Plasma and tissue fatty acid profiles of growing pigs fed structured or non-structured triacylglycerides containing medium-chain and marine oil fatty acids

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Twenty-four growing pigs (18.3 kg body weight) were used in a 19-day metabolism trial to determine the effect of n-3/medium-chain triacylglyceride (MCT)-structured triacylglyceride on plasma and tissue lipid profiles. Pigs were allotted to four liquid diets, each providing 19.5% protein (dry matter basis) as Na- and Ca-caseinate + soybean protein isolate. Lipid composition (percentage by weight of total lipid) by treatment was as follows: (I) corn oil:soybean oil:MCT oil (40:10:50), (II) MCT oil:menhaden oil (60:40 as structured triacylglyceride), (III) MCT oil:menhaden oil (60:40 non-structured, physical mixture), and (IV) structured triacylglyceride (as in II):safflower oil:canola oil (80:10:10). Blood samples were obtained on days 0, 12, and 19 with liver and muscle samples obtained on day 19. With the exception of arachidonic acid, plasma fatty acid profiles reflected dietary fatty acid profiles. Plasma arachidonic acid percentage was higher ($P < 0.05$) in pigs consuming diet I despite a lower dietary content of this fatty acid relative to diets II, III, and IV. Platelets from pigs consuming diets II or III had lower percentage linoleic and arachidonic acid percentages on day 19 compared with diet I, which appeared to be compensated for by elevated eicosapentaenoic and docosahexaenoic acids. Diets II, III, and IV resulted in higher ($P < 0.05$) percentages of myristic, pentadecenoic, palmitic, palmitoleic, heptadecanoic, heptadecenoic, stearic, linoleic, eicosapentaenoic, docosahexaenoic, and nervonic acids in the liver and elevated ($P < 0.05$) myristic, palmitic, palmitoleic, stearic, linoleic, and eicosapentaenoic acids in the muscle. Physical form (structured versus non-structured triacylglyceride) did not affect fatty acid profiles.

Keywords: swine; structured triacylglycerides; menhaden oil; n-3 fatty acids; medium-chain triacylglycerides; fatty acid profiles

Introduction

Medium-chain triacylglycerides (MCT) are synthetic acylglycerols composed primarily of caprylic and capric acids and small amounts of caproic acid. Unique digestive, absorptive, and metabolic characteristics of MCT including (1) a more rapid and complete intestinal hydrolysis[1,2] and absorption[3] relative to long-chain triacylglycerides (LCT), (2) direct absorption of the MCT hydrolysis products into the portal vein,[4] (3) subsequent mitochondrial uptake of medium-chain fatty acids via a carnitine-independent mechanism,[5] and (4) rapid catabolism and limited reesterification of medium-chain fatty acids compared with long-chain fatty acids[6,7] have stimulated the investigation of MCT use in a variety of nutritional applications.

One such nutritional application is the formation of MCT-structured triacylglycerides comprised of medium-chain fatty acids and long-chain polyunsaturated fatty acids esterified to the same glycerol molecule. Some of the distinctive digestive, absorptive, and metabolic characteristics of MCT are retained in the MCT-structured triacylglyceride. The efficiency of intestinal

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hydrolysis and absorption of MCT-structured triacylglycerides is intermediate between that for MCT and LCT.[8] Furthermore, MCT-structured triacylglycerides are more readily oxidized and less adipogenic than LCT.[4] Finally, the incorporation of long-chain polyunsaturated fatty acids into MCT-structured triacylglycerides also provides a source of essential fatty acids, a capacity not possible with MCT.

The application of MCT-structured triacylglycerides in a nutritional regimen has been tested primarily through the use of the burned rat model. Nitrogen balance was improved as a result of enteral[8,9] or parenteral[7–10] administration of MCT-structured triacylglycerides containing up to 60% medium-chain fatty acids by weight. Although MCT or LCT (i.e., soybean oil) provided alone or in combination did not affect nitrogen balance, DeMichele et al.[8] noted that a physical mixture of fatty acids identical to the MCT-structured triacylglyceride improved nitrogen balance. Increased protein synthesis in liver and muscle in concert with decreased protein catabolism appear responsible for the ameliorated nitrogen retention.

Ingestion of increasing quantities of n-3 polyunsaturated fatty acids can shift the balance of prostacyclin, thromboxane, and leukotriene synthesis toward one favoring vasodilation and anti-aggregation.[11–13] This has been hypothesized to assist in the prevention of atherosclerosis by minimizing platelet thrombi formation on the vascular endothelium, reducing subsequent release of platelet-derived growth factor, and limiting the inflammatory response to vascular wall injury.[14]

There is currently no information available regarding the use of dietary MCT/marine oil-structured triacylglycerides in the growing pig. The present study was conducted using the growing pig to (1) evaluate MCT/marine oil-structured triacylglyceride in a liquid diet relative to a commercially available liquid diet containing corn oil, soybean oil, and MCT oil; (2) compare liquid diets containing MCT/marine oil structured triacylglyceride with a physical mixture of fatty acids equivalent to MCT/marine oil structured triacylglyceride; and (3) evaluate MCT/marine oil-structured triacylglyceride in a liquid diet when mixed with the minimal quantity of canola oil and safflower oil necessary to provide dietary essential fatty acids. The present paper will focus on the effect of dietary lipid source on plasma and tissue fatty acid profiles.

Methods and materials

Animals and dietary treatments

A 19-day metabolism trial was conducted in two replicates using a total of 24 crossbred (Yorkshire × Landrace × Duroc) barrows weighing approximately 18.3 kg. Pigs were individually housed in stainless-steel metabolism cages in an environmentally controlled room and were allowed a 5-day adjustment period prior to the start of the experiment. During this adjustment period and during the experimental period the treatment diets were provided ad libitum via Kane baby pig waterers. No additional feed or water source was provided. The protocol for this study was approved by the Institutional Laboratory Animal Care and Use Committee.

The experimental animals were allotted by weight and ancestry to four isotonic liquid diets differing only in lipid source. Diets contained either (I) corn oil + soybean oil + MCT oil (Corn/Soy/MCT), (II) MCT/marine oil structured triacylglyceride (ST), (III) MCT/marine oil physical mixture (PM), or (IV) MCT/marine oil structured triacylglyceride + canola oil + safflower oil (ST/Can/Saf) *(Table 1)*. The MCT/marine oil structured lipid was a mixture (by weight) of 60% MCT (Captex 300, Karlshamns Lipid Specialties USA, Columbus, OH USA) and 40% menhaden oil randomly transesterified into composite MCT-structured lipid molecules. The PM was a similar mixture of MCT and menhaden oil without random transesterification. *Table 1* presents the complete diet composition.

Sample collection and preparation

All pigs were weighed on days 1, 7, 12, 14, and 19. A 12-mL blood sample was obtained from all pigs by puncture of the anterior vena cava on days 1, 12, and 19 of the study following an overnight fast. At the termination of the experiment, all pigs were killed and a liver and muscle (semimembranosus) sample frozen at −20° C for subsequent analysis. Approximately 2 mL of blood was placed in a glass test tube for the determination of clotting time by stirring the blood with a wire loop and recording the time required for clot formation.[15] The remaining 10 mL of blood was gently mixed in a plastic centrifuge tube containing 1.1 mL of 38 g/L sodium citrate. A 1.2 mL aliquot of the citrated blood was removed and held at room temperature prior to the determination of platelet aggregation. The remaining citrated blood was placed on ice until centrifugation to obtain platelets and plasma.

