EFFECT OF DIETARY n-3 FATTY ACID SOURCE ON PLASMA, RED BLOOD CELL AND MILK COMPOSITION AND IMMUNE STATUS OF MARES AND FOALS

By

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A THESIS PRESENTED TO THE GRADUATE SCHOOL
OF THE UNIVERSITY OF FLORIDA IN PARTIAL FULFILLMENT
OF THE REQUIREMENTS FOR THE DEGREE OF
MASTER OF SCIENCE

UNIVERSITY OF FLORIDA

2006

This document is dedicated to my mother and step-father, Melanie and Jim Eisenhour, for their unconditional love and support even when they had no idea what I was doing, and to my husband Alex, who I could not have survived graduate school without.

ACKNOWLEDGMENTS

First and foremost I want to thank my husband Alex for his never ending patience, love and understanding through these first years of our marriage. His faith in me has oftentimes exceeded the faith I have in myself, and without his constant encouragement I could not have completed this work. I am so proud of him for completing his doctoral degree this summer, while at the same time walking me through my first experiences of graduate school. I am extremely lucky to have a husband who is also my best friend.

I owe great gratitude to Dr. Lori Warren, my committee chair. Her guidance and wisdom have been invaluable to me, both inside the classroom and out. She has been instrumental in my choices of future paths, and I thank her for this direction. I would also like to thank Drs. Lokenga Badinga and Steeve Giguere, who served on my committee and dedicated their time to improving my project and thesis. Joel McQuagge also deserves my appreciation for his encouragement, humor and friendship.

My sample analysis could not have been completed had it not been for the supervision and instruction of Jan Kivipelto. Jan also receives my debt of gratitude for being a shoulder to lean on and an open ear to talk to. I owe thanks as well to Steve Vargas and the employees of the University of Florida Horse Research Center, especially Cher Jackson. Their help, both in taking samples and organizing data, was crucial to this project. I would also like to thank the horses of the Horse Research Center and Horse Teaching Unit. Without their cooperation and patience I could not have completed this work. A special thank you goes to “Buster Buckley,” who always made me smile.

I am fortunate enough to have friends throughout the department willing to offer help and smiles during the past few years. Many thanks go to Sarah Dilling, Kelly Spearman, Drew Cotton, Aimee Holton, Sarah White and Liz Greene for their assistance. I also appreciate the support of the crew at the Horse Teaching Unit, especially Justin Calahan and Kristin Detweiler.

Last, but most definitely not least, I would like to thank my grandmother, Lois VanNatta; my "Baba," Dena Lovacheff; my sister- and brother-in-law, Jennifer and Todd Schwent; and the rest of Alex's and my family. I would like to lend a special thank you to my in-laws, Lynne and Michael Stelzleni, for their unconditional love, support and encouragement of Alex and me. Most importantly, I want to thank my mother, Melanie Eisenhour, for showing me the kind of woman I want to be and my step-father, Jim Eisenhour, for taking me in and loving me like his own daughter. They have offered me nothing but undying love and support and have been my biggest fans. I am forever indebted to them for all they have done.

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EFFECT OF DIETARY n-3 FATTY ACID SOURCE ON PLASMA, RED BLOOD CELL AND MILK COMPOSITION AND IMMUNE STATUS OF MARES AND FOALS

By

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August 2006

Chair: Lori K. Warren
Major Department: Animal Sciences

Supplementing the diets of horses with fat is a popular trend in today's equine industry. However, little focus has been given to the effect of supplementing with omega-3 fatty acids (FA) in the broodmare and her suckling foal. To study these effects, 36 Thoroughbred and Quarter Horse mares with an average bodyweight of 580.9 ± 3.5 kg (mean ± SE) were randomly assigned to one of three treatment groups: 1) basal diet with no supplementation (CON, n = 12); 2) basal diet plus milled flaxseed supplementation (FLAX, n = 12); or 3) basal diet plus encapsulated fish oil supplementation (FISH, n = 12) from 28 days prior to expected foaling date until 84 days after foaling. The flaxseed and fish oil supplements were fed to mares in amounts to provide 6 g total n-3 FA/100 kg BW per day. The basal diet consisted of a commercial grain-based concentrate, Coastal bermudagrass hay and bahiagrass pasture.

Blood samples were obtained from mares at 28 d pre-partum and milk and blood samples were obtained from mares and foals at foaling, 36 h and 14, 28, 56 and 84 d post-partum to determine FA and IgG content. On d 84, mares and foals received paired intradermal injections of phytohemagluttinin (PHA) and skin thickness was determined over a 48 h period as a measure of the inflammatory response. Bodyweights were obtained from mares and foals at 14 d intervals throughout the trial.

Treatment had no effect on gestation length (P = 0.84), mare bodyweight (P = 0.80) or foal bodyweight (P = 0.76). Mares fed FLAX had higher plasma alpha-linolenic acid (ALA) (P=0.06) than mares fed FISH or CON mares. Mares fed FISH had higher plasma eicosapentaenoic acid (EPA), docosahexanoic acid (DHA) and total n-3 (P=0.03) than FLAX and CON mares. Across treatments, total milk n-3 increased (P=0.0005) and total n-6 decreased (P=0.0001) from foaling to d 84. Milk from FLAX mares had higher ALA (P=0.01) than milk from FISH and CON mares. Milk from FISH mares had higher EPA and DHA and a lower n-6:n-3 ratio (P=0.007) than milk from FLAX and CON mares. Foals suckling FLAX mares had higher plasma ALA (P=0.04) than foals suckling FISH and CON mares. Foals suckling FISH mares had higher plasma EPA, DHA and total n-3 and a lower plasma n-6:n-3 ratio (P=0.002) than FLAX and CON foals. Treatment did not affect colostrum, milk or foal serum IgG. Response to PHA injection was greater (P=0.0001) in mares compared to foals but similar between treatments. Although the addition of n-3 FA to the mare's diet altered the FA content of mare milk and mare and foal plasma, changes in total IgG and PHA intradermal responses were not detected..

CHAPTER 1
INTRODUCTION

Supplementing the diet with fat is a popular trend in the horse industry. Fat is commonly fed to horses to improve the hair coat, improve body condition and increase the energy density of the diet. However, most of the research that has examined fat supplementation of the horse has been performed with little regard to the type of fatty acids (FA) provided. In addition, most of this research has focused on mature performance horses; relatively little information is available on fat supplementation of mares and the effects on the suckling foal.

Corn oil, soybean oil, and rice bran are common sources of fat added to horse rations; however, these feeds are high in omega-6 FA. High levels of n-6 FA have been associated with more pronounced inflammatory responses in humans (Meydani et al., 1993; Simopoulos, 1999); therefore, potential exists for such diets to also have negative biological effects in the horse.

Based on the immunomodulatory effects of n-3 FA in humans and other animals (Simopoulos, 1999; Anderson and Fritsche, 2002), there is interest in determining whether n-3 FA supplementation can modify inflammatory and immune responses in horses. In addition, the dietary source of n-3 FA may be important for eliciting the desired health benefits. Flaxseed is an excellent source of alpha-linolenic acid (ALA), whereas fish oil is a good source of eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA). Although both are rich in total n-3 FA, fish oil may be a more effective means of providing biologically active n-3 FA than flax.

The immune status of foals is a vital concern for horse breeders, as suckling foals are susceptible to many health problems including diarrhea and septicemia. These health problems can cause significant veterinary expense, as well as endanger the life of the foal. Previous research has shown that supplementation of broodmares with linseed oil or a mix of corn and linseed oil increases the n-3 content of her milk and the n-3 content of her foal's blood (Duvaux-Ponter et al, 2004; Spearman et al., 2005). Therefore, it seems possible to enhance the concentration of n-3 FA in the foal by manipulation of the mare's diet.

The objectives of this research were to 1) examine the effect of dietary n-3 supplementation on the FA composition of mare milk and mare and foal plasma; 2) examine the efficiency with which ground flaxseed or encapsulated fish oil augment the presence of EPA and DHA in the mare and foal; 3) examine the effects of supplementation with flaxseed vs. fish oil on increasing colostrum, milk and foal plasma IgG; and 4) examine the effects of feeding flaxseed vs. fish oil on the inflammatory response in the mare and foal. We hypothesize that supplementing the mare with fish oil will increase the EPA and DHA concentrations in mare milk and mare and foal blood to a higher extent than will flaxseed, will increase the IgG in mare colostrum and foal blood, and will decrease the inflammatory response in mares and foals.

CHAPTER 2
REVIEW OF LITERATURE

Fatty Acid Structure, Digestion and Metabolism

Fatty Acid Structure

Fatty acids (FA) consist of carbon (C), hydrogen (H) and oxygen (O) arranged in a carbon chain with a carboxyl group (-COOH) at one end and a methyl group ($-CH_3$) at the other. FA are classified and named by their chain lengths and their degree of unsaturation, or number of double bonds. Unsaturated FA can be monounsaturated (only one double bond) or polyunsaturated (two or more double bonds), whereas saturated fatty acids have no double bonds. Fatty acids are also classified as short, medium or long chain, with short chain FA having less than 8 carbons, medium chain FA having 8 to 16 carbons and long chain FA having greater than 16 carbons. The numbering sequence of the carbons in a fatty acid chain begins at the carboxyl end, with the carboxyl carbon being C1. An older system used Greek letters to identify carbon atoms. In this system, C2 (the first carbon after the carboxyl carbon) was the α-carbon, C3 was the β-carbon and so on, ending with the last carbon in the chain at the methyl end as the ω-carbon (Gurr et al., 2002).

Currently, the numbering system is the preferred method of naming individual FA. In this system, the number of carbon atoms in the FA chain is given followed by a colon and the number of double bonds. For example, stearic acid, a saturated FA of 18C, is identified as C18:0. Linoleic acid, a polyunsaturated FA of 18C with two double bonds, is identified as C18:2. While the current numbering system is preferred, the older system

is used to identify ω-6 and ω-3 fatty acids, where the last double bond in the fatty acid chain is six and three carbons away from the ω-carbon, respectively (Greene, 2006). Newer research may substitute the ω with an *n*, but the meaning does not change.

The presence of double bonds in a fatty acid chain also allows for positional and geometric isomerism. Positional isomerism refers to a different location of double bonds in the carbon chain. Geometric isomerism refers to the orientation of the hydrogen atoms around the carbon-carbon double bond. A *cis* configuration results when both hydrogen atoms are on the same side of the bond, while a *trans* configurations results when hydrogen atoms are on opposite sides of the bond. Most natural unsaturated FA are in the *cis* configuration (Spallholz et al., 1999).

Fatty Acid Digestion

Dietary fats exist mostly as triglycerides (TG) which are made up of three FA attached to a glycerol backbone (Mu and Hoy, 2004). The digestion of these TG begins in the stomach by the action of gastric lipase released from the gastric mucosa. In humans and rats, lingual lipase from the von Ebner glands, a group of serous glands on the tongue, also aids in FA digestion in the stomach. This lipase is transferred with the food bolus into the stomach where its activity begins (Mu and Hoy, 2004). Secretion of the lingual lipase occurs continuously but is stimulated by dietary (high fat) and mechanical (suckling) factors (Carey et al., 1983; Tso, 1989). Digestion by both lipases produces free FA and diglycerides (Carey et al., 1983; Tso, 1989). The lingual lipase is especially important in the newborn, as pancreatic lipase activity is not fully developed at birth. In addition, the short and medium-chain TG present in milk fat are readily hydrolyzed by the lingual lipase (Tso, 1989). Horse saliva, however, does not possess

this lingual lipase (Frape, 1998; Ellis and Hill, 2005). In fact, it is currently thought that equine saliva does not contain any enzyme activity (Ellis and Hill, 2005). Therefore, equine saliva is not as important in beginning digestion but is vital in providing feed lubrication (Frape, 1998) and buffering of the feed-saliva mixture (Ellis and Hill, 2005).

While only 10-30% of dietary fat is hydrolyzed in the stomach, the majority of FA digestion takes places in the small intestine, especially in the duodenum. In animals with a gall bladder, the action of the food bolus entering the duodenum stimulates gall bladder emptying, secretion of pancreatic lipase and the release of cholecystokinin (CCK). Bile acids are also released from the gall bladder or directly from the liver to emulsify the fat (Mu and Hoy, 2004). The horse, however, does not have a gall bladder, but this does not seem to affect the digestion of fat (Cunha, 1991). In the horse, bile continuously drains from the liver into the small intestine to facilitate the emulsion of fat (Frape, 1998). Furthermore, the peristaltic and segmental contractions present in the intestine supply mechanical energy to reduce the fat particle size and increase the interfacial area of the fat droplets (Carey et al., 1983). The action of pancreatic lipase on a triglyceride molecule releases two free FA and a 2-monoglyceride. These compounds, along with biliary salts, form micelles that are absorbed into the intestinal mucosal cells by passive diffusion (Doreau and Chilliard, 1997). In the horse, pancreatic lipase is secreted in high amounts and increases as fat is added to the diet (Frank et al., 2004). Therefore, the horse is able to digest high amounts of fat in the diet. Horses have been fed diets with 20% of the DE provided by oil with good results and no negative effect on digestibility (Cunha, 1991).

Once the monoglycerides and free FA are absorbed into the intestinal cell, the long-chain fatty acids (LCFA) must be transported to the endoplasmic reticulum, the major site of absorbed lipid metabolism. One explanation for how the LCFA reach the endoplasmic reticulum is by the action of fatty acid-binding protein (FABP). FABP is present in the intestinal mucosa, liver, kidney, and adipose tissue and has no affinity for short or medium-chain FA (Tso, 1985). It has been postulated that FABP may be responsible for removing LCFA acids from their binding to the cytosolic side of the luminal membrane and transferring them to the endoplasmic reticulum (Carlier et al., 1991). Unlike LCFA, short and medium-chain FA are transferred directly from the intestinal cell into the portal blood as free FA bound to albumin (Carlier et al., 1991).

Once inside the endoplasmic reticulum, LCFA and monoglycerides are recombined into triglycerides by the monoglyceride pathway. The enzyme complex that makes up this pathway is known as "triglyceride synthetase." This complex consists of three enzymes: acyl-CoA synthetase, MG transacylase and diglyceride transacylase. The acyl-CoA synthetase, in the presence of CoA, activates the LCFA to form acyl-CoA. The acyl-CoA is then used for the reacylation of monoglyceride to diglycerides and finally to triglycerides (Tso, 1985). The resulting triglycerides are then packaged with cholesterol esters and phospholipids into chylomicrons, which are large lipoproteins that act as carriers of dietary triglycerides. Chylomicron formation is activated by the addition of apoproteins, which are proteins that play an important role in the formation and secretion of chylomicrons by the enterocytes. Once chylomicrons are formed, they are released by exocytosis into the lymphatic system where they can enter the blood stream via the thoracic duct and be transported to the rest of the body (Carlier et al., 1991).

De novo Fatty Acid Synthesis

There are two primary sources of FA in the body: FA provided by the diet and FA made by the animal via *de novo* synthesis (Lehner and Kuksis, 1996). The pathways for *de novo* FA synthesis exist in the animal during the well-fed state and in monogastrics occur primarily in the liver. Most of the carbon used for *de novo* FA formation is supplied through the pyruvate pool and from the end product of glycolysis. There are three substances needed for FA synthesis: acetyl CoA, malonyl CoA and NADPH. The first step in the synthesis of FA is the formation of acetyl CoA from pyruvate in the mitochondrial matrix by the action of pyruvate dehydrogenase. The acetyl CoA must then be moved out of the mitochondria and into the cytosol where FA synthesis takes place. Because the inner mitochondrial membrane is not permeable to acetyl CoA, the acetyl CoA is combined with oxaloacetate to form citrate. Citrate is then translocated to the cytosol where it is cleaved back to oxaloacetate and acetyl CoA by ATP:citrate lyase (Gurr et al., 2002). This mechanism of moving acetyl CoA into the cytosol in the form of citrate is called the citrate shuttle.

Once acetyl CoA reaches the cytosol, *de novo* FA synthesis begins. The first reaction of this mechanism, which is also the rate limiting reaction, involves the formation of malonyl CoA by the enzyme acetyl-CoA carboxylase (ACC) (Knowles, 1989). The malonyl CoA forms the source of the vast majority of the carbons of a FA chain. The enzyme complex that synthesizes LCFA from acetyl and malonyl CoA is fatty acid synthase (FAS). This enzyme complex has synthase, reductase and dehydrase actions. The typical end product of animal FAS action is palmitic acid (C16:0) (Greene, 2006).

Once produced, palmitic acid can be elongated and desaturated. Type III synthases (commonly called elongases) lengthen FA preformed in the animal 2C at a time. The principal elongation reactions occur in the endoplasmic reticulum membranes and involve acyl-CoA as a primer, malonyl-CoA as a donor of 2C units and NADPH as the reducing coenzyme. This system is capable of producing FA chain with an excess of 20 carbons (Suneja et al., 1990). Desaturation, or the addition of double bonds, occurs mainly by oxidative desaturation, a process by which a double bond is introduced directly into the LCFA with O_2 and NADH as cofactors (Scheuerbrandt and Bloch, 1962). Mammalian desaturases are only able to introduce double bonds in the Δ9, Δ6 and Δ5 positions. Plant desaturases can introduce additional double bonds at the Δ12 and Δ15 positions, therefore creating n-6 and n-3 FA. All double bonds introduced by the process of oxidative desaturation are in the *cis* configuration (Lehner and Kuksis, 1996).

Fatty Acid Degradation

The mobilization and oxidation of FA occur primarily during fasting, physical exercise and stress in the animal in order to break down dietary or stored TG into FA to provide energy. The mobilization of FA occurs via lipolysis in the adipose tissue, in which FA are cleaved from their glycerol backbone mainly by hormone sensitive lipase (HSL) and released into circulation (Johnson and Greenwood, 1998). The main forms of FA oxidation are termed alpha (α), beta (β) and omega (ω), referring to which carbon on the acyl chain is attacked. Of these three, β-oxidation is the most prevalent. In β-oxidation, there is a stepwise removal of 2C units from the carboxyl end of the FA (Greene, 2006).

The mitochondria and microbodies (peroxisomes and glyoxysomes) are capable of performing β-oxidation. The process begins by converting the FA into fatty acyl-CoA as soon as it enters the cytosol of the cell. The inner mitochondrial membrane, however, is impermeable to fatty acyl-CoA. In order to move this molecule across the membrane, the enzyme carnitine:palmitoyl transferase (CPT_1), located on the outer mitochondrial membrane, combines the fatty acyl-CoA with carnitine. The resulting acyl carnitine is then transported across the membrane, crossing the inner membrane by a carnitine:acylcarnitine translocase (Pande, 1975). Once the acyl carnitine is inside the mitochondrial matrix, CPT2 transfers the acyl group from carnitine to CoA, therefore reforming acyl-CoA as a substrate for further β-oxidation (Bieber, 1988).

The process of β-oxidation involves a repeated sequence of four reactions resulting in the removal of 2C from the acyl chain. First, acyl-CoA dehydrogenase acts on the acyl-CoA to form trans-3,3-enoyl-CoA. Enoyl hydratase then acts on the product of the first reaction to form 3-hydroxy acyl-CoA. The third reaction is catalyzed by the enzyme 3-hydroxy acyl-CoA dyhyrogenase which works with NAD^+ to form 3-oxoacyl-CoA. The final reaction involves 3-oxoacyl-CoA thiolase which produces a shorter fatty acyl-CoA and acetyl-CoA (Bieber, 1988). The resulting acyl-CoA is recycled back into β-oxidation for the removal of additional 2 carbon units, while the acetyl-CoA can be used in the TCA cycle to produce energy (Gurr et al., 2002).

Polyunsaturated Fatty Acids

n-6 and n-3 Polyunsaturated Fatty Acids

By definition, n-6 polyunsatured fatty acids (PUFA) have the last double bond in the FA chain six carbons from the methyl (omega) end. The two most physiologically

import n-6 PUFA are linoleic acid (LA; C18:2) and arachidonic acid (AA; C20:4). Of these, LA is considered a dietary essential fatty acid because it cannot be synthesized by mammals. Sources of LA include vegetable oils such as corn, sunflower, peanut, and soy oils (Carlier et al., 1991). Linoleic acid can be elongated and desaturated in the body to produce AA in a mechanism that is discussed later in this chapter. Omega-6 PUFA, with AA as the principal component, predominate in organs and tissues performing storage functions (adipose tissue), chemical processing (liver), excretion (kidney) and mechanical work (muscle) (Innis, 1991). In addition, plasma lipoproteins contain high amounts of LA in triglycerides, cholesterol esters and phospholipids (Innis, 1992a). A very important feature of n-6 PUFA is their effect on the body. In general, n-6 PUFA have proinflammatory, prothrombotic and proaggregatory effects, characterized by increases in blood viscosity, vasospasm, vasoconstriction and decreases in bleeding time (Simopoulos, 1999).

Omega-3 PUFA have the last double bond in their carbon chains three carbons from the methyl end. The dietary essential PUFA from the n-3 family is α-linolenic acid (ALA; 18:3), but other physiologically important n-3 PUFA include eicosapentaenoic acid (EPA; 20:5) and docosahexaenoic acid (DHA; 22:6) (Innis, 1992a). Using elongase and desaturase actions similar to those in n-6 PUFA, ALA can be transformed into EPA, which can be further transformed into DHA. Alpha-linolenic acid is found primarily in the chloroplast of green leafy vegetables and in seeds of flax, linseed and walnuts. Fatty fish and fish oils, however, are the main sources of EPA and DHA (Benatti et al., 2004). The primary sites of n-3 PUFA accumulation in the body include the nervous tissue, reproductive organs and retina membranes (Innis, 1991). Unlike the plasma

concentrations of LA, tissue and plasma triglyceride and cholesterol ester levels of ALA are usually quite low (<1-2% FA) (Innis, 1992a). Polyunsaturated FA of the n-3 family are known to have anti-inflammatory, antithrombotic, antiarrhythmic, hypolipidemic and vasodilatory effects on the body (Simopoulos, 1999). To obtain optimal health, it is important to have adequate dietary amounts of PUFA of both the n-6 and n-3 families, but it may also be important to have a proper ratio between the two. A ratio of 4-5:1 of n-6:n-3 has been suggested as most beneficial for humans, but most investigation in this area has been conducted in lab animals (Wiseman, 1997).

The efficiency at which the horse converts ALA to EPA and DHA is unknown. In addition, while a recommendation for a beneficial n-6:n-3 ratio exists for humans, the optimal ratio for horses is unknown. Most horse feeds today are high in n-6 FA, with the horse's major n-3 FA intake obtained from forages. The FA composition of common grains and fat supplements fed to horses are presented in Table 2-1 and the FA composition of common forages fed to horses are presented in Table 2-2.

Table 2-1. Fatty acid composition of common feeds and fat supplements fed to horses

	Feed						
Fatty acid[1]	Textured Grain[2]	Oats[3]	Corn Oil[4]	Flaxseed Oil[5]	Fish Oil[4]	Rice Bran Oil[6]	Soybean Meal[3]
C14:0	0.14	Trace	0.2	0.1	5.6	0.43	Trace
C16:0	NA[7]	22.1	10.8	5.4	21.6	16.27	10.7
C18:0	NA	1.3	20.6	3.6	9.0	1.84	1.5
C18:1	NA	38.1	10.2	0.0	15.5	41.92	21.4
C18:2n-6	38.82	24.9	54.8	15.2	1.5	35.44	14.2
C18:3n-3	3.72	2.1	1.1	53.6	1.4	1.24	7.0
C20:4n-6	0.03	NA	NA	0.1	NA	NA	NA
C20:5n-3	0.08	NA	0.3	0.0	13.5	NA	NA
C22:6n-3	0.06	NA	0.2	0.0	11.5	NA	NA

[1] Presented as g fatty acid/100 g fat

[2] Commercial grain mix (Hallway Feeds, Lexington, KY) containing barley, corn, soybean meal, molasses, oats and supplemental pellet; 14.8% CP, 6.5% fat; from O'Connor et al., 2004.

[3] From Ellis and Hill, 2005.

[4] From Chen et al., 2006.

[5] From Francois et al., 2003.

[6] From Sierra et al., 2005.

[7] NA = information not available.

Table 2-2. Fatty acid composition of common forages fed to horses

Fatty acid[1]	Forage			
	Fresh Bahiagrass[2]	Fresh Perennial Rye Grass[3]	Bermudagrass Hay[2]	Timothy Hay[4]
C14:0	0.00	0.4	0.00	1.63
C16:0	22.56	14.6	39.14	NA
C18:0	4.28	1.2	6.72	NA
C18:1	3.00	1.7	7.05	NA
C18:2n-6	21.32	10.6	23.35	15.76
C18:3n-3	46.21	68.4	15.93	26.68
C20:4n-6	0.00	NA[5]	0.00	0.35
C20:5n-3	0.00	NA	0.00	0.36
C22:6n-3	0.00	NA	0.00	0.25

[1] Presented as g fatty acid/100 g fat.
[2] From the present study.
[3] From Elgersma et al., 2003.
[4] From O'Connor et al., 2004.
[5] NA = information not available.

Elongation of and Competition Between n-6 and n-3 Families

As stated earlier, both LA and ALA can be elongated and desaturated to form their longer chain derivatives (Figure 2-1). This conversion happens in the endoplasmic reticulum (Benatti et al., 2004). The first step of the mechanism converting LA to AA is catalyzed by Δ^6-desaturase, the rate-limiting step of the pathway. This enzyme acts on LA to insert a double bond between carbons 6 and 7. This product is then elongated by the addition of two carbon units to form dihomo-γ-linoleic acid (C20:3). Further desaturation by Δ^5-desaturase inserts a double bond between carbons 5 and 6, thereby creating AA. Arachidonic acid can then be elongated to form adrenic acid (C22:4). The enzyme Δ^6-desaturase inserts a double bond between carbons 4 and 5 of adrenic acid to form ω6-docosapentaenoic acid (C22:5) (Innis, 1991).

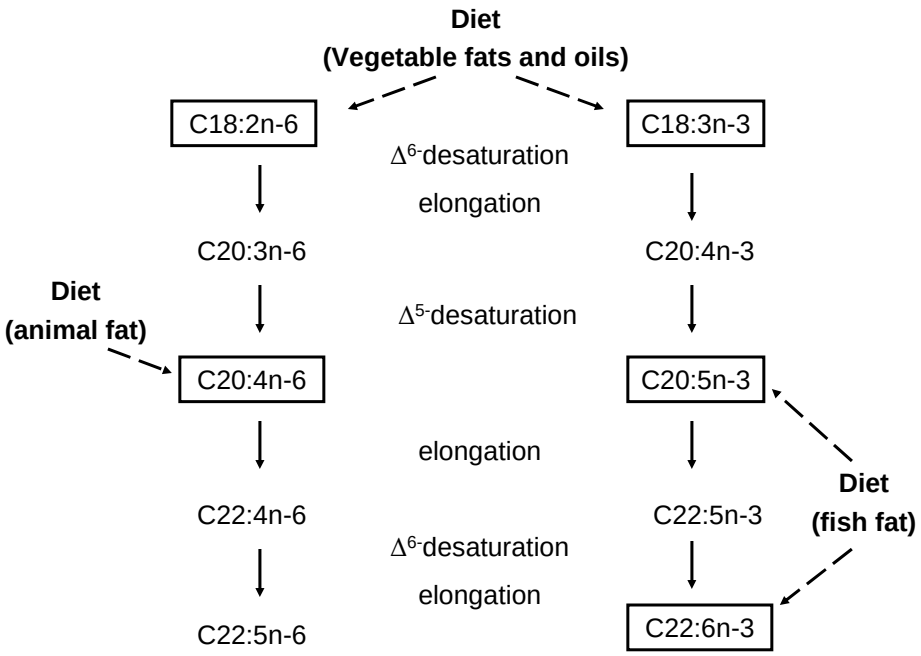

Figure 2-1. Essential fatty acid metabolism. Adapted from Innis, 1992a.

The conversion of ALA to its longer chain derivatives uses the same pathway and enzymes as LA. The enzyme Δ^6-desaturase acts first on ALA to form stearidonic acid (C18:4). This acid is then elongated and desaturated by Δ^5-desaturase to form EPA. To form DHA, EPA is elongated to form ω3-docosapentaenoic acid (DPA; C22:5), which is then desaturated by Δ^6-desaturase to form DHA (Innis, 1991). However, there is a marked inefficiency of conversion of ALA to EPA, with only about 0.2% of plasma ALA fated for synthesis of EPA in human blood (Pawlosky et al., 2001). There is 10-fold greater rate of transfer, however, of EPA to DHA than there is from ALA to EPA, showing that the initial desaturation/elongation to EPA is the most restrictive (Pawlosky et al., 2001). The difficulty of conversion of ALA to DHA has been shown in rats, where maternal rats were fed a diet high made in ALA by the addition of flaxseed oil (Bowen and Clandinin, 2000). Maternal rats were started on the experimental diet on the day of

parturition and their pups were sacrificed at two weeks of age. Bowen and Clandinin (2000) showed that supplementing maternal rats with a high ALA diet did not increase the DHA content of the whole body, skin, epididymal fat pads or muscles in rat pups, therefore suggesting that the conversion of ALA to DHA is inefficient in the rat. However, the efficiency at which the horse converts ALA to EPA and DHA is unknown. Therefore, providing animals with a dietary source of EPA and DHA (such as fish or fish oil) may be a better way to ensure incorporation of these FA into the body than feeding a source of ALA.

Because the n-6 and n-3 families use the same enzymes in the process of desaturation to their longer chain derivatives, there is competition between them. The major site of competition occurs at the site of Δ^6-desaturase action, the rate limiting reaction for PUFA desaturation. There is a strong preferential substrate affinity of the Δ^6-desaturase for n-3 PUFA, especially ALA over LA (Innis, 1991; Drevon, 1992). Therefore, feeding animals a source of n-3 PUFA will often decrease the amount of n-6 PUFA processed in the body, as more of the Δ^6-desaturase will act on the n-3 PUFA and less on the n-6. Studies have shown, in both animals and humans, that providing a dietary source of n-3 PUFA reduces the amount of AA found in the blood (Fritsche et al., 1993; Sauerwald et al., 1996). This competition between n-3 and n-6 PUFA has been established in sows assigned to diets containing 7% added fat where menhaden fish oil was substituted for lard at 0, 3.5 and 7% of the total dietary fat (Fritsche et al., 1993). Sows were fed from 107 days of gestation to 28 days of lactation. The substitution of fish oil for lard at both 3.5 and 7% decreased serum levels of AA by approximately 50% in sow serum (Fritsche et al., 1993). However, the opposite phenomenon has been

documented as well. Extensive research has shown that providing a diet rich in LA but poor in ALA will result in the accumulation of AA and very little EPA and DHA (Wiseman, 1997).

Eicosanoid Production and Function

Eicosanoids are a large family of oxygenated 20-carbon FA (Smith, 1989) that act as local hormones to modulate the intensity and duration of inflammatory and immune responses (Yaqoob, 2004). The family is made up of three groups: the prostanoids (prostaglandins and thromboxanes) which are synthesized by cyclooxygenase (COX), the leukotrienes which are synthesized by lipoxygenase (LOX) and the epoxides synthesized by epoxygenase. Eicosanoids are produced from 20-carbon PUFA containing three to five *cis*, methylene-interrupted double bonds. These PUFA include AA, a member of the n-6 family, and EPA, a member of the n-3 family. Linoleic acid (18 carbons) and DHA (22 carbons) can be converted to eicosanoid homologues, but these are not actual eicosanoids and are thought to have limited biological function. Because AA is the most abundant C_{20} polyunsaturate in mammalian systems, it is the major precursor of eicosanoids (Smith, 1989). Macrophages and monocytes are important sources of eicosanoids, as their membranes typically contain large amounts of AA (Yaqoob, 2004).

Arachidonic acid and EPA each produce eicosanoids of a different series. Arachidonic acid is a substrate for the 2-series prostaglandins (PG), namely prostaglandin E_2 (PGE_2) and prostaglandin F_2 (PGF_2) and the 4-series leukotrienes (LT), namely leukotriene B_4 (LTB_4). Prostaglandin E_2 and LTB_4 have powerful proinflammatory actions (James et al., 2000). Prostaglandin E_2 induces fever and increases vascular permeability, vasodilation, pain and edema. However, PGE_2 also suppresses the production of inflammatory cytokines TNF-α, IL-1 and IL-6 by macrophages and T cells.

Leukotriene B_4 increases vascular permeability and blood flow, is a chemotactic agent for leukocytes, induces the release of neutrophil lysosomal enzymes, and enhances the generation of reactive oxygen species. Leukotriene B_4 also increases production of TNF-α, IL-1 and IL-6 by macrophages (Calder, 2001; Yaqoob, 2004). In contrast to AA, EPA is a substrate for the 3-series PG, namely PGE_3, and the 5-series LT, namely LTB_5. These eicosanoids have the same types of inflammatory effects as those generated from AA, but they are far less biologically potent (Calder, 2001). Therefore, production of eicosanoids from EPA could modulate the immune response.

The Immune System

Acquired Immunity

The acquired immune system is capable of recognizing and selectively inhibiting specific foreign antigens (Goldsby et al., 2003). T cells, B cells, antigen-presenting cells, the major histocompatibility complex (MHC), and immunoglobulins all play important roles in the acquired immune system. This system of immunity is classified as acquired because the immune cells must be exposed to an antigen once to develop, or acquire, memory for that antigen. A second exposure to the same antigen will trigger an enhanced state of immune reactivity (Goldsby et al., 2003).

Immunoglobulins

Along with playing an important role in acquired immunity, immunoglobulins are also an important part of humoral immunity, or the type of immunity pertaining to extracellular fluids including the plasma and lymph (Goldsby et al., 2003). Humoral immunity is driven by B cells, which originate and mature in the bone marrow (Kuby, 1992). Immunoglobulins are a group of large glycoproteins found on B-cell membranes or secreted by plasma cells. They are found most prevalently in blood serum but are also

present in mucosal tissues and external secretions such as milk. An antibody is an immunoglobulin (Ig) that exhibits antigen-binding ability. Therefore, all antibodies are Ig, but not all Ig are necessarily antibodies. The two terms, however, are often used interchangeably. Antibodies have a wide range of functions, including targeting infectious organisms, neutralization of toxins and removal of foreign antigens from body circulation (Peakman and Vergani, 1997). Antibodies can serve as diagnostic tools for clinical evaluations of immune diseases or disorders. For example, immunoglobulin G (IgG) is measured in the serum of foals to determine if there has been a successful transfer of maternal antibodies.

In horses, the major immunoglobulins are IgG, IgM, IgA and IgE (Nezlin, 1998). Average concentrations of immunoglobulins in the serum of mature horses are presented in Table 2-1.

Table 2-1. Immunoglobulin concentrations of serum and milk in mature horses[1]

Sample	IgG	IgM	IgA
Adult horse serum	1000-1500	100-200	60-350
Mare colostrum	1500-5000	100-350	500-1500
Mare milk	20-50	5-10	50-100

[1] From Tizard, 1996; presented as mg/dL.

IgG is synthesized and secreted from plasma cells found in the spleen, lymph nodes and bone marrow (Tizard, 1996). IgG molecules have a long half-life (23-25 days) and there is a continuous high-level stimulation for IgG production. As a result, the concentration of IgG in blood and colostrum is higher than any other immunoglobulin (Tizard, 1996). IgG is the smallest of the immunoglobulin classes, so it is therefore more able to move through the body to travel to needed areas (Widmann and Itatani, 1998). Four immunoglobulin subclasses have been described in horses: IgGa, IgGb, IgGc and

IgG(T) (Sheoran et al. 2000). IG(T) has also been divided into the subclasses IgG(Ta) and IgG(Tb) (Tizard, 1996). These subclasses are distinguished from one another by molecular structural differences and slight variations in biological function (Kuby, 1992).

Like IgG, IgM is also made and secreted from plasma cells in the spleen, lymph nodes and bone marrow. It is found in the second highest concentration in serum, following IgG (Tizard, 1996). IgM is the first immunoglobulin class produced by the maturing B cell, and the first class synthesized by the neonate (Kuby, 1992). It is also the first antibody produced in a primary response to an antigen (Widmann and Itatani, 1998; Kuby, 1992). IgM is more efficient than other immunoglobulins in binding antigens because of its larger molecular size (largest of the immunoglobulin classes) and its larger number of binding sites. Because of its higher efficiency, IgM is also more able to neutralize viral infectivity, cause agglutination and activate compliment than IgG (Goldsby et al., 2003).

The immunoglobulin IgA is produced mainly by plasma cells in muscosa-associated lymphoid tissues beneath surface epithelium (Widmann and Itatani, 1998). While it is manufactured more than any other immunoglobulin class, serum concentration of IgA is relatively low. This low concentration is due to the secretion of IgA in fluids present on the epithelial surfaces of the alimentary, respiratory and reproductive tracts and in such fluids as urine, saliva, tears and milk (Widmann and Itatani, 1998). Because IgA is the major immunoglobulin in the external secretions of horses, it plays a vital role in protecting the intestinal tract, respiratory tract, urogenital tract, mammary gland and eyes against microbial invasion (Tizard, 1996).

Like IgM, IgE is produced predominantly by plasma cells located beneath body surfaces. It is found in very low concentrations in the serum of healthy animals, partially because the molecule is fairly unstable and has the shortest half-life of all the classes of immunoglobulins (Tizard, 1996). IgE antibodies mediate immediate (type I) hypersensitivity reactions that cause the symptoms of hay fever, asthma, hives and anaphylactic shock (Kuby, 1992). IgE is also thought to be largely responsible for immunity against parasitic worms (Tizard, 1996).

Passive Immunity in the Foal

Due to the mare's diffuse epitheliochorial placenta, there is no significant transfer of immunoglobulins to the fetal circulation during pregnancy (Jeffcott, 1972, 1974a; Erhard et al., 2001). Therefore, foals are born with a near absence of circulating immunoglobulins and an easily compromised immune system. Although they are able to produce their own antibodies soon after birth, foals will not produce levels approaching those of the adult horse until 3-4 months of age (Jeffcott, 1974a). Foals receive the needed antibodies via passive transfer from colostrum, or the mare's first milk. Prior to birth, the mare's mammary gland is capable of selecting and concentrating a wide range of serum Ig into the colostrum (Jeffcott, 1974a, 1975). When foals suckle this colostrum after birth, they take the antibody-rich fluid into their digestive tracts where the Ig can be absorbed into the circulating blood.

For the transfer of passive immunity to be successful, the mare's colostrum must contain adequate amounts of the appropriate immunoglobulins, especially IgG (Rooke and Bland, 2002). In addition, the immunoglobulins must be delivered intact to the site of absorption and absorbed intact (Rooke and Bland, 2002). Because of the very low level of proteolytic activity in the digestive tract of young foals, most immunoglobulins

are kept intact as they pass with the colostrum through the foal's stomach and small intestine. Trypsin inhibitors found in colostrum further reduce the degradation of immunoglobulins in the foals digestive tract (Kruse, 1983; Tizard, 1996).

The immunoglobulins in colostrum are rapidly absorbed by non-specific pinocytosis into the small intestine enterocytes. Maximum absorption occurs soon after birth and declines thereafter, completely ceasing by 24 hours after birth (Raidal et al., 2000). The foal's intestine shows selective permeability, with a greater affinity for IgG and IgM (Tizard, 1996). Once inside the enterocyte, individual immunoglobulins merge together to form one or more larger globules. These larger globules pass from the enterocyte into the local lymphatics and later reach the systemic circulation (Jeffcott, 1974a).

The critical event in the transfer of intact immunoglobulin to the foal's circulation is cessation of transfer across the enterocyte basolateral membrane. For this reason, "gut closure" is the term used to define the cessation of transfer of IgG to the foal's circulation. Gut closure, usually reached around 24 hours of age (Rooke and Bland, 2002), is characterized by a replacement of the immature epithelial cells with more mature cells that no longer engage in pinocytosis (Kruse, 1983).

IgG displays a unique behavior in foal serum. At birth, before the foal has suckled, foal serum IgG may be as low as 30 mg/dL (Erhard et al., 2001). However, foal IgG rises rapidly after colostrum is ingested. Peak IgG values in foal serum occur between 18 and 24 hours after birth (Jeffcott, 1974a) and have been reported to be as high as 2,160 mg/dL (McGuire and Crawford, 1973). The IgG values in foal plasma appear to stay at near peak levels for at least the first two days after foaling (Jeffcott, 1974b; Duvaux-Ponter et

al., 2004). After this peak, passively derived IgG molecules will gradually decline until they are completely absent by around 5 months of age (Jeffcott, 1974a). Foals may begin to process their own IgG molecules as early as 2 weeks of age, but levels reaching those of the adult horse are not seen until around 4 months of age (Jeffcott, 1974a). Erhard et al. (2001) reported that 7 day old foals had a mean IgG value of 1000 ml/dL. This value then decreased, indicating the elimination of maternal IgG, and reached the lowest level of 790 mg/dL at 35 days of age. However, foal serum IgG increased to around 1100 mg/dL at 42 days after birth, indicating that endogenous IgG production was increasing in the foal (Erhard et al., 2001). Therefore, behavior of IgG in the foal seems to begin with a very low presuckle value, experience a dramatic rise after colostrum ingestion, undergo a steady decline as maternal IgG is eliminated and shows an increase as the foal begins to produce its own IgG.

Failure of Passive Transfer

Failure of passive transfer (FPT) is defined as the failure of absorption of maternal immunoglobulins by the neonatal foal, a condition that predisposes the foal to life-threatening infections (Kohn et al., 1989). Failure of passive transfer is the most commonly recognized immune deficiency in horses and may predispose affected foals to septicemia, infective arthritis and pneumonia (Raidal, 1996; Raidal et al., 2000). There are conflicting views in the literature as to what antibody levels actually constitute failure of passive transfer. Liu (1980) and McGuire et al. (1977) defined failure of passive transfer as less than 200 mg IgG/dL serum and partial failure as between 200 and 400 mg/dL. LeBlanc et al. (1986) suggested failure of passive transfer as levels below 400 mg/dL, while Raidal (1996) and Tyler-McGowan et al. (1997) noticed an increased susceptibility of foals to disease when IgG levels dropped below 800 mg/dL. Today, it is

common to define failure of passive transfer as IgG levels below 400 mg/dL serum (Tizard, 1996; Erhard et al., 2001) and partial failure as levels between 400 and 800 mg/dL. IgG levels above 800 mg/dL are considered necessary to provide optimal immune function (Erhard et al., 2001). In order to prevent failure of passive transfer, the minimal concentration of colostral immunoglobulin required has been estimated to be between 1,000 and 3,000 mg IgG/dL colostrum (LeBlanc et al., 1986). The possibility of failure of passive transfer cannot be evaluated until the foal is about 18 hours of age, as antibody absorption is essentially complete at this time (Tizard, 1996). Therefore, the standard industry practice of testing foal IgG levels at 12 hours after foaling may give misleading results, as complete antibody absorption has not happened yet. This practice may still be needed, however, in order to be able to administer plasma or colostrum to the foal before the foal's ability of absorption is completed.

Possible causes of failure of passive transfer fall into three categories: production failure, ingestion failure and absorption failure (Tizard, 1996). A failure of the mammary gland to concentrate immunoglobulins from the blood into colostrum can occur in maiden mares foaling for the first time. Premature lactation, however, is the most common production failure cause of failure of passive transfer. In this case, initial colostrum may be of adequate amount with adequate IgG concentration, but the mare will commence lactation prior to parturition. This steady leak of colostrum may occur for several hours or several days before birth and significantly reduces the amount of IgG available to the foal (Jeffcott, 1974a).

While less common, inabilities of ingestion and absorption are additional failure of passive transfer causes. Ingestion failures can arise from a mare not allowing her foal to

nurse, weak or deformed foals that take longer than normal to stand, or a delayed or defective suckling reflex (Jeffcott, 1974a; Tizard, 1996). Foals usually overcome these factors, but it may take longer than the 24-hour period of intestinal permeability to IgG. Absorption failures can be linked to stress at the time of parturition. The adrenal hormones play important roles in the onset of parturition and can influence changes in the permeability of small intestine cells after birth (Jeffcott, 1972). In conditions of stress at parturition, the mare or foal could produce abnormal amounts of corticosteroids which would therefore have a detrimental affect on the foal's antibody absorption (Jeffcott, 1974a).

Innate Immunity

Innate immunity is the first line of defense in fighting an invading organism (Goldsby et al., 2003). The skin, mucosal surfaces, macrophages and neutrophils all play important roles in the innate immune system. The skin and mucosal surfaces act as barriers against infection, and the macrophage and neutrophils act to phagocytize and kill invading foreign cells. Inflammation is also an important part of innate immunity and functions to draw immune cells to areas of injury or antigen attack. Because the innate immune system is less specific than the acquired immune system, the innate system can act quickly to begin an immune response (Goldsby et al., 2003).

Inflammation

By definition, inflammation is the response of tissue to the presence of microorganisms or injury (Tizard, 1996). Inflammation is a vital protective mechanism and the means by which defensive molecules and phagocytic cells gain access to the sites of tissue microbial invasion or damage. Inflammation is classified according to its severity and duration, with acute inflammation developing in less than an hour after

tissue damage and chronic inflammation occurring a much slower rate and being more constant. There are five symptoms of acute inflammation: heat, redness, swelling, pain and loss of function. These symptoms are a result of changes in the small blood vessels in the damaged tissue (Tizard, 1996; Goldsby et al., 2003).

Immediately following microbial invasion or injury, blood flow to the effected area greatly increases. This increase is due to a transient constriction of local arterioles and dilation of all the small blood vessels in the area. The blood vessel permeability is also increased, allowing fluid to move from the blood into the tissues where it causes edema and swelling (Tizard, 1996). The changes in blood vessels allow an influx of lymphocytes, neutrophils, monocytes and other immune cells into the area to participate in clearance of the antigen (Kuby, 1992). Neutrophils are the first immune cells to arrive in the inflamed tissues, followed by the slower moving monocytes. Once within the inflamed tissues, these cells are attracted to sites of bacterial growth and tissue damage and phagocytize and destroy any foreign material present. Monocytes will also remove dead and dying tissue (Tizard, 1996).

Cytokines play an important role in the acute-phase inflammatory response. These low-molecular-weight proteins secreted by macrophages exert a variety of effects on lymphocytes and other immune cells to regulate the intensity and duration of an immune response. The three cytokines that play the largest role in acute inflammation are tumor necrosis factor-α (TNF-α), interleukin 1 (IL-1) and interleukin 6 (IL-6). All three cytokines act locally on endothelial cells to induce coagulation and increase vascular permeability (Kuby, 1992). TNF-α, IL-1 and IL-6 also act on the brain to induce fever and suppress appetite and on skeletal muscle to drive protein catabolism and mobilize

available amino acids. In addition, these cytokines operate on liver cells to increase protein synthesis and secretion of clotting factors, complement components and protease inhibitors, all of which aid in the host defense (Tizard, 1996). All three cytokines activate B and T cells, while IL-6 can also increase immunoglobulin synthesis (Goldsby et al, 2003).

Effects of Dietary PUFA Supplementation on Inflammation and Immune Function

Blood and Tissue Responses to Experimental Feeding of n-3 PUFA

Numerous studies have investigated the levels of different n-3 PUFA resulting in the blood when feeding ALA and DHA, either alone or in combination. In humans, many studies have looked at providing adults with dietary fish oil, a good source of EPA and DHA. Helland et al. (1998) supplemented pregnant women with cod liver oil for 14 days, between three and eight weeks post partum. The women were divided into four groups: Group 1 served as the control and received no supplementation, Group 2 received 2.5 mL of cod liver oil/day, Group 3 received 5 mL of cod liver oil/day, and Group 4 received 10 mL of cod liver oil/day. Helland et al. (1998) found that the pregnant women in Groups 3 and 4 (receiving 5 and 10 mL of cod liver oil/day, respectively) showed a decreased plasma LA content and an increased ALA and DHA plasma content. When EPA and DHA intake were computed on a bodyweight basis, the women receiving 5 mL of cod liver oil were consuming the equivalent of 14 mg EPA and DHA/kg of bodyweight and the women receiving 10 mL of cod liver oil were consuming the equivalent of 28 mg of EPA and DHA/kg bodyweight.

Henderson et al. (1992) also supplemented pregnant women with EPA and DHA, but not in the form of fish oil. Henderson and coworkers supplemented pregnant women with six capsules of Bio-EFA for a total supplement weight of six grams of

supplementation. This supplement provided women with 1080 mg EPA and 720 mg DHA per day. Assuming an average bodyweight of 70 kg, this dosage provided 15.43 mg EPA/kg BW and 10.29 mg DHA/kg BW. Similar to Helland et al. (1998), however, Henderson et al. (1992) also started their supplementation period after lactation had already commenced, supplementing women between two and five weeks post partum for a total of 21 days. The results of Henderson et al. (1992) showed that daily supplementation of lactating women with 6 g of an EPA and DHA source increased the women's red blood cell content of EPA, DPA and total n-3 PUFA. Although it was not significant, there was also a trend toward red blood cell DHA increase. Infant red blood cells were also affected by supplementation of the mother, as EPA and DPA concentrations of infant red blood cells significantly increased after the supplementation period. However, similar to maternal results, there was no significant change in infant red blood cell DHA. Unfortunately, this study only used five women and their infants, so it may have been hampered by a small sample size.

Further evidence suggests that infants breast fed from omnivorous mothers have a higher DHA concentration in their red blood cells than do infants of vegan mothers (Sanders and Reddy, 1992). In fact, the difference in infant red blood cell DHA was quite large in this study, with infants feeding from vegan mothers having 1.9% of the total FA found in their red blood cells as DHA and infants feeding from omnivorous mothers having 6.2% of their total red blood cell FA as DHA. The content of DHA in breast milk reported by Sanders and Reddy (1992) was also significantly lower in vegan women when compared to omnivorous women (0.14 and 0.37% of fat, respectively). In addition, infants fed conventional formula (low in DHA) have consistently lower plasma

and red blood cell levels of DHA than infants fed breast milk, which is higher in DHA (Innis, 1991; Innis, 1992b).

The ability to increase blood concentrations of n-3 PUFA by feeding sources of these FA has also been documented in animals. Bauer et al. (1998) fed adult dogs either ground flax or sunflower seeds for 84 days and showed that plasma ALA, EPA and DPA were elevated when dogs were fed flaxseed, compared to when dogs were fed sunflower seeds. Plasma DHA, however, remained unchanged in the flax fed dogs, showing the difficulty in converting ALA to DHA. The flax fed dogs also showed a plasma reduction in AA and 22:5 n-6, providing evidence of competition between n-3 and n-6 PUFA for the Δ^6-desaturase in dogs (Bauer et al., 1998). However, the exact amount of supplement Bauer and coworkers added to the diets of the dogs was not stated, so it is therefore difficult to compare levels of supplementation to other studies.

In horses, Hansen et al. (2002) examined the effects of ALA supplementation on equine fatty acid status by feeding adult horses a diet consisting of 8% flaxseed oil for 18 weeks. Hansen and coworkers found that the flaxseed oil supplemented horses showed an increased plasma ALA and EPA compared to horses that did not receive any fat supplementation. On the other hand, there were no increases in DHA noticed. A weakness of this study, however, is the low sample size of only 12 horses (6 horses in the control group, 6 horses in the supplemented group). Duvaux-Ponter et al. (2004) also tested the effects of ALA on horses, but used pregnant mares and young foals as subjects. In this study, 26 pregnant mares were divided into two groups. The first group acted as the control and was fed extruded rapeseed (high in n-6 FA), while the second group was fed extruded linseed (high in n-3 FA). The mares were supplemented 1.5 months prior to

foaling until one month after foaling. While mare blood was not tested, supplementation with extruded linseed caused an increase in the ALA content of foal plasma from foaling until 4 weeks post parturition, and this increase was greater than the increase seen in foals nursing the mares given rapeseed (Duvaux-Ponter et al., 2004). However, the exact amount of linseed provided to the mares is unclear, as it was not stated in the paper.

The effects of feeding sources of EPA and DHA have also been documented in horses. Hall et al. (2004a) fed ten adult mares either menhaden fish oil or corn oil for a period of 14 weeks. Mares fed the menhaden fish oil consumed 22.93 g EPA per day and 19.58 g DHA per day. These amounts equated to a daily intake of 4.6 g EPA/100 kg bodyweight and 3.9 g DHA/100 kg bodyweight. As a result of this supplementation, Hall et al. (2004a) noticed higher plasma ALA, EPA and DHA and lower plasma LA in the mares fed fish oil compared to the mares fed corn oil. Brinsko et al. (2005) examined the effects of feeding a DHA source to stallions to determine the effects of FA supplementation on semen. In this study, eight stallions were used in a 2 x 2 crossover design. Stallions were fed a grain mix top-dressed daily with either 250 g of a commercial nutriceutical containing 30% n-3 FA (resulting in 75 g of n-3 FA) or a grain mix with no supplementation. Stallions were fed their respective diets for 14 weeks, separated by a 14 week wash out period, before treatments were switched for another 14 weeks of supplementation. The authors found that when stallions were supplemented with n-3 FA, their semen had almost three times the levels of DHA/billion sperm compared to when stallions were not supplemented. Even though the relative percentage of DHA in semen fatty acids was not significantly different between the two groups, treated stallions showed a 1.5-fold increase in their semen DHA:DPA ratio when they

received n-3 supplementation (Brinsko et al., 2005). Therefore, it seems that supplementation of DHA in horses may have the ability to affect the fatty acid composition of body constituents other than just the plasma.

Effects of PUFA supplementation on the acquisition of passive immunity in the foal

In order for IgG to be maximally absorbed into the foal's intestinal cells, the cell membranes must be fluid enough to allow the molecules to navigate through the membrane. Membrane bilayers tend to exist at the transition point between a fluid and solid-like (gel) state. The phospholipid fatty acyl chains present in membranes are one of the key chemical determinants of this balance. PUFA of the *cis* configuration tend to increase the fluidity of the membrane (Murphy, 1990; Mills et al., 2005). The fatty acid composition of membrane phospholipids is also easily changed by manipulation of dietary fat (Murphy, 1990), which, in turn, can influence membrane fluidity.

Brasitus et al. (1985) showed that adjusting the fatty acid composition of the diet in rats changed the composition of their enterocyte membranes. To do so, rats were fed either unsaturated or saturated triglycerides provided by corn oil or butter fat, respectively, for 6 weeks. The supplementation of corn oil, which is rich in LA, caused an enhanced fluidity of the membranes of several intestinal cells (Brasitus et al., 1985). When humans were supplemented with dietary n-3 PUFA, the fluidity of their erythrocyte membranes was substantially increased (Lund et al., 1999). In this study, 17 adults were supplemented with three 1 g capsules of fish oil per day for 42 days. Fluidity of the red blood cell membrane was determined by measuring the lateral diffusion coefficient of the fluorophore ODAF by fluorescence recovery after photobleaching. The results of Lund et al. (1999) suggest that supplementation with fish oil increased the lateral diffusion coefficient of ODAF, therefore increasing the membrane fluidity. This

increase in fluidity was seen at 21 days after supplementation and continued to rise until the termination of the study at 42 days (Lund et al., 1999).

