EFFECTS OF FLAXSEED SUPPLEMENTATION AND EXOGENOUS HORMONES ON FINISHING PERFORMANCE, CARCASS CHARACTERISTICS, AND PLASMA AND LONGISSIMUS MUSCLE FATTY ACID PROFILES IN FINISHING CATTLE

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ABSTRACT

The effects of supplementing forms of flaxseed on plasma and longissimus muscle (LM) fatty acid (FA) composition, finishing performance, and carcass characteristics were evaluated in five studies. In study 1, steers were fed diets with soy oil (SO), ground flaxseed (Flaxseed), or urea formaldehyde condensation polymer treated flaxseed (UFCP). In study 2, steers were fed diets with SO, linseed oil (LO), or a combination of flaxseed and field peas that was extruded (LinPro). Feeding flaxseed products increased ($P < 0.01$) α-linolenic acid (ALA), omega-3 FA, and decreased ($P < 0.01$) n-6:n-3 in LM compared to cattle fed SO. Feeding LinPro increased ($P < 0.01$) ALA, omega-3 FA, and decreased ($P < 0.01$) n-6:n-3 in LM compared to steers fed SO or LO. In studies 3 and 4, steers were fed diets with and without Flaxseed and implanted or not. Implanting improved ($P \leq 0.05$) DMI, ADG, feed efficiency, HCW, and LM area compared to cattle not implanted. In study 4, cattle fed Flaxseed had increased ($P < 0.01$) ALA and omega-3 FA, and decreased ($P < 0.01$) n-6:n-3 in LM compared to cattle fed SO. In study 5, heifers were fed diets with 0% or 5% linseed meal, and administered with or without exogenous hormones (NHTC). Administering exogenous hormones improved ($P \leq 0.02$) DMI, ADG, G:F, and HCW compared to NHTC cattle. Omega-3 FA increased in LM when cattle were supplemented with flaxseed products. Cattle fed LinPro achieved the highest levels of ALA and omega-3 FA. Flaxseed products did not interact with implants as a natural growth promoter in finishing cattle.

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FORWARD

This thesis was written in the form of three separate manuscripts which serve as Chapter 2, Chapter 3, and Chapter 4. Chapter 2, 3, and 4 contain data obtained from Holstein steers evaluating the effects of flaxseed supplementation in several forms on finishing performance and carcass characteristics. Chapter 4 also evaluated the effect of implant status on finishing performance and carcass characteristics. Chapter 4 also contains data obtained from crossbred heifers evaluating the effects of the interaction between linseed meal supplementation and management regime on finishing performance and carcass characteristics. These chapters were prepared to follow the guidelines suggested for contributors to the *Journal of Animal Science*.

CHAPTER 1: A REVIEW OF THE LITERATURE

Flaxseed

Flaxseed *(Linum usitatissiumum),* also known as linseed, is grown throughout the Northern United States and Canada. The small, dark oilseeds are processed to release the oil from the inner seed. Cold press extraction uses only mechanical pressure to extract the oils, leaving approximately 8% oil residues in the remaining solids. Prepress solvent extraction uses mechanical pressure and solvents to more effectively remove the oil, with as much as 1.5% oil left in the residues. The resulting flaxseed oil contains elevated levels of α-linolenic acid (αLA) and may be utilized as a feedstuff rich in energy. The residual solids after oil extraction are called linseed meal. The protein of flaxseed is concentrated into the linseed meal, making it a good source of protein in livestock diets. Depending on the oil extraction method and if solvent soaps are discarded into the solids, linseed meal may contain nearly 0 to 8% oil. The remaining seed coat in linseed meal contains lignan, a kind of phytoestrogen capable of manipulating estrogenic biological activities. The entire seed may also be ground and referred to as ground flaxseed. Ground flaxseed contains both αLA and lignan, and provides protein and energy as a feedstuff. Flaxseed also can be extruded, a process where heat and pressure are applied as the seeds pass through a die. Extruded flaxseed contains both αLA and lignan. The heat applied protects the protein from ruminal degradation, providing bypass protein for livestock diets. Overall, flaxseed in its many forms may be a good source of energy and protein in livestock diets.