The citrated blood was centrifuged for 15 min (120 × g, 5° C) and the resulting platelet-rich plasma removed to another centrifuge tube. The red blood cell pellet and the platelet-rich plasma were then centrifuged for 10 min (100 × g, 5° C) and the resulting plasma layers pooled within pig and frozen until analyzed. The platelet pellet was washed twice in suspending buffer A[16], resuspended to a final concentration of approximately 10^{11} platelets/L, and frozen at −20° C in a plastic tube until analyzed for fatty acid profile. Platelet concentration in the final suspension was determined with a Coulter counter (Coulter Electronics, Hialeah, FL USA).

Analytical methods

Platelet aggregation was conducted in a single channel aggregometer (Model 500-VS, Chronolog Corp., Haverton, PA USA) at 37° C with continuous stirring. Collagen was selected as the agonist because preliminary assays demonstrated it to be a more potent agonist than adenosine diphosphate (ADP). Two microliters of agonist (Collagen #385, Chronolog Corp.) were added to a final concentration of 2 mg/L to a mixture of 500 μL of citrated whole blood and 500 μL of 8.5 g/L NaCl. Platelet aggregation was measured as increased impedance between a pair of microelectrodes.

Plasma fatty acid profiles were determined using the one-step methylation procedure described by Sukhija and Palmquist[17] with the following modifications: 1 mL of nonadecanoic acid (19:0) in benzene (40 mg/L) was added as the internal standard to 300 μL of plasma, 1.5 mL of freshly prepared methanolic HCl (1:10, acetyl chloride:redistilled methanol) was the methylating agent, samples were methylated for 2 hr at 90° C, and the samples extracted into 500 μL of hexane. The methyl esters were analyzed on a Hewlett Packard gas

Table 1 Composition of experimental diets expressed as g/kg of diet (dry basis)

Ingredient	Diet			
	Corn/Soy/MCT*	ST*	PM*	ST/Can/Saf*
Corn oil	62.67	—	—	—
Soybean oil	15.59	—	—	—
MCT oil	78.30	—	93.79	—
MCT-structured lipid†	—	156.32	—	125.06
Menhaden oil	—	—	62.53	—
Canola oil	—	—	—	15.63
Safflower oil	—	—	—	15.63
Sodium caseinate	175.45	175.45	175.45	175.45
Isolated soy protein	26.71	26.71	26.71	26.71
Glucose polymer	590.53	590.76	590.76	590.76
Taurine	0.64	0.64	0.64	0.64
L-carnitine	0.55	0.55	0.55	0.55
Calcium lecithin	6.90	6.90	6.90	6.90
Vitamin premix‡	0.55	0.55	0.55	0.55
Trace mineral premix§	0.97	0.97	0.97	0.97
Ascorbic acid	2.21	2.21	2.21	2.21
Choline chloride	2.30	2.30	2.30	2.30
Magnesium chloride	4.78	4.78	4.78	4.78
Potassium chloride	2.39	2.39	2.39	2.39
Potassium citrate	9.79	9.79	9.79	9.79
Potassium hydroxide	0.55	0.55	0.55	0.55
Dipotassium phosphate	1.89	1.89	1.89	1.89
Sodium citrate	3.49	3.49	3.49	3.49
Sodium hydroxide	2.34	2.34	2.34	2.34
Tricalcium phosphate	9.33	9.33	9.33	9.33
Viscosity enhancers	2.07	2.07	2.07	2.07

*Abbreviations used: Corn, corn oil; Soy, soybean oil; MCT, medium-chain triacylglyceride; ST, MCT/marine oil-structured triacylglyceride; PM, Physical mixture of fatty acids equivalent to the MCT/marine oil-structured triacylglyceride; Can, canola oil; Saf, safflower oil.
†A mixture (by weight) of 60% MCT (Captex 300, Karlshamns Lipid Specialties USA, Columbus, OH USA) and 40% menhaden oil randomly transesterified into composite MCT/marine oil structured triacylglyceride molecules.
‡Supplied per kg of diet (DM): niacin, 120.1 mg; pantothenic acid, 77.7 mg; thiamin, 3.7 mg; riboflavin, 19.8 mg; folic acid, 19.1 mg; biotin, 2.3 mg; vitamin B_6, 15.5 mg; vitamin B_{12}, 53.0 mg; vitamin A, 25,507 IU; vitamin D_3, 1,518 IU; vitamin E, 169.1 IU; vitamin K, 310.5 IU.
§Supplied, mg/kg of diet (DM): Zn, 80.4; Fe, 61.1; Mn, 17.1; Cu, 7.2; Se, 0.26; Cr, 0.29; Mo, 0.57.

chromatograph (Model 5890, Avondale, PA USA) with a flame ionization detector and a 30 m × 0.25 mm id capillary column packed with 10% SP-2380 fused silica (Supleco Inc., Bellefonte, PA USA). The oven temperature was held at 100° C for 5 min then increased by 2° C/min to 200° C and maintained for 10 min. The injector and detector temperatures were 260° C and 275° C, respectively. Fatty acids were identified by comparing retention times to those of commercial standards (Nu-Check Prep, Elysian, MN USA).

An 800-μL aliquot of diluted platelets was lyophilized prior to methylation and nonadecanoic acid (12 mg/L) incorporated as previously described for plasma. Equipment was as previously described with the exception that the final oven temperature was 220° C and held for 5 min. Lyophilized feces (250 mg) were analyzed for fatty acid profile using conditions identical to those for platelets.

Muscle (1 g) or liver (600 mg) samples were homogenized in 5 mL of methanolic HCl prior to the addition of 2 mL of lauric acid (12:0) in benzene (500 mg/L) as an internal standard. Methylation was as previously described. Analytical conditions were as previously described for platelet fatty acid analyses.

Diets were analyzed for fatty acid profile following the lyophilization of a 500 μL sample and addition of 1 mL of pentadecanoic acid (15:0) in benzene (400 mg/L). Methylation and experimental conditions were as previously described for platelet analyses.

Colorimetric determination of plasma triacylglyceride concentration (Sigma Chemical Co., St. Louis, MO USA) and plasma non-esterified fatty acid concentration (Wako Chemicals USA, Inc., Dallas, TX USA) were conducted using commercially available kits.

Plasma, liver, and muscle samples were prepared for total cholesterol analyses as previously described[18] and based on the procedure of Duncan et al.[19] The chromatographic system was as previously described by Lepine et al.[18]

Statistics

Because preliminary statistical analyses indicated that some of the data (muscle and liver fatty acid profiles) were not normally distributed, these data were analyzed with nonparametric statistics. Data were first transformed to ranks and then analyzed with a one-way analysis of variance (ANOVA) on the ranks, in essence a Kruskal-Wallis Test. Day 1 data that were normally distributed were analyzed with a one-way ANOVA to assess comparability of the two replications. Nonesterified fatty acids and several of the platelet fatty acids exhibited a significant effect due to replication and were therefore analyzed with a two-way ANOVA with the main effects of treatment and replication. The remaining data were analyzed with either a one-way ANOVA (if normally distributed) or a Kruskal-Wallis Test (if non-normally distributed) in a manner similar to the initial statistical analyses of the replications. Analyses were conducted separately at days 12 and 19 because the experimental objective was to determine the effect of

Table 2 Fatty acid profiles of experimental diets (expressed as percentage of total fatty acids)*

Item	Lipid source			
	Corn/Soy/MCT†	ST†	PM†	ST/Can/Saf†
Caproic (6:0)	0.29	1.83	1.77	1.40
Caprylic (8:0)	33.64	47.28	44.26	36.82
Capric (10:0)	14.88	13.53	13.44	10.92
Lauric (12:0)	0.18	0.22	0.25	0.22
Myristic (14:0)	0.19	2.90	3.25	2.45
Palmitic (16:0)	6.12	7.91	8.87	8.27
Palmitoleic (16:1)	0.07	3.67	4.00	3.04
Heptadecanoic (17:0)	—	0.18	0.09	0.16
Stearic (18:0)	1.41	0.52	0.56	0.43
Oleic (18:1)	12.35	4.52	5.05	8.42
Linoleic (18:2n-6)	29.91	2.74	3.13	15.11
Linolenic (18:3n-3)	0.22	0.13	0.14	0.11
Arachidic (20:0)	—	0.08	0.10	0.08
Eicosenoic (20:1)	0.11	0.07	0.60	0.53
Eicosadienoic (20:2n-6)	—	0.09	0.09	0.08
Eicosatrienoic (20:3n-6)	0.50	0.59	0.64	0.59
Arachidonic (20:4n-6)	0.13	0.41	0.44	0.42
Eicosapentaenoic (20:5n-3)	—	5.71	5.60	4.67
Erucic (22:1)	—	0.11	0.14	0.12
Docosahexaenoic (22:6n-3)	—	6.12	6.14	5.01
Lignoceric (24:0)	—	0.37	0.39	0.31
Nervonic (24:1)	—	1.03	1.05	0.84

*Diets were analyzed by gas chromatography following lyophilization of a 500 μL sample and addition of 1 mL of pentadecanoic acid (15:0) in benzene (400 mg/L). Fatty acid methyl esters were formed based on the procedure of Sukhija and Palmquist.[17]
†Abbreviations used: Corn, corn oil; Soy, soybean oil; MCT, medium-chain triacylglyceride; ST, MCT/marine oil structured triacylglyceride; PM, Physical mixture of fatty acids equivalent to the MCT/marine oil structured triacylglyceride; Can, canola oil; Saf, safflower oil.

dietary lipid source and the effect of time on these responses. If significant differences were observed, Tukey's Studentized Range Test (HSD) was used to determine where the differences occurred. Results were considered to be statistically significant at the $P < 0.05$ level.

Results

Diet fatty acids

The analyzed fatty acid composition of the experimental diets was similar to that anticipated based on the lipid sources incorporated into each respective diet *(Table 2)*. The predominant fatty acids in corn oil (palmitic acid, 10.9%; oleic acid, 24.2%; and linoleic acid, 58.0%), soybean oil (palmitic acid, 10.3%; oleic acid, 22.8%; linoleic acid, 51.0%; and linolenic acid, 6.8%), and MCT oil (caproic acid, 3.8%; caprylic acid, 75.3%; and capric acid, 20.8%) determined the fatty acid pattern of the Corn/Soy/MCT diet and reflected the relative quantity of the lipid sources (40:10:50, wt:wt:wt) in the diet. Likewise, the fatty acid profiles of the ST and PM diets were as expected based on the predominant fatty acids in menhaden oil (myristic acid, 7.5%; palmitic acid, 18.3%; palmitoleic acid, 10.7%; oleic acid, 13.2%; arachidonic acid, 3.3%; eicosapentaenoic acid, 10.6%; and docosahexaenoic acid, 10.1%) and the MCT oil source as previously described. The fatty acid profiles for the ST and PM diets indicated that the desired similarity in overall fatty acid composition between these two diets was achieved. The fatty acid pattern of the ST/Can/Saf was similar to the ST and PM diets, but was higher in oleic acid and linoleic acid as a result of the substitution of 20% of the ST source with a 50:50 (wt:wt) mixture of canola oil and safflower oil. Canola oil contains 53.8% and 22.1% of oleic and linoleic acids, respectively, while safflower oil contains a high percentage (74.1%) of linoleic acid.

Plasma fatty acids

A small but significant ($P < 0.05$) increase in plasma caprylic acid concentration was noted with the PM diet compared with the Corn/Soy/MCT diet at day 12 *(Figure 1)*. However, by day 19 no effect ($P > 0.05$) of dietary treatment on plasma profile of the medium-chain fatty acids could be detected, despite the fact that these fatty acids comprised 49–63% of the total fatty acid of the dietary treatments.

With the exception of the medium-chain fatty acids, plasma fatty acid profiles reflected the dietary fatty acid profile. Dietary treatment had no effect ($P > 0.05$) on the plasma profile of palmitic, heptadecenoic, stearic, linolenic, arachidic, eicosenoic, eicosadienoic, eicosatrienoic, and erucic acids. Dietary treatment affected ($P < 0.05$) the plasma profile of myristic, palmitoleic, heptadecanoic, oleic, linoleic, arachidonic, eicosapentaenoic, docosahexaenoic, lignoceric, and nervonic acids *(Figures 1 and 2)*. The percentage of heptadecanoic acid in the plasma of pigs consuming the ST, PM, or ST/Can/Saf diets was increased ($P < 0.05$) on day 12 relative to those fed Corn/Soy/MCT. Heptadecanoic acid was not present in the Corn/Soy/MCT diet. The percentage of myristic acid

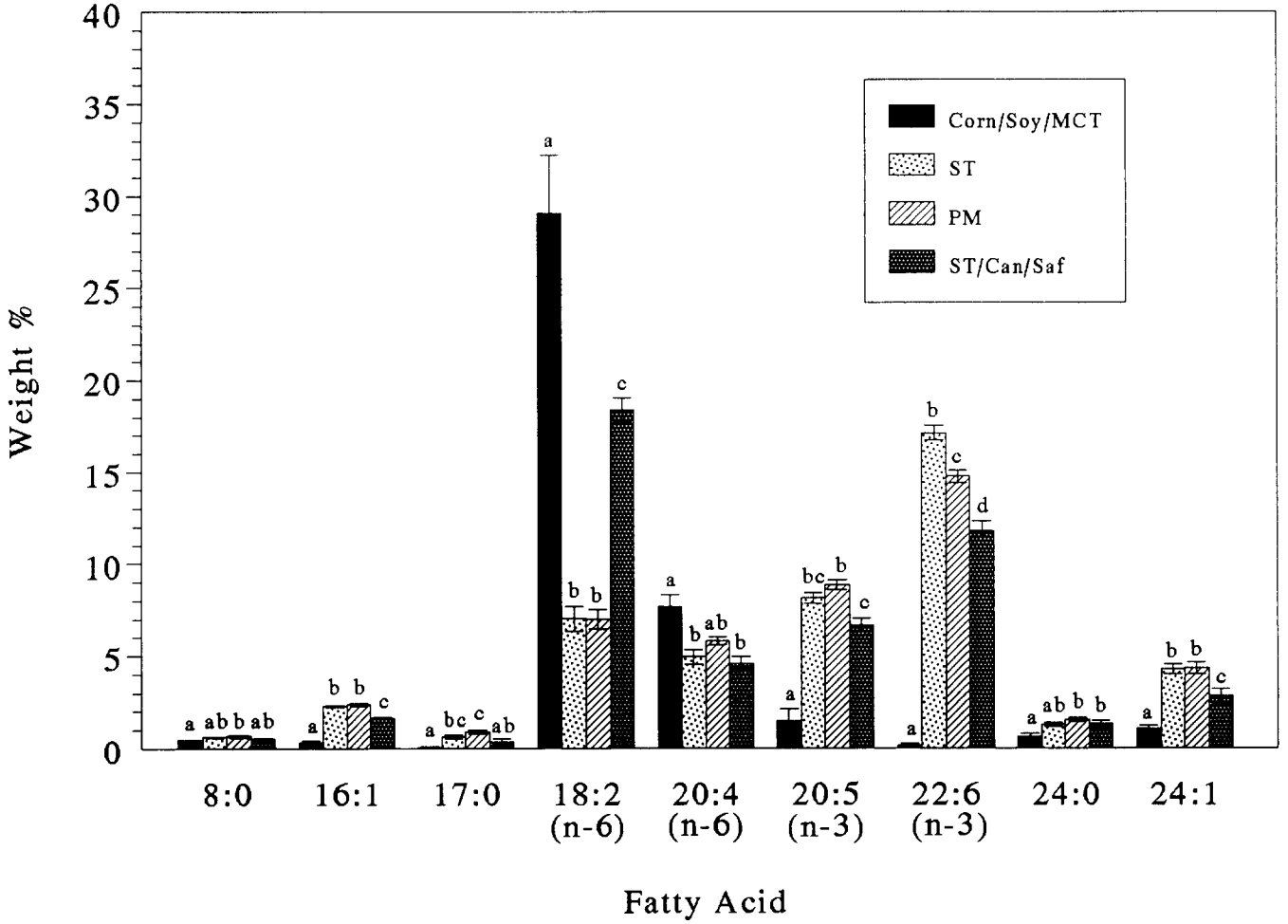

Figure 1 Plasma fatty acids affected by dietary treatment (day 12). Fatty acid methyl esters were formed by the procedure of Sukhija and Palmquist[17] following the addition of 1 mL of nonadecanoic acid (19:0) in benzene (40 mg/L) to 300 μL of plasma and subsequently analyzed by gas chromatography. Each bar is the mean ± SEM for six animals. Data were analyzed using a one-way ANOVA. Tukey's Studentized Range Test (HSD) was used if treatment differences were detected. Bars within fatty acid with different letters are different ($P < 0.05$).

in the plasma of pigs fed the ST, PM, or ST/Can/Saf diets was increased ($P < 0.05$) on day 19 relative to those consuming the Corn/Soy/MCT diet, reflecting the content of this fatty acid in menhaden oil. A similar response is evident on days 12 and 19 for the palmitoleic acid profile and is a function of dietary level. Specifically, the percentage of palmitoleic acid in the plasma of pigs fed the ST, PM, or ST/Can/Saf diets was increased ($P < 0.05$) on days 12 and 19 compared with pigs consuming the Corn/Soy/MCT diet. The percentage of palmitoleic acid in the plasma of pigs fed ST or PM was increased ($P < 0.05$) on days 12 and 19 compared with the ST/Can/Saf diet. Menhaden oil did indeed result in significant decreases in plasma linoleic (days 12 and 19) and oleic acid (day 19) levels in the present study relative to the Corn/Soy/MCT diet. This effect was due to the low level of these fatty acids in the ST and PM diet, which was directly reflected in the plasma. Linoleic acid was less depressed in plasma from pigs fed the ST/Can/Saf diet as a result of the relatively high concentration of this fatty acid in safflower oil. The effect of diet on plasma fatty acid composition is most evident for the long-chain and polyunsaturated fatty acids. Diets containing menhaden oil (ST, PM, and ST/Can/Saf) resulted in elevated ($P < 0.05$) plasma eicosapentaenoic, docosahexaenoic, lignoceric, and nervonic acid and a decreased ($P < 0.05$) plasma arachidonic acid percentage. The response of plasma eicosapentaenoic, docosahexaenoic, lignoceric, and nervonic acids percentages was the result of greater intakes of these fatty acids while plasma arachidonic acid response was antithetical to that of dietary intake.

Higher ($P < 0.05$) plasma concentrations of docosahexaenoic acid at day 12 in pigs fed ST relative to those fed PM suggests a possible effect of physical form on the intestinal absorption of this n-3 fatty acid *(Figure 1)*. The plasma level of eicosapentaenoic acid, the other prominent n-3 fatty acid in fish oil, was not affected by the physical form of the dietary lipid. Plasma eicosapentaenoic acid was decreased to a greater extent in pigs consuming safflower oil (ST/Can/Saf diet), potentially resulting from the lower percentage of marine oil in this diet relative to the ST and PM diets.

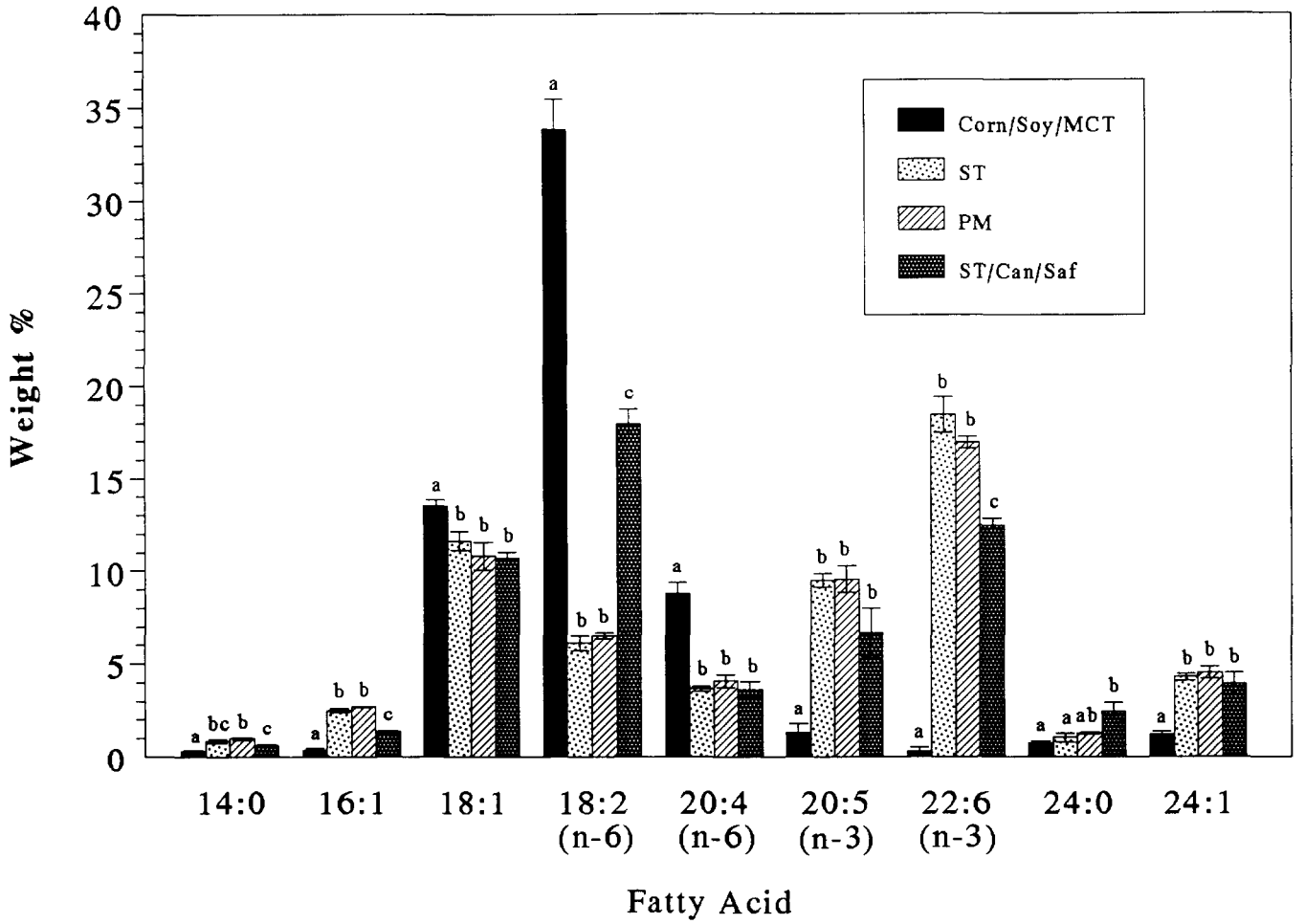

Figure 2 Plasma fatty acids affected by dietary treatment (day 19). Fatty acid methyl esters were formed by the procedure of Sukhija and Palmquist[17] following the addition of 1 mL of nonadecanoic acid (19:0) in benzene (40 mg/L) to 300 μL of plasma and subsequently analyzed by gas chromatography. Each bar is the mean ± SEM for six animals. Data were analyzed using a one-way ANOVA. Tukey's Studentized Range Test (HSD) was used if treatment differences were detected. Bars within fatty acid with different letters are different ($P < 0.05$).

Table 3 Effect of lipid source on serum triacylglyceride and nonesterified fatty acid (NEFA) concentrations in the growing pig*

Item	Lipid source			
	Corn/Soy/MCT†	ST†	PM†	ST/Can/Saf†
Triacylglyceride (mmol/L)				
Day 1	0.58 ± 0.10	0.62 ± 0.10	0.74 ± 0.10	0.74 ± 0.15
Day 12	0.55 ± 0.06	0.68 ± 0.07	0.66 ± 0.08	0.80 ± 0.09
Day 19	0.58 ± 0.02	0.55 ± 0.12	0.65 ± 0.14	0.75 ± 0.08
NEFA (mg/L)				
Day 1	15.0 ± 1.6	20.6 ± 2.7	18.1 ± 3.6	18.6 ± 3.2
Day 12	39.1 ± 9.2	42.1 ± 14.0	26.5 ± 5.7	36.1 ± 7.4
Day 19	78.6 ± 11.2	56.6 ± 11.9	46.2 ± 8.8	66.3 ± 8.9

*Values represent means ± SEM. Triacylglycerides were statistically analyzed by a one-way ANOVA. NEFA were statistically analyzed using a two-way ANOVA with the main effects of treatment and replication. Analyses were conducted separately at each time point (days 1, 12, and 19).
†Abbreviations used: Corn, corn oil; Soy, soybean oil; MCT, medium-chain triacylglyceride; ST, MCT/marine oil structured triacylglyceride; PM, Physical mixture of fatty acids equivalent to the MCT/marine oil structured triacylglyceride; Can, canola oil; Saf, safflower oil.

The effect of dietary lipid source on plasma triacylglyceride concentration is presented in *Table 3*. The initial plasma triacylglyceride concentration was typical for pigs approximately 60 days of age and fed diets not supplemented with lipid.[20] No effect ($P > 0.05$) of dietary treatment on plasma triacylglyceride concentrations was noted.

As with plasma triacylglyceride concentration, plasma nonesterified fatty acid concentration was not affected ($P > 0.05$) by dietary treatment *(Table 3)*.

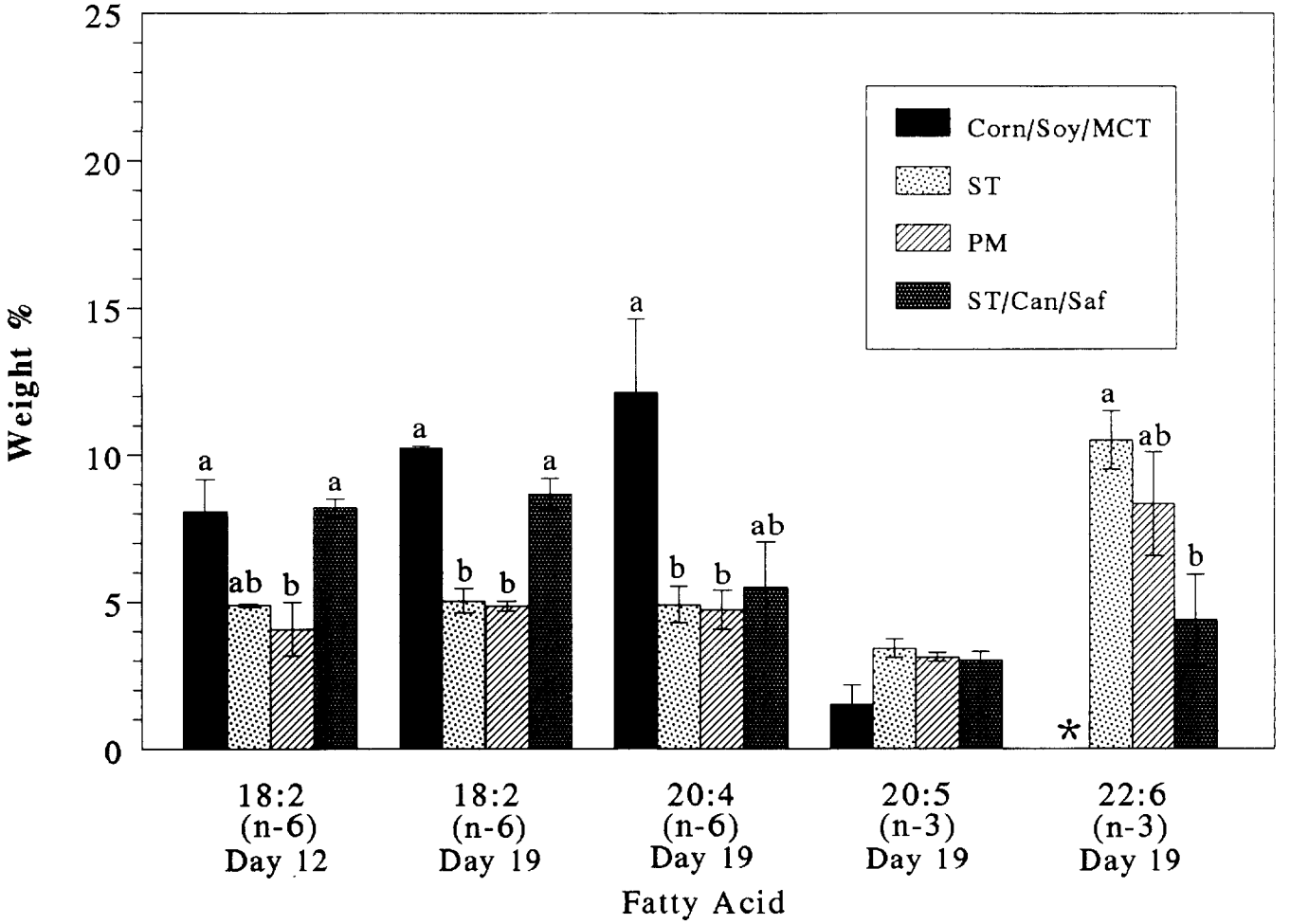

Figure 3 Platelet fatty acids affected by dietary treatment. Fatty acid methyl esters were formed by the procedure of Sukhija and Palmquist[17] following the addition of 1 mL of nonadecanoic acid (19:0) in benzene (40 mg/L) to 300 μL of plasma and subsequently analyzed by gas chromatography. Each bar is the mean ± SEM for six animals. Data were analyzed using a two-way ANOVA with the main effects of treatment and replication. Tukey's Studentized Range Test (HSD) was used if treatment differences were detected. Bars within fatty acid with different letters are different ($P < 0.05$). *Not detectible.

However, there was a marked elevation above baseline plasma nonesterified fatty acid concentration following the provision of any of the dietary lipid sources.

Platelet fatty acid profiles

Only linoleic, arachidonic, and docosahexaenoic acid were significantly affected by dietary treatment *(Figure 3)*. Platelets from pigs fed the ST or PM diets had lower ($P < 0.05$) linoleic and arachidonic acid percentages (day 19) as compared with Corn/Soy/MCT pigs. This change in fatty acid composition was compensated for by an increased ($P < 0.05$) docosahexaenoic acid level (day 19) and a trend for an increased eicosapentaenoic acid level ($P > 0.05$). A treatment by replicate interaction was observed for eicosapentaenoic acid at day 12 with higher values for PM- and ST-fed pigs in replicate 1 and higher values for pigs consuming Corn/Soy/MCT or Can/Saf/ST diets in replicate 2. The explanation for this interaction is unclear and this interaction was not present at day 19.

Tissue fatty acid profiles

In the present study, dietary treatments containing menhaden oil (ST, PM, and ST/Can/Saf) increased ($P < 0.05$) the percentage of pentadecenoic, palmitic, heptadecanoic, heptadecenoic, stearic, linolenic, eicosapentaenoic, docosahexaenoic, and nervonic acids and decreased ($P < 0.05$) the percentage of linoleic, eicosadienoic, and arachidonic acids in the liver relative to the diet devoid of menhaden oil (Corn/Soy/MCT) *(Table 4)*. The ST/Can/Saf diet was less effective than the ST and PM diets in lowering linoleic acid and elevating eicosapentaenoic acid.

The effect of diet on muscle fatty acid profile was less pronounced than for the liver although the pattern of change was generally similar. Dietary effects included an increased percentage of myristic, palmitic, stearic, linolenic, and eicosapentaenoic acids and a decreased percentage of linoleic and eicosadienoic acids in the muscle of pigs fed diets containing menhaden oil *(Table 5)*. The physical form of the dietary lipid source (ST

Table 4 The effect of dietary lipid source on liver fatty acid profile in the growing pig (expressed as percentage of identifiable acids)*

Item	Lipid source			
	Corn/Soy/MCT†	ST†	PM†	ST/Can/Saf†
Caprylic (8:0)	0.01 ± 0.01	0.02 ± 0.02	0.04 ± 0.03	0.03 ± 0.02
Capric (10:0)	0.13 ± 0.01	0.17 ± 0.02	0.20 ± 0.03	0.16 ± 0.02
Tridecanoic (13:0)	0.02 ± 0.02	0.03 ± 0.03	—	—
Myristic (14:0)	0.33 ± 0.02^{a}	0.45 ± 0.12^{b}	0.61 ± 0.04^{b}	0.50 ± 0.03^{ab}
Pentadecanoic (15:0)	—	0.02 ± 0.02	—	—
Pentadecenoic (15:1)	0.30 ± 0.02^{a}	0.46 ± 0.02^{b}	0.43 ± 0.02^{b}	0.35 ± 0.07^{ab}
Palmitic (16:0)	12.51 ± 0.18^{a}	17.19 ± 0.34^{b}	17.14 ± 0.30^{b}	15.79 ± 0.67^{b}
Palmitoleic (16:1)	0.32 ± 0.04^{a}	1.53 ± 0.03^{b}	1.58 ± 0.06^{b}	0.92 ± 0.04^{c}
Heptadecanoic (17:0)	0.42 ± 0.02^{a}	0.89 ± 0.05^{b}	0.90 ± 0.03^{b}	0.78 ± 0.03^{b}
Heptadecenoic (17:1)	0.51 ± 0.02^{a}	0.91 ± 0.04^{b}	0.91 ± 0.02^{b}	0.77 ± 0.02^{c}
Stearic (18:0)	29.25 ± 0.06^{a}	41.87 ± 0.35^{b}	40.78 ± 0.51^{b}	38.24 ± 0.69^{c}
Oleic (18:1)	9.17 ± 0.57	8.82 ± 0.12	8.88 ± 0.48	8.28 ± 0.16
Linoleic (18:2n-6)	21.25 ± 0.47^{a}	5.03 ± 0.34^{b}	5.38 ± 0.26^{b}	13.65 ± 0.17^{c}
Linolenic (18:3n-3)	0.39 ± 0.03^{a}	0.64 ± 0.03^{b}	0.57 ± 0.03^{bc}	0.53 ± 0.01^{c}
Arachidic (20:0)	0.20 ± 0.01^{a}	0.31 ± 0.01^{b}	0.22 ± 0.04^{ab}	0.18 ± 0.03^{a}
Eicosenoic (20:1)	0.14 ± 0.01^{a}	0.19 ± 0.05^{b}	0.17 ± 0.02^{ab}	0.22 ± 0.01^{b}
Eicosadienoic (20:2n-6)	0.72 ± 0.04^{a}	0.14 ± 0.04^{b}	0.18 ± 0.01^{b}	0.29 ± 0.01^{c}
Eicosatrienoic (20:3n-6)	0.99 ± 0.14^{a}	0.61 ± 0.04^{b}	0.62 ± 0.02^{b}	0.84 ± 0.05^{a}
Arachidonic (20:4n-6)	19.37 ± 0.53^{a}	9.19 ± 0.28^{b}	9.82 ± 0.23^{b}	9.18 ± 0.28^{b}
Eicosapentaenoic (20:5n-3)	2.12 ± 0.08^{a}	9.14 ± 0.39^{b}	9.39 ± 0.43^{b}	6.51 ± 0.46^{c}
Docosahexaenoic (22:6n-3)	0.34 ± 0.02^{a}	0.42 ± 0.01^{ab}	0.44 ± 0.01^{b}	1.02 ± 0.62^{b}
Lignoceric (24:0)	1.24 ± 0.03	1.38 ± 0.04	1.27 ± 0.09	1.31 ± 0.06
Nervonic (24:1)	0.29 ± 0.02^{a}	0.60 ± 0.18^{b}	0.46 ± 0.05^{b}	0.46 ± 0.10^{ab}

*Values represent means ± SEM. These data were determined to be nonparametric and thus analyzed using a Kruskal-Wallis Test. Means in a row with different superscripts are significantly different ($P < 0.05$).
†Abbreviations used: Corn, corn oil; Soy, soybean oil; MCT, medium-chain triacylglyceride; ST, MCT/marine oil structured triacylglyceride; PM, Physical mixture of fatty acids equivalent to the MCT/marine oil structured triacylglyceride; Can, canola oil; Saf, safflower oil.

Table 5 The effect of dietary lipid source on muscle fatty acid profile in the growing pig (expressed as percentage of identifiable acids)*

Item	Lipid source			
	Corn/Soy/MCT†	ST†	PM	ST/Can/Saf†
Caprylic (8:0)	—	0.01 ± 0.01	0.02 ± 0.01	0.01 ± 0.01
Capric (10:0)	0.27 ± 0.02	0.27 ± 0.01	0.27 ± 0.01	0.28 ± 0.01
Tridecanoic (13:0)	0.02 ± 0.02	—	0.04 ± 0.04	0.04 ± 0.04
Myristic (14:0)	1.07 ± 0.06^{a}	1.37 ± 0.03^{b}	1.28 ± 0.08^{ab}	1.32 ± 0.05^{b}
Pentadecanoic (15:0)	—	0.06 ± 0.03	0.04 ± 0.03	0.06 ± 0.02
Pentadecenoic (15:1)	2.01 ± 0.37	2.34 ± 0.32	2.22 ± 0.42	2.47 ± 0.39
Palmitic (16:0)	20.23 ± 0.81^{a}	23.04 ± 0.47^{b}	22.77 ± 0.58^{b}	22.14 ± 0.58^{ab}
Palmitoleic (16:1)	2.52 ± 0.21^{ab}	3.63 ± 0.11^{bc}	3.84 ± 0.21^{c}	2.99 ± 0.19^{b}
Heptadecanoic (17:0)	0.26 ± 0.03	0.42 ± 0.07	0.38 ± 0.05	0.43 ± 0.04
Heptadecenoic (17:1)	1.11 ± 0.22	1.42 ± 0.25	1.25 ± 0.24	1.33 ± 0.20
Stearic (18:0)	12.93 ± 0.19^{a}	15.39 ± 0.40^{b}	14.26 ± 0.47^{ab}	14.45 ± 0.50^{b}
Oleic (18:1)	32.28 ± 2.44	34.09 ± 2.39	36.18 ± 2.84	30.62 ± 2.40
Linoleic (18:2n-6)	19.12 ± 1.73^{a}	9.24 ± 1.15^{b}	9.44 ± 1.38^{b}	15.43 ± 1.25^{a}
Linolenic (18:3n-3)	0.21 ± 0.01^{a}	0.39 ± 0.02^{b}	0.36 ± 0.02^{bc}	0.32 ± 0.02^{c}
Arachidic (20:0)	0.16 ± 0.02	0.16 ± 0.02	0.14 ± 0.03	0.16 ± 0.02
Eicosenoic (20:1)	0.57 ± 0.05	0.58 ± 0.07	0.50 ± 0.12	0.49 ± 0.09
Eicosadienoic (20:2n-6)	0.50 ± 0.03^{a}	0.23 ± 0.03^{b}	0.25 ± 0.03^{b}	0.33 ± 0.03^{b}
Eicosatrienoic (20:3n-6)	0.58 ± 0.08	0.56 ± 0.07	0.53 ± 0.09	0.62 ± 0.08
Arachidonic (20:4n-6)	5.25 ± 0.89	4.44 ± 0.67	4.23 ± 0.84	4.53 ± 0.59
Eicosapentaenoic (20:5n-3)	0.58 ± 0.10^{a}	1.98 ± 0.23^{b}	1.75 ± 0.19^{b}	1.61 ± 0.14^{b}
Lignoceric (24:0)	0.26 ± 0.04	0.31 ± 0.05	0.30 ± 0.06	0.30 ± 0.04
Nervonic (24:1)	0.08 ± 0.03	0.07 ± 0.03	0.04 ± 0.03	0.07 ± 0.03

*Values represent means ± SEM. These data were determined to be nonparametric and thus analyzed using a Kruskal-Wallis Test. Means in a row with different superscripts are significantly different ($P < 0.05$).
†Abbreviations used: Corn, corn oil; Soy, soybean oil; MCT, medium-chain triacylglyceride; ST, MCT/marine oil structured triacylglyceride; PM, Physical mixture of fatty acids equivalent to the MCT/marine oil structured triacylglyceride; Can, canola oil; Saf, safflower oil.

versus PM) did not affect the fatty acid profiles of the liver or muscle. Plasma, liver, and muscle cholesterol concentrations were not affected ($P > 0.05$) by dietary lipid source (data not shown).

Discussion

Physical form (structured versus non-structured triacylglyceride) did not affect plasma fatty acid profiles as anticipated based on the potential advantage associated with the incorporation of medium-chain fatty acids into structured triacylglycerides. It can be noted however that plasma nonesterified fatty acids concentrations tended to be higher for the ST and ST/Can/Saf treatments compared with the PM treatment *(Table 3)*, suggesting that total plasma fatty acid concentration may have been affected by using the structured triacylglyceride treatments. Because further study is required to evaluate this response, the remaining discussion will focus on the effect of dietary fatty acid profiles on plasma and tissue responses.

The results of the present study are in contrast to those of Cera et al.,[21] who observed approximately 28–30% of plasma fatty acids as caprylic acid and capric acid following the incorporation of 8% medium-chain fatty acids into the diet of weanling pigs. The medium-chain fatty acid content of the diets in the present experiment is similar to that of Cera et al.[21] (7.58% for the Corn/Soy/MCT and ST/Can/Saf diets; 6.25% for the ST and PM diets) but less effective in altering plasma medium-chain fatty acid levels. The pigs in the study conducted by Cera et al.[21] were not fasted prior to blood sampling, which most likely accounts for the disparity in the plasma medium-chain fatty acid profiles compared with those observed in the present study. Additionally, age differences, diet formulation, or frequency of diet consumption may have influenced the differential plasma fatty acid response between these studies.

Plasma fatty acid responses similar to those reported here have been reported in healthy human subjects supplemented with fish oil concentrates.[22] Specifically, dietary fish oil has been demonstrated to significantly increase plasma nervonic, eicosapentaenoic, and docosahexaenoic acid concentrations in humans, while concurrently decreasing plasma concentration of oleic and linoleic acids.[23]

In contrast to the present study previous investigators[23,24] have observed no response of n-3 fatty acids on the plasma percentage of arachidonic acid. The explanation for this disparity is unclear, but Thomson et al.[25] demonstrated an effect of n-3 fatty acids on intestinal mucosa weight resulting in a depressed palmitic acid absorption in the jejunum. The effect on arachidonic acid absorption was not evaluated.

In contrast to the present study, Bronsgeest-Schoute et al.[24] and Jensen et al.[22] demonstrated lowered plasma triacylglyceride concentrations in normotriacylglyceridemic humans supplemented with n-3 polyunsaturated fatty acids. This hypotriacylglyceridemia reported in humans results from a lowered hepatic triacylglyceride synthesis and subsequently reduced very low density lipoprotein production.[11,26] Although relatively high dietary intakes of n-3 polyunsaturated fatty acids (docosahexaenoic + eicosapentaenoic acids) were required (5.979 g/day; 8.19 g/day)[22,26] to lower plasma triacylglyceride concentrations in humans, this does not account for the lack of hypotriacylglyceridemic effect of fish oil in the present study. Estimated daily intakes of eicosapentaenoic and docosahexaenoic acids for pigs consuming fish oil-supplemented diets (ST, PM, and ST/Can/Saf) ranged from 9.95 to 14.63 g/d. It is likely that the mixture of the fish oil with other lipid sources such as medium-chain fatty acids may have masked the hypotriacylglyceridemic response of the n-3 polyunsaturated fatty acids. Indeed, Kritchevsky and Tepper[27] and Leveille et al.[28] demonstrated hepatic fatty acid synthesis to be enhanced in rats fed a medium-chain triacylglyceride source relative to rats fed corn oil. Enhanced lipogenesis resulted not only from chain elongation but also from fatty acid synthesis from acetyl CoA provided by the rapidly oxidizable medium-chain fatty acids.

Cera et al.[21] observed an increased plasma nonesterified fatty acid concentration in postweaning (nonfasted) pigs supplemented with medium-chain fatty acids relative to pigs supplemented with tallow. The lack of any treatment effect in the present study probably relates to the mixture of medium-chain and long-chain fatty acids in each of the treatment diets and fasting prior to blood sampling.

In general agreement with the present study, Mueller and Talbert[11] observed that the ingestion of n-3 polyunsaturated fatty acids increased the incorporation of eicosapentaenoic and docosahexaenoic acids into tissue phospholipids, including platelets. Likewise, Knapp et al.[29] and Galloway et al.[30] found increased eicosapentaenoic acid concentration in platelets from rats supplemented with dietary eicosapentaenoic acid. The increased platelet eicosapentaenoic acid concentration appeared to be at the expense of arachidonic acid in the tissue of the rat. Wahle and Brown[31] speculated that the effects of dietary fish oil on platelet fatty acid composition are caused by a competitive interaction between eicosapentaenoic acid and arachidonic acid for incorporation into phospholipids and further metabolism to eicosanoids. Indeed, ingestion of n-3 polyunsaturated fatty acids leads to the partial replacement of arachidonic acid with eicosapentaenoic acid and docosahexaenoic acid into tissue phospholipids.[32]

Many studies have demonstrated a diminished platelet aggregation[10,33–36] and prolonged bleeding time[29,33,35,37,38] as a result of dietary n-3 polyunsaturated fatty acids shifting the prostaglandin, thromboxane, and leukotriene balance toward one favoring vasodilation and anti-aggregation. In contrast to these reports, the effects of dietary lipid source on platelet fatty acid profile noted in the present study did not significantly alter platelet aggregation or blood clotting time *(Table 6)*. The effect of dietary lipid source on plasma concentrations of prostaglandins, leukotrienes, and thromboxanes were not determined in the present study. The daily intake of n-3 polyunsaturated fatty acid in the present study may have been below that required to

Table 6 Effect of lipid source on platelet aggregation and blood clotting time in the growing pig*

Item	Lipid source			
	Corn/Soy/MCT†	ST†	PM†	ST/Can/Saf†
Platelet aggregation (Ω)				
Day 1	14.12 ± 2.27	12.45 ± 2.15	9.25 ± 1.27	13.53 ± 1.68
Day 12	15.44 ± 3.41	10.96 ± 1.62	12.60 ± 1.51	18.04 ± 2.61
Day 19	9.81 ± 1.34	8.54 ± 0.79	6.76 ± 1.18	8.53 ± 0.47
Clotting time(s)				
Day 1	89.3 ± 9.7	83.8 ± 12.8	79.5 ± 12.0	90.8 ± 7.6
Day 12	73.4 ± 14.3	63.0 ± 13.5	73.0 ± 10.5	88.5 ± 17.4
Day 19	86.7 ± 14.4	100.2 ± 5.6	73.3 ± 14.1	85.3 ± 8.5

*Values represent means ± SEM. Data were statistically analyzed by a one-way ANOVA. Analyses were conducted separately at each time point (days 1, 12, and 19).
†Abbreviations used: Corn, corn oil; Soy, soybean oil; MCT, medium-chain triacylglyceride; ST, MCT/marine oil structured triacylglyceride; PM, Physical mixture of fatty acids equivalent to the MCT/marine oil structured triacylglyceride; Can, canola oil; Saf, safflower oil.

produce antiplatelet effects because these effects appear to be dose-dependent.[30,39] Additionally, a more pronounced effect on platelet fatty acid profiles and platelet aggregation may have resulted if the duration of feeding in the present study had been extended.

Although the ability of dietary fat source to change the tissue fatty acid composition of swine has been clearly documented,[40–42] the effect of dietary fish oil on the tissue fatty acid composition of swine has not been determined. The results of the present study are in general agreement with those reported in mice[43] and rats[44] supplemented with dietary fish oil. Inclusion of safflower oil[45,46] or canola oil[47,48] into the diet has been demonstrated to enhance the deposition of linoleic acid into pig tissues as a consequence of the increased dietary concentration of this unsaturated fatty acid, a finding consistent with the ST/Can/Saf diet in the present study.

The present study demonstrates that serum and tissue fatty acid profiles of the growing pig are affected by dietary fatty acid profile but only marginally affected by the physical nature (structured or non-structured) of the triacylglycerides employed here. The reason for the relative lack of effect of physical form on fatty acid profile is uncertain and requires an evaluation of intestinal absorption and subsequent metabolism of the fatty acids provided by these lipid sources.

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