A correlation between membrane fluidity and permeability was shown in young rats by Meddings and Theisen (1989). These researchers examined the changes that naturally occur in the membrane of jejunal microvilli in rats as they aged from 9 to 25 days. Study results showed a decreasing lipid fluidity as the rats aged, assessed by a steady-state fluorescence polarization technique. This decrease in membrane fluidity correlated to a decrease in membrane permeability (Meddings and Theisen, 1989). This correlation may suggest that if it is possible to enhance membrane fluidity by feeding n-3 FA, it may therefore be possible to augment the amount of IgG that travels both into the mare's mammary gland and across the foal's intestine.

To determine the effects of feeding PUFA on the IgG content of mare colostrum, Kruglik et al. (2005) fed mares either corn oil (rich in LA) or encapsulated fish oil (rich in EPA and DHA) from 60 days before foaling to 21 days after foaling. The mares fed encapsulated fish oil consumed 8.6 g of EPA and 10.4 g of DHA per day. The results of Kruglik et al. (2005) showed a higher presuckle colostrum IgG content in the fish oil fed mares, suggesting that supplementation with fish oil may have improved the fluidity and permeability of mammary epithelial cells. However, Kruglik et al. (2005) did not specify the amount of colostrum collected, and it has been reported that IgG amounts can vary between the first 250 and 500 mL of colostrum (Lavoie et al., 1989). Therefore, if the volume of colostrum gathered was different between mares, the differences seen in IgG may have been attributed to colostrum volume and not to treatment. Studies have also been performed that suggest that n-6 PUFA may increase mare colostrum IgG. Hoffman

et al. (1998) fed mares a high fat diet (10 % fat) through gestation and lactation. The fat in this diet was provided primarily by corn oil, which is high in LA. Colostrum IgG levels were higher in the mares fed the high fat diet, even though the dietary fat was rich in n-6 and not n-3 FA (Hoffman et al., 2004). However, the volumes of harvested colostrum were not stated in this study. In addition, colostrum was collected between 6 and 12 hours after foaling. Because the IgG of colostrum can vary dramatically in the first 12 hours after foaling (Pearson et al., 1984), colostrum IgG values obtained from mares in this study may have shown differences that were due to time and not treatment.

Studies have also been executed to determine the effect of feeding PUFA on foal IgG. Kruglik et al. (2005) showed that mares supplemented with fish oil produced foals that showed no differences in IgG levels when compared to foals born to corn oil fed dams, even though the same study showed a higher colostrum IgG in the mares fed fish oil. Kruglik et al. (2005) sampled foal plasma at 24 hours, so peak IgG content should have been reached. Therefore, it is unclear as to why the higher IgG levels in fish oil fed mare colostrum did not cause an increase in the plasma of foals born to these mares. Duvaux-Ponter et al. (2004) also failed to show a difference in foal serum IgG when mares were supplemented with a source of n-3 FA. In this study, mares fed extruded linseed did not produce foals with a higher serum IgG than mares fed extruded rapeseed (Duvaux-Ponter et al., 2004). Foal blood was again sampled at 24 hours after foaling in this study, so peak foal IgG should have been recorded. However, mare colostrum IgG was not tested, so it is unclear if ALA could increase colostrum IgG. More research needs to be done to determine the capability of dietary PUFA on modifying mare mammary and foal

enterocyte membrane composition and fluidity on the enhancement of passive transfer of IgG.

Effects of PUFA supplementation on the inflammatory response

Numerous studies have shown anti-inflammatory effects of n-3 PUFA. Sadeghi et al. (1999) fed groups of mice either a low fat diet (2.5% fat provided by corn oil) or diets high fat diets providing 20% fat by coconut (rich in medium chain FA), olive (rich in C18:1n-6), safflower (rich in LA) or fish oils. After 5 weeks on diet, mice were injected with 1.0 mL of phosphate-buffered saline containing endotoxin from *E. coli*. In the mice receiving the fish oil diet, lower plasma concentrations of the proinflammatory cytokines TNF-α, IL-1 and IL-6 were seen after the injection of endotoxin when compared to mice fed olive or safflower oils. However, coconut oil fed rats also showed decreased amounts of these cytokines (Sadeghi et al., 1999). Therefore, it is unclear if fish oil was specifically responsible for the decreased proinflammatory cytokine production, or if this difference was caused by a lack of n-6 PUFA. Unfortunately, the amount of feed offered to the mice was not provided by the authors, so the amount of consumed FA could not be calculated. Billiar et al. (1988) fed fish oil to rats for 6 weeks and observed a lower *in vitro* production of IL-1 and TNF-α by macrophages. Unfortunately, the fish oil fed rats in this study were compared to rats fed either corn or safflower oil, which are both high in n-6 FA. Therefore, it is again unclear if fish oil reduces proinflammatory cytokine production or if the feeding of n-6 FA increases cytokine production.

To test the inflammatory effects of PUFA in horses, Hall et al. (2004a) fed 10 adult mares 3.0% of their total diet (as-fed basis) with either corn oil or menhaden fish oil. After 14 weeks of supplementation, the fish oil supplemented mares had neutrophils

with a 78-fold greater concentration of the lesser inflammatory LTB_5 when compared to the neutrophils of mares fed corn oil (Hall et al., 2004a). In the same group of mares during the same supplementation period, production of TNF-α by bronchoalveolar lavage fluid (BALF) cells was increased in both groups, but only the corn oil fed mares had an increased production of inflammatory PGE_2 their BALF cells (Hall et al., 2004b). When mare BALF cells were stimulated with lipopolysaccharide, mares fed corn oil also showed a higher production of inflammatory PGE_2 (Hall et al., 2004b). Similar to the studies discussed previously, however, Hall et al. (2004a, 2004b) compared horses fed fish oil to horses fed corn oil and did not include a control group fed a diet without n-6 or n-3 FA supplementation. Because the diets of both groups of horses were altered through fat supplementation, it is unclear if the reported decreases in pro-inflammatory eicosanoids seen in horses fed fish oil would have resulted if these horses had been compared to a control group.

The delayed-type hypersensitivity (DTH) response has also been used to test the anti-inflammatory effects of n-3 PUFA. Meydani et al. (1993) supplemental adult humans with a low-fat, high-fish diet (26% calories from fat, 1.23 g EPA and DHA combined per day) or a low-fat, low-fish diet (25% calories from fat, 0.27 g EPA and DHA combined per day) for 24 weeks. This treatment period was compared to a previous 6 week period where subjects had been eating a current American diet of 35% of calories from fat and 0.8% of calories from n-3 FA. A delayed-type hypersensitivity (DTH) test was administered before and after the supplementation period using several different antigens, including tetanus toxoid and *Streptococcus* (group C). Results of Meydani et al. (1993) showed that the DTH response of adults consuming the low-fat,

high-fish diet was significantly less than the response of those consuming the low-fat, low-fish diet, with diameter measurements of the DTH reactions of the low-fat, high-fish diet participants being reduced by half.

Changing the n-6:n-3 ratio in dogs was also shown to effect the inflammatory response (Wander et al., 1997). In this study, dogs were fed diets containing n-6:n-3 ratios of 31:1, 5.4:1 or 1.4:1 for 16 weeks, where the n-6 FA was provided by corn oil and the n-3 FA was provided by fish oil. Dietary ratios were changed mostly by a reduction in LA simultaneous to an increase in EPA and DHA. When the diameter of a DTH response to keyhole limpet hemocyanin (KLH) was measured, dogs fed a n-6:n-3 ratio of 1.4 showed a much smaller reaction compared to the dogs fed ratios of 34:1 and 5.4:1 (Wander et al., 1997). Because the amount of n-6 FA in these diets decreased as the n:6-n:3 ratios decreased (as opposed to holding the amount of n-6 FA stable and increasing the amount of n-3), it is again unclear if differences seen in DTH response were strictly caused by the increase in n-3 FA. It is quite plausible that these differences may have been influenced by the decreasing n-6 FA. In contrast to dogs, the DTH response of horses sensitized with KLH showed no differences between horses fed 3% of the total diet (as-fed basis) either fish oil or corn oil (Hall et al., 2004b).

Effects of PUFA supplementation on disease resistance and survival

The majority of studies on inflammation and PUFA supplementation have shown positive results with n-3 PUFA, particularly when n-3 supplementation reduces n-6 FA in the diet. However, studies on the effect of PUFA on disease resistance show conflicting results. When guinea pigs were fed diets high in either n-3 FA (1.4% and 0.9% fat calories from EPA and DHA, respectively) or n-6 FA (15.4% fat calories from LA) for 13 weeks and infected with *M. tuberculosis*, the guinea pigs fed a diet high in n-3 FA

showed a higher number of mycobacteria recovered from the spleen, the most pronounced progression of the disease and a higher mean size of the tuberculin reaction (Paul et al., 1997). The authors suggested that possible explanations for these results may include the lower production of inflammatory mediators and the impairment in release of lysosomal enzymes that kill mycobacteria (Paul et al., 1997). The study performed by Paul et al. (1997) is possibly one of the best studies done to examine n-3 FA effects on disease resistance, as animals consuming both fat supplemented diets were compared to a no fat added control diet. In addition, the study utilized animals consuming the experimental diets but that were not infected. These animals therefore acted as uninfected controls within each diet. The benefits of a study design such as this is that a direct comparison can be made between the n-3 FA supplemented group and the control group, which in turn helps to determine disease effects are due strictly to the addition of dietary n-3 FA.

Another well designed study compared disease responses of mice infected with influenza (Byleveld et al., 1999). Challenging fish oil fed mice with influenza virus produced a higher lung viral load, lower body weights and impaired production of IgG and lung IgA when compared to mice fed beef tallow. Mice were fed fish oil or beef tallow at 20% of dietary fat for 14 days, after which half of the mice from each treatment were infected with influenza while the other half served as noninfected controls. D'ambola et al. (1991) supplemented newborn rabbits with high (5 g/kg) or low (0.22 g/kg) doses of fish oil, safflower oil or saline for 7 days after birth. When the young rabbits were supplemented with the higher levels of fat, both the fish and sunflower oil supplemented rabbits had an impaired ability to clear *Staphylococcus aureus* when

compared to the saline control group (D'ambola et al., 1991). However, the low doses of fish and safflower oil did not produce the same impaired ability to clear the bacteria. In light of these results, the authors of this study concluded that high does of both n-3 and n-6 FA can reduce the host's ability to kill *S. aureus* (D'ambola et al., 1991).

Positive results of supplementing with n-3 PUFA were shown when neonatal rat pups were infected with group B *streptococcus* (Rayon et al., 1997). In this study, researchers fed gestating rats a control diet (no fat added) or diets supplemented with either corn or menhaden fish oil. Supplementation was begun on day 2 of gestation and continued through lactation, but the amounts of diets and supplements fed were not provided by the authors. Rat pups were then infected with the *streptococcus* bacteria at 7 days of age. The results of Rayon et al. (1997) showed that pups from mothers who had been fed fish oil during gestation showed a significantly higher rate of survival (79%) than those born to corn oil fed dams (49%), though this difference was not significant. In this study, the lowered production of inflammatory mediators by fish fed rats when compared to corn oil fed rats may have been responsible for the higher survival rates, as group B streptococcal infections induce elevated levels of proinflammatory cytokines that lead to septic shock (Rayon et al., 1997).

Feeding fish oil to weanling mice has been shown to prolong mice survival to a murine retrovirus-induced immunodeficiency syndrome (MAIDS) that mimics human AIDS (Fernandes, et al., 1992). Mice in this study were fed diets consisting of 5% corn oil fed at an energy restriction of 40%, or diets fed ad libitum consisting of 5% corn oil, 20% corn oil or 20% menhaden fish oil. Mice were fed for 8 weeks before being injected with the MAIDS plaque-forming units (Fernandes et al., 1992). Mice fed both the 5%

corn oil energy restricted and 20% fish oil diets showed significantly longer survival rates than mice consuming the other diets. The authors explained the increase in survival rates of these two groups as a result of a slowed the progression of the MAIDS disease (Fernandes et al., 1992).

Thors et al. (2004) also showed positive immune effects on mice when feeding fish oil. In this study, 120 female mice were fed a standardized, control diet for 6 weeks before being divided into four groups and fed two different diets. The first two groups were fed a diet enriched with fish oil at 10% of total diet weight, and the remaining two groups were fed a diet enriched with corn oil at 10% of the total diet weight (Thors et al., 2004). However, the amount of time these diets were fed was not clear. Mice were intranasally inoculated with either Klebsiella or Streptococcus pneumoniae and the inoculum was aspirated into the lungs. Survival rates of the mice fed a fish oil diet and infected with Klebsiella pneumoniae were significantly higher than the rates seen in corn oil fed mice infected with the same disease. However, survival rates of mice infected with Streptococcus pneumoniae did not differ between the fish or corn oil fed mice (Thors et al., 2004).

In general, the conflicting results of studies examining the effects of dietary PUFA supplementation on disease resistance may be caused in part by study differences in the type of animal used, type and amount of pathogen utilized, route of pathogen infection and amount and duration of dietary PUFA supplementation. Because many of the above studies did not clearly state this information, it is difficult to establish which differences in disease response between studies could be attributed to PUFA treatment and which could be attributed to differences in experimental design. However, Anderson and

Fritsche (2002) suggest that conflicting results may be rooted in the host's ability to find a proper balance between the necessary and excessive production of various proinflammatory mediators.

Characteristics of Mare Milk

Mare Colostrum

Colostrum is the mare's first milk and is vital in transferring immunity to the newborn foal. It has a much thicker, stickier consistency than milk and is often a pale to deep yellow in color. Colostrum, produced in the mammary gland during the last trimester of pregnancy, is only secreted for a very short time (Lavoie et al., 1989). By 24-96 hours after foaling, mammary secretions have completely transitioned from colostrum to milk (Ullrey et al., 1966). Compositionally, colostrum is higher than milk in fat content (Csapó et al., 1995). The most important colostrum constituent, however, is the immunoglobulins. Colostrum has high concentrations IgG but lower IgM and IgA (Lavoie et al., 1989). Colostral IgG declines within the first 24 hours after birth. This decline often corresponds to the change of a thick, pale yellow fluid to one of a thinner consistency with a gray-white color (Pearson et al., 1984). Average colostral Ig concentrations are shown in Table 2-1.

Factors Affecting Mare Colostrum IgG Content

Premature lactation, or "prelactation," is considered the most important cause of failure of passive transfer in foals, as it is one of the main determinants of colostral IgG levels (Jeffcott, 1974). Causes of premature lactation include placentitis and/or placental separation, but the condition can occur without obvious placental pathology (Jeffcott, 1974b, 1975). Mares that experience prelactation for longer than 24 hours before foaling tend to have lower colostral IgG concentrations than those who lactate normally (Koterba

et al., 1990). Morris et al. (1985) found that as the proportion of mares on a breeding farm experiencing prelactation increased, so did the proportion of mares with low colostral IgG concentrations. In addition, the proportion of foals with low serum IgG concentration also increased.

Breed of mare may also affect colostral IgG concentration. Pearson et al. (1984) found a significantly higher IgG concentration of more than 5,000 mg IgG/dL colostrum in Arabian mares when compared to Thoroughbred mares. Average time from birth until colostrum IgG concentration declined to 1,000 mg/dL (the IgG concentration that cannot prevent failure of passive transfer) was 19.1 hours for the Arabians and only 8.9 hours for the Thoroughbreds. LeBlanc et al. (1992) founder higher IgG colostral concentrations in Thoroughbreds and Arabians when compared to Standardbreds. However, in another study, LeBlanc et al. (1986) reported no differences between IgG colostral concentrations in Thoroughbred, Quarter Horse, Arabian and Standardbred mares. The conflicting results seen between these two studies should not have been due to different sampling times or colostrum amounts taken, as both studies tested 10 mL of presuckle colostrum. Therefore, the conflicting results may be explained by differences in body size and weight between breeds. Larger breeds can sometimes produce larger volumes of colostrum, and this large volume may lead to a dilution effect. However, the age of the mare, number of lactations and herd management are factors that probably influence colostral IgG concentration. Additionally, there is large individual variation in colostral IgG content, making it difficult to attribute differences in colostral IgG as purely breed oriented (Pearson et al., 1984). Further studies are needed to examine what, if any, influence breed has on colostral IgG content.

Conflicting results exist in regard to the connection between a mare's age and her colostrum quality. In a study involving Standardbred, Thoroughbred and Arabian mares, mares between the ages of 3 and 10 years had the highest colostral IgG concentration and FPT was most prevalent in foals born to dams of over 15 years (LeBlanc et al., 1992). However, Morris et al. (1985) and Erhard et al. (2001) saw no significant effects of age on IgG in mares of varying breeds. Both LeBlanc et al. (1992) and Erhard et al. (2001) sampled colostrum before the foal had been allowed to suckle. However, Morris et al. (1985) sampled colostrum during the first 2 hours after foaling. Since Morris et al. (1985) sampled colostrum at a later time than the other two studies, any difference seen in the colostrum of this study could have been attributed to time. However, time should not have affected the values of LeBlanc et al. (1992) and Erhard et al. (2001). Therefore, discrepancies in data reflecting the effect of mare age of colostrum IgG could be explained by outside factors such as individual mare variation and management differences.

Composition of Mare Milk

In mares kept without human influence, lactation lasts about one year, and drying of the udder occurs several weeks to several days before the next foaling. There have been, however, extreme cases noted of 2- or 3-year-old suckling foals (Feist and McCullough, 1976). Today, the drying process is initiated by weaning foals at 4-6 months of age. Actual daily lactation yields of nursing mares are not well known, but are estimated to be between 10 and 30 kg for light breed nursing mares (Doreau and Boulot, 1989). Peak lactation seems to occur at about two months postpartum (Bouwman and van der Shee, 1978).

Compositionally, the fat content of mare milk is very low (Doreau and Boulot, 1989) but can be influenced by diet. Milk fat is also influenced by mare body condition at foaling, with fat mares producing milk with a higher fat content than thin mares. The increased lipid mobilization of fat mares may be explained this phenomenon (Doreau et al., 1993). Crude protein in milk exists at between 1.7 and 3.0% (Doreau and Boulot, 1989) and decreases throughout lactation (Oftedal et al., 1983). Mare milk is different from the milk of other species as it contains higher amounts of the amino acids cystine and glycine (Doreau and Boulot, 1989). Milk carbohydrates are almost entirely made of lactose, with very low levels of free glucose. Mare milk is also extremely low in ash, with 0.7% as extreme (Doreau and Boulot, 1989). Milk is also different than colostrum in the amounts of immunoglobulins present. Levels of IgG, IgA and IgM all decrease as colostrum transitions into milk and IgA becomes the predominant Ig present (Norcross, 1982). Average immunoglobulin concentrations in mare milk are shown in Table 2-1.

Peak colostrum IgG content is observed at foaling and rapidly declines during the first 24 hours after foaling (Lavoie et al., 1989). In colostrum sampled within 2 hours after foaling, mean IgG values were shown to be 16,583 mg/dL (Lavoie et al., 1989). At 4 hours post foaling, another study showed mean IgG values that were at lower levels of 5,450 mg/mL, and these values fell even further to 1,010 mg/dL by 9-12 hours after foaling (Erhard et al., 2001). Colostrum IgG fell below 1,000 mg/dL by 13-16 hours post foaling and continued to decrease until day 14 (Erhard et al., 2001). Duvaux-Ponter et al. (2004) showed that these low milk IgG levels did not show any changes by 21 days after foaling, suggesting that mare milk IgG levels stay at this low level for the duration of lactation.

Effect of Diet on Fat and Fatty Acid Composition of Milk

Milk fatty acids are either synthesized *de novo* by acetyl-CoA carboxylase and fatty acid synthase or are supplied exogenously. The mammary epithelial cells of lactating animals are highly active in triglyceride biosynthesis (Clegg et al., 2001). If the FA are not synthesized in the mammary epithelial cells, they can enter the cells either from albumin in the plasma or from hydrolysis of chylomicron triglycerides by lipoprotein lipase. Once inside the cell, FA are bound to fatty acid binding protein in the cytoplasm or activated with acetyl-coenzyme A (CoA) and used for triglyceride synthesis. The endoplasmic reticulum synthesizes microlipid droplets that fuse to form cytoplasmic droplets which move to the apical membrane where they are enveloped to form the milk fat globule. This globule is then secreted in a membrane-bound form into the milk (Neville and Picciano, 1997).

Mare milk contains relatively little fat, with triglycerides as the predominate lipid class (Dils, 1986). Mare milk naturally contains very small quantities of stearic (C18:0) and palmitoleic (C16:0) acids and high quantities of linolenic (C18:3n-3) and linoleic (C18:2n-6) acids (Csapó et al., 1995). The higher amounts of unsaturated FA are explained by the fact that horses consume large amounts of forages rich in unsaturated FA (Csapó et al., 1995). Milk composition, however, may be changed by manipulating the diet, with the largest effects seen in the fat content (Sutton and Morant, 1989). Mare milk long-chain FA composition is strongly related to the FA composition of the diet, as no microbial FA hydrogenation occurs before intestinal absorption in horses (Doreau et al., 1992; Hoffman et al., 1998).

The ratio of forage to grain in the mare's diet can effect her milk composition. Generally, fat content decreases as the percentage of grain increases (Doureau and

Boulot, 1989). Doreau et al. (1992) fed nursing mares diets containing either 95% hay and 5% grain or 50% hay and 50% concentrate. Milk fat concentrations were higher for the mares fed the 95:5 forage:grain diet compared to the 50:50 forage:grain diet. The mares eating mostly forage also had higher linolenic and lower linoleic acid milk contents than those eating mostly grain (Doreau et al., 1992). This effect is understandable considering the fact that forage is high in linolenic acid. However, because exact amounts of hay and grain fed and the fat composition of the diet ingredients was not given, it is difficult to determine accurate values for percent fat of each diet.

Studies in humans have also examined the effect of dietary fat on milk fat composition. Henderson et al. (1992) found that supplementing pregnant women with 6 g of an EPA and DHA supplement for 21 days significantly increased EPA, DPA and DHA and decreased total n-6 PUFA levels in breast milk when compared to pre-supplementation levels. Helland et al. (1998) observed an increase in EPA and DHA in breast milk when women were supplemented with 5 and 10 mL cod liver oil daily for 14 days compared to women receiving 5 mL of cod liver oil/day and those receiving no supplementation. The changes in breast milk FA composition reported by Henderson et al. (1992) and Helland et al. (1998) were noted as early as day two of supplementation. Interestingly, daily supplementation of women with 20 g of flaxseed oil (approximately 10.7 g ALA/d) for 4 weeks increased the EPA and DPA breast milk content but failed to produce an increase in DHA (Francois et al., 2003). The authors speculated that the excess ALA supplied from flax oil may have competitively inhibited Δ^6-desaturase from converting DPA to DHA (Francois et al., 2003).

In dogs, feeding fat supplements with varying ratios between ALA and the sum of EPA and DHA produced milk fat compositions highly correlated to the diet fed (Bauer et al., 2004). Dogs were fed one of four diets containing 15% total fat as beef tallow and varying amounts of linseed and menhaden fish oil to provide specific levels of ALA, EPA and DHA. The diets were formulated as follows: the Lo/Lo diet contained 0.14% ALA and 0.04% EPA and DHA, the Lo/Mod diet contained 0.29% ALA and 0.24% EPA and DHA, the Lo/Hi diet contained 0.20% ALA and 0.66% EPA and DHA and the Hi/Lo diet contained 6.82% ALA and 0.04% EPA and DHA (fatty acids are expressed as a percentage of dry matter). Bitches fed the Hi/Lo diet had the highest milk ALA content, while bitches fed the Lo/Hi diet had the highest EPA and DHA milk content. Milk responses of EPA, DPA and DHA content were seen as a function of increasing dietary n-3 PUFA content. There was no enrichment of DHA when the Hi/Lo diet was fed, showing that ALA is inefficiently converted to DHA in the dog (Bauer et al., 2004).

Davidson et al. (1991) showed that mares fed a diet with 5% added fat produced milk with a higher fat content than mares who were not supplemented with fat (2-3% dietary fat). However, no differences in milk fat production were noted when mares were fed a sugar and starch diet with 2.4% fat compared to a fat (corn oil) and fiber diet with 10.4% fat (Hoffman et al., 1998). Nonetheless, the FA composition of the milk mirrored the FA supplied by the diet. The mares eating the high fat diet showed higher milk concentrations of LA and lower concentrations of ALA, which can be explained, in part, by the n-6 PUFA content of the corn oil (Hoffman et al., 1998). Spearman et al. (2005) found that feeding gestating mares a mix of corn oil and linseed oil increased milk ALA content when compared to mares fed corn oil. Duvaux-Ponter et al. (2004) observed

higher levels of ALA in mare milk when mares were supplemented with linseed oil. Feeding mares 454 g of encapsulated fish oil per day increased EPA and DHA in the milk but did not affect the ALA content when compared to mares fed corn oil (Kruglik et al., 2005). Together, these studies show that the fat content and FA composition of the mare's diet can influence milk composition.

Fatty Acid Transfer across the Placenta

The placenta is a pivotal organ in providing the developing fetus with essential fatty acids. During the last trimester of pregnancy, fetal requirements for AA and DHA are especially high due to rapid synthesis of brain tissue. To obtain these FA, the fetus depends upon placental transfer, and thus on the FA status of the mother (Al et al., 2000). Much of the research of placental FA transfer has been performed in humans, who possess a discoid hemochorial placenta. In these studies, there has been considerable evidence of transfer of ALA, EPA and DHA across the placenta (Innis, 2005). This transfer is a multi-step process of FA uptake by fatty acid binding proteins and intracellular translocation of the FA from the maternal to fetal environment. The fatty acid binding proteins that facilitate this process favor the uptake of n-6 and n-3 PUFA over non-essential FA (Innis, 2005).

Human placental preference for transfer of FA has been reported by one author to be DHA>ALA>LA>AA (Haggarty et al., 1997), while others have speculated that DHA and AA are preferred over all other FA (Campbell, 1996; Crawford, 2000).
Fetal plasma concentrations of AA and DHA are reported to be 300- to 400-fold higher than maternal plasma levels while their LA and ALA levels are lower (Elias and Innis, 2001). However, human placenta does contain Δ^6- and Δ^5-desaturases (Innis, 2005), so

the higher concentration of AA and DHA in fetal circulation may be partially produced by placental conversion of these FA from their 18 carbon precursors.

Human studies have shown that the maternal dietary intake of n-6 and n-3 PUFA influences placental transfer of AA and DHA. Connor et al. (1996) supplemented pregnant women with sardines and fish oil from the 26th to the 35th week of pregnancy in amounts to provide 2.6 g of n-3 FA per day. When DHA blood levels of newborn infants born to supplemented women were compared to those of newborn infants born to unsupplemented women, newborn babies born to supplemented mothers had 35.2% more DHA in red blood cells. Infants from supplemented women also showed a plasma DHA content 45.5% higher than infants from unsupplemented mothers, concluding that placental transfer of DHA in women is increased by maternal supplementation with DHA (Connor et al., 1996). However, de Groot et al. (2004) reported that supplementing pregnant women with ALA did not increase umbilical cord blood DHA, suggesting that the placenta could not efficiently convert ALA to DHA. In this study, pregnant women were supplemented daily with either 9.02 g LA and 2.82 g ALA (experimental group) or 10.94 g LA and 0.03 g ALA (control group) in the form of margarine. Supplementation was provided from week 14 of pregnancy until delivery (de Groot et al., 2004). While the umbilical venous plasma obtained from the subjects at delivery showed no differences in DHA content between groups, the experimental group did show an EPA concentration twice that of the control group, suggesting that conversion of ALA to EPA in the human placenta may be possible (de Groot et al., 2004).

In spite of its complex six-layered placenta, the transfer fatty acids from mare to fetus is possible. Equine studies, while few in number, have shown a positive correlation

between maternal and umbilical vein plasma free FA levels (Stammers et al., 1991). However, the same study also showed a difference in FA composition between maternal and umbilical vein plasma. The phospholipids portion of the umbilical venous plasma contained more longer chain derivatives of LA and ALA than was found in maternal plasma, suggesting that these longer chain FA were of placenta origin, because maternal plasma phospholipids in the horse contain very little longer chain PUFA (Stammers et al., 1991).

The presence of Δ^6 or Δ^5-desaturase, to the author's knowledge, has not been established in the equine placenta. However, many studies have produced results that would imply these enzymes are present. In natural situations, long-chain PUFA (particularly DHA) are virtually absent from maternal circulation and in very low concentrations in other maternal lipid compartments. In spite of this occurrence, foal plasma phospholipids are rich in long-chain PUFA which must therefore be provided to the foal by placental formation and transfer (Stammers et al., 1987). Stammers et al. (1988) showed that foals had higher plasma concentrations of AA, EPA and DHA than did their dams. A 30 hour fast of the pregnant mares resulted in an even greater fetal concentration of these fatty acids, resulting from the increased lipid mobilization in the mares (Stammers et al., 1988). When Stammers et al. (1994) incubated equine placenta in media enriched with LA, the lipid fractions released from the placenta consisted of long-chain PUFA derivatives of LA such as C20:3n-6, C20:4n-6 and C22:6n-6. This finding suggests that these PUFA would be seen in the umbilical plasma lipids rather than the maternal plasma lipids. No studies exist in mares to test the ability of manipulation of

dietary fat to influence placental FA transfer, so much research needs to be done in this area.

Conclusions

Because many of the studies investigating the effects of feeding n-3 FA to the horse have not utilized a true control group that received no fat supplementation, research is needed to compare the effects of n-3 supplementation with no n-3 supplementation (i.e., unaltered diet). This is especially important considering the fact that high forage diets contain significant quantities of n-3 FA, but the addition of grain to the diet shift the proportion of FA in favor of n-6. Studies comparing n-3 FA supplementation to baseline diets are needed to validate that the biological effects observed when feeding n-3 FA are truly due to the increase in these FA, and not to a decrease in n-6 FA.

In addition, little research has addressed responses yielded by different n-3 FA (e.g., ALA, EPA, DHA) to determine if differences in dietary FA source can influence biological responses in the horse. In particular, little data exists that compares the effects of different n-3 FA sources fed to the mare and the subsequent response of her nursing foal. It is unclear if supplementing the mare during gestation with n-3 FA can affect the IgG composition of her colostrum and milk and subsequently the IgG concentration in her foal. Furthermore, it is unknown if increasing the gestating mare's n-3 FA intake can result in greater placental transfer of n-3 FA, therefore allowing the foal to be born with an already elevated level of these FA.

Lastly, clear effects of supplementation with ALA or an EPA/DHA combination on the inflammatory response in horses have yet to be elucidated. Therefore, in an attempt to answer some of these questions, the objectives of this study were:

1. Examine the effect of dietary n-3 supplementation of mares on the FA composition of mare milk and mare and foal plasma and red blood cells;
2. Examine the difference of efficiencies of ground flaxseed (ALA) and encapsulated fish oil (EPA and DHA) in augmenting EPA and DHA in the mare and foal;
3. Determine if n-3 FA supplementation of the mare can increase the IgG content of colostrum, milk and foal plasma.
4. Determine in supplementation with flaxseed or fish oil can alter the inflammatory response in mares and foals.

CHAPTER 3
MATERIALS AND METHODS

Animals

This trial used 36 pregnant Thoroughbred (n=24) and Quarter Horse (n=8) mares and their subsequent foals. Mare age ranged from 4 to 20 years with a mean of 10.5 ± 4.1 years (mean ± SE). Mares were paired according to breed and stratified according to expected foaling date before being assigned to three treatment groups. The order of treatment assignment was determined by numbering three pennies, each penny corresponding to a separate treatment, and placing them into a hat. Pennies were then drawn at random to determine the order of treatment assignment. Treatment groups were then balanced for mare age and parity.

For the duration of this trial, mares and foals were housed at the University of Florida's Horse Research Center in Ocala, Florida. Pregnant mares were housed on pasture until signs of foaling were evident. At this time, mares were moved into small paddocks until foaling. All mares, with the exception of one, foaled outside. After foaling, mares and foals were kept in a box stall for 24 hours and then turned out in a small paddock for one week before being returned to pasture. A routine vaccination and anthelmintic schedule was followed for all animals. This experiment was performed in accordance with the regulations and approval of the Institutional Animal Care and Use Committee of the University of Florida.

Diets and Treatments

The basal diet for all treatment groups consisted of a commercial grain-based concentrate (Gest-O-Lac; Ocala Breeders Sales, Ocala, Florida) and pasture or hay. The grain-based concentrate was offered at 1.0% BW in late gestation and 1.0-2.0% BW during lactation in order to maintain bodyweight and a minimum body condition score of 5. The concentrate was formulated to meet or slightly exceed nutrient requirements for late gestation and lactation based on NRC recommendations (NRC, 1989). From December to March, mares were fed Coastal bermudagrass hay *ad-libitum* and had access to dormant bahiagrass pasture. From April to June, mares only had access to bahiagrass pasture. Trace mineralized salt blocks were available at all times. Foals were provided with access to the same grain-based concentrate that was fed to mares via creep feeders that were placed in the pasture.

Mares received one of three treatments: 1) basal diet with no supplementation (CON, n = 12); 2) basal diet supplemented with milled flaxseed (Pizzey's Milling, Manitoba, Canada; FLAX, n=12); or 3) basal diet supplemented with encapsulated fish oil (United Feeds, Inc., Indiana; FISH, n = 12). Both FLAX and FISH were fed to mares in amounts to provide 6 g total n-3 FA/100 kg BW per day. This level of supplementation was chosen based on the studies of O'Connor et al. (2004) and Siciliano et al. (2003) which demonstrated changes in plasma fatty acid composition when horses were supplemented with similar levels of fish oil. Mares and foals were brought in from pasture at 0700 and 1500 h each day, placed into box stalls and individually fed the grain mix concentrate. Half of the daily allotment of flaxseed or fish oil supplement was hand mixed into the grain provided in the morning feeding and the remaining half of the supplements were mixed into the grain provided in the afternoon feeding. Foals had the

opportunity to share the mares' feed, but this depended upon the individual temperament of each mare. Supplementation began 28 days before the expected foaling date and continued until 84 days post-partum.

The nutrient composition of the grain-based concentrate and the flaxseed and encapsulated fish oil supplements is presented in Table 3-1. The nutrient content of the Coastal bermudagrass hay and bahiagrass pasture is presented in Table 3-2.

Table 3-1. Nutrient composition of the grain mix concentrate and the milled flaxseed and encapsulated fish oil supplements

Nutrient[1]	Concentrate	Flaxseed	Fish Oil
DM, %[2]	92.7	91.5	91.2
DE, Mcal/kg[3]	3.41	3.0	3.7
CP, %	15.5	22.9	11.8
ADF, %	11.9	19.0	5.9
NDF, %	26.3	40.0	9.9
Fat, %	4.2	37.7	21.5
Ca, %	1.06	0.24	0.33
P, %	0.70	0.74	0.15
Zn, mg/kg	248	41	33
Cu, mg/kg	64	11	4

[1] Values are presented on a 100% DM basis (except DM).
[2] DM, dry matter; DE, digestible energy; CP, crude protein; ADF, acid detergent fiber; NDF, neutral detergent fiber.
[3] Calculated using the equation: DE (Mcal/kg) = 4.07 – 0.055(%ADF) (NRC, 1989).

Table 3-2. Nutrient composition of the bahiagrass pasture (by month) and Coastal bermudagrass hay

	Pasture							
Nutrient[1]	Dec.	Jan.	Feb.	March	April	May	June	Hay
DM, %[2]	45.2	64.1	65.0	44.1	30.5	23.0	17.5	90.8
DE,Mcal/kg[3]	1.9	1.9	2.0	1.9	2.2	2.4	2.3	1.9
CP, %	11.2	10.7	11.7	11.8	16.0	19.2	19.4	8.9
ADF, %	42.6	41.4	40.2	43.5	35.4	34.4	36.6	38.1
NDF, %	69.3	68.5	64.5	67.7	54.7	59.4	63.0	71.4
Fat, %	1.9	2.1	2.5	2.4	3.1	3.1	3.0	1.5
Ca, %	0.53	0.61	0.68	0.64	0.72	.50	0.40	0.35
P, %	0.29	0.24	0.25	0.25	0.36	0.39	0.40	0.21
Zn,mg/kg	27	38	36	40	32	33	30	46
Cu,mg/kg	6	6	6	8	8	8	9	5

[1] Presented on a 100% DM basis (except DM).

[2] DM, dry matter; DE, digestible energy; CP, crude protein; ADF, acid detergent fiber; NDF, neutral detergent fiber.

[3] Calculated using the equation: DE (Mcal/kg) = 4.22 – 0.11(%ADF) + 0.0332(%CP) + 0.00112(%ADF)^2 (NRC, 1989).

Bodyweights

Mares were weighed at 28 and 14 d prior to expected foaling date (d-28, d-14), at foaling (d0) and every 14 days thereafter. Foals were weighed at birth (d0) and every 14 days thereafter. A digital livestock scale with an accuracy of ± 0.5 kg was used to obtain body weights.

Blood Sample Collection and Processing

Blood samples were collected from mares by jugular venipuncture at 28 and 14 d prior to expected foaling, at foaling (d0), and at 28, 56 and 84 d after foaling for acquisition of plasma, serum and/or red blood cells. Blood samples were collected from

foals via jugular venipuncture at birth before the foal was allowed to nurse (d0), 36 h post-parturition, and 7, 28, 56 and 84 d post-foaling for acquisition of plasma, serum and/or red blood cells. A square patch of hair was shaved over the foal's jugular vein to allow for easier blood sampling. Precision Glide Vacutainer brand blood collection needles (20G, 1 ½ in. for mares; 20G, 1 in. for foals) were used to collect blood into Beckton Dickinson Vacutainers containing sodium heparin, to facilitate harvesting of plasma and red blood cells, or tubes containing no anticoagulant for harvesting of serum. With the exception of samples obtained at birth or 36 h post-parturition, all blood samples were collected between 0700 and 0900 h and prior to the mare's morning grain feeding. After collection, blood samples were immediately placed on ice and transported to the Animal Nutrition Laboratory for further processing.

In the laboratory, blood samples for obtaining serum were allowed to clot for 30 min to 1 h and then centrifuged at 5590 x g for 7 min to allow for separation of serum. Serum was collected with plastic disposable pipets and aliquoted into polypropylene cryogenic vials (2-3 vials, 0.5-1.0 mL each). Samples were frozen at -80°C until further analysis for IgG using a commercially available single radial immunodiffusion kit (SRID Kit, VMRD, Inc., Pullman, WA). See Appendix B for a description of the IgG analysis.

Blood samples for obtaining plasma and red blood cells were first used to determine the hematocrit (packed cell volume). Hematocrit values were determined in duplicate using whole blood drawn into a microcapillary tube, centrifuged and read on a microcapillary reader. After determination of hematocrit, each vacutainer was gently rotated and 5.0 mL of whole blood was pipetted into a separate glass tube, labeled, and centrifuged at 5590 x g for 15 minutes to separate the plasma and red blood cells. A

pipet was used to transfer 1.0 mL of plasma to each of four polypropylene cryogenic vials. Samples were frozen on a slant at -20°C to increase the surface area and ensure more efficient freeze drying before being stored at -80°C until further analysis of fatty acids.

Once plasma had been removed, an aspirator was used to remove any additional plasma and the thin layer of while blood cells lining the top of the red blood cells in each tube. Two mL of cold saline was then added to each tube and the tubes were gently mixed and centrifuged at 5590 x g for 7 min. After centrifuging, the supernatant was aspirated off and an additional 2.0 mL of cold saline was added. The tubes were again mixed and centrifuged at 5590 x g for 7 min. This procedure was repeated once more for a total of three saline washes. After the supernatant of the final wash had been aspirated off, exactly 2.0 mL of cold saline was added to the remaining red blood cells in each tube. The tubes were mixed well before 2.0 mL of the red blood cell suspension was transferred into labeled polypropylene cryogenic vials. These tubes were frozen at an angle at -20°C before being placed into storage at -80°C until analyzed for fatty acid content.

Colostrum and Milk Collection and Processing

Colostrum was obtained from the mare within 1 h of birth and before the foal had suckled (d0). Approximately 120 mL of colostrum was recovered into a pre-labeled, pre-weighed plastic cup. The cup was covered with a lid and stored at 4°C until transfer to the Animal Nutrition Laboratory for processing.

Milk samples were obtained 36 h post-partum and between 0700 and 0900 h on 7, 14, 28, 56 and 84 d post-foaling for determination of fatty acid and IgG content. To facilitate milk collection, foals were muzzled for approximately 30 min to allow the

mare's udder to fill. The entire udder was then milked out into a pre-labeled, pre-weighed plastic cup. If the udder contained more milk than one cup could hold, the udder was milked out into multiple cups whose content was then mixed in a larger container and approximately 120 mL was transferred to the pre-labeled, pre-weighed sample cup. The excess milk was discarded. After collection, milk samples were immediately placed on ice and transported to the Animal Nutrition Laboratory for further processing.

In the laboratory, colostrum and milk samples were gently swirled to mix and strained through four layers of cheesecloth to remove any dirt and debris in the sample. The samples were then returned back to the original pre-weighed sample cups. After straining, the sample was mixed again and approximately 1.0 mL was aliquoted into each of three pre-labeled polypropylene cryogenic vials. These vials were then stored at -80°C until further analysis for IgG content. The remaining colostrum or milk sample was weighed to determine a wet sample weight and then freeze dried. Freeze dried milk samples were stored at -20°C until used for the determination of fatty acid composition.

Fatty Acid Analysis

Fatty acids in plasma and red blood cells were extracted and methylated using the procedure of Folch et al. (1957). Fatty acids were analyzed by gas chromatography (CP-3800 Gas Chromatograph, Varian, Inc., Palo Alto, CA) using a WCOT fused silica column (CP-SIL 88, length100 m, internal diameter 0.25 mm, flow rate 5.0 mL/min, Varian, Inc., Palo Alto, CA). The carrier gas was helium with a pressure of 29.5 psi (1 min), 35.4 psi (0.42 psi/min, total of 45 min) and 37.9 psi (0.17 psi/min, held for 50 min, total of 110 min). The temperature program was 120°C for 1 min, increased to 190°C at 5°C/min and held at 190°C for 30 min (total of 45 min), increased to 220°C at 2°C/min and held at 220°C for 50 min, giving a total run time of 110 min. Fatty acids were

identified by comparison of peak retention times for samples and reference standards (Nu-Chek Prep, Inc., Elysian, MN). The FA identified included C8:0, C10:0, C12:0, C14:0, C14:1, C16:0, C16:1, C17:0, C17:1, C18:0, C18:1n-9, C18:2n-6 (LA), C18:3n-3 (ALA), C20:0, C20:1, C20:2, C20:3, C20:4n-6 (AA), C20:5n-3 (EPA), C22:0, C22:5n-3, C22:6n-3 (DHA) and C24:1. Nonadecanoic acid (C19:0) was added to the samples and used as an internal standard to assess FA recovery. Total n-6 FA were defined as the sum of C18:2 n-6 and C20:4n-6 while total n-3 FA were defined as the sum of C18:3n-3, C20:5n-3, C22:5n-3 and C22:6n-3.

Intradermal Skin Test

To examine the effect of n-3 FA supplementation on the inflammatory response, mares and foals were sensitized with phytohemagglutinin (PHA; Lectin from *Phaseolus vulgaris*, Sigma-Aldrich, Inc., St. Louis, Missouri) at 84 d post-partum. Twenty-five milligrams of PHA was reconstituted in 16.7 mL of phosphate buffered saline (PBS) to give a final concentration of 150 μg/100 μL. A 4 x 4 cm patch of hair was surgically clipped on the midsection of both sides of the neck on mares and foals and injected intradermally with 100 μL of the PHA suspension. Precision Glide brand intradermal injection needles (26 G, 3/8 in.) were used to deliver the PHA. Needles were changed between each injection site on the right and left side of the neck. Skin thickness measurements were obtained by pinching the skin between the thumb and forefinger and measuring the skin fold thickness in mm with an electronic digital micrometer (Marathon Watch Company, Ltd., Ontario, Canada). Measurements of each injection site were obtained after clipping but before injecting (h 0) and at 2, 4, 6, 8, 12, 24 and 48 h after injection. Skin thickness measurements from the right and left sides of the neck were averaged to give a single thickness measurement for each time point.

Supplement and Feed Sample Analysis

The same batch of milled flaxseed, encapsulated fish oil and Coastal bermudagrass hay were available for the duration of the trial. However, the source of commercial grain mix was replenished approximately every 2 wk due to storage limitations and the volume of feed needed. Samples of the flaxseed, encapsulated fish oil and grain mix were obtained at 4 wk intervals. These samples were then dried at 60°C and stored at 20°C for later analysis. Samples of bahiagrass pasture were obtained at 4 wk intervals from four, 16 ha pastures. Pasture grass clippings were only obtained from areas where grazing was evident. At each 4-wk collection, clippings from the four pastures were composited, dried at 60°C and stored at 20°C for later analysis. Throughout the trial, each round bale of Coastal bermudagrass hay that was offered to the mares was core sampled (5 cores per bale), dried at 60°C and stored at 20°C. After the completion of the trial, all samples of Coastal bermudagrass hay were composited into one sample for analysis. All feeds and supplements were analyzed for fatty acid content using the method described above. In addition, feeds were analyzed for DM, DE, CP, NDF, ADF, total fat, Ca, P, Mg, Zn and Cu by wet chemistry (Dairy One Forage Analysis Lab, Ithaca, NY).

Statistical Analysis

Four mares either delivered dead foals or their foals died shortly after birth. Only pre-foaling data was used from these mares. One mare experienced a red bag during foaling and her foal was subsequently given plasma, so IgG data from this mare and foal were not included in the statistical analysis. One foal was euthanized at 30 d of age, so only data taken up to that time point were used. The final number of mare and foal pairs successfully completing this study was 11 CON, 11 FLAX and 9 FISH.

The MIXED procedure of SAS (V. 9.1, SAS Inst., Inc., Cary, NC) was used to analyze fatty acid composition of colostrum, milk, plasma and all feeds, IgG content of colostrum, milk, mare serum and foal serum, and mare and foal bodyweights. The sources of variation included treatment, time and treatment x time interaction. Breed effects were also tested for mare and foal bodyweights, mare serum IgG, mare colostrum and milk IgG and foal serum IgG. Sex effects were tested for foal serum IgG. In addition, principle forage source (hay or pasture) was examined as a main effect for mare plasma and red blood cell fatty acids. Fatty acids analyzed included linoleic (LA), α-linolenic (ALA), arachidonic (AA), eicosapentaenoic (EPA) and docosahexaenoic (DHA) acids as well as total n-3 and total n-6 fatty acids and the ratio of n-6:n-3 fatty acids. For IgG and fatty acid analysis, horse nested within treatment was considered as a random variable and used as an error term to test the effects of all sources of variation. Dunnett's test was used to separate means. The homogeneity of regression for skin thickness values was evaluated using the GLM and MIXED procedures in SAS. Horse nested within treatment was used as an error term.

Due to missing values and unbalanced treatment groups after foaling, all data are expressed as least square means ± SE unless otherwise stated. Values were considered significant at $p \leq 0.05$ and trends were considered at $p \leq 0.10$.

CHAPTER 4
RESULTS

Feed and Supplement Analysis

The fatty acid composition of the grain mix concentrate and the flaxseed and encapsulated fish oil supplements is presented in Table 4-1. The fatty acid composition of pasture forage was similar from December through March (Appendix A). Therefore, the fatty acid composition of pasture samples collected in December, January, February and March was averaged and presented as "winter" pasture (Table 4-2). Similarly, the fatty acid composition of pasture forage was not different between the months of April, May and June (Appendix A). Thus, the fatty acid composition of pasture samples from these months was also averaged and presented as "spring" pasture (Table 4-2).

Spring pasture contained lower quantities of C18:0 ($P = 0.01$) and C18:1 ($P = 0.004$) and higher quantities of ALA ($P = 0.03$) and total n-3 FA ($P = 0.03$) than winter pasture (Table 4-2). Hay contained higher concentrations of C16:0 ($P = 0.0001$), C18:0 ($P = 0.003$) and C18:1 ($P = 0.0001$) compared to winter and spring pasture forage (Table 4-2). In addition, hay contained lower concentrations of ALA ($P = 0.0001$) resulting in a lower total n-3 FA content ($P = 0.0001$) and a higher n-6:n-3 FA ratio ($P = 0.0001$) in hay compared to winter and spring pasture (Table 4-2). No differences were observed in LA, AA, EPA, DHA or total n-6 FA content between hay and pasture forage. When principle forage source (hay or pasture) was included in the model as a main effect, forage source did not influence FA levels in any of the milk or blood samples examined in this study ($P = 0.11$).

Mare Fatty Acid Intake

Horses were housed on bahiagrass pastures throughout the trial. Because of winter dormancy and reduced quantity of pasture forage, mares were offered unlimited access to Coastal bermudagrass hay during the months of December, January, February and March. During this four month period, hay was assumed to be the primary forage source. Limited evidence of grazing in pastures and reasonable consumption of hay (based on the number of round bales fed and expected DM intake) during this period support this assumption. All hay was removed from pastures in the first week of April. Therefore, pasture served as the primary forage source from April until the conclusion of the trial in late June.

Average daily intake of long chain FA by mares on each treatment from December to March (when Coastal bermudagrass hay was the primary forage source) and from April to June (when bahiagrass pasture was the primary forage source) is shown in Tables 4-3 and Table 4-4, respectively.

From December to March (when hay was the primary forage source), FLAX mares consumed 255% more n-3 FA and FISH mares consumed 257% more n-3 FA than CON mares. From April to June (when pasture was the primary forage source), FLAX and FISH mares consumed 138% and 137% more n-3 FA than CON mares, respectively. This change in percentages reflects the higher n-3 FA content of spring bahiagrass pasture compared to Coastal bermudagrass hay. From December to March, the total diet provided a total n-3 FA intake of 4.3 g n-3 FA/100 kg BW in CON mares, 11.3 g n-3 FA/100 kg BW in FLAX mares and 11.3 g n-3 FA/100 kg BW in FISH mares. From April to June, the total diet provided a total n-3 FA intake of 17.4 g n-3 FA/100 kg BW in CON mares, 24.8 g n-3 FA/100 kg BW in FLAX mares and 24.3 g n-3 FA/100 kg BW in

FISH mares. Within the supplemented groups, the milled flaxseed and encapsulated fish oil supplied 6.5 to 7.0 g total n-3 FA/100 kg BW, or approximately 65% of the total n-3 FA intake in the winter and 30% of the total n-3 FA intake in the spring.

For the duration of the trial, FLAX mares consumed higher ALA ($P = 0.0001$) than FISH and CON mares while FISH mares consumed higher EPA ($P = 0.0001$) and DHA ($P = 0.0001$) than FLAX and CON mares. There were no differences between the consumption of total n-3 FA between FISH and FLAX mares ($P = 0.94$), but both treatments consumed more total n-3 FA than CON mares ($P = 0.0001$). No treatment effect was observed for total n-6 FA intake ($P = 0.12$).

Mare and Foal Bodyweight

Treatment had no effect on mare BW at any time during the trial (Table 4-5). CON mares foaled 3 colts and 9 fillies, FLAX mares foaled 7 colts and 5 fillies and FISH mares foaled 4 colts and 8 fillies. There were no differences in foal BW due to sex ($P = 0.58$), breed ($P = 0.62$) or treatment ($P = 0.75$). At birth, CON foals weighed 51.0 ± 4.1 kg and gained 107.3 ± 4.1 kg over the trial period. FLAX foals weighed 54.4 ± 3.5 kg at birth and gained 103.9 ± 3.5 kg, and FISH foals weighed 56.3 ± 3.8 kg at birth and gained 107.6 ± 3.9 kg over the trial period (Table 4-6). There was a significant effect of time ($P = 0.0001$) on foal BW, which reflected an increase in BW as foals grew from birth to 84 d of age.

Mare Plasma Fatty Acid Composition

Omega-6 Fatty Acids

The FA found in the highest concentration in mare plasma was LA, which made up almost half of the total FA found in plasma (Table 4-7). An overall treatment effect was noted for mare plasma LA, AA and total n-6 FA (Table 4-7). Before supplementation

began (d-28), plasma n-6 FA concentrations were similar between treatments (Table 4-8). In response to supplementation, mares fed FISH had lower plasma LA (P = 0.05), greater plasma AA (P = 0.03) and tended to have lower plasma total n-6 FA (P = 0.10) than FLAX or CON mares (Tables 4-7 and 4-8).

An overall effect of time was detected for plasma LA (P = 0.07), AA (P = 0.01) and total n-6 FA (P = 0.08) and may have reflected the effects of both parturition and dietary treatment (Table 4-8). Plasma LA declined from baseline (d-28) to foaling (d0) in CON (P = 0.05) and FISH mares (P = 0.02). After foaling, plasma LA returned to pre-treatment levels in CON mares but remained lower in FISH mares. Plasma AA increased (P = 0.01) from baseline to foaling in FISH mares but did not change in CON mares. Total plasma n-6 FA decreased from d-28 to d0 in CON (P = 0.05) and FISH mares (P = 0.04, Figure 4-1). In contrast to the responses seen in FISH and CON mares, plasma LA, AA and total n-6 FA did not change over the course of the trial in FLAX mares.

Omega-3 Fatty Acids

The n-3 FA found in the highest concentration in mare plasma was ALA, with concentrations ranging from 2.99 to 3.65 g ALA/100 g fat (Table 4-7). Overall treatment effects were detected for plasma ALA, EPA, DHA and total n-3 FA (Table 4-9). Before supplementation, no differences in plasma n-3 concentrations were observed between treatment (Table 4-9). In response to dietary treatment, FISH mares had higher plasma EPA (P = 0.0001), higher plasma DHA (P = 0.0001) and higher plasma total n-3 FA (P = 0.01) than CON mares (Table 4-7). When mares were fed FLAX, plasma ALA tended to be higher (P = 0.09) than that observed in the plasma of CON or FISH and total n-3 FA plasma content was similar to both FISH and CON mares (Table 4-7).