The Phytoestrogen Lignan

Lignan is a class of phytoestrogen found in plants. Members include lariciresinol, isolariciresinol, matairseinol and secoisolariciresinol (SECO). These lignans possess a

2,3-dibenzylbutane skeleton, distinguishing them from other phytoestrogens. The hull found of flaxseed contains approximately 28 to 369 mg/100 g (Raffaelii et al, 2002) of the lignan precursor secoisolariciresinol diglucoside (SDG), which is lacking in estrogenic activity.

Metabolism of Secoisolariciresinol Diglucoside

The metabolism of SDG produces the metabolites enterodiol (ED) and enterolactone (EL). These metabolites are formed by mammalian gut microbes, and therefore are coined as mammalian lignans or enterolignans. The metabolic process of forming ED and EL begins with SECO. The conversion of SDG releases SECO and initiates the metabolism of enterolignans. In a study by Thompson et al. (1991), lignan production from flaxseed SDG was shown to generate the highest concentration of enterolignans when compared to 67 other food products.

The metabolism of SDG has been studied primarily in humans and mice. The site of conversion and the complex interactions between several bacterial strains has been established through *in vitro* and *in vivo* experiments. The *in vitro* findings from Wang et al. (2000) helped identify the conversion pathway of SDG shown in Figure 1.

First, two glucoses are removed from SDG to form SECO. Strains of *Bacteroides distasonis, B. fragilis, B. ovatus, Clostridium cocleatum* and *C. ramosum* have the ability to perform this deglycosylation steps (Clavel et al, 2006). Next, a methyl group is removed, yielding demethylated metabolite 22. Bacterial strains *Butyribacterium methylotrophicum, Eubacterium callanderi, E. limosum* and *Peptostreptococcus productus* are capable of this demethylation (Clavel et al., 2006). From metabolite 22, the pathway diverges to two different paths, but both result in formation of the same end

product. Following the left pathway, the demethylated metabolites 22 and 24 appear within 20 to 30 h after SDG incubation (Wang, 2000). Taking the right pathway, the dehydroxylated metabolites 23 and 25 materialize after 48 h (Wang, 2000). The timeline established by metabolite appearance suggests demethylation occurs first. After diverting to the left, metabolite 22 is demethylated a second time to form metabolite 24. Following demethylation, metabolite 24 is dehydrolyzed by *Eubacterium* sp. strain SDG-2 to form metabolite 25. Metabolite 25 is again dehydrolyzed by *Eubacterium* sp. strain SDG-2 to yield ED. Finally ED is dehydrogenated to form EL (Clavel et al., 2006).

These metabolic steps have not been confirmed in ruminants. A metabolism study with fistulated goats has revealed SDG metabolism does occur in the rumen, though the steps are unknown (Zhou et al., 2009). After administering SDG via jugular catheter at 1 mg/kg of BW, ED and EL appeared in both rumen fluid and serum. This suggests ruminal microbes can metabolize SDG into enterolignans that can be absorbed into the bloodstream. Supplementation of SDG affected ruminal metabolism, increasing microbal CP concentration and total VFA concentrations, while decreasing ruminal pH and ammonia–nitrogen concentration. Microbial profile of the rumen also was affected by SDG supplementation. These results demonstrate that SDG supplementation influences ruminal bacterial composition and the metabolism of carbohydrates and nitrogenous compounds in goats. Previous research has demonstrated that feeding flaxseed can lead to formation of ED and EL. Thangavelu et al. (2008) observed dairy cows supplemented with whole flaxseed to have increased fecal concentrations of SDG and ED than those supplemented with saturated fatty acids or sunflower seeds. Petit and Gagnon (2009) fed various concentrations of linseed meal (0, 50, 100 or 150 g/kg of diet DM) to lactating

dairy cows. Milk concentrations of EL increased ($P < 0.05$) linearly with increasing concentrations of linseed meal compared to cows fed control diets. The enterolignan ED was not detectable in milk, suggesting ED was completely dehydrogenated to form EL, that it was not absorbed, or that it was metabolized post-absorptively. Petit et al. (2009) fed 0 or 20% flax hulls to ruminally-fistulated Holstein cows. Fistulated cows fed flax hulls had increased ($P < 0.05$) concentrations of EL in ruminal fluid and milk compared to control cows, implying that SDG of flax hulls is metabolized by ruminal microbes and transferred into the bloodstream. The works of Thangavelu et al. (2008), Petit and Gagnon (2009), and Petit et al. (2009) demonstrate the SDG in flaxseed, fed as the whole seed, linseed meal, or as flax hulls, is metabolized in the rumen and absorbed into the bloodstream.

