EFFECTS OF FATTY ACIDS ON REPRODUCTION IN THE DAIRY COW: THE GOOD AND THE BAD

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INTRODUCTION

Recently, there has been a great deal of interest in feeding fat to dairy cows in order to increase energy density of the diet and improve reproduction. It is known that cows fed supplemental fat may experience improved energy balance and begin to cycle sooner because of enhanced follicular growth and development (Grummer and Carroll, 1991). However, Lucy et al. (1992) suggested that it was fatty acids, and not the additional energy provided by the fatty acids, that stimulated ovarian function. Recently, new information has been published that demonstrates that the type of dietary fatty acids is important as individual fatty acids do not have the same effects on reproduction of the dairy cow.

FATTY ACID TERMINOLOGY

A fatty acid molecule is shaped like a caterpillar with two different ends: a methyl group and a water-soluble end that is the carboxyl end. There are different families of fatty acids in feed: omega-3, omega-6, omega-7, and omega-9. The most common numbering system is called the omega system. This system numbers carbon atoms in sequence, starting from the methyl end. The other commonly used system, called the delta (*d*) system, starts at the acid end and numbers the carbon atoms in reverse direction.

The omega-7 family of fatty acids is synthetised from palmitic acid (C16:0) while the omega-9 fatty acid family is synthetised from stearic acid (C18:0) via oleic acid (C18:1, Figure 1). These two families are not considered essential as they are produced in the body.

The omega-3 and omega-6 fatty acids are essential because both are vital to health but cannot be made by our cells and must, therefore, be provided by foods.

Linoleic acid (C18:2) belongs to the omega-6 family while linolenic acid (C18:3) belongs to the omega-3 family (Figure 2). The system used to name fatty acids consider the number of carbons in the chain (e.g. 18 for linoleic acid), the number of double bonds in the chain (2 for linoleic acid) and where in the chain the first double bond is located from the methyl end (1st double bond between carbons 6 and 7 for linoleic acid): C18:2.

Figure 1. Schematic pathway of omega-7 and omega-9 fatty acid synthesis.

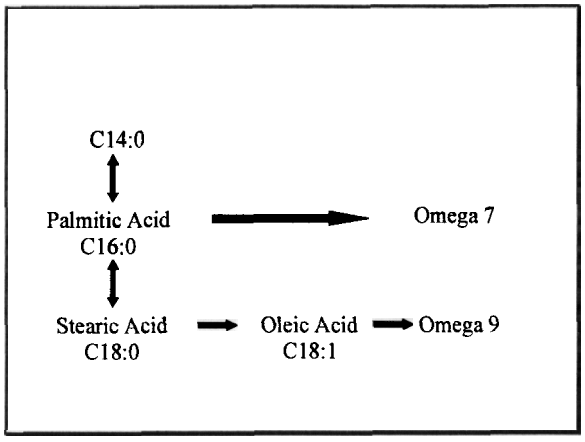
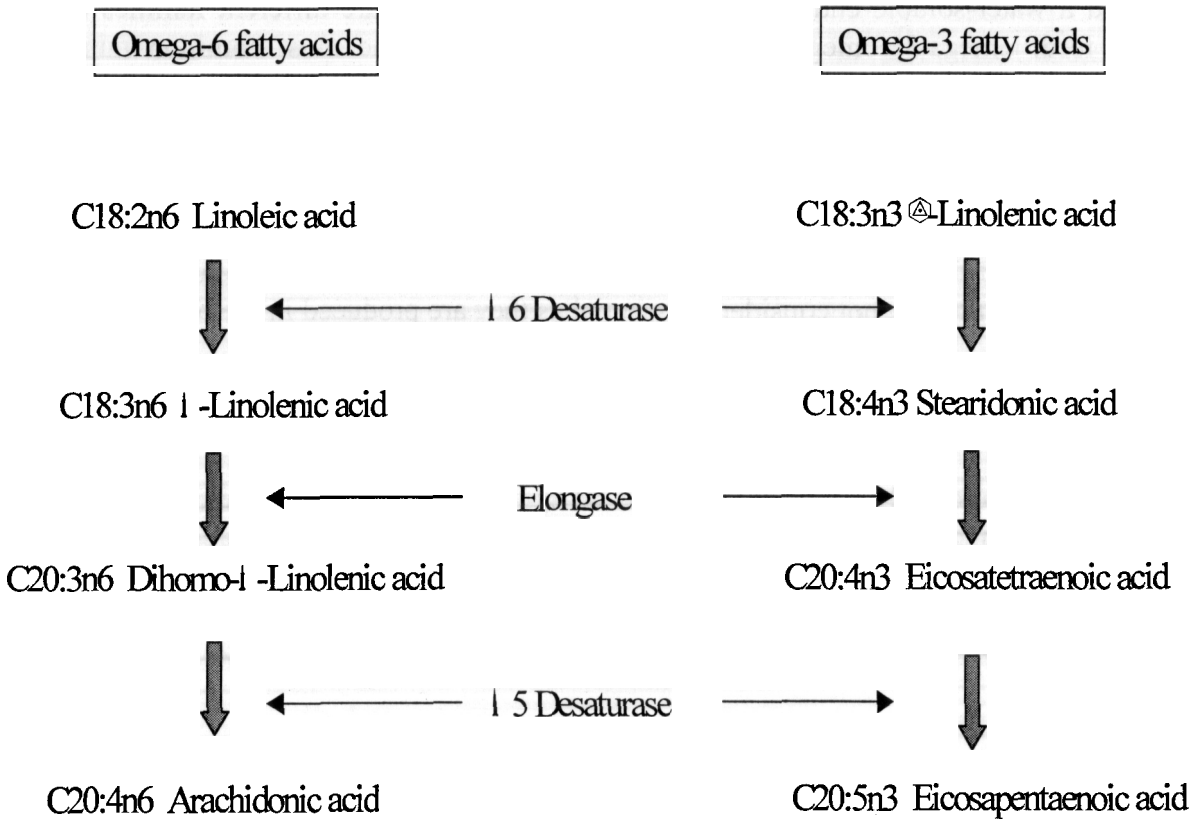