Overall effects of time were detected in plasma ALA (P = 0.02), EPA (P = 0.0001), DHA (P = 0.001) and total n-3 FA (P = 0.0005; Table 4-9). From d-28 to d0, plasma ALA increased in FLAX mares (P = 0.05) but decreased in FISH mares (P = 0.02). After foaling, the plasma ALA of FISH mares returned to baseline levels while the plasma ALA of FLAX mares remained at an elevated level. Plasma ALA did not change over time in CON mares. Plasma EPA increased (P = 0.002) through d+28 in mares fed FISH, after which this FA stabilized at levels above CON and FLAX mares. Plasma DHA and total n-3 increased (P = 0.001) in response to FISH, but remained unchanged in the plasma of CON or FLAX mares for the duration of the trial (Table 4-9; Figure 4-2). In contrast to the effects observed in FISH mares, plasma EPA, DHA and total n-3 FA did not change in response to dietary treatment in FLAX or CON mares (Table 4-9; Figure 4-2).

Treatment did not affect the ratio of n-6:n-3 FA in mare plasma (P = 0.24; Table 4-10). However, an overall effect of time was detected, as the ratio of plasma n-6:n-3 FA decreased over the course of the trial in all treatments (P = 0.02; Table 4-10).

Mare Colostrum and Milk Fatty Acid Composition

Treatment had no effect on the total fat content of mare colostrum (P = 0.95) or milk (P = 0.12, Table 4-11). As colostrum transitioned into milk, the total fat content increased (P = 0.0003) for all treatments (Table 4-11). The FA found in the highest concentrations in mare colostrum and milk were C16:0, C18:1 and LA (Table 4-12).

An overall effect of time was detected for all FA examined in mare milk (Tables 4-13 and 4-14). All treatments experienced a decrease in total milk n-6 FA (P = 0.0001) and an increase in total milk n-3 FA (P = 0.0001) as lactation progressed (Tables 4-13 and 4-14). As a result, the ratio of n-6:n-3 FA decreased in mare milk from foaling

through 84 d post-foaling (P = 0.0001; Table 4-10). From 36h to through 14 d post-foaling, milk LA and total n-6 FA decreased in FLAX (P = 0.0005) and FISH mares (P = 0.0003), and then remained constant for the duration of the trial (Table 4-13). Milk from mares fed FISH showed an increase in EPA (P = 0.0001) and DHA (P = 0.0001) content from 36h to through 14 d post-foaling, and these levels remained steady until 84 d post-foaling (Table 4-14).

Overall effects of treatment were not noted for any of the n-6 FA examined in mare milk (Table 4-12, Figure 4-3). However, overall effects of treatment were observed for milk ALA (P = 0.0001), EPA (P = 0.0001), DHA (P = 0.0001) and n-6:n-3 FA ratio (P = 0.01; Tables 4-10 and 4-12). At foaling, the colostrum of FLAX mares contained higher levels of ALA (P = 0.05) than CON and FISH mares (Table 4-14, Figure 4-4). As lactation progressed, FLAX mares continued to have a higher ALA content in their milk compared to FISH or CON mares (P = 0.01). FISH mares had a higher colostrum DHA content than CON and FLAX mares (P = 0.03) and had higher EPA (P = 0.0001) and DHA (P = 0.0001) concentrations in milk than CON or FLAX mares as lactation progressed (Table 4-14, Figure 4-4). The colostrum of FLAX mares contained greater total n-3 FA than the colostrum of CON mares (P = 0.05), but total n-3 FA was not different than FISH mares. Over the course of lactation, the n-6:n-3 FA ratio tended to be lower in the milk of FLAX mares (P = 0.09) when compared to the milk of CON and FISH mares (Table 4-10).

Foal Plasma Fatty Acid Composition

Omega-6 Fatty Acids

Similar to the mare, the FA found in the highest concentration in foal plasma was LA, making up roughly one third of the total FA found in plasma (Table 4-15). An

overall treatment effect was not observed for any of the n-6 FA examined in foal plasma (Table 4-16, Figure 4-5). However, at foaling (d0), plasma AA concentrations were highest in foals born to FLAX mares (P = 0.04, Table 4-16). At 14 d post-foaling, foals suckling FISH mares showed a higher plasma AA concentration than foals suckling CON mares (P = 0.04), but were similar to foals suckling FLAX mares (P = 0.30). No other effects of treatment on n-6 FA were detected at any time point over the course of the trial.

An overall effect of time was detected in plasma LA (P = 0.0001), AA (P = 0.0001) and total n-6 FA (P = 0.0001; Table 4-16, Figure 4-4). Plasma LA and total n-6 FA increased (P = 0.0001) and plasma AA decreased (P = 0.0001) from foaling to 14 d post-foaling in all treatments (Table 4-16). Plasma LA and total n-6 FA increased from 14 to 84 d of age in foals suckling CON (P = 0.005) and FISH mares (P = 0.008), but remained stable in foals nursing FLAX mares. Plasma AA increased from 14 to 84 d of age in

Omega-3 Fatty Acids

The n-3 FA found in the highest concentration in foal plasma was ALA, with levels ranging from 2.48 to 3.33 g ALA/100 g fat (Table 4-15). An overall effect of treatment was observed in foal plasma ALA, EPA, DHA and total n-3 FA (Table 4-15, Figure 4-6). At foaling, foals born to FISH mares tended to have a higher total n-3 FA plasma content than foals born to CON or FLAX mares (P = 0.09; Table 4-17, Figure 4-6). Foals suckling FLAX mares had higher plasma ALA (P = 0.04) than foals suckling CON mares and foals nursing FISH mares had higher plasma EPA (P = 0.0001), DHA (P = 0.0001) and total n-3 FA (P = 0.002) than foals nursing both CON and FLAX mares (Table 4-15 and 4-17).

An overall effect of time was detected in all n-3 FA in foal plasma (P = 0.0001; Table 4-17, Figure 4-6). From foaling to 84 d of age, the plasma ALA and total n-3 FA

content increased in foals, regardless of mare treatment (P = 0.0001). Plasma EPA increased in FLAX foals from foaling to 28 d of age (P = 0.0001), but returned to foaling levels by 56 d of age (Table 4-17). Plasma EPA increased from birth to 14 d of age in FISH foals (P = 0.0001), but decreased from 28 to 56 d of age. However, the plasma EPA concentration in FISH foals was still higher at 84 d of age than the concentrations at foaling (P = 0.0005). From birth to 14 d of age, plasma DHA decreased in CON (P = 0.0001) and FLAX (P = 0.0001) foals and then remained steady until the end of the trial. However, the DHA concentration in the plasma of FISH foals remained elevated through 56 d of age, declining slightly by 84 d (Table 4-17).

Omega-6:Omega-3 Fatty Acid Ratios

An overall effect of treatment on the n-6:n-3 ratio was observed in foal plasma (Table 4-10). At birth, no difference in n-6: n-3 FA ratio was detected between treatments. After suckling treated mares, FISH foals had a lower n-6:n-3 FA ratio (P = 0.002) than FLAX or CON foals (Table 4-10). An overall effect of time on the n-6:n-3 FA ratio was also detected in foal plasma (P = 0.001, Table 4-10). From birth to 14 d of age, the plasma n-6:n-3 ratio increased in CON (P = 0.001) and FLAX (P = 0.01) foals. The plasma n-6:n-3 FA ratio returned to levels seen at foaling by 28 d of age in FLAX foals, while the plasma n-6:n-3 FA ratio of CON foals did not return to baseline levels until 56 d of age. The plasma n-6:n-3 ratio in FISH foals remained steady over the course of the trial (Table 4-10).

Fatty Acid Correlations

Positive correlations (P = 0.0001) between mare plasma and milk FA were found for ALA, EPA, DHA and total n-3 FA, while a negative, but weak correlation between mare plasma and mare milk was found for total n-6 FA (P = 0.003; Table 4-18). No

correlation was found between mare plasma LA and mare milk LA, while mare plasma AA and mare milk AA tended to be negatively correlated (P = 0.10).

Mare milk ALA, EPA, DHA and total n-3 FA were positively correlated (P = 0.0001) with foal plasma ALA, EPA, DHA and total n-3 FA (Table 4-18). Mare milk AA and total n-6 FA were negatively correlated to foal plasma AA (P = 0.0001) and total n-6 FA (P = 0.03). No correlation between milk LA and foals plasma LA was detected.

Fatty Acid Composition of Red Blood Cells

Mare Red Blood Cell Fatty Acids

Fatty acids with chain lengths longer than 18 carbons were not detected in mare red blood cells, and LA was the only n-6 FA observed (Table 4-19). An overall effect of treatment was noted for LA (Table 4-19, Figure 4-7). While no differences in LA concentration were found in mare red blood cells before supplementation, there was a tendency (P = 0.10) for FISH mares to have a higher red blood cell LA content than both CON and FLAX mares (P = 0.10, Table 4-19). An overall effect of time was not detected for mare red blood cell n-6 FA content (Table 4-20, Figure 4-7). However, the red blood cell LA content of FISH mares increased from pre-supplementation to foaling (P = 0.02; Table 4-20). The LA content of CON and FLAX red blood cells did not fluctuate during the study (Table 4-20).

Foal Red Blood Cell Fatty Acids

Linoleic acid was the only n-6 FA and ALA was the only n-3 FA found in foal red blood cells; fatty acids with chain lengths longer than 18 carbons were not detected (Table 4-21). An overall effect of treatment was detected for red blood cell LA, but not ALA (Table 4-21). At foaling, no differences were observed in red blood cell LA content (Table 4-22, Figure 4-8). In response to suckling supplemented mares, FISH foals had a

higher red blood cell LA content than both CON and FLAX foals (P = 0.04, Table 4-21, Figure 4-8). Foals belonging to FLAX mares had higher (P = 0.03) ALA in red blood cells at birth than foals belonging to CON mares, but had similar red blood cell ALA as foals born to FISH mares. Treatment of the mare did not affect ALA content of foal red blood cells at any other time point during the study (Table 4-22, Figure 4-9).

An overall effect of time was found in foal red blood cell LA (P = 0.03) but not ALA (Table 4-22). The LA content of red blood cells increased in FISH foals from foaling to 14 d of age (P = 0.01) and stayed elevated for the duration of the study. In contrast, the LA in red blood cells of CON and FLAX foals did not change during the trial (Table 4-22).

Mare Serum, Colostrum and Milk IgG

An overall effect of treatment was not detected in mare serum or colostrum IgG concentrations (Table 4-23). Mare breed (P = 0.78) or age (P = 0.56) did not affect serum IgG content. Similarly, colostrum IgG was not affected by mare breed (P = 0.67) or age (P = 0.58).

Milk IgG was not affected by treatment (P = 0.65) or breed (P = 0.67, Table 4-24). However, an overall effect of time on milk IgG was detected (P = 0.0001, Table 4-24). Milk from all mares showed a decline in IgG from 36 h through 84 d post-foaling (Table 4-24). The overall decline in milk IgG concentration from 36 h to 84 d post-partum was 139.4 ± 630.7 mg/dL for CON mares, 157.0 ± 677.5 mg/dL for FLAX mares and 132.5 ± 659.2 mg/dL for FISH mares.

Mare serum IgG at foaling was not correlated with mare age (Table 4-25). Similarly, colostrum IgG was not correlated with mare age, although FLAX mares tended to show an inverse relationship (r = -0.58, P = 0.06) between mare age and colostrum IgG

(Table 4-25). Mare serum IgG at foaling was not correlated to colostrum IgG, and colostrum IgG was not correlated to foal serum IgG at 36 h post-foaling. However, a weak correlation between mare serum IgG at foaling and foal serum IgG at 36 h post-foaling was detected across treatments ($r = 0.42$, $P = 0.02$; Figure 4-10). Within treatments, d0 serum IgG from CON and FLAX mares showed no correlation with foal 36h serum IgG content, but serum IgG from FISH mares at d0 was correlated to FISH foals serum IgG at 36 h post-foaling ($r = 0.63$, $P = 0.05$, Table 4-25).

Foal Serum IgG

All foals had serum IgG concentrations that were very low at birth and reflected the pre-suckle status of the foal (Figure 4-11). The IgG content of foal serum increased to, and peaked at, 36 h post-foaling, indicating that passive transfer of IgG had taken place. An overall effect of time was noted for foal serum IgG ($P = 0.0001$), as the IgG of all foals steadily declined from 36 h to 84 d post-foaling (Table 4-25, Figure 4-12).

No overall effect of treatment was observed for foal serum IgG (Table 4-25). However, foals suckling FISH mares tended to have lower serum IgG than foals nursing FLAX mares at 36h ($P = 0.09$) and 7 d post-foaling ($P = 0.10$). In addition, FISH foals tended to have lower IgG than CON foals at 28 d post-foaling ($P = 0.10$).

Mare and Foal Responses to the Intradermal Skin Test

Mare Response to PHA

An overall effect of treatment was not detected in the skin thickness of mares in response to a paired intradermal skin test using PHA as the stimulant ($P = 0.89$; Table 4-26). However, an overall effect of time was observed ($P = 0.0001$; Table 4-26). Before injection, no differences were observed in mare skin thickness ($P = 0.56$; Figure 4-13). All mares experienced a significant increase in skin thickness from 0 to 2 h ($P = 0.0001$)

and from 2 to 4 h post-injection (P = 0.0001; Table 4-26, Figure 4-13). Skin thickness was greatest between 4 and 8 h post-injection and then decreased. At 48 h, skin thickness was still elevated above that measured before PHA injection at 0 h (P = 0.0001).

Foal Response to PHA

An overall effect of time (P = 0.0001) on skin thickness in foals in response to PHA injection was detected, reflecting an inflammatory response (Figure 4-14). Foal skin thickness increased (P = 0.0001) from 0 to 4 h, remained elevated through 8 h post-injection and then declined through 48 h post-injection (P = 0.0001, Figure 4-14). At 48 h post-injection, skin thickness had not yet declined to baseline thickness measured before PHA injection (P = 0.0001).

An overall effect of treatment on foal skin thickness was not detected (P = 0.58; Table 4-27). However, CON foals peaked at 4 h (P = 0.0001), whereas skin thickness remained elevated in FLAX and FISH foals through 6 h (P = 0.0001; Figure 4-14, Table 4-27). At 6 h post-injection, FLAX foals had greater skin thickness than CON foals (P = 0.02), while the skin thickness of FISH foals was intermediate between FLAX and CON foals.

Comparing Mare and Foal Responses to PHA

Across treatments, skin thickness in response to PHA injection was different between mares and foals (P = 0.0001; Table 4-28, Figure 4-15). Although thickness was not different before injection of PHA (0h), mares exhibited a greater (P = 0.0001) inflammatory response to intradermal PHA compared to foals (Table 4-28, Figure 4-15). The skin thickness of neither the mares or the foals returned to pre-injection values by 48 h post-injection (P = 0.0001).

Table 4-1. Fatty acid composition of the grain mix concentrate and the milled flaxseed and encapsulated fish oil supplements[1]

Fatty acid	Grain mix	Flaxseed	Fish Oil
C8:0	ND	ND	ND
C10:0	ND	ND	ND
C12:0	ND	ND	ND
C14:0	ND	ND	8.62
C16:0	17.18	5.60	21.14
C16:1	0.21	ND	13.77
C17:0	ND	ND	ND
C17:1	ND	ND	ND
C18:0	2.20	2.77	3.79
C18:1	26.28	13.90	7.87
C18:2n-6 (LA)	49.63	16.31	7.23
C18:3n-3 (ALA)	3.72	61.20	2.35
C20:4n-6 (AA)	ND	ND	0.69
C20:5n-3 (EPA)	ND	ND	15.03
C22:5 n-3 (DPA)	ND	ND	2.11
C22:6n-3 (DHA)	ND	ND	12.54
Total n-6[2]	49.63	16.31	7.92
Total n-3[3]	3.72	61.20	32.03
n-6:n-3	13.34	0.27	0.25

[1] Presented as g fatty acid per 100 g fat; ND = not detected in the feedstuff.
[2] Calculated as C18:2 + C20:4.
[3] Calculated as C18:3 + C20:5 + C22:5 + C22:6.

Table 4-2. Fatty acid composition of winter and spring bahiagrass pasture and Coastal bermudagrass hay[1]

	Pasture		
Fatty acid	Winter[2]	Spring[3]	Hay
C8:0	ND	ND	ND
C10:0	ND	ND	ND
C12:0	ND	ND	ND
C14:0	ND	ND	ND
C16:0	22.07 ± 0.69[a]	23.22 ± 0.67[a]	39.30[b]
C16:1	ND	ND	ND
C17:0	0.93 ± 0.05[a]	0.53± 0.28[a]	0.00[b]
C17:1	ND	ND	ND
C18:0	4.97 ± 0.38[a]	3.36 ± 0.22[b]	6.65[c]
C18:1	4.21 ± 0.60[a]	1.39 ± 0.02[b]	7.08[c]
C18:2n-6 (LA)	23.71 ± 1.96	18.13 ± 1.55	23.48
C18:3n-3 (ALA)	41.52 ± 3.28[a]	52.47 ± 1.76[b]	15.90[c]
C20:4n-6 (AA)	ND	ND	ND
C20:5n-3 (EPA)	ND	ND	ND
C22:5 n-3 (DPA)	ND	ND	ND
C22:6n-3 (DHA)	ND	ND	ND
Total n-6[4]	23.71 ± 1.90	18.13 ± 1.55	23.48
Total n-3[5]	41.52 ± 3.28[a]	52.47 ± 1.76[b]	15.90[c]
n-6:n-3	0.59 ± 0.08[a]	0.35 ± 0.04[a]	1.45[b]

[1] Presented as g fatty acid per 100 g fat; ND = not detected in the forage.
[2] Winter = Mean of December, January, February and March.
[3] Spring = Mean of April, May and June.
[4] Calculated as C18:2 + C20:4.
[5] Calculated as C18:3 + C20:5 + C22:5 + C22:6.
[a,b,c] Values in the same row having different superscripts differ at $P < 0.05$.

Table 4-3. Mare average daily fatty acid intake from December-March[1]

		Mare diet			
Fatty acid	Treatment[2]	Grain	Hay[3]	Supplement	Total diet
C18:2n-6 (LA)	CON	135.49	21.37	ND	156.86
	FLAX	129.24	20.66	10.45	160.35
	FISH	137.57	21.37	8.7	167.64
C18:3n-3 (ALA)	CON	10.16	14.58	ND	24.73
	FLAX	9.69	14.10	39.22	63.01
	FISH	10.31	14.58	2.83	27.72
C20:4n-6 (AA)	CON	ND	ND	ND	ND
	FLAX	ND	ND	ND	ND
	FISH	ND	ND	0.83	0.83
C20:5n-3 (EPA)	CON	ND	ND	ND	ND
	FLAX	ND	ND	ND	ND
	FISH	ND	ND	18.10	18.10
C22:6n-3 (DHA)	CON	ND	ND	ND	ND
	FLAX	ND	ND	ND	ND
	FISH	ND	ND	15.10	15.10
Total n-6[4]	CON	135.49	21.37	ND	156.86
	FLAX	129.24	20.66	10.45	160.35
	FISH	137.57	21.37	9.54	168.48
Total n-3[5]	CON	10.16	14.58	ND	24.73
	FLAX	9.69	14.10	39.22	63.01
	FISH	10.31	14.58	38.56	63.45
n-6:n-3	CON	13.34:1	1.47:1	ND	6.34:1
	FLAX	13.34:1	1.47:1	0.27:1	2.54:1
	FISH	13.34:1	1.47:1	0.25:1	2.66:1

[1] Presented as g fatty acid/d; ND = not detected in any of the feedstuffs.

[2] CON = no supplement, FLAX = supplemented with milled flaxseed, FISH = supplemented with encapsulated fish oil.

[3] Hay intake estimated at 1.0% BW (DM basis).

[4] Calculated as C18:2 + C20:4.

[5] Calculated as C18:3 + C20:5 + C22:5 + C22:6.

Table 4-4. Mare average daily fatty acid intake from April-June[1]

		Mare diet			
Fatty acid	Treatment[2]	Grain	Spring pasture[3]	Supplement	Total diet
C18:2n-6 (LA)	CON	135.49	30.91	ND	166.40
	FLAX	129.24	31.47	9.84	170.55
	FISH	137.57	30.91	8.24	176.72
C18:3n-3 (ALA)	CON	10.16	89.46	ND	99.62
	FLAX	9.69	91.09	36.91	137.69
	FISH	10.31	89.46	2.68	102.45
C20:4n-6 (AA)	CON	ND	ND	ND	ND
	FLAX	ND	ND	ND	ND
	FISH	ND	ND	0.82	0.82
C20:5n-3 (EPA)	CON	ND	ND	ND	ND
	FLAX	ND	ND	ND	ND
	FISH	ND	ND	17.06	17.16
C22:6n-3 (DHA)	CON	ND	ND	ND	ND
	FLAX	ND	ND	ND	ND
	FISH	ND	ND	14.34	14.34
Total n-6[4]	CON	135.49	30.91	ND	166.40
	FLAX	129.24	31.47	9.84	170.55
	FISH	137.57	30.91	9.03	177.51
Total n-3[5]	CON	10.16	89.46	ND	99.62
	FLAX	9.69	91.09	36.91	137.69
	FISH	10.31	89.46	36.50	136.27
n-6:n-3	CON	13.34:1	0.35:1	ND	1.67:1
	FLAX	13.34:1	0.35:1	0.27:1	1.24:1
	FISH	13.34:1	0.35:1	0.25:1	1.30:1

[1] Presented as g fatty acid/d; ND = not detected in any of the feedstuffs.

[2] CON = no supplement, FLAX = supplemented with milled flaxseed, FISH = supplemented with encapsulated fish oil.

[3] Pasture intake estimated at 1.0% BW (DM basis).

[4] Calculated as C18:2 + C20:4.

[5] Calculated as C18:3 + C20:5 + C22:5 + C22:6.

Table 4-5. Mare bodyweights[1]

	Treatment[2]			
Time[3]	CON	FLAX	FISH	SEM
d-28	629.2	619.0	635.9	13.66
d-14	631.3	626.3	639.7	13.51
d0	554.9	544.2	559.2	8.83
d+14	553.0	551.7	559.3	8.81
d+28	556.0	550.7	562.5	8.81
d+42	560.9	552.9	567.5	8.87
d+56	561.7	548.2	567.4	8.84
d+70	556.6	542.8	557.7	9.00
d+84	556.6	552.4	565.0	8.90

[1] Presented in kg.
[2] CON = no supplement, FLAX = supplemented with milled flaxseed, FISH = supplemented with encapsulated fish oil.
[3] d-28 to d-14 = d before expected foaling; d0 = foaling; d+14 to d+84 = d post-foaling.

Table 4-6. Foal bodyweights[1,2]

	Treatment[3]			
Time[4]	CON	FLAX	FISH	SEM
d0	51.0[a]	54.4[a]	56.3[a]	2.21
d+14	74.1[b]	75.5[b]	75.4[b]	2.20
d+28	91.9[c]	95.4[c]	95.0[c]	2.20
d+42	109.4[d]	116.0[d]	115.0[d]	2.25
d+56	125.1[e]	127.6[e]	131.5[e]	2.23
d+70	144.8[f]	142.4[f]	144.4[f]	2.34
d+84	158.3[g]	158.5[g]	163.9[g]	2.25

[1] Presented in kg.
[2] Effect of time (P = 0.0001), effect of treatment (P = 0.75), effect of treatment x time (P = 0.31).
[3] CON = no supplement, FLAX = supplemented with milled flaxseed, FISH = supplemented with encapsulated fish oil.
[4] d0 = foaling; d+14 to d+84 = d post-foaling.
[a,b,c,d,e,f,g] Values in the same column having different subscripts are different at P < 0.05.

Table 4-7. Overall effect of treatment on the fatty acid composition of mare plasma[1]

	Treatment[2]				
Fatty Acid	CON	FLAX	FISH	SEM	P-value
C8:0	ND	ND	ND	NA	NA
C10:0	ND	ND	ND	NA	NA
C12:0	ND	ND	ND	NA	NA
C14:0	ND	ND	ND	NA	NA
C16:0	16.15	16.01	16.48	0.25	0.42
C16:1	0.88	0.88	1.10	0.09	0.17
C17:0	0.70	0.73	0.73	0.04	0.78
C17:1	ND	ND	ND	NA	NA
C18:0	20.08	20.18	20.45	0.32	0.72
C18:1	10.28	9.78	9.70	0.28	0.29
C18:2n-6 (LA)	46.43	46.87	44.37	0.71	0.05
C18:3n-3 (ALA)	2.99	3.65	3.06	0.23	0.09
C20:4n-6 (AA)	1.50	1.33	1.82	0.13	0.03
C20:5n-3 (EPA)	0.02	0.02	0.56	0.06	0.0001
C22:6n-3 (DHA)	0.05	0.03	0.61	0.05	0.0001
Total n-6[3]	48.64	48.92	47.04	0.64	0.10
Total n-3[4]	3.03	3.69	4.22	0.26	0.01
n-6:n-3	16.33	16.21	13.32	1.35	0.24

[1] Presented as g fatty acid per 100 g fat, ND = not detected in plasma, NA = not applicable.

[2] CON = no supplement, FLAX = supplemented with milled flaxseed, FISH = supplemented with encapsulated fish oil.

[3] Calculated as C18:2 + C20:4.

[4] Calculated as C18:3 + C20:5 + C22:5 + C22:6.

Table 4-8. Omega-6 fatty acid content of mare plasma[1]

	Time[2]						P-values		
Fatty acid	d-28	d0	d+28	d+56	d+84	SEM	Treatment	Time	Treatment x Time
C18:2 (LA)							0.05	0.07	0.79
CON	47.0^{y}	43.1^{z}	47.5^{y}	46.9^{y}	47.6^{a,y}	0.70			
FLAX	45.8	45.5	47.2	47.7	48.1^{a}	0.73			
FISH	46.7^{y}	42.3^{z}	44.6^{z}	44.9^{z}	43.4^{b,z}	0.73			
C20:4 (AA)							0.03	0.01	0.60
CON	1.7^{y,z}	2.2^{a,b,y}	1.3^{z}	1.2^{z}	1.2^{z}	0.12			
FLAX	1.8	1.5^{a}	1.2	1.1	1.1	0.13			
FISH	1.7^{y}	2.7^{b,z}	1.4^{y}	1.7^{y}	1.6^{y}	0.13			
Total n-6[3]							0.10	0.08	0.79
CON	49.2^{y}	45.8^{z}	49.5^{y}	48.9^{y}	49.7^{y}	0.63			
FLAX	48.4	47.6	49.1	49.5	50.0	0.65			
FISH	49.2^{y}	45.7^{z}	46.8^{y,z}	47.6^{y,z}	45.9^{z}	0.65			

[1] Presented as g fatty acid per 100 g fat.
[2] d-28 = d before expected foaling; d0 = foaling; d+28 to d+84 = d post-foaling.
[3] Calculated as C18:2 + C20:4.
[a,b] Values in the same column for each fatty acid not sharing a common superscript are different at $P < 0.05$.
[y,z] Values in the same row not sharing a common superscript are different at $P < 0.05$.

Table 4-9. Omega-3 fatty acid content of mare plasma[1]

	Time[2]						P-values		
Fatty acid	d-28	d0	d+28	d+56	d+84	SEM	Treatment	Time	Treatment x Time
C18:3 (ALA)							0.09	0.02	0.12
CON	3.0	2.3^{a}	2.8	3.4	3.4	0.23			
FLAX	2.5^{x}	3.3^{b,y}	3.6^{y}	4.2^{y}	4.5^{y}	0.23			
FISH	3.2^{x}	2.2^{a,y}	3.3^{x}	2.9^{x}	3.8^{x}	0.24			
C20:5 (EPA)							<0.0001	<0.0001	<0.0001
CON	0.0	0.01	0.0^{a}	0.02^{a}	0.03^{a}	0.06			
FLAX	0.0	0.0	0.1^{a}	0.0^{a}	0.0^{a}	0.06			
FISH	0.0^{x}	0.3^{y}	1.1^{b,z}	1.0^{b,z}	0.5^{b,y}	0.06			
C22:6 (DHA)							<0.0001	0.001	0.0004
CON	0.0	0.17^{a}	0.0^{a}	0.04^{a}	0.04^{a}	0.05			
FLAX	0.0	0.0^{a}	0.1^{a}	0.0^{a}	0.03^{a}	0.05			
FISH	0.0^{x}	0.64^{b,y}	0.95^{b,y}	0.89^{b,y}	0.71^{b,y}	0.05			
Sum n-3[3]							0.01	0.0005	0.01
CON	2.9	2.5	2.9^{a}	3.4^{a}	3.5^{a}	0.25			
FLAX	2.6	3.3	3.8^{a}	4.2^{a,b}	4.5^{a,b}	0.26			
FISH	2.9^{x}	3.0^{x}	5.3^{b,y}	4.8^{b,y}	5.1^{b,y}	0.26			

[1] Presented as g fatty acid per 100 g fat.
[2] d-28 = d before expected foaling; d0 = foaling; d+28 to d+84 = d post-foaling.
[3] Calculated as C18:3 + C20:5 + C22:5 + C22:6.
[a,b] Values in the same column for each fatty acid not sharing a common superscript are different at $P < 0.05$.
[y,z] Values in the same row not sharing a common superscript are different at $P < 0.05$.

Table 4-10. Omega-6:omega-3 fatty acid ratios in mare and foal plasma and mare milk[1]

	Time[2]								P-values	
Sample	d-28	d0	36h	d+14	d+28	d+56	d+84	SEM	Treatment	Time	Treatment x Time
Mare plasma									0.24	0.02	0.79
CON	19.0^{y,z}	22.7^{y}	---	---	15.0^{z}	12.2^{z}	12.8^{z}	1.35			
FLAX	21.6^{y,z}	17.2^{y,z}	---	---	14.5^{y,z}	15.0^{y,z}	12.8^{z}	1.40			
FISH	17.5^{y}	17.2^{y}	---	---	9.1^{z}	11.8^{y}	10.9^{y,z}	1.40			
Mare milk									0.01	0.0001	0.66
CON	---	3.4^{a,y}	3.2^{a,y}	1.6^{z}	1.2^{z}	1.2^{z}	1.0^{z}	0.12			
FLAX	---	2.4^{b,y}	2.4^{b,y}	1.4^{z}	1.0^{z}	0.7^{z}	0.7^{z}	0.12			
FISH	---	3.6^{a,y}	3.0^{a,b,y}	1.5^{z}	0.9^{z}	1.0^{z}	1.0^{z}	0.12			
Foal plasma									0.002	0.001	0.03
CON	---	11.1^{y}	---	19.7^{a,z}	17.2^{a,z}	10.4^{y}	11.5^{y}	1.08			
FLAX	---	11.4^{y}	---	17.1^{a,z}	9.9^{b,y}	9.7^{y}	9.1^{y}	0.94			
FISH	---	11.0	---	8.3^{b}	6.5^{b}	6.5	7.8	1.00			

[1] Calculated as total n-6 / total n-3.

[2] d-28 = d before expected foaling; d0 = foaling; 36h = h post-foaling; d+14 to d+84 = d post-foaling.

[a,b] Values in the same column for each fatty acid having different superscripts are different at $P < 0.05$.

[y,z] Values in the same row having different superscripts are different at $P < 0.05$.

Table 4-11. Overall effect of treatment on the total fat content of mare colostrum and milk[1]

	Treatment[2]				
Sample	CON	FLAX	FISH	SEM	P - Value
Colostrum	0.90[a]	0.99[a]	0.96[a]	0.20	0.95
Milk	1.54[b]	1.42[b]	1.33[b]	0.07	0.12

[1] Presented as g fat/100 g milk (wet basis).
[2] CON = no supplement, FLAX = supplemented with milled flaxseed, FISH = supplemented with encapsulated fish oil.
[a,b] Values in the same column having different superscripts are different at $P < 0.05$.

Table 4-12. Overall effect of treatment on the fatty acid composition of mare colostrum and milk[1]

	Treatment[2]				
Fatty acid	CON	FLAX	FISH	SEM	P-value
C8:0	5.69	6.20	6.23	0.18	0.08
C10:0	10.00	10.78	10.78	0.41	0.32
C12:0	8.53	9.26	9.23	0.38	0.34
C14:0	5.74	5.92	6.01	0.21	0.67
C16:0	17.73	16.99	17.69	0.22	0.04
C16:1	5.74	5.02	5.37	0.18	0.03
C17:0	0.05	0.05	0.08	0.01	0.33
C17:1	0.31	0.29	0.26	0.02	0.31
C18:0	1.42	1.49	1.53	0.05	0.33
C18:1	16.16	14.69	14.58	0.50	0.06
C18:2n-6 (LA)	15.82	14.52	15.03	0.58	0.31
C18:3n-3 (ALA)	11.71	13.06	10.87	0.48	0.01
C20:4n-6 (AA)	0.19	0.20	0.20	0.03	0.96
C20:5n-3 (EPA)	0.01	0.02	0.43	0.03	0.0001
C22:6n-3 (DHA)	0.03	0.03	0.62	0.04	0.0001
Total n-6[3]	16.46	15.22	15.59	0.60	0.34
Total n-3[4]	11.74	13.11	12.02	0.48	0.12
n-6:n-3	1.95	1.40	1.83	0.12	0.09

[1] Presented as g fatty acid per 100 g fat.
[2] CON = no supplement, FLAX = supplemented with milled flaxseed, FISH = supplemented with encapsulated fish oil.
[3] Calculated as C18:2 + C20:4.
[4] Calculated as C18:3 + C20:5 + C22:5 + C22:6.

Table 4-13. Omega-6 fatty acid content of mare colostrum and milk[1]

	Time[2]							P-values		
Fatty acid	d0	36h	d+14	d+28	d+56	d+84	SEM	Treatment	Time	Treatment x Time
C18:2 (LA)								0.31	0.0001	0.50
CON	20.71^{x}	16.52^{y}	$15.24^{a,y}$	14.05^{y}	15.05^{y}	13.32^{y}	0.59			
FLAX	19.86^{x}	17.25^{y}	$11.58^{b,z}$	12.43^{z}	13.61^{z}	12.38^{z}	0.59			
FISH	21.85^{x}	17.34^{y}	$11.98^{b,z}$	12.37^{z}	12.49^{z}	14.16^{z}	0.59			
C20:4 (AA)								0.96	0.0001	0.72
CON	0.02^{x}	0.01^{x}	0.35^{y}	0.12^{z}	0.39^{y}	0.26^{y}	0.03			
FLAX	0.02^{x}	0.07^{x}	0.35^{y}	0.15^{x}	0.38^{y}	0.24^{z}	0.03			
FISH	0.02^{x}	0.10^{x}	0.30^{y}	0.03^{x}	0.48^{z}	0.28^{y}	0.03			
Total n-6[3]								0.34	0.0001	0.46
CON	21.38^{x}	16.84^{y}	$16.15^{a,y}$	14.71^{y}	15.80^{y}	13.87^{y}	0.59			
FLAX	20.73^{x}	17.80^{y}	$12.47^{b,z}$	13.06^{z}	14.34^{z}	12.93^{z}	0.59			
FISH	22.57^{x}	17.72^{y}	$12.65^{b,z}$	12.92^{z}	13.13^{z}	14.57^{z}	0.60			

[1] Presented as g fatty acid per 100 g fat.

[2] d0 = colostrum collected at foaling; 36h = h post-foaling; d+14 to d+84 = d post-foaling.

[3] Calculated as C18:2 + C20:4.

[a,b] Values in the same column for each fatty acid having different superscripts are different at $P < 0.05$.

[x,y,z] Values in the same row having different superscripts are different at $P < 0.05$.

Table 4-14. Omega-3 fatty acid content of mare colostrum and milk[1]

	Time[2]							P-values		
Fatty acid	d0	36h	d+14	d+28	d+56	d+84	SEM	Treatment	Time	Treatment x Time
C18:3 (ALA)								0.01	0.0001	0.17
CON	6.73^{a,w}	4.70^{a,w}	10.25^{x}	13.33^{y}	15.81^{a,y}	19.44^{z}	0.48			
FLAX	9.49^{b,w}	8.01^{b,w}	10.54^{x}	13.02^{y}	19.53^{b,z}	17.74^{z}	0.48			
FISH	6.89^{a,w}	6.30^{a,b,w}	8.44^{w}	12.92^{x}	14.34^{a,x}	16.34^{x}	0.49			
C20:5 (EPA)								0.0001	0.002	0.005
CON	0.00	0.00^{a}	0.05^{a}	0.00^{a}	0.00^{a}	0.00^{a}	0.03			
FLAX	0.00	0.00^{a}	0.00^{a}	0.09^{a}	0.00^{a}	0.00^{a}	0.03			
FISH	0.23^{x}	0.28^{b,x,z}	0.63^{b,y}	0.69^{b,y}	0.43^{b,y}	0.27^{b,z}	0.03			
C22:6 (DHA)								0.0001	0.0004	0.003
CON	0.00^{a}	0.00^{a}	0.05^{a}	0.03^{a}	0.08^{a}	0.00^{a}	0.04			
FLAX	0.00^{a}	0.00^{a}	0.00^{a}	0.12^{a}	0.06^{a}	0.00^{a}	0.04			
FISH	0.29^{b,x}	0.37^{b,x,z}	0.94^{b,y}	0.95^{b,y}	0.66^{b,y}	0.47^{b,z}	0.04			
Total n-3[3]								0.12	0.0001	0.20
CON	6.70^{a,w}	4.66^{a,w}	10.32^{x}	13.40^{y}	15.87^{a,y}	19.48^{z}	0.48			
FLAX	9.47^{b,w}	7.99^{b,w}	10.57^{x}	13.22^{y}	19.63^{b,z}	17.80^{z}	0.47			
FISH	7.38^{a,b,w}	6.94^{a,b,w}	10.18^{x}	14.58^{y}	15.69^{a,y}	17.37^{y}	0.48			

[1] Presented as g fatty acid per 100 g fat.

[2] d0 = colostrum collected at foaling; 36h = h post-foaling; d+14 to d+84 = d post-foaling..

[3] Calculated as C18:3 + C20:5 + C22:5 + C22:6.

[a,b] Values in the same column for each fatty acid having different superscripts are different at $P < 0.05$.

[w,x,y,z] Values in the same row having different superscripts are different at $P < 0.05$.

Table 4-15. Overall effect of treatment on the fatty acid composition of foal plasma[1]

	Treatment[2]				
Fatty acid	CON	FLAX	FISH	SEM	P-value
C8:0	ND	ND	ND	NA	NA
C10:0	0.17	0.23	0.36	0.11	0.50
C12:0	0.68	0.75	0.69	0.07	0.70
C14:0	0.63	0.64	0.60	0.06	0.88
C16:0	17.96	17.54	18.11	0.21	0.16
C16:1	2.27	2.13	2.12	0.08	0.28
C17:0	0.35	0.36	0.43	0.03	0.19
C17:1	0.15	0.19	0.15	0.03	0.48
C18:0	20.83	21.18	20.51	0.25	0.20
C18:1	10.90	10.67	10.03	0.20	0.01
C18:2n-6 (LA)	36.99	36.45	33.67	0.85	0.56
C18:3n-3 (ALA)	2.48	3.33	2.72	0.23	0.04
C20:4n-6 (AA)	2.79	3.01	2.97	0.20	0.74
C20:5n-3 (EPA)	0.05	0.09	0.89	0.06	0.0001
C22:6n-3 (DHA)	0.48	0.53	1.74	0.11	0.0001
Total n-6[3]	40.76	39.95	39.12	0.75	0.34
Total n-3[4]	3.10	4.12	5.80	0.30	0.0001
n-6:n-3	13.97	11.43	8.03	0.96	0.002

[1] Presented as g fatty acid per 100 g fat, ND = not detected in plasma, NA = not applicable.

[2] CON = no supplement, FLAX = supplemented with milled flaxseed, FISH = supplemented with encapsulated fish oil.

[3] Calculated as C18:2 + C20:4.

[4] Calculated as C18:3 + C20:5 + C22:5 + C22:6

.Table 4-16. Omega-6 fatty acid content of foal plasma[1]

	Time[2]								
Fatty acid	d0	d+14	d+28	d+56	d+84	SEM	Treatment	Time	Treatment x Time
C18:2 (LA)							0.56	0.0001	0.60
CON	18.79^{w}	39.32^{x}	39.92^{x}	$42.31^{x,y}$	44.62^{y}	0.90			
FLAX	16.92^{w}	39.98^{x}	40.07^{x}	41.97^{x}	43.32^{x}	0.76			
FISH	19.89^{w}	37.73^{x}	37.27^{x}	$40.41^{x,y}$	43.06^{y}	0.84			
C20:4 (AA)							0.74	0.0001	0.003
CON	$6.00^{a,w}$	$1.40^{a,x}$	2.34^{y}	2.32^{y}	$1.89^{x,y}$	0.22			
FLAX	$6.92^{b,w}$	$1.88^{a,b,x}$	2.13^{x}	2.17^{x}	1.95^{x}	0.18			
FISH	$5.30^{a,w}$	$2.35^{b,x}$	2.73^{x}	2.31^{x}	2.14^{x}	0.20			
Total n-6[3]							0.34	0.0001	0.81
CON	24.98^{w}	42.11^{x}	$43.85^{x,y}$	$45.47^{y,z}$	47.41^{z}	0.80			
FLAX	23.78^{w}	42.72^{x}	42.81^{x}	44.75^{x}	45.68^{x}	0.69			
FISH	25.20^{w}	40.66^{x}	40.61^{x}	$43.40^{x,y}$	45.76^{y}	0.74			

[1] Presented as g fatty acid per 100 g fat.
[2] d0 = foaling; d+14 to d+84 = d post-foaling.
[3] Calculated as C18:2 + C20:4.
[a,b] Values in the same column for each fatty acid having different superscripts are different at $P < 0.05$.
[w,x,y,z] Values in the same row having different superscripts are different at $P < 0.05$.

Table 4-17. Omega-3 fatty acid content of foal plasma[1]

	Time[2]						P-values		
Fatty acid	d0	d+14	d+28	d+56	d+84	SEM	Treatment	Time	Treatment x Time
C18:3 (ALA)							0.04	0.0001	0.42
CON	0.18^{x}	2.16^{y}	$2.72^{a,y}$	$3.78^{a,z}$	$3.55^{a,z}$	0.25			
FLAX	0.1^{x}	2.2^{y}	$4.2^{b,z}$	$5.0^{b,z}$	$5.1^{b,z}$	0.21			
FISH	0.28^{x}	1.62^{y}	$3.24^{a,z}$	$4.19^{a,z}$	$4.5^{a,z}$	0.24			
C20:5 (EPA)							0.0001	0.0001	0.0001
CON	0.01	0.13^{a}	0.02^{a}	0.06^{a}	0.02^{a}	0.07			
FLAX	0.03^{x}	$0.11^{a,x,y}$	$0.25^{a,y}$	$0.01^{a,x}$	$0.02^{a,x}$	0.06			
FISH	0.21^{x}	$1.26^{b,y}$	$1.41^{b,y}$	$0.91^{b,z}$	$0.65^{b,z}$	0.06			
C22:6 (DHA)							0.0001	0.0001	0.0001
CON	1.52^{x}	$0.36^{a,y}$	$0.20^{a,y}$	$0.23^{a,y}$	$0.08^{a,y}$	0.12			
FLAX	1.57^{x}	$0.37^{a,y}$	$0.48^{a,y}$	$0.14^{a,y}$	$0.06^{a,y}$	0.10			
FISH	1.82^{x}	$1.91^{b,x}$	$2.09^{b,x}$	$1.55^{b,xy}$	$1.34^{b,y}$	0.11			
Total n-3[2]							0.0001	0.0001	0.01
CON	$1.89^{d,x}$	$2.78^{a,x,y}$	$2.97^{a,y}$	$4.19^{a,z}$	$3.67^{a,y,z}$	030			
FLAX	$1.75^{d,x}$	$2.94^{a,y}$	$5.13^{b,z}$	$5.31^{a,z}$	$5.48^{b,z}$	0.26			
FISH	$2.85^{e,x}$	$5.28^{b,y}$	$7.25^{c,z}$	$7.15^{b,z}$	$6.49^{b,z}$	0.28			

[1] Presented as g fatty acid per 100 g fat.
[2] d0 = foaling, d+14 = 14 d post-foaling, d+28 = 28 d post-foaling, d+56 = 56 d post-foaling, d+84 = 84 d post-foaling.
[3] Calculated as C18:3 + C20:5 + C22:5 + C22:6.
[a,b,c] Values in the same column for each fatty acid having different superscripts are different at $P < 0.05$.
[d,e] Values in the same column for each fatty acid having different superscripts are different at $P < 0.10$.
[x,y,z] Values in the same row having different superscripts are different at $P < 0.05$.

Table 4-18. Correlations between mare milk and mare plasma fatty acid concentrations and mare milk and foal plasma fatty acid concentrations

Correlation	r-value	P-value
Mare milk and plasma		
C18:2 (LA)	-0.01	0.90
C18:3 (ALA)	0.63	<0.0001
C20:4 (AA)	-0.18	0.10
C20:5 (EPA)	0.92	<0.0001
C22:6 (DHA)	0.69	<0.0001
Total n-6	-0.32	0.003
Total n-3	0.55	<0.0001
Mare milk and foal plasma		
C18:2 (LA)	-0.04	0.69
C18:3 (ALA)	0.70	<0.0001
C20:4 (AA)	-0.43	<0.0001
C20:5 (EPA)	0.78	<0.0001
C22:6 (DHA)	0.58	<0.0001
Total n-6	-0.20	0.03
Total n-3	0.50	<0.0001

Table 4-19. Overall effect of treatment on the fatty acid content of mare red blood cells[1]

	Treatment[2]				
Fatty acid	CON	FLAX	FISH	SEM	P-value
C8:0	ND	ND	ND	NA	NA
C10:0	ND	ND	ND	NA	NA
C12:0	ND	ND	ND	NA	NA
C14:0	ND	ND	ND	NA	NA
C16:0	41.48	41.20	39.57	0.87	0.28
C16:1	0.44	0.25	0.29	0.11	0.42
C17:0	0.37	0.26	0.28	0.06	0.39
C17:1	ND	ND	ND	NA	NA
C18:0	28.43	28.47	27.18	0.52	0.17
C18:1	24.33	24.32	24.97	0.52	0.62
C18:2n-6 (LA)	4.85	5.41	7.57	0.89	0.10

[1] Presented as g fatty acid per 100 g fat, ND = not detected in red blood cells, NA = not analyzed.

[2] CON = no supplement, FLAX = supplemented with milled flaxseed, FISH = supplemented with encapsulated fish oil.

Table 4-20. Linoleic acid content of mare red blood cells[1]

	Time[2]						P-values		
Fatty acid	d-28	d0	d+28	d+56	d+84	SEM	Treatment	Time	Treatment x Time
C18:2 (LA)							0.10	0.31	0.43
CON	4.92	4.84[a]	4.41	3.90[a]	6.20	0.88			
FLAX	5.94	5.81[a]	4.51	4.33[a]	6.50	0.88			
FISH	5.89[y]	9.85[b,z]	6.85[y,z]	8.34[b,y,z]	6.92[y,z]	0.92			

[1] Presented as g fatty acid per 100 g fat.
[2] d-28 = d before expected foaling date; d0 = foaling; d+28 to d+84 = d post-foaling.
[a,b] Values in the same column for each fatty acid having different superscripts are different at $P < 0.05$.
[y,z] Values in the same row having different superscripts are different at $P < 0.05$.

Table 4-22. Linoleic and alpha-linolenic acid contents of foal red blood cells[1]

	Time[2]						P-values		
Fatty acid	d0	d+14	d+28	d+56	d+84	SEM	Treatment	Time	Treatment x Time
C18:2 (LA)							0.04	0.03	0.31
CON	5.82	7.61^{ab}	9.63	7.69	7.96^{a}	1.39			
FLAX	4.03	4.73^{b}	7.56	7.50	6.25^{a}	1.38			
FISH	6.53^{x}	11.50^{a,y}	10.92^{y}	12.62^{y}	15.49^{b,y}	1.47			
C18:3 (ALA)							0.16	0.12	0.68
CON	0.08^{a}	0.01	0.00	0.00	0.00	0.08			
FLAX	0.60^{b,y}	0.39^{y,z}	0.00^{z}	0.25^{y,z}	0.00^{z}	0.08			
FISH	0.24^{a,b}	0.00	0.00	0.33	0.00	0.09			

[1] Presented as g fatty acid per 100 g fat.
[2] d0 = foaling; d+14 to d+84 = d post-foaling.
[a,b] Values in the same column for each fatty acid having different superscripts are different at $P < 0.05$.
[x,y,z] Values in the same row having different superscripts are different at $P < 0.05$.

Table 4-21. Overall treatment effect on the fatty acid composition of foal red blood cells[1]

	Treatment2				
Fatty acid	CON	FLAX	FISH	SEM	P-value
C8:0	ND	ND	ND	NA	NA
C10:0	ND	ND	ND	NA	NA
C12:0	ND	ND	ND	NA	NA
C14:0	ND	ND	ND	NA	NA
C16:0	36.80	37.18	33.95	1.0	0.07
C16:1	2.97	2.87	2.90	0.33	0.97
C17:0	0.15	0.18	0.08	0.08	0.66
C17:1	0.18	0.50	0.24	0.21	0.51
C18:0	21.85	21.60	19.78	0.57	0.03
C18:1	28.65	27.90	28.54	0.92	0.82
C18:2n-6 (LA)	7.74	6.01	11.41	1.41	0.04
C18:3n-3 (ALA)	0.02	0.25	0.11	0.09	0.16

[1] Presented as g fatty acid per 100 g fat, ND = not detected in red blood cells, NA = not analyzed.

[2] CON = no supplement, FLAX = supplemented with milled flaxseed, FISH = supplemented with encapsulated fish oil.

Table 4-23. Overall effect of treatment on mare serum and colostrum IgG content at foaling[1]

	Treatment[2]				
Sample type	CON	FLAX	FISH	SEM	P-value
Serum	2015.5	2406.7	2295.7	590.9	0.48
Colostrum	15570.0	15703.0	13560.0	1544.3	0.55

[1] Presented as mg/dL.
[2] CON = no supplement, FLAX = supplemented with milled flaxseed, FISH = supplemented with encapsulated fish oil.

Table 4-24. IgG content of mare milk[1,2]

	Treatment[3]			
Time post-foaling[4]	CON	FLAX	FISH	SEM
36h	211.8[a]	236.8[a]	212.7[a]	381.0
d+14	143.2[b]	149.8[b]	139.7[b]	381.0
d+28	108.2[b]	117.4[b]	105.4[b]	388.1
d+56	95.0[b]	95.0[b]	101.1[b]	414.5
d+84	72.4[b]	79.8[b]	80.2[b]	387.0

[1] Presented as mg/dL.
[2] Overall effect of treatment (P = 0.65), overall effect of time (P = 0.0001), overall effect of treatment x time (P = 0.82).
[3] CON = no supplement, FLAX = supplemented with milled flaxseed, FISH = supplemented with encapsulated fish oil.
[4] 36h = h post-foaling; d+14 to d+84 = d post-foaling.
[a,b] Values in the same column having different superscripts are different at P < 0.05.

Table 4-25. Correlations between IgG content of mare and foal serum, colostrum, and mare age

Correlation	r	P-value
Mare age and d0 serum		
Across treatments	0.13	0.50
CON	-0.13	0.74
FLAX	0.31	0.38
FISH	0.15	0.68
Mare age and colostrum		
Across treatments	-0.10	0.58
CON	-0.10	0.78
FLAX	-0.58	0.06
FISH	0.27	0.46
Mare d0 serum and colostrum		
Across treatments	0.24	0.21
CON	-0.06	0.87
FLAX	0.24	0.50
FISH	0.54	0.11
Mare d0 serum and foal 36h serum		
All treatments	0.42	0.02
CON	0.32	0.40
FLAX	0.32	0.37
FISH	0.63	0.05

Table 4-26. IgG content of foal serum[1,2]

	Treatment[3]			
Time[4]	CON	FLAX	FISH	SEM
d0	31.3[a]	88.5[a]	72.6[a]	103.1
36h	2603.1[b]	2608.4[b]	2198.8[b]	102.9
d+7	2160.8[c]	2355.1[b]	1933.3[b]	102.9
d+28	1603.5[d]	1430.1[c]	1184.1[c]	102.1
d+56	933.0[e]	1101.6[d]	705.0[d]	104.9
d+84	734.0[e]	761.6[d]	708.4[d]	104.2

[1] Presented as mg/dL.
[2] Overall effect of treatment (P = 0.32), overall effect of time (P = 0.0001), overall effect of treatment x time (P = 0.48).
[3] CON = no supplement, FLAX = supplemented with milled flaxseed, FISH = supplemented with encapsulated fish oil.
[4] d0 = foaling; 36h = h post-foaling; d+7 to d+84 = d post-foaling.
[a,b,c,d,e] Values in the same column having different superscripts are different at P < 0.05.

Table 4-27. Skin thickness of mares in response to an intradermal injection of phytohemagglutinin[1,2]

	Treatment[3]			
Hour	CON	FLAX	FISH	SEM
0	4.7[a]	4.5[a]	4.2[a]	0.34
2	12.0[b]	11.4[b]	12.3[b]	0.34
4	15.6[c]	14.6[c]	16.1[c]	0.34
6	15.9[c]	14.9[c]	15.8[c]	0.34
8	15.1[c]	15.3[c]	15.6[c]	0.34
12	12.5[b]	13.0[b]	12.8[b]	0.34
24	12.6[b]	12.6[b]	12.2[b]	0.34
48	9.0[d]	8.8[d]	8.7[d]	0.34

[1] Presented in mm.
[2] Overall effect of treatment (P = 0.89), overall effect of time (P = 0.0001), overall effect of treatment x time (P = 0.95).
[3] CON = no supplement, FLAX = supplemented with milled flaxseed, FISH = supplemented with encapsulated fish oil.
[a,b,c,d] Values in the same column having different subscripts are different at P < 0.05.

Table 4-28. Skin thickness of foals in response to an intradermal injection of phytohemagglutinin[1,2]

	Treatment[3]			
Hour	CON	FLAX	FISH	SEM
0	4.3^{u}	3.9^{u}	4.1^{u}	0.20
2	10.3^{v}	10.3^{vw}	9.9^{v}	0.20
4	13.2^{w}	14.0^{x}	13.2^{w}	0.20
6	$12.4^{a,x}$	$13.9^{b,x}$	$12.9^{a,b,w}$	0.20
8	12.1^{x}	13.0^{y}	11.8^{x}	0.20
12	10.0^{v}	10.8^{v}	10.6^{v}	0.20
24	9.6^{v}	9.7^{w}	10.2^{v}	0.20
48	6.4^{y}	6.7^{z}	7.0^{y}	0.20
SEM	0.46	0.44	0.49	0.20

[1] Presented in mm.

[2] Overall effect of treatment (P = 0.58), overall effect of time (P = 0.0001), overall effect of treatment x time (P = 0.15).

[3] CON = no supplement, FLAX = supplemented with milled flaxseed, FISH = supplemented with encapsulated fish oil.

[a,b] Values in the same row having different subscripts are different at $P < 0.05$.

[u,v,w,x,y,z] Values in the same column having different superscripts are different at $P < 0.05$.

Table 4-29. Skin response of mares and foals pooled across treatments to an intradermal skin test using phytohemagglutinin as the stimulant[1,2]

Hour	Mare	Foal	SEM
0	$4.5^{a,v}$	$4.1^{a,v}$	0.36
2	$11.9^{a,w}$	$10.2^{b,w}$	0.36
4	$15.4^{a,x}$	$13.5^{b,x}$	0.36
6	$15.5^{a,x}$	$13.1^{b,x}$	0.36
8	$15.3^{a,x}$	$12.3^{b,y}$	0.36
12	$12.7^{a,y}$	$10.4^{b,w}$	0.36
24	$12.5^{a,w,y}$	$9.8^{b,w}$	0.36
48	$8.9^{a,z}$	$6.7^{b,z}$	0.36

[1] Presented in mm.
[2] Overall effect of age (P = 0.0001), overall effect of time (P = 0.0001).
[a,b] Values in the same row having different subscripts are significant at P < 0.05.
[v,w,x,y,z] Values in the same column having different superscripts are different at P < 0.05.

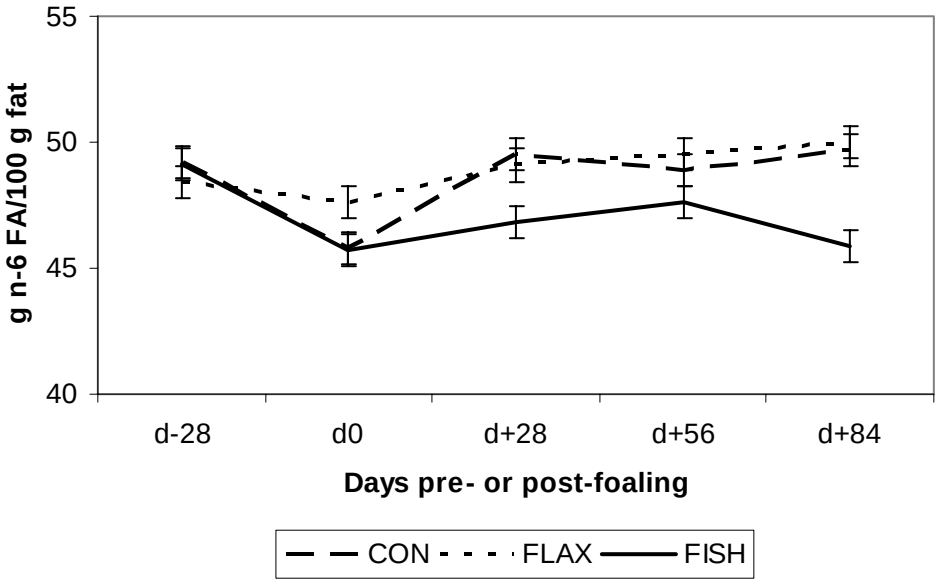

Figure 4-1. Total omega-6 fatty acid content in mare plasma from 28 d pre-partum to 84 d post-foaling.

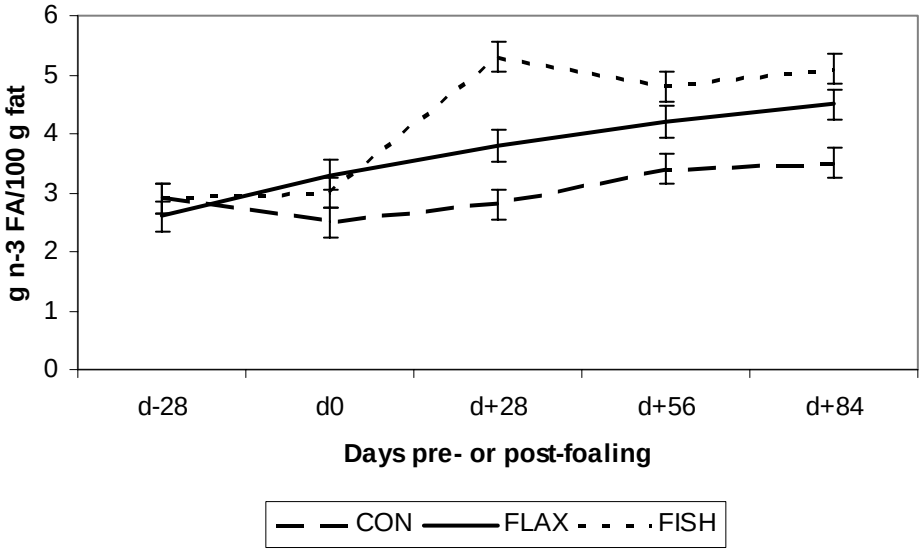

Figure 4-2. Total omega-3 fatty acid content in mare plasma from 28 d pre-partum to 84 d post foalingOmega-6:Omega-3 Fatty Acid Ratios

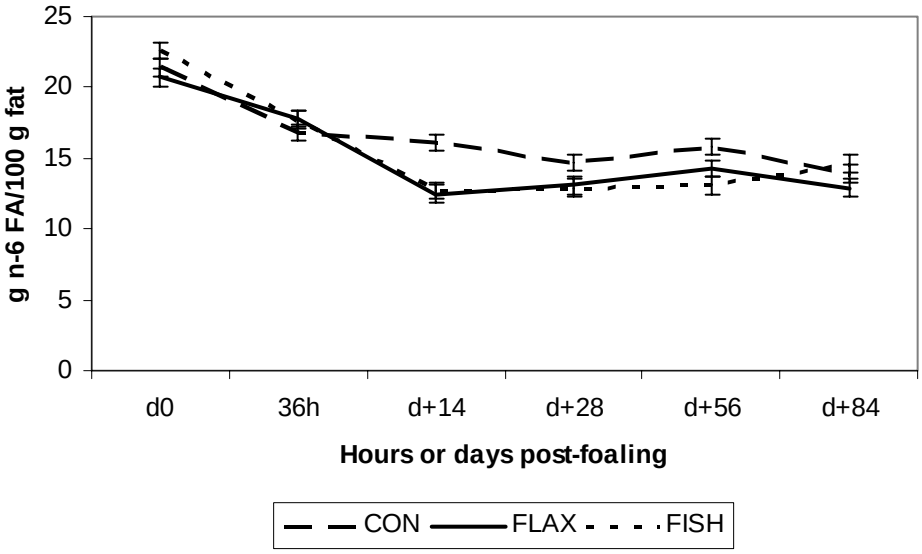

Figure 4-3. Total omega-6 fatty acid content of mare milk from foaling (d0) through 84 d post-foaling.

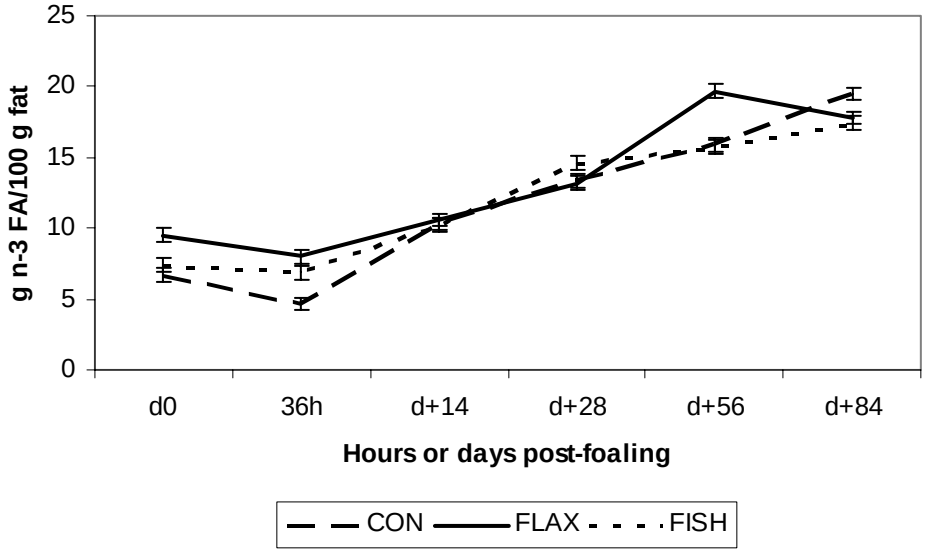

Figure 4-4. Total omega-3 FA content of mares milk from foaling (d0) through 84 d post-foaling.

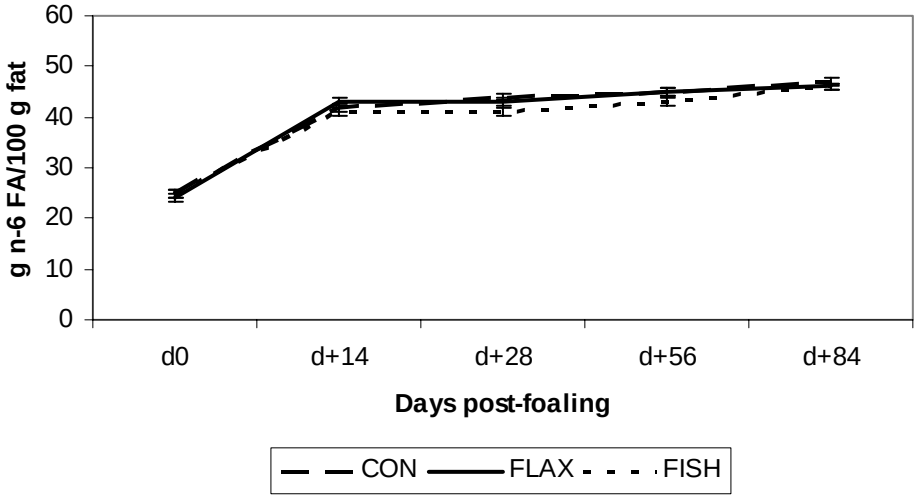

Figure 4-5. Total omega-6 fatty acid content of foal plasma from birth (d0) through 84 d of age.

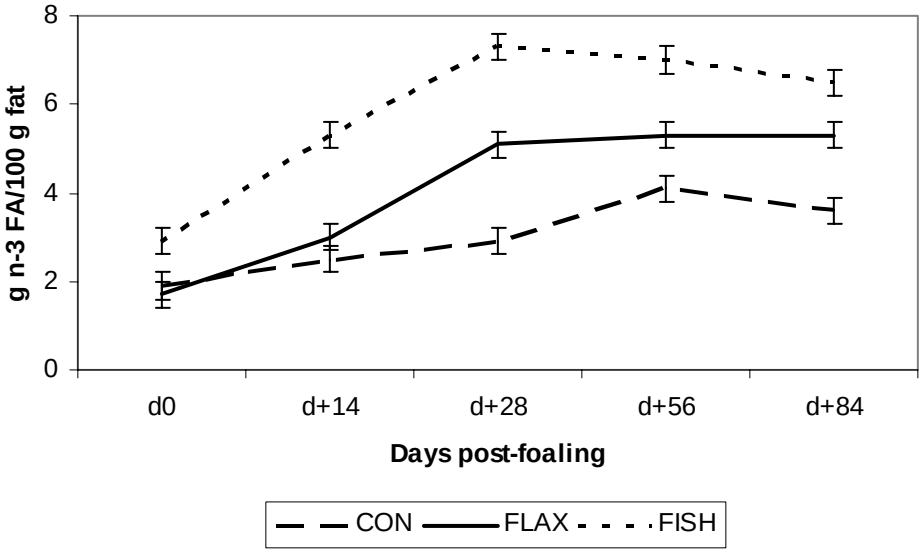

Figure 4-6. Total omega-3 fatty acid content of foal plasma from birth (d0) through 84 d of age.

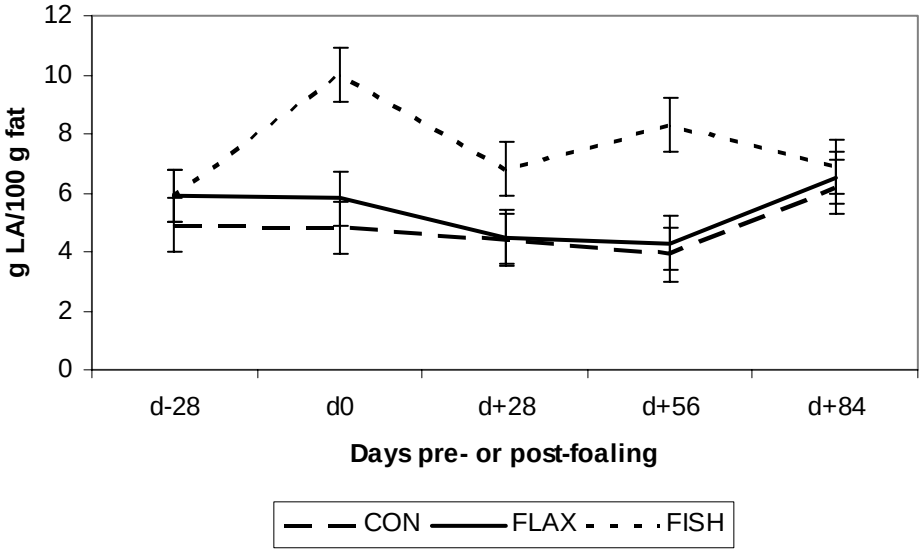

Figure 4-7. Linoleic acid content of mare red blood cells from 28 d pre-partum to 84 d post-foaling.

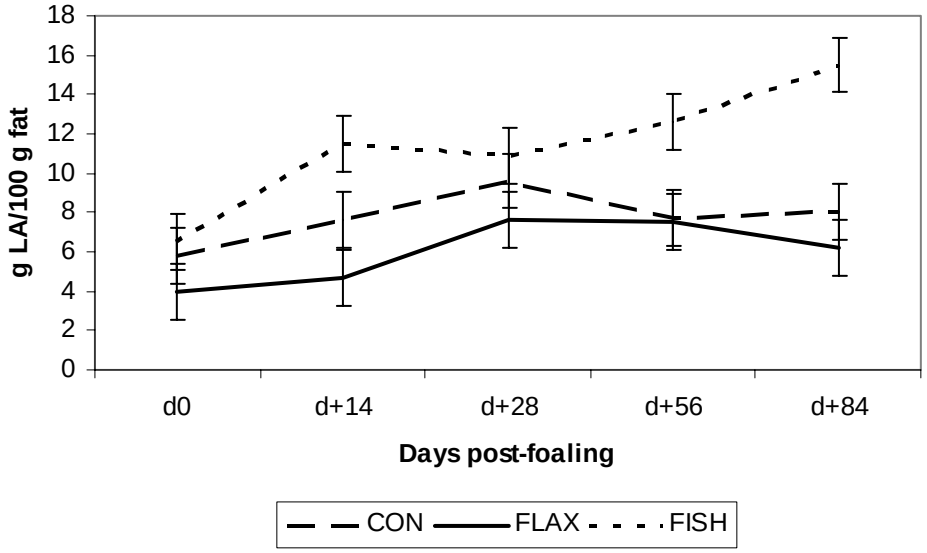

Figure 4-8. Linoleic acid content of foal red blood cells from birth (d0) to 84 d of age.

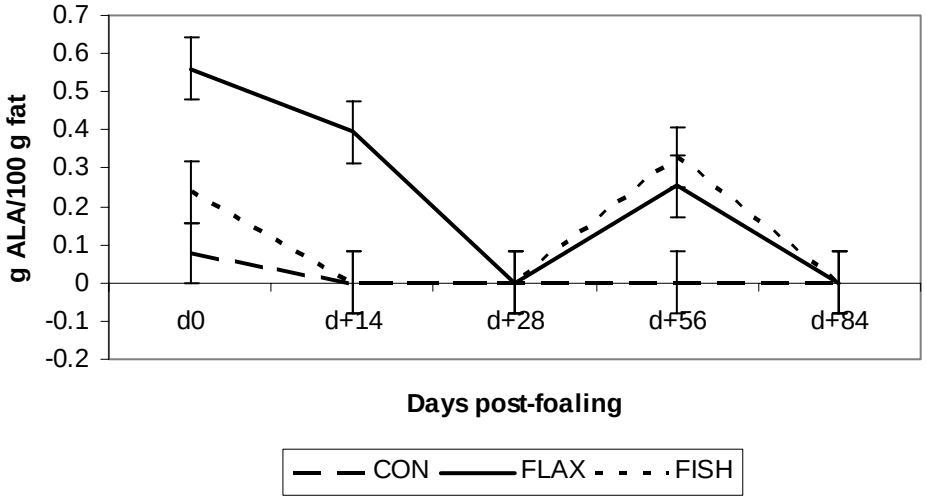

Figure 4-9. Alpha-linolenic acid content of foal red blood cells from birth (d0) to 84 d of age

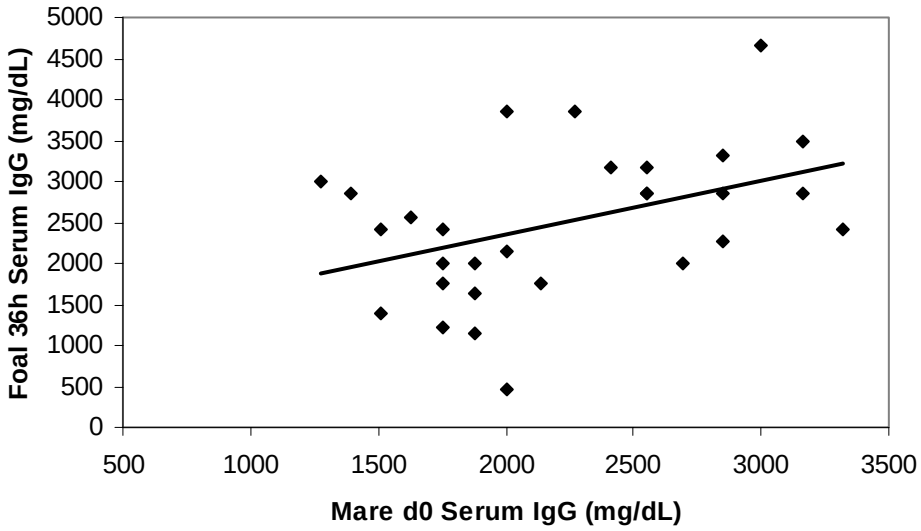

Figure 4-10. Correlation between mare serum IgG concentration at foaling (d0) and foal serum IgG concentration 36 h post-foaling. The equation of the line is y = 0.6543x + 1042.1, r = 0.42, P = 0.02.

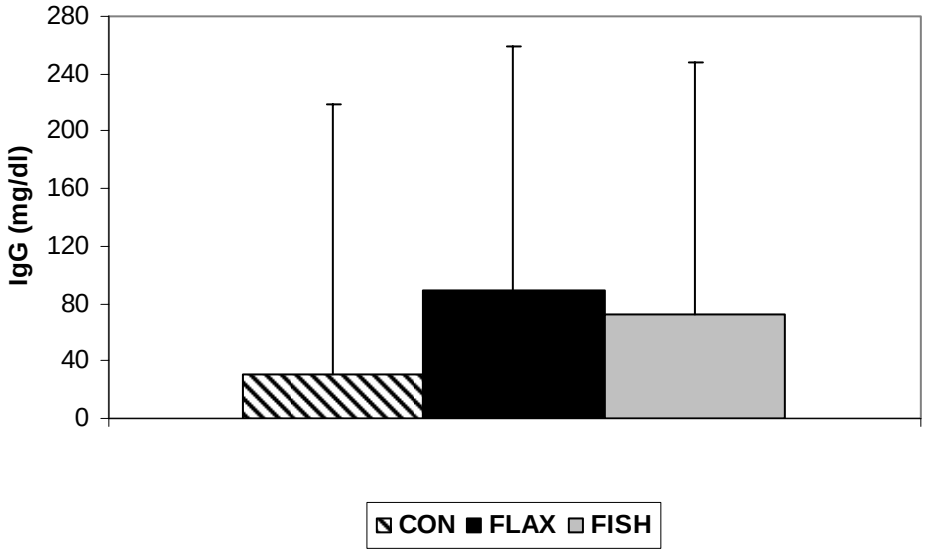

Figure 4-11. Foal serum IgG concentration at birth and before nursing.

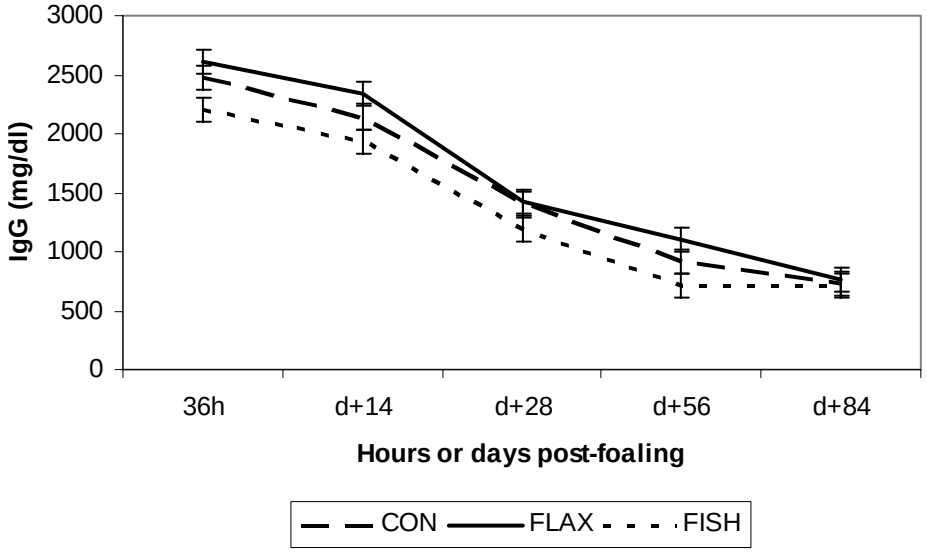

Figure 4-12. Foal serum IgG content after colostrum ingestion from 36 h to 84 d post-foaling.

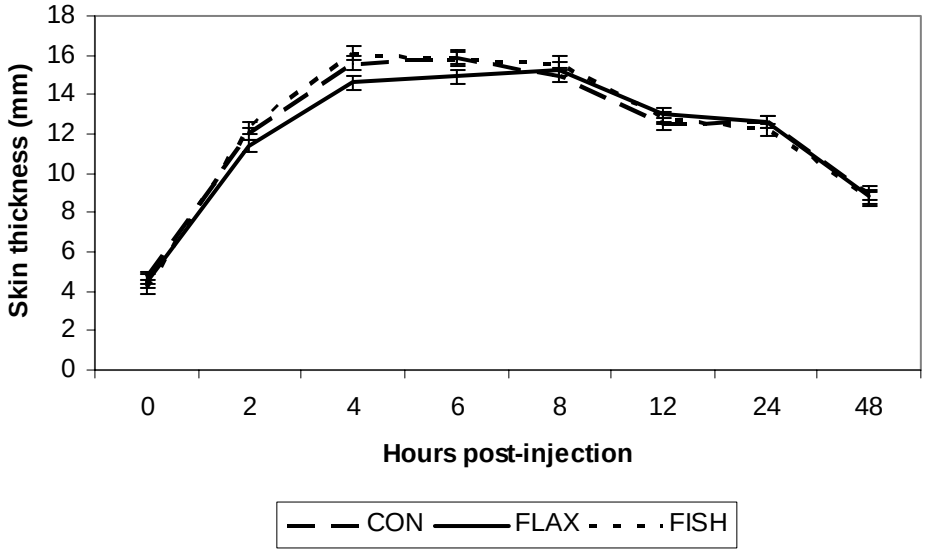

Figure 4-13. Skin thickness of mares in response to an intradermal injection of phytohemagglutinin.

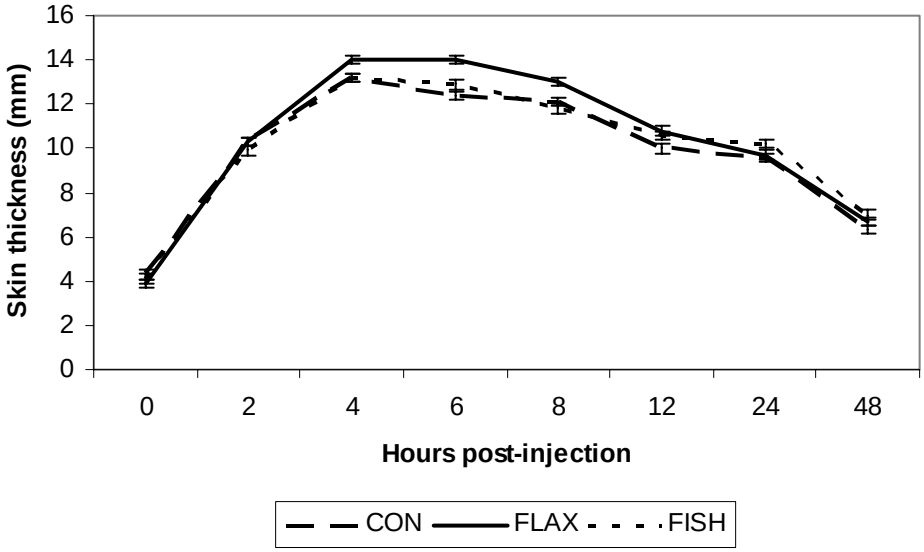

Figure 4-14. Skin thickness of foals in response to an intradermal injection of phytohemagglutinin.

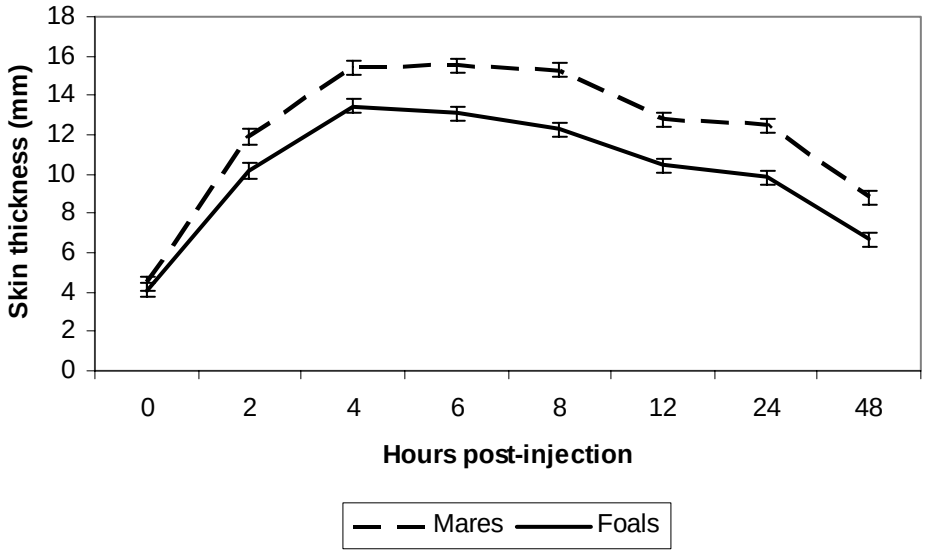

Figure 4-15. Skin thickness of mares and foals in response to an intradermal injection of phytohemagglutinin.

CHAPTER 5
DISCUSSION

Fatty Acid Composition of Feeds and Supplements

Almost half of the total FA in the basal grain mix concentrate was made up of LA. In contrast, ALA made up a very small proportion of the total FA in the grain mix. Commercial feeds formulated for horses typically contain cereal grains, such as oats, corn and barley, as well as soybean meal. Despite being relatively low in total fat (~2-3% of total DM), these ingredients contain a greater proportion of n-6 to n-3 FA (Ellis and Hill, 2005; Chen et al., 2006). The addition of fat to the diet is a common trend in the horse industry. Fat is typically provided in the form of soybean oil or corn oil, which increases the total amount of FA provided, but does not necessarily alter the FA composition of the grain mix. For example, O'Connor et al. (2004) offered horses a commercial grain mix concentrate in which soybean oil had been added (6.5% crude fat), but the relative proportions of n-6 and n-3 FA were similar to that found in the grain mix of the present study, even though oil was not included in the basal mix (3% crude fat). Ultimately, grain mixes can be a significant source of n-6 FA, particularly if horses consume a high grain, low forage diet.

In contrast to the grain mix, flaxseed is an excellent source of ALA. The milled flaxseed used in the present study contained 61.20 g ALA/100 g fat, which was slightly higher but within range of previous reported values (Bauer et al., 1998; Francois et al., 2003). The milled flax fed to horses in the current study was a human food-grade

product. Flax of higher quality can be expected to have greater levels of ALA, as well as increased resistance to lipid peroxidation.

Although low in ALA, fish oil is also a significant source of n-3 FA, predominantly EPA, DPA and DHA. The EPA concentration of the encapsulated fish oil supplement used in the current study was 1.4 to 1.6 times values reported for menhaden fish oil (Hall et al., 2004a, 2004b; O'Connor et al., 2004) and cod liver oil (Helland et al., 1998), whereas the DHA concentration of the encapsulated fish was 1.5 times that found in menhaden fish oil (Hall et al., 2004a, 2004b; O'Connor et al, 2004), but similar to that found in cod liver oil (Helland et al., 1998). The encapsulated fish oil used in the present study was derived from menhaden fish oil; however, it is likely that slight variations in FA content between different sources of fish oil accounted for the differences in EPA and DHA between this study and others.

The ALA content of bahiagrass pasture increased from winter to spring months in the current trial. In an evaluation of several species of temperate grasses, Dewhurst et al. (2001) reported a decline in ALA from April to June/July, with a slight rebound in ALA concentration from September through November. However, in a later study, Dewhurst et al. (2002) observed a steady increase in ALA of perennial ryegrass pasture samples from May through November. The authors explained this difference in ALA response was likely due to the interruption of flowering and inflorescence emergence, because pasture samples were obtained with greater frequency in the later trial (20-30 d regrowth), compared to their previous study (40 d regrowth).

In the current study, bahiagrass pasture samples were obtained at approximately 30 d intervals. More importantly, samples were obtained from areas where there was

evidence of grazing. Horses are selective grazers, preferring short, vegetative growth and avoiding tall, more mature grasses. Therefore, pasture samples were primarily made up of pre-florescent plant material, which could explain the increase in ALA noted between winter and spring months. In addition, the growth of bahiagrass slows or ceases completely in the winter months in north central Florida. Changes in the FA content of pasture forages are more pronounced when the plant is actively growing (Dewhurst et al., 2001 and 2002).

Although bahiagrass is a warm-season forage, the ALA content of bahiagrass pasture was within the range of values reported by Dewhurst et al. (2001) for temperate pasture grasses, including orchardgrass, tall fescue, meadow fescue, timothy, annual ryegrass and perennial ryegrass. However, the LA content of bahiagrass pasture was almost double of that observed by Dewhurst et al. (2001) for temperate grasses. These differences in LA may be due to differences in FA accumulation between cool-season (temperate) and warm-season grasses. O'Kelly and Reich (1976) reported the fatty acid composition of several tropical grasses and legumes harvested from pastures in Australia. The LA content of these tropical pasture forages was similar to the LA found in the bahiagrass in the current study; however, the ALA content of the Australian tropical forage was substantially lower than bahiagrass. To the author's knowledge, the current study is the first to report the FA composition of freshly cut bahiagrass.

The FA profile of the Coastal bermudagrass hay fed in the current study contained 25% more LA and 45% less ALA than the timothy hay fed to horses by O'Connor et al. (2004). When compared to meadow bromegrass hay, Coastal bermudagrass had a similar ALA concentration, but about 40% more LA (P.D. Siciliano, unpublished data). Both the

timothy and meadow brome hays had more total n-3 FA than total n-6 FA (n-6:n-3 ratio of 0.6 to 0.8:1), whereas the Coastal bermudagrass hay had more total n-6 FA (n-6:n-3 ration of 1.45:1). The difference in the FA composition of these hays may not only be due to species, but also to morphological differences between cool-season and warm-season grasses. Unfortunately, the available literature on the FA composition of warm-season hays is lacking. Nonetheless, tropical forage grasses collected from Australian pastures in the winter months (July-September) also contained more n-6 FA than n-3 FA, yielding an n-6:n-3 FA ration of 1.2 to 2.1:1 (O'Kelly and Reich, 1976). Although these tropical grasses were obtained from pasture clippings, the authors noted the grasses were "dormant and dried," which may provide a similar comparison to dried hay. Additional study is necessary to better characterize the FA profiles of warm season grasses commonly fed to horses in the southern United States.

Mare and Foal Bodyweight

After foaling, all mares were able to maintain BW, indicating that DE intake was likely sufficient to meet the needs of lactation in all treatments. The amounts of DE provided by the milled flaxseed and encapsulated fish oil fed in this study were relatively small. The milled flaxseed contributed approximately 0.5 Mcal/d and the encapsulated fish oil contributed approximately 2.0 Mcal/day, equating to between 1.0 and 5.0% of the total DE requirement for lactating broodmares (NRC, 1989). Because CON mares showed no difference in bodyweight when compared to FLAX and FISH mares and there was no difference in grain intake between treatments, it is reasonable to assume that CON mares were able to adjust their hay and grazing intake to meet their DE needs.

Foal bodyweight was also not affected by treatment of the mare. In part, this can be explained by the fact that gestation length did not differ between CON (351 ± 1.89

days), FLAX (354 ± 1.82 days) or FISH mares (350 ± 2.35 days) in the present experiment (P = 0.84). The lack of effect on length of gestation is contrary to the positive correlation noted between dietary fish oil intake for the entire duration of pregnancy and a longer gestation length in rats (Olsen et al., 1990). In sheep, the continual infusion of fish oil for 24 hours prior to induction of premature delivery also delayed the onset of labor and time of delivery (Baguma-Nibasheka et al., 1999).

Eicosanoids, particularly those derived from AA, regulate gestation length and the onset of parturition (Allen and Harris, 2001). Because n-3 FA compete with n-6 FA for desaturation enzymes, a high dietary intake of n-3 FA can lead to an increased production of the 3-series prostaglandins from EPA and a decreased production of the 2-series prostaglandins from AA, ultimately inhibiting the onset of parturition (Mattos et al., 2000). Omega-3 FA also promote vasodilatation and reduce blood viscosity, both of which facilitate placental blood flow and improve fetal growth, thus allowing for an increased birth weight (Baguma-Nibasheka et al., 1999). However, the lack of differences for length of gestation and foal bodyweights seen in the present study are supported by studies performed on sows and mares that also showed no differences in gestation length or newborn bodyweights when dams were fed n-3 FA from 7 to 45 days prior to parturition (Fritsche et al., 1993; Duvaux-Ponter et al., 2004).

Mare Plasma Fatty Acid Content

This study showed that mare plasma n-3 FA profiles were altered after 28 days of FA supplementation. Siciliano et al. (2003) found significant changes in plasma FA levels within 14 d of supplementing with flax or fish oil.

The fatty acid profiles of mare plasma in the present study mirrored the PUFA content of the supplements fed. FISH mares had higher plasma concentrations of EPA

and DHA, reflecting the content of the encapsulated fish oil supplement. Similarly, the FLAX mares, who consumed a supplement rich in ALA, tended to have higher ALA. FLAX and FISH mares had similar plasma total n-3 FA concentrations, reflecting the nearly equal quantities of total n-3 FA consumed by the two groups. The transition in principle forage source from hay to spring pasture resulted in a proportional increase in total n-3 FA intake for all mares. Nonetheless, mares supplemented with flax or fish oil continued to receive approximately 30% more total n-3 FA than unsupplemented mares. In addition, the differences in plasma FA between treatments were maintained over the course of the trial and were unaffected when forage source was included as a variable in the statistical analyses. Therefore, the effects of FA supplementation observed in this study can be primarily attributed to the flax or fish oil consumed by the mares.

All mares consumed similar levels of LA, while only FISH mares consumed AA. In the blood, LA increased over time in CON and FLAX mares but stayed level in FISH mares. Plasma AA was elevated only at foaling in CON and FISH mares. An explanation of the elevated AA concentration at foaling could be that the onset of foaling caused an increase in the synthesis of prostaglandins. Prostaglandins, particularly $PGF_{2\alpha}$ and PGE_2, are present at high concentrations immediately before and during parturition (Mattos et al., 2000) and are synthesized from AA. Therefore, more LA would need to be converted to AA to fuel the prostaglandin synthesis. However, prostaglandins were not determined in the present experiment. Differences in LA and AA at foaling between treatments could also be related to differences in ALA in the diet. FLAX mares had the highest ALA intake and the highest ALA plasma levels, and this ALA could have competed with LA for conversion enzymes, particularly Δ^6-desaturase. Therefore, the

increased ALA in FLAX mares could explain why these mares did not have elevated plasma AA at foaling.

Mare Milk Fatty Acid Content

Data in the current study confirmed that mare milk is rich in short and medium chain FA, making up approximately 30% of all FA detected, which agrees with reports in other studies (Doreau et al., 1993; Csapo et al., 1995; Duvaux-Ponter et al., 2004). The present study also confirmed the low proportion (1-2%) of stearic acid (C18:0) in mare milk and the relatively high proportion (5-6 %) of palmitoleic acid (C16:1) when compared to cow's milk (Doreau and Boulot, 1989). The presence of odd chain length FA (C17:0 and C17:1; present at less than 1%) in mare could reflect the comparatively high pH in the horse stomach, which would allow for bacterial hydrogenation and the formation of odd chain FA (Doreau et al., 1993; Hoffman et al., 1998; Duvaux-Ponter et al., 2004).

The LCFA detected in the greatest quantity in mare milk was LA, which agrees with data from other studies (Doreau and Boulot, 1989; Csapó et al., 1995). After supplementation, mares fed flaxseed had higher milk ALA than FISH mares and CON mares. In response to dietary treatment, FISH mares had a greater milk EPA and DHA content than FLAX mares. This occurrence supports that diet influences milk FA composition. The low levels of EPA and DHA in FLAX mares also supports the idea that the conversion of ALA to EPA and DHA is difficult in the mare udder and occurs at a low rate. Studies in women have supported this idea by showing that supplementation with flaxseed oil during lactation did not increase the women's breast milk DHA content (Francois et al., 2003). When using dogs as subjects, supplementation during gestation and lactation with menhaden fish oil promoted an increase in DHA milk content, while

supplementation with linseed oil did not (Bauer et al., 2004). This same occurrence was observed in mares, where supplementation with linseed oil during gestation or and lactation did not increase mare milk DHA (Duvaux-Ponter et al., 2004; Spearman et al., 2005). Consequently, it seems that supplementing the mare with ALA is not an effective method of increasing EPA and DHA in milk. The only way of increasing EPA and DHA in milk appears to be to supply it in the diet of the mare.

Strong correlations were observed between mare plasma and milk for ALA, EPA and DHA, but not for AA. In women, plasma EPA and DHA have been correlated to breast milk EPA and DHA (Helland et al., 1998). The strong correlation between mare plasma and milk ALA, EPA and DHA seen in the present study may suggest that EPA and DHA are entering the mammary gland from the plasma. In contrast, the lack of correlation between mare plasma and milk AA suggests that the mammary gland may be capable of synthesizing AA from LA.

Foal Plasma Fatty Acid Content

At birth, a complete absence of short and medium-chain FA in the plasma of foals suggests that these FA are preferentially used as energy sources by the suckling foal. A similar finding was reported in newborn calves (Hocquette and Bauchart, 1999). Fatty acids with chain lengths of less than 16 carbons are transported directly to the liver via the portal vein where they are catabolized (Duvaux-Ponter et al., 2004). While plasma concentrations of these short- and medium-chain FA increased slightly as the foals aged, overall plasma levels were still quite low. This phenomenon, coupled with the fact that mare milk contained relatively high amounts of short and medium chain FA, shows that foals may use these FA for energy production through 84 days of age.

Supplementation of the mare also appeared to affect circulating concentrations of longer chain PUFA in the newborn foal. Prior to suckling, foals born to mares supplemented with flaxseed had an elevated level of AA in plasma. One explanation for this occurrence may be that FLAX foals converted more LA to AA while in utero. Blood in the umbilical vein, which drains the placenta, passes through the foal's liver. Hence, the fetal horse liver could use the enzyme Δ^6-desaturase to covert LA to AA (Duvaux-Ponter et al., 2004). Direct assessment of placental transfer of FA was not undertaken in this study; however, it is also possible that FLAX mares transferred more AA across the placenta than did mares of other treatments. Mares exhibited lower plasma AA at foaling when supplemented with flaxseed, which could have resulted from sending more of the mare's plasma AA across the placenta to the foal. However, it is unclear as to why any of these events would take place in response to flaxseed vs. fish oil or no supplementation.

In contrast to Stammers et al. (1987) but in agreement with Kruglik et al. (2005), the present study showed the presence of EPA and DHA in the jugular blood of newborn foals. Since foals had not yet had access to mares' milk, these FA must have resulted from a transfer across the placenta from mare to foal. Work by Stammers et al. (1987, 1991) has demonstrated that FA are capable of crossing the placenta from mare to foal. Foals born to fish oil supplemented mares showed a tendency to have a higher plasma total n-3 FA content at foaling, primarily due to an increase in DPA. This increased plasma DPA was likely a result of the high amount of DPA present in the fish oil supplement fed to the mares.

The feeding of both flaxseed or encapsulated fish oil to mares significantly modified the plasma PUFA profiles of foals suckling those mares. Foals suckling FLAX mares had higher plasma ALA, while foals nursing FISH mares had higher plasma EPA and DHA, as well as a higher plasma total n-3 FA content. These results agree with those described for foals suckling mares fed linseed (Duvaux-Ponter et al., 2004) and foals suckling mares fed a marine-derived protected n-3 FA source (Kruglik et al., 2005). The FA composition of foal plasma mirrored that of mare milk, as FLAX mares had higher milk ALA and FISH mares had higher milk EPA and DHA. However, FISH foals had the highest plasma total n-3 FA, even though mare milk did not differ in total n-3 FA content. It is possible that since foals had the opportunity to consume the mare's grain, both FLAX and FISH foals were able to consume some of the mare's supplement. Foals may not have been as capable of digesting the flaxseed with the same efficiency as the fish oil, which could have altered plasma n-3 FA concentrations.

The plasma of all foals also showed dramatic increases in FA from birth to 14 d of age. Similar observations were noted in foals (Duvaux-Ponter et al., 2004) and piglets (Fritsche et al., 1993). The increases seen in n-3 PUFA in foal plasma suggest that the foal is capable of digesting and absorbing long chain FA from milk.

Mare and Foal Red Blood Cell Fatty Acid Content

Although n-3 PUFA have been found in adult human (Makrides et al., 1996; Francois et al., 2003) and adult horse RBC (King et al., 2005), these FA were not detected in the RBC of mares in the present study. Linoleic acid was the longest chain PUFA discovered in mare RBC, and FISH mares had a higher LA content. This higher LA content cannot easily be explained, as it disagrees with the results of past research

examining human and horse red blood cell FA contents after dietary supplementation with DHA (Makrides et al., 1996; King et al., 2005).

To the author's knowledge, no studies exist that have examined the FA content of newborn foal RBC. However, studies on humans have shown that the n-3 FA concentrations of infant RBC increase when the concentrations of these FA increase in the diet (Henderson et al, 1992; Innis, 1992b). Studies conducted in rats have shown that increasing maternal dietary ALA increases the ALA content of rat pup whole body and tissue (Bowen and Clandinin, 2000). The only PUFA found in foal RBC in the present study were LA and ALA. Similar to the results of mare RBC, FISH foals had a significantly higher LA red blood cell content, and no treatment differences were noted in ALA. It is again unclear as to why FISH foals had a higher LA content, especially considering there was no difference in foal plasma LA content. Further research is needed to determine how FA supplementation of the mare can influence uptake and incorporation of FA into foal cell membranes.

Evidence of FA transfer across the placenta was also demonstrated by the FA composition of red blood cells in newborn foals. Foals born to FLAX mares had higher ALA in red blood cells in samples obtained before they first suckled. The finding of ALA, but not EPA or DHA, in newborn foal RBC suggests that the high requirement for long-chain PUFA, particularly DHA for fetal brain development in late gestation, may preferentially direct these FA into brain tissue rather than RBC membranes. However, supporters of this theory also hold that since ALA is a precursor to EPA and DHA, ALA would not be present in RBC membranes either as it would be converted into its longer chain derivatives (Duvaux-Ponter et al., 2004). The findings of ALA in newborn foal

RBC of the present study may show that not all ALA is preferentially incorporated into brain tissue and some may be retained in RBC membranes. It is also possible that the ALA supplied from the mare's intake of flaxseed may have exceeded amounts required for fetal development and was therefore stored in all membranes.

In the present study, the ability to accurately determine mare and foal red blood cell FA content may have been undermined because of laboratory procedures. The centrifuge used to process RBC was non-refrigerated. In order to process the large number of horses that were sampled at each time period, the centrifuge was frequently in use for long periods of time, which may have allowed excessive heating of the sample. In light of research done in other laboratories that has shown the occurrence of PUFA in animal RBC and of further tests performed in our laboratory, we believe the heating action of the RBC in the centrifuge could have resulted in the degradation of the small amounts of PUFA that may have been present in the sample.

Effect of n-3 Supplementation on IgG

The IgG content of mare colostrum was much higher than observed by others (Duvaux-Ponter et al., 2004; Kohn et al., 1989; Pearson et al., 1984), but was similar to that measured from mares in a previous study conducted at the same facility (Spearman, 2004). Breed and age did not appear to affect mare colostrum IgG. Breed effects on colostrum IgG have been noted in other studies, which the authors attributed to differences in body size between breeds (LeBlanc et al., 1986, 1992). Larger breeds are capable of greater colostrum production, resulting in a dilution of IgG (Pearson et al., 1984). However, BW did not differ between Thoroughbred and Quarter Horse mares in this project; therefore, body size most likely did not contribute to colostrum IgG differences. Age has also been shown to effect mare colostrum IgG, with older mares

having lower IgG concentrations (LeBlanc et al., 1992). However, other studies have shown age to have no effect on colostrum IgG (Morris et al., 1985; Erhard et al., 2001). The effects of breed and age on colostrum IgG content are not well defined, partly because of the high variation seen between individual mares. In addition, colostrum IgG varies dramatically according to when it is sampled. Colostrum IgG decreases markedly during the first 12 hours after birth, with a decrease of approximately 20% possible in the first 6 hours after birth (Pearson et al., 1984). Therefore, differences in sampling times may explain the differences in IgG values seen across studies. In the current study, colostrum was obtained with the first hour after birth.

The fact that FISH mares had numerically lower colostrum IgG content in this study cannot easily be explained. Hoffman et al. (1998) observed higher IgG concentrations in colostrum from mares supplemented with corn oil, which is rich in LA. In contrast, Duvaux-Ponter et al. (2004) found that supplementation of mares with linseed oil did not affect the IgG in mare colostrum. More recently, Kruglik et al. (2005) fed mares a marine-derived protected n-3 FA source rich in EPA and DHA and compared their response to mares fed an isocaloric amount of corn oil. Supplementation with EPA and DHA significantly increased colostrum IgG at foaling, but not at 12 or 24 hours after foaling (Kruglik et al., 2005).

Low colostrum IgG has been associated with maiden mares (Jeffcott, 1972; Erhard et al., 2001) and prelactation (Jeffcott, 1947; Morris et al., 1985; LeBlanc et al., 1992); however, the FISH treatment group did not have a higher number of maiden mares and none of the FISH mares were observed dripping milk prior to parturition. Three of the mares receiving FISH supplementation had dramatically lower colostrum IgG than

any other mare on the study. When data from these mares was removed, the mean colostrum IgG of FISH mares was comparable to that observed in CON and FLAX mares.

With the exception of two foals, all foals had serum IgG levels high enough to suggest passive transfer (at least 800 mg/dL) at 12 h post-foaling. The two foals that had IgG levels lower than 800 mg/dL were not given any colostrum or plasma transfusions but were closely monitored for the first 48 hours after foaling. Neither foal developed any problems.

The values for foal serum IgG seen in the present study were higher than those reported by others (Erhard et al., 2001; Duvaux-Ponter et al., 2004). Nonetheless, the decline in foal serum IgG before 28 d of age agrees with that commonly observed by others (Erhard et al., 2001; Duvaux-Ponter et al., 2004). No increases in serum IgG content were seen in foals at any time point during the present study, suggesting that initiation of IgG production by the foals had not begun by 84 d of age. Erhard et al. (2001) reported an increase in foal serum IgG at 47 days of age, suggesting that foals were beginning to synthesize their own IgG. However, other studies have shown that foals do not begin to produce their own IgG until four months of age (Jeffcott, 1974a, 1974b, 1975).

Omega-3 supplementation of mares had no effect in foal serum IgG in the current study, which is likely due to the lack of treatment effect on colostrum IgG. Although Kruglik et al. (2005) found higher colostrum IgG in mares supplemented with fish oil, the serum IgG concentration of foals was not different from foals who were nursing mares supplemented with corn oil.

In the present study, no relationships were found between mare serum IgG and colostrum IgG at foaling or between mare colostrum IgG and foal serum IgG at 36 h after birth. This lack of relationship agrees with the work of Lavoie et al. (1989) but disagrees with Erhard et al. (2001), who reported a correlation between colostrum and foal serum IgG. Erhard et al. (2001) also reported that mare serum at foaling was significantly correlated to foal serum at 36 hours, which was similar to that observed in the present study. The lack of correlation between mare colostrum and foal serum, but the presence of a correlation between mare and foal serum IgG, suggests that factors other than colostrum IgG concentration determine foal serum IgG concentrations, including the volume of colostrum produced, colostrum intake by foals and the efficiency of IgG transfer across the foal's intestine.

Mare and Foal Inflammatory Response

In the current study, inflammatory response in mares and foals was evaluated *in vivo* using an intradermal injection of PHA. In mares, peak skin thickness responses to PHA were observed between 4 and 8 hours post-injection, whereas peak skin thickness responses were observed at 4 hours post-injection in foals. The time frame of these responses agrees with Ward et al. (1993), who observed a peak inflammatory response at 4 h. Mature animals have a more developed immune system with a greater reaction capability than do young animals (Calder, 2001); therefore, it was not surprising that mares reacted to a greater extent than did foals. Supplementation with flaxseed or fish oil had no detectable effects on the inflammatory response to PHA in mares or foals. Hall et al. (2004b) was also unable to demonstrate an effect of n-3 supplementation on *in vivo* inflammatory response as horses fed fish oil had a delayed-type hypersensitivity response similar to horses fed corn oil when challenged with keyhole limpet hemocyanin.

Most of the work done to address the effects of fish oil supplementation on inflammatory responses in horses has focused on *in vitro* cellular production of eicosanoids. The intradermal skin test performed in this study, while widely used, is somewhat crude. This skin test measures the cumulative effects of inflammation, but cannot directly measure immune cell responses or eicosanoid production, so there may have been changes at the skin level that this test could not detect. It has been reported that the skin epidermis relies on the PUFA content of the blood and that altering blood PUFA levels by dietary manipulation can alter the synthesis of epidermal eicosanoids (Wright, 1991). When adult horses were fed Purple Viper's Bugloss oil (contains ALA and C18:4n-3), the skin of these horses showed a significant increase in n-3 FA (Bergero et al., 2002). The skin of these horses also showed a selective inclusion of FA with a strong preference for n-3 FA over n-6 FA (Bergero et al., 2002). Therefore, it may be possible that the increase in horses supplemented with flaxseed or fish oil in the present study could correlate to an increase of these FA in their skin, which could potentially alter eicosanoid production in response to an antigen.

CHAPTER 6
IMPLICATIONS

This study demonstrated that n-3 FA supplementation of mares one month before parturition can influence FA available to the foal *in utero*. After birth, but before suckling, foals from flax supplemented mares had higher red blood cell ALA concentrations, and foals from fish oil supplemented mares had elevated plasma levels of DPA and total n-3 FA. The transfer of n-3 FA across the placenta has the potential to modulate the immune response of the foal. Nonetheless, augmenting the n-3 FA content of the mare's diet with milled flaxseed or encapsulated fish oil did not increase IgG content in mare colostrum, nor did it appear to enhance uptake of IgG by the suckling foal. Perhaps a longer period of n-3 FA supplementation during gestation is needed to allow for greater incorporation of EPA and DHA into cell membranes. The resulting increase in membrane fluidity and permeability could permit greater entry of IgG into mammary tissue, as well as greater absorption of IgG by foal enterocytes, thereby facilitating passive transfer of immunity to the naïve foal.

Supplementing the mare with 6 g total n-3 FA/100 kg BW led to the enrichment of n-3 FA in milk, and subsequently, the plasma of the suckling foal. Although flaxseed increased the quantity of ALA available to the foal, supplementation with fish oil appears to be a more effective method of increasing EPA and DHA in plasma and milk.

Despite alterations in circulation n-3 FA, supplementation of the mare with flaxseed or fish oil did not modify the inflammatory response to PHA in mares or foals as

measured by skin thickness. Although crude, the method does allow cumulative assessment of innate immune response *in vivo*.

Future study should characterize the effects of different sources of n-3 FA on eicosanoid and cytokine production at the cellular level. In addition, optimal amounts of dietary n-3 FA and/or ratios of n-6:n-3 FA needed to confer immune benefits in horses deserves further investigation. Such information is especially relevant to the health of lactating mares and growing horses, which are typically fed large amounts of grain-based feeds that are high in n-6 FA in order to meet their high nutrient requirements. Furthermore, the popularity of fat-added feeds, most of which contain oils rich in n-6 FA, as well as reduced access to grazing, may have important biological consequences as a result of reduced n-3 FA supply.

