Effects of essential fatty acids on ruminant animal: A Review

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Abstract: Previous work on the relationship between chemical structure and biological activity of fatty acids was based on the theory that either the 6-term. (That is, a double bond between the 6th and 7th carbonatom counted from the terminal methyl group) or the 9-term. Double bonds or both were fundamental for essential fatty acid activity. By means of a new quantitative bioassay a number of fatty acids were tested and the results were in favors of the theory that both the 6-term. And the 9-term. Positions are essential. Although essential fatty acids (EFA) are not vitamins by definition, a deficiency disease or condition with dietary insufficiency does result, and in some ways, a similarity to vitamin deficiencies can be seen. The finding that components of fat, other than the fat-soluble vitamins, are dietary essentials is of nutritional and medical importance. Studies are reevaluating the beneficial effects of linolenic acid in species that previously were considered to need only linoleic acid as a dietary essential.

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Introduction

Although essential fatty acids (EFA) are not vitamins by definition, a deficiency disease or condition with dietary insufficiency does result, and in some were, a similarity to vitamin deficiencies can be seen. The finding that components of fat, other than the fat-soluble vitamins, are dietary essentials is of nutritional and medical importance. Excellent re- views in the literature of EFA have been prepared by Holman [26, 27 and 35] and Hansen [20, 35].

Knowledge that carbohydrates can be readily converted into fat and that essential lipid constituent such as phospholipids and cholesterol can be made in the body led to the view that dietary lipids were not required.

Evans and Burr [35, 15], changed this viewpoint by reporting that the total deprivation of fat in the diet of rats induced a syndrome of deficiency that could be corrected by certain components of fat. The EFAs originally included linoleic, linolenic, and arachidonic acids. However, arachidonic was later found to be synthesized from linoleic acid. Most species have a dietary requirement for linoleic acid, while others (e.g., fish) require linolenic acid. Studies are reevaluating the beneficial effects of linolenic acid in species that previously were considered to need only linoleic acid as a dietary essential

CHEMICAL STRUCTURE

Chemical structures of linoleic, linolenic, and arachidonic acids as well as other fatty acids associated with EFA, are shown in Fig1. There are three common families of unsaturated 18-carbon fatty acids and one family of unsaturated 16-carbon fatty acids. The exact structure of an unsaturated fatty acid is given by three numbers: (1)the number of carbon atoms in the chain, (2) the number of double bonds, and (3) the omega (ω) number, which indicates the number of carbon atoms from the terminal methyl group to the carbon atom of the first double bond. The omega system, which was originated by Holman, designated those unsaturated fatty acids belonging to each series. Another system substitutes n for ω (e.g., n-6 versus ω-6). The ω-9 and ω-7 series can be derived from endogenously synthesized oleic acid (18:1ω-9) and palmitoleic acid (16:1ω-7), respectively. The ω-6 series is derived from linoleic acid (18:2ω-6) and the ω-3 series from linolenicacid (18:3ω-3). These latter two fatty acids are considered essential as they are products of plants and cannot be synthesized by animals. Thus, it appears that linoleic acid (18:2ω-6) is essential for most species because of the inability of animals to synthesize a double bond between carbons 6 and 7 counting from the terminal methyl group. The polyunsaturated fatty acids are liquids at room temperature. Double bonds of natural fatty acids would normally be found in nature as the cis-form. Ruminant animals ingest unsaturated fatty acids with their plant foods. Subsequently, bacteria in the rumen use these

unsaturated fatty acids as acceptors for excess hydrogen produced during bacterial anaerobic fermentation, thus resulting in saturation of the fatty acids. However, in some cases, the end product is not a saturated bond but an unsaturated one with a different configuration—*Trans* instead of *cis*—or in a different position, or both. As a consequence, the body and milk fat of ruminants contain trans-fatty acids, about 2 to 9% in the case of butterfat. A similar process, which uses hydrogen gas and a nickel catalyst, is employed industrially to turn liquid edible oils into solid fats. During the hydrogenation process, some of the naturally occurring *cis*-double bonds are isomerized to *trans*-conformation, resulting in decreased bond angle and an acyl chain resembling a saturated fatty acid [34, 35].

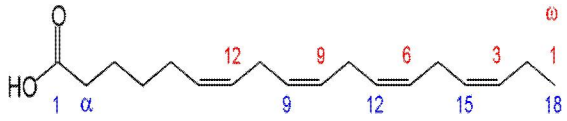

Fig1: Structures of essential fatty acids and other unsaturated fatty acids. [35].

Hydrogenation also results in the saturation of a portion of the existing double bonds in the fatty acyl chain, thereby decreasing the polyunsaturated fatty acid (PUFA) and increasing the saturated and monounsaturated fatty acid content of the fat. The *trans*-fatty acid content of commercial edible fats may vary from 0% for diet margarines high in linoleic acid to more than 50% of fatty acids for certain shortenings and frying fats [28, 35]

One school of thought regarding trans- versus *cis*-fatty acids is that dietary substitution of vegetable fats for animal fats reduces the risk of cardiovascular heart disease (CHD). However, all forms of vegetable fat are not alike, and research indicates that consumption of hydrogenated rather than hydrogenated vegetable oils may negatively influence plasma lipids and risk of CHD. Low-density lipoprotein (LDL) cholesterol was raised by both *trans*-fatty acids and saturated fatty acids compared to a diet high in oleic acid [28, 35]. Also, *trans*-fatty acids depressed high-density lipoprotein (HDL) cholesterol, the so called good cholesterol," whereas saturates did not have this effect.

New forms of linoleic acid, conjugated linoleate acid (CLA) have received considerable attention as chemo preventive agents since they have been shown to inhibit rat mammary tumorigenesis, mouse fore stomach neoplastic, and mouse skin carcinogenesis [2, 42, 35]. These compounds are linoleic acid derivatives with cis-9, Trans-11-; *trans*-9, *cis*-11-; *trans*-9, *Trans*-11-; *trans*-10, *Trans*-12-; and *trans*-10, *cis*-12-octadecadienoic acids accounting for the major isomers. The CLAs are found predominantly in foods from ruminants, first identified as an anticarcinogen following isolation from grilled ground beef extracts [19].

Linoleic acid is colorless oil that melts at -12^{O}C. It is soluble in ether, absolute alcohol, and other fat solvents and oils. It has an iodine value of 181 and a molecular weight of 280.44. Arachidonic acid is oil that melts at -49.5^{O}C, has an iodine value of 333.5, and has a molecular weight of 304.46 [44, 35].

METABOLISM and FUNCTION

Fats and fatty acid metabolism in relation to digestion, absorption, and excretion are discussed elsewhere [14]. Fatty acid-binding proteins (FABPs) have been identified as a family of cytosolic proteins found in heart, liver, and epithelial cells lining the small intestine [1]. FABPs are believed to be integrally involved in the cellular uptake as well as intracellular transportand/or compartmentalization of fatty acids. It has been postulated that the intestinal FABPs may actually participate in cellular fatty acid transport across the intestinal mucosa, as well as in selected intracellular events. In humans, placental transfer of fat to the fetus occurs late in pregnancy. The late second trimester or early third trimester fetus has only 1.7% of its body weight as fat, compared with 15% in the full-term infant [46]. After fat absorption in mono gastric animals, fatty acid composition of body fat is directly related to fatty acid composition of the diet. In ruminants, however, polyunsaturated fatty acids are hydrogenated to a large extent by ruminal microorganisms, resulting in more saturated body fat of the animal. In all species, certain fatty acids form structural components and serve indispensable biochemical functions. Members of a particular family may be metabolically converted to more proximally unsaturated (toward the carboxyl group) or chain-elongated fatty acids, but no con- version from one ω family to another occurs in mammals. For example, linoleic acid (18:2ω-6) is converted to

arachidonic acid (20:4ω-6) in animals, and linolenic acid (18:3ω-3) may be converted to eicosapen-taenoic (20:5ω-3) and docosahaexenoic acid (22:6ω-3). Members of the ω-6 and ω-3 families are considered essential fatty acids for mammals, because they cannot be synthesized de novo.

Linoleic acid and linolenic acid are the precursors of the entire ω-6 and ω-3 families of polyunsaturated fatty acids, respectively. All members of the ω-6 and ω-3 families are active as essential fatty acids, and many have been shown to be more active than their original precursor. Studies employing graded dose levels of arachidonic acid fed to rats have revealed that the deposition of arachidonic acid in liver is greater when arachidonic acid itself is fed in the diet than when linoleic acid is fed. This indicates that the conversion of 18:2ω-6 acid to arachidonic acid for deposit in tissue lipids is a less efficient process than the deposition of dietary 20:4ω-6 acid directly into tissue lipids, and that the potency of 20:4ω-6 is greater than that of 18:2ω-6 [26, 35].

The same phenomenon has been observed in a large variety of tissues and species, the differences being mainly in magnitude [25, 26, and 35]. Changes of greatest magnitude have occurred in heart and liver lipids. At zero intake of linoleic acid, the major differences in fatty acid composition are in the polyunsaturated acids themselves. In linoleic acid deficiency, 18:2ω-6, 20:4ω-6 and 22:5ω-6 is much lower than found in normal animals. Palmitoleic acid, 16:1ω-7, and oleic acid, 18:1ω-9, are higher than normal, but the most striking increase is in 20:3ω-9, which is formed endogenously from oleic acid. This acid, which is a normal component of tissue lipids in trace amounts,increases very dramatically in linoleic acid deficiency. It is found in the phospholipids in the 2 position, the same position in which arachidonic acid, 22:5ω-6 and other polyunsaturated acids are normally found. Similar studies have been made with graded dose levels of linolenic acid as the sole fatty acid supplement to a fat-free diet [36, 35]. Supplementation of the fat-free diet with 18:3ω-3 causes dramatic increases in 20:5ω-3, 22:5ω-3, and 22:6ω-3 in comparison with the amounts found in the lipids of fat-deficient animals. [15, 35] noted that polyunsaturated fatty acids in muscle phospholipids from rats fed high ω-6 or ω-3 fatty acid diets reflected the composition of their respective diets.

Studies on dose of a single fatty acid versus response of several fatty acids in tissues have shown that each family of fatty acids suppresses metabolism of other families of fatty acids [22, 23 and 35].

In the absence of the main ω-6 (linoleic) and ω-3 (linolenic) families in the diet, animals are capable of synthesizing some polyunsaturated acids from endogenous precursors. Both oleic (18:1ω-9) and palmitoleic (16:1ω-7) acids themselves, and their respective families, are enhanced in the tissue lipids. None of these polyunsaturated acids is fully efficacious in meeting the requirement for polyunsaturation, for although they are present in enhanced quantity in linoleic acid deficiency, the animals often die. The enzymatic systems that perform chain elongation, desaturation, and insertion of fatty acids into various lipid molecules apparently handle all groups of fatty acids, for there is competition between substrates at every step in each of these processes. The ω-3 family effectively suppresses metabolism of the ω-6 family. Likewise, the ω-6 family is able to suppress metabolism of the ω-3 family, but less effectively. The ω-6 family, however, suppresses the formation of polyunsaturated acids from oleic acid, as is manifested in linoleic acid deficiency. Ability of the precursor acids to compete for these enzyme systems is in the order linolenic> linoleic > oleic.

Depending on animal species, different EFAs are not equal in relationship to requirements or in ability to prevent all signs of EFA deficiency. For most species, linolenic acid does not fully relieve dermal signs of linoleic acid deficiency, even at high levels [26, 35]. However, arachidonic acid (20:4ω-6) is twice as active as its precursor, linoleic acid, in reducing dermal signs attributed to the deficiency. The eicosanoids (*eikosi*=20 in Greek), which are important in the regulation of widely diverse physiological processes, are derived from ω-6 (20:4) and ω-3 (20:5) fatty acids. They have different biological activity depending on the precursor molecule. Prostaglandins, thromboxane, prostacyclin, leukotriene, and hydroxyl fatty acids are among the eicosanoids that can be formed by enzymatic conversion of di-homo-γ-linolenic, arachidonic, and eicosapentaenoic acids. One of the most important specific metabolic functions of EFAs is as precursors for a diverse group of "local hormones" called prostaglandins.

Prostaglandins are formed by elongation and desaturation of linoleic acid to dihomo-γ-linoleic acid (20:3ω-6) (DHGL) and to arachidonic acid (20:4ω-6), and from long-chain fatty acids of the linolenic family (20:5ω-3), eicosapentaenoic acid.

These fatty acids are found in membrane phospholipids. Practically all cells are capable either of producing or of being influenced by prostaglandins. A large number of known biologically active prostaglandins have been identified. Prostaglandins formed from DHGL without further desaturation to arachidonate comprise the 1 series of prostaglandins. Prostaglandins formed from arachidonate comprise the 2 series. The 3 series of prostaglandins is formed from eicosapentaenoic acid (20:5ω-3). The discovery of prostaglandin-like molecules in human urine and plasma [35, 37] pointed to a non-enzymatic peroxidation of arachidonate, resulting in many different oxygenated products with prostaglandin-like structures.They are called is oprostanes because many of them resemble the prostaglandins, with some differences in stereochemistry, for example, 8-epi-prostaglandin F_{2a} versus prostaglandin F_{2-a} [4, 20, 35].

Generally, prostaglandins and leukotrienes constitute a group of extracellular mediator molecules that are part of an organism's defense system [7, 35]. Prostaglandins and leukotrienes are formed during the inflammatory process, and if the inflammation is caused by invading bacteria, the formation of prostaglandins and leukotrienes will stimulate macrophages and other leukocytes to begin the process of destroying the bacteria. Lipid sources could alter the development of autoimmune disease and the life span of short-lived animals. Many investigators have observed that polyunsaturated lipids that hinder cardiovascular disease (when compared to saturated fats) can be proinflammatory [16, 35]. Macrophages are involved in immune and inflammatory functions and possess the enzymes necessary for prostaglandin and leukotriene biosynthesis. Consuming high levels of ω-3 fatty acids may provide considerable health benefits in relation to inflammatory diseases, such as atopic dermatitis and rheumatoid arthritis [35, 13]. The mechanism behind the potentially beneficial effect of ω-3 fatty acids on some inflammatory diseases may be related to altered eicosanoid formation. Reduction of eicosanoid biosynthesis by inflammatory cells is of clinical interest because of the immunosuppressive potential of elevated levels of prostaglandin E (PGE) (Kinsella et al.,1990) and possibly leukotriene B_4 (LTB_4) [18, 35]. For instance, production of leukotriene derived from 20:5ω-3 (LTB_5) is an immunosuppressive result for one of the more potent inflammatory agents (LTB_4), which is derived from 20:4ω-6 [13, 35].

Many age-associated diseases, including malignancy and autoimmune disease with a viral etiology, appear to be exacerbated by high-fat diets with a large proportion of vegetable oils high in ω-6 fatty acids. These oils could increase autoimmune disease by increasing free radical formation and decreasing levels of antioxidant enzyme mRNA, thus further decreasing immune function, in particular by inhibiting the development of anti-inflammatory cytokines such as interleukin (IL-2) and transforming growth factor (TGFb). In contrast, ω-3 lipids could protect against autoimmunity by enhancing TGFb mRNA levels and preventing an increase in oncogene expression [35, 16].

Polyunsaturated fatty acids have a structural function as an integral part of phospholipids, the building unit of bio membranes. This is inferred from the specific composition of the fatty acids in these phospholipids (the β position normally being esterified with the highly unsaturated members of the EFA families) and from the fact that in EFA deficiency, these fatty acids are replaced by eicosatrienoic acid (20:3ω-9), biosynthesized from oleic acid (18:1ω-9), with the known concomitant deleterious effects on bio membrane function and integrity. The phospholipids of cell membranes will influence membrane viscosity and permeability and thereby possibly the enzyme activity of membrane proteins. Different types of eicosanoids are formed from different essential fatty acids. The general rule is that eicosanoids derived from eicosapen-taenoic acid are less potent than the corresponding compounds derived from arachidonic acid [13, 35].

It has been suggested that EFA deficiency and replacement of the linoleic acid family in membrane structures may cause a disruption in spatial arrangements in mitochondria that results in less efficient oxidative phosphorylation and a derangement of basal metabolism. Such a process may be the partial uncoupling of oxidative phosphorylation in mitochondria. In poultry, presence of linoleic acid may not be absolutely necessary in the body since a deficiency will not always result in death. Fatty acids that replace the linoleic acid family in tissue lipids seem to cause a reduction in the metabolic efficiency and functioning of the animal, but life often can still be maintained [35, 44].

A disturbed water balance is a characteristic defect of EFA deficiency and can include increased water loss through the skin, increased urinary arginine-vasopressin loss, increased water intake, and reduced urine output [26, 21, 20, 35]. Increased water loss through skin results from a defect in the permeability barriers of skin, which is an indication that EFAs are involved in membrane structure. Histological

studies have shown many changes in skin structure as a result of the deficiency. Additional functions of EFAs include provision of adequate fluidity to sustain cellular function and for lipid transport [27, 35]. Phospholipids and cholesteryl esters containing an abnormally high proportion of saturated fatty acids would tend to be more rigid or less fluid than would similar compounds with high proportions of polyunsaturated acids. Ethanol can penetrate the lipid bilayer of the cell membrane and can cause changes in the structure and organization of the fatty acid core, thus changing the membrane fluidity [35, 24]. Ethanol may affect the uptake of very long chain fatty acids from the diet and/or their incorporation into lipids [47, 35]. One of the functions of polyunsaturated acids is to provide lipids that are fluid at body temperature. Alloxan diabetes, hyperthyroidism, dietary cholesterol, saturated-fat diet, or mineral oil all involves the transport of a nonessential lipid in large quantities. These conditions have been found to accelerate EFA deficiency significantly. Studies suggest that one function of polyunsaturated acids is to provide necessary structural components for circulating lipoproteins [26, 35].

In a paper, [35, 49] summarized the beneficial effects of adequate linoleic acid: (1) decreased blood cholesterol and triglyceride levels, (2) decreased thrombotic tendency of platelets,(3) preventive and curative effects in sodium-induced hypertension, (4) improvement of the physiological function of the heart, and (5) normalization of the biochemical abnormalities in obesity and maturity onset diabetes. Mechanisms of these responses are not clearly established; however, many of the biological effects are derived from the eicosanoids synthesized from arachidonic acid (20:4ω-6), including prostaglandins, thromboxane, prostacyclin, and leukotriene. Linoleic and linolenic acids stimulate the growth of mammary epithelium in normal rats. Dietary linoleate seems to be necessary to the development of mammary ducts and alveoli in immature mice and to their maintenance in adult animals [31, 35]. Using rats, Ollivier-Bousquet et al. [40, 35] suggested that only ω-6 fatty acids (versus ω-3) are required for the optimal functioning of lactating mammary epithelial cells. CLA (see Chemical Structure and Properties) has been identified as an anticarcinogen. The CLA has several unique structural and functional properties, resulting in chemical and physiological effects that are different from those of all-*cis*-nonconjugated polyunsaturated fatty acids. In turn, these unique qualities appear to modulate cellular processes involved in carcinogenesis [2, 35].

Synthesis of CLA requires the presence of free linoleic acid (the substrate), a free radical-generating species, and proteins rich in sulfur residues [12, 35]. These conditions occur *in vivo* (through oxidative pathways and enzymatic isomerization) and *in vitro* (treatment of foods with heat) [8, 35]. The major dietary sources of CLA are foods derived from ruminants, such as beef and cheese. More than 85% of the CLA in animal tissues is the *cis*-9, *trans*-11 isomer form. They have been found in triglycerides, lipoproteins, and cell membrane phospholipids in several tissues of rodents, rabbits, and humans. Intestinal bacterial flora of rats is capable of converting free linoleic acid to *cis*-9, *trans*-11, and *trans*-9, *cis*-11 CLA isomers [9, 35] Using isolated working heart models from rats, Pepe and McLennan [35, 43] reported that dietary fish oil prevented the initiation and reduced the severity of arrhythmias in the isolated hearts in response to a variety of stimuli. These results establish that irrespective of any effects on blood pressure or platelet function *in vivo*, dietary fish oil directly affects myocardial properties, which may contribute to observed clinical reductions in cardiac mortality associated with fish consumption.

From studies on postinfarct transcutaneous angioplasty, there are some conflicting data on the effect of very long chain ω-3 fatty acids on the frequency of re-stenosis, while a prospective study on mortality after coronary artery infarction reported a significant decrease (29%) in all-cause mortality after 2 years for patients using at least two fatty fish meals weekly [3, 35]. Sudden cardiac death accounts for about 50% of total coronary disease mortality in westernized industrial countries. The lack of early symptoms for this disorder makes prevention the preferred strategy. In a rat model of cardiac ischemia, dietary ω-6 (sunflower seed oil) and ω-3 (fish oil) polyunsaturated fatty acids were shown to protect against arrhythmia compared with saturated fat; greatest protection was observed with fish oil [35, 48].

Studies have shown that one aspirin every other day reduced CHD events by 47% in 5 years in 23,000 men. Aspirin interferes with platelet aggregation by inhibiting the enzyme cyclooxygenase in platelets. ω-3 Fatty acid supplements have been shown to inhibit the same enzyme and to inhibit platelet aggregation [33, 35].

The brain, as well as most of the nervous system, is a well-protected organ, especially with regard to

polyunsaturated fatty acids. The nervous system has the greatest concentration of lipids after adipose tissue. Interestingly, these lipids are practically all structural and are not related to energy production; they participate directly in the functioning of cerebral membranes. Brain lipids are formed of polyunsaturated fatty acids derived from dietary essential linoleic and linolenic acids. On average, one fatty acid out of three in the brain is polyunsaturated, and these fatty acids participate in the structure of phospholipids. The ω-6 fatty acids are required for normal fetal growth, but ω-3 fatty acids are beneficial for surfactant synthesis [35, 50]. Surfactant is a phospholipid-rich substance that lines the air-alveolar inter- face in the lung and prevents alveolar collapse, a major cause of mortality and morbidity in premature infants.

The long-chain ω-3 fatty acids are found in high proportions in reproductive and nervous tissues. The elongated docosahexaenoic acid (22:6ω-3) is the most abundant fatty acid in the ethanolamine phospholipids of cerebral gray matter and the retina [7, 9 and 35]. The need for ω-3 fatty acids in developing visual acuity was presented as evidence for a functional requirement for ω-3 fatty acids in primates [35, 39]. Differences in physical activity and ability to learn have been related to low content of 22:6ω-3 in brains of rats produced by feeding a diet low in linolenic acid [32, 35].

A diet deficient in linolenic acid alters nerve-ending fluidity and enzymatic activities, reduces the amplitude of electrophysiological parameters such as the electroretino gram, alters the resistance of the nervous system to poisons, and reduces the performance of learning tasks [3, 35]. It has been shown that changes in lipid membrane composition can modulate the binding of neurotransmitters or hormones to membrane receptors at the periphery [38, 35]. Modifications of the neurotransmission pathways might induce the behavioral disturbances that are seen in animals [13, 35].

Diets rich in PUFAs have been associated with promotion of tumor growth in rodents under certain conditions. However, studies in rats and mice [8, 35] have shown that not all PUFAs should be incriminated. Increasing the fat contribution from ω-6 PUFAs (in vegetable oils) from 0 to 4% dietary energy routinely enhances tumorigenesis in rodents, while diets with equivalent levels of long-chain highly unsaturated ω-3 fatty acids found in fish oil often diminish formation of certain tumors.

Epidemiologic studies suggest that individuals who consume diets rich in fat are at higher risk for colon cancer, whereas consumption of fish products rich in ω-3 fatty acids, such as eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA), is associated with low incidence of colorectal cancer [35, 51]. Fish oil seems to protect against cancers that are most likely linked to diet—those of the colon, pancreas, breast, and ovary.

Leaf [33] reviewed the effect of ω-3 fatty acids in reducing cardiovascular disease; additional physiologic and pharmacologic effects of fish oils were referenced as follows: (1) decreases blood pressure in normal and moderately hypertensive subjects, (2) decreases blood viscosity, (3) decreases microvascular albumin leakage in insulin dependent diabetics, (4) decreases plasma triglycerides, (5) decreases vascular response to norepinephrine, (6) decreases ventricular fibrillation from ischemia, (7)decreases cardiac toxicity of cardiac glycosides, (8) decreases platelet adhesion, (9) decreases leucocyte endothelium interactions, (10) increases vascular compliance, and (11) increases platelet survival.

Effects of Deficiency

Ruminants

The EFA deficiency of ruminants has been less extensively researched than that of nonruminants, with the deficiency in adult ruminant's not readily demonstrated [35, 41]. The microbial population appears to provide enough EFA to meet the requirements; however, studies with lambs suggest that the required level of EFA may be elevated in the presence of host microflora [5, 35]. Gullickson et al. [17] reported that calves fed a low-fat diet did not develop EFA deficiency signs, but growth was suppressed. Cunningham and Loosli [31] reported that calves receiving fat-free synthetic milk developed leg weakness and muscular twitches within 1 to 5 weeks and died unless a source of fat was supplied. Lambert et al. [31] also studied the effect of a "lipid-free," semisynthetic milk fed to dairy calves. They reported the following clinical signs: growth retardation after 3 weeks on trial; scaly dandruff; long dry hair; dull hair coat; excessive loss of hair on the back, shoulders, and tail; and diarrhea.

Weanling lambs fed a fat-free diet for 7 months showed no evidence of skin lesions or other clinical signs typical of fat deficiency. In a

second experiment, 2-day-old lambs and kids were given fat-free synthetic milk. The lambs and kids receiving the fat-free diets became weak and died within 1 to 7 weeks, while controls were raised successfully on the same milk with 2% added lard. Delivery of fatty acids at various levels for metabolism can influence events important in dairy cow reproduction. A soybean oil emulsion (50% linoleic acid) was infused intravenously to Holstein heifers [34, 35]. This resulted in increased plasma concentrations of prostaglandin F 2-α ($PGF_{2\alpha}$) metabolite and increased ovarian follicles, and the size of the largest follicle was greater. In a second study, feeding rumen-protected fat to lactating dairy cows increased the numbers of 3- to 5 mm follicles and follicles greater than 15mm in diameter, and increased the size of the preovulatory follicle of a synchronized estrus cycle during the early postpartum period [34, 35] fed rumen protected fat (0.5kg/day), which improved conception rates of lactating Holstein cows from 52 to 86%. Inclusion of fish oil in the diet appears to result in an alteration in regression dynamics of the corpus luteum as evidenced by a greater proportion of cows having elevated concentration of plasma progesterone after injection of $PGF_{2\alpha}$ [6, 35]. Perhaps the increase in concaption rate (39.5 versus 30.6%) could be attributable to increased survival of the embryo at the time of pregnancy recognition (e.g., when $PGF_{2\alpha}$ secretion is suppressed). It would appear that fatty acids are important to both stimulated follicles (elevated $PGF_{2\alpha}$) to bring about pregnancy but later decrease PGF_2, which would result in greater progesterone production and maintenance of pregnancy.

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