Estrogenic Properties of Enterolignans

The metabolites ED and EL can exert estrogenic effects, interacting with ligands that bind estradiol. These ligands include estrogen receptors (ER) and proteins that modulate availability of estrogen to estrogen-dependent tissues. The mammalian lignans ED and EL may be either agonistic or antagonistic (Kuiper et al., 1998), and the estrogenic effect conferred is influenced by several factors, including phytoestrogen potency, binding affinity to ER, and the type of ER. Compared to other phytoestrogens, EL may appear to be ineffectual with low potency and weak ER binding (Mueller et al., 2004). Though ED and EL are relatively weak phytoestrogens, studies have shown their interaction with estrogen-binding compounds may lead to significant outcomes.

Effects of Enterolignan Binding to Estrogen Receptors (ER)

The binding of an ER ligand activates ER, causing a genomic reaction that upregulates or downregulates specific genes of mRNA. Proteins are produced from the translated mRNA to function in hormone-dependent tissues regulating cell proliferation, differentiation, and homeostasis (Offermans and Rosenthal, 2008). The sub-type of ER affected, whether ERα or ERβ, and the tissues containing the ER dictate the actions that follow.

The enterolignan EL has been shown to bind more strongly to ERα than to ERβ (Mueller et al., 2004), suggesting EL is more likely to affect tissues containing primarily ERα. The distribution of ER sub-types has been determined in bovine gastrointestinal compartments (Pfaffl et al., 2003), various organs and skeletal muscles (Pfaffl et al., 2001), identifying ERα as the predominant sub-type in the jejunum, whereas ERβ is more prevalent in the liver, rumen, and skeletal muscles. These results suggest EL may exert stronger effects on jejunum gene expression and function than compared to the ruminant stomach or skeletal muscles. To explore this, O'Neil et al. (2009) used ovariectomized ewes fed 12.5% linseed meal for 0, 1, 7, or 14 d and implanted with estradiol-17β (E_2) for 0, 6, or 24 h before collecting the jejunum for analysis of jejunal mucosa and mRNA expression. Estradiol-17β increased jejunum mass, whereas linseed meal interacted with E_2 to decrease jejunal cellular proliferation and influence jejunal gene expression. The authors speculated the enterolignans from the metabolism of linseed meal caused this interaction, affecting ERα activity. O'Neil et al. (2009) suggested further investigation as to how this interaction may affect nutrient absorption in females exposed to estrogen.

Though EL binds more strongly to ERα than ERβ, EL has been shown to interact with ERβ of rat skeletal muscle (Zhou, et al. 2009). Feeding flaxseed lignans (50 ppm) with another phytoestrogen, daidzein at 5 ppm, upgraded ERβ expression in soleus muscle and the hypothalamus, increased the growth of the femoral muscle, and increased serum testosterone levels. Zhou et al. (2009) suggested the phytoestrogens may have regulated serum testosterone levels by binding to ERβ in the hypothalamus, resulting in decreased protein catabolism and increased hypertrophy of skeletal muscle cells. Bovine skeletal muscles are comprised primarily of ERβ (Pfaffl et al., 2001). The ERβ from bovine skeletal muscle may respond in a manner similar to that of ERβ in rat muscle, stimulating the growth of bovine skeletal muscle. Studies have not been completed determining if bovine skeletal muscle growth is altered through the effects of flaxseed lignans on bovine ERβ.

Enterolignan Binding to Estrogenic Ligands

Flax enterolignans have estrogenic properties, allowing them to interfere with estrogenic ligands and circulating estradiol. How and with which compounds enterolignans interact to exert these effects has been studied using mouse and human models. Sex hormone binding globulin (SHBG) is the main plasma sex hormone transport protein and bind readily with endogenous estrogens (Rosner, 1990). The lignan EL has been shown to increase levels of SHBG (Aldecreutz et al., 1992). By increasing SHBG, more estrogen would be bound, thus decreasing the amount of free estrogen available to bind to other tissues. The lignan EL also prevents binding of estrogenically steroid binding protein (SBP), thus allowing estrogen to circulate (Benassayag et al., 1994). The SBP could also bind with EL and transport it into cells, where EL could

interfere with estrogen-dependent processes through competitive binding at receptor sites (Benassayag et al., 1994). Yet another ligan, α-fetoprotein (AFP), is inhibited by enterolignans. The protein AFP modulates activity of estrogens and regulates growth of estrogen-sensitive cells (Jacobson et al., 1990). Enterolignans have been shown to interfere with this binding (Garreau et al., 1991), affecting AFP-related tumor growth. These works demonstrate that enterolignans affect estrogenic ligands SHBG, SBP, and AFP, thereby influencing available estrogen and its ability to affect target tissues.

