Essential fatty acids in ruminant diets

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Introduction

The essentiality of the unsaturated fatty acids, linoleic and linolenic, was described by Burr and Burr in a series of papers from the late 1920’s to the early 1930’s (Burr et al., 1932). They are incorporated into membranes as such, and are precursors for other unsaturated fatty acids that are key for metabolic regulation and cell membrane function and are essential to life for all mammals. Progress on nutrition and physiology of essential fatty acids (EFA) was slow until the near simultaneous development of gas liquid chromatography to separate and quantify fatty acids rapidly (late 1950’s) and the discovery of prostaglandins (1964), and EFA as their precursors.

Chemistry of EFA

EFA are characterized by the position and number of cis double bonds in the molecule. Each cis double bond introduces a bend of 120° into the carbon chain, so that with increasing number of double bonds the chain becomes increasingly coiled. When fatty acid chains are incorporated into membranes, straight-chain saturated fatty acids pack tightly, whereas unsaturated fatty acids pack more loosely. More open packing decreases melting point and introduces flexibility into membranes. Structures and pathways for conversion of linoleic and linolenic acids to their very long chain fatty acid products are shown in Figure 1. Note that desaturation of the elongation products of both linoleic and linolenic acids is mediated by Δ-6 desaturase, and thus these fatty acids are competitive inhibitors of each other. The affinity of Δ-6 desaturase for the ω-3 fatty acids is higher, however. Also, high concentrations of either (or both) fatty acids, as well as increased concentrations of the long chain fish oil fatty acids, inhibit further chain elongation and desaturation of linoleic and linolenic acids to their eicosanoid products.

Physiology and metabolism

Although EFA may be stored in tissues as triacylglycerols, their key roles in metabolism are incorporation into membrane phospholipids and conversion to very long chain fatty acids and eicosanoids, commonly known as prostaglandins. For this brief discussion, the ω-6 linoleic acid is converted to arachidonic acid (AA), which is a precursor for the 2-series of prostaglandins, whereas the ω-3 linolenic acid is converted to eicosapentaenoic acid (EPA), a precursor of the 3-series of prostaglandins. Generally, physiological effects of the 2 and 3 series are antagonistic. Also, EPA can
be elongated and desaturated to docosahexaenoic acid (DHA), which is not a precursor for prostaglandin synthesis, but is an important component of membranes.

The EFA and eicosanoids are important regulators of physiological functions and gene expression (Figure 2), the latter by binding to various regulatory molecules associated with genes. Arachidonic acid metabolites PGE2 and the 4-series of leukotrienes, among others, are generally proinflammatory, inducing fever, pain, vasodilation and vascular permeability, whereas EPA metabolites have opposing or lesser effects on these processes.

Deficiency symptoms and requirements for EFA

Calves that were fed fat-free diets exhibited overt symptoms of EFA deficiency, though specific deficiencies were not demonstrated. These symptoms are associated with impaired functions of membranes, such as rough hair coat, alopecia, dry, scaly skin, dermatitis, excess water loss, etc., and impaired nervous function (Figure 2; Cunningham and Loosli, 1954a,b; Lambert et al., 1954). Deficiencies associated with eicosanoid effects on physiological functions or regulation of gene expression are more subtle. There is no defined EFA requirement for ruminants. Holman (1960) reported a biochemical definition for EFA deficiency as a ratio of eicosatrienoic acid:eicosatetraenoic acid in blood erythrocyte membranes of >0.4. In EFA adequate conditions, linoleic acid provides the eicosatetraenoic acid (arachidonic acid, AA, 20:4ω-6) needed for membrane synthesis. In absence of adequate linoleic acid, the non-essential ω-9 oleic acid is desaturated and elongated to form eicosatrienoic acid (20:3ω-9, Mead acid), which is incorporated into membranes. Holman (1960) observed that when the ratio of these fatty acids in phospholipids of young rat plasma or red cell membranes exceeded 0.4, that overt EFA symptoms became apparent. When plotted graphically, deficiency symptoms appeared when linoleic acid was < 2% of energy intake. The triene/tetraene ratio in blood phospholipids has become the accepted standard for defining EFA deficiency in various species.