Figure 2. Schematic pathway of omega-6 and omega-3 fatty acid synthesis.



SOURCES OF FATTY ACIDS

The main sources of short chain fatty acids are cottonseed and palm oils. All sources of fat contain long chain fatty acids. The main sources of linolenic acid (C18 :3T3) are flaxseed, hemp, canola, soybean, nuts and dark green forages. Ryegrass silage contains as much as 60% of linolenic acid as a percentage of total fatty acids (Dewhurst and King, 1998), which would encourage high forage systems to increase dietary linolenic acid content. Omega-3 fatty acids are found also in cold water and salt water fish (salmon, trout, makerel, sardines). The main sources of linoleic acid (C18 :2T6) are sunflower ssed, safflower, hemp, soybean, nuts, pumpkin seeds, sesame seeds and flaxseed. Gamma-linolenic acid (C18 :3T6) is found in evening primose oil, grape seeds and borage. Dihomogamma-linolenic acid (C20 :3T6) is found in maternal milk while arachidonic acid (C20 :4T6) is occurs mainly in meat and animal products. Oleic acid (C18 :1) is found in olive, almond, avocado, peanut, pecan, cashew, macadamia nut and butter. Omega 7 in the form of palmitoleic acid (C16 :1) is found in tropical oils (coconut, palm). Composition in C18 fatty acids of some edible vegetable oils is reported in Table 1.

OIL	C18 :0	C18 :1	C18 :2	C18 :3
Peanut	2	47	32	0
Canola	2	64	19	8
Safflower	2	12	77	0
Cottonseed	25	21	50	0
Linseed (Flax)	4	19	14	58
Crn	2	25	60	1
Tallow	15	41	8	1
Fishmeal (10 to 12 % Fat)	2	25	4	45
Hi Linolenic Ryegrass	6	4	14	43
Olive	2	76	8	0
Palm	4	39	10	1
Sesame	2	42	45	0
Soybean	4	24	53	7
Sunflower	5	20	69	0
Megalac®	3.5	32.3	7.8	0.3

Adapted from Erasmus (1993)

FATTY ACIDS AND FERTILITY

Supplementary fats are likely to affect fertility because fatty acids are the precursors both of prostaglandins (**PG**) and, via cholesterol, the steroid hormones. In general, feeding supplemental fat such as calcium soaps of long chain fatty acids, fish meal, and tallow increases conception rates. However, a lowered conception rate at first service has been reported when there was a paralleled increase in milk production (range of 2.2 to 4.5 kg/d).