Antioxidant Effects

Enterolignans also may be classified as antioxidants due to their capacity for free radical scavenging. Vitamin E supplemented in diets is an antioxidant well known for increasing shelf life in meat products (Faustman et al., 1989 and Arnold et al., 1993), but the lignan of flaxseed actually has antioxidant power 5-fold greater than that of vitamin E (Prasad, 2000). The strong antioxidant property of flaxseed may increase shelf life similar to supplemented vitamin E. However, research has shown shelf life is improved in steaks from cattle fed flaxseed when fed vitamin E compared to steaks from cattle fed flaxseed without vitamin E (Good et al., 2004; LaBrune et al., 2008). This suggests the antioxidant properties of flaxseed do not improve shelf life as well as vitamin E.

α- linolenic Acid

The inside of flaxseed contains the oil fraction, of which over 50% may contain high levels of the omega-3 αLA. This essential long chain FA (LCFA) is a precursor for the production of eicosapentaenoic acid (EPA), a precursor to the production of docosahexaenoic acid (DPA). Health benefits are associated with diets containing αLA,

including proper eye and brain development (Greiner et al., 1997) and decreased inflammatory responses (Alexander, 1998). Another essential LCFA is linoleic acid (LA), a precursor for arachidonic acid (AA) known to produce pro-inflammatory agents (Blok et al., 1996).

n:6 to n:3 Ratio

Western diets often contain higher amounts of n-6 FA, creating a ratio of n-6:n-3 ranging from 10:1 to 25:1 (Simopoulos, 2000). This elevated ratio may be unhealthy, as the recommended n-6:n-3 intake is between 5:1 to 10:1 (WHO and FAO, 1995). Therefore it would benefit citizens to consume foods with lower n-6 FA content and/or greater n-3 FA content. In the United States, the average fish intake per capita is only 4.58 g/d (EPA, 2002). However, Americans eat more beef (81.6 g/d per capita; USDA). Consequently, n-3 enriched beef could be useful as an alternative source of desirable FA in Western diets. Tissue FA composition can be altered by the diet fed to the animal (Alexander, 1998). Therefore, it may be possible to feed diets with greater αLA than LA as a means of producing salable meat with more desirable fatty FA composition. Flaxseed has very low n-6:n-3 of 0.3:1 due to high αLA content (Morris, 2003). Thus, feeding flaxseed products may enrich beef products with αLA and help increase n-3 dietary intake in Western diets, restoring a more proper balanced n-6:n-3.

Biohydrogenation of α-linolenic Acid

Feeding αLA to cattle will not ensure its enrichment in beef products. The ruminant stomach often alters the composition of diet through biohydrogenation, a process whereby microbes saturate unsaturated fatty acids (USFA), yielding saturated

fatty acids (SFA). Biohydrogenation limits the amount of USFA leaving the rumen. The chemical structure of αLA, like that found in flaxseed, undergoes extensive biohydrogenation, with only 7.9% bypassing the rumen on average (Scollan et al., 2001). The hull of flaxseed offers little protection from biohydrogenation. Biohydrogenation proceeds by disrupting the structure of the FA. The FA enters the rumen as part of a triacylglyceride, a glycerol molecule connected with ester bonds to three FA. Lipolysis breaks the ester bonds, freeing FA from the glycerol. But when high concentrate diets are fed ruminal pH is lowered, affecting pH-dependent lypolysis and therefore allowing some polyunsaturated FA (PUFA) to bypass the rumen (Harfoot and Hazelwood, 1998). Nevertheless, biohydrogenation is a significant challenge to enriching beef, making it difficult for n-3 FA to pass through the rumen intact. Some form of protection from biohydrogenation is necessary to increase intact n-3 FA for tissue enrichment.