APPENDIX A
RAW DATA

Mare Expected and Actual Foaling Dates and Dates Started on Trial

Table A-1. FISH mare expected foaling dates, actual foaling dates and dates started on trial

Mare	Expected foaling date	Actual foaling date	Date on trial
B46	1/21/2005	2/18/2005	12/23/2004
W69	1/24/2005	2/7/2005	12/27/2004
B33	1/30/2005	2/14/2005	1/3/2005
B14	2/8/2005	2/14/2005	1/10/2005
B01	2/20/2005	2/18/2005	1/25/2005
B31	3/2/2005	3/14/2005	2/2/2005
A59	3/6/2005	3/6/2005	2/7/2005
B47	3/10/2005	3/22/2005	2/10/2005
B35	3/20/2005	3/27/2005	2/21/2005
B24	3/24/2005	4/8/2005	2/24/2005
B23	3/25/2005	3/29/2005	2/24/2005
A66	4/11/2005	4/16/2005	3/14/2005

Table A-2. FLAX mare expected foaling dates, actual foaling dates and dates started on trial

Mare	Expected foaling date	Actual foaling date	Date on trial
B6	1/27/2005	2/15/2005	12/30/2004
B19	1/28/2005	2/15/2005	12/30/2004
A65	2/11/2005	2/21/2005	1/13/2005
B44	2/17/2005	2/22/2005	1/20/2005
C1	2/27/2005	3/16/2005	1/31/2005
A62	3/1/2005	3/18/2005	1/31/2005
B21	3/3/2005	3/23/2005	2/2/2005
C2	3/5/2005	3/25/2005	2/2/2005
B28	3/7/2005	3/8/2005	2/7/2005
C6	3/8/2005	3/17/2005	2/7/2005
B32	3/24/2005	4/10/2005	2/24/2005
B30	4/8/2005	4/24/2005	3/11/2005

Table A-3. CON mare expected foaling dates, actual foaling dates and dates started on trial

Mare	Expected foaling date	Actual foaling date	Date on trial
A61	1/16/2005	1/24/2005	12/20/2004
B26	1/20/2005	1/28/2005	12/23/2004
B41	2/9/2005	3/1/2005	1/10/2005
C4	2/13/2005	3/6/2005	1/17/2005
A54	2/27/2005	3/3/2005	1/31/2005
B13	3/3/2005	3/2/2005	2/2/2005
B43	3/5/2005	3/24/2005	2/2/2005
B18	3/8/2005	3/19/2005	2/7/2005
B29	3/9/2005	3/22/2005	2/10/2005
A64	3/18/2005	3/26/2005	2/18/2005
B45	3/24/2005	4/5/2005	2/24/2005
B36	4/9/2005	4/19/2005	3/11/2005

Fatty Acid Composition of Monthly Pasture Samples

Table A-4. Fatty acid composition of bahiagrass pasture (by month) and Coastal bermudagrass hay

	Pasture						
Fatty acid[1]	Dec.	Jan.	Feb.	March	April	May	June
C8:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C10:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C12:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C14:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C16:0	22.38	23.35	20.11	22.42	22.53	22.59	24.53
C16:1	0.00	1.70	0.00	0.00	0.00	0.00	0.00
C17:0	0.99	1.02	0.84	0.86	0.70	0.00	0.90
C17:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C18:0	3.94	5.47	5.60	4.87	3.05	3.26	3.77
C18:1	2.64	5.54	4.57	4.07	1.37	1.42	1.37
C18:2n-6	18.17	25.72	26.52	24.43	21.07	15.98	17.33
C18:3n-3	50.27	34.42	40.13	41.25	50.55	55.92	50.94
C20:4n-6	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C20:5n-3	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C22:5 n-3	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C22:6n-3	0.00	0.00	0.00	0.00	0.00	0.00	0.00
Total n-6[2]	18.17	25.72	26.52	24.43	21.07	15.98	17.33
Total n-3[3]	50.27	34.42	40.13	41.25	50.55	55.92	50.94
n-6:n-3	0.36	0.75	0.66	0.59	0.42	0.29	0.34

[1] Presented as g fatty acid per 100 g fat.
[2] Calculated as C18:2 + C20:4.
[3] Calculated as C18:3 + C20:5 + C22:5 + C22:6.

Mare Fatty Acid Intake

Table A-5. Mare daily intake of forage, grain and supplement by month[1]

	Forage[2]			Grain			Supplement	
Month	CON	FLAX	FISH	CON	FLAX	FISH	FLAX	FISH
Dec.	6.57 ± 0.65	5.71 ± 0.16	6.21 ± 0.05	6.57 ± 0.65	5.71 ± 0.16	6.21 ± 0.05	0.16 ± 0.00	0.55 ± 0.01
Jan.	6.00 ± 0.24	6.29 ± 0.18	6.45 ± 0.22	6.00 ± 0.24	6.29 ± 0.18	6.45 ± 0.22	0.17 ± 0.01	0.57 ± 0.01
Feb.	6.14 ± 0.16	6.05 ± 0.13	6.09 ± 0.13	6.55 ± 0.28	6.09 ± 0.26	6.52 ± 0.13	0.18 ± 0.01	0.57 ± 0.01
March	5.70 ± 0.22	5.57 ± 0.29	5.59 ± 0.15	6.70 ± 0.35	6.85 ± 0.45	7.14 ± 0.35	0.17 ± 0.01	0.54 ± 0.01
April	5.47 ± 0.14	5.64 ± 0.09	5.53 ± 0.10	7.83 ± 0.26	8.17 ± 0.28	8.08 ± 0.19	0.16 ± 0.01	0.53 ± 0.01
May	5.71 ± 0.14	5.62 ± 0.08	5.56 ± 0.13	8.57 ± 0.21	8.34 ± 0.13	8.43 ± 0.20	0.16 ± 0.01	0.53 ± 0.01
June	5.25 ± 0.30	5.60 ± 0.12	5.50 ± 0.11	7.87 ± 0.43	8.41 ± 0.18	8.23 ± 0.16	0.16 ± 0.01	0.53 ± 0.01

[1] Presented in kg as means ± SEM.
[2] Estimated at 1% of mare BW.

Mare Bodyweight

Table A-6. FISH mare bodyweights[1]

	Time								
Mare	d-28	d-14	d0	d+14	d+28	d+42	d+56	d+70	d+84
B46	1401	1408	1246	1251	1259	1262	1287	NT[2]	1272
W69	1331	1343	1172	1222	1224	1215	1215	NT	1211
B33	1314	1326	1130	1176	1172	1174	1180	NT	1187
B14	1539	1572	1346	1389	1398	1436	1425	NT	1396
B01	1430	1428	1245	1287	1306	NT	1305	NT	1282
B31	1302	1306	1133	1089	1106	1121	1106	1072	1075
A59	1530	1551	1341	1219	1261	1290	1275	1290	1316
B47	1366	1364	1156	1203	1198	1191	1209	1136	1234
B35	1363	1378	Off trial – Dead foal (dystocia)						
B24	1412	1401	1280	1228	1200	Off trial –Foal euthanized			
B23	1395	1420	Off trial – Dead foal (dystocia)						
A66	1425	1412	1267	1256	1265	1287	1282	1302	1253

[1] Presented in lb.
[2] NT = not taken.

Table A-7. FLAX mare bodyweights[1]

	Time								
Mare	d-28	d-14	d0	d+14	d+28	d+42	d+56	d+70	d+84
B6	1246	1250	1122	1118	1135	1133	1132	NT[2]	1096
B19	1266	1267	1140	1127	1158	1147	1136	NT	1111
A65	1487	1524	1393	1379	1375	1391	1377	NT	1374
B44	1343	1383	1221	1241	1244	1242	NT	NT	1219
C1	1286	1329	NT	1081	1082	1142	1124	1136	1144
A62	1537	1554	1330	1376	1382	1378	1394	1405	1396
B21	1396	1387	1213	1226	1216	NT	1204	1147	1215
C2	1296	1328	1110	1175	1177	NT	1165	1138	1227
B28	1444	1445	1306	1269	1253	1266	1260	1192	1246
C6	1284	1294	1077	1082	1072	1083	1070	1105	1113
B32	1438	1447	1204	1292	1248	1217	1196	1260	1243
B30	1337	1346	Off trial – Dead foal (foal suffocated)						

[1] Presented in lb.
[2] NT = not taken.

Table A-8. CON mare bodyweights[1]

	Time								
Mare	d-28	d-14	d0	d+14	d+28	d+42	d+56	d+70	d+84
A61	1588	1579	1425	1419	1445	1436	1426	NT[2]	1438
B26	1301	1324	1124	1077	1084	1138	1103	NT	1135
B41	1338	1374	1211	1246	1255	NT	1266	1253	1250
C4	1258	1296	1103	1117	1107	1127	1115	1104	1122
A54	1135	1143	968	979	980	982	993	977	978
B13	1347	1360	1169	1152	1094	1160	1148	1126	1147
B43	1290	1267	Off trial – Dead foal (dystocia)						
B18	1435	1404	1214	1217	1241	1214	1231	1242	1225
B29	1485	1531	1378	1343	1378	1338	1355	1368	1370
A64	1422	1410	NT	1185	1225	NT	1275	NT	1260
B45	1449	1451	1285	1297	1289	1291	1312	1328	1278
B36	1583	1548	1376	1366	1374	NT	1386	1286	NT

[1] Presented in lb.
[2] NT = not taken.

Foal Bodyweight

Table A-9. FISH foal bodyweights[1]

		Time						
Foal	Sex[2]	d0	d+14	d+28	d+42	d+56	d+70	d+84
5W69	F	120	163	205	245	284	NT[3]	364
5B33	F	123	161	203	240	287	NT	364
5B14	F	123	179	200	271	316	NT	396
5B46	C	118	136	209	256	300	NT	373
5B01	F	100	133	165	NT	242	NT	305
5A59	C	141	198	238	292	337	360	386
5B31	F	109	157	200	240	236	284	322
5B47	F	130	173	214	250	287	292	342
5B24	C	123	170	204	Foal euthanized (joint ill)			
5A66	F	118	156	219	246	284	312	360
5B35	Foal died at birth (dystocia)							
5B23	Foal died at birth (dystocia)							

[1] Presented in lb.
[2] C = colt, F = filly.
[3] NT = not taken.

Table A-10. FLAX foal bodyweights[1]

		Time						
Foal	Sex	d0	d+14	d+28	d+42	d+56	d+70	d+84
5B06	F	108	162	207	250	291	NT	355
5B19	C	127	159	195	239	278	NT	353
5A65	C	122	177	225	272	324	NT	398
5B44	C	110	153	197	271	NT	NT	312
5B28	C	112	161	208	238	276	306	327
5C1	C	NT	140	169	200	236	267	298
5C6	C	116	173	203	252	252	292	323
5A62	F	130	184	239	306	330	372	413
5B21	C	119	167	209	NT	272	283	331
5C2	F	113	138	199	NT	260	297	347
5B32	F	136	187	233	265	266	316	354
5B30	Foal died at birth (suffocated)							

[1] Presented in lb.
[2] C = colt, F = filly.
[3] NT = not taken.

Table A-11. CON foal bodyweights[1]

		Time						
Foal	Sex[2]	d0	d+14	d+28	d+42	d+56	d+70	d+84
5A61	F	124	198	247	287	334	NT[3]	444
5B26	F	110	183	195	229	262	NT	363
5B41	C	121	161	197	NT	275	314	326
5B13	F	126	177	220	262	307	335	360
5A54	C	106	149	190	237	275	322	340
5C4	F	123	160	176	212	230	264	289
5B18	F	114	150	198	244	278	303	328
5B29	F	115	167	215	249	284	323	365
5A64	F	NT	145	210	NT	272	NT	354
5B45	F	120	163	202	234	264	304	334
5B36	F	123	188	222	NT	296	350	NT
5B43	Foal died at birth (dystocia)							

[1] Presented in lb.
[2] C = colt, F = filly.
[3] NT = not taken.

Mare Serum IgG

Table A-12. Serum IgG content of FISH mares at foaling

Mare	IgG[1]
A59	1879.0
A66	3000.4
B01	2007.2
B14	1879.0
B24	3323.7
B31	3159.4
B33	1879.0
B46	1509.5
B47	2007.2
W69	2138.3

[1] Presented as mg/dL.

Table A-13. Serum IgG content of FLAX mares at foaling

Mare	IgG[1]
A62	2410.1
A65	3159.4
B06	2007.2
B19	2846.3
B21	2696.7
B28	2272.5
B32	1390.5
B44	1509.5
C2	1753.5
C6	1753.5

[1] Presented as mg/dL.

Table A-14. Serum IgG content of CON mares at foaling

Mare	IgG[1]
A54	1630.4
A61	1753.5
B13	2551.5
B18	1273.2
B26	2551.5
B29	2846.3
B36	1753.5
B41	2846.3
C4	2551.5

[1] Presented as mg/dL.

Mare Colostrum and Milk IgG

Table A-15. IgG content of colostrum and milk from FISH mares[1]

	Time					
Mare	d0	36h	d+7	d+28	d+56	d+84
A59	7014.10	183.40	102.20	112.00	84.31	61.36
A66	21447.20	279.10	133.30	84.31	NT[2]	84.31
B01	8028.80	183.40	157.10	102.20	93.00	93.00
B14	8553.10	157.10	133.30	93.00	102.20	93.00
B24	13294.90	169.90	144.90	112.00	NT	NT
B31	21447.20	279.10	144.90	122.40	112.00	76.15
B33	18634.70	228.00	157.10	112.00	112.00	84.31
B46	14679.00	169.90	133.30	102.20	102.20	93.00
B47	12001.60	279.10	157.10	112.00	102.20	68.51
W69	10787.00	197.60	133.30	102.20	NT	68.51

[1] Presented as mg/dL.
[2] NT = not taken.

Table A-16. IgG content of colostrum and milk from FLAX mares[1]

	Time					
Mare	d0	36h	d+7	d+28	d+56	d+84
A62	16956.50	197.60	133.30	133.30	93.00	54.69
A65	16513.80	279.10	144.90	112.00	93.00	122.40
B06	14679.00	197.60	157.10	122.40	102.20	68.51
B19	14679.00	503.70	228.00	133.30	112.00	102.20
B21	15409.30	197.60	133.30	112.00	93.00	84.31
B28	25286.90	261.30	144.90	112.00	93.00	84.31
B32	15409.30	228.00	133.30	102.20	93.00	68.51
B44	16956.50	133.30	133.30	112.00	93.00	68.51
C2	10205.90	157.10	144.90	112.00	93.00	68.51
C6	10205.90	212.40	144.90	122.40	84.31	76.15

[1] Presented as mg/dL.

Table A-17. IgG content of colostrum and milk from CON mares[1]

	Time					
Mare	d0	36h	d+7	d+28	d+56	d+84
A54	17778.00	197.60	133.30	102.20	84.31	84.31
A61	10205.90	197.60	157.10	112.00	102.20	76.15
A64	12001.60	279.10	169.90	NT[2]	84.31	61.36
B13	12001.60	228.00	122.40	112.00	93.00	84.31
B18	18634.70	169.90	133.30	102.20	84.31	48.49
B26	10787.00	157.10	144.90	93.00	102.20	84.31
B29	26505.70	244.30	157.10	112.00	93.00	48.49
B36	18634.70	183.40	122.40	102.20	NT	61.36
B41	12001.60	228.00	144.90	122.40	102.20	102.20
B45	19529.60	183.40	144.90	112.00	102.20	61.36
C4	14679.00	261.30	144.90	112.00	102.20	84.31

[1] Presented as mg/dL.
[2] NT = not taken.

Foal Serum IgG

Table A-18. IgG content of serum from FISH foals[1]

	Time					
Foal	d0	36h	d+7	d+28	d+56	d+84
5A59	122.40	1157.20	1630.40	963.10	789.90	695.30
5A66	76.15	4658.70	3669.70	2138.30	815.20	1275.70
5B01	84.31	469.80	407.60	289.30	318.30	318.30
5B14	54.69	1630.40	1509.50	1111.10	637.90	534.60
5B24	84.31	2410.10	2007.20	1273.20	NT[2]	NT
5B31	61.36	3493.70	2846.30	1753.50	1273.20	939.50
5B33	54.69	2007.20	2007.20	1059.80	750.10	568.10
5B46	102.20	2410.10	1879.00	1273.20	711.60	637.90
5B47	133.30	2138.30	2007.20	1157.20	917.40	695.30
5W69	93.00	1753.50	1509.50	963.10	232.00	830.90

[1] Presented as mg/dL.
[2] NT = not taken.

Table A-19. IgG content of serum from FLAX foals[1]

	Time					
Foal	d0	36h	d+7	d+28	d+56	d+84
5A62	122.40	3159.40	2551.50	2138.30	1273.20	815.20
5A65	84.31	2846.30	3000.40	1630.40	1273.20	928.10
5B06	68.51	3852.30	3669.70	1879.00	NT[2]	928.10
5B19	68.51	2846.30	2551.50	1509.50	1273.20	789.90
5B21	61.36	2007.20	1753.50	963.10	674.20	464.10
5B28	84.31	3852.30	3323.70	1879.00	1509.50	1069.10
5B32	102.20	2846.30	2272.50	1390.50	1069.10	695.30
5B44	93.00	1390.50	1630.40	1059.80	789.90	711.60
5C2	68.51	1220.60	534.60	637.90	963.10	521.50
5C6	76.15	2007.20	2007.20	1157.20	750.10	636.60

[1] Presented as mg/dL.
[2] NT = not taken.

Table A-20. IgG content of serum from CON foals[1]

	Time					
Foal	d0	36h	d+7	d+28	d+56	d+84
5A54	76.15	2551.50	2007.20	1157.20	830.90	876.80
5A61	76.15	1753.50	1753.50	2138.30	674.20	637.90
5A64	NT[2]	NT	1273.20	1273.20	830.90	939.50
5B13	61.36	2846.30	2410.10	1273.20	830.90	578.60
5B18	102.20	3000.40	2551.50	1879.00	928.10	876.80
5B26	68.51	2846.30	2551.50	3000.40	711.60	602.50
5B29	68.51	3323.70	NT	1879.00	1273.20	695.30
5B36	61.36	2410.10	1879.00	1273.20	1500.20	636.60
5B41	122.40	2272.50	2272.50	1273.20	1042.30	815.20
5B45	76.15	2551.50	2272.50	1509.50	1003.60	636.60
5C4	93.00	3159.40	2551.50	1273.20	928.10	1069.10

[1] Presented as mg/dL.
[2] NT = not taken.

Fatty Acid Composition of Plasma from FISH Mares

Table A-21. Fatty acid composition of FISH mare plasma at 28 d prior to expected foaling date

	Mare									
FA[1]	B47	W69	A66	B31	A59	B33	B24	B46	B14	B01
C8:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C10:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C12:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C14:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C14:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C16:0	15.50	15.35	15.08	16.99	17.03	15.24	16.18	15.73	13.46	15.97
C16:1	1.60	1.15	1.69	1.68	1.49	1.11	1.44	1.16	1.24	1.18
C17:0	0.66	0.74	0.56	0.00	0.64	0.89	0.00	0.72	0.68	0.95
C17:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C18:0	18.05	19.34	18.20	18.49	16.66	19.55	19.99	19.26	21.83	19.36
C18:1	10.50	12.15	10.41	12.05	12.04	10.47	10.91	10.21	10.57	12.38
C18:2	46.29	45.89	47.98	44.65	47.59	46.44	45.64	48.07	47.39	43.55
C20:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C18:3	5.12	2.70	3.73	3.07	1.58	3.36	3.86	2.48	1.89	4.17
C20:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C20:2	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C22:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C20:3	0.71	0.81	0.73	1.03	0.89	0.78	0.00	0.61	0.73	0.83
C20:4	1.57	1.88	1.63	2.04	2.09	2.17	1.98	1.77	2.21	1.61
C24:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C20:5	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C22:5	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C22:6	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00

[1] FA = fatty acid, presented as g FA per 100 g fat.

Table A-22. Fatty acid composition of FISH mare plasma at foaling

	Mare									
FA[1]	B47	W69	A66	B31	A59	B33	B24	B46	B14	B01
C8:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C10:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C12:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C14:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C14:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C16:0	18.27	15.40	15.24	18.86	23.46	17.04	16.49	17.46	15.99	16.58
C16:1	1.62	0.96	1.30	1.72	2.53	1.01	1.28	1.33	1.63	1.46
C17:0	0.64	0.66	0.58	0.77	0.00	0.79	0.73	0.61	0.66	0.75
C17:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C18:0	17.63	21.76	23.42	19.45	21.90	20.46	21.44	19.97	20.89	19.53
C18:1	11.94	9.61	9.71	11.69	18.64	9.07	9.68	9.13	10.77	8.79
C18:2	43.15	47.32	44.72	41.12	18.41	46.22	45.17	46.92	43.62	43.03
C20:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C18:3	4.37	1.30	2.64	2.49	0.00	2.35	2.42	1.50	1.59	2.49
C20:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C20:2	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C22:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C20:3	0.75	0.79	0.74	0.82	1.03	0.66	0.73	0.57	0.69	0.72
C20:4	1.63	2.21	1.66	1.94	10.00	2.41	2.06	1.93	1.90	2.11
C24:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C20:5	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.66	2.47
C22:5	0.00	0.00	0.00	0.00	0.78	0.00	0.00	0.00	0.00	0.00
C22:6	0.00	0.00	0.00	1.14	2.23	0.00	0.00	0.58	1.13	2.07

[1] FA = fatty acid, presented as g FA per 100 g fat.

Table A-23. Fatty acid composition of FISH mare plasma at 28 d post-foaling

	Mare									
FA[1]	B47	W69	A66	B31	A59	B33	B24	B46	B14	B01
C8:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C10:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C12:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C14:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C14:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C16:0	16.42	16.55	15.90	16.69	18.17	17.23	16.35	17.91	14.34	16.35
C16:1	0.00	0.65	1.17	1.47	1.25	1.18	1.16	2.12	0.00	1.21
C17:0	0.91	0.81	0.75	0.73	0.91	1.06	0.73	0.62	0.00	0.89
C17:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C18:0	22.27	21.43	21.27	20.49	19.15	20.03	19.98	19.33	23.61	19.56
C18:1	7.69	8.36	7.82	10.11	9.82	9.75	7.63	10.82	7.02	8.38
C18:2	44.60	46.15	44.66	43.32	44.52	42.63	46.92	41.36	48.21	43.29
C20:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.57
C18:3	3.57	1.14	4.15	3.10	3.21	2.33	3.01	3.83	3.76	4.58
C20:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C20:2	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C22:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C20:3	0.90	0.96	0.83	0.98	0.93	1.01	0.75	0.66	0.00	0.85
C20:4	1.33	1.39	1.40	1.28	1.18	1.65	1.68	1.47	1.65	1.30
C24:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C20:5	1.25	1.39	1.12	0.85	0.00	1.63	0.92	0.88	1.41	1.90
C22:5	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C22:6	1.06	1.17	0.93	0.98	0.86	1.51	0.86	1.00	0.00	1.11

[1] FA = fatty acid, presented as g FA per 100 g fat.

Table A-24. Fatty acid composition of FISH mare plasma at 56 d post-foaling

	Mare									
FA[1]	B47	W69	A66	B31	A59	B33	B24	B46	B14	B01
C8:0	0.00	NA[2]	0.00	0.00	0.00	NA	OT[3]	0.00	NA	0.00
C10:0	0.00	NA	0.00	0.00	0.00	NA	OT	0.00	NA	0.00
C12:0	0.00	NA	0.00	0.00	0.00	NA	OT	0.00	NA	0.00
C14:0	0.00	NA	0.00	0.00	0.00	NA	OT	0.00	NA	0.00
C14:1	0.00	NA	0.00	0.00	0.00	NA	OT	0.00	NA	0.00
C16:0	15.67	NA	15.32	16.19	16.68	NA	OT	16.72	NA	16.18
C16:1	1.04	NA	0.71	1.43	0.86	NA	OT	0.00	NA	0.00
C17:0	0.93	NA	0.82	0.68	0.82	NA	OT	0.83	NA	1.03
C17:1	0.00	NA	0.00	0.00	0.00	NA	OT	0.00	NA	0.00
C18:0	19.97	NA	20.40	21.00	20.72	NA	OT	21.99	NA	22.14
C18:1	9.09	NA	7.59	10.03	8.19	NA	OT	8.34	NA	7.91
C18:2	43.73	NA	46.87	44.42	47.25	NA	OT	46.40	NA	44.60
C20:0	0.00	NA	0.00	0.00	0.00	NA	OT	0.00	NA	0.00
C18:3	5.72	NA	3.98	3.96	3.36	NA	OT	1.09	NA	2.20
C20:1	0.00	NA	0.00	0.00	0.00	NA	OT	0.00	NA	0.00
C20:2	0.00	NA	0.00	0.00	0.00	NA	OT	0.00	NA	0.00
C22:0	0.00	NA	0.00	0.00	0.00	NA	OT	0.00	NA	0.00
C20:3	0.95	NA	0.95	0.99	0.86	NA	OT	0.91	NA	0.99
C20:4	1.19	NA	1.37	1.29	1.27	NA	OT	1.70	NA	1.49
C24:1	0.00	NA	0.00	0.00	0.00	NA	OT	0.00	NA	0.00
C20:5	0.89	NA	1.06	0.00	0.00	NA	OT	0.97	NA	2.04
C22:5	0.00	NA	0.00	0.00	0.00	NA	OT	0.00	NA	0.00
C22:6	0.84	NA	0.92	0.00	0.00	NA	OT	1.05	NA	1.43

[1] FA = fatty acid, presented as g FA per 100 g fat.
[2] NA = not analyzed.
[3] OT = off trial.

Table A-25. Fatty acid composition of FISH mare plasma at 84 d post-foaling

	Mare									
FA[1]	B47	W69	A66	B31	A59	B33	B24	B46	B14	B01
C8:0	0.00	0.00	0.00	0.00	0.00	0.00	OT[2]	0.00	0.00	0.00
C10:0	0.00	0.00	0.00	0.00	0.00	0.00	OT	0.00	0.00	0.00
C12:0	0.00	0.00	0.00	0.00	0.00	0.00	OT	0.00	0.00	0.00
C14:0	0.00	0.00	0.00	0.00	0.00	0.00	OT	0.00	0.00	0.00
C14:1	0.00	0.00	0.00	0.00	0.00	0.00	OT	0.00	0.00	0.00
C16:0	16.00	15.13	16.20	16.41	25.51	16.25	OT	16.20	13.80	14.83
C16:1	0.68	0.81	1.45	1.66	0.00	0.82	OT	1.24	1.01	0.95
C17:0	0.98	0.61	0.68	0.87	1.45	1.00	OT	0.73	0.76	0.86
C17:1	0.00	0.00	0.00	0.00	0.00	0.00	OT	0.00	0.00	0.00
C18:0	19.37	19.24	18.57	19.66	31.98	19.23	OT	19.90	19.81	20.58
C18:1	8.85	9.06	10.02	10.29	12.51	7.23	OT	8.38	7.97	8.02
C18:2	47.04	49.92	46.36	44.89	19.05	48.24	OT	45.67	50.05	46.71
C20:0	0.00	0.00	0.00	0.00	0.00	0.00	OT	0.55	0.00	0.00
C18:3	4.02	3.10	4.95	2.76	6.20	2.97	OT	3.75	3.81	4.24
C20:1	0.00	0.00	0.00	0.00	0.00	0.00	OT	0.00	0.00	0.00
C20:2	0.00	0.00	0.00	0.00	0.00	0.00	OT	0.00	0.00	0.00
C22:0	0.00	0.00	0.00	0.00	0.00	0.00	OT	0.00	0.00	0.00
C20:3	1.04	0.86	0.78	1.18	1.29	0.75	OT	0.73	0.73	0.89
C20:4	0.81	1.27	0.99	1.38	2.00	1.67	OT	1.53	1.34	1.32
C24:1	0.00	0.00	0.00	0.00	0.00	0.00	OT	0.00	0.00	0.00
C20:5	0.00	0.00	0.00	0.00	0.00	0.78	OT	0.56	0.72	0.76
C22:5	0.00	0.00	0.00	0.00	0.00	0.00	OT	0.00	0.00	0.00
C22:6	1.20	0.00	0.00	0.91	0.00	1.05	OT	0.77	0.00	0.84

[1] FA = fatty acid, presented as g FA per 100 g fat.
[2] OT = off trial.

Fatty Acid Composition of Plasma from FLAX Mares

Table A-26. Fatty acid composition of FLAX mare plasma at 28 d before expected foaling date

	Mare										
FA[1]	B21	A62	B32	C2	C6	A65	B06	C1	B44	B28	B19
C8:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C10:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C12:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C14:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C14:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C16:0	15.71	16.43	18.05	17.09	17.91	16.07	17.82	14.90	14.18	16.37	14.62
C16:1	0.75	1.84	1.77	0.00	0.81	1.50	0.73	1.58	0.00	1.29	1.34
C17:0	0.66	0.60	0.59	0.77	0.82	0.62	1.01	0.66	0.00	0.88	0.64
C17:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C18:0	20.11	19.09	18.17	19.16	16.25	18.56	18.87	20.55	24.52	18.20	18.98
C18:1	10.45	10.86	12.72	11.36	12.25	10.95	10.87	11.60	10.78	10.99	11.28
C18:2	47.88	42.96	43.44	47.14	46.48	44.91	45.38	43.84	47.43	46.51	49.45
C20:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C18:3	2.01	5.77	2.66	1.43	2.84	5.13	2.92	4.31	3.09	2.18	1.59
C20:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C20:2	0.00	0.00	0.37	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C22:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C20:3	0.77	0.69	0.53	0.91	0.80	0.74	0.88	0.71	0.00	0.88	0.61
C20:4	1.66	1.77	1.70	2.14	1.84	1.54	1.52	1.86	0.00	2.69	1.47
C24:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C20:5	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C22:5	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C22:6	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00

[1] FA = fatty acid, presented as g FA per 100 g fat.

Table A-27. Fatty acid composition of FLAX mare plasma at foaling

	Mare										
FA[1]	B21	A62	B32	C2	C6	A65	B06	C1	B44	B28	B19
C8:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA[2]	0.00	0.00	0.00
C10:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA	0.00	0.00	0.00
C12:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA	0.00	0.00	0.00
C14:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA	0.00	0.00	0.00
C14:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA	0.00	0.00	0.00
C16:0	15.06	17.83	17.98	16.68	17.00	17.07	17.88	NA	14.24	16.93	13.94
C16:1	1.39	1.95	1.61	1.04	1.08	1.72	1.14	NA	0.68	0.00	0.87
C17:0	0.63	0.71	0.60	0.58	0.73	0.61	0.81	NA	0.89	0.00	0.00
C17:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA	0.00	0.00	0.00
C18:0	23.28	19.07	18.05	19.16	19.52	18.47	19.04	NA	20.78	20.50	23.12
C18:1	9.25	11.37	12.84	11.01	9.46	10.63	9.86	NA	10.19	10.49	9.68
C18:2	44.93	40.42	43.04	45.96	46.76	43.52	46.16	NA	46.90	49.27	49.55
C20:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA	0.00	0.00	0.00
C18:3	3.01	6.55	3.70	2.91	2.89	6.04	2.65	NA	4.00	2.81	1.13
C20:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA	0.00	0.00	0.00
C20:2	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA	0.00	0.00	0.00
C22:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA	0.00	0.00	0.00
C20:3	0.72	0.67	0.66	0.73	0.71	0.59	0.80	NA	0.75	0.00	0.00
C20:4	1.73	1.45	1.52	1.92	1.86	1.35	1.65	NA	1.57	0.00	1.72
C24:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA	0.00	0.00	0.00
C20:5	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA	0.00	0.00	0.00
C22:5	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA	0.00	0.00	0.00
C22:6	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA	0.00	0.00	0.00

[1] FA = fatty acid, presented as g FA per 100 g fat.

[2] NA = not analyzed.

Table A-28. Fatty acid composition of FLAX mare plasma at 28 d post-foaling

	Mare										
FA[1]	B21	A62	B32	C2	C6	A65	B06	C1	B44	B28	B19
C8:0	0.00	0.00	0.00	0.00	0.00	NA	0.00	0.00	NA	0.00	0.00
C10:0	0.00	0.00	0.00	0.00	0.00	NA	0.00	0.00	NA	0.00	0.00
C12:0	0.00	0.00	0.00	0.00	0.00	NA	0.00	0.00	NA	0.00	0.00
C14:0	0.00	0.00	0.00	0.00	0.00	NA	0.00	0.00	NA	0.00	0.00
C14:1	0.00	0.00	0.00	0.00	0.00	NA	0.00	0.00	NA	0.00	0.00
C16:0	15.35	16.57	16.26	16.63	18.02	NA	16.33	14.46	NA	16.53	15.42
C16:1	0.60	0.87	0.99	0.00	0.00	NA	0.97	0.87	NA	0.91	1.08
C17:0	0.73	0.77	0.72	0.81	0.83	NA	0.86	0.77	NA	0.90	0.74
C17:1	0.00	0.00	0.00	0.00	0.00	NA	0.00	0.00	NA	0.00	0.00
C18:0	23.28	22.34	19.95	20.32	18.98	NA	18.82	22.35	NA	22.30	21.16
C18:1	7.21	7.74	9.73	8.46	9.10	NA	8.49	9.95	NA	7.77	9.97
C18:2	47.28	45.94	46.73	48.78	49.25	NA	47.13	46.57	NA	45.39	45.55
C20:0	0.57	0.00	0.00	0.00	0.00	NA	0.00	0.00	NA	0.00	0.61
C18:3	3.25	3.59	3.81	3.05	2.74	NA	5.41	2.69	NA	1.68	3.48
C20:1	0.00	0.00	0.00	0.00	0.00	NA	0.00	0.00	NA	0.00	0.00
C20:2	0.00	0.00	0.00	0.00	0.00	NA	0.00	0.00	NA	0.00	0.00
C22:0	0.00	0.00	0.00	0.00	0.00	NA	0.00	0.00	NA	0.00	0.00
C20:3	0.71	0.88	0.68	0.82	0.00	NA	0.77	0.91	NA	0.85	0.81
C20:4	1.01	1.30	1.13	1.13	1.08	NA	1.21	1.43	NA	1.73	1.18
C24:1	0.00	0.00	0.00	0.00	0.00	NA	0.00	0.00	NA	0.00	0.00
C20:5	0.00	0.00	0.00	0.00	0.00	NA	0.00	0.00	NA	0.96	0.00
C22:5	0.00	0.00	0.00	0.00	0.00	NA	0.00	0.00	NA	0.00	0.00
C22:6	0.00	0.00	0.00	0.00	0.00	NA	0.00	0.00	NA	0.98	0.00

[1] FA = fatty acid, presented as g FA per 100 g fat.
[2] NA = not analyzed.

Table A-29. Fatty acid composition of FLAX mare plasma at 56 d post-foaling

	Mare										
FA[1]	B21	A62	B32	C2	C6	A65	B06	C1	B44	B28	B19
C8:0	NA	0.00	0.00	0.00	0.00	NA	NA	0.00	0.00	0.00	0.00
C10:0	NA	0.00	0.00	0.00	0.00	NA	NA	0.00	0.00	0.00	0.00
C12:0	NA	0.00	0.00	0.00	0.00	NA	NA	0.00	0.00	0.00	0.00
C14:0	NA	0.00	0.00	0.00	0.00	NA	NA	0.00	0.00	0.00	0.00
C14:1	NA	0.00	0.00	0.00	0.00	NA	NA	0.00	0.00	0.00	0.00
C16:0	NA	16.13	15.79	15.68	17.99	NA	NA	14.09	14.08	15.71	15.18
C16:1	NA	0.87	1.04	0.95	0.79	NA	NA	0.00	0.00	1.23	1.13
C17:0	NA	0.82	0.60	0.83	0.82	NA	NA	0.96	0.90	0.78	0.73
C17:1	NA	0.00	0.00	0.00	0.00	NA	NA	0.00	0.00	0.00	0.00
C18:0	NA	19.82	21.04	19.71	17.71	NA	NA	21.59	23.21	21.51	22.45
C18:1	NA	8.36	9.63	9.78	10.73	NA	NA	8.02	9.12	8.57	8.76
C18:2	NA	46.82	47.68	47.10	46.65	NA	NA	48.33	47.37	47.58	45.88
C20:0	NA	0.00	0.00	0.00	0.00	NA	NA	0.59	0.00	0.00	0.59
C18:3	NA	5.42	2.19	3.90	3.29	NA	NA	4.39	3.31	2.43	3.67
C20:1	NA	0.00	0.00	0.00	0.00	NA	NA	0.00	0.00	0.00	0.00
C20:2	NA	0.00	0.00	0.00	0.00	NA	NA	0.00	0.00	0.00	0.00
C22:0	NA	0.00	0.00	0.00	0.00	NA	NA	0.00	0.00	0.00	0.00
C20:3	NA	0.72	0.81	0.87	0.87	NA	NA	0.82	0.76	0.77	0.64
C20:4	NA	1.03	1.23	1.19	1.16	NA	NA	1.21	1.26	1.42	0.97
C24:1	NA	0.00	0.00	0.00	0.00	NA	NA	0.00	0.00	0.00	0.00
C20:5	NA	0.00	0.00	0.00	0.00	NA	NA	0.00	0.00	0.00	0.00
C22:5	NA	0.00	0.00	0.00	0.00	NA	NA	0.00	0.00	0.00	0.00
C22:6	NA	0.00	0.00	0.00	0.00	NA	NA	0.00	0.00	0.00	0.00

[1] FA = fatty acid, presented as g FA per 100 g fat.

[2] NA = not analyzed.

Table A-30. Fatty acid composition of FLAX mare plasma at 84 d post-foaling

	Mare										
FA[1]	B21	A62	B32	C2	C6	A65	B06	C1	B44	B28	B19
C8:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C10:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C12:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C14:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C14:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C16:0	14.76	16.65	17.68	15.63	17.48	14.09	16.42	14.82	13.59	15.80	14.58
C16:1	0.84	1.49	1.22	0.51	0.90	1.01	0.58	0.56	0.00	0.59	0.68
C17:0	0.73	0.83	0.72	0.85	0.88	0.65	0.98	1.01	0.99	0.98	0.71
C17:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C18:0	20.30	18.11	18.84	19.48	18.21	18.60	19.24	20.63	22.96	18.57	20.07
C18:1	7.75	10.44	11.56	9.69	9.93	7.65	8.60	10.18	8.45	8.69	9.42
C18:2	48.41	45.19	44.51	48.45	47.61	50.46	47.13	46.26	48.55	48.63	48.75
C20:0	0.58	0.00	0.00	0.00	0.00	0.00	0.00	0.57	0.00	0.44	0.00
C18:3	4.89	5.37	3.66	3.47	3.02	5.65	4.62	3.89	3.13	3.42	3.67
C20:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C20:2	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C22:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C20:3	0.68	0.84	0.87	0.77	0.84	0.82	0.95	0.78	0.82	0.63	0.92
C20:4	1.06	1.08	0.95	1.16	1.13	1.07	1.47	1.30	1.50	1.25	1.20
C24:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C20:5	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.45	0.00
C22:5	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C22:6	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.55	0.00

[1] FA = fatty acid, presented as g FA per 100 g fat.

[2] NA = not analyzed.

Fatty Acid Composition of Plasma from CON Mares

Table A-31. Fatty acid composition of CON mare plasma at 28 d before expected foaling date

	Mare										
FA[1]	B29	B36	B13	B41	B45	A54	C4	A64	B18	B26	A61
C8:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA[2]	NA
C10:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA	NA
C12:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA	NA
C14:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA	NA
C14:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA	NA
C16:0	14.71	14.82	16.35	16.24	15.86	17.72	15.11	15.14	16.36	NA	NA
C16:1	0.89	0.00	1.06	1.63	1.29	1.92	1.11	1.33	0.00	NA	NA
C17:0	0.81	0.70	0.82	0.76	0.80	0.63	0.71	0.63	0.92	NA	NA
C17:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA	NA
C18:0	17.80	22.49	18.58	19.52	20.60	16.84	19.74	18.51	19.48	NA	NA
C18:1	10.79	9.43	12.79	12.24	10.78	11.28	11.51	9.97	10.82	NA	NA
C18:2	48.76	48.69	44.62	44.04	45.08	44.01	44.47	50.02	47.68	NA	NA
C20:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA	NA
C18:3	2.94	1.10	2.83	3.21	2.92	5.40	5.29	1.82	2.63	NA	NA
C20:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA	NA
C20:2	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA	NA
C22:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA	NA
C20:3	0.89	0.83	0.84	0.79	0.77	0.69	0.70	0.77	0.00	NA	NA
C20:4	2.40	1.96	2.11	1.56	1.91	1.50	1.35	1.79	2.10	NA	NA
C24:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA	NA
C20:5	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA	NA
C22:5	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA	NA
C22:6	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA	NA

[1] FA = fatty acid, presented as g FA per 100 g fat.
[2] NA = not analyzed.

Table A-32. Fatty acid composition of CON mare plasma at foaling

	Mare										
FA[1]	B29	B36	B13	B41	B45	A54	C4	A64	B18	B26	A61
C8:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA	0.00	0.00	0.00
C10:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA	0.00	0.00	0.00
C12:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA	0.00	0.00	0.00
C14:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA	0.00	0.00	0.00
C14:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA	0.00	0.00	0.00
C16:0	16.70	15.97	16.34	15.90	15.26	18.95	22.80	NA	17.29	16.84	15.84
C16:1	0.00	1.19	1.92	0.85	1.03	2.19	3.82	NA	1.29	1.40	0.68
C17:0	0.00	0.70	0.52	0.73	0.89	0.44	0.00	NA	0.70	0.64	0.82
C17:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA	0.00	0.00	0.00
C18:0	19.13	22.53	19.33	21.75	22.07	17.05	20.33	NA	18.69	20.81	21.20
C18:1	12.26	10.51	10.71	9.06	10.55	10.88	27.20	NA	11.43	9.56	9.33
C18:2	46.22	43.44	45.84	48.84	45.76	44.62	15.27	NA	45.18	46.75	48.16
C20:0	0.00	0.00	0.00	0.00	0.00	0.00	1.14	NA	0.00	0.00	0.00
C18:3	3.82	3.16	3.22	1.07	1.87	4.02	0.00	NA	3.11	1.53	1.30
C20:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA	0.00	0.00	0.00
C20:2	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA	0.00	0.00	0.00
C22:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA	0.00	0.00	0.00
C20:3	0.00	0.77	0.57	0.00	0.70	0.58	0.73	NA	0.65	0.66	0.74
C20:4	1.87	1.73	1.55	1.80	1.88	1.28	6.13	NA	1.66	1.81	1.93
C24:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA	0.00	0.00	0.00
C20:5	0.00	0.00	0.00	0.00	0.00	0.00	0.77	NA	0.00	0.00	0.00
C22:5	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA	0.00	0.00	0.00
C22:6	0.00	0.00	0.00	0.00	0.00	0.00	1.83	NA	0.00	0.00	0.00

[1] FA = fatty acid, presented as g FA per 100 g fat.

[2] NA = not analyzed.

Table A-33. Fatty acid composition of CON mare plasma at 28 d post-foaling

	Mare										
FA[1]	B29	B36	B13	B41	B45	A54	C4	A64	B18	B26	A61
C8:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C10:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C12:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C14:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C14:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C16:0	15.68	17.09	15.11	15.84	16.76	16.97	15.25	14.53	17.87	15.88	13.94
C16:1	0.64	1.24	0.00	1.13	0.00	0.99	0.71	0.00	0.00	0.93	0.92
C17:0	0.96	0.00	0.71	0.70	0.00	0.73	0.83	0.00	0.91	0.68	0.90
C17:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C18:0	21.47	22.06	23.53	20.01	20.75	20.19	20.93	22.29	21.17	21.49	20.31
C18:1	9.53	10.82	8.88	8.93	9.22	8.43	9.53	8.30	9.10	8.51	9.98
C18:2	45.92	43.75	47.87	47.66	48.15	48.02	46.46	50.40	45.82	49.99	48.97
C20:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C18:3	3.70	3.66	1.93	3.76	3.64	2.68	4.38	2.29	2.89	0.00	2.63
C20:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C20:2	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C22:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C20:3	0.83	0.00	0.82	0.83	0.00	0.86	0.73	0.96	0.94	0.94	0.93
C20:4	1.26	1.37	1.16	1.15	1.48	1.12	1.19	1.24	1.30	1.59	1.42
C24:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C20:5	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C22:5	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C22:6	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00

[1] FA = fatty acid, presented as g FA per 100 g fat.

Table A-34. Fatty acid composition of CON mare plasma at 56 d post-foaling

	Mare										
FA[1]	B29	B36	B13	B41	B45	A54	C4	A64	B18	B26	A61
C8:0	0.00	0.00	0.00	NA[2]	0.00	0.00	0.00	0.00	0.00	NA	NA
C10:0	0.00	0.00	0.00	NA	0.00	0.00	0.00	0.00	0.00	NA	NA
C12:0	0.00	0.00	0.00	NA	0.00	0.00	0.00	0.00	0.00	NA	NA
C14:0	0.00	0.00	0.00	NA	0.00	0.00	0.00	0.00	0.00	NA	NA
C14:1	0.00	0.00	0.00	NA	0.00	0.00	0.00	0.00	0.00	NA	NA
C16:0	15.31	15.37	15.60	NA	17.18	17.25	15.95	14.48	17.07	NA	NA
C16:1	0.95	0.88	1.24	NA	0.00	1.12	0.94	0.00	0.59	NA	NA
C17:0	0.90	0.87	0.68	NA	1.03	0.68	0.95	0.60	0.96	NA	NA
C17:1	0.00	0.00	0.00	NA	0.00	0.00	0.00	0.00	0.00	NA	NA
C18:0	20.94	19.95	21.35	NA	19.09	18.65	20.78	20.90	19.23	NA	NA
C18:1	10.25	9.01	9.59	NA	9.59	8.47	10.93	7.81	9.19	NA	NA
C18:2	46.75	47.15	46.33	NA	46.61	47.33	44.82	51.96	45.89	NA	NA
C20:0	0.00	0.00	0.51	NA	0.00	0.60	0.00	0.00	0.00	NA	NA
C18:3	4.01	4.64	2.95	NA	3.91	4.44	3.70	2.17	4.99	NA	NA
C20:1	0.00	0.00	0.00	NA	0.00	0.00	0.00	0.00	0.00	NA	NA
C20:2	0.00	0.00	0.00	NA	0.00	0.00	0.00	0.00	0.00	NA	NA
C22:0	0.00	0.00	0.00	NA	0.00	0.00	0.00	0.00	0.00	NA	NA
C20:3	0.00	0.96	0.77	NA	0.98	0.68	0.77	0.79	0.86	NA	NA
C20:4	0.90	1.17	0.98	NA	1.60	0.77	1.15	1.29	1.20	NA	NA
C24:1	0.00	0.00	0.00	NA	0.00	0.00	0.00	0.00	0.00	NA	NA
C20:5	0.00	0.00	0.00	NA	0.00	0.00	0.00	0.00	0.00	NA	NA
C22:5	0.00	0.00	0.00	NA	0.00	0.00	0.00	0.00	0.00	NA	NA
C22:6	0.00	0.00	0.00	NA	0.00	0.00	0.00	0.00	0.00	NA	NA

[1] FA = fatty acid, presented as g FA per 100 g fat.

[2] NA = not analyzed.

Table A-35. Fatty acid composition of CON mare plasma at 84 d post-foaling

	Mare										
FA[1]	B29	B36	B13	B41	B45	A54	C4	A64	B18	B26	A61
C8:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C10:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C12:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C14:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C14:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C16:0	14.72	15.95	15.71	15.73	15.56	16.88	16.00	14.93	16.40	16.29	16.06
C16:1	0.89	0.76	0.70	0.00	0.84	1.13	0.57	0.67	0.81	0.93	0.69
C17:0	0.94	0.84	0.82	0.90	0.98	0.71	0.95	0.70	1.01	0.92	0.83
C17:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C18:0	17.09	20.43	19.95	18.91	18.08	18.25	19.52	20.80	18.25	18.92	19.59
C18:1	10.43	9.11	10.57	8.77	9.37	8.63	10.51	8.62	10.43	9.23	10.21
C18:2	49.57	45.74	46.95	49.28	47.76	48.29	46.93	50.25	46.47	47.72	47.12
C20:0	0.00	0.00	0.00	0.00	0.00	0.56	0.00	0.00	0.58	0.00	0.57
C18:3	4.37	4.97	2.63	4.32	4.99	4.01	3.66	2.08	3.70	4.04	3.04
C20:1	0.00	0.00	0.59	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C20:2	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C22:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C20:3	0.84	0.97	0.83	0.94	1.04	0.64	0.74	0.74	0.90	0.85	0.80
C20:4	1.15	1.24	1.24	1.15	1.39	0.89	1.11	1.19	1.46	1.09	1.08
C24:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C20:5	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C22:5	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C22:6	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00

[1] FA = fatty acid, presented as g FA per 100 g fat.

[2] NA = not analyzed.

Fatty Acid Composition of Colostrum and Milk from FISH Mares

Table A-36. Fatty acid composition of FISH mare colostrum

	Mare									
FA[1]	B24	A66	W69	B31	A59	B33	B46	B14	B01	B47
C8:0	3.16	3.05	3.94	5.26	2.84	3.05	5.25	3.69	6.11	4.19
C10:0	6.87	5.71	12.90	12.69	6.13	7.80	11.67	7.90	13.44	9.46
C12:0	5.29	3.42	11.37	9.29	4.12	5.42	9.18	6.11	10.36	6.52
C14:0	2.94	2.65	7.06	6.14	3.83	2.85	6.49	4.09	7.21	4.40
C14:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C16:0	23.05	22.84	18.77	17.83	24.84	20.41	20.55	19.98	17.25	20.12
C16:1	4.11	4.91	3.88	3.22	5.97	4.03	5.32	5.22	4.22	3.89
C17:0	0.00	0.00	0.00	0.30	0.00	0.00	0.00	0.00	0.34	0.00
C17:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C18:0	3.31	2.20	1.83	2.21	2.04	2.15	1.69	2.38	2.24	2.34
C18:1	20.86	22.22	16.00	14.51	22.17	20.51	17.78	20.27	12.98	18.10
C18:2	22.53	24.90	19.43	21.29	23.41	24.43	16.33	24.86	15.38	21.47
C20:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C18:3	7.25	7.43	3.80	5.24	4.65	8.43	4.94	3.52	5.33	8.81
C20:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.53	0.00	0.00
C20:2	0.63	0.66	1.03	0.76	0.00	0.91	0.79	0.72	0.61	0.69
C22:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C20:3	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C20:4	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.34	0.00
C24:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C20:5	0.00	0.00	0.00	0.41	0.00	0.00	0.00	0.33	1.53	0.00
C22:5	0.00	0.00	0.00	0.27	0.00	0.00	0.00	0.00	0.74	0.00
C22:6	0.00	0.00	0.00	0.58	0.00	0.00	0.00	0.40	1.92	0.00

[1] FA = fatty acid, presented as g FA per 100 g fat.

Table A-37. Fatty acid composition of FISH mare milk at 36 h post-foaling

	Mare									
FA[1]	B24	A66	W69	B31	A59	B33	B46	B14	B01	B47
C8:0	5.46	3.89	5.90	3.05	5.00	6.07	4.99	6.38	5.27	6.95
C10:0	12.21	5.75	12.93	5.44	7.72	11.14	11.28	14.68	11.81	14.50
C12:0	11.96	3.71	12.37	3.90	5.09	9.11	10.47	13.59	10.77	12.15
C14:0	8.36	3.80	8.58	4.34	4.44	6.19	7.78	9.33	7.70	8.20
C14:1	0.77	0.31	0.97	0.26	0.23	0.53	0.19	0.53	0.48	0.67
C16:0	20.29	21.55	18.52	23.43	22.27	16.96	23.02	19.03	21.37	18.14
C16:1	5.21	5.65	5.15	6.18	6.75	4.89	5.63	4.99	4.96	4.59
C17:0	0.00	0.00	0.00	0.29	0.00	0.22	0.00	0.00	0.00	0.19
C17:1	0.00	0.00	0.00	0.48	0.00	0.27	0.00	0.00	0.38	0.19
C18:0	1.61	2.41	1.03	2.01	1.90	1.33	1.65	1.52	1.67	1.63
C18:1	13.53	21.08	13.26	22.45	21.53	15.66	16.05	10.59	14.15	12.53
C18:2	16.30	20.61	15.74	20.39	20.03	19.13	13.54	14.00	13.27	15.95
C20:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C18:3	3.98	10.87	3.72	7.22	4.52	6.38	4.28	2.35	6.19	3.97
C20:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C20:2	0.33	0.37	0.28	0.43	0.34	0.37	0.00	0.28	0.22	0.25
C22:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C20:3	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C20:4	0.00	0.00	0.00	0.00	0.00	0.00	0.68	0.00	0.00	0.19
C24:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C20:5	0.00	0.00	0.66	0.00	0.00	0.67	0.00	0.93	0.57	0.00
C22:5	0.00	0.00	0.28	0.00	0.00	0.29	0.00	0.27	0.33	0.00
C22:6	0.00	0.00	0.77	0.00	0.00	0.80	0.00	1.37	0.76	0.00

[1] FA = fatty acid, presented as g FA per 100 g fat.

Table A-38. Fatty acid composition of FISH mare milk at 14 d post-foaling

	Mare									
FA[1]	B24	A66	W69	B31	A59	B33	B46	B14	B01	B47
C8:0	7.60	7.54	7.62	6.65	5.94	8.28	8.37	8.68	9.09	7.21
C10:0	14.91	12.16	9.62	9.76	8.54	16.68	13.77	17.76	16.70	13.43
C12:0	13.94	8.82	11.36	7.71	5.73	15.88	11.23	16.18	13.22	11.09
C14:0	9.40	5.51	7.78	5.55	4.29	10.42	7.21	10.75	7.65	7.13
C14:1	0.86	0.34	0.98	0.58	0.69	1.34	0.71	1.44	0.38	0.48
C16:0	19.44	15.03	17.31	15.50	17.32	19.53	16.31	18.91	18.40	16.84
C16:1	6.29	3.84	6.05	6.15	7.65	6.87	5.20	6.16	3.07	4.09
C17:0	0.18	0.20	0.00	0.00	0.16	0.28	0.00	0.16	0.30	0.24
C17:1	0.34	0.21	0.26	0.24	0.35	0.35	0.26	0.29	0.27	0.31
C18:0	1.26	1.99	1.03	1.52	1.42	1.19	1.43	1.32	1.82	1.60
C18:1	9.10	12.33	14.69	14.85	19.16	8.89	12.72	8.72	10.15	11.04
C18:2	8.77	13.16	14.84	17.39	20.39	6.95	12.14	6.33	7.34	10.94
C20:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C18:3	5.60	17.23	8.45	10.64	5.46	1.49	7.87	1.50	9.62	13.36
C20:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C20:2	0.26	0.33	0.34	0.36	0.36	0.24	0.32	0.19	0.00	0.29
C22:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C20:3	0.00	0.32	0.23	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C20:4	0.32	0.30	0.30	0.37	0.41	0.33	0.39	0.34	0.39	0.36
C24:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C20:5	0.49	0.32	0.48	0.87	0.95	0.53	0.75	0.59	0.78	0.55
C22:5	0.28	0.00	0.24	0.34	0.31	0.23	0.25	0.22	0.00	0.25
C22:6	0.95	0.37	0.68	1.56	1.13	0.85	1.24	1.00	0.82	0.79

[1] FA = fatty acid, presented as g FA per 100 g fat.

Table A-39. Fatty acid composition of FISH mare milk at 28 d post-foaling

	Mare									
FA[1]	B24	A66	W69	B31	A59	B33	B46	B14	B01	B47
C8:0	6.38	NA[2]	9.41	7.37	7.23	8.90	5.03	8.59	6.30	NA
C10:0	10.91	NA	15.92	11.99	11.39	14.00	6.55	15.18	9.28	NA
C12:0	10.06	NA	14.25	10.38	9.12	12.82	4.97	13.60	7.28	NA
C14:0	6.82	NA	8.42	6.51	5.63	7.59	4.05	8.20	4.71	NA
C14:1	0.83	NA	0.85	0.52	0.49	0.71	0.43	0.87	0.34	NA
C16:0	16.99	NA	16.98	16.22	16.07	16.23	18.45	15.37	15.14	NA
C16:1	5.89	NA	4.94	5.39	4.89	6.24	7.57	4.62	4.60	NA
C17:0	0.30	NA	0.00	0.00	0.00	0.00	0.00	0.19	0.00	NA
C17:1	0.00	NA	0.30	0.35	0.40	0.45	0.46	0.28	0.38	NA
C18:0	1.42	NA	0.97	1.49	1.38	1.32	1.60	1.25	1.55	NA
C18:1	11.46	NA	11.04	12.68	13.93	11.94	19.64	8.22	13.93	NA
C18:2	12.52	NA	7.83	14.30	14.15	10.14	17.07	7.84	14.37	NA
C20:0	0.00	NA	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA
C18:3	13.83	NA	7.39	10.04	14.24	7.17	12.23	13.72	18.14	NA
C20:1	0.00	NA	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA
C20:2	0.32	NA	0.22	0.38	0.31	0.32	0.34	0.00	0.32	NA
C22:0	0.00	NA	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA
C20:3	0.27	NA	0.00	0.00	0.00	0.00	0.00	0.25	0.31	NA
C20:4	0.36	NA	0.30	0.00	0.00	0.00	0.00	0.26	0.29	NA
C24:1	0.00	NA	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA
C20:5	0.61	NA	0.49	0.75	0.28	0.81	0.73	0.64	1.20	NA
C22:5	0.24	NA	0.00	0.31	0.00	0.00	0.00	0.25	0.43	NA
C22:6	0.88	NA	0.74	1.28	0.44	1.23	0.84	0.77	1.41	NA

[1] FA = fatty acid, presented as g FA per 100 g fat.

[2] NA = not analyzed.

Table A-40. Fatty acid composition of FISH mare milk at 56 d post-foaling

	Mare									
FA[1]	B24	A66	W69	B31	A59	B33	B46	B14	B01	B47
C8:0	OT[2]	6.35	NA[3]	6.56	7.03	7.47	NA	7.32	10.07	3.60
C10:0	OT	12.79	NA	8.43	11.29	9.72	NA	11.47	15.30	7.38
C12:0	OT	11.04	NA	6.90	9.91	9.13	NA	10.40	11.82	7.37
C14:0	OT	6.54	NA	4.66	5.97	5.32	NA	6.09	5.86	4.89
C14:1	OT	1.04	NA	0.56	0.66	0.77	NA	1.01	0.30	0.84
C16:0	OT	15.08	NA	16.03	16.25	14.64	NA	14.79	16.64	15.80
C16:1	OT	4.54	NA	6.90	5.13	5.82	NA	5.24	3.69	5.23
C17:0	OT	0.18	NA	0.00	0.00	0.00	NA	0.22	0.00	0.27
C17:1	OT	0.35	NA	0.39	0.36	0.44	NA	0.34	0.48	0.52
C18:0	OT	0.97	NA	1.16	1.32	1.17	NA	1.28	1.20	1.04
C18:1	OT	9.41	NA	16.40	12.97	11.85	NA	10.30	13.50	15.56
C18:2	OT	10.49	NA	17.01	14.85	14.37	NA	13.25	8.96	13.69
C20:0	OT	0.00	NA	0.00	0.00	0.00	NA	0.00	0.00	0.00
C18:3	OT	19.64	NA	14.17	12.76	17.41	NA	16.34	9.99	22.42
C20:1	OT	0.00	NA	0.00	0.00	0.00	NA	0.00	0.00	0.00
C20:2	OT	0.27	NA	0.33	0.36	0.36	NA	0.28	0.00	0.37
C22:0	OT	0.00	NA	0.00	0.00	0.00	NA	0.00	0.00	0.00
C20:3	OT	0.00	NA	0.00	0.00	0.00	NA	0.00	0.00	0.00
C20:4	OT	0.41	NA	0.00	0.50	0.42	NA	0.45	0.00	0.55
C24:1	OT	0.00	NA	0.00	0.00	0.00	NA	0.00	0.00	0.00
C20:5	OT	0.49	NA	0.00	0.29	0.41	NA	0.66	0.86	0.29
C22:5	OT	0.21	NA	0.00	0.00	0.00	NA	0.00	0.00	0.00
C22:6	OT	0.60	NA	0.45	0.41	0.73	NA	0.83	1.18	0.44

[1] FA = fatty acid, presented as g FA per 100 g fat.
[2] OT = off trial.
[3] NA = not analyzed.

Table A-41. Fatty acid composition of FISH mare milk at 84 d post-foaling

	Mare									
FA[1]	B24	A66	W69	B31	A59	B33	B46	B14	B01	B47
C8:0	OT[2]	6.32	5.94	4.70	NA[3]	6.67	6.47	6.55	5.17	6.19
C10:0	OT	8.90	8.43	6.81	NA	8.98	8.29	9.50	6.37	9.43
C12:0	OT	7.31	8.16	5.99	NA	9.03	7.25	8.73	5.23	9.12
C14:0	OT	4.52	5.21	4.68	NA	5.23	4.28	5.24	2.95	5.71
C14:1	OT	0.53	1.04	1.35	NA	1.20	0.66	0.84	0.37	0.99
C16:0	OT	15.98	16.02	16.16	NA	14.29	15.64	14.93	15.71	16.25
C16:1	OT	5.65	5.71	7.44	NA	6.18	7.07	6.16	5.01	6.01
C17:0	OT	0.00	0.00	0.16	NA	0.00	0.00	0.00	0.00	0.00
C17:1	OT	0.00	0.42	0.51	NA	0.48	0.40	0.54	0.56	0.51
C18:0	OT	1.36	1.17	0.94	NA	1.15	1.33	1.43	1.77	1.04
C18:1	OT	13.91	16.35	19.67	NA	11.49	16.21	13.54	16.41	12.85
C18:2	OT	14.99	14.12	19.12	NA	13.83	14.97	13.18	16.47	12.23
C20:0	OT	0.00	0.00	0.00	NA	0.00	0.00	0.00	0.00	0.00
C18:3	OT	20.56	16.34	11.44	NA	19.74	15.78	17.58	22.76	19.80
C20:1	OT	0.00	0.00	0.00	NA	0.00	0.00	0.00	0.00	0.00
C20:2	OT	0.00	0.44	0.43	NA	0.41	0.00	0.30	0.00	0.00
C22:0	OT	0.00	0.00	0.00	NA	0.00	0.00	0.00	0.00	0.00
C20:3	OT	0.00	0.00	0.00	NA	0.00	0.00	0.00	0.00	0.00
C20:4	OT	0.00	0.54	0.00	NA	0.00	0.59	0.55	0.00	0.00
C24:1	OT	0.00	0.00	0.00	NA	0.00	0.00	0.00	0.00	0.00
C20:5	OT	0.00	0.00	0.41	NA	0.47	0.43	0.38	0.45	0.00
C22:5	OT	0.00	0.00	0.27	NA	0.30	0.00	0.00	0.00	0.00
C22:6	OT	0.00	0.44	0.65	NA	0.85	0.62	0.53	0.69	0.00

[1] FA = fatty acid, presented as g FA per 100 g fat.
[2] OT = off trial.
[3] NA = not analyzed.

Fatty Acid Composition of Colostrum and Milk from FLAX Mares

Table A-42. Fatty acid composition of FLAX mare colostrum

	Mare										
FA[1]	B21	B32	A62	C2	C6	A65	B44	B28	B19	B06	C1
C8:0	3.88	4.41	4.95	4.52	3.18	5.10	4.89	3.80	4.69	4.05	NA[2]
C10:0	10.96	9.73	10.04	9.34	6.83	10.67	11.87	7.34	9.75	10.03	NA
C12:0	8.53	6.94	6.70	6.17	4.47	6.52	8.62	6.13	6.52	7.18	NA
C14:0	5.35	4.54	4.27	3.92	3.44	3.81	4.95	4.52	4.42	4.73	NA
C14:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA
C16:0	19.21	19.52	18.93	17.70	19.21	16.32	15.51	18.96	19.05	18.20	NA
C16:1	3.78	3.64	4.90	3.44	2.89	3.11	2.38	5.81	3.51	2.67	NA
C17:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA
C17:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA
C18:0	2.66	2.41	1.87	1.91	3.01	1.97	2.34	1.81	2.61	2.52	NA
C18:1	17.84	18.46	16.31	18.90	18.34	16.07	16.08	18.95	18.56	16.86	NA
C18:2	18.96	21.89	18.35	25.79	0.79	22.84	22.37	22.66	23.04	22.71	NA
C20:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA
C18:3	7.81	7.80	13.26	7.26	10.42	12.30	10.14	9.44	7.31	10.08	NA
C20:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA
C20:2	1.01	0.66	0.41	1.06	0.63	0.82	0.86	0.59	0.52	0.96	NA
C22:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA
C20:3	0.00	0.00	0.00	0.00	0.00	0.46	0.00	0.00	0.00	0.00	NA
C20:4	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA
C24:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA
C20:5	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA
C22:5	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA
C22:6	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA

[1] FA = fatty acid, presented as g FA per 100 g fat.
[2] NA = not analyzed.

Table A-43. Fatty acid composition of FLAX mare milk at 36 h post-foaling

	Mare										
FA[1]	B21	B32	A62	C2	C6	A65	B44	B28	B19	B06	C1
C8:0	4.40	3.41	6.64	6.60	3.92	5.66	6.03	4.24	6.45	4.93	NA[2]
C10:0	9.41	6.68	11.94	12.53	8.11	11.29	12.35	9.70	14.09	11.57	NA
C12:0	8.07	5.16	9.45	9.77	6.70	9.06	10.60	9.50	11.87	10.86	NA
C14:0	6.41	4.71	6.17	5.79	5.24	6.40	6.98	7.06	7.55	8.50	NA
C14:1	0.44	0.35	0.38	0.39	0.24	0.33	0.42	0.72	0.34	0.54	NA
C16:0	22.30	22.04	17.32	18.50	19.99	17.98	17.05	20.14	18.49	21.47	NA
C16:1	5.57	5.18	5.90	1.05	4.07	4.18	3.35	4.80	3.10	4.33	NA
C17:0	0.00	0.26	0.00	0.00	0.23	0.00	0.00	0.20	0.00	0.00	NA
C17:1	0.00	0.31	0.34	0.00	0.31	0.00	0.00	0.30	0.00	0.23	NA
C18:0	1.88	2.29	1.30	1.68	1.98	1.76	1.49	1.47	1.88	1.58	NA
C18:1	19.09	22.03	15.31	16.68	18.20	14.37	14.64	15.28	12.91	13.11	NA
C18:2	15.53	20.32	13.49	17.16	21.22	16.18	18.76	18.78	15.55	15.68	NA
C20:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA
C18:3	6.54	6.46	11.33	6.41	9.35	12.05	7.82	7.50	6.95	6.87	NA
C20:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA
C20:2	0.34	0.45	0.00	0.44	0.36	0.39	0.39	0.39	0.34	0.34	NA
C22:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA
C20:3	0.00	0.00	0.30	0.00	0.00	0.33	0.00	0.00	0.00	0.00	NA
C20:4	0.00	0.35	0.00	0.00	0.00	0.00	0.00	0.00	0.38	0.00	NA
C24:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA
C20:5	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA
C22:5	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA
C22:6	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA

[1] FA = fatty acid, presented as g FA per 100 g fat.

[2] NA = not analyzed.

Table A-44. Fatty acid composition of FLAX mare milk at 14 d post-foaling

	Mare										
FA[1]	B21	B32	A62	C2	C6	A65	B44	B28	B19	B06	C1
C8:0	7.09	7.40	8.50	9.96	7.34	8.23	5.87	NA[2]	7.24	NA	6.63
C10:0	12.11	12.54	15.38	16.92	13.69	14.50	11.72	NA	13.11	NA	12.12
C12:0	10.48	11.03	13.23	14.74	12.30	12.80	11.83	NA	10.50	NA	10.05
C14:0	6.61	7.19	7.81	8.26	8.17	7.93	8.13	NA	6.90	NA	7.04
C14:1	0.63	1.18	0.80	0.75	0.83	1.09	0.88	NA	0.51	NA	0.50
C16:0	16.69	15.59	16.47	14.50	17.96	14.85	18.18	NA	18.36	NA	16.98
C16:1	5.84	4.86	5.01	4.41	4.88	7.38	6.42	NA	4.42	NA	5.00
C17:0	0.00	0.17	0.00	0.00	0.00	0.00	0.00	NA	0.29	NA	0.13
C17:1	0.38	0.31	0.26	0.37	0.29	0.22	0.38	NA	0.38	NA	0.27
C18:0	1.36	1.15	1.35	0.88	1.21	1.20	1.16	NA	1.82	NA	1.45
C18:1	16.20	11.31	10.09	9.87	12.44	8.80	10.90	NA	16.30	NA	14.78
C18:2	14.30	10.77	9.32	9.45	12.63	10.34	9.01	NA	13.07	NA	15.44
C20:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA	0.00	NA	0.00
C18:3	7.13	16.01	11.09	8.93	7.52	15.03	14.85	NA	6.40	NA	8.81
C20:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA	0.00	NA	0.00
C20:2	0.47	0.33	0.26	0.37	0.33	0.34	0.00	NA	0.37	NA	0.31
C22:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA	0.00	NA	0.00
C20:3	0.33	0.38	0.34	0.30	0.24	0.47	0.31	NA	0.00	NA	0.23
C20:4	0.37	0.26	0.27	0.27	0.30	0.25	0.31	NA	0.36	NA	0.27
C24:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA	0.00	NA	0.00
C20:5	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA	0.00	NA	0.00
C22:5	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA	0.00	NA	0.00
C22:6	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA	0.00	NA	0.00

[1] FA = fatty acid, presented as g FA per 100 g fat.

[2] NA = not analyzed.

Table A-45. Fatty acid composition of FLAX mare milk at 28 d post-foaling

	Mare										
FA[1]	B21	B32	A62	C2	C6	A65	B44	B28	B19	B06	C1
C8:0	5.87	NA[2]	9.00	8.51	8.33	9.78	5.11	7.48	7.58	NA	7.41
C10:0	9.20	NA	15.36	12.92	14.03	15.04	8.26	11.67	11.61	NA	13.03
C12:0	8.05	NA	13.22	11.59	13.38	12.74	6.54	10.15	9.58	NA	11.44
C14:0	5.48	NA	7.76	6.79	8.15	7.08	4.38	6.31	5.71	NA	7.05
C14:1	0.67	NA	0.76	1.34	1.00	0.69	0.34	0.71	0.42	NA	0.50
C16:0	15.86	NA	16.16	15.03	16.94	15.00	15.40	16.46	15.01	NA	16.84
C16:1	5.84	NA	4.46	5.23	5.48	7.70	4.51	6.12	4.49	NA	4.73
C17:0	0.00	NA	0.00	0.21	0.00	0.00	0.00	0.00	0.00	NA	0.00
C17:1	0.43	NA	0.22	0.38	0.37	0.25	0.37	0.36	0.43	NA	0.36
C18:0	1.43	NA	1.39	1.07	1.24	1.31	1.56	1.67	1.56	NA	1.57
C18:1	14.99	NA	8.82	11.26	10.17	10.24	17.15	14.40	15.01	NA	13.94
C18:2	15.05	NA	8.46	12.71	9.67	8.92	18.99	13.22	12.85	NA	11.69
C20:0	0.00	NA	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA	0.00
C18:3	15.80	NA	13.99	12.65	10.72	9.99	16.76	9.95	15.32	NA	10.71
C20:1	0.00	NA	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA	0.00
C20:2	0.56	NA	0.20	0.49	0.28	0.28	0.36	0.33	0.34	NA	0.38
C22:0	0.00	NA	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA	0.00
C20:3	0.58	NA	0.32	0.34	0.27	0.30	0.27	0.00	0.00	NA	0.29
C20:4	0.32	NA	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA	0.00
C24:1	0.00	NA	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA	0.00
C20:5	0.00	NA	0.00	0.00	0.00	0.33	0.00	0.49	0.00	NA	0.00
C22:5	0.00	NA	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA	0.00
C22:6	0.00	NA	0.00	0.00	0.00	0.40	0.00	0.66	0.00	NA	0.00

[1] FA = fatty acid, presented as g FA per 100 g fat.

[2] NA = not analyzed.

Table A-46. Fatty acid composition of FLAX mare milk at 56 d post-foaling

	Mare										
FA[1]	B21	B32	A62	C2	C6	A65	B44	B28	B19	B06	C1
C8:0	4.56	NA[2]	7.52	NA	NA	NA	5.58	5.67	6.83	7.23	7.43
C10:0	6.88	NA	11.44	NA	NA	NA	8.43	7.40	9.08	11.61	11.04
C12:0	5.93	NA	10.27	NA	NA	NA	7.95	6.39	7.41	10.63	9.87
C14:0	4.25	NA	5.91	NA	NA	NA	4.88	4.27	3.62	6.20	5.70
C14:1	0.85	NA	0.69	NA	NA	NA	0.63	0.55	0.64	1.17	0.60
C16:0	15.27	NA	15.71	NA	NA	NA	16.06	16.34	15.14	14.36	15.23
C16:1	6.23	NA	5.12	NA	NA	NA	4.78	7.42	4.53	4.36	4.32
C17:0	0.19	NA	0.00	NA	NA	NA	0.00	0.00	0.28	0.21	0.00
C17:1	0.53	NA	0.38	NA	NA	NA	0.46	0.51	0.47	0.34	0.38
C18:0	1.01	NA	0.86	NA	NA	NA	1.43	1.34	1.36	1.08	1.03
C18:1	17.94	NA	10.30	NA	NA	NA	14.56	18.71	16.10	9.47	10.56
C18:2	16.15	NA	10.58	NA	NA	NA	14.60	16.35	14.39	12.62	9.70
C20:0	0.00	NA	0.00	NA	NA	NA	0.00	0.00	0.00	0.00	0.00
C18:3	19.36	NA	20.68	NA	NA	NA	19.60	14.19	19.26	20.22	23.55
C20:1	0.00	NA	0.00	NA	NA	NA	0.00	0.00	0.00	0.00	0.00
C20:2	0.56	NA	0.00	NA	NA	NA	0.42	0.42	0.44	0.37	0.00
C22:0	0.00	NA	0.00	NA	NA	NA	0.00	0.00	0.00	0.00	0.00
C20:3	0.00	NA	0.00	NA	NA	NA	0.00	0.00	0.00	0.00	0.00
C20:4	0.68	NA	0.56	NA	NA	NA	0.62	0.00	0.62	0.62	0.58
C24:1	0.00	NA	0.00	NA	NA	NA	0.00	0.00	0.00	0.00	0.00
C20:5	0.00	NA	0.00	NA	NA	NA	0.00	0.00	0.00	0.00	0.00
C22:5	0.00	NA	0.00	NA	NA	NA	0.00	0.00	0.00	0.00	0.00
C22:6	0.00	NA	0.00	NA	NA	NA	0.00	0.39	0.00	0.00	0.00

[1] FA = fatty acid, presented as g FA per 100 g fat.

[2] NA = not analyzed.

Table A-47. Fatty acid composition of FLAX mare milk at 84 d post-foaling

	Mare										
FA[1]	B21	B32	A62	C2	C6	A65	B44	B28	B19	B06	C1
C8:0	5.67	5.17	NA[2]	7.37	NA	NA	4.20	NA	7.53	6.43	5.47
C10:0	8.56	6.42	NA	10.23	NA	NA	6.96	NA	9.94	10.26	10.10
C12:0	8.25	5.74	NA	9.62	NA	NA	6.94	NA	8.36	10.81	10.93
C14:0	5.33	3.64	NA	5.55	NA	NA	5.30	NA	4.35	7.08	7.88
C14:1	1.26	0.60	NA	1.07	NA	NA	2.26	NA	0.58	1.30	3.36
C16:0	14.86	15.43	NA	15.18	NA	NA	16.47	NA	14.99	18.19	18.23
C16:1	5.86	6.60	NA	5.69	NA	NA	5.72	NA	4.57	7.66	8.33
C17:0	0.00	0.00	NA	0.00	NA	NA	0.29	NA	0.00	0.00	0.14
C17:1	0.49	0.38	NA	0.64	NA	NA	0.64	NA	0.45	0.00	0.65
C18:0	0.90	1.29	NA	0.92	NA	NA	1.06	NA	1.40	0.99	0.64
C18:1	12.77	18.59	NA	14.02	NA	NA	17.73	NA	17.01	12.84	13.38
C18:2	12.68	20.19	NA	10.21	NA	NA	13.88	NA	12.89	8.11	8.17
C20:0	0.00	0.00	NA	0.00	NA	NA	0.00	NA	0.00	0.00	0.00
C18:3	22.81	15.41	NA	19.18	NA	NA	19.11	NA	17.59	15.85	14.33
C20:1	0.00	0.00	NA	0.00	NA	NA	0.00	NA	0.00	0.00	0.00
C20:2	0.49	0.46	NA	0.29	NA	NA	0.48	NA	0.35	0.00	0.25
C22:0	0.00	0.00	NA	0.00	NA	NA	0.00	NA	0.00	0.00	0.00
C20:3	0.00	0.00	NA	0.00	NA	NA	0.00	NA	0.00	0.00	0.00
C20:4	0.58	0.00	NA	0.33	NA	NA	0.53	NA	0.00	0.57	0.28
C24:1	0.00	0.00	NA	0.00	NA	NA	0.00	NA	0.00	0.00	0.00
C20:5	0.00	0.00	NA	0.00	NA	NA	0.00	NA	0.00	0.00	0.00
C22:5	0.00	0.00	NA	0.00	NA	NA	0.00	NA	0.00	0.00	0.00
C22:6	0.00	0.00	NA	0.00	NA	NA	0.00	NA	0.00	0.00	0.00

[1] FA = fatty acid, presented as g FA per 100 g fat.

[2] NA = not analyzed.

Fatty Acid Composition of Colostrum and Milk from CON Mares

Table A-48. Fatty acid composition of CON mare colostrum

	Mare										
FA[1]	B29	B36	B41	A64	B13	B45	A54	B18	C4	B26	A61
C8:0	3.99	3.35	4.75	1.52	3.33	3.44	3.20	3.30	5.47	5.75	5.49
C10:0	9.13	7.84	11.69	1.84	19.90	9.04	7.99	8.25	12.86	14.85	12.83
C12:0	8.89	6.16	7.73	1.40	5.19	7.91	5.79	6.49	9.22	11.09	9.06
C14:0	6.04	4.85	4.61	3.47	4.30	5.51	4.91	3.63	5.22	6.19	5.66
C14:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C16:0	18.53	21.71	18.29	29.23	18.76	20.03	22.90	21.15	16.57	17.39	19.86
C16:1	5.42	3.74	3.51	8.13	5.36	3.58	6.19	3.73	2.41	2.84	3.33
C17:0	0.00	0.00	0.00	0.00	0.00	0.37	0.00	0.00	0.00	0.00	0.00
C17:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C18:0	1.52	2.36	2.13	2.38	1.61	3.39	1.83	2.09	2.10	1.99	2.18
C18:1	16.34	19.02	19.41	25.52	16.37	18.67	18.93	19.28	15.88	15.17	16.90
C18:2	21.93	21.42	21.44	20.61	17.47	22.14	19.81	23.73	23.63	20.08	18.32
C20:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C18:3	8.22	8.23	5.51	5.89	6.72	4.92	8.46	7.31	5.72	3.61	5.73
C20:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C20:2	0.00	0.92	0.93	0.00	0.71	0.03	0.00	1.03	0.94	1.04	0.65
C22:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C20:3	0.00	0.40	0.00	0.00	0.29	0.00	0.00	0.00	0.00	0.00	0.00
C20:4	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C24:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C20:5	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C22:5	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C22:6	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00

[1] FA = fatty acid, presented as g FA per 100 g fat.

Table A-49. Fatty acid composition of CON mare milk at 36 h post-foaling

	Mare										
FA[1]	B29	B36	B41	A64	B13	B45	A54	B18	C4	B26	A61
C8:0	NA[2]	5.26	5.35	2.63	NA	7.90	5.83	6.02	4.86	6.35	5.09
C10:0	NA	10.44	11.65	3.91	NA	14.66	13.06	11.59	9.38	13.85	11.01
C12:0	NA	9.39	10.72	2.97	NA	12.26	13.68	10.17	7.93	12.23	10.25
C14:0	NA	6.58	7.86	3.68	NA	8.09	9.35	6.43	5.99	8.61	8.03
C14:1	NA	0.34	0.73	0.29	NA	0.00	0.92	0.36	0.39	0.61	0.63
C16:0	NA	21.22	19.80	24.07	NA	19.23	20.77	18.22	19.01	20.55	21.93
C16:1	NA	5.54	5.29	7.98	NA	5.62	5.99	4.11	4.47	5.03	5.12
C17:0	NA	0.00	0.17	0.00	NA	0.20	0.00	0.00	0.00	0.00	0.00
C17:1	NA	0.00	0.28	0.00	NA	0.37	0.00	0.00	0.00	0.00	0.00
C18:0	NA	1.75	1.43	2.14	NA	1.59	1.19	1.79	1.57	1.25	1.40
C18:1	NA	17.40	15.85	26.14	NA	16.25	11.10	16.16	17.93	13.76	15.91
C18:2	NA	14.16	16.40	20.28	NA	13.84	14.70	20.25	21.26	14.54	15.01
C20:0	NA	0.00	0.00	0.00	NA	0.00	0.00	0.00	0.00	0.00	0.00
C18:3	NA	7.65	4.15	5.31	NA	0.00	3.00	4.20	6.70	2.88	5.19
C20:1	NA	0.00	0.00	0.00	NA	0.00	0.00	0.00	0.00	0.00	0.00
C20:2	NA	0.00	0.33	0.39	NA	0.00	0.21	0.54	0.42	0.35	0.27
C22:0	NA	0.00	0.00	0.00	NA	0.00	0.00	0.00	0.00	0.00	0.00
C20:3	NA	0.00	0.00	0.00	NA	0.00	0.00	0.00	0.00	0.00	0.17
C20:4	NA	0.00	0.00	0.00	NA	0.00	0.00	0.00	0.00	0.00	0.00
C24:1	NA	0.00	0.00	0.00	NA	0.00	0.00	0.00	0.00	0.00	0.00
C20:5	NA	0.00	0.00	0.00	NA	0.00	0.00	0.00	0.00	0.00	0.00
C22:5	NA	0.00	0.00	0.00	NA	0.00	0.00	0.00	0.00	0.00	0.00
C22:6	NA	0.00	0.00	0.00	NA	0.00	0.00	0.00	0.00	0.00	0.00

[1] FA = fatty acid, presented as g FA per 100 g fat.

[2] NA = not analyzed.

Table A-50. Fatty acid composition of CON mare milk at 14 d post-foaling

	Mare										
FA[1]	B29	B36	B41	A64	B13	B45	A54	B18	C4	B26	A61
C8:0	5.93	4.39	6.97	5.41	5.52	8.09	5.97	8.32	6.00	3.39	5.77
C10:0	8.96	7.14	12.12	8.52	9.01	14.32	9.90	14.46	9.69	4.34	10.37
C12:0	6.52	6.27	10.40	6.90	7.44	12.90	7.87	12.51	8.08	2.95	8.98
C14:0	4.43	5.14	6.49	5.35	5.24	8.38	5.89	7.47	5.35	3.32	6.88
C14:1	0.36	0.59	0.52	0.83	0.56	1.10	0.63	1.04	0.68	0.50	0.55
C16:0	16.99	18.43	16.06	18.37	18.45	17.74	18.06	15.91	15.19	19.32	19.51
C16:1	5.44	7.95	5.21	7.34	7.12	5.93	7.06	4.98	4.96	9.11	6.33
C17:0	0.00	0.00	0.00	0.00	0.00	0.18	0.00	0.17	0.00	0.00	0.00
C17:1	0.53	0.35	0.30	0.39	0.39	0.35	0.30	0.32	0.28	0.53	0.42
C18:0	1.97	1.29	1.44	1.50	1.20	1.39	1.28	1.35	1.30	1.36	1.69
C18:1	19.89	18.83	13.09	21.14	16.82	13.00	15.50	11.59	15.09	25.37	17.83
C18:2	18.58	15.51	13.90	18.69	15.53	9.33	14.40	10.07	18.61	21.38	13.64
C20:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C18:3	9.54	13.02	12.73	4.95	11.68	6.70	11.63	11.17	13.82	7.52	7.30
C20:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C20:2	0.45	0.37	0.31	0.49	0.35	0.35	0.25	0.38	0.46	0.57	0.33
C22:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C20:3	0.00	0.37	0.00	0.00	0.34	0.22	0.00	0.35	0.31	0.00	0.00
C20:4	0.41	0.36	0.40	0.33	0.35	0.29	0.27	0.25	0.33	0.35	0.41
C24:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C20:5	0.00	0.00	0.00	0.00	0.00	0.00	0.51	0.00	0.00	0.00	0.00
C22:5	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C22:6	0.00	0.00	0.00	0.00	0.00	0.00	0.55	0.00	0.00	0.00	0.00

[1] FA = fatty acid, presented as g FA per 100 g fat.

Table A-51. Fatty acid composition of CON mare milk at 28 d post-foaling

	Mare										
FA[1]	B29	B36	B41	A64	B13	B45	A54	B18	C4	B26	A61
C8:0	7.08	4.05	7.28	NA[2]	NA	7.11	9.29	9.83	4.82	NA	6.61
C10:0	10.65	5.67	10.32	NA	NA	10.21	16.65	15.44	6.66	NA	12.03
C12:0	9.85	4.73	8.42	NA	NA	9.09	14.57	13.80	5.66	NA	10.78
C14:0	5.91	3.80	4.74	NA	NA	5.31	8.31	7.60	3.82	NA	7.61
C14:1	0.81	0.39	0.32	NA	NA	0.71	0.88	0.87	0.53	NA	0.59
C16:0	14.41	18.82	14.43	NA	NA	15.43	16.13	15.52	14.55	NA	19.56
C16:1	4.70	8.79	4.89	NA	NA	5.26	5.45	5.25	5.31	NA	5.77
C17:0	0.00	0.00	0.00	NA	NA	0.00	0.00	0.00	0.00	NA	0.00
C17:1	0.45	0.57	0.34	NA	NA	0.29	0.27	0.33	0.46	NA	0.43
C18:0	1.35	1.04	1.87	NA	NA	1.40	1.19	1.33	1.55	NA	1.55
C18:1	11.64	21.87	15.44	NA	NA	14.67	9.33	10.75	17.09	NA	14.09
C18:2	13.43	17.39	16.57	NA	NA	13.88	9.85	7.42	19.25	NA	10.57
C20:0	0.00	0.00	0.00	NA	NA	0.00	0.00	0.00	0.00	NA	0.00
C18:3	18.74	12.26	14.83	NA	NA	15.88	7.55	11.01	18.98	NA	10.15
C20:1	0.00	0.00	0.00	NA	NA	0.00	0.00	0.00	0.00	NA	0.00
C20:2	0.37	0.41	0.41	NA	NA	0.41	0.27	0.25	0.53	NA	0.26
C22:0	0.00	0.00	0.00	NA	NA	0.00	0.00	0.00	0.00	NA	0.00
C20:3	0.44	0.00	0.00	NA	NA	0.35	0.00	0.31	0.41	NA	0.00
C20:4	0.28	0.00	0.00	NA	NA	0.00	0.00	0.31	0.37	NA	0.00
C24:1	0.00	0.00	0.00	NA	NA	0.00	0.00	0.00	0.00	NA	0.00
C20:5	0.00	0.00	0.00	NA	NA	0.00	0.00	0.00	0.00	NA	0.00
C22:5	0.00	0.00	0.00	NA	NA	0.00	0.00	0.00	0.00	NA	0.00
C22:6	0.00	0.00	0.00	NA	NA	0.00	0.26	0.00	0.00	NA	0.00

[1] FA = fatty acid, presented as g FA per 100 g fat.

[2] NA = not analyzed.

Table A-52. Fatty acid composition of CON mare milk at 56 d post-foaling

	Mare										
FA[1]	B29	B36	B41	A64	B13	B45	A54	B18	C4	B26	A61
C8:0	NA[2]	NA	7.43	5.30	6.01	NA	7.03	6.56	NA	4.78	NA
C10:0	NA	NA	11.36	7.64	8.37	NA	10.74	9.97	NA	6.54	NA
C12:0	NA	NA	10.41	7.14	6.59	NA	9.02	9.59	NA	5.08	NA
C14:0	NA	NA	6.24	5.07	4.32	NA	6.02	6.24	NA	3.56	NA
C14:1	NA	NA	1.06	1.33	0.58	NA	0.58	0.88	NA	0.44	NA
C16:0	NA	NA	16.49	15.91	16.77	NA	16.96	16.24	NA	16.98	NA
C16:1	NA	NA	4.89	6.78	7.00	NA	5.47	5.65	NA	6.50	NA
C17:0	NA	NA	0.30	0.17	0.00	NA	0.00	0.00	NA	0.00	NA
C17:1	NA	NA	0.50	0.45	0.38	NA	0.00	0.50	NA	0.38	NA
C18:0	NA	NA	1.15	0.85	1.15	NA	0.94	0.82	NA	1.49	NA
C18:1	NA	NA	14.15	18.55	17.43	NA	11.61	11.88	NA	19.57	NA
C18:2	NA	NA	11.86	15.26	17.15	NA	14.21	9.65	NA	19.00	NA
C20:0	NA	NA	0.00	0.00	0.00	NA	0.00	0.00	NA	0.00	NA
C18:3	NA	NA	13.70	15.25	13.26	NA	17.14	21.19	NA	14.34	NA
C20:1	NA	NA	0.00	0.00	0.00	NA	0.00	0.00	NA	0.00	NA
C20:2	NA	NA	0.39	0.46	0.47	NA	0.29	0.30	NA	0.36	NA
C22:0	NA	NA	0.00	0.00	0.00	NA	0.00	0.00	NA	0.00	NA
C20:3	NA	NA	0.00	0.00	0.00	NA	0.00	0.00	NA	0.00	NA
C20:4	NA	NA	0.46	0.41	0.59	NA	0.00	0.60	NA	0.48	NA
C24:1	NA	NA	0.00	0.00	0.00	NA	0.00	0.00	NA	0.00	NA
C20:5	NA	NA	0.00	0.00	0.00	NA	0.00	0.00	NA	0.00	NA
C22:5	NA	NA	0.00	0.00	0.00	NA	0.00	0.00	NA	0.00	NA
C22:6	NA	NA	0.00	0.00	0.00	NA	0.00	0.00	NA	0.46	NA

[1] FA = fatty acid, presented as g FA per 100 g fat.

[2] NA = not analyzed.

Table A-53. Fatty acid composition of CON mare milk at 84 d post-foaling

	Mare										
FA[1]	B29	B36	B41	A64	B13	B45	A54	B18	C4	B26	A61
C8:0	5.19	5.05	4.73	NA[2]	6.20	5.64	NA	NA	6.49	NA	NA
C10:0	6.77	7.27	8.19	NA	11.36	7.69	NA	NA	9.61	NA	NA
C12:0	5.79	8.05	8.06	NA	11.07	7.36	NA	NA	9.53	NA	NA
C14:0	3.70	5.24	5.58	NA	7.22	4.65	NA	NA	6.08	NA	NA
C14:1	0.65	0.95	3.03	NA	3.24	1.13	NA	NA	2.79	NA	NA
C16:0	13.86	16.92	15.25	NA	15.91	14.18	NA	NA	15.20	NA	NA
C16:1	5.75	8.07	5.87	NA	6.14	6.11	NA	NA	6.19	NA	NA
C17:0	0.00	0.00	0.26	NA	0.20	0.00	NA	NA	0.26	NA	NA
C17:1	0.60	0.46	0.59	NA	0.58	0.67	NA	NA	0.80	NA	NA
C18:0	1.05	1.15	0.89	NA	0.65	1.02	NA	NA	0.64	NA	NA
C18:1	15.36	13.47	15.05	NA	14.98	14.11	NA	NA	15.06	NA	NA
C18:2	17.47	12.83	13.25	NA	10.61	12.82	NA	NA	11.08	NA	NA
C20:0	0.00	0.00	0.00	NA	0.00	0.00	NA	NA	0.00	NA	NA
C18:3	23.51	20.44	20.39	NA	13.46	24.30	NA	NA	17.24	NA	NA
C20:1	0.00	0.00	0.00	NA	0.00	0.00	NA	NA	0.00	NA	NA
C20:2	0.38	0.00	0.43	NA	0.39	0.30	NA	NA	0.43	NA	NA
C22:0	0.00	0.00	0.00	NA	0.00	0.00	NA	NA	0.00	NA	NA
C20:3	0.00	0.00	0.00	NA	0.00	0.00	NA	NA	0.00	NA	NA
C20:4	0.00	0.00	0.69	NA	0.30	0.32	NA	NA	0.41	NA	NA
C24:1	0.00	0.00	0.00	NA	0.00	0.00	NA	NA	0.00	NA	NA
C20:5	0.00	0.00	0.00	NA	0.00	0.00	NA	NA	0.00	NA	NA
C22:5	0.00	0.00	0.00	NA	0.00	0.00	NA	NA	0.00	NA	NA
C22:6	0.00	0.00	0.00	NA	0.00	0.00	NA	NA	0.00	NA	NA

[1] FA = fatty acid, presented as g FA per 100 g fat.

[2] NA = not analyzed.

Fatty Acid Composition of Plasma from FISH Foals

Table A-54. Fatty acid composition of FISH foal plasma at birth

	Foal									
FA[1]	5B24	5A66	5W69	5B31	5A59	5B14	5B33	5B46	5B01	5B47
C8:0	NA[2]	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C10:0	NA	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C12:0	NA	0.56	0.59	0.00	0.77	0.64	0.00	0.84	0.00	0.00
C14:0	NA	0.00	0.00	0.00	0.00	0.00	0.00	0.65	0.00	0.00
C14:1	NA	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C16:0	NA	15.86	17.56	17.08	17.03	15.02	16.07	18.32	17.13	16.70
C16:1	NA	2.08	1.59	1.46	1.60	1.55	1.26	2.82	1.57	1.41
C17:0	NA	0.48	0.53	0.00	0.58	0.54	0.57	0.54	0.63	0.72
C17:1	NA	0.00	0.46	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C18:0	NA	18.10	19.13	20.61	21.08	22.63	21.14	18.12	20.41	20.51
C18:1	NA	8.28	8.30	7.36	7.09	6.92	6.38	8.95	8.30	7.23
C18:2	NA	44.60	41.21	45.51	43.17	42.93	44.78	36.87	40.32	45.85
C20:0	NA	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C18:3	NA	5.63	3.99	2.39	4.44	3.97	2.34	6.12	4.37	3.43
C20:1	NA	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C20:2	NA	0.51	0.63	0.61	0.81	0.53	0.56	0.62	0.55	0.00
C22:0	NA	0.80	0.81	0.00	0.00	0.95	0.00	0.78	0.80	0.00
C20:3	NA	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C20:4	NA	1.76	2.20	2.44	2.52	2.40	2.88	2.11	2.51	2.17
C24:1	NA	0.00	0.54	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C20:5	NA	0.46	0.56	0.80	0.00	0.63	1.52	0.84	0.96	0.69
C22:5	NA	0.00	0.59	0.00	0.00	0.00	0.60	0.66	0.53	0.00
C22:6	NA	0.88	1.32	1.74	0.90	1.29	1.90	1.76	1.91	1.29

[1] FA = fatty acid, presented as g FA per 100 g fat.
[2] NA = not analyzed.

Table A-55. Fatty acid composition of FISH foal plasma at 14 d of age

	Foal									
FA[1]	5B24	5A66	5W69	5B31	5A59	5B14	5B33	5B46	5B01	5B47
C8:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C10:0	0.38	0.57	0.54	0.42	0.00	0.00	0.00	0.65	0.39	0.42
C12:0	1.21	1.10	1.32	0.89	0.64	0.64	0.62	1.77	0.84	1.03
C14:0	1.70	1.19	1.61	1.06	0.86	0.93	0.92	1.99	1.23	1.12
C14:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C16:0	19.22	15.93	18.42	16.98	19.21	17.13	17.50	17.87	18.11	17.80
C16:1	2.61	1.79	2.07	2.37	3.97	1.83	2.14	2.39	1.50	1.57
C17:0	0.51	0.56	0.48	0.42	0.47	0.56	0.52	0.50	0.84	0.69
C17:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.43
C18:0	21.51	20.87	21.01	20.97	18.69	24.59	21.87	20.72	22.92	22.05
C18:1	7.03	8.38	7.85	9.26	12.81	6.77	7.60	8.29	8.21	7.20
C18:2	36.04	39.76	39.03	38.49	37.27	38.07	38.60	36.17	35.33	36.57
C20:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.42
C18:3	1.29	3.22	1.36	2.19	1.51	0.00	0.00	1.85	1.41	2.19
C20:1	0.00	0.00	0.40	0.00	0.40	0.00	0.00	0.00	0.00	0.00
C20:2	0.50	0.66	0.62	0.60	0.63	0.48	0.51	0.65	0.45	0.58
C22:0	1.03	0.77	0.66	0.82	0.51	0.89	0.92	0.65	0.91	0.77
C20:3	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C20:4	2.96	2.55	1.54	1.69	1.32	3.39	3.67	2.55	2.06	3.18
C24:1	0.00	0.00	0.00	0.40	0.00	0.39	0.00	0.00	0.00	0.00
C20:5	1.46	0.92	1.08	1.04	0.63	1.46	1.56	1.42	2.25	1.32
C22:5	0.62	0.46	0.48	0.48	0.00	0.54	0.73	0.59	0.68	0.52
C22:6	1.92	1.28	1.54	1.92	1.09	2.32	2.85	1.95	2.87	2.14

[1] FA = fatty acid, presented as g FA per 100 g fat.

Table A-56. Fatty acid composition of FISH foal plasma at 28 d of age

	Foal									
FA[1]	5B24	5A66	5W69	5B31	5A59	5B14	5B33	5B46	5B01	5B47
C8:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C10:0	0.54	0.59	0.61	0.00	0.00	0.45	0.00	0.45	0.00	0.00
C12:0	1.63	1.38	1.60	0.69	0.78	1.24	0.95	1.13	0.94	1.20
C14:0	1.77	1.11	1.17	0.75	0.73	1.23	0.94	1.16	0.95	0.76
C14:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C16:0	18.45	15.64	18.59	17.44	17.32	17.14	17.56	17.95	16.76	17.79
C16:1	2.63	1.94	1.97	1.71	1.59	1.99	2.00	3.13	1.89	1.47
C17:0	0.51	0.59	0.58	0.49	0.63	0.55	0.55	0.54	0.64	0.63
C17:1	0.00	0.56	0.48	0.00	0.00	0.00	0.87	0.00	0.00	0.58
C18:0	19.45	20.44	20.62	21.52	23.71	22.53	21.86	18.51	20.43	22.32
C18:1	6.72	7.00	7.89	6.69	6.79	6.12	7.02	9.94	8.19	7.02
C18:2	35.69	36.01	36.50	41.68	39.76	36.57	37.17	34.37	34.70	38.31
C20:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C18:3	4.50	6.01	1.20	1.22	1.97	3.60	0.73	4.61	5.57	1.82
C20:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C20:2	0.62	0.66	0.60	0.57	0.62	0.56	0.57	0.61	0.55	0.54
C22:0	0.90	0.74	0.90	0.93	0.78	0.99	1.03	0.88	0.94	1.07
C20:3	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C20:4	2.64	2.56	2.82	2.74	2.84	3.05	3.44	2.66	2.93	3.07
C24:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C20:5	1.36	1.93	1.68	1.05	0.83	1.38	1.93	1.16	1.88	1.37
C22:5	0.60	0.66	0.63	0.56	0.00	0.57	0.77	0.68	0.80	0.00
C22:6	1.98	2.19	2.16	1.97	1.65	2.02	2.61	2.23	2.83	2.06

[1] FA = fatty acid, presented as g FA per 100 g fat.

Table A-57. Fatty acid composition of FISH foal plasma at 56 d of age

	Foal									
FA[1]	5B24	5A66	5W69	5B31	5A59	5B14	5B33	5B46	5B01	5B47
C8:0	OT[2]	NA[3]	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C10:0	OT	NA	0.40	0.00	0.46	0.42	0.00	0.73	0.44	0.00
C12:0	OT	NA	0.79	0.99	1.15	0.91	0.47	2.09	1.02	0.00
C14:0	OT	NA	0.70	0.00	0.85	0.84	0.39	1.51	0.58	0.00
C14:1	OT	NA	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C16:0	OT	NA	16.91	16.81	17.12	16.31	17.16	18.86	16.73	16.27
C16:1	OT	NA	1.74	2.23	2.10	1.86	1.69	2.91	1.18	1.40
C17:0	OT	NA	0.57	0.00	0.50	0.55	0.46	0.50	0.68	0.74
C17:1	OT	NA	0.38	0.68	0.41	0.45	0.49	0.00	0.00	0.65
C18:0	OT	NA	19.46	20.60	19.60	19.28	18.29	18.23	20.34	20.85
C18:1	OT	NA	8.40	7.79	7.16	6.65	6.57	8.71	7.22	7.31
C18:2	OT	NA	39.45	39.95	40.52	40.50	43.35	36.19	39.89	41.66
C20:0	OT	NA	0.36	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C18:3	OT	NA	4.95	4.06	4.23	4.68	3.04	3.70	2.76	4.02
C20:1	OT	NA	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C20:2	OT	NA	0.67	0.63	0.74	0.68	0.52	0.57	0.74	0.63
C22:0	OT	NA	0.64	1.04	0.77	0.76	0.82	0.71	0.90	0.90
C20:3	OT	NA	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C20:4	OT	NA	2.10	2.76	2.34	2.59	2.64	2.18	3.10	2.48
C24:1	OT	NA	0.00	0.00	0.00	0.00	0.49	0.00	0.00	0.00
C20:5	OT	NA	0.66	0.66	0.58	1.17	1.17	0.97	1.52	1.03
C22:5	OT	NA	0.51	0.00	0.47	0.54	0.45	0.48	0.66	0.50
C22:6	OT	NA	1.31	1.81	1.00	1.81	2.00	1.67	2.22	1.57

[1] FA = fatty acid, presented as g FA per 100 g fat.
[2] OT = off trial.
[3] NA = not analyzed.

Table A-58. Fatty acid composition of FISH foal plasma at 84 d of age

	Foal									
FA[1]	5B24	5A66	5W69	5B31	5A59	5B14	5B33	5B46	5B01	5B47
C8:0	OT[2]	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C10:0	OT	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C12:0	OT	0.56	0.59	0.00	0.77	0.64	0.00	0.84	0.00	0.00
C14:0	OT	0.00	0.00	0.00	0.00	0.00	0.00	0.65	0.00	0.00
C14:1	OT	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C16:0	OT	15.86	17.56	17.08	17.03	15.02	16.07	18.32	17.13	16.70
C16:1	OT	2.08	1.59	1.46	1.60	1.55	1.26	2.82	1.57	1.41
C17:0	OT	0.48	0.53	0.00	0.58	0.54	0.57	0.54	0.63	0.72
C17:1	OT	0.00	0.46	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C18:0	OT	18.10	19.13	20.61	21.08	22.63	21.14	18.12	20.41	20.51
C18:1	OT	8.28	8.30	7.36	7.09	6.92	6.38	8.95	8.30	7.23
C18:2	OT	44.60	41.21	45.51	43.17	42.93	44.78	36.87	40.32	45.85
C20:0	OT	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C18:3	OT	5.63	3.99	2.39	4.44	3.97	2.34	6.12	4.37	3.43
C20:1	OT	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C20:2	OT	0.51	0.63	0.61	0.81	0.53	0.56	0.62	0.55	0.00
C22:0	OT	0.80	0.81	0.00	0.00	0.95	0.00	0.78	0.80	0.00
C20:3	OT	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C20:4	OT	1.76	2.20	2.44	2.52	2.40	2.88	2.11	2.51	2.17
C24:1	OT	0.00	0.54	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C20:5	OT	0.46	0.56	0.80	0.00	0.63	1.52	0.84	0.96	0.69
C22:5	OT	0.00	0.59	0.00	0.00	0.00	0.60	0.66	0.53	0.00
C22:6	OT	0.88	1.32	1.74	0.90	1.29	1.90	1.76	1.91	1.29

[1] FA = fatty acid, presented as g FA per 100 g fat.
[2] OT = off trial.

Fatty Acid Composition of Plasma from FLAX Foals

Table A-59. Fatty acid composition of FLAX foal plasma at birth

	Foal										
FA[1]	5B21	5B32	5C2	5A62	5B28	5C6	5A65	5B44	5C1	5B19	5B06
C8:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C10:0	0.00	0.00	0.00	0.42	0.00	0.44	0.00	0.00	0.00	0.00	0.00
C12:0	0.88	0.00	0.86	1.07	0.51	1.12	0.00	0.00	1.02	0.58	0.00
C14:0	0.53	0.00	0.48	0.93	0.00	0.45	0.00	0.00	0.92	0.00	0.00
C14:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C16:0	15.65	17.24	16.38	17.62	16.83	17.06	14.18	16.14	17.89	15.21	15.75
C16:1	2.04	1.85	1.73	2.72	1.66	1.77	1.56	1.49	2.60	1.21	1.78
C17:0	0.57	0.00	0.62	0.56	0.60	0.53	0.53	0.68	0.68	0.51	0.52
C17:1	0.41	0.00	0.60	0.00	0.00	0.00	0.00	0.52	0.00	0.00	0.00
C18:0	19.01	18.89	20.06	18.71	21.32	20.23	22.30	22.19	20.71	21.43	23.77
C18:1	8.53	9.46	8.61	8.32	7.75	8.14	7.08	8.02	8.26	7.99	6.63
C18:2	40.85	46.29	40.84	41.05	43.07	42.88	44.95	43.10	39.37	46.42	44.11
C20:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C18:3	7.92	4.47	6.08	6.46	3.49	4.26	5.97	4.19	4.23	3.92	3.03
C20:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C20:2	0.76	0.00	0.63	0.65	0.60	0.72	0.64	0.68	0.61	0.52	0.53
C22:0	0.57	0.00	0.67	0.00	0.00	0.00	0.85	0.72	0.65	0.00	0.83
C20:3	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C20:4	1.80	1.81	2.01	1.50	2.06	1.93	1.93	2.25	2.17	2.20	2.43
C24:1	0.46	0.00	0.42	0.00	0.50	0.48	0.00	0.00	0.45	0.00	0.62
C20:5	0.00	0.00	0.00	0.00	0.37	0.00	0.00	0.00	0.00	0.00	0.00
C22:5	0.00	0.00	0.00	0.00	0.48	0.00	0.00	0.00	0.45	0.00	0.00
C22:6	0.00	0.00	0.00	0.00	0.76	0.00	0.00	0.00	0.00	0.00	0.00

[1] FA = fatty acid, presented as g FA per 100 g fat.

Table A-60. Fatty acid composition of FLAX foal plasma at 14 d of age

	Foal										
FA[1]	5B21	5B32	5C2	5A62	5B28	5C6	5A65	5B44	5C1	5B19	5B06
C8:0	0.00	0.00	NA[2]	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C10:0	0.53	0.59	NA	0.99	0.50	0.00	0.40	0.54	0.25	0.00	0.53
C12:0	1.17	1.42	NA	1.88	1.14	0.80	1.03	1.14	0.44	0.63	1.17
C14:0	1.34	1.49	NA	1.73	1.28	1.00	1.06	1.40	0.75	0.81	1.34
C14:1	0.00	0.00	NA	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C16:0	16.49	17.45	NA	16.30	16.87	16.70	15.21	16.58	17.30	17.94	16.49
C16:1	2.24	2.02	NA	2.21	2.17	1.29	1.53	2.08	2.21	1.35	2.24
C17:0	0.51	0.52	NA	0.42	0.57	0.46	0.53	0.63	0.53	0.55	0.51
C17:1	0.00	0.00	NA	0.00	0.37	0.38	0.00	0.00	0.18	0.00	0.00
C18:0	21.81	22.61	NA	22.70	21.98	23.19	24.66	22.56	23.05	22.52	21.81
C18:1	9.97	8.62	NA	8.24	7.92	7.40	6.67	7.95	11.00	8.92	9.97
C18:2	39.92	38.54	NA	39.37	39.26	42.94	41.43	39.85	36.37	38.65	39.92
C20:0	0.39	0.00	NA	0.00	0.31	0.39	0.38	0.32	0.22	0.38	0.39
C18:3	1.73	3.74	NA	3.76	1.73	1.27	2.96	3.26	1.59	1.12	1.73
C20:1	0.00	0.00	NA	0.00	0.00	0.00	0.00	0.00	0.30	0.00	0.00
C20:2	0.80	0.71	NA	0.67	0.58	0.64	0.79	0.65	0.57	0.56	0.80
C22:0	0.45	0.53	NA	0.39	0.51	0.40	0.63	0.46	0.65	0.93	0.45
C20:3	0.00	0.00	NA	0.00	0.00	0.00	0.00	2.26	0.00	0.00	0.00
C20:4	2.29	1.30	NA	0.90	2.61	2.33	1.35	0.00	3.45	3.93	2.29
C24:1	0.35	0.45	NA	0.00	0.00	0.37	0.00	0.32	0.42	0.00	0.35
C20:5	0.00	0.00	NA	0.00	0.75	0.00	0.37	0.00	0.00	0.34	0.00
C22:5	0.00	0.00	NA	0.00	0.37	0.00	0.37	0.00	0.27	0.38	0.00
C22:6	0.00	0.00	NA	0.45	1.05	0.44	0.63	0.00	0.46	1.01	0.00

[1] FA = fatty acid, presented as g FA per 100 g fat.

[2] NA = not analyzed.