Thatcher and Staples (2000) wrote an excellent review on the subject. There are two main families of essential fatty acids, omega-3 and omega-6 fatty acids, that could affect fertility. The main source of omega-6 fatty acids is dietary linoleic acid (C18:2n-6) and this is converted to arachidonic acid (C20:4n-6), which *inter alia* is the precursor of the dienoic (2-series) PG, such as PGF_2. The same elongase and desaturase enzymes also convert the main dietary omega-3 fatty acids (-linolenic acid; C18:3n-3) to eicosapentaenoic acid (**EPA**; C20:5n-3), the precursor of the trienoic (3-series) PG, such as PGF_3 (Abayasekara and Wathes, 1999). Competition between omega-3 and omega-6 precursors for desaturation and elongation as well as at the site of PG synthetase means that increasing the supply of omega-3 fatty acids will decrease production of dienoic PG (Barnouin and Chassagne, 1991). In many cases the trienoic PG have lower biological activity than the corresponding dienoic PG (Fly and Johnston, 1990) and this may directly affect aspects of fertility. For example, treatments that reduce ovarian and endometrial synthesis of PGF_2, at the expense of PGF_3, may contribute to a reduction in embryonic mortality (Mattos et al., 2000). There is some evidence for different effects of -linolenic acid and the omega-3 fatty acids from fish oil (EPA and docosahexaenoic acid (**DHA**), C22 :6n-3) on eicosanoid (interleukin) synthesis, perhaps because of differences in the way in which these fatty acids incorporate into cell membranes (Wu et al., 1996).

Supplementary fats can also reduce the total synthesis of PG by affecting the activity of PG synthase (Thatcher et al., 1995). Diets rich in linoleic acid (C18:2) increase arachidonic acid concentration (C20:4) in tissues and diets rich in linolenic acid (C18:3) increase concentration of eicosapentaenoic acid (C20:5) (Béréziat, 1978). Moreover, eicosapentaenoic acid (C20:5) is a competitive inhibitor of the enzyme complex involved in the synthesis of prostaglandins from arachidonic acid (C20:4) (Leat and Northrop, 1979; Holman, 1986). Therefore, this would suggest that a diet with a low linoleic to linolenic acid ratio (C18:2:C18:3, omega-6:omega-3) could decrease prostaglandin secretion or prostaglandin activity as suggested by Barnouin and Chassagne (1991), which would thus have important effects on reproduction and immunity in the dairy cow.

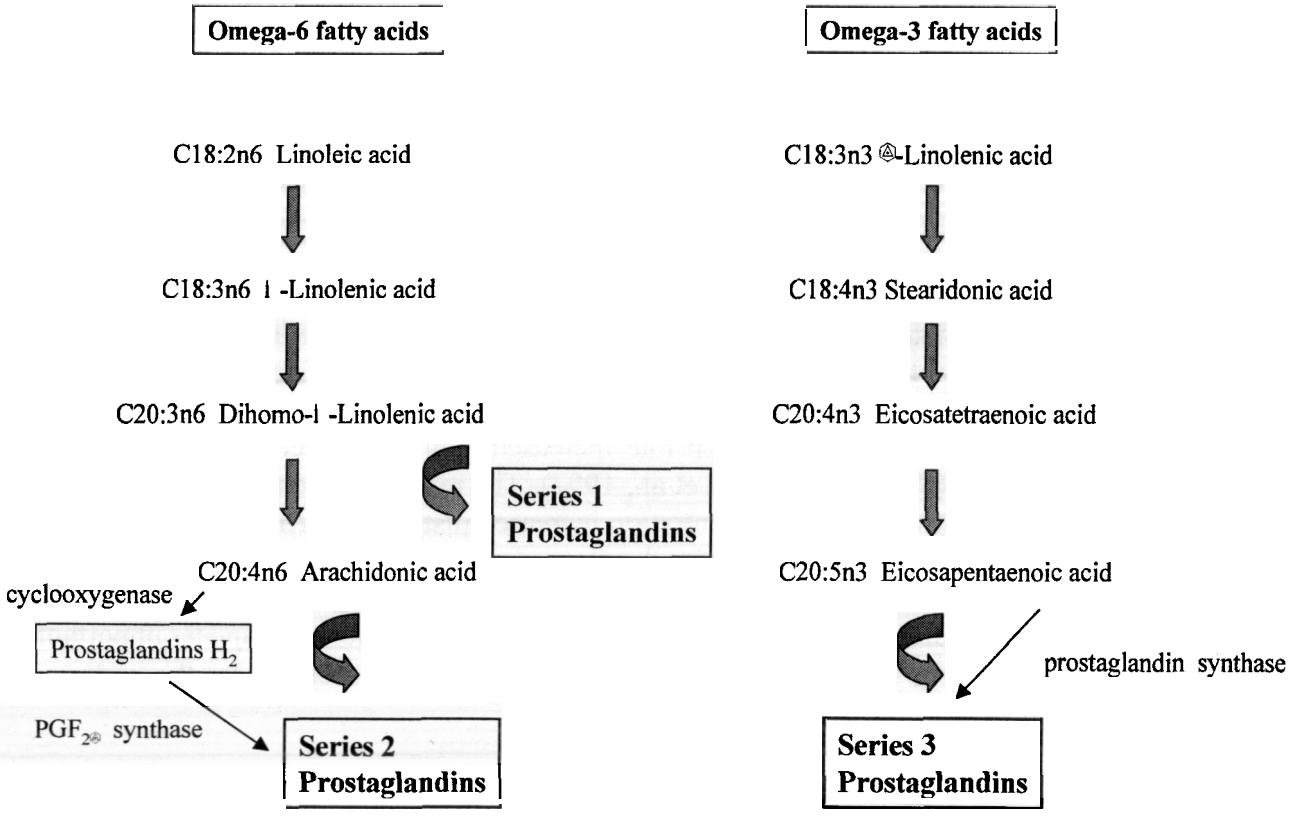
PROSTAGLADINS SYNTHESIS

There are two main pathways (Figure 3) used to synthesize **PG**: one is used by most dietary fat (e. g. corn and soybean, sources of omega-6 fatty acids) and leads to series 1 and 2 PG while the other one is more specific to fish products and flax (sources of omega-3 fatty acids) and leads to series 3 PG. Thus, depending on the pathway used for PG synthesis, the type and role of the resulting PG will differ. PG of series 2 are important at calving; they increase platelet agglutination and blood clot formation, they increase salt retention in kidneys, water retention, and blood pressure. PG of series 2 cause also inflammation, which lead to their role of "bad guys" among the different PG series. PG of series 1 improve the immune sytem of T cells, prevent platelet agglutination and heart attack, contribute to remove the excess of Na and water in kidneys, decrease the inflammatory response and contribute in controling arthritis and decreasing cholesterol production. PG of the series 3 have a very weak platelet agglutination power and they prevent fabrication of PG of the series 2; they also prevent heart attack, water retention, and inflammation. PG of the series 1 and 3 are thus considered as "good guys" contrary to those of the series 2. In fact, our preliminary results (Gagnon et al., 2000) showed

that some immune parameters were affected by the type of dietary fatty acids at the time of embryo implantation.

Some polyunsaturated fatty acids (PUFA) can serve as a substrate for the synthesis of PGF_2. These include cis-linoleic acid (C18:2) that is commonly found in natural fat sources. It can be desaturated and elongated to form arachidonic acid which serves as an immediate precursor for the series 2 PG of which PGF_2 is a key member. Key regulatory enzymes for these conversions include six desaturase and cyclooxygenase. These same fatty acids also can inhibit PG synthesis by competitive inhibition with these key enzymes. Linoleic acid has been shown to be an inhibitor of PG synthesis that is produced by the endometrium in response to the presence of a conceptus in order to preserve the integrity of the conceptus (Thatcher et al., 1994). Other fatty acids besides linoleic acid can play inhibitory roles. EPA and docosahexanoic acid (C22:6) ha ve been shown to inhibit cyclooxygenase activity, which is an enzyme involved in the synthesis of PGF_2.

Figure 3. Metabolic pathway of series 1, 2, and 3 prostaglandins



FATTY ACIDS, CHOLESTEROL, AND PROGESTERONE

Cholesterol serves as a precursor for the synthesis of progesterone by ovarian luteal cells. Secretion of progesterone is the main function of the corpus luteum. Progesterone not only prepares the uterus for implantation of the embryo but also helps maintain pregnancy by providing nourishment to the conceptus. The successful establishment and maintenance of pregnancy (before day 16 post AI) requires the maintenance of progesterone secretion through the critical period of the maternal recognition of pregnancy when luteolysis occurs in the non-pregnant animal (Lamming and Royal, 2001). Between 25 and 55% of mammalian embryos die in early gestation. Increased concentrations of plasma progesterone have been associated with improved conception rates of lactating ruminants. Similarly, progesterone concentration prior to AI has been associated with greater fertility. In a field study involving 426 lactating dairy cows, blood was sampled on 58 d postpartum for multiparous cows and 72 for primiparous cows and then analyzed for progesterone. Cows were bred approximately 3 d later in a synchronized estrus scheme. Conception rate increased 1.44% for every 1 ng/ml increase in plasma progesterone ($r^2 = 0.11$, Staples et al., 1997). The recovery of embryos 7 d after

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It has been suggested that improved conception rate could be a result of increased concentrations in plasma cholesterol (Spicer et al. 1993), although this hypothesis was not supported by our results. In fact, cows fed formaldehyde-treated flaxseed had lower plasma cholesterol concentration and better conception rate than those fed Megalac® (Petit et al., 2001). Other studies have reported no relationship between cholesterol concentrations in blood and reproductive measures (Ferguson et al. 1990; Spicer et al. 1990).

The fatty acid profile of the dietary fat may influence the propensity of animals to increase plasma progesterone. Mature ewes were infused intravenously with saline, soybean

oil, or olive oil for 5h on d 9 through 13 of an estrous cycle (Burke et al., 1996). Serum cholesterol was increased by fat infusates, and olive oil was more effective than soybean oil (127, 141, and 153 mg/dl for saline, soybean oil, and olive oil, respectively). However, soybean oil infusion resulted in greater progesterone response than did infusion of olive oil at 2.5h postinfusion. Therefore, the greatest concentration of serum cholesterol did not coincide with the greatest concentration of serum progesterone.

FATTY ACIDS AND PROSTAGLANDINS SECRETION

It is known that there is a negative relationship between concentration of PGF_2 and that of progesterone. For example, at calving, PGF_2 concentration increases while that of progesterone decreases. Similarly, during gestation, PGF_2 concentration decreases and that of progesterone increases. Progesterone is secreted by the corpus lutem and synthesized by steroids. Therefore, an increase in PGF_2 concentration is paralleled with a decrease in progesterone concentration and vice versa. In theory, it could thus be possible to modulate concentrations of PGF_2 and progesterone by different feeding strategies! In fact, in the experiment we carried out in UK, we observed a tendency ($P = 0.09$) for greater progesterone concentration in the blood of cows fed formaldehyde-treated flax compared to those fed Megalac (Petit et al., 2002). This may partly explain the greater gestation rate observed for cows fed formaldehyde-treated flax (87.5%) compared to those fed Megalac (50.0%) in a companion study (Petit et al., 2001).

Better conception rate for cows fed formaldehyde-treated flaxseed compared to those fed Megalac® could result from different prostaglandins synthesis. In fact, linolenic acid in flaxseed uses the eicosapentaenoic acid metabolic pathway while fatty acids in Megalac uses partly the arachidonic acid pathway (Cunnane 1995) and it is known that eicosapentaenoic acid inhibits prostaglandins synthesis (Spicer et al. 1993). Therefore, ingestion of linolenic acid contained in flaxseed could potentially inhibit PGF_2 synthesis (Cunnane 1995). Thatcher et al. (1997) has shown that PGF_2 secretion is decreased in dairy cows fed fish meal. In fact, fish meal, which would lead to eicosapentaenoic acid and docosahexaenoic acid formation, has been shown to increase gestation rate of dairy cows and to alter corpus luteum regression as shown by greater plasma concentrations of progesterone (Burke et al. 1997). This would agree with the tendency observed in one of our experiment (Petit et al., 2002) for greater milk progesterone concentration, expressed as the area under the curve, for cows fed formaldehyde-treated flaxseed compared to those fed Megalac®. However, it is not known if the greater conception rate observed for cows fed formaldehyde-treated flaxseed in the experiment of Petit et al. (2001) was a result of a decrease in embryo mortality or better fertilization of the ova as pregnancy was confirmed only once at d 45 post AI. More research is required to determine the reasons for better conception rate for cows fed a source rich in omega-3 fatty acids. The potential to improve reproduction of dairy cows through dietary manipulation is an exciting concept and needs to be further addressed.

One of the rate-limiting precursors for PGF_2 synthesis is arachidonic acid. It is known that the essential fatty acid linoleic acid acts as a competitive inhibitor of PG synthase (Thatcher et al., 1994) and that the uterus of pregnant cows at day 17 are enriched with non-esterified linoleic acid (Thatcher et al., 1995). An increase in the linoleic pool in blood would suggest that linoleic acid becomes a competitive inhibitor with arachidonic acid for the

prostaglandin synthase enzyme system. In addition, linoleic acid can be converted to a shunt metabolite, eicosadienoic acid (C20:2), rather than to arachidonic acid (Kaduce et al., 1982) when excess linoleic acid is present, thereby reducing synthesis of series 2 prostaglandins. A decrease in arachidonic acid biosynthesis by inhibition of .6 and .5 desaturase enzymes that are necessary for conversion of linoleic acid to arachidonic acid would decrease PGF_2 secretion. Duodenal infusion of yellow grease (enriched in linoleic acid) depressed peak plasma concentrations of PGFM (Oldick et al., 1997). Moreover, feeding diets containing 2.6, 5.2 and 7.8% Menhaden fish meal to lactating dairy cows reduced uterine secretion of PGF_2, (Thatcher et al., 2001a).

Dietary supplementation with -linolenic acid (C18:3, n-6) or EPA reduced the synthesis in vitro of PGE_2 and PGF_2 from human endometrial samples collected 6 months after initiation of dietary treaments (Graham et al., 1994). Infusion of a fat source rich in linoleic acid (17%) into the abomasum of lactating dairy cows resulted in a significant attenuation in the release of PGFM, as measured in peripheral plasma, in response to an injection of oxytocin on day 15 of a synchronized oestrous cycle (Oldick et al., 1997).

Dietary PUFAs can decrease PGF_2 synthesis by different actions, which include decreasing the availability of precursor arachidonic acid, increasing the concentration of fatty acids that compete with arachidonic acid for series 2 PG, and inhibiting PG synthase. Reduced availability of arachidonic acid in the uterine phospholipid membranes for conversion to series 2 PG can occur through a reduction in the synthesis of arachidonic acid or through displacement of existent arachidonic acid from the phospholipid membranes by other fatty acids. This can be achieved through dietary supplementation with fish oil (rich en EPA and DHA) or linseed oil as they are major inhibitors of desaturation and elongation in liver cells leading to arachidonic acid formation (Bezard et al., 1994). Moreover, as there is a preferential processing of n-3 fatty acids by .6 desaturase at the expense of desaturation of n-6 fatty acids (Sprecher, 1981), feeding n-3 fatty acids would lead to a reduction in arachidonic acid formation. In summary, inhibition of PG secretion can be achieved through: 1) reduced synthesis of arachidonic acid by .6 and .5 desaturase enzymes necessary for conversion of linoleic acid to arachidonic acid; 2) alteration in fatty acid profile (in favour of omega-3 in membrane phospholipids which may or may not be precursors of other eicosanoids; 3) inhibition of synthesis and activity of cyclooxygenase enzymes responsible for the synthesis of PGF_2; and 4) inbition of gene expression involved in the synthesis of series 2 PG (Mattos et al., 2000).

MATERNAL RECOGNITION OF PREGNANGY

The dialogue between the conceptus and uterine endometrium leads to maintenance of the corpus luteum. The ability of embryonic interferon-9 to inhibit uterine secretion of PGF_2 is critical to the establishment of pregnancy in cattle. Up to 40% of total embryonic losses are estimated to occur beween day 8 and day 17 of pregnancy (Thatcher et al., 1994). This high proportion of losses is coincident with the period of conceptus inhibition of uterine PGF_2 secretion, suggesting that some loss may be occurring because certain conceptuses are unable to inhibit secretion PGF_2. Future strategies to improve embryo survival during this critical period will be based upon a through understanding of the factors regulating "a better communication between the embryo and the mother at the embryo interface".

The success of early pregnancy in the mated cow is dependant on the successful maternal recognition of pregnancy (Thatcher et al., 1995; Mann et al., 1999). To achieve this the embryo must prevent the demise of the corpus luteum by the timely production of interferon tau, the embryonic signal which acts to inhibit the development of the maternal luteolytic mechanism. Interferon tau acts locally in the uterus to suppress the development of oxytocin receptors in the endometrium and thereby suppress the secretion of luteolytic episodes of PGF_2 generated by the binding of oxytocin to its receptors (Mann et al., 1999). It has been shown that the pattern and level of ovarian steroid hormones in early pregnancy can influence both embryo development and survival and the timing and intensity of the mothers luteolytic drive. For example low progesterone levels or high oestradiol levels during the luteal phase increase the strength of the luteolytic drive while low post ovulatory progesterone levels result in retarded embryo development (Mann et al., 1999). Moreover, lower estradiol concentrations may prevent premature regression of the corpus luteum and prevent early embryonic death (Staples et al., 1998).

Oxytocin induces release of PGF_2 (Tysseling et al., 1998). Increased prostaglandin synthesis induced by oxytocin during days 5 to 8 of pregnancy reduced pregnancy rates of beef cows at 30 days after AI from 80 to 30% (Lemaster et al., 1999). Treating cows concomitantly with an inhibitor of prostanoid synthesis neutralized the effect of oxytocin and restored pregnancy rates to 80%. The implication is that increased PGF_2 secretion during early pregnancy causes embryonic loss and supports the hypothesis that reducing PGF_2 during this period reduces embryonic loss and improves pregnancy rates. Arachidonic acid is the rate limiting fatty acid for the synthesis of PGF_2 via the action of PGF_2 synthase. The same enzymes also are capable of processing other fatty acids, such as EPA, which is the precursor for the synthesis of prostanoids of the 3 series. Increased availability of EPA in membrane phospholipids could displace arachidonic acid, leading to increased synthesis of prostanoids of the 3 series at the expense of prostanoids of the 2 series, such as PGF_2. Prostanoids of the 3 series are less bioactive, and there appears to be no evidence for their role in ruminant luteolysis. Gamma-linolenic acid (GLA, C18:3n6) and EPA has been shown to reduce the synthesis in vitro of PGF_2 and PGE_2 (Graham et al., 1994).

Both EPA and INF-ϑ inhibit secretion of $PGF_{2\forall}$ through different mechanisms. Interferon-ϑ, but not EPA, reduced levels of enzyme gene expression (cyclooxygenase-2) and thus modulates $PGF_{2\forall}$ production (Thatcher et al., 2001b). On the other hand, EPA does not seem to affect enzyme gene expression but would be involved in competition of precursors for processing by the cyclooxygenase enzymes, and regulation of enzyme activity. The implication of these findings is that supplementation with inhibitory fatty acids such as EPA during early pregnancy by dietary or parenteral means may further enhance the suppression of $PGF_{2\forall}$ secretion in concert with the action of embryonic IFN-ϑ. Because a significant proportion of bovine embryos are thought to be lost due to inadequate inhibition of uterine $PGF_{2\forall}$ secretion, further inhibition by exogenous means may result in increased embryo survival. This hypothesis is supported by the findings of Burke et al. (1997), in which feeding lactating dairy cows of low fertility a source of EPA and DHA in fish meal incresed pregnancy rates from 31.9 to 41.3%.

FATTY ACIDS, PARTURITION, AND RETAINED PLACENTA

Parturition is a process that is accompanied by the massive release of prostaglandins. Alterations of fatty acids in the endometrium have been described in normal parturition, and manipulations of fatty acid content used experimentally to delay onset of parturition. Fatty acids of the omega-3 family have been shown to affect uterine activity during parturition in rats and sheep, and to delay the onset of parturition in humans (Olsen et al., 1992). Supplementing linolenic acid to a diet deficient in essential fatty acids resulted in an impairment of parturition rates (Leat and Horthrop, 1979). This also occurred when fish oil was given to rats as the major dietary essential fatty acid source, and an inhibition of uterine synthesis of PGE2 was detected (Leaver et al., 1986). In pre-term pregnant sheep, intravenous infusion of a 20% omega-3 fatty acid emulsion resulted in a delay in the onset of induced labour and delivery compared with a control group infused with an emulsion of soybean oil containing 7% omega-3 fatty acids.

Forages could also affect reproduction. Chassagne and Barnouin (1992) reported that cows fed grass silage had lower blood PG concentrations than those fed corn silage. Grass silage had greater concentration of linolenic acid and lower concentration of linoleic acid than corn silage. As a result, the incidence of retained placenta in cows fed grass silage was higher than in cows fed corn silage. As linolenic acid is an inhibitor of PG secretion, a high linolenic to linoleic acid ratio (grass silage) therefore could result in retained placenta. Kemp et al. (1998) reported that cows requiring more time to expulse their placenta also had lower PG metabolite blood concentrations at calving. However, in their experiment, the linolenic to linoleic acid ratio (C18:3:C18:2) had no effect on delivery time of placenta probably because the difference in the linolenic to linoleic acid ratio (C18:3:C18:2) between flaxseed and sunflower seed based diets was not large enough. This would suggest that fatty acid composition of forages and diets could have important effects on cow reproduction. So far, we have observed (Benchaar et al., unpublished results) that a grass silage based diet decreases the omega 6 to omega 3 fatty acid ratio in milk compared to a corn silage based diet; we have no data however on the effects of these two diets on reproduction parameters.

FATTY ACIDS AND REPRODUCTION FUNCTION

In theory, feeding omega 3 fatty acids would delay the return to cyclicity after calving due to a decrease in the synthesis of series 2 prostaglandins, which could increase the number of days to first service. Synthesis of series 2 prostaglandins is required after calving for uterine involution, which lead to the return of normal cyclicity. On the other hand, feeding omega 3 fatty acids would improve maternal recognition and thus decrease embryo mortality. Taken altogether, this would strongly suggest that feeding omega 3 fatty acids would delay the return to cyclicity but lead to a better gestation rate when cows are bred. We are currently conducting an experiment to study this hypothesis and our preliminary results (Petit and Twagiramungu, 2002) show that cows fed omega 3 fatty acids have no embryo mortality compared to those fed micronized soybeans or calcium salts of palm oil.

Therefore, feeding omega 3 fatty acids should improve the overall reproductive function of cows as a result of better gestation rate, decreased embryo mortality, and decreased service per conception. However, we still need to do more research on this topic as there are almost no published data regarding the effects of specific fatty acids on the overall reproduction of dairy

cows. There is a specific need to develop different feeding strategies according to the reproductive stage of cows; fatty acids required for better maternal recognition (omega 3) won't be necessary the same as those required for easier calving (omega 6). We should be balancing diets for specific fatty acids for optimum reproduction performance. The only problem is that polyunsaturated fatty acids (e.g. omega 3 and omega 6) are biohydrogenated by rumen microbes and these fatty acids must bypass the rumen to have any effect on reproduction. These fatty acids must therefore be protected against the attack of rumen microbes but they must remain digestible in the intestine and this is even more important for free oils. Oils contained in fish meal (EPA and DHA) escape partially biohydrogenation in the rumen (Ashes et al., 1992).

CONCLUSIONS

In a practical manner, we could summarize five possible strategies to improve reproduction of the cow:

1) Generate a larger corpus luteum: it is known that a larger corpus luteum will secrete more progesterone and this may have a positive effect on pregnancy recognition and consequently pregnancy rates. Feeding flaxseed has increased corpus luteum diameter and progesterone concentration in dairy cows (Petit et al. 2002).

2) Increase progesterone concentration: a greater progesterone concentration leads to a better maternal recognition of pregnancy (Staples et al., 1997). Feeding omega 3 fatty acids increases progesterone concentration.

3) Decrease PGF_2 secretion: inhibition of PGF_2 secretion would increase pregnancy recognition. Linoleic acid is an endometrial PG synthesis inhibitor through cyclooxygenase-2 activity inhibition. A possible strategy to decrease PGF_2 secretion would be to increase the linoleic to arachidonic acid ratio in the uterus. Feeding cows diets rich in linoleic acid constitutes a practical alternative to inhibit PGF_2 synthesis in the uterus. However, linoleic acid is biohydrogenated in the rumen and special care must be taken with choosing feed ingredients.

4) Increase series 3 prostaglandin secretion: feeding fish meal (Burke et al., 1997) and flaxseed (Petit et al., 2002) inhibit PGF_2 synthesis as both will lead to the synthesis of series 3 prostaglandins. Competition for the same key enzymes will lead to a lower synthesis of PGF2.

5) Inhibition of cyclooxygenase activity: Cyclooxygenase is the enzyme leading to the synthesis of PGF_2. Eicosapentaenoic and docosahexanoic acids have been shown to inhibit cyclooxygenase activity. High concentrations of 20-carbon fatty acids (such as dihomo--linolenic acid, C20:3 and EPA, C20:5) other than arachidonic acid (C20:4n6) can compete with arachidonic acid for active sites of prostaglandin-endoperoxide synthase complex, therefore, reducing the conversion of arachidonic acid to the series 2 PG (Weber and Sellmayer, 1990).

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