Further research by Holman’s group showed that female rats have 1.3 to 1.6 times more unsaturation of fatty acids in tissues than male rats; despite the greater unsaturation, female rats consistently required lower linoleic acid to meet requirements, 0.5% of energy vs 1.3% of energy for males. This was found to be a true sex difference; castrated males supplemented with estrogen had requirements similar to females (Pudelkewicz et al., 1968). It is known (Williams and Burdge, 2006) that conversion of linolenic acid to DHA is upregulated in pregnant women, attributed to the effects of estrogen. Whether this occurs in ruminants or other mammals is not known. Pudelkewicz et al. (1968) observed also that the optimum range for intakes of linoleic and linolenic acids is rather narrow, as determined by decreased growth when intakes were excessive (for linoleic acid, 1.2% of energy for female rats), with no maximum determined for males, though growth decreased at high intakes. No maximum was suggested for linolenic acid, though it probably exists. The observation of optimum intake ranges is consistent with the fact that these two fatty acids utilize the same pathway of Δ-6 desaturation and chain elongation as they are converted to eicosanoic
fatty acids, and thus are competitive inhibitors of each other. The Δ-6 desaturase enzyme is rate limiting for such conversions (Holman, 1986; Sprecher et al., 2000).

Excessive intake of unsaturated fatty acids without adequate Vitamin E induces severe muscular myopathy in calves, similar to that observed in selenium deficiency (Blaxter, 1953; Adams et al., 1959).

Studies of EFA metabolism by Noble and coworkers showed that young lambs are born with a biochemical deficiency of EFA (20:3/20:4 >0.4) that was reversed by 8 days of suckling, and that 100% of linoleic acid consumed in dam’s milk during the first 3 weeks of suckling was retained in the body (Noble et al., 1972) so that the lambs were no longer deficient. In similar studies (Palmquist et al., 1977a) newborn lambs had a biochemical deficiency at birth that was reversed after one day of suckling. Apparently, allowing newborn ruminants to suckle for only a few days is sufficient to prevent symptoms of deficiency even when maintained on a fat free diet (Cunningham and Loosli, 1954a,b; Lambert et al., 1954). When we maintained wethers for 4 weeks on a high glucose, fat-free solution infused parenterally (Palmquist et al., 1977b) we failed to show any accumulation of 20:3 in the plasma, whereas similar infusions in humans initiate a biochemical deficiency within 3 to 7 days (Wene et al., 1975).

Whereas unsaturated fatty acids are preferentially oxidized in non-ruminants, Lindsay and Leat (1977) showed that linoleic acid is oxidized in sheep at less than 5% of the extent of palmitic, stearic and oleic acids. Labeled linoleic acid was incorporated into plasma phospholipids and cholesteryl esters to a much greater extent than was stearic acid. Further, on starvation linoleic acid was mobilized to a much lower extent than other fatty acids. Forty to 50% of the linoleic acid absorbed from the intestine is incorporated into the phospholipids of intestinal lipoproteins (Mattos and Palmquist, 1977), preserving it from oxidation or secretion into milk. Thus, by incorporating a large part of linoleic acid into phospholipids and cholesteryl esters, rather than non-esterified fatty acids and triacylglycerol, ruminants have the capacity to retain EFA very efficiently and survive on much lower EFA intake than non-ruminants. Because all the available data pertain to linoleic acid, one can only assume that they apply also to linolenic acid. Detailed studies of absorption, transport and metabolism of linolenic acid in ruminants would be very useful.

We recovered one-half of a bolus dose of radiolabeled linoleic acid in milk fat within 10 days of dosing lactating cows. Using estimated absorption of linoleic acid, we concluded that the cows retained sufficient linoleic acid to exceed requirements, compared on a metabolic body basis with young female rats at maintenance intake (Mattos and Palmquist, 1977). Roy (1980) concluded that young calves may require 1% of energy intake as linoleic acid.

