduct; SORB) on production and composition of milk, body weight, body condition score (BCS) and DM intakes of grain supplement, SORB and TMR during the first 14 wk of lactation.

| | Treatment | | | | | | |
|---|---|---|---|---|---|---|---|
| | Pasture | | Barn | | | P < [1] | |
| Measurement | - Fat | + Fat | - Fat | + Fat | SE | Fat | Mgt | Fat x Mgt |
| Milk yield, kg/d | 36.4 | 35.7 | 38.2 | 38.5 | 1.4 | 0.93 | 0.25 | 0.80 |
| Milk fat, % | 3.27 | 3.05 | 3.42 | 3.36 | 0.08 | 0.12 | * | 0.39 |
| Milk fat, kg/d | 1.2 | 1.1 | 1.3 | 1.3 | 0.1 | 0.46 | * | 0.64 |
| 3.5% FCM, kg/d | 34.7 | 32.9 | 37.6 | 37.3 | 2.1 | 0.63 | † | 0.73 |
| | | | | | | | | |
| Milk protein, % | 2.88 | 2.91 | 2.90 | 3.05 | 0.06 | 0.15 | 0.18 | 0.29 |
| Milk protein, kg/d | 1.0 | 1.0 | 1.1 | 1.2 | 0.1 | 0.68 | * | 0.47 |
| MUN,[2] mg/100 ml | 12.4 | 10.2 | 12.8 | 11.0 | 1.0 | † | 0.58 | 0.83 |
| | | | | | | | | |
| Supp. intake, kg/d | 12.4 | 12.1 | --- | --- | | | | |
| SORB intake, kg/d | 0 | 0.47 | 0 | 0.49 | | | | |
| TMR intake, kg/d | --- | --- | 23.0 | 25.0 | | | | |
| | | | | | | | | |
| BCS | 2.92 | 3.30 | 3.05 | 3.21 | 0.15 | † | 0.90 | 0.46 |
| BCS change, units/wk | -0.07 | -0.05 | -0.06 | -0.01 | 0.03 | 0.25 | 0.36 | 0.55 |
| BW, kg | 565 | 662 | 594 | 636 | 24 | * | 0.94 | 0.27 |
| BW change, kg/wk | -3.1 | -5.9 | 0.4 | -5.1 | 1.3 | † | * | 0.74 |

[1] Mgt = effect of managing in confinement or on pasture; Fat x Mgt = interaction of fat supplementation and management.

* P = 0.05

† P = 0.10

[2] Milk urea nitrogen

Table 4. Effect of housing management and fat supplementation (soybean oil refining byproduct) on breeding and conception rates from day 45 to 100 postpartum.

| | Pasture | | Barn | |
|---|---|---|---|---|
| Measurement | No SORB | + SORB | No SORB | + SORB |
| Number of cows (n =) | 9 | 8 | 9 | 9 |
| Anestrous cows (n =) [1] | 2 | 0 | 0 | 1 |
| Estrous cycle: | | | | |
| Time of first ovulation, days [a] | 43.6 | 21.4 | 41.2 | 32.1 |
| Ovulation interval, days | 19.6 | 23.9 | 21.9 | 18.6 |
| Peak progesterone, ng/ml [b] | 7.2 | 6.7 | 4.8 | 4.4 |
| Breeding opportunities: | | | | |
| Ovulations (n =) | 19 | 13 | 15 | 18 |
| Estruses detected with ovulation [2] | 10 | 8 | 14 | 10 |
| Ovulations detected with estrus [2], % | 52.6 | 61.5 | 93.3 | 55.6 |
| Breeding: | | | | |
| Cows inseminated, (n =) | 5 | 6 | 7 | 6 |
| Inseminations per group, (n =) | 8 | 7 | 8 | 7 |
| Ovulations detected and AI[3], % | 80.0 | 87.5 | 57.1 | 70.0 |
| Pregnancy: | | | | |
| Number of cows | 2 | 5 | 2 | 0 |
| Of cows inseminated [d,e], % | 40.0 | 83.3 | 28.6 | 0.0 |
| Of total cows in group [d,e], % | 22.2 | 62.5 | 22.2 | 0.0 |
| Services per conception | 1.5 | 1 | 1.5 | --- |

[1] Anaestrous defined as cows having no cycles prior to 85 days postpartum.
[2] Estrus accompanying ovulations were detected using HeatWatch® and/or visual observations of standing heats.
[3] Artificial inseminations that preceded a confirmed ovulation using plasma progesterone values that could have resulted in pregnancy.
[b] Housing management, P = 0.01.
[c] SORB by housing management, P < 0.03.
[d] Housing management, P < 0.03.
[e] SORB by housing management, P < 0.05.

## Summary

Growing evidence indicates that the design and delivery of supplemental fatty acids to the lower gut (primarily linoleic acid, EPA and DHA) may target reproductive tissues to improve reproductive function and fertility. Improvement in embryo survival may be associated with suppression of uterine prostaglandin secretion via linoleic acid or other longer chain unsaturated fatty acids. Although not discussed in this paper, changes in follicular dynamics can be affected by fat supplementation and may lead to a more fertile ovulation. This improvement may be due to alterations in metabolic hormones like IGF-I and growth hormone or hormonal clearance.

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