Table A-61. Fatty acid composition of FLAX foal plasma at 28 d of age

	Foal										
FA[1]	5B21	5B32	5C2	5A62	5B28	5C6	5A65	5B44	5C1	5B19	5B06
C8:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C10:0	0.00	0.53	0.62	1.00	0.57	0.71	0.60	0.00	0.30	0.62	0.45
C12:0	0.82	1.56	1.63	2.33	1.42	2.01	1.45	0.65	0.67	1.34	1.25
C14:0	0.95	1.27	1.35	1.99	1.33	1.91	1.32	0.80	0.93	1.39	0.96
C14:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C16:0	16.32	16.78	16.38	17.04	17.67	17.90	16.33	15.17	17.01	17.25	16.21
C16:1	2.34	2.39	2.04	2.37	2.33	2.35	2.23	2.00	1.94	1.54	1.74
C17:0	0.00	0.49	0.50	0.49	0.49	0.00	0.44	0.47	0.57	0.59	0.46
C17:1	0.48	0.00	0.44	0.43	0.35	0.00	0.00	0.45	0.21	0.41	0.48
C18:0	20.51	18.76	21.22	19.97	19.52	18.87	21.13	20.69	22.55	18.43	22.98
C18:1	8.72	9.39	7.66	7.21	8.47	7.91	7.30	8.38	9.64	8.51	7.02
C18:2	42.53	39.38	41.96	37.59	38.60	40.42	40.40	40.44	39.63	39.92	39.55
C20:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.30	0.31	0.00	0.00
C18:3	3.20	5.69	2.50	5.85	2.36	5.00	2.42	5.66	1.81	5.36	5.11
C20:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C20:2	0.79	0.71	0.82	0.59	0.54	0.00	0.72	0.67	0.76	0.56	0.76
C22:0	0.83	0.68	0.70	0.63	0.74	0.64	0.72	0.66	0.58	0.58	0.81
C20:3	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C20:4	2.50	2.37	2.19	0.49	2.47	2.29	2.38	2.22	2.50	2.32	2.20
C24:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C20:5	0.00	0.00	0.00	0.60	0.88	0.00	0.87	0.54	0.00	0.00	0.00
C22:5	0.00	0.00	0.00	0.47	0.58	0.00	0.51	0.34	0.34	0.39	0.00
C22:6	0.00	0.00	0.00	0.96	1.68	0.00	1.18	0.56	0.24	0.80	0.00

[1] FA = fatty acid, presented as g FA per 100 g fat.

Table A-62. Fatty acid composition of FLAX foal plasma at 56 d of age

	Foal										
FA[1]	5B21	5B32	5C2	5A62	5B28	5C6	5A65	5B44	5C1	5B19	5B06
C8:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA[2]
C10:0	0.00	0.00	0.00	0.65	0.00	0.41	0.58	0.00	0.53	0.00	NA
C12:0	0.46	0.00	0.50	1.63	0.62	1.24	1.36	0.55	1.24	0.51	NA
C14:0	0.00	0.00	0.00	0.94	0.00	1.11	0.89	0.00	0.97	0.50	NA
C14:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA
C16:0	16.26	16.01	16.08	16.74	16.18	17.68	15.61	15.20	16.00	15.87	NA
C16:1	1.76	1.74	1.71	2.23	2.01	2.09	1.71	1.29	1.98	1.30	NA
C17:0	0.55	0.00	0.56	0.51	0.50	0.42	0.44	0.58	0.54	0.53	NA
C17:1	0.52	0.83	0.67	0.00	0.55	0.43	0.00	0.58	0.42	0.36	NA
C18:0	20.72	20.92	20.15	19.53	20.90	17.84	20.94	22.29	20.52	18.88	NA
C18:1	8.91	9.54	8.35	7.35	8.31	8.23	7.59	7.74	7.31	8.45	NA
C18:2	41.80	43.14	41.23	38.77	40.50	43.01	42.32	43.74	39.23	45.51	NA
C20:0	0.36	0.00	0.36	0.00	0.37	0.00	0.38	0.00	0.33	0.00	NA
C18:3	4.59	2.49	6.12	7.96	3.51	3.66	4.97	4.11	6.59	5.18	NA
C20:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA
C20:2	0.88	0.73	0.85	0.64	0.73	0.55	0.62	0.61	0.68	0.46	NA
C22:0	0.68	1.02	0.68	0.62	0.79	0.66	0.75	0.68	0.62	0.59	NA
C20:3	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA
C20:4	2.12	2.89	2.33	1.91	2.43	2.22	1.83	2.20	2.10	2.08	NA
C24:1	0.40	0.70	0.41	0.00	0.52	0.46	0.00	0.43	0.53	0.40	NA
C20:5	0.00	0.00	0.00	0.00	0.39	0.00	0.00	0.00	0.00	0.00	NA
C22:5	0.00	0.00	0.00	0.00	0.51	0.00	0.00	0.00	0.39	0.38	NA
C22:6	0.00	0.00	0.00	0.52	1.17	0.00	0.00	0.00	0.00	0.00	NA

[1] FA = fatty acid, presented as g FA per 100 g fat.

[2] NA = not analyzed.

Table A-63. Fatty acid composition of FLAX foal plasma at 84 d of age

	Foal										
FA[1]	5B21	5B32	5C2	5A62	5B28	5C6	5A65	5B44	5C1	5B19	5B06
C8:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C10:0	0.00	0.00	0.00	0.42	0.00	0.44	0.00	0.00	0.00	0.00	0.00
C12:0	0.88	0.00	0.86	1.07	0.51	1.12	0.00	0.00	1.02	0.58	0.00
C14:0	0.53	0.00	0.48	0.93	0.00	0.45	0.00	0.00	0.92	0.00	0.00
C14:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C16:0	15.65	17.24	16.38	17.62	16.83	17.06	14.18	16.14	17.89	15.21	15.75
C16:1	2.04	1.85	1.73	2.72	1.66	1.77	1.56	1.49	2.60	1.21	1.78
C17:0	0.57	0.00	0.62	0.56	0.60	0.53	0.53	0.68	0.68	0.51	0.52
C17:1	0.41	0.00	0.60	0.00	0.00	0.00	0.00	0.52	0.00	0.00	0.00
C18:0	19.01	18.89	20.06	18.71	21.32	20.23	22.30	22.19	20.71	21.43	23.77
C18:1	8.53	9.46	8.61	8.32	7.75	8.14	7.08	8.02	8.26	7.99	6.63
C18:2	40.85	46.29	40.84	41.05	43.07	42.88	44.95	43.10	39.37	46.42	44.11
C20:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C18:3	7.92	4.47	6.08	6.46	3.49	4.26	5.97	4.19	4.23	3.92	3.03
C20:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C20:2	0.76	0.00	0.63	0.65	0.60	0.72	0.64	0.68	0.61	0.52	0.53
C22:0	0.57	0.00	0.67	0.00	0.00	0.00	0.85	0.72	0.65	0.00	0.83
C20:3	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C20:4	1.80	1.81	2.01	1.50	2.06	1.93	1.93	2.25	2.17	2.20	2.43
C24:1	0.46	0.00	0.42	0.00	0.50	0.48	0.00	0.00	0.45	0.00	0.62
C20:5	0.00	0.00	0.00	0.00	0.37	0.00	0.00	0.00	0.00	0.00	0.00
C22:5	0.00	0.00	0.00	0.00	0.48	0.00	0.00	0.00	0.45	0.00	0.00
C22:6	0.00	0.00	0.00	0.00	0.76	0.00	0.00	0.00	0.00	0.00	0.00

[1] FA = fatty acid, presented as g FA per 100 g fat.

Fatty Acid Composition of Plasma from CON Foals

Table A-64. Fatty acid composition of CON foal plasma at birth

	Foal										
FA[1]	5B29	5B36	5A64	5B41	5B13	5B45	5A54	5B18	5C4	5B26	5A61
C8:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C10:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C12:0	0.00	1.05	0.00	0.00	1.03	0.00	0.53	0.65	0.73	0.00	0.00
C14:0	0.00	0.57	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C14:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C16:0	15.42	18.10	16.14	16.13	16.63	14.85	16.41	16.75	16.57	16.47	16.32
C16:1	1.66	3.15	1.36	1.23	1.65	1.44	1.20	1.82	1.61	1.40	1.30
C17:0	0.70	0.00	0.57	0.61	0.00	0.00	0.49	0.57	0.63	0.00	0.63
C17:1	0.00	0.00	0.00	0.00	0.00	0.65	0.00	0.00	0.00	0.53	0.00
C18:0	21.17	17.02	20.26	22.44	22.68	21.99	22.03	21.84	21.01	21.54	21.50
C18:1	7.65	9.30	9.09	8.28	7.99	7.04	6.37	7.22	8.11	7.70	9.23
C18:2	45.74	40.90	44.98	44.04	44.12	47.20	46.88	44.45	44.47	45.63	43.74
C20:0	4.70	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C18:3	0.00	7.57	4.39	3.52	3.08	4.13	3.00	3.56	3.95	2.92	3.63
C20:1	0.66	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C20:2	0.00	0.79	0.68	0.69	0.65	0.00	0.53	0.59	0.78	0.65	0.00
C22:0	0.00	0.00	0.62	0.00	0.00	0.00	0.00	0.00	0.00	0.88	0.77
C20:3	2.30	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C20:4	0.00	1.54	1.91	2.42	2.16	2.70	1.97	2.06	2.13	2.29	2.33
C24:1	0.00	0.00	0.00	0.64	0.00	0.00	0.57	0.50	0.00	0.00	0.55
C20:5	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C22:5	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C22:6	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00

[1] FA = fatty acid, presented as g FA per 100 g fat.

Table A-65. Fatty acid composition of CON foal plasma at 14 d of age

	Foal										
FA[1]	5B29	5B36	5A64	5B41	5B13	5B45	5A54	5B18	5C4	5B26	5A61
C8:0	NA[2]	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C10:0	NA	0.00	0.57	0.65	0.00	0.00	0.54	0.80	0.50	0.00	0.81
C12:0	NA	0.76	1.28	1.57	0.84	0.84	1.14	1.89	1.19	0.00	1.56
C14:0	NA	1.05	1.76	1.75	0.92	1.17	1.34	2.00	1.37	0.53	2.09
C14:1	NA	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C16:0	NA	17.78	18.82	16.55	17.05	17.04	18.04	17.47	16.30	16.75	18.44
C16:1	NA	2.81	2.98	2.19	2.32	1.93	2.21	2.12	1.79	2.50	2.61
C17:0	NA	0.42	0.39	0.44	0.00	0.61	0.45	0.51	0.53	0.47	0.61
C17:1	NA	0.00	0.28	0.00	0.00	0.36	0.00	0.26	0.33	0.40	0.19
C18:0	NA	21.92	18.61	21.24	22.91	23.85	22.01	22.30	21.99	22.22	19.89
C18:1	NA	9.92	13.00	8.70	9.07	7.57	7.22	8.14	9.33	10.90	10.89
C18:2	NA	39.83	37.54	38.97	42.49	40.98	39.27	37.06	40.63	40.41	36.97
C20:0	NA	0.35	0.00	0.39	0.00	0.35	0.00	0.33	0.00	0.00	0.29
C18:3	NA	2.68	1.22	3.91	2.12	1.16	2.20	3.08	3.36	1.06	1.70
C20:1	NA	0.00	0.36	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C20:2	NA	0.76	0.71	0.65	0.66	0.68	0.50	0.76	0.82	0.83	0.65
C22:0	NA	0.51	0.50	0.75	0.48	0.63	0.67	0.47	0.50	0.88	0.66
C20:3	NA	0.00	0.00	1.97	0.00	0.00	0.00	0.29	0.00	0.00	0.00
C20:4	NA	1.21	1.42	0.00	1.15	2.83	1.52	2.20	1.34	2.65	2.15
C24:1	NA	0.00	0.30	0.28	0.00	0.00	0.42	0.33	0.00	0.41	0.24
C20:5	NA	0.00	0.00	0.00	0.00	0.00	0.86	0.00	0.00	0.00	0.00
C22:5	NA	0.00	0.00	0.00	0.00	0.00	0.49	0.00	0.00	0.00	0.25
C22:6	NA	0.00	0.27	0.00	0.00	0.00	1.13	0.00	0.00	0.00	0.00

[1] FA = fatty acid, presented as g FA per 100 g fat.

[2] NA = not analyzed.

Table A-66. Fatty acid composition of CON foal plasma at 28 d of age

	Foal										
FA[1]	5B29	5B36	5A64	5B41	5B13	5B45	5A54	5B18	5C4	5B26	5A61
C8:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C10:0	0.00	0.00	0.70	0.00	0.77	0.00	0.76	0.59	0.00	0.57	0.71
C12:0	0.00	0.64	1.41	0.36	1.89	1.18	1.75	1.62	0.70	1.60	1.99
C14:0	0.49	0.69	1.47	0.38	1.55	1.15	1.76	1.48	0.78	1.41	2.07
C14:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C16:0	15.99	17.82	16.59	16.09	18.03	16.66	18.70	16.88	14.90	17.80	19.33
C16:1	1.30	2.82	2.16	1.53	2.23	2.16	1.91	2.32	2.17	2.72	2.79
C17:0	0.61	0.00	0.46	0.49	0.00	0.48	0.48	0.38	0.51	0.39	0.54
C17:1	0.49	0.63	0.00	0.50	0.48	0.43	0.00	0.00	0.68	0.36	0.00
C18:0	22.51	21.61	19.06	21.67	22.31	20.48	20.04	22.59	19.74	21.44	19.24
C18:1	6.92	10.18	9.50	9.19	8.56	8.12	6.89	7.70	9.27	10.76	9.33
C18:2	46.02	39.29	41.19	43.94	38.65	39.93	41.65	40.39	41.71	37.88	37.64
C20:0	0.39	0.00	0.27	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C18:3	1.85	2.21	4.02	1.91	1.96	5.37	1.60	3.03	5.28	0.86	2.59
C20:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C20:2	0.74	0.70	0.79	0.76	0.64	0.70	0.57	0.61	0.63	0.76	0.57
C22:0	0.00	0.78	0.53	0.74	0.66	0.75	0.77	0.00	0.84	0.68	0.66
C20:3	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C20:4	2.69	2.64	1.84	2.44	2.26	2.60	2.16	2.13	2.79	2.36	2.54
C24:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C20:5	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C22:5	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C22:6	0.00	0.00	0.00	0.00	0.00	0.00	0.95	0.00	0.00	0.40	0.00

[1] FA = fatty acid, presented as g FA per 100 g fat.

Table A-67. Fatty acid composition of CON foal plasma at 56 d of age

	Foal										
FA[1]	5B29	5B36	5A64	5B41	5B13	5B45	5A54	5B18	5C4	5B26	5A61
C8:0	0.00	NA[2]	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C10:0	0.00	NA	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.59
C12:0	0.00	NA	0.76	0.66	0.66	1.00	0.66	0.62	0.80	0.34	1.26
C14:0	0.00	NA	0.59	0.00	0.00	0.97	0.47	0.00	0.56	0.47	1.31
C14:1	0.00	NA	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C16:0	15.29	NA	17.36	16.93	16.72	15.70	16.92	14.69	17.09	17.18	18.24
C16:1	1.53	NA	1.93	1.39	2.11	2.47	1.44	1.52	1.93	2.09	2.36
C17:0	0.68	NA	0.46	0.54	0.00	0.47	0.52	0.55	0.56	0.48	0.48
C17:1	0.67	NA	0.00	0.00	0.00	0.50	0.00	0.00	0.00	0.42	0.00
C18:0	21.06	NA	20.16	21.98	21.25	20.10	20.39	22.96	20.61	18.62	19.11
C18:1	8.21	NA	8.36	8.55	8.85	7.96	6.84	6.63	9.07	9.12	8.87
C18:2	43.84	NA	42.84	42.53	42.73	41.15	43.62	44.23	40.59	43.72	39.96
C20:0	0.38	NA	0.00	0.00	0.00	0.00	0.40	0.00	0.00	0.00	0.00
C18:3	3.88	NA	3.78	3.46	3.40	5.14	4.27	3.98	4.22	1.77	4.03
C20:1	0.00	NA	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C20:2	0.77	NA	0.73	0.71	0.71	0.56	0.65	0.67	0.78	0.57	0.42
C22:0	0.67	NA	0.57	0.83	0.71	0.76	0.79	0.87	0.81	0.75	0.64
C20:3	0.00	NA	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C20:4	2.56	NA	1.96	2.43	2.33	2.79	2.05	2.65	2.43	2.38	2.33
C24:1	0.45	NA	0.49	0.00	0.54	0.43	0.48	0.62	0.54	0.00	0.40
C20:5	0.00	NA	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.48	0.00
C22:5	0.00	NA	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.49	0.00
C22:6	0.00	NA	0.00	0.00	0.00	0.00	0.49	0.00	0.00	1.12	0.00

[1] FA = fatty acid, presented as g FA per 100 g fat.

[2] NA = not analyzed.

Table A-68. Fatty acid composition of CON foal plasma at 84 d of age

	Foal										
FA[1]	5B29	5B36	5A64	5B41	5B13	5B45	5A54	5B18	5C4	5B26	5A61
C8:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C10:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C12:0	0.00	1.05	0.00	0.00	1.03	0.00	0.53	0.65	0.73	0.00	0.00
C14:0	0.00	0.57	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C14:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C16:0	15.42	18.10	16.14	16.13	16.63	14.85	16.41	16.75	16.57	16.47	16.32
C16:1	1.66	3.15	1.36	1.23	1.65	1.44	1.20	1.82	1.61	1.40	1.30
C17:0	0.70	0.00	0.57	0.61	0.00	0.00	0.49	0.57	0.63	0.00	0.63
C17:1	0.00	0.00	0.00	0.00	0.00	0.65	0.00	0.00	0.00	0.53	0.00
C18:0	21.17	17.02	20.26	22.44	22.68	21.99	22.03	21.84	21.01	21.54	21.50
C18:1	7.65	9.30	9.09	8.28	7.99	7.04	6.37	7.22	8.11	7.70	9.23
C18:2	45.74	40.90	44.98	44.04	44.12	47.20	46.88	44.45	44.47	45.63	43.74
C20:0	4.70	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C18:3	0.00	7.57	4.39	3.52	3.08	4.13	3.00	3.56	3.95	2.92	3.63
C20:1	0.66	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C20:2	0.00	0.79	0.68	0.69	0.65	0.00	0.53	0.59	0.78	0.65	0.00
C22:0	0.00	0.00	0.62	0.00	0.00	0.00	0.00	0.00	0.00	0.88	0.77
C20:3	2.30	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C20:4	0.00	1.54	1.91	2.42	2.16	2.70	1.97	2.06	2.13	2.29	2.33
C24:1	0.00	0.00	0.00	0.64	0.00	0.00	0.57	0.50	0.00	0.00	0.55
C20:5	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C22:5	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C22:6	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00

[1] FA = fatty acid, presented as g FA per 100 g fat.

Fatty Acid Composition of Red Blood Cells from FISH Mares

Table A-69. Fatty acid composition of FISH mare red blood cells at 28 d before expected foaling date

	Mare									
FA[1]	B47	W69	A66	B31	A59	B33	B24	B46	B14	B01
C8:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C10:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C12:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C14:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C14:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C16:0	36.31	34.40	35.64	41.46	41.78	38.40	42.53	39.76	35.58	38.03
C16:1	1.36	0.00	1.12	0.00	0.89	0.00	0.00	1.46	0.00	0.00
C17:0	1.51	0.00	0.78	0.00	0.72	0.00	1.21	0.00	0.00	0.00
C17:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C18:0	28.07	27.93	31.50	32.67	31.75	27.01	30.05	26.66	29.82	32.13
C18:1	27.16	27.29	23.15	22.23	21.56	26.59	22.30	26.68	25.60	25.07
C18:2	5.58	10.37	7.81	3.63	3.30	8.00	3.92	5.43	9.00	4.77
C20:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C18:3	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C20:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C20:2	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C22:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C20:3	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C20:4	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C24:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C20:5	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C22:5	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C22:6	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00

[1] FA = fatty acid, presented as g FA per 100 g fat.

Table A-70. Fatty acid composition of FISH mare red blood cells at foaling

	Mare									
FA[1]	B47	W69	A66	B31	A59	B33	B24	B46	B14	B01
C8:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C10:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C12:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C14:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C14:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C16:0	36.50	39.54	18.90	41.48	28.46	45.04	38.00	45.39	42.75	35.99
C16:1	0.00	0.00	1.23	0.00	2.92	0.00	0.00	0.79	0.00	4.54
C17:0	1.21	0.00	0.64	0.00	0.35	1.52	1.74	1.55	1.43	1.42
C17:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C18:0	26.40	30.72	17.60	29.66	16.85	25.92	28.09	25.89	28.61	25.87
C18:1	29.63	24.40	22.99	24.24	33.25	22.76	26.92	21.31	22.41	25.36
C18:2	6.27	5.34	38.64	4.62	18.16	4.77	5.25	5.06	4.80	6.82
C20:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C18:3	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C20:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C20:2	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C22:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C20:3	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C20:4	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C24:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C20:5	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C22:5	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C22:6	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00

[1] FA = fatty acid, presented as g FA per 100 g fat.

Table A-71. Fatty acid composition of FISH mare red blood cells at 28 d post-foaling

	Mare									
FA[1]	B47	W69	A66	B31	A59	B33	B24	B46	B14	B01
C8:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C10:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C12:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C14:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C14:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C16:0	37.06	41.49	27.50	41.43	41.99	41.70	41.47	41.91	42.72	39.81
C16:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C17:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C17:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C18:0	25.27	28.00	21.91	29.53	33.92	26.02	28.90	25.65	29.69	29.68
C18:1	30.52	25.42	25.50	25.06	21.14	26.93	25.02	27.05	24.37	24.37
C18:2	7.15	5.09	25.09	3.98	2.95	5.34	4.61	5.39	3.22	6.15
C20:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C18:3	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C20:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C20:2	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C22:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C20:3	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C20:4	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C24:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C20:5	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C22:5	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C22:6	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00

[1] FA = fatty acid, presented as g FA per 100 g fat.

Table A-72. Fatty acid composition of FISH mare red blood cells at 56 d post-foaling

	Mare									
FA[1]	B47	W69	A66	B31	A59	B33	B24	B46	B14	B01
C8:0	0.00	0.00	0.00	0.00	0.00	0.00	OT[2]	0.00	0.00	0.00
C10:0	0.00	0.00	0.00	0.00	0.00	0.00	OT	0.00	0.00	0.00
C12:0	0.00	0.00	0.00	0.00	0.00	0.00	OT	0.00	0.00	0.00
C14:0	0.00	0.00	0.00	0.00	0.00	0.00	OT	0.00	0.00	0.00
C14:1	0.00	0.00	0.00	0.00	0.00	0.00	OT	0.00	0.00	0.00
C16:0	39.97	43.51	26.25	38.66	42.46	45.16	OT	47.13	43.29	38.41
C16:1	0.00	0.00	0.00	0.00	0.00	0.00	OT	0.00	0.00	0.00
C17:0	0.00	0.00	0.00	0.00	0.00	0.00	OT	0.00	0.00	0.00
C17:1	0.00	0.00	0.00	0.00	0.00	0.00	OT	0.00	0.00	0.00
C18:0	24.60	26.18	17.14	26.65	31.52	26.53	OT	27.00	28.59	27.55
C18:1	29.42	25.22	20.64	24.86	22.86	24.52	OT	23.09	24.60	27.98
C18:2	6.01	5.09	35.98	9.82	3.15	3.78	OT	2.78	3.51	6.07
C20:0	0.00	0.00	0.00	0.00	0.00	0.00	OT	0.00	0.00	0.00
C18:3	0.00	0.00	0.00	0.00	0.00	0.00	OT	0.00	0.00	0.00
C20:1	0.00	0.00	0.00	0.00	0.00	0.00	OT	0.00	0.00	0.00
C20:2	0.00	0.00	0.00	0.00	0.00	0.00	OT	0.00	0.00	0.00
C22:0	0.00	0.00	0.00	0.00	0.00	0.00	OT	0.00	0.00	0.00
C20:3	0.00	0.00	0.00	0.00	0.00	0.00	OT	0.00	0.00	0.00
C20:4	0.00	0.00	0.00	0.00	0.00	0.00	OT	0.00	0.00	0.00
C24:1	0.00	0.00	0.00	0.00	0.00	0.00	OT	0.00	0.00	0.00
C20:5	0.00	0.00	0.00	0.00	0.00	0.00	OT	0.00	0.00	0.00
C22:5	0.00	0.00	0.00	0.00	0.00	0.00	OT	0.00	0.00	0.00
C22:6	0.00	0.00	0.00	0.00	0.00	0.00	OT	0.00	0.00	0.00

[1] FA = fatty acid, presented as g FA per 100 g fat.
[2] OT = off trial.

Table A-73. Fatty acid composition of FISH mare red blood cells at 84 d post-foaling

	Mare									
FA[1]	B47	W69	A66	B31	A59	B33	B24	B46	B14	B01
C8:0	0.00	0.00	0.00	0.00	0.00	0.00	OT[2]	0.00	0.00	NA[3]
C10:0	0.00	0.00	0.00	0.00	0.00	0.00	OT	0.00	0.00	NA
C12:0	0.00	0.00	0.00	0.00	0.00	0.00	OT	0.00	0.00	NA
C14:0	0.00	0.00	0.00	0.00	0.00	0.00	OT	0.00	0.00	NA
C14:1	0.00	0.00	0.00	0.00	0.00	0.00	OT	0.00	0.00	NA
C16:0	37.20	43.41	34.88	42.89	42.56	44.01	OT	45.73	46.00	NA
C16:1	0.00	0.00	0.00	0.00	0.00	0.00	OT	0.00	0.00	NA
C17:0	0.00	0.00	0.00	0.00	0.00	0.00	OT	0.00	0.00	NA
C17:1	0.00	0.00	0.00	0.00	0.00	0.00	OT	0.00	0.00	NA
C18:0	23.47	28.63	24.69	27.32	28.53	22.39	OT	26.81	29.70	NA
C18:1	29.84	22.54	26.95	23.74	22.36	21.38	OT	23.90	24.30	NA
C18:2	9.49	5.42	13.48	6.04	6.55	12.22	OT	3.56	0.00	NA
C20:0	0.00	0.00	0.00	0.00	0.00	0.00	OT	0.00	0.00	NA
C18:3	0.00	0.00	0.00	0.00	0.00	0.00	OT	0.00	0.00	NA
C20:1	0.00	0.00	0.00	0.00	0.00	0.00	OT	0.00	0.00	NA
C20:2	0.00	0.00	0.00	0.00	0.00	0.00	OT	0.00	0.00	NA
C22:0	0.00	0.00	0.00	0.00	0.00	0.00	OT	0.00	0.00	NA
C20:3	0.00	0.00	0.00	0.00	0.00	0.00	OT	0.00	0.00	NA
C20:4	0.00	0.00	0.00	0.00	0.00	0.00	OT	0.00	0.00	NA
C24:1	0.00	0.00	0.00	0.00	0.00	0.00	OT	0.00	0.00	NA
C20:5	0.00	0.00	0.00	0.00	0.00	0.00	OT	0.00	0.00	NA
C22:5	0.00	0.00	0.00	0.00	0.00	0.00	OT	0.00	0.00	NA
C22:6	0.00	0.00	0.00	0.00	0.00	0.00	OT	0.00	0.00	NA

[1] FA = fatty acid, presented as g FA per 100 g fat.
[2] OT = off trial.
[3] NA = not analyzed.

Fatty Acid Composition of Red Blood Cells from FLAX Mares

Table A-74. Fatty acid composition of FLAX mare red blood cells at 28 d before expected foaling date

	Mare										
FA[1]	B21	A62	B32	C2	C6	A65	B06	C1	B44	B28	B19
C8:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C10:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C12:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C14:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C14:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C16:0	40.27	40.26	43.51	43.75	46.31	35.36	35.70	40.02	35.46	42.09	32.74
C16:1	0.00	0.00	0.67	0.00	0.00	1.37	0.00	0.00	1.46	0.00	1.49
C17:0	0.00	0.00	0.68	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.95
C17:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C18:0	32.74	30.75	32.62	30.62	29.27	27.00	24.06	32.00	28.99	33.70	28.21
C18:1	23.48	25.35	19.05	23.03	21.33	27.82	25.43	23.73	26.92	21.13	24.11
C18:2	3.51	3.64	3.46	2.59	3.10	8.44	14.81	4.25	7.17	3.08	12.50
C20:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C18:3	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C20:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C20:2	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C22:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C20:3	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C20:4	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C24:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C20:5	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C22:5	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C22:6	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00

[1] FA = fatty acid, presented as g FA per 100 g fat.

Table A-75. Fatty acid composition of FLAX mare red blood cells at foaling

	Mare										
FA[1]	B21	A62	B32	C2	C6	A65	B06	C1	B44	B28	B19
C8:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA[2]	0.00	0.00	0.00
C10:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA	0.00	0.00	0.00
C12:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA	0.00	0.00	0.00
C14:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA	0.00	0.00	0.00
C14:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA	0.00	0.00	0.00
C16:0	37.71	36.31	42.21	42.32	48.96	29.40	47.08	NA	38.97	37.68	42.97
C16:1	2.54	0.00	0.00	1.86	0.00	1.60	0.00	NA	0.00	2.21	0.00
C17:0	1.26	1.67	1.32	1.64	1.24	1.04	1.71	NA	1.42	0.00	0.00
C17:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA	0.00	0.00	0.00
C18:0	28.72	31.15	28.54	28.64	25.18	24.67	24.85	NA	28.48	29.31	30.95
C18:1	25.39	27.01	23.22	21.73	21.31	25.27	22.47	NA	24.57	25.24	21.14
C18:2	4.36	3.86	4.72	3.80	3.31	18.02	3.90	NA	6.56	5.56	4.94
C20:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA	0.00	0.00	0.00
C18:3	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA	0.00	0.00	0.00
C20:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA	0.00	0.00	0.00
C20:2	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA	0.00	0.00	0.00
C22:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA	0.00	0.00	0.00
C20:3	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA	0.00	0.00	0.00
C20:4	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA	0.00	0.00	0.00
C24:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA	0.00	0.00	0.00
C20:5	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA	0.00	0.00	0.00
C22:5	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA	0.00	0.00	0.00
C22:6	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA	0.00	0.00	0.00

[1] FA = fatty acid, presented as g FA per 100 g fat.

[2] NA = not analyzed.

Table A-76. Fatty acid composition of FLAX mare red blood cells at 28 d post-foaling

	Mare										
FA[1]	B21	A62	B32	C2	C6	A65	B06	C1	B44	B28	B19
C8:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C10:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C12:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C14:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C14:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C16:0	44.13	40.75	41.27	45.81	45.32	40.44	43.21	38.43	39.97	41.75	39.81
C16:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C17:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C17:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C18:0	30.84	28.58	27.45	25.17	26.94	30.23	25.56	30.72	31.88	30.78	30.89
C18:1	22.13	25.82	25.86	23.98	23.73	25.27	25.87	25.87	24.48	23.41	23.88
C18:2	2.90	4.85	5.43	5.04	4.01	4.07	5.36	4.98	3.67	4.06	5.42
C20:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C18:3	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C20:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C20:2	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C22:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C20:3	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C20:4	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C24:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C20:5	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C22:5	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C22:6	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00

[1] FA = fatty acid, presented as g FA per 100 g fat.

Table A-77. Fatty acid composition of FLAX mare red blood cells at 56 d post-foaling

	Mare										
FA[1]	B21	A62	B32	C2	C6	A65	B06	C1	B44	B28	B19
C8:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C10:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C12:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C14:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C14:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C16:0	45.97	32.68	42.37	48.00	50.33	39.28	46.37	39.68	39.32	53.79	43.24
C16:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C17:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C17:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C18:0	30.94	21.34	28.87	28.47	26.03	29.09	24.85	27.98	30.22	35.86	28.21
C18:1	23.09	28.73	24.17	23.53	23.65	27.65	24.98	26.35	26.65	5.60	24.97
C18:2	0.00	17.25	4.59	0.00	0.00	3.98	3.80	5.99	3.80	4.75	3.58
C20:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C18:3	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C20:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C20:2	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C22:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C20:3	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C20:4	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C24:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C20:5	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C22:5	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C22:6	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00

[1] FA = fatty acid, presented as g FA per 100 g fat.

Table A-78. Fatty acid composition of FLAX mare red blood cells at 84 d post-foaling

	Mare										
FA[1]	B21	A62	B32	C2	C6	A65	B06	C1	B44	B28	B19
C8:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA[2]	0.00	0.00	0.00
C10:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA	0.00	0.00	0.00
C12:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA	0.00	0.00	0.00
C14:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA	0.00	0.00	0.00
C14:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA	0.00	0.00	0.00
C16:0	40.47	38.78	36.96	46.98	46.48	38.12	45.57	NA	36.21	42.62	35.07
C16:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA	0.00	0.00	0.00
C17:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA	0.00	0.00	0.00
C17:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA	0.00	0.00	0.00
C18:0	29.08	24.59	25.63	26.79	24.75	29.02	25.86	NA	27.89	29.42	26.34
C18:1	24.47	27.66	27.95	22.95	23.74	27.11	23.91	NA	29.48	23.48	28.44
C18:2	5.98	8.97	9.46	3.29	5.03	5.75	4.66	NA	6.43	4.48	10.15
C20:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA	0.00	0.00	0.00
C18:3	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA	0.00	0.00	0.00
C20:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA	0.00	0.00	0.00
C20:2	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA	0.00	0.00	0.00
C22:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA	0.00	0.00	0.00
C20:3	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA	0.00	0.00	0.00
C20:4	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA	0.00	0.00	0.00
C24:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA	0.00	0.00	0.00
C20:5	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA	0.00	0.00	0.00
C22:5	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA	0.00	0.00	0.00
C22:6	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA	0.00	0.00	0.00

[1] FA = fatty acid, presented as g FA per 100 g fat.

[2] NA = not analyzed.

Fatty Acid Composition of Red Blood Cells from CON Mares

Table A-79. Fatty acid composition of CON mare red blood cells at 28 d before expected foaling date

	Mare										
FA[1]	B29	B36	B13	B41	B45	A54	C4	A64	B18	B26	A61
C8:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C10:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C12:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C14:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C14:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C16:0	44.05	47.94	41.03	32.81	50.39	45.04	36.77	42.78	42.15	37.59	35.88
C16:1	0.00	0.97	0.00	1.43	1.05	0.00	0.00	1.48	0.00	1.95	1.30
C17:0	0.00	1.44	0.00	1.07	1.71	0.00	0.00	1.22	0.00	0.00	1.36
C17:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C18:0	29.84	38.63	32.87	27.69	34.36	29.09	31.15	29.98	32.99	27.69	29.63
C18:1	23.58	7.30	22.62	26.69	7.36	21.93	26.28	21.52	21.76	25.52	24.55
C18:2	2.54	3.71	3.48	10.31	5.14	3.94	5.80	3.02	3.11	7.25	7.28
C20:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C18:3	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C20:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C20:2	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C22:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C20:3	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C20:4	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C24:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C20:5	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C22:5	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C22:6	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00

[1] FA = fatty acid, presented as g FA per 100 g fat.

Table A-80. Fatty acid composition of CON mare red blood cells at foaling

	Mare										
FA[1]	B29	B36	B13	B41	B45	A54	C4	A64	B18	B26	A61
C8:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA[2]	0.00	0.00	0.00
C10:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA	0.00	0.00	0.00
C12:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA	0.00	0.00	0.00
C14:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA	0.00	0.00	0.00
C14:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA	0.00	0.00	0.00
C16:0	39.73	40.84	39.47	42.14	36.04	45.00	45.03	NA	40.81	39.32	36.17
C16:1	0.00	0.00	0.00	0.00	1.45	1.36	5.05	NA	0.00	4.25	2.21
C17:0	1.38	1.26	1.54	1.44	1.40	1.28	0.00	NA	1.59	0.64	1.47
C17:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA	0.00	0.00	0.00
C18:0	27.23	31.34	31.86	24.69	28.79	25.93	17.65	NA	30.54	17.41	27.41
C18:1	26.69	22.43	23.51	24.62	26.83	19.70	30.50	NA	23.98	32.08	26.02
C18:2	4.97	4.13	3.63	7.10	5.49	6.73	1.76	NA	3.08	6.29	6.71
C20:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA	0.00	0.00	0.00
C18:3	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA	0.00	0.00	0.00
C20:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA	0.00	0.00	0.00
C20:2	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA	0.00	0.00	0.00
C22:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA	0.00	0.00	0.00
C20:3	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA	0.00	0.00	0.00
C20:4	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA	0.00	0.00	0.00
C24:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA	0.00	0.00	0.00
C20:5	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA	0.00	0.00	0.00
C22:5	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA	0.00	0.00	0.00
C22:6	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA	0.00	0.00	0.00

[1] FA = fatty acid, presented as g FA per 100 g fat.

[2] NA = not analyzed.

Table A-81. Fatty acid composition of CON mare red blood cells at 28 d post-foaling

	Mare										
FA[1]	B29	B36	B13	B41	B45	A54	C4	A64	B18	B26	A61
C8:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C10:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C12:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C14:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C14:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C16:0	41.84	41.31	37.11	39.50	41.12	42.73	40.43	40.78	41.81	44.04	39.24
C16:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C17:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C17:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C18:0	29.35	30.84	31.04	28.48	29.33	25.64	32.74	29.13	30.28	28.88	27.47
C18:1	25.55	24.17	27.33	26.56	25.22	25.16	23.35	26.04	25.02	21.99	27.72
C18:2	3.26	3.68	4.53	5.45	4.34	6.47	3.48	4.05	2.89	5.08	5.57
C20:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C18:3	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C20:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C20:2	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C22:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C20:3	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C20:4	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C24:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C20:5	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C22:5	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C22:6	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00

[1] FA = fatty acid, presented as g FA per 100 g fat.

Table A-82. Fatty acid composition of CON mare red blood cells at 56 d post-foaling

	Mare										
FA[1]	B29	B36	B13	B41	B45	A54	C4	A64	B18	B26	A61
C8:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C10:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C12:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C14:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C14:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C16:0	41.52	41.50	38.15	37.78	43.13	42.30	40.38	48.30	50.47	46.87	49.18
C16:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C17:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C17:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C18:0	29.47	30.10	30.86	27.39	28.67	27.40	31.29	29.47	25.72	28.59	24.26
C18:1	24.29	23.99	25.96	29.46	23.41	23.92	24.17	22.24	23.81	21.05	22.18
C18:2	4.73	4.41	5.03	5.36	4.79	6.39	4.16	0.00	0.00	3.48	4.38
C20:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C18:3	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C20:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C20:2	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C22:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C20:3	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C20:4	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C24:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C20:5	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C22:5	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C22:6	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00

[1] FA = fatty acid, presented as g FA per 100 g fat.

Table A-83. Fatty acid composition of CON mare red blood cells at 84 d post-foaling

	Mare										
FA[1]	B29	B36	B13	B41	B45	A54	C4	A64	B18	B26	A61
C8:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C10:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C12:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C14:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C14:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C16:0	39.00	38.26	39.83	38.69	36.35	43.91	42.27	41.91	41.50	46.16	45.23
C16:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C17:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C17:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C18:0	25.50	24.82	29.62	26.29	23.78	27.09	29.04	27.38	25.44	29.21	26.71
C18:1	27.78	29.26	25.90	29.42	28.54	22.23	25.72	24.08	26.35	20.82	23.70
C18:2	7.72	7.66	4.65	5.59	11.34	6.77	2.97	6.63	6.71	3.82	4.36
C20:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C18:3	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C20:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C20:2	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C22:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C20:3	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C20:4	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C24:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C20:5	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C22:5	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C22:6	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00

[1] FA = fatty acid, presented as g FA per 100 g fat.

Fatty Acid Composition of Red Blood Cells from FISH Foals

Table A-84. Fatty acid composition of FISH foal red blood cells at birth

	Foal									
FA[1]	5B47	5W69	5A66	5B31	5A59	5B33	5B24	5B46	5B14	5B01
C8:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C10:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	1.18	0.00	0.00
C12:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C14:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C14:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C16:0	41.10	38.53	22.45	37.99	41.96	25.87	27.04	36.20	39.10	37.91
C16:1	6.30	2.70	2.73	2.25	4.13	4.27	2.32	2.34	2.89	5.24
C17:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C17:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C18:0	16.47	15.18	14.07	15.81	28.25	12.92	12.95	19.71	18.77	20.36
C18:1	32.60	32.70	34.09	36.40	20.78	36.71	35.74	32.17	35.99	31.50
C18:2	1.90	3.60	25.12	3.51	3.23	15.33	5.40	2.30	1.57	3.31
C20:0	1.64	3.03	0.97	2.05	1.65	1.33	1.51	1.58	1.69	1.68
C18:3	0.00	0.00	0.57	0.00	0.00	0.89	0.91	0.00	0.00	0.00
C20:1	0.00	4.27	0.00	2.00	0.00	2.68	12.47	4.53	0.00	0.00
C20:2	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C22:0	0.00	0.00	0.00	0.00	0.00	0.00	1.65	0.00	0.00	0.00
C20:3	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C20:4	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C24:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C20:5	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C22:5	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C22:6	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00

[1] FA = fatty acid, presented as g FA per 100 g fat.

Table A-85. Fatty acid composition of FISH foal red blood cells at 14 d of age

	Foal									
FA[1]	5B47	5W69	5A66	5B31	5A59	5B33	5B24	5B46	5B14	5B01
C8:0	0.00	0.00	0.00	NA[2]	NA	0.00	0.00	0.00	0.00	0.00
C10:0	0.00	0.00	0.00	NA	NA	0.00	0.00	0.00	0.00	0.00
C12:0	0.00	0.00	0.00	NA	NA	0.00	0.00	0.00	0.00	0.00
C14:0	0.00	0.00	0.00	NA	NA	0.00	0.00	0.00	0.00	0.00
C14:1	0.00	0.00	0.00	NA	NA	0.00	0.00	0.00	0.00	0.00
C16:0	37.42	31.63	19.03	NA	NA	19.88	35.15	25.94	26.29	30.06
C16:1	2.74	2.93	3.16	NA	NA	3.38	4.20	3.45	2.03	1.68
C17:0	0.00	0.83	0.59	NA	NA	0.00	0.94	0.00	0.00	0.00
C17:1	0.00	0.00	0.00	NA	NA	0.00	0.00	0.00	0.00	0.00
C18:0	21.19	17.88	15.64	NA	NA	13.95	18.16	19.63	17.96	20.35
C18:1	30.28	29.56	28.64	NA	NA	31.08	32.85	29.66	30.16	29.14
C18:2	3.49	8.22	31.41	NA	NA	24.68	7.14	8.53	5.22	6.13
C20:0	2.98	2.13	1.52	NA	NA	0.00	1.55	1.48	1.76	1.26
C18:3	0.00	0.00	0.00	NA	NA	0.00	0.00	0.00	0.00	0.00
C20:1	0.00	5.02	0.00	NA	NA	5.69	0.00	11.32	14.80	9.79
C20:2	1.89	1.80	0.00	NA	NA	1.33	0.00	0.00	1.78	1.59
C22:0	0.00	0.00	0.00	NA	NA	0.00	0.00	0.00	0.00	0.00
C20:3	0.00	0.00	0.00	NA	NA	0.00	0.00	0.00	0.00	0.00
C20:4	0.00	0.00	0.00	NA	NA	0.00	0.00	0.00	0.00	0.00
C24:1	0.00	0.00	0.00	NA	NA	0.00	0.00	0.00	0.00	0.00
C20:5	0.00	0.00	0.00	NA	NA	0.00	0.00	0.00	0.00	0.00
C22:5	0.00	0.00	0.00	NA	NA	0.00	0.00	0.00	0.00	0.00
C22:6	0.00	0.00	0.00	NA	NA	0.00	0.00	0.00	0.00	0.00

[1] FA = fatty acid, presented as g FA per 100 g fat.

[2] NA = not analyzed.

Table A-86. Fatty acid composition of FISH foal red blood cells at 28 d of age

	Foal									
FA[1]	5B47	5W69	5A66	5B31	5A59	5B33	5B24	5B46	5B14	5B01
C8:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C10:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C12:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C14:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C14:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C16:0	55.63	39.02	33.48	50.39	39.35	33.79	51.56	38.40	37.88	33.94
C16:1	3.78	3.09	4.33	5.81	3.88	4.96	5.50	3.62	3.13	2.64
C17:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C17:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C18:0	24.02	21.97	24.47	22.56	22.56	21.25	25.58	21.68	21.28	20.41
C18:1	7.73	27.50	3.68	4.26	29.37	32.33	7.23	31.66	27.91	33.04
C18:2	8.83	5.42	34.04	16.98	4.84	7.67	10.13	4.64	6.67	9.97
C20:0	0.00	3.00	0.00	0.00	0.00	0.00	0.00	0.00	3.13	0.00
C18:3	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C20:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C20:2	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C22:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C20:3	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C20:4	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C24:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C20:5	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C22:5	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C22:6	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00

[1] FA = fatty acid, presented as g FA per 100 g fat.

Table A-87. Fatty acid composition of FISH foal red blood cells at 56 d of age

	Foal									
FA[1]	5B47	5W69	5A66	5B31	5A59	5B33	5B24	5B46	5B14	5B01
C8:0	0.00	NA[2]	0.00	0.00	0.00	0.00	OT[3]	0.00	0.00	0.00
C10:0	0.00	NA	0.00	0.00	0.00	0.00	OT	0.00	0.00	0.00
C12:0	0.00	NA	0.00	0.00	0.00	0.00	OT	0.00	0.00	0.00
C14:0	0.00	NA	0.00	0.00	0.00	0.00	OT	0.00	0.00	0.00
C14:1	0.00	NA	0.00	0.00	0.00	0.00	OT	0.00	0.00	0.00
C16:0	39.04	NA	25.16	30.52	36.66	36.35	OT	42.90	43.25	24.44
C16:1	2.21	NA	2.15	2.12	2.41	2.51	OT	0.00	0.00	1.73
C17:0	0.00	NA	0.00	0.00	0.00	0.00	OT	0.00	0.00	0.00
C17:1	0.00	NA	0.00	0.00	0.00	0.00	OT	0.00	0.00	0.00
C18:0	20.98	NA	18.21	16.66	23.73	16.10	OT	21.39	23.28	14.34
C18:1	30.28	NA	28.25	30.58	28.88	33.83	OT	30.68	31.22	31.36
C18:2	7.49	NA	26.22	20.14	8.32	8.58	OT	5.03	2.26	28.13
C20:0	0.00	NA	0.00	0.00	0.00	0.00	OT	0.00	0.00	0.00
C18:3	0.00	NA	0.00	0.00	0.00	2.63	OT	0.00	0.00	0.00
C20:1	0.00	NA	0.00	0.00	0.00	0.00	OT	0.00	0.00	0.00
C20:2	0.00	NA	0.00	0.00	0.00	0.00	OT	0.00	0.00	0.00
C22:0	0.00	NA	0.00	0.00	0.00	0.00	OT	0.00	0.00	0.00
C20:3	0.00	NA	0.00	0.00	0.00	0.00	OT	0.00	0.00	0.00
C20:4	0.00	NA	0.00	0.00	0.00	0.00	OT	0.00	0.00	0.00
C24:1	0.00	NA	0.00	0.00	0.00	0.00	OT	0.00	0.00	0.00
C20:5	0.00	NA	0.00	0.00	0.00	0.00	OT	0.00	0.00	0.00
C22:5	0.00	NA	0.00	0.00	0.00	0.00	OT	0.00	0.00	0.00
C22:6	0.00	NA	0.00	0.00	0.00	0.00	OT	0.00	0.00	0.00

[1] FA = fatty acid, presented as g FA per 100 g fat.
[2] NA = not analyzed.
[3] OT = off trial.

Table A-88. Fatty acid composition of FISH foal red blood cells at 84 d of age

	Foal									
FA[1]	5B47	5W69	5A66	5B31	5A59	5B33	5B24	5B46	5B14	5B01
C8:0	0.00	0.00	0.00	0.00	0.00	0.00	OT[2]	0.00	0.00	0.00
C10:0	0.00	0.00	0.00	0.00	0.00	0.00	OT	0.00	0.00	0.00
C12:0	0.00	0.00	0.00	0.00	0.00	0.00	OT	0.00	0.00	0.00
C14:0	0.00	0.00	0.00	0.00	0.00	0.00	OT	0.00	0.00	0.00
C14:1	0.00	0.00	0.00	0.00	0.00	0.00	OT	0.00	0.00	0.00
C16:0	33.44	26.20	24.82	23.64	35.66	27.91	OT	39.07	38.30	23.89
C16:1	0.00	17.87	0.00	1.76	1.92	0.00	OT	0.00	0.00	0.00
C17:0	0.00	0.00	0.00	0.00	0.00	0.00	OT	0.00	0.00	0.00
C17:1	0.00	0.00	5.42	0.00	0.00	0.00	OT	0.00	0.00	5.97
C18:0	20.97	23.69	28.11	14.92	24.25	11.90	OT	23.23	22.49	23.72
C18:1	32.93	21.96	27.25	28.53	29.29	29.66	OT	31.93	31.29	28.33
C18:2	12.66	10.28	14.40	31.15	8.88	30.53	OT	5.77	7.93	18.08
C20:0	0.00	0.00	0.00	0.00	0.00	0.00	OT	0.00	0.00	0.00
C18:3	0.00	0.00	0.00	0.00	0.00	0.00	OT	0.00	0.00	0.00
C20:1	0.00	0.00	0.00	0.00	0.00	0.00	OT	0.00	0.00	0.00
C20:2	0.00	0.00	0.00	0.00	0.00	0.00	OT	0.00	0.00	0.00
C22:0	0.00	0.00	0.00	0.00	0.00	0.00	OT	0.00	0.00	0.00
C20:3	0.00	0.00	0.00	0.00	0.00	0.00	OT	0.00	0.00	0.00
C20:4	0.00	0.00	0.00	0.00	0.00	0.00	OT	0.00	0.00	0.00
C24:1	0.00	0.00	0.00	0.00	0.00	0.00	OT	0.00	0.00	0.00
C20:5	0.00	0.00	0.00	0.00	0.00	0.00	OT	0.00	0.00	0.00
C22:5	0.00	0.00	0.00	0.00	0.00	0.00	OT	0.00	0.00	0.00
C22:6	0.00	0.00	0.00	0.00	0.00	0.00	OT	0.00	0.00	0.00

[1] FA = fatty acid, presented as g FA per 100 g fat.
[2] OT = off trial.

Fatty Acid Composition of Red Blood Cells from FLAX Foals

Table A-89. Fatty acid composition of FLAX foal red blood cells at birth

	Foal										
FA[1]	5B21	5A62	5B32	5C2	5C6	5A65	5B06	5B44	5B28	5B19	5C1
C8:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA[2]
C10:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA
C12:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA
C14:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA
C14:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA
C16:0	33.93	29.79	36.93	44.22	31.45	24.80	40.11	29.89	27.17	37.09	NA
C16:1	3.69	4.14	2.58	2.25	3.11	3.01	2.89	2.53	2.33	7.00	NA
C17:0	0.00	0.00	0.00	0.00	0.00	0.00	0.98	0.00	0.00	0.00	NA
C17:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA
C18:0	18.45	12.73	16.33	18.19	14.35	14.81	18.52	14.67	12.64	17.35	NA
C18:1	37.04	31.49	34.29	31.36	32.01	34.56	33.15	31.90	31.72	34.06	NA
C18:2	5.00	5.60	2.92	1.84	4.38	9.42	2.49	3.59	1.94	3.04	NA
C20:0	1.88	12.25	1.58	2.15	1.72	0.96	1.87	1.78	1.87	1.45	NA
C18:3	0.00	4.01	0.00	0.00	0.86	0.73	0.00	0.00	0.00	0.00	NA
C20:1	0.00	0.00	5.36	0.00	12.11	11.69	0.00	15.63	22.32	0.00	NA
C20:2	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA
C22:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA
C20:3	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA
C20:4	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA
C24:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA
C20:5	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA
C22:5	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA
C22:6	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA

[1] FA = fatty acid, presented as g FA per 100 g fat.
[2] NA = not analyzed.

Table A-90. Fatty acid composition of FLAX foal red blood cells at 14 d of age

	Foal										
FA[1]	5B21	5A62	5B32	5C2	5C6	5A65	5B06	5B44	5B28	5B19	5C1
C8:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C10:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C12:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C14:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C14:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C16:0	32.20	28.97	33.76	37.95	29.92	33.25	25.42	31.82	31.14	33.81	19.14
C16:1	4.31	4.12	3.77	4.93	4.43	3.22	2.26	2.18	2.92	3.52	9.38
C17:0	0.00	0.92	0.00	0.00	1.10	0.00	0.00	0.00	1.10	0.00	0.00
C17:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C18:0	21.31	20.24	21.15	22.31	19.26	26.62	17.68	20.49	21.03	22.21	18.01
C18:1	35.46	34.05	34.48	30.02	35.63	33.16	29.44	32.31	28.72	33.13	25.12
C18:2	5.63	6.74	3.82	2.01	5.81	3.75	4.66	5.78	2.79	6.20	4.89
C20:0	0.00	2.40	3.02	2.78	1.73	0.00	1.39	0.00	2.80	0.00	11.24
C18:3	0.00	0.86	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	3.47
C20:1	0.00	0.00	0.00	0.00	0.00	0.00	17.14	5.55	7.66	0.00	3.49
C20:2	1.10	1.71	0.00	0.00	2.11	0.00	2.01	1.86	1.84	1.12	5.26
C22:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C20:3	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C20:4	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C24:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C20:5	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C22:5	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C22:6	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00

[1] FA = fatty acid, presented as g FA per 100 g fat.

Table A-91. Fatty acid composition of FLAX foal red blood cells at 28 d of age

	Foal										
FA[1]	5B21	5A62	5B32	5C2	5C6	5A65	5B06	5B44	5B28	5B19	5C1
C8:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C10:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C12:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C14:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C14:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C16:0	53.87	53.08	49.36	54.67	55.73	36.14	38.45	35.70	53.14	39.02	52.83
C16:1	6.43	4.94	5.47	6.02	4.51	2.20	3.17	2.65	6.45	3.29	3.96
C17:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C17:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C18:0	26.51	28.53	26.84	26.74	25.16	24.19	22.11	26.37	27.51	22.57	25.75
C18:1	6.82	5.11	3.51	7.79	6.74	32.45	31.05	26.39	5.16	27.41	4.95
C18:2	6.37	8.34	14.82	4.79	4.75	5.02	5.22	5.83	7.75	7.72	12.51
C20:0	0.00	0.00	0.00	0.00	3.12	0.00	0.00	3.05	0.00	0.00	0.00
C18:3	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C20:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C20:2	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C22:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C20:3	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C20:4	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C24:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C20:5	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C22:5	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C22:6	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00

[1] FA = fatty acid, presented as g FA per 100 g fat.

Table A-92. Fatty acid composition of FLAX foal red blood cells at 56 d of age

	Foal										
FA[1]	5B21	5A62	5B32	5C2	5C6	5A65	5B06	5B44	5B28	5B19	5C1
C8:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C10:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C12:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C14:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C14:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C16:0	38.57	33.52	39.54	29.77	39.47	37.55	37.98	39.89	41.05	35.24	30.58
C16:1	2.70	0.00	2.25	11.17	0.00	2.86	1.61	2.02	0.00	0.00	2.35
C17:0	0.00	0.00	0.00	5.56	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C17:1	0.00	0.00	0.00	7.08	0.00	0.00	0.00	0.00	0.00	7.31	0.00
C18:0	21.91	21.03	24.50	22.64	22.28	25.14	23.77	23.33	23.11	20.06	16.96
C18:1	30.84	32.02	28.56	19.15	30.52	30.67	31.59	28.87	30.09	30.07	29.52
C18:2	5.97	13.43	5.14	4.63	7.73	3.78	2.26	5.89	5.76	7.32	20.59
C20:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C18:3	0.00	0.00	0.00	0.00	0.00	0.00	2.78	0.00	0.00	0.00	0.00
C20:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C20:2	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C22:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C20:3	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C20:4	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C24:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C20:5	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C22:5	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C22:6	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00

[1] FA = fatty acid, presented as g FA per 100 g fat.

Table A-93. Fatty acid composition of FLAX foal red blood cells at 84 d of age

	Foal										
FA[1]	5B21	5A62	5B32	5C2	5C6	5A65	5B06	5B44	5B28	5B19	5C1
C8:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C10:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C12:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C14:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C14:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C16:0	35.45	40.13	35.70	44.63	43.37	37.09	38.19	34.58	39.40	34.17	39.01
C16:1	1.90	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C17:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C17:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	13.18	0.00	0.00	0.00
C18:0	23.78	23.10	22.78	22.86	23.78	26.90	22.71	24.72	23.67	21.95	23.83
C18:1	32.10	31.26	33.18	28.15	29.04	31.89	30.72	22.48	31.02	32.74	31.77
C18:2	6.77	5.51	8.34	4.36	3.81	4.13	8.39	5.04	5.91	11.14	5.39
C20:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C18:3	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C20:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C20:2	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C22:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C20:3	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C20:4	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C24:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C20:5	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C22:5	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C22:6	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00

[1] FA = fatty acid, presented as g FA per 100 g fat.

Fatty Acid Composition of Red Blood Cells from CON Foals

Table A-94. Fatty acid composition of CON foal red blood cells at birth

	Foal										
FA[1]	5B29	5B36	5B13	5B41	5B45	5A54	5C4	5B18	5B26	5A61	5A64
C8:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA[2]
C10:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA
C12:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA
C14:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA
C14:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA
C16:0	40.50	36.66	35.52	24.43	32.16	42.54	40.82	21.76	39.10	38.22	NA
C16:1	4.19	3.63	5.38	2.28	2.50	6.80	0.00	2.60	1.56	5.25	NA
C17:0	1.02	0.00	1.06	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA
C17:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA
C18:0	17.84	15.42	17.94	15.31	13.19	19.65	31.41	12.09	29.21	14.39	NA
C18:1	32.93	34.79	35.60	36.65	28.13	27.99	23.94	33.17	23.22	36.39	NA
C18:2	1.76	2.96	3.14	20.72	2.03	1.78	3.83	15.32	5.07	4.50	NA
C20:0	1.76	1.24	1.37	0.61	1.55	1.25	0.00	1.13	1.84	1.25	NA
C18:3	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.77	0.00	0.00	NA
C20:1	0.00	5.31	0.00	0.00	20.44	0.00	0.00	13.17	0.00	0.00	NA
C20:2	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA
C22:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA
C20:3	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA
C20:4	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA
C24:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA
C20:5	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA
C22:5	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA
C22:6	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA

[1] FA = fatty acid, presented as g FA per 100 g fat.
[2] NA = not analyzed.

Table A-95. Fatty acid composition of CON foal red blood cells at 14 d of age

	Foal										
FA[1]	5B29	5B36	5B13	5B41	5B45	5A54	5C4	5B18	5B26	5A61	5A64
C8:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA[2]	0.00	0.00	0.00
C10:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA	0.00	0.00	0.00
C12:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA	0.00	0.00	0.00
C14:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA	0.00	0.00	0.00
C14:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA	0.00	0.00	0.00
C16:0	38.00	32.86	32.48	23.08	38.58	39.55	33.23	NA	23.38	37.14	40.83
C16:1	2.55	4.56	3.54	2.34	4.10	3.29	3.50	NA	2.49	2.26	4.93
C17:0	0.00	0.00	0.00	0.66	0.00	1.29	1.06	NA	0.65	1.10	0.00
C17:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA	0.00	0.00	2.59
C18:0	20.45	19.66	25.20	17.91	19.15	26.42	24.30	NA	17.95	18.93	19.42
C18:1	33.43	36.85	33.95	31.10	31.41	26.45	30.18	NA	28.56	28.87	25.57
C18:2	4.29	3.82	4.83	24.90	3.25	3.01	2.59	NA	15.62	5.64	2.80
C20:0	0.00	2.24	0.00	0.00	3.51	0.00	2.98	NA	0.76	0.00	2.57
C18:3	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA	0.00	0.00	0.00
C20:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA	10.59	4.80	0.00
C20:2	1.29	0.00	0.00	0.00	0.00	0.00	2.16	NA	0.00	1.26	1.28
C22:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA	0.00	0.00	0.00
C20:3	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA	0.00	0.00	0.00
C20:4	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA	0.00	0.00	0.00
C24:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA	0.00	0.00	0.00
C20:5	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA	0.00	0.00	0.00
C22:5	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA	0.00	0.00	0.00
C22:6	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA	0.00	0.00	0.00

[1] FA = fatty acid, presented as g FA per 100 g fat.

[2] NA = not analyzed.

Table A-96. Fatty acid composition of CON foal red blood cells at 28 d of age

	Foal										
FA[1]	5B29	5B36	5B13	5B41	5B45	5A54	5C4	5B18	5B26	5A61	5A64
C8:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA[2]	0.00	0.00	0.00
C10:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA	0.00	0.00	0.00
C12:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA	0.00	0.00	0.00
C14:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA	0.00	0.00	0.00
C14:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA	0.00	0.00	0.00
C16:0	38.00	32.86	32.48	23.08	38.58	39.55	33.23	NA	23.38	37.14	40.83
C16:1	2.55	4.56	3.54	2.34	4.10	3.29	3.50	NA	2.49	2.26	4.93
C17:0	0.00	0.00	0.00	0.66	0.00	1.29	1.06	NA	0.65	1.10	0.00
C17:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA	0.00	0.00	2.59
C18:0	20.45	19.66	25.20	17.91	19.15	26.42	24.30	NA	17.95	18.93	19.42
C18:1	33.43	36.85	33.95	31.10	31.41	26.45	30.18	NA	28.56	28.87	25.57
C18:2	4.29	3.82	4.83	24.90	3.25	3.01	2.59	NA	15.62	5.64	2.80
C20:0	0.00	2.24	0.00	0.00	3.51	0.00	2.98	NA	0.76	0.00	2.57
C18:3	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA	0.00	0.00	0.00
C20:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA	10.59	4.80	0.00
C20:2	1.29	0.00	0.00	0.00	0.00	0.00	2.16	NA	0.00	1.26	1.28
C22:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA	0.00	0.00	0.00
C20:3	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA	0.00	0.00	0.00
C20:4	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA	0.00	0.00	0.00
C24:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA	0.00	0.00	0.00
C20:5	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA	0.00	0.00	0.00
C22:5	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA	0.00	0.00	0.00
C22:6	0.00	0.00	0.00	0.00	0.00	0.00	0.00	NA	0.00	0.00	0.00

[1] FA = fatty acid, presented as g FA per 100 g fat.

[2] NA = not analyzed.

Table A-97. Fatty acid composition of CON foal red blood cells at 56 d of age

	Foal										
FA[1]	5B29	5B36	5B13	5B41	5B45	5A54	5C4	5B18	5B26	5A61	5A64
C8:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C10:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C12:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C14:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C14:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C16:0	43.78	36.70	39.48	32.69	41.96	44.46	38.00	36.93	34.70	39.13	31.16
C16:1	3.17	2.63	2.63	0.00	0.00	0.00	0.00	2.19	1.94	0.00	2.37
C17:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C17:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C18:0	21.84	21.31	23.12	19.76	23.42	25.23	25.40	21.30	24.26	21.06	19.61
C18:1	27.98	32.22	29.75	32.52	29.28	25.49	30.70	31.32	33.85	34.18	27.92
C18:2	3.24	7.14	5.02	15.03	5.33	4.82	5.91	8.26	5.25	5.64	18.94
C20:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C18:3	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C20:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C20:2	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C22:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C20:3	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C20:4	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C24:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C20:5	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C22:5	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C22:6	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00

[1] FA = fatty acid, presented as g FA per 100 g fat.

Table A-98. Fatty acid composition of CON foal red blood cells at 84 d of age

	Foal										
FA[1]	5B29	5B36	5B13	5B41	5B45	5A54	5C4	5B18	5B26	5A61	5A64
C8:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C10:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C12:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C14:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C14:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C16:0	35.47	35.31	41.74	29.73	30.76	42.27	39.77	30.81	36.44	44.32	35.88
C16:1	0.00	2.62	0.00	0.00	0.00	0.00	0.00	1.97	9.00	0.00	2.08
C17:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C17:1	6.27	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C18:0	30.71	21.69	27.83	20.70	21.12	26.61	27.35	20.62	29.93	24.38	21.52
C18:1	27.56	33.46	26.10	32.78	31.87	22.39	28.24	31.60	24.63	28.01	28.90
C18:2	0.00	6.93	4.33	16.80	16.25	8.73	4.64	15.00	0.00	3.30	11.62
C20:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C18:3	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C20:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C20:2	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C22:0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C20:3	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C20:4	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C24:1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C20:5	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C22:5	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
C22:6	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00

[1] FA = fatty acid, presented as g FA per 100 g fat.

Response of Mares during an Intradermal Skin Test

Table A-99. Skin thickness values of FISH mares during an intradermal skin test[1]

	Time[2]							
Mare	0h	2h	4h	6h	8h	12h	24h	48h
A61	3.93	11.39	16.71	17.47	14.48	15.98	18.33	11.83
B26	4.42	11.68	18.05	16.78	22.09	19.44	15.91	19.50
B41	3.91	9.83	13.59	11.17	16.79	13.98	10.09	15.52
C4	4.23	11.03	15.43	10.33	14.68	12.51	14.79	12.24
A54	3.78	13.52	22.67	23.52	24.36	23.94	19.63	23.87
B13	4.78	13.28	16.68	15.47	16.25	15.86	14.78	20.07
B18	5.37	12.92	13.84	15.78	13.61	14.69	18.63	13.43
B29	3.18	10.56	15.57	14.73	13.69	14.21	15.34	14.87
B45	4.25	9.75	12.16	11.73	12.21	11.97	10.79	11.01
B36	3.93	11.39	16.71	17.47	14.48	15.98	18.33	11.83

[1] Presented in mm.
[2] Measured as h post injection.

Table A-100. Skin thickness values of FLAX mares during an intradermal skin test[1]

	Time[2]							
Mare	0h	2h	4h	6h	8h	12h	24h	48h
B06	3.78	11.71	10.17	10.94	14.54	18.57	16.56	14.82
B19	3.83	16.46	12.78	14.62	18.55	17.56	18.06	22.46
A65	4.45	7.45	6.84	7.15	14.29	13.30	13.80	17.74
B44	4.00	10.51	12.59	11.55	12.46	13.19	12.83	13.27
C1	4.35	14.51	14.21	14.36	16.13	16.54	16.33	18.62
A62	5.88	12.53	12.37	12.45	14.77	13.73	14.25	12.81
B21	4.54	7.84	7.97	7.91	9.88	11.03	10.45	9.10
C2	4.09	10.90	10.11	10.51	14.89	14.27	14.58	18.25
B28	4.52	13.81	10.92	12.36	17.72	12.45	15.08	19.97
C6	4.91	12.79	12.07	12.43	15.62	18.22	16.92	15.81
B32	4.68	11.54	10.46	11.00	11.34	11.45	11.40	9.73

[1] Presented in mm.
[2] Measured as h post injection.

Table A-101. Skin thickness values of CON mares during an intradermal skin test[1]

	Time[2]							
Mare	0h	2h	4h	6h	8h	12h	24h	48h
A61	4.61	11.29	11.52	11.41	15.11	16.80	15.96	16.18
B26	4.59	9.88	9.13	9.51	19.80	18.25	19.03	20.17
B41	5.03	12.68	9.56	11.12	15.73	10.08	12.90	18.14
C4	4.38	13.56	10.84	12.20	12.78	12.36	12.57	12.68
A54	4.82	11.58	11.55	11.57	13.23	12.62	12.93	15.94
B13	4.81	13.34	11.42	12.38	17.89	17.45	17.67	21.08
B18	5.35	16.36	13.85	15.11	21.19	20.00	20.59	21.20
B29	4.80	13.78	10.58	12.18	16.78	12.09	14.43	15.95
B45	3.92	10.54	10.46	10.50	12.80	13.48	13.14	10.30
B36	4.37	12.95	14.99	13.97	15.55	18.14	16.85	14.75

[1] Presented in mm.
[2] Measured as h post injection.

Response of Foals during an Intradermal Skin Test

Table A-102. Skin thickness values of FISH foals during an intradermal skin test[1]

	Time[2]							
Foal	0h	2h	4h	6h	8h	12h	24h	48h
5W69	4.76	4.55	4.66	9.81	9.15	9.48	11.02	10.30
5B33	3.60	3.83	3.72	8.34	9.79	9.07	11.21	11.34
5B14	4.57	4.38	4.48	9.66	10.35	10.01	12.14	14.92
5B46	4.22	3.78	4.00	10.86	7.86	9.36	14.61	12.56
5B1	4.66	4.54	4.60	10.01	10.48	10.25	13.55	15.53
5A59	4.69	4.36	4.52	12.64	10.63	11.63	15.87	16.40
5B31	3.63	3.52	3.57	10.50	9.85	10.17	13.13	13.28
5B47	3.81	4.17	3.99	10.99	8.45	9.72	15.25	10.42
5A66	3.76	3.82	3.79	7.95	11.27	9.61	10.23	16.50

[1] Presented in mm.
[2] Measured as h post injection.

Table A-103. Skin thickness values of FLAX foals during an intradermal skin test[1]

	Time[2]							
Foal	0h	2h	4h	6h	8h	12h	24h	48h
5B06	4.00	3.06	3.53	11.45	11.92	11.69	14.85	14.26
5B19	3.75	4.45	4.10	10.46	13.54	12.00	13.79	16.67
5A65	3.58	3.56	3.57	9.26	11.18	10.22	12.13	15.00
5B44	3.29	3.49	3.39	8.68	9.84	9.26	11.66	13.77
5B28	4.56	4.42	4.49	11.02	10.71	10.86	14.00	12.07
5C1	4.61	3.62	4.11	11.48	9.94	10.71	15.30	17.30
5C6	4.72	3.36	4.04	9.24	9.26	9.25	13.55	12.01
5A62	4.29	3.80	4.05	9.76	9.04	9.40	14.62	12.13
5B21	3.60	3.47	3.54	7.55	7.38	7.46	13.30	13.55
5C2	3.92	4.22	4.07	12.53	11.68	12.10	15.13	14.95
5B32	3.76	3.49	3.63	10.41	9.46	9.94	14.28	14.32

[1] Presented in mm.
[2] Measured as h post injection.

Table A-104. Skin thickness values of CON foals during an intradermal skin test[1]

	Time[2]							
Foal	0h	2h	4h	6h	8h	12h	24h	48h
5A61	5.12	4.94	5.03	11.37	10.25	10.81	13.90	12.50
5B26	4.01	3.99	4.00	8.07	7.66	7.87	12.60	10.47
5B41	4.73	4.63	4.68	8.80	9.15	8.97	9.16	13.65
5B13	4.62	4.59	4.60	12.09	9.72	10.91	15.61	13.51
5A54	5.35	4.35	4.85	13.50	10.71	12.10	13.29	12.37
5C4	4.67	4.75	4.71	12.89	12.83	12.86	14.34	17.64
5B18	3.82	4.11	3.96	10.35	9.61	9.98	14.99	14.23
5B29	4.21	3.87	4.04	12.51	8.28	10.39	16.78	7.97
5B45	3.14	2.88	3.01	10.63	9.45	10.04	12.74	12.40
5B36	4.21	4.17	4.19	9.30	8.95	9.13	10.57	14.66

[1] Presented in mm.
[2] Measured as h post injection.

APPENDIX B
PROCEDURE FOR IMMUNOGLOBULIN G ANALYSIS

Immunoglobulin G (IgG) content was analyzed using a single radial immunodiffusion (SRID) kit (VMRD, Inc., Pullman, WA) with a detection range of 200 to 1600 mg/dL. The kit included four SRID plates (12 wells each) containing monospecific antisera in buffered agarose, four reference standards (described at the end of this appendix) preserved with 0.09% sodium azide and a 3 μL precision pipette and plunger. SRID plates were shipped and stored upside down in mylar pouches. The mechanism at work in this analysis is as follows: the antigen in the sample added to the plate diffuses into the gel containing the antibody, and a precipitation ring forms that is proportional to the concentration of the antigen. This analysis is time and temperature dependent. The procedure used for IgG determination was as follows:

1. Samples were allowed to come to room temperature and vortexed thoroughly.
2. Samples with high expected IgG values were diluted with deionized water (diH_2O) to ensure that readings would be within measurable levels. Samples were diluted by pipetting 250 μL of sample into a clean vial and adding the appropriate amount of diH_20. Samples were vortexed thoroughly after dilution. Dilutions were as follows:
    - Colostrum: 1 part sample, 15-17 parts diH_2O
    - Mare serum (d0): 1 part sample, 3 parts diH_2O
    - Foal serum (36h through d+84): 1 part sample, 3 parts diH_2O
    - Mare milk (36h through d+84, foal serum (d0): No dilution
3. Three μL of Standards A through D were pipetted (using the included precision pipette and plunger) into wells 1 through 4 of plate 1. Once the filling process had begun, the pipette was lifted off the bottom of the well to ensure the liquid did not overflow the well.

4. The remaining wells of all plates were filled with 3 µL of each sample to be tested, following the same pipetting technique as above. Identification and dilution rate of the sample in each well were recorded on the data sheet included in the kit.

5. Plate covers were firmly reattached and plates were left undisturbed, rightside up at room temperature for 18-24 hours outside of their mylar pouches.

6. After 18-24 hours, ring diameter was read in mm using a monocular comparator (VMRD, Inc., Pullman, WA). Diameter readings were recorded on the data sheet included in the kit. Used plates were inverted, returned to their mylar pouches and stored at 4-8°C.

7. To determine IgG concentrations, standard and sample diameters and dilution rates were entered into the computer program MetraFIT (Metra Biosystems, Inc., Mountain View, CA). Concentrations were calculated using a 4 parameter model using a standard equation provided by the program. The equation used was IgG concentration = (a - d) / (1 + (RD / c) ^ b) + d, where a = 2.968, b = 1.245, c = 1278.2, d = 9.829 and RD = ring diameter.

STANDARDS

- **Standard A**: 200 mg IgG/dL
- **Standard B**: 400 mg IgG/dL
- **Standard C**: 800 mg IgG/dL
- **Standard D**: 1600 mg IgG/dL

APPENDIX C
PROCEDURE FOR FATTY ACID ANALYSIS

Fatty acids were extracted from plasma, milk or red blood cells as described by Folch et al. (1957). The fatty acid extraction procedure was as follows:

8. Colostrum, milk and plasma samples were dried in a freeze dryer in preparation for fatty acid (FA) extraction. Colostrum and milk samples were dried for 5 days and plasma samples were dried for 24-48 hours. Every 48-72 hours, samples were removed from the freeze dryer and stored at -20°C while the freeze dryer was defrosted. At the end of defrosting (approximately 30 minutes), samples were placed back into the freeze dryer to continue drying. Grass, hay, and concentrate samples were dried in a 60°C oven for 3 days in preparation for FA analysis.

9. After drying, colostrum and milk samples were weighed in their sample cups to determine dry sample weights. These weights were then used to calculate colostrum and milk dry matter. After weighing, the dry milk samples were transferred from the samples cups to plastic Whirl Pack bags. Blood samples were kept in their original vials. All samples were stored at -20°C until extraction.

10. To prepare for FA extraction, 40 mL screw cap vials were weighed without caps to determine a tare weight used later to calculate the amount of total fat extracted. These vials were labeled as "T-Tubes." Special care was taken to not allow bare skin to touch these vials as skin oils could add additional weight. After weighing, Teflon lined caps were placed on the vials and the vials were set aside for later use.

11. Weighted or measured sample amounts were placed into another set of 40 mL screw cap vials and the weights/volumes of samples was recorded. Target weights/volumes used were:

    - 1 g freeze dried milk
    - 2 g freeze dried colostrum
    - 2 mL freeze dried plasma
    - 2 mL freeze dried red blood cells
    - 2-3 g dried hay or grass
    - 1-3 g dried concentrate
    - 0.3 g fish oil or flaxseed supplement

12. Twenty mL of Folch 1 and 50 µL of a C19:0 internal standard were added to all samples (see end of appendix for reagent explanation). Vials were then vortexed for 2 minutes.

13. Samples were left at room temperature over night (at least 16 hours).

14. The next morning, a nitrogen gas evaporator apparatus was prepared by adding distilled water (dH_2O) to the water bath and heating it with a heating rod to 37°C. The water bath in the methylation tank was also filled and heated to 90°C.

15. Samples were vortexed and filtered through #40 Whatman filter paper (150 mm) into the T-Tubes.

16. To recover any FA remaining in the original sample vial, 10-20 mL of Folch 1 were added to the original vial. Vials were again vortexed and filtered. The filter paper was then rinsed with 1-2 mL of clean Folch 1.

17. Once the samples were finished filtering, 0.1 mL of 10% BHT was pipetted into each sample and the vials were gently swirled to mix.

18. Samples were dried under nitrogen gas flow in a 37°C water bath until all liquid had disappeared (approximately 1.5-2 hours).

19. Vials were removed from the drying apparatus, dried with a paper towel and allowed to cool completely.

20. When the tubes were cool, they were weighed to determine the amount of fat in the original sample. This was accomplished using a digital scale. A small beaker was placed on the scale, its weight tared and the vials were placed in the beaker and weighed without their caps. The original T-Tube weights were subtracted from these weights to give the weight of actual fat recovered.

21. Teflon tape was wrapped around the threads of each vial to prevent evaporation during methylation.

22. Two mL of 4% H_2SO_4 in methanol were added to each vial containing the dried fat. Caps were replaced and checked for tightness of fit.

23. Samples were heated in a 90°C bath for 15 minutes to permit methylation of the fatty acid.

24. Samples were allowed to cool completely after methylation before being uncapped. The Teflon tape was removed from the vial threads and 1.0 mL hexane was added to the sample by pipet. Caps were quickly replaced to avoid hexane evaporation.

25. Samples were vortexed and transferred to 8 mL glass vials with Teflon septa screw caps.

26. Two mL of double distilled water (ddH_2O) were added to the vials via needle and syringe. Vials were vortexed, inverted and left at room temperature for at least 30 minutes to allow for layer separation.

27. The bottom layer (made up of water and chemicals) was removed using a needle and syringe. Great care was taken not to remove any of the top layer (made of hexane and fat). Therefore, a small amount of water was left after each removal to ensure no hexane was accidentally removed.

28. Two mL ddH_2O was again added to the vials via needle and syringe and the vials were vortexed, inverted and left to stand at room temperature for at least 30 minutes.

29. The removal of the water layer was repeated, again leaving a small amount of water to ensure no hexane removal.

30. Steps 19-22 were repeated, for a total of three washes.

31. Two mL of ddH_2O was added to the sample. The sample was vortexed and left standing upright for at least 30 minutes at room temperature. (Note: If the hexane did not separate from the water or was cloudy, samples were stored at 4-8°C overnight to ensure separation).

32. The hexane layer was transferred by glass pipet into a 2 ml glass GC vial for FA analysis. If less than 0.5 mL of hexane was recovered, the hexane was transferred to a 200 μL polypropylene tube FA analysis.

33. Samples were stored at -20°C and allowed to come to room temperature before being placed on the GC autosampler. After analysis, samples were stored at -20°C.

Gas Chromatograph Information

Gas chromatography (GC) was performed using a CP-3800 Gas Chromatograph (Varian, Inc., Palo Alto, CA). A WCOT fused silica column (CP-SIL 88, length 100 m, internal diameter 0.25 mm, flow rate 5.0 mL/min, Varian, Inc., Palo Alto, CA) was used. The carrier gas was helium with a pressure of 29.5 psi (one minute), 35.4 psi (0.42 psi/min, total of 45 minutes) and 37.9 psi (0.17 psi/min, held for 50 minutes, total of 110 minutes). The temperature program was 120°C for one minute, increased to 190°C at 5°C/minute and held at 190°C for 30 minutes (total of 45 minutes), increased to 220°C at 2°C/minute and held at 220°C for 50 minutes, giving a total run time of 110 minutes. Fatty acids were identified by comparison of peak retention times for samples and reference standards (Nu-Chek Prep, Inc., Elysian, MN).

REAGENTS

- **10% BHT**: 20 g butylated hydrototulene in 200 mL total volume with methanol; antioxidant
- **4% H_2SO_4**: 8.33 mL H_2SO_4 in 200 mL total volume with methanol; for methylation
- **Folch 1**: 1 part methanol, 2 parts chloroform; for fat homogenization
- **Double distilled water (ddH2O)**: replaced daily; for washing

LIST OF REFERENCES

Al, M.D.M., A.C. van Houwelingen, and G. Hornstra. 2000. Long-chain polyunsaturated fatty acids, pregnancy, and pregnancy outcome. Am. J. Clin. Nutr. 71:285S-291S.

Allen, K.G.D., and M.A. Harris. 2001. The role of n-3 fatty acids in gestation and parturition. Exp. Biol. Med. 226:498-506.

Anderson, M., and K.L. Fritsche. 2002. (n-3) fatty acids and infectious disease resistance. J. Nutr. 132:3566-3576.

Baguma-Nibasheka, M., J. T. Brenna, and P.W. Nathanielsz. 1999. Delay of preterm delivery in sheep by omega-3 long-chain polyunsaturates. Biol. Reprod. 60:698-701.

Bauer, J.E., B.L. Dunbar, and K.E. Bigley. 1998. Dietary flaxseed in dogs results in differential transport and metabolism of (n-3) polyunsaturated fatty acids. J. Nutr. 128:2641S-2644S.

Bauer, J.E., K.M. Heinemann, K.E. Bigley, G.E. Lees, and M.K. Waldron. 2004. Maternal diet α-linolenic acid during gestation and lactation does not increase docosahexaenoic acid in canine milk. J. Nutr. 134:2035S-2038S.

Benatti, P., G. Peluso, R. Nicolai, and M. Calvani. 2004. Polyunsaturated fatty acids: biochemical, nutritional and epigenetic properties. J. Am. Coll. Nutr. 23:281-302.

Bergero, D., N. Miraglia, M. Polidori, M. Ziino, and D. Gagliardi. 2002. Blood serum and skin fatty acid levels in horses and the use of dietary polyunsaturated fatty acids. Anim. Res. 51:157-163.

Bieber, L. L. 1988. Carnitine. Ann. Rev. Biochem. 57:261-283.

Billiar, T., P. Bankey, B. Svingen, R.D. Curran, M.A. West, R.T. Holman, R.L. Simmons, and F.B. Cerra. 1988. Fatty acid uptake and Kupffer cell function: fish oil alters eicosanoid and monokine production to endotoxin stimulation. Surgery. 104:343-349.

Bouwman, H., and W. van der Shee. 1978. Composition and production of milk from Dutch warmblood saddle horse mares. Z. Tierphysiol. Tierernaehr. Futtermittelkd. 40:39-53.

Bowen, R.A.R., and M.T. Clandinin. 2000. High dietary 18:3n-3 increases the 18:3n-3 but not the 22:6n-3 content in the whole body, brain, skin, epididymal fat pads, and muscles of suckling rat pups. Lipids. 35:389-394.

Brasitus, T.A., N.O. Davidson, and D. Schachter. 1985. Variations in dietary triacylglycerol saturation alter the lipid composition and fluidity of rat intestinal plasma membranes. Biochim. Biophys. Acta. 812:460-472.

Brinsko, S.P., D.D. Varner, C.C. Love, T.L. Blanchard, B.C. Day, and M.E. Wilson. 2005. Effect of feeding a DHA-enriched nutriceutical on the quality of fresh, cooled and frozen stallion semen. Theriogenology 63:1519-1527.

Byleveld, P.M, G.T. Pang, R.L. Clancy, and D.C.K. Roberts. 1999. Fish oil feeding delays influenza virus clearance and impairs production of interferon-γ and virus-specific immunoglobulin A in the lungs of mice. J. Nutr. 129:328-335.

Calder, P.C. 2001. Polyunsaturated fatty acids, inflammation, and immunity. Lipids. 36:1007-1024.

Campbell, F.M., M.J. Gordon, and A.K. Dutta-Roy. 1996. Preferential uptake of long chain polyunsaturated fatty acids by isolated placental membranes. Mol. Cell. Biochem. 155:77-83.

Carey, M.C., D.M. Small, and C.M Bliss. 1983. Lipid digestion and absorption. Ann. Rev. Physiol. 45:651-677.

Carlier, H., A. Bernard, and C. Caselli. 1991. Digestion and absorption of polyunsaturated fatty acids. Reprod. Nutr. Dev. 31:475-500.

Chen, C.C., H.C. Chaung, M.Y. Chung, and L.T. Huang. In press. Menhaden fish oil improves spatial memory in rat pups following recurrent pentylenetetrazole-induced seizures. Epilepsy Behav. In press.

Clegg, R.A., M.C. Barber, L. Pooley, I. Ernens, Y. Larondelle, and M.T. Travers. 2001. Milk fat synthesis and secretion: molecular and cellular aspects. Livest. Prod. Sci. 70:3-14.

Connor, W.E., R. Lowensohn, and L. Hatcher. 1996. Increased docosahexaenoic acid levels in newborn infants by administration of sardines and fish oil during pregnancy. Lipids. 31:S183-S187.

Crawford, M.A. 2000. Placental delivery of arachidonic and docosahexaenoic acids: implications for the lipid nutrition of preterm infants. Am. J. Clin. Nutr. 71(Suppl.):275S-284S.

Csapó, J., J. Stefler, T.G. Martin, S. Makray, and Z. Csapó-Kiss. 1995. Composition of mares' colostrum and milk. Fat content, fatty acid composition and vitamin content. Int. Dairy J. 5:393-402.

Cunha, T.J. 1991. Horse feeding and nutrition. 2nd ed. Academic Press, Inc., New York, NY.

D'ambola, J.B., E.E. Aeberhard, N. Trang, S. Gaffar, C.T. Barrett, and M.P. Sherman. 1991. Effect of dietary (n-3) and (n-6) fatty acids on in vivo pulmonary bacterial clearance by neonatal rabbits. J. Nutr. 121:1262-1269.

Davidson, K.E., G.D. Potter, L.W. Greene, J.W. Evans, and W.C. McMullan. 1991. Lactation and reproductive performance of mares fed added dietary fat during late gestation and early lactation. J. Eq. Vet. Sci. 11:111-115.

de Groot, R.H., G. Hornstra, A.C. van Houwelingen, and F. Roumen. 2004. Effect of alpha-linolenic supplementation during pregnancy on maternal and neonatal polyunsaturated fatty acid status and pregnancy outcome. Am. J. Clin. Nutr. 79:251-260.

Dewhurst, R.J., N.D. Scollan, S.J. Youell, J.K.S. Tweed, and M.O. Humphreys. 2001. Influence of species, cutting date and cutting interval on the fatty acid composition of grasses. Grass Forage Sci. 56:68-74.

Dewhurst, R.J., J.M. Moorby, N.D. Scollan, J.K.S. Tweed, and M.O Humphreys. 2002. Effects of a stay-green trait on the concentrations and stability of fatty acids in perennial ryegrass. Grass Forage Sci. 57:360-366.

Dils, R.R. 1986. Comparative aspects of milk fat synthesis. J. Dairy Sci. 69:904-910.

Doreau, M., and S. Boulot. 1989. Recent knowledge on mare milk production: a review. Livest. Prod. Sci. 22:213-235.

Doreau, M., S. Boulot, D. Bauchart, J.P. Barlet, and W. Martin-Rosset. 1992. Voluntary intake, milk production and plasma metabolites in nursing mares fed two different diets. J. Nutr. 122:992-999.

Doreau, M., S. Boulot, and Y. Chilliard. 1993. Yield and composition of milk from lactating mares: effect of body condition at foaling. J. Dairy Res. 60:457-466.

Doreau, M., and Y. Chilliard. 1997. Digestion and absorption of dietary fat in farm animals. Br. J. Nutr. 78(Suppl. 1):S15-S35.

Drevon, C.A. 1992. Marine oils and their effects. Nutr. Rev. 50:38-45.

Duvaux-Ponter, C., M. Tournié, L. Detrimont, F. Clément, C. Ficheux, and A.A. Ponter. 2004. Effect of a supplement rich in linolenic acid added to the diet of mares on the fatty acid composition of mammary secretions and the acquisition of passive immunity in the foal. Anim. Sci. 78:399-407.

Elgersma, A., G. Ellen, H. van der Horst, B.G. Muuse, H. Boer, and S. Tamminga. 2003. Comparison of the fatty acid composition of fresh and ensiled perennial ryegrass (*Lolium perenne* L.), affected by cultivar and regrowth interval. Anim. Feed Sci. Tech. 108:191-205.

Elias, S.L. and S.M. Innis. 2001. Infant plasma *trans*, n-6, and n-3 fatty acids and conjugated linoleic acids are related to maternal plasma fatty acids, length of gestation, and birth weight and length. Am. J. Clin. Nutr. 73:807-814.

Ellis, A.D., and J. Hill. 2005. Nutritional Physiology of the Horse. Nottingham University Press, Nottingham, UK.

Erhard, M.H., C. Luft, H.P. Remler, and M. Stangassinger. 2001. Assesment of colostral transfer and systemic availability of immunoglobulin G in new-born foals using a newly developed enzyme-linked immunosorbent assay (ELISA) system. J. Anim. Physiol. Anim. Nutr. (Berl.). 85:164-173.

Feist, J.D., and D. McCullough. 1976. Behaviour patterns and communication in feral horses. Z Tierpsychol. 41:337-371.

Fernandes, G., V. Tomar, M.N. Venkatraman, and J.T. Venkatraman. 1992. Potential of diet therapy of murine AIDS. J. Nutr. 122:716-722.

Folch, J., M. Lees, and G.H. Sloane Stanley. 1957. A simple method for the isolation and purification of total lipids from animal tissues. J. Biol. Chem. 226:497-506.

Francois, C.A., S.L. Connor, L.C. Bolewicz, and W.E. Connor. 2003. Supplementing lactating women with flaxseed oil does not increase docosahexaenoic acid in their milk. Am. J. Clin. Nutr. 77:226-233.

Frank, N., J.E. Sojka, and M.A. Latour. 2004. Effect of hyperthyroidism on the blood lipid response to higher dietary fat intake in mares. J. Anim. Sci. 82:2640-2646.

Frape, D. 1998. Equine Feeding and Nutrition. 2nd ed. Blackwell Sci., Oxford, UK.

Fritsche, K.L., S. Huang, and N.A. Cassity. 1993. Enrichment of omega-3 fatty acids in suckling pigs by maternal dietary fish oil supplementation. J. Anim. Sci. 71:1841-1847.

Goldsby, R.A., T.J. Kindt, B.A. Osborne, and J. Kuby. 2003. Immunology. 5th ed. W.H. Freeman and Company, New York.

Greene, E. 2006. Effect of long-chain fatty acids on lipid metabolizing genes and high-density lipoprotein cholesterol production in cultured human and rat hepatocytes. Ph.D. Diss., University of Florida, Gainesville.

Gurr, M.I., J.L. Harwood, and K.N. Frayn. 2002. Lipid Biochemistry. 5th ed. Blackwell Science Ltd., Oxford, UK.

Haggerty, P., K. Page, D.R. Abramovich, J. Ashton, and D. Brown. 1997. Long-chain polyunsaturated fatty acid transport across the perfused human placenta. Placenta. 18:635-642.

Hall, J.A., R.J. Van Saun, and R.C. Wander. 2004a. Dietary (n-3) fatty acids from menhaden fish oil alter plasma fatty acids and leukotriene B synthesis in healthy horses. J. Vet. Intern. Med. 18:871-879.

Hall, J.A., R.J. Van Saun, S.J. Tornquist, J.L. Gradin, E.G. Pearson, and R.C. Wander. 2004b. Effect of type of dietary polyunsaturated fatty acid supplement (corn oil or fish oil) on immune response in healthy horses. J. Vet. Intern. Med. 18:880-886.

Hansen, R.A., C.J. Savage, K. Reidlinger, J.L. Traub-Dargatz, G.K. Ogilvie, D. Mitchell, and M.J. Fettman. 2002. Effects of dietary flaxseed oil supplementation on equine plasma fatty acid concentrations and whole blood platelet aggregation. J. Vet. Intern. Med. 16:457-463.

Helland, I.B., K. Saarem, O.D. Saugstad, and C.A. Drevon. 1998. Fatty acid composition in maternal milk and plasma during supplementation with cod liver oil. Eur. J. Clin. Nutr. 52:839-845.

Henderson, R.A., R.G. Jensen, C.J. Lammi-Keefe, A.M. Ferris, and K.R. Dardick. 1992. Effect of fish oil on the fatty acid composition of human milk and maternal and infant erythrocytes. Lipids. 27:863-869.

Hocquette, J.F., and D. Bauchart. 1999. Intestinal absorption, blood transport and hepatic and muscle metabolism of fatty acids in preruminant and ruminant animals. Reprod. Nutr. Dev. 39:27-48.

Hoffman, R.M., D.S. Kronfeld, J.H. Herbein, W.S. Swecker, W.L. Cooper, and P.A. Harris. 1998. Dietary carbohydrates and fat influence milk composition and fatty acid profile of mare's milk. J. Nutr. 128:2708S-2711S.

Innis, S.M. 1991. Essential fatty acids in growth and development. Prog. Lipid Res. 30:39-103.

Innis, S.M. 1992a. Essential fatty acid requirements in human nutrition. Can. J. Physiol. Pharmacol. 71:699-706.

Innis, S.M. 1992b. Plasma and red blood cell fatty acid values as indexes of essential fatty acids in the developing organs of infants fed with milk or formulas. J. Pediatr. 120:S78-S86.

Innis, S.M. 2005. Essential fatty acid transfer and fetal development. Placenta. 26:S70-S75.

James, M.J., R.A. Gibson, and L.G. Cleland. 2000. Dietary polyunsaturated fatty acids and inflammatory mediator production. Am. J. Clin. Nutr. 71:343S-348S.

Jeffcott, L.B. 1972. Passive immunity and its transfer with special reference to the horse. Biol. Rev. 47:439-464.

Jeffcott, L.B. 1974a. Some practical aspects of the transfer of passive immunity to newborn foals. Equine Vet. J. 6:109-115.

Jeffcott, L.B. 1974b. Studies on passive immunity in the foal. J. Comp. Path. 84: 93-101.

Jeffcott, L.B. 1975. The transfer of passive immunity to the foal and its relation to immune status after birth. J. Reprod. Fert. 23(Suppl.):727-733.

Johnson, P.R., and M.R.C. Greenwood. 1998. Cell and Tissue Biology: A Textbook of Histology. 6th ed. Urban and Schwarzenberg, Baltimore, MD.

King, S.S., K.L. Jones, G.A. Apgar, and A. AbuGhazaleh. 2005. Fatty acid profiles from red blood cells in response to three levels of dietary omega-3 fatty acid supplementation. Proc. 19th Equine Sci. Soc. Symp., Tucson, Arizona:135-136.

Knowles, J.R. 1989. The mechanism of biotin-containing enzymes. Ann. Rev. Biochem. 58:195-221.

Kohn, C.W., D.K. Knight, W.H. Hueston, R. Jacobs, and S.M. Reed. 1989. Colostral and serum IgG, IgA, and IgM concentrations in Standardbred mares and their foals at parturition. J. Am. Vet. Med. Assoc. 195:64-68.

Koterba, A.M., W.H. Drummond, and P.C. Kosch. 1990. Equine Clinical Neonatology. Lea & Febiger, Philadelphia, Pennsylvania.

Kruglik, V.L., J.M. Kouba, C.M. Hill, K.A. Skjolasss-Wilson, C. Armendariz, J.E. Minton, and S.K. Webel. 2005. Effect of feeding protected n-3 polyunsaturated fatty acids on plasma and milk fatty acid levels and IgG concentrations in mares and foals. Proc. 19th Equine Sci. Soc. Symp., Tucson, Arizona:135-136.

Kruse, P.E. 1983. The importance of colostral immunoglobulins and their absorption from the intestine of the newborn animals. Ann. Rech. Vet. 14:349-353.

Kuby, J. 1992. Immunology. W.H. Freeman and Company, New York.

Lavoie, J.P., M.S. Spensley, B.P. Smith, and J. Mihalyi. 1989. Colostral volume and immunoglobulin G and M determinations in mares. Am. J. Vet. Res. 50:466-470.

LeBlanc, M.M, B.I. McLaurin, and R. Boswell. 1986. Relationships among serum immunoglobulin concentration in foals, colostral specific gravity, and colostral immunoglobulin concentration. J. Am. Vet. Med. Assoc. 189:57-60.

LeBlanc, M.M., T. Thang, J.L. Baldwin, and E.L. Pritchard. 1992. Factors that influence passive transfer of immunoglobulins in foals. J. Am. Vet. Med. Assoc. 200:179-183.

Lehner, R., and A. Kuksis. 1996. Biosynthesis of triacylglycerols. Prog. Lipid Res. 35:169-201.

Liu, I.K.M. 1980. Systemic diseases of the newborn foal. Vet. Clin. North. Amer. Large. Anim. Pract. 2:361-375.

Lund, E.K., L.F. Harvey, S. Ladha, D.C. Clark, and I.T. Johnson. 1999. Effects of dietary fish oil supplementation on the phospholipid composition and fluidity of cell membranes from human volunteers. Ann. Nutr. Metab. 43:290-300.

Makrides, M., M.A. Neumann, and R.A. 1996. Effect of maternal docosahexaenoic acid (DHA) supplementation on breast milk composition. Eur. J. Clin. Nutr. 50:352-357.

Mattos, R., C.R. Staples, and W.T. Thatcher. 2000. Effects of dietary fatty acids on reproduction in ruminants. Rev. Reprod. 5:38-45.

McGuire, T.C., and T.B. Crawford. 1973. Passive immunity in the foal: measurement of immunoglobulin classes and specific antibody. Am. J. Vet. Res. 34:1299-1302.

McGuire, T.C., T.B. Crawford, and A.L. Halliwell. 1977. Failure of colostral immunoglobulin transfer as an explanation for most infections and deaths of neonatal foals. J. Am. Vet. Med. Assoc. 170:1302-1304.

Meddings, J.B, and S. Theisen. 1989. Development of rat jejunum: lipid permeability, physical properties, and chemical composition. Am. J. Physiol. 256:931-940.

Meydani, S.N., A.H. Lichtenstein, S. Cornwall, M. Meydani, B.R. Goldin, H. Rasmussen, C.A. Dinarello, and E.J. Schaefer. 1993. Immunologic effects of National Cholesterol Education Panel step-2 diets with and without fish-derived n-3 fatty acid enrichment. J. Clin. Invest. 92:105-113.

Mills, S.C., A.C. Windsor, and S.C. Knight. 2005. The potential interactions between polyunsaturated fatty acids and colonic inflammatory disease. Clin. Exp. Immunol. 142:216-228.

Morris, D.D., D.A. Meirs, and G.S. Merryman. 1985. Passive transfer failure in horses: incidence and causative factors on a breeding farm. Am. J. Vet. Res. 46:2294-2299.

Mu, H. and C. Hoy. 2004. The digestion of dietary triacylglycerols. Prog Lipid Res. 43:105-133.

Murphy, M.G. 1990. Dietary fatty acids and membrane protein functions. J. Nutr. Biochem. 1:68-76.

National Research Council (NRC). 1989. Nutrient requirements of horses. 5th ed. National Academy Press, Washington, DC.

Neville, M.C., and M.F. Picciano. 1997. Regulation of milk lipid secretion and composition. Annu. Rev. Nutr. 17:159-184.

Nezlin, R. 1998. The immunoglobulins: structure and function. Academic Press, San Diego, CA.

O'Connor, C.I., L.M. Lawrence, A.S. St. Lawrence, K.M. Janicki, L.K. Warren, and S. Hayes. 2004. The effect of dietary fish oil supplementation on exercising horses. J. Anim. Sci. 82:2978-2984.

O'Kelly, J.C., and H.P. Reich. 1976. The fatty acid composition of tropical pastures. J. Agric. Sci. Camb. 86:427-429.

Oftedal, O.T., H.F. Hintz , and H.F. Schryver. 1983. Lactation in the horse: milk composition and intake by foals. J. Nutr. 113:2096-2106.

Olsen, S.F., H.S. Hansen, and B. Jensen. 1990. Fish oil versus arachis oil food supplementation in relation to pregnancy duration in rats. Prostaglandins Leukot. Essent. Fatty Acids. 40:255-260.

Pande, S. V. 1975. A mitochondrial carnitine acylcarnitine translocase system. Proc. Natl. Acad. Sci. U.S.A. 72:883-887.

Paul, K.P., M. Leichsenring, M. Pfisterer, E. Mayatepek, D. Wagner, M. Domann, H.G. Sonntag, and H.J. Bremer. 1997. Influence of n-6 and n-3 polyunsaturated fatty acids on the resistance to experimental tuberculosis. Metabolism. 46:619-624.

Pawlosky, R.J., J.R. Hibbeln, J.A. Novotny, and N. Salem, Jr. 2001. Physiological compartmental analysis of α-linolenic acid metabolism in adult humans. J. Lipid Res. 42:1257-1265.

Peakman, M., and D. Vergani. 1997. Basic and clinical immunology. Churchill Livingstone, New York.

Pearson, R.C., A.L. Hallowell, W.M. Bayly, R.L. Torbeck, and L.E. Perryman. 1984. Times of appearance and disappearance of colostral IgG in the mare. Am. J. Vet. Res. 45:186-190.

Raidal, S.L. 1996. The incidence and consequences of failure of passive transfer of immunity on a Thoroughbred breeding farm. Aust. Vet. J. 73:201-206.

Raidal, S.L., C. McTaggart, J.V. Yovich, and J. Penhale. 2000. Effect of withholding macromolecules on the duration of intestinal permeability to colostral IgG in foals. Proc. Amer. Assoc. Equine Prac. 46:260-263.

Rayon, J.I., J.D. Carver, L.E. Wyble, D. Wiener, S.S. Dickey, V.J. Benford, L.T. Chen, and D.V. Lim. 1997. The fatty acid composition of maternal diet affects the lung prostaglandin E_2 levels and survival from group B streptococcal sepsis in neonatal rat pups. J. Nutr. 127:1989-1992.

Rooke, J.A., and I.M. Bland. 2002. The acquisition of passive immunity in the new-born piglet. Livest. Prod. Sci. 78:13-23.

Sadeghi, S., F.A. Wallace, and P.C. Calder. 1999. Dietary lipids modify the cytokine response to bacterial lipopolysaccharide in mice. Immunology. 96:404-410.

Sanders, T.A.B, and K.M. Reddy. 1992. The influence of a vegetarian diet on the fatty acid composition of human milk and the essential fatty acid status of the infant. J. Pediatr. 120:S71-S77.

Sauerwald, T.U., D.L Hachey, C.L. Jensen, H. Chen, R.E. Anderson, and W.C. Heird. 1996. Effect of dietary α-linolenic acid intake on incorporation of docosahexaenoic and arachidonic acids into plasma phospholipids of term infants. Lipids. 31 (Suppl.) S131-S135.

Scheuerbrandt, G. and K. Bloch. 1962. Unsaturated fatty acids in microorganisms. J. Biol. Chem. 237:2064-2068.

Sheoran, A.S, J.F. Timoney, M.A. Holmes, S.S. Karzenski, and M.V. Crisman. 2000. Immunoglobulin isotypes in sera and nasal mucosal secretions and their neonatal transfer and distribution in horses. Am. J. Vet. Res. 61:1099-1105.

Siciliano, P.D., S.K. Webel, L.S. Brown, L.K. Warren, T.E. Engle, and P.D. Burns. 2003. Effect of n-3 polyunsaturated fatty acid source on plasma fatty acid profiles of horses. J. Anim. Sci. 81(Suppl. 1):72. (Abstr.)

Sierra, S., F. Lara-Villoslada, M. Olivares, J. Jiménez, J. Boza, and J. Xaus. 2005. Increased immune response in mice consuming rice bran oil. Eur. J. Nutr. 44:509-516.

Simopoulos, A.P. 1999. Essential fatty acids in health and chronic disease. Am. J. Clin. Nutr. 70 (Suppl.):560S-569S.

Smith, W.L. 1989. The eicosanoids and their biological mechanisms of action. Biochem. J. 259:315-324.

Spallholz, J.E, L.M. Boylan, and J.A. Driskell. 1999. Lipids. Pages 24-40 in Nutrition: Chemistry and Biology. 2nd ed. CRC Press LLC, Boca Raton, FL.

Spearman, K.R. 2004. Effect of mannan oligosaccharide (MOS) supplementation on the immune status of mares and their foals. M.S. Thesis, University of Florida, Gainesville.

Spearman, K.R., E.A. Ott, J. Kivipelto, and L.K. Warren. 2005. Effect of fatty acid supplementation of the mare on milk and foal plasma composition and foal growth. Paper presented at Proc. 19th Equine Sci. Soc. Symp., Tucson, Arizona.

Stammers, J.P., D.P. Leadon, and D. Hull. 1987. Fatty acid composition of the plasma lipids of the maternal and newborn horse. J. Reprod. Fert. 35(Suppl.):615-622.

Stammers, J.P., M. Silver, and A.L. Fowden. 1988. Effects of nutrition on uterine and umbilical venous plasma lipids in chronically catheterized mares in late gestation. Eq. Vet. J. 5(Suppl.):37-40.

Stammers, J.P., M. Silver, and A.L. Fowden. 1991. Maternal and umbilical venous plasma lipid concentrations at delivery in the mare. Eq. Vet. J. 23:119-122.

Stammers, J.P., D. Hull, M. Silver, A.L. Fowden, J. Ousey, and P.D. Rossdale. 1994. Release of lipid from the equine placenta during in vitro incubation. Placenta. 15:857-872.

Suneja, S. K., P. Osei, L. Cook, M.N. Nagi, and D.L. Cinti. 1990. Enzyme site-specific changes in hepatic microsomal fatty acid chain elongation in streptozotocin-induced diabetic rats. Biochim. Biophys. Acta. 1042:81-85.

Sutton, J.D., and S.V. Marant. 1989. A review of the potential of nutrition to modify milk fat and protein. Livest. Prod. Sci. 23:219-237.

Thors, V.S., A. Porisdottir, H. Erlendsdottir, I. Einarsson, S. Gudmundsson, E. Gunnarsson, and A. Haraldsson. 2004. The effect of dietary fish oil on survival after infection with Klebsiella pneumoniae or Streptococcus pneumoniae. Scand. J. Infect. Dis. 36:102-105.

Tizard, I.R. 1996. Veterinary immunology: an introduction. 5th ed. W.B. Saunders Company, Philadelphia, Pennsylvania.

Tso, P. 1989. Gastrointestinal digestion and absorption of lipid. Adv. Lipid Res. 21:143-186.

Tyler-McGowan, C.M., J.L. Hodgson, and D.R. Hodgson. 1997. Failure of passive transfer in foals: incidence and outcome on four studs in New South Wales. Aust. Vet. J. 75:56-59.

Ullrey, D.E., R.D. Struthers, D.G. Hendricks, and B.E. Brent. 1966. Composition of mare's milk. J. Anim. Sci. 25:217-221.

Wander, R.C., J.A. Hall, J.L. Grandin, S.H. Du, and D.E. Jewell. 1997. The ratio of dietary (n-6) to (n-3) fatty acids influences immune system function, eicosanoid metabolism, lipid peroxidation and vitamin E status in aged dogs. J. Nutr. 127:1198-1205.

Ward, J.D., J.W. Spears, and E.B. Kegley. 1993. Effect of copper level and source (copper lysine vs. copper sulfate) on copper status, performance, and immune response in growing steers fed diets with or without supplemental molybdenum and sulfur. J. Anim. Sci. 71:2748-2755.

Widmann, F.K., and C.A. Itatani. 1998. An introduction to clinical immunology and serology. 2nd ed. F.A. Davis Company, Philadelphia, Pennsylvania.

Wiseman, M.J. 1997. Fish oils and human diet. Br. J. Nutr. 78(Suppl.):S5-S13.

Wright, S. 1991. Essential fatty acids and the skin. Br. J. Dermatol. 125:503-515.

Yaqoob, P. 2004. Fatty acids and the immune system: from basic science to clinical applications. Proc. Nutr. Soc. 63:89-104.

BIOGRAPHICAL SKETCH

Elizabeth Lindsay Stelzleni was born in Gainesville, Florida, on March 2, 1982. She was raised a strict Gator fan and always knew she would attend the University of Florida. Elizabeth loved horses from a very young age and was given riding lessons for her seventh birthday. It was then that her devotion to horses truly began.

Elizabeth graduated fourth in her class from Gainesville High School in 2000 with a GPA of 4.5. During her high school years she bought Handsome Slew, her Thoroughbred gelding whom she proceeded to win many area and national awards on in the sport of eventing. In her senior year of high school, Elizabeth began training young horses and retraining problem horses in the sports of eventing, dressage and hunter/jumper.

Elizabeth entered Santa Fe Community College in the fall of 2000 and achieved her Associate of Arts degree in 2002. She then moved to the University of Florida to begin her Bachelor of Science degree program in animal sciences (equine science specialization) and graduated cum laude in 2004. At graduation, she was honored as a Two-Year Scholar by the University. Around the same time that Elizabeth was preparing to graduate, Dr. Lori Warren had just come to the University as a professor of equine nutrition. Dr. Warren agreed to take Elizabeth on as a graduate student, and Elizabeth began her Master of Science degree in the fall of 2004. During her time as a graduate student, Elizabeth worked as a teaching assistant for many equine science classes and continued to train young and problem horses for various sports.

During the first year of her graduate program, Elizabeth married Alexander Stelzleni on December 18, 2004. Alexander was working on his doctoral degree in meat science and beef production when the two met and plans on graduating with his degree in May of 2006. Upon graduation, Alexander plans on pursuing his goal of securing a job as a professor of meat science and animal production classes, while Elizabeth will pursue her goal of working for a feed company as an equine specialist.