EFA requirements for physiological functions

**Growth.** From literature cited above, there is no evidence that growing ruminants have a higher requirement for EFA than is provided in conventional diets.
Based on Pudelkewicz and Holman (1968) and applying the comparison of rats and ruminants used by Mattos and Palmquist (1977), growing animals require 88 mg of linoleic acid/kg body weight \( \frac{1}{4} \). Using this comparison, examples of requirements for various body weights are summarized in Table 2. Even considering that cell membranes of larger animals, as ruminants, contain a greater proportion of linoleic acid than those of small animals (rats), the amount available seems to be more than adequate to satisfy requirements (Table 3).

**Lactation.** For lactation requirements one must consider the amount of linoleic acid secreted in the milk. Ruminal biohydrogenation of dietary unsaturated fatty acids is relatively constant (86 and 82%, respectively, for linoleic and linolenic acids; (Jenkins and Bridges, 2007), whereas secretion of these in milk is more variable (30 to 60% of absorbed; (Chilliard et al., 2007; Moate et al., 2008). Regulation of the proportions of absorbed fatty acids secreted is mostly unknown and uncontrollable; whereas the amounts consumed are controlled by management. Thus, one must feed more unsaturated fatty acids to achieve greater absorption. Assuming 50% of absorbed EFA are secreted into milk (Palmquist and Mattos, 1978; LaCount et al., 1994), then one-half of absorbed EFA are available for oxidation and physiological functions. In a study of fatty acid content of diets in Ohio dairy herds (Timmons et al., 2001), linoleic acid constituted 1.3 to 2.6 % of dry matter in diets containing 0 to 15% of roasted whole soybeans, whereas linolenic acid content did not differ with soybean content (approximately 0.3% of diet dry matter). Calculated amounts available above milk secretion are in Table 3. High producing cows would ordinarily be fed some fat supplement such as whole cottonseed, whole soybeans or tallow that would increase the linoleic acid available above that indicated for zero roasted soybeans. Sources of linolenic acid are fresh forage or whole linseed. Sources of preformed EPA and DHA are fish oil or algae. These calculations suggest that lactating cows consume adequate amounts of EFA to support normal physiological functions. Research in human subjects suggests that consumption of greater amounts of n-3 fatty acids, especially those from fish oil, improve health status during periods of physiological stress, including cancer, heart disease, diseases of the immune system and surgery. It remains to be determined whether these fatty acids can improve the physiological status of dairy cattle.

In a continuing study at the University of Florida (Santos, personal communication, 2010) primiparous and multiparous cows were fed low fat diets (<2% fatty acids) or diets supplemented with 1.7% saturated (Energy Booster 100®) or 1.7% unsaturated fatty acids (Megalac®) for 60 days prepartum and continuing for 90 days postpartum. The unsaturated fat diet decreased dry matter intake of both primiparous and multiparous cows prepartum, but only of older cows postpartum. Post-partum energy balance (EB) of both groups of cows was improved by feeding saturated fatty acids, and unsaturated fatty acids showed an interaction, with improved EB of primiparous cows, and decreased EB in older cows. Whereas fat supplements caused differences in milk yields and fat percentages, no differences occurred in yields of energy-corrected milk among treatments. Among reproductive measures, preliminary
data showed that supplemental fat decreased rectal temperatures and puerperal metritis.

**Reproduction.** Often dietary fat influences the reproductive status of dairy cows, including increasing the number and size of ovulatory follicles, plasma concentration of progesterone, and decreasing the secretion of prostaglandin metabolite, resulting in increased lifespan of the corpus luteum and improved fertility (Staples et al., 1998). Many of these effects can be related to increasing intake of EFA, through effects on prostaglandin synthesis; this has led to increased research on the role and requirement for EFA in reproduction. To this time, data are not conclusive enough to define specific intake requirements for improved reproduction.

Two approaches have been taken—1) feeding increased amounts of fat in the dry period or early lactation, or both, to increase total blood lipids. Because the primary carriers of blood lipids are low- and high-density lipoproteins, higher blood lipids largely increase phospholipids and cholesteryl esters that are rich in unsaturated fatty acids and cholesterol. 2) targeting specific dietary fatty acids, linoleic or linolenic acids, or more commonly, the 20- and 22-carbon fatty acids of fish oil, EPA and DHA, respectively.

Higher cholesterol concentrations, as the precursor for progesterone synthesis, assure adequate synthesis of this key regulatory hormone, although research suggests that increasing blood lipid concentrations causes increased circulating progesterone by decreasing its clearance from the blood (Staples et al., 1998). Fouladi Nashta et al., (2007) synchronized estrous cycles in cows fed a silage-based diet supplemented with either low (200 g/day) or high (800 g/day) fat. Oocytes were collected, matured, fertilized, and cultured to the blastocyst stage in vitro. The high fat diet reduced numbers of small and medium follicles without effect on the quality of oocytes or cleavage rate, and improved blastocyst production from matured and cleaved oocytes. Blastocysts from the high fat group had significantly more total and inner cell mass and trophoderm cells than the low fat group. Higher milk yields were associated with reduced developmental potential of oocytes in cows given a low fat diet. Provision of a high fat diet buffered oocytes against these effects, resulting in significantly improved developmental potential.

Modifying intakes of ω-6 and ω-3 fatty acids regulates the balance of 2- and 3-series prostaglandins. Establishing pregnancy after fertilization requires continued progesterone secretion from the corpus luteum (CL); high concentrations of prostaglandin F2α cause regression of the CL, with loss of the conceptus before it is established in the uterus. Increasing the availability of linoleic or linolenic acids decreases synthesis of AA (Chagas et al., 2007), the precursor for PGF2α, in turn decreasing its synthesis. Also, higher linolenic acid increases synthesis of EPA, a precursor of the 3-series of prostaglandins that counteract the effects of PGF2α. Providing EPA directly by feeding fish oil has a similar effect. More subtle effects of EFA in these processes have been reviewed (Chagas et al., 2007; Wathes et al., 2007).
The fatty acid composition of different reproductive tissues varies (Adiamak et al., 2006; Wonnacott et al., 2009). Wonnacott et al., (2009) fed sunflower oil or linseed plus salmon oil to ewes for 6 weeks. Ovaries were harvested and oocytes were fertilized. Follicle number and size were unaltered by diet, but follicular-fluid progesterone concentrations were greater in n-3 than n-6 polyunsaturated fatty acids (PUFA) fed ewes. Though blastocyst formation was not influenced by treatment, development to the blastocyst stage and quality of embryos from ewes fed sunflower oil (n-6 treatment) was clearly inferior to those from the n-3 treatment. The content of fatty acid groups in plasma, granulosa cells and oocytes are summarized in Table 4. It is clear that tissues take up fatty acids selectively and that ovarian cells are much more saturated than most tissues. Whether dietary fatty acid availability for ovarian tissues is regulatory is not established. The experimental diets clearly influenced the proportions of n-3 and n-6 fatty acids in granulosa cells and oocytes, and correspondingly changed the n-3/n-6 ratios, which could influence proportions of prostaglandins in these tissues. Bilby et al., (2006) reported the fatty acid profiles of numerous tissues in cows fed diets containing whole cottonseed or calcium salts enriched with fish oil fatty acids. Endometrium from the cows fed fish oil had increased proportions of EPA and DHA, whereas AA was decreased. Such effects would be positive for maintaining low PGF$_2$α and other products of the PG2 series, which would maintain the CL and high progesterone concentration, resulting in improved possibility for implantation of the embryo.