Tissue Deposition of α-linolenic Acid

Intact PUFA are deposited into tissue through several steps. First, PUFA is esterified into triacylglycerides (TAG) and phospholipids (PL). Then TAG and PL are assimilated into chylomicrons and very low density lipoproteins (VLDL) to be carried into the lymph and blood. Finally, lipoproteins transport FA to the adipose and muscle tissues for deposition into the membrane phospholipids (Demeyer and Doreau, 1999).

Diets have been shown to alter tissue FA composition across several species. Unlike ruminants, monogastric digestive systems have limited abilities to cause biohydrogenation, so it is easier to keep PUFA intact for tissue deposition. When fed 10 to 20% ground flaxseed, chickens were able to produce omega-3 enriched eggs without a discernable difference in taste (Scheideler et al., 1997). Feeding flaxseed has also

increased omega-3 content in pork products (Warnants et al., 2001 and Matthews et al., 2000). Though when fed at 3% of the diet for 65-d, concentrations of thiobarbituric acid reactive substrates were increased, indicating a greater propensity for oxidation, though levels were unlikely to be detectable by consumers (Riley et al., 2000).

Increasing omega-3 content of ruminant tissues is more challenging due to ruminal biohydrogenation of αLA. Despite the extensive biohydrogenation, changes in FA composition and increases in omega-3 content of adipose tissue (Casutt et al., 2000 and Aharoni et al., 2004) and lean tissue (La Brune et al., 2008 and Maddock et al., 2006) have been reported. LaBrune et al. (2008) supplemented ground flaxseed in finishing diets, resulting in increased ($P < 0.05$) plasma and muscle αLA in cattle fed flaxseed compared to control cattle. Maddock et al. (2006) supplemented flaxseed, whole, rolled and ground, and found all forms of flaxseed supplementation to increase ($P < 0.05$) muscle αLA content compared to control cattle. Kronberg et al. (2006) reported similar findings with ground flaxseed supplementation increasing muscle αLA in supplemented cattle compared to control cattle, regardless of breed. Extruded flaxseed supplementation also has been shown to increase muscle αLA in supplemented cattle in several studies (Dawson et al., 2010; Barton et al., 2007; and Raes et al., 2004). An increase in the n-3 FA αLA is often followed by an increase in total omega-3 content. When supplemented with ground flaxseed (Maddock et al., 2006; Kronberg et al., 2006) or extruded flaxseed (Dawson et al., 2010; Barton et al., 2007; and Raes et al., 2004) supplemented cattle had increased muscle omega-3 compared to control animals.

A few studies have shown a decrease in muscle n-6 FA when cattle were supplemented with whole, rolled, ground or extruded flaxseed (Maddock et al., 2006;

Raes et al., 2004). Most studies show muscle omega-6 to not be affected by flaxseed supplementation (Dawson et al., 2010; Barton et al., 2007; Kronberg et al., 2006). The results of Brenner (1989) imply that metabolism of n-3 FA is preferred over n-6 FA. Therefore in the presence of n-3 FA, n-6 FA would not be used as a substrate and maintain levels of n-6 FA. Perhaps the results of Benner (1989) explain why some studies do or don't have decreased n-6 FA following flaxseed supplementation.

Overall, changes in n-6 FA and n-3 FA alter the n-6:n-3 ratio. Previous studies consistently demonstrate a decreased muscle n-6:n-3 in cattle supplemented with flaxseed (whole, rolled, ground, or extruded) compared to control cattle (Dawson et al., 2010; Barton et al., 2007; Maddock et al., 2006 and Kronberg et al., 2006). The ratio changes are due to the consistent increase in omega-3, followed by the occasional decrease in omega-6.

The amount of saturated FA can be an indicator to the degree of ruminal biohydrogenation. Previous research with whole, rolled, and ground flaxseed (Maddock et al., 2006; Kronberg et al., 2006) or extruded flaxseed (Dawson et al., 2010; Barton et al., 2007; and Raes et al., 2004) has not affected the amount of monounsaturated FA (MUFA) or saturated FA (SFA) deposited in muscle tissues compared to control cattle. Thus, ruminal biohydrogenation does not appear to be affected by flaxseed supplementation.

Generally previous research (Barton et al., 2007; Maddock et al., 2006; Raes et al., 2004; Scollan et al., 2001) has shown extruded flaxseed supplementation to not affect polyunsaturated FA (PUFA) deposition in muscle. In contrast, Kronberg et al. (2006) showed extruded flaxseed supplementation to increase PUFA muscle compared to control

animals. Yet a diet effect on muscle PUFA is generally not expected since concentrations of muscle PUFA are primarily impacted by genetics and are only minorly influenced by nutrition (De Smet et al., 2003).

Flaxseed supplementation, whether fed whole, rolled, ground, or extruded, shifts FA deposition in muscle to create a more desirable beef product with elevated levels of n-3 FA and a more properly balanced n-6:n-3. The seed coat or heat from extrusion may provide partial protection against biohydrogenation, allowing for some FA deposition into the muscle tissues. Even greater protection from biohydrogenation would be warranted so muscle could be further enriched with n-3 FA from flaxseed supplementation.

Effects of Flaxseed on Feedlot Performance

Generally feeding flaxseed has not affected finishing performance in previous research. Maddock et al. (2006) and LaBrune et al. (2008) recorded no effect of ground flaxseed fed at 8% and 10%, respectively, on DMI. In contrast, Drouillard et al. (2004) reported ground flaxseed supplementation at 5% to increase ($P < 0.05$) DMI. So long as flaxseed doesn't influence the diet to exceed fat levels greater than 7%, ground flaxseed doesn't appear to impair DMI. Studies feeding ground flaxseed at 5% diet DM (Drouillard et al., 2004) and 10% diet DM (LaBrune et al., 2008) have also recorded no effect of flaxseed inclusion on ADG or G:F. However, flaxseed at an intermediate level of 8% diet DM has been shown to increase ADG and G:F (Maddock et al., 2006), but likely resulted from increased energy density in the flaxseed diet. The form of flaxseed doesn't appear to affect performance either. Dawson et al. (2010), Barton et al. (2007), and Raes et al. (2004) demonstrated extruded flaxseed does not affect performance. To

our knowledge LSM has not been evaluated for its affects on finishing performance. Flaxseed supplementation in many forms does not impair finishing performance.

Effect of Flaxseed on Carcass Characteristics

Previous studies have shown many carcass characteristics, such as yield grade, LM area, 12^{th} rib fat thickness, incidence of liver abcesses, and KPH, are not affected by feeding flaxseed products (Dawson et al., 2010; Barton et al., 2007; LaBrune et al., 2008; Maddock et al., 2006; Raes et al., 2004).

On the other hand, some studies have shown HCW) marbling scores, and quality grade to be affected by flaxseed supplementation. Most report flaxseed supplementation to have no affect on HCW (Dawson et al., 2010; Barton et al., 2007; LaBrune et al., 2008; Raes et al., 2004), yet Maddock et al. (2006) had steers fed flaxseed report heavier ($P < 0.05$) HCW. Supplemented steers may have had heavier HCW as a result of the elevated energy from their diets compared to control cattle.

Previous research shows no effect of extruded flaxseed supplementation on marbling scores (Dawson et al., 2010; Barton et al., 2007; Raes et al., 2004). In contrast, ground flaxseed has given mixed effects on marbling scores reported in previous research (Maddock et al., 2006 and La Brune et al., 2008). Maddock et al. (2006) fed whole, rolled and ground flaxseed and resulted in increased marbling scores compared to cattle not fed flaxseed. Again, the increased energy density of diets fed to cattle supplemented with flaxseed may have contributed to the increased marbling scores. LaBrune et al. (2008) fed ground flaxseed, tending to decrease marbling scores in supplemented cattle compared to control cattle.

Work with extruded flaxseed has shown no effect on quality grade (Dawson et al., 2010; Barton et al., 2007; Raes et al., 2004). LaBrune et al. (2008) fed ground flaxseed and also reported no effect of ground flaxseed supplementation on carcasses grading Choice or upper Choice compared to control cattle. In contrast, Maddock et al. (2006) reported increased carcasses grading upper Choice in cattle supplemented with flaxseed compared to control cattle. This increase in upper Choice carcasses followed an increase in marbling scores in cattle fed flaxseed. The increased energy density of the flaxseed diets may have contributed to the elevated marbling scores, subsequently increasing carcasses grading upper Choice. To our knowledge LSM has not been evaluated for its affects on carcass characteristics.

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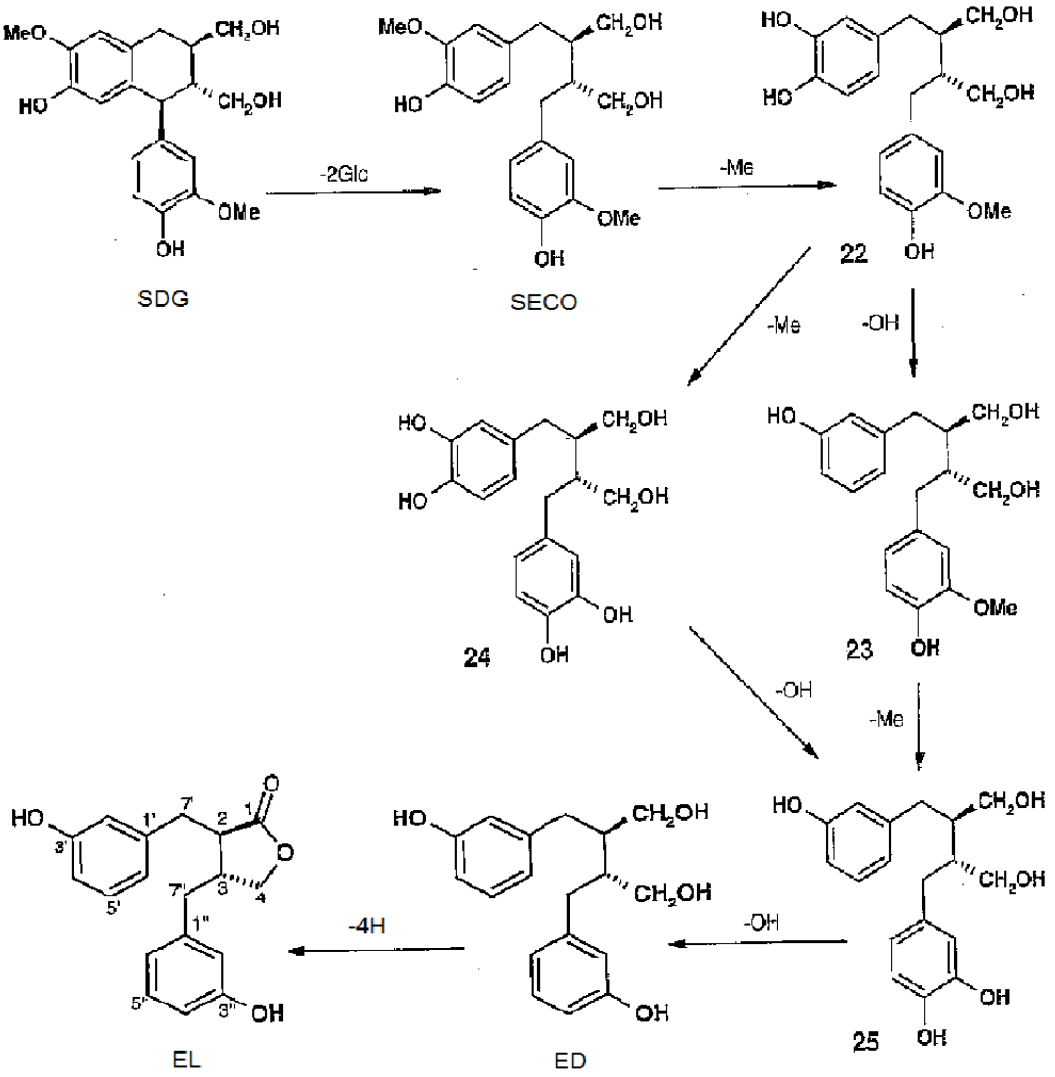
Figure 1. Metabolic pathway of secoisolariciresiol diglucoside.



SDG conversion pathway provided by Wang et al. (2000)

CHAPTER 2: Treatment of flaxseed with urea formaldehyde condensation polymer (UFCP): effects on fatty acid profiles of plasma and longissimus muscle in finishing Holstein steers.

ABSTRACT

Holstein steers (n = 30; initial BW = 499 ± 46 kg) were used in a finishing study to evaluate the effects of treating flaxseed with urea-formaldehyde condensation polymer (UFCP) on lipid profiles of plasma and LM. Experimental treatments included corn-based diets containing soy oil (SO), ground flaxseed (Flaxseed), or ground flaxseed treated with UFCP (UFCP Flaxseed). Steers were blocked by initial BW and these treatments were replicated across 5 individually-fed steers in each block. Residual feed, plasma, and BW were determined every 28-d period. Steers were harvested after 89 or 119 d on feed (heavy and light blocks, respectively). Body weights were recorded prior to transport to the abattoir. Following a 48-h chill, full rib primals were collected from one side of each carcass. Plasma and LM tissues were analyzed for fatty acid (FA) profiles. Finishing performance and carcass characteristics were not affected ($P > 0.16$) by diet. Cattle fed either flaxseed had greater LM α-linolenic acid and total omega-3 FA than LM from control cattle ($P < 0.05$). Application of UFCP to flaxseed did not affect ($P > 0.05$) degree of saturation in either tissue. Supplementing flaxseed, with or without UFCP treatment, increases omega-3 FA in beef products without compromising finishing performance or carcass quality.

Key words: beef, fatty acid, finishing, flaxseed, urea formaldehyde condensation polymer

Introduction

Many Americans do not consume the recommended amounts of n-3 FA (Kris-Etherton et al., 2000). In the U.S., the consumption of fish is relatively low (4.58 g/d per capita; EPA, 2002) compared to that of beef (81.6 g/d per capita; USDA ERS 2005). Consequently, n-3 enriched beef could be useful as an alternative source of n-3 FA in Western diets.

Previous studies have investigated the use of forage diets to increase n-3 FA in beef (Lorenz et al., 2002). Most cattle in the U.S. are finished on grain-based diets, yielding beef products with organoleptic properties deemed desirable by U.S. consumers (Medeiros et al., 1987), but with relatively low levels of n-3 FA. Previous research has investigated feeding strategies using fish oils (Scollan et al., 2001) and vegetable oils with high n-3 content (Clinquart et al., 1991) to alter beef FA composition. However, these fats often are altered by ruminal microbes through biohydrogenation (BH), limiting n-3 FA assimilation and deposition into tissues.

Flaxseed is rich in the n-3 FA α-linolenic acid (ALA). Feeding flaxseed can increase concentrations of n-3 FA in adipose (Casutt et al., 2000) and lean tissues (Maddock et al., 2006; La Brune et al., 2008), suggesting some FA escape biohydrogenation. Protection of these FA against ruminal BH could increase FA that are available for tissue deposition.

Urea-formaldehyde condensation polymer (UFCP) has been effective as a method to decrease susceptibility of plant protein meals to microbial degradation (Lebo and Winowiski, 2008). Briefly, UFCP is combined with plant proteins and water, and then heated to induce covalent bonding of UFCP to proteins. Conceivably, this same

technology could be applied to oilseeds, thus encapsulating lipids with ruminally undegraded protein to inhibit microbial BH. The objective of our study was to evaluate UFCP treatment of flaxseed as a method to decrease BH of lipids, thereby increasing the proportion of n-3 FA available for deposition into meat.

Materials and Methods

This study was conducted in accordance with procedures approved by the Kansas State University Institutional Animal Care and Use Committee.

Animals and Diets

Experimental treatments included corn-based diets containing soy oil (SO), ground flaxseed (Flaxseed), or UFCP Flaxseed. Composition of diets is summarized in Table 1. The fatty acid profile of diet ingredients is summarized in Table 2.

To minimize differences in gastrointestinal tract fill, steers were fed a common diet based on dry rolled corn for several days before initiation of the experiment. Cattle (n = 30; initial BW = 499 ± 46 kg) were blocked by individual BW into 2 groups (heavy and light) and assigned randomly within block to 1 of 3 dietary treatments. Steers were housed within barns consisting of 20 individual partially-covered concrete pens per barn with each pen measuring 1.5 m × 6 m. On d 1, steers were implanted (Revalor XS; 200 mg of trenbolone acetate, 40 mg of estradiol; Intervet Inc., Millsboro, DE), dewormed (Safeguard; Intervet, Inc., Millsboro, DE), individually weighed and identified with uniquely numbered ear tags. Baseline blood samples were collected in heparinized vacuum tubes (BD; Franklin Lakes, NJ) via jugular puