Fat and Fat-Soluble Vitamin Supplementation for Improving Reproduction of the Dairy Cow

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Introduction

The scientific literature contains evidence that the nutrient status of lactating dairy cows can have a direct bearing on reproductive status. Pregnancy rate has been improved by manipulating the mineral (Hurley and Doane, 1989), vitamin (Seymour, 2001), energy (Butler, 2001), protein (Butler, 1998), and lipid fractions (Staples et al., 1998) of the diet. Nevertheless the amount of knowledge in this area is not great. Collective efforts of nutritionists, reproductive physiologists, immunologists, and veterinary practitioners and researchers are needed in order to advance our understanding of both the extent of potential impact and the physiological mechanisms by which these nutrients act in vivo. The challenge to characterize the factors contributing to conception and embryo development as well as developing strategies to use to improve embryo survival is complex involving steroidogenesis, cell proliferation, follicle development, ovulation, fertilization, corpus luteum development and maintenance, oviductal and uterine functions, embryo implantation and subsequent fetal growth. Indeed our current daily production and reproductive management systems impact all of these coordinated events and need to be optimized if reproductive efficiency in lactating dairy cows is to be enhanced. Dietary vitamins A and E, protein, fat, and phosphorus are nutrients selected to briefly review in this paper in terms of their potential impact on reproductive health and fertility of dairy cows.

Vitamins A and E – The Immune System, Health, and Reproduction

The incidence of diseases and disorders can have a negative impact on reproductive performance. In a study involving 2087 cows, those that had clinical mastitis during the first 45 d postpartum were at 2.7 times greater risk of abortion within the next 90 d compared to those without mastitis (Risco et al., 1999). Coliform organisms that can cause mastitis liberate lipopolysaccharide endotoxin which in turn can cause an inflammatory response by the cow so that she releases PGF$_{2\alpha}$. High enough levels of PGF$_{2\alpha}$ in the blood can result in luteolysis and therefore embryo loss. Although mastitis-causing gram positive bacteria do not produce endotoxins, the peptidoglycans comprising their cell wall can elicit an inflammatory response by the cow as well. Cows having mastitis after their first AI required an extra AI for pregnancy, thus having more days open than those without mastitis (Barker et al., 1998). Incidence of mastitis occurring close to first AI resulted in lower pregnancy rates for those cows

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compared to cows without such untimely mastitis in the Netherlands (Loeffler et al., 1999). Also the ‘risk’ of pregnancy (odds ratio) was reduced if cows experienced displaced abomasum (0.25; P = 0.036), retained fetal membranes (RFM) (0.55; P = 0.004), and loss of 1 BCS (0.80; P = 0.007) but not milk fever (0.85; P = 0.12) (Loeffler et al., 1999).

Management and nutritional efforts that maintain a healthy immune system may reap benefits for reproduction. The periparturient period is a time of significant stress on the immune system (Goff and Horst, 1997). The nutritional effects on immunity have not received a lot of research attention although vitamins A (retinol) and E (α-tocopherol) may have received the most. Plasma concentrations of retinol, β-carotene, and α-tocopherol decreased by at least 50% from approximately 4 wk prepartum to the time of calving, to levels that may be considered below chronic deficiency concentrations (Michal et al., 1994). Additional supplementation around this time period may have benefits for improved immunity.

Vitamin A

Vitamin A is necessary for maintenance of skeletal muscle and epithelial tissue as well as for normal immune function, vision, growth, and spermatogenesis (NRC, 2001). The 1989 NRC’s published requirement for vitamin A was based upon studies conducted between 1937 and 1957 in which cows showed normal reproductive efficiency until the intake of β-carotene (72 IU of vitamin A) dropped below 0.18 mg/kg of body weight as supplied by prairie grass hay. Incidence of abortions and RFM increased when intakes dropped below that amount. Milk production averaged ~8000 lb in 292 d of lactation. Diets were very fibrous containing no starchy grains. It is reasonable to assume that today’s cows producing 3 to 4 times more milk and consuming lower fiber diets that result in greater ruminal destruction of retinol would require more dietary vitamin A. The new dairy NRC (2001) increased the daily vitamin A requirement from 76 to 110 IU/kg of body weight for both the dry and lactating cow for these reasons as well as for the potential for improvement in mammary gland health. A 650 kg cow supplemented at the current recommended guideline would consume 71,500 IU daily. Clinical signs of a vitamin A deficiency do not appear until plasma concentrations drop below 10 ug/dl; <20 ug/dl may indicate a subclinical deficiency (McDowell, 2000). Liver values above 2 to 3 ppm indicate adequacy status. However plasma concentrations are generally a poor indicator of vitamin A intake because the liver stores vitamin A and supplies vitamin A to the blood stream, carried by retinol binding protein. Relationships between concentrations of plasma retinol and immune status, mammary gland health, or reproduction have been weak (Weiss, 1998).

Vitamin A and immune system

The literature contains only limited documentation of improved immune responses and/or reduced incidence of clinical mastitis due to vitamin A supplementation. Cows supplemented with 120,000 IU/d of vitamin A starting 4 wk prior to calving date had an improved nonspecific, cellular host defense system than cows
given 0 IU/d, as evidenced by increased killing of S. aureus by blood polymorphonuclear neutrophils at wk 0 and 1 postpartum; however, supplementation did not affect mammary host defense (Michal et al., 1994). In another study in which the control cows were supplemented at 53,000 IU/d from 6 wk prior to dry off through 2 wk after dry off, increasing the vitamin A supplementation to 213,000 IU/d had no effect on neutrophil function on wk -6, 0, and 2 in relation to time of dry-off (Tjoelker et al., 1990).

Vitamin A and mastitis

In a study involving 326 Canadian Holsteins, blood samples were collected weekly from 1 wk before expected calving date to 1 wk postpartum (LeBlanc et al., 2004). Using logistic regression, authors determined that cows having a 100 ng/ml greater concentration of serum retinol the week before parturition were 2.5 times less likely to have mastitis (n=23) in the first 30 d postpartum (n=303 nonmastitic cows). Thirty Finnish herds showed no such relationship between serum vitamin A concentration and the incidence of clinical mastitis although the mean vitamin A concentration was a good bit higher than that of the Canadian cows (Jukola et al., 1996). Workers at Penn State did not report a significant positive benefit to mammary gland health by increased vitamin A supplementation when the control cows were supplemented also. Increasing the vitamin A intake from 50,000 to 170,000 IU/d from approximately the last 2 week of lactation through the first 6 weeks of a new lactation (120 to 140 d total) did not affect the number of new intramammary infections or the cases of clinical mastitis of Holstein cows (Oldham et al., 1991). However, production of 4% FCM was increased by 6.2 lb/d at the higher supplementation rate. All cows in this study had good concentrations of serum retinol even on the day of calving (>34.5 ug/dl).

Vitamin A and reproduction

Vitamin A is clearly present at the ovarian level and in steroidogenesis. Higher vitamin A concentrations are found in non-atretic follicles and this might indicate a role of vitamin A in follicular development (Schweigert and Zucker, 1988). The synthesis of progesterone was depressed markedly in vitamin-deficient compared to normal rats (Jayaram et al., 1973). It is known that vitamin A influences the Cholesterol Side Chain Cleavage Enzyme (CSCCE) that converts cholesterol to pregnenolone (Ganguly et al., 1980) and also the enzyme $\Delta^{5}$-3$\beta$-hydroxyesteroid dehydrogenase that converts pregnenolone to progesterone (Islabão, 1982). When incubated in vitro with retinol, bovine luteal cells had a 3 to 10 fold increase in progesterone concentration over controls (Talavera and Chew, 1987). Although based upon very limited studies, it appears that supplying vitamin A in amounts much above NRC (2001) recommendations has not benefited reproductive performance of cows bred normally. Increasing supplemental vitamin A (100,000 or 1 million IU/d) to cows (n=78) the first 120 d postpartum did not affect the number of days to first service (63 d) nor conception rates at first AI service (28%) although the estrus detection rate following prostaglandin treatment was greater for cows fed the higher amount of vitamin A (60 vs. 26%) (Tharnish and Larson, 1992). A follow up study (n=52) using the same dietary
treatments failed to find any reproductive benefit for cows consuming 1 million IU/d nor were circulating progesterone concentrations changed. When cows undergo superovulation, additional vitamin A has proved beneficial. Shaw et al. (1995) reported that vitamin A (retinol palmitate) injection at 1 million IU at the first superovulatory dose of FSH increased the number of transferable embryos (5.87 vs. 3.13) in comparison to a control group injected with a placebo solution. The total number of embryos was not affected by vitamin A injection (11.1 vs. 8.2 for vitamin A and control groups, respectively). Amaral (2004) injected four different amounts of vitamin A (0, 500,000, 1,000,000 and 1,500,000 IU of retinol palmitate) into donor nonlactating *Bos indicus* cows (n = 64) grazing brachiaria forage and supplemented with millet silage and concentrate without vitamin A supplementation. Injections were given along with the first superovulatory injection of FSH. The number of viable embryos recovered in the group given vitamin A increased (3.6 vs. 6.1, 6.5 and 6.7 for 0, 500,000, 1,000,000 and 1,500,000 IU of retinol palmitate, respectively). An increase in the number of viable embryos recovered also was reported when supplementing a source of β-carotene to donor nonlactating dairy cows (n = 33) fed a TMR without a vitamin A supplement (Amaral et al., 2001). Nonlactating cows were supplemented with 3 kg (as-fed) of pumpkin per day during 20 days before flushing. Cows that consumed pumpkin produced more (P < 0.02) viable embryos than the control group (6.4 vs. 5.3). Supplemental β-carotene or animal conversion of β-carotene in pumpkin into vitamin A possibly improved embryo quality. Unfortunately the vitamin A status of the control cows was not determined in any of these studies.

**Summary**

Evidence is lacking to support the supplementing of vitamin A above NRC (2001) recommendations in order to reduce the incidence of mastitis and, in turn, improve pregnancy rate. However superovulated cows may produce a greater number of healthy transferable embryos if injected with vitamin A.

**Vitamin E**

Vitamin E is a lipid soluble cellular antioxidant having important roles in maintenance of cellular membranes, immunity, and reproduction (NRC, 2001). The form that is most common in feeds and is most biologically active is α-tocopherol. Unlike vitamin A, it is not thought to be degraded by ruminal microorganisms. A specific requirement for vitamin E has not been defined yet because titration studies are lacking. The recommended rate of supplemental vitamin E is 1.6 and 0.8 IU/kg of body weight for pregnant dry cows and lactating cows, respectively. A 650 kg cow supplemented at the recommended guideline of the 2001 Dairy NRC would consume daily ~1000 IU prepartum and ~ 500 IU postpartum. Cows fed fresh forages will require less supplemental vitamin E than this. Unlike plasma retinol concentrations, plasma α-tocopherol concentrations do reflect vitamin E intake. Based upon optimizing neutrophil function and minimizing clinical mastitis, the minimal acceptable concentration of plasma α-tocopherol for the dairy cow within a day or two after calving is 3 to 3.5 ug/ml
Cows at later stages of lactation may have a different minimal acceptable concentration as they may be under less immunological stress. The ratio of α-tocopherol to cholesterol in blood may be a better indicator of a cow’s vitamin E status because α-tocopherol is transported by lipoproteins.

Increased supply of vitamin E to cell membranes may improve immune function by protecting neutrophils from oxidative damage following their intracellular killing of ingested bacteria. Neutrophil function in blood was improved in cows 1) fed 500 IU/d for 30 d postpartum compared to unsupplemented cows, 2) fed 3000 IU/d from 8 wk prepartum to 4 wk postpartum compared to unsupplemented cows, 3) fed 3000 IU/d from 4 wk prepartum to 8 wk postpartum & injected with 5000 IU at 1 wk prepartum, and 4) injected with 3000 IU at 10 and 5 d before expected calving in blood collected at calving (supplementing 0 or 1040 IU/d had no effect) as reviewed by Weiss (1998).

Vitamin E & Retained Fetal Membranes (RFM)

If immune function is improved, then incidence of RFM and mastitis might decline. Indeed impaired neutrophil function has been reported to occur in cows having RFM (Kimura et al., 2002). A reduction in the incidence of RFM has been a consistent benefit of Se-sufficient cows fed supplemental vitamin E daily during the dry period (1000 IU/d, Miller et al., 1997; 740 IU/d, Harrison et al., 1984) compared to those not supplemented. Supplementing at 2000 IU/d was not superior to supplementing at 1000 IU/d starting at 14 d prepartum in reducing RFM (Baldi et al., 2000). Supplementing at 1000 IU/d the last 6 wk prepartum did not reduce RFM but the amount of vitamin E offered may not have been sufficient, as plasma α-tocopherol only averaged 1.15 ug/ml in the supplemented cows (Campbell and Miller, 1998). A one-time injection of a relatively small amount of vitamin E (700 IU) and 50 mg Se at ~21 d prepartum also reduced RFM (3 vs. 10.1%) in a large study (Arechiga et al., 1994) but not in other studies using one injection of similar small amounts (500 to 700 IU) (see Harrison et al., 1984 Introduction). One larger injection of 3000 IU of vitamin E at ~14 d prepartum reduced RFM (6.4 vs. 12.5%) and metritis (3.9 vs. 8.8%) in a 420 cow study (Erskine et al., 1997). Injecting 3000 IU at ~ 7 d prepartum tended to reduce the risk of RFM by ~44% in primiparous but not multiparous cows in a 1142 cow study (LeBlanc et al., 2002). Pregnant heifers may have benefited from the vitamin E injection more than pregnant cows because heifers consume less DM and therefore less vitamin E, they may not receive a vitamin fortified diet in transition, or they may take up vitamin E into tissues better due to less tissue mobilization compared to cows. From this same study but using a subset of cows (n=138), authors determined that for every 1 ug/ml increase in serum α-tocopherol prepartum, the risk of RFM decreased by 21%. However, one injection of 3000 IU of tocopherol acetate raised serum α-tocopherol only by 0.4 to 0.5 ug/ml (LeBlanc et al., 2004). In addition authors reported that there was no consistent threshold of circulating α-tocopherol that “neatly and repeatedly classifies cows as to risk of RFM.” This is not surprising since the cause of RFM is multifactorial including endocrine, nutrient, and immune factors (Goff and Horst, 1997).
Supplementing vitamin E at 1000 IU/d during the dry period has reduced SCC, clinical mastitis, and/or duration of clinical mastitis compared to control cows supplemented at 0 or 100 IU/day as reviewed by Weiss (1998) and Seymour (2001). However when Se status was suspect (plasma Se concentrations < 50 ng/ml), feeding 1000 IU/d did not improve mammary health. Even when intake of vitamin E was at 1000 IU/d prepartum and 500 or 1000 IU/d postpartum, mammary gland health was improved when intakes of vitamin E were increased to 2000 or 4000 IU/d (Baldi et al., 2000; Weiss et al., 1997). Weiss et al. (1997) reported that cows having a concentration of plasma α-tocopherol of < 3.0 ug/ml at calving were 9.4 times more likely to have clinical mastitis the first 7 d postpartum than those at > 3.0 ug/ml.

**Vitamin E and reproduction**

Cows and heifers fed 1000 IU of vitamin E daily for only 6 wk prepartum had fewer days to first observed estrus (42 vs. 62 d), to first AI (62 vs. 72d), and to pregnancy (113 vs. 145 d) compared to animals receiving no supplemental vitamin E (Campbell and Miller, 1998). Injecting 500 IU of vitamin E and 40 mg of Se reduced RFM (13.3 vs. 30%) and days to first AI (60 vs. 103 d) (Kim et al., 1997). Increasing vitamin E intake from 1000 to 2000 IU/d from 2 wk prepartum to 1 wk postpartum reduced the number of days open (84 vs. 111 d) and the number of AI per conception (1.3 vs. 2.2) (Baldi et al., 2000). Pregnancy rate and concentration of serum α-tocopherol were highly positively correlated in beef heifers. Pregnancy rate was not improved once serum α-tocopherol exceeded 3 ug/ml (Laflamme and Hidiroglou, 1991).

**Summary**

Cows in good Se status and supplemented with vitamin E at or above the dairy NRC (2001) guidelines show improved immune status and reduced incidence of RFM compared to unsupplemented cows. Giving a one-time injection of 3000 IU at 7 to 14 d before expected calving date reduced the incidence of or the risk of RFM, with only heifers benefitting in one study. Supplementing with vitamin E at NRC (2001) rates or at 2 to 4 times the NRC (2001) rates reduced mammary gland infections. Plasma concentrations of α-tocopherol may be a reliable indicator as to whether cows will reap a health or reproductive benefit from supplemental vitamin E.

**Supplemental Fat Feeding**

The supplementing with some sources of fat to lactating dairy cows has improved reproductive performance. In several studies, lactating cows fed a basal diet containing whole cottonseed (~9% C18:2) and further supplemented with Ca salts of palm oil (CaPO) (~8% C18:2) experienced a better rate of conception or pregnancy than cows fed the diet containing only whole cottonseeds (Staples et al., 1998). Lactating cows fed tallow (4.3% C18:2) 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). Grazing dairy cows...
supplemented with soybean oil soapstock (53% C18:2) at ~2% of dietary DM experienced a greater pregnancy rate than controls (62.5 vs. 22.2%) whereas those fed fat and housed in a free stall barn had lower pregnancy rates than controls (0 vs. 22.2%) (Boken, 2001). Primiparous beef heifers also have experienced greater pregnancy rates (94, 90, 91, and 79%) from being fed rolled and cracked safflower seeds, soybeans, or sunflower seeds, all high in C18:2 concentration (Bellows et al., 1999). The inclusion of fish meal in the diet also has stimulated fertility in several studies (n = 4) (Staples et al., 1998). The oils in the fish are hypothesized to be responsible for this positive response, hence their inclusion in the current discussion. What accounts for this improved fertility of cows supplemented with fat?

**How Might the Feeding of Additional Fat Improve Fertility?**

Some have suggested that the feeding of additional energy in the form of fat reduces the cow’s negative energy status so that she returns to estrus earlier after calving and therefore conceives sooner. However, the energy status of cows supplemented with fat is unchanged most of the time because of a nonsignificant depression in feed intake and/or an increase in milk production (Table 1; Staples et al., 1998). In fact, dairy cows fed tallow at 3% of dietary DM had a greater pregnancy rate despite having a more negative calculated mean net energy status from weeks 2 to 12 postpartum compared to controls (Son et al., 1996).

A second hypothesis is that cows fed fat have higher circulating concentrations of progesterone, a hormone necessary for the implantation and nutrition of the newly formed embryo. Progesterone is called the hormone of pregnancy; that is, progesterone is continually synthesized during pregnancy. The corpus luteum formed by the ovulated follicle remains on the ovary throughout pregnancy and is responsible for synthesizing progesterone. Increased concentrations of plasma progesterone have been associated with improved conception rates of lactating ruminants (Butler et al., 1996). A number of studies have reported that dairy cows fed supplemental fat (tallow, CaPO, prilled fatty acids, or whole cottonseeds) had elevated concentrations of blood progesterone (Staples et al., 1998). This may result from a reduced clearance of progesterone from the blood or an increased production by larger or a more productive corpus luteum. Feeding fat often increases the size of the dominant follicle (Staples et al., 1998). In addition, concentrations of progesterone were higher in follicular fluid of ruminants fed supplemental fat (Staples et al., 1998). In summary, fat supplementation can increase the concentration of fat, cholesterol, and progesterone in blood and ovarian structures of ruminants as well as increase the size of ovulating follicles. Improved fertility may result from more progesterone being available to improve embryo survival and health of fat-fed cows.

A third explanation of improved fertility of cows supplemented with fat is that specific individual long chain fatty acids found in some fats inhibit the production or release of prostaglandin F$_2$ (PGF$_2$) by the uterus. This prevents the regression of the corpus luteum on the ovary so that the newly formed embryo survives. The omega-3 long chain, polyunsaturated fatty acids may exert their effect in this way; namely
Linolenic acid (C18:3), eicosapentaenoic acid (EPA, C20:5) and docosahexaenoic acid (DHA, C22:6). All three fatty acids have a double bond located between the third and fourth carbon counting from the methyl end of the molecule, thus are classified as omega-3 fatty acids. These latter two fatty acids are found in marine products such as algae, fish meal, fish oil, and some seafood byproducts. Linolenic acid is the main fatty acid found in some vegetable oils such as linseed and in pasture forages.

Linolenic acid may have been responsible for the improvement in conception rate (87.5 vs. 50.0%) of lactating dairy cows (n = 35) fed formaldehyde-treated whole flaxseed (17% of dietary DM) compared to those fed CaPO (5.6% of dietary DM) from 9 to 19 weeks postpartum (Petit et al., 2001). Supplementing diets of lactating dairy cows with fish meal has improved conception rates (Staples et al., 1998). First service conception rate tended to be greater (P = 0.14) for lactating primiparous beef cows (n=82) fed fish meal compared to corn gluten meal (75.6 vs. 61.5%) (Bonnette et al., 2001). Serum progesterone concentrations after insemination were similar between the two groups of cows.

The synthesis of PGF2α is from arachidonic acid (C20:4) and is regulated by the key enzyme, prostaglandin endoperoxide synthase (PGHS) (Figure 1). The feeding of C20:5 may aid in the suppression of synthesis of PGF2α by the uterus by competing for PGHS. Dihomo-γ-linolenic acid also can compete for PGHS when it is converted to the series one prostaglandins. Although C22:6 is not a substrate for PGHS, it is a strong inhibitor of PGHS activity. Therefore when intake of C18:3, C20:4, or C22:5 increases, conversion of C20:4 to PGF2α can be reduced, thus potentially increasing the chances of preserving the life of a newly formed embryo. In addition, the increased presence of C20:5 and C22:6 can inhibit the synthesis of C20:4 from C18:2 by inhibiting the desaturation and elongation enzymes required for that conversion (Figure 1). Linolenic acid also can compete with C18:2 for the desaturase enzymes so that more C20:5 and less C20:4 are synthesized (Figure 1). In addition, the omega-3 fatty acids can displace C20:4 in the phospholipids of cell membranes thus reducing availability of C20:4. Therefore increasing the dietary intake of the omega-3 fatty acids can potentially reduce the production of PGF2α. Evidence supporting this mechanism is a slower regression of the corpus luteum in cows fed fish meal (Burke et al., 1997) and a reduced response of the uterus to secrete PGF2α by lactating dairy cows fed fish meal (Mattos et al., 2002) and by periparturient dairy cows fed fish oil (Mattos et al., 2004). If the omega-3 fatty acids are performing as described, embryo survival should be increased. Holstein cows (n = 141) were allotted to one of three dietary treatments initiated at calving (Petit and Twagiramungu, 2002). Diets were isonitrogenous, isoenergetic, and isolipidic. Diets contained whole flaxseed, CaPO, or micronized soybeans. Flaxseeds are ~32% oil of which 57% is C18:3, 14% is C18:2, and 18% is C18:1. The diameter of the CL of the cows fed flaxseed was larger than that of cows fed soybeans (19.7 vs.16.9 mm) but not larger than that of cows fed CaPO (17.5 mm). Embryo mortality from day 30 to 50 after AI tended to be lower (P < 0.11) when cows were fed flaxseed (0%) compared to CaPO (15.4%) or soybeans (13.6%).
A fourth reason offered is that supplemental fats are alleviating an essential fatty acid (EFA) deficiency (linoleic acid [C18:2] and C18:3) of the modern high-producing dairy cow. Deficiencies of EFA have reduced reproductive performance of nonruminants. Using the recent fat sub-model developed for use in the CPM-Dairy model, Sanchez and Block (2002) suggested that the amount of C18:2 excreted in 100 lb of milk daily exceeds the post ruminal uptake from typical diets. Therefore fat sources that supply additional EFA may minimize the need to mobilize EFA from tissues, thus protecting their functional integrity. Cows fed a calcium salt of fatty acids containing 30% linoleic acid (Megalac-R®, Arm and Hammer, Princeton, NJ) tended to have better first service conception rates than cows not fed a fat supplement (58.1 vs. 27.8%) (Cullens et al., 2004). According to the scientific literature dealing with human and lab animal nutrition, a ratio of C20:3 to C20:4 in tissues/serum that exceeds 0.4 is indicative of a C18:2 deficiency or an imbalance of C18:2 to C18:3. If the ratio of C20:3 to C20:5 exceeds 0.4, a deficiency of C18:3 is suspected. The rational behind this ratio is that the synthesis of C20:3 n-9 from oleic acid increases when C18:2 or C18:3 are deficient. It might be productive if these same ratios could be relevant to identify situations, if any, in which supplemental EFA would benefit the bovine.

Lastly, an improved fertilization rate and embryo quality may also result when lactating cows are supplemented with select fat sources. Dairy cows supplemented with a calcium salt blend of linoleic acid and monoenoic trans fatty acids or a calcium salt of palm oil (Bioproducts, Inc. Fairlawn, OH) from 25 d before calving through ~55 d postpartum were timed AI and flushed 5 d after AI with recovered structures evaluated (Santos et al., 2004). Cows fed the linoleic acid and monoenoic trans fatty acids tended to have (P = 0.11) a greater fertilization rate (87 vs. 73%), had more accessory sperm per structure collected (34 vs. 21), and tended to have (P = 0.06) a greater proportion of embryos classified as high quality (73 vs. 51%). In an accompanying study, conception rate at first AI was greater for cows fed the linoleic and trans acid salt (38.9 vs. 25.9%).

Sources of Fat Supplements

Only calcium salts of long chain fatty acids and fish meal have been evaluated in repeated studies for their reproductive effects, both having improved pregnancy or conception rates in a limited number of studies. The unique fatty acids in fish meal may be responsible for enhanced fertility. Animal tallow, flaxseed, safflower seeds, soybeans, sunflower seeds, and oil originating from soybeans have proven beneficial for ruminants in even more limited work. Obviously more studies are needed with these fat sources. If linoleic acid is a limiting fatty acid postruminally, then fat sources containing high concentrations of this fatty acid (e.g. soybeans and Megalac-R, Arm and Hammer Nutrition, Princeton, NJ), would be a good choice. Soybeans appear to deliver more linoleic acid to the small intestine than cottonseeds. Roasting of soybeans may be an effective way to reducing biohydrogenation in the rumen, thus increasing the delivery of EFA to the small intestine for absorption.
Evidence is accumulating that the design and delivery of supplemental unsaturated fatty acids to the lower gut for absorption (specifically linoleic acid, linolenic acid, EPA, and DHA) may target reproductive tissues to improve reproductive function and fertility. Improvement in pregnancy may be associated with improved embryo survival due to increased production and/or decreased clearance of progesterone as well as the suppression of uterine prostaglandin secretion by omega-3 fatty acids. Further work is needed to determine if the modern high-producing dairy cow is in a negative EFA balance.

Summary

Dietary nutrients not only affect productive but also reproductive performance. Supplying sufficient amounts of vitamins A and E may improve the immune status of the periparturient cow thus reducing the incidence of mastitis and/or retained fetal membranes, which in turn may improve pregnancy rates. Based upon a limited number of studies, the current feeding recommendation (dairy NRC, 2001) for vitamin A (110 IU/kg body weight) appears sufficient whereas that for vitamin E (1.6 IU and 0.8 IU per kg of body weight for pre- and postpartum cows, respectively) may be conservative in situations where plasma concentrations of α-tocopherol are <3.0 to 3.5 ug/ml. The recovery of healthy embryos may improve from cows undergoing superovulation if vitamin A is injected. Evidence is accumulating that the design and delivery of supplemental unsaturated fatty acids to the lower gut for absorption (specifically linoleic acid, linolenic acid, EPA, and DHA) may target reproductive tissues to improve reproductive function and fertility. It is unclear whether these improvements are mediated through the endocrine system, by alleviating an EFA nutrient deficiency, by changing the phospholipid composition of membranes, or by some other avenue.

References


Cow Symp. XVI. Greeley, CO. Coop. Ext. Serv. of CO, SD, WY, and NE.

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**Table 1.** Effect of fat on performance factors related to energy status in studies reporting improved fertility due to feeding of tallow (Son et al., 1996) or calcium salts of long chain fatty acids (all other references).

<table>
<thead>
<tr>
<th>Reference</th>
<th>DM intake, lb/day</th>
<th>Fat corrected milk, lb/day</th>
<th>Body weight or energy status (ES)</th>
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<tbody>
<tr>
<td>Son et al., 1996</td>
<td>↓ 2.6</td>
<td>↑ 1.3</td>
<td>More negative ES</td>
</tr>
<tr>
<td>Sklan et al., 1991</td>
<td>↓ 0.2</td>
<td>↑ 3.7</td>
<td>↑ loss of weight</td>
</tr>
<tr>
<td>Scott et al., 1995</td>
<td>N. R. ¹</td>
<td>↑ 2.9</td>
<td>No change</td>
</tr>
<tr>
<td>Garcia-Bojalil et al., 1998</td>
<td>↓ 0.2</td>
<td>↑ 3.5</td>
<td>No change</td>
</tr>
<tr>
<td>Sklan et al., 1989</td>
<td>N.R.</td>
<td>↑ 3.1</td>
<td>↑ loss, ↑ gain weight</td>
</tr>
<tr>
<td>Schneider et al., 1988</td>
<td>↓ 2.0</td>
<td>↑ 6.4</td>
<td>↑ gain of weight</td>
</tr>
</tbody>
</table>

¹ N.R. = not reported

**Figure 1.** Synthesis of the various prostaglandin (PG) series from fatty acid precursors.

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**Omega-6 Family**
- Linoleic acid, C18:2
- γ-Linolenic acid, C18:3
- Dihomo-γ-linolenic acid, C20:3
- Arachidonic acid, C20:4

**Omega-3 Family**
- α-linolenic acid, C18:3
- Stearidonic acid, C18:4
- Eicosatetraenoic acid, C20:4
- Eicosapentaenoic acid, C20:5

Δ6 desaturase
Elongase
Δ5 desaturase

PG-1 series
PGH Synthase

PG-2 series
PGH Synthase

PG-3 series
PGH Synthase

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