An early return to ovarian activity post partum may not be desirable in dairy cows. PGF$_2$α is necessary in the post-partum process of resorption of the uterus and restoration of tissues for the next pregnancy. Absence of ovarian activity during this period appears to enhance the process of involution. Thatcher et al. (2006) suggested that a strategy of dietary supplementation with functional nutrients to increase availability of prostaglandins that would enhance general immunocompetence and neutrophil function could be an attractive means to manage uterine infections and subsequent infertility. Cows that developed endometritis had lower PGFM concentrations during the early post-partum period (i.e., 0–14 days postpartum) perhaps contributing to a reduction in neutrophil function that compromises the ability of the uterus to prevent and/or manage infections. After completion of uterine involution, sequential ovulations would be a goal towards normal fertility. To test these concepts, summarized in Thatcher et al., (2006), cows were supplemented for 28 days prepartum or only postpartum with a calcium salt of fatty acids containing 28% linoleic acid. Those cows supplemented prepartum had significantly higher PGF metabolite in serum for the first 12 days of lactation than cows in other groups (Figure 3) and fewer health problems in the first 10 days postpartum, whereas conception to first insemination after induced ovulation at 72 days was greater (P <0.09) for all supplemented groups. A caution must be noted: if supplementation of linoleic acid prepartum is excessive, desaturation and chain elongation to AA, the precursor of PGF$_2$α, will be inhibited.

A further concept of manipulating EFA availability in reproductive management is to feed a high linoleic fatty acid source from 28 days prepartum to 28 days postpartum, followed by a high n-3 source, preferably fish oil, from 28 to 100 days of lactation. The first would provide an environment as described above, whereas following with a high n-
source should decrease the 2-series and increase the 3-series of prostaglandins to improve the environment for embryo implantation and survival.

Collectively, these studies suggest that feeding fats enriched in selected unsaturated fatty acids, beginning in the dry period and continuing in the post-partum period, improves post-partum health and milk production, as well as the development of bovine embryos and subsequent pregnancy rates. The beneficial effects on reproductive responses may be due to a hastened restoration of the post-partum reproductive system to support embryo development (Thatcher et al., 2006).

**Modifying immune response.** Septicemia occurs in one-fourth to one-third of calves with acute diarrhea, and greater than one-half of these do not survive, attributed to an overaggressive systemic acute phase response that is characterized by a period of hyperinflammation, followed by immune paralysis caused by a strong counter anti-inflammatory response (Ballou et al., 2008). Because feeding fish oil had been reported to decrease the acute phase response and increase survival in several animal models, Ballou et al. (2008) postulated that supplemental fish oil may lead to a more balanced acute phase response, circumventing the hyperinflammation and immune paralysis response induced by LPS endotoxin injection in dairy calves. Injection of LPS caused a dramatic rise in respiratory rate; the overall effects were that adding fish oil to milk replacer attenuated many aspects of the acute phase response, and the effects were linear in the range of 5 to 10% of the lipid replaced as fatty acids from fish oil. Many of the effects may have been mediated by fish oil fatty acid modulation of tissue prostaglandin concentrations, though these were not reported.

**Other Interests**

Because of the increased interest for higher intakes of \( \omega-3 \) fatty acids, there is interest also to increase these in human foods. Approaches to accomplish this in animal products have been reviewed recently (Palmquist, 2009). In addition to supplementing animal diets with traditional sources high in \( \omega-3 \) fatty acids, new products are becoming available through gene engineering of plants (Whelan, 2009); several plants, including soybeans, have had genes introduced to increase the content of **stearidonic acid** (18:4\( \omega-3 \)). The limiting \( \Delta-6 \) desaturase enzyme is bypassed when stearidonic acid is the precursor for chain elongation to the eicosanoids; this fatty acid is four times as effective as linolenic acid as a precursor for EPA synthesis (James et al., 2003).

**Summary.** Linoleic and linolenic acids are essential for life of all mammals, functioning as components of membranes and as precursors for synthesis of the prostaglandins or for other very long chain fatty acids that function mainly in membranes. Thus, the EFA are essential both for cell structure and regulation. There are no direct measures of dietary EFA requirements for ruminants; however, research and observation suggest that typical diets provide amounts adequate for normal functions. There is no detailed information on metabolism of linolenic acid in ruminants; such research could prove to be fruitful. Evidence suggests that supplemental EFA may modulate reproductive and
immune functions positively. Research continues to refine feeding management of essential fatty acids to achieve improved reproductive performance.

References


Table 1. Overt symptoms of essential fatty acid deficiency in newborn ruminants.

<table>
<thead>
<tr>
<th>Symptoms</th>
<th>Notes</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Leg weakness, muscular twitches, death within 5 wk on synthetic diet.</td>
<td>Calves given colostrum 1-2 days —lard allowed survival</td>
<td>Cunningham and Loosli, 1954b</td>
</tr>
<tr>
<td>Growth retardation, scaly dandruff, alopecia, diarrhea at 8 wk of synthetic diet.</td>
<td>Calves—allowed one milk feeding after birth</td>
<td>Lambert et al., 1954</td>
</tr>
<tr>
<td>Leg weakness, listless, fluid retention, death at 5 – 7 wk on fat free diet. Survived with 0.25% lard added. “Weanling” lambs survived 7 mo on fat-free synthetic diet.</td>
<td>Lambs—0.25% lard reversed symptoms</td>
<td>Cunningham and Loosli, 1954a</td>
</tr>
</tbody>
</table>

Table 2. Linoleic acid required for growth and maintenance of ruminants, assuming requirement of 88 mg/kg body weight ⅓ (Mattos and Palmquist, 1977).

<table>
<thead>
<tr>
<th>Body weight, kg</th>
<th>Metabolic body size, kg⅓</th>
<th>Linoleic acid required, g/day</th>
</tr>
</thead>
<tbody>
<tr>
<td>50</td>
<td>18.8</td>
<td>1.6</td>
</tr>
<tr>
<td>100</td>
<td>31.6</td>
<td>2.8</td>
</tr>
<tr>
<td>200</td>
<td>53.2</td>
<td>4.7</td>
</tr>
<tr>
<td>300</td>
<td>72.1</td>
<td>6.3</td>
</tr>
<tr>
<td>400</td>
<td>89.4</td>
<td>7.9</td>
</tr>
<tr>
<td>500</td>
<td>105.7</td>
<td>9.3</td>
</tr>
<tr>
<td>600</td>
<td>121.2</td>
<td>10.7</td>
</tr>
</tbody>
</table>
**Table 3. a)** Calculated linoleic (18:2) and linolenic (18:3) acids available for metabolism (in excess of amount secreted in milk) in lactating cows at 20 and 25 kg/d intake of dry matter (DMI), and containing varying amounts of whole roasted soybeans (SB)\(^1\); **b)** Comparison with estimates from Staples et al. (1998).

<table>
<thead>
<tr>
<th>DMI, kg/d</th>
<th>Whole Roasted SB, % of DM</th>
<th>Absorbed, g/d</th>
<th>Available above milk, g/d</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>18:2</td>
<td>18:3</td>
</tr>
<tr>
<td>20</td>
<td>0</td>
<td>36.4</td>
<td>10.8</td>
</tr>
<tr>
<td></td>
<td>5</td>
<td>49.1</td>
<td>10.8</td>
</tr>
<tr>
<td></td>
<td>10</td>
<td>60.8</td>
<td>10.8</td>
</tr>
<tr>
<td></td>
<td>15</td>
<td>72.4</td>
<td>10.8</td>
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<tr>
<td>25</td>
<td>5</td>
<td>46.9</td>
<td>13.5</td>
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<tr>
<td></td>
<td>10</td>
<td>61.3</td>
<td>13.5</td>
</tr>
<tr>
<td></td>
<td>15</td>
<td>75.9</td>
<td>13.5</td>
</tr>
</tbody>
</table>

Assumptions: Biohydrogenation of 18:2 = 86%, and of 18:3 = 82% (Jenkins and Bridges, 2007). Secretion in milk is 50% of absorbed (LaCount et al., 1994). Fatty acid content of diets from Timmons et al. (2007).

**b)**

<table>
<thead>
<tr>
<th>Fat source</th>
<th>Amount fed, kg/d</th>
<th>Linoleic acid fed, g/d</th>
<th>Linoleic acid available for absorption from fat source(^1)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Whole cottonseed</td>
<td>2.8</td>
<td>300</td>
<td>30</td>
</tr>
<tr>
<td>Whole soybeans</td>
<td>2.8</td>
<td>300</td>
<td>30</td>
</tr>
<tr>
<td>Yellow grease</td>
<td>0.45</td>
<td>77</td>
<td>8</td>
</tr>
<tr>
<td>Tallow</td>
<td>0.45</td>
<td>23</td>
<td>2</td>
</tr>
<tr>
<td>Megalac</td>
<td>0.45</td>
<td>38</td>
<td>25(^2)</td>
</tr>
</tbody>
</table>

Table adapted from Staples et al., 1998.

\(^1\) Assumes 90% ruminal biohydrogenation of linoleic acid.

\(^2\) Assumes 33% ruminal biohydrogenation of linoleic acid.
### Table 4. Saturated and unsaturated fatty acids in plasma and some reproductive tissues

<table>
<thead>
<tr>
<th>Fatty acid group</th>
<th>Plasma (µg/ml)</th>
<th>Granulosa cells (µg/pellet)</th>
<th>Oocytes (ng/oocyte)</th>
<th>Dietary treatment¹, ²</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>n-3</td>
<td>n-6</td>
<td>n-3</td>
<td>n-6</td>
</tr>
<tr>
<td>Saturated</td>
<td>30.9</td>
<td>42.1</td>
<td>39.1</td>
<td>39.7</td>
</tr>
<tr>
<td>Unsaturated</td>
<td>59.3</td>
<td>49.4</td>
<td>49.7</td>
<td>52.1</td>
</tr>
<tr>
<td>MUFA</td>
<td>21.5</td>
<td>18.0</td>
<td>20.9</td>
<td>23.9</td>
</tr>
<tr>
<td>PUFA</td>
<td>37.8</td>
<td>31.4</td>
<td>28.9</td>
<td>28.2</td>
</tr>
<tr>
<td>n-6 PUFA</td>
<td>25.4</td>
<td>28.9</td>
<td>8.5³</td>
<td>24.1</td>
</tr>
<tr>
<td>n-3 PUFA</td>
<td>12.4</td>
<td>2.5</td>
<td>20.4</td>
<td>4.1</td>
</tr>
<tr>
<td>Ratio of n-6: n-3</td>
<td>2.1</td>
<td>11.5</td>
<td>0.4</td>
<td>6.2</td>
</tr>
</tbody>
</table>

Adapted from Wonnacott et al. (2009).

MUFA = monounsaturated fatty acids; PUFA = polyunsaturated fatty acids.

¹ 6.4% oil in the diet supplemented from linseed oil

² 5.7% oil in the diet supplemented from sunflower oil

³ Important effects due to dietary fatty acid are in bold

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**Figure 1.** Biosynthesis of Series-1, 2, and 3 Prostaglandins from essential fatty acid precursors. Modified from Wathes et al. (2007).
Figure 2. Nuclear mechanism for PUFA regulation of gene expression. FA = fatty acids, NF-Y = nuclear factor Y; PPAR = peroxisome proliferator-activated receptor; PPRE = peroxisome proliferator-activated response element; SP-1 = stimulatory protein 1; SREBP 1 = sterol regulatory element-binding protein-1; TG = triglycerides.

Figure 3. Effect of Megalac-® supplementation on plasma 13–14 dihydro, 15 keto-PGF2 (PGFM; ng/mL) between 2 and 14 days in milk (DIM). Lactating dairy cows did not receive Megalac-R1 supplementation (control; n = 23), received supplement beginning at 28 days prior to expected parturition (Fat Pre; n = 12) or received supplement at the time of parturition (Fat 1 DIM; n = 12). From Thatcher et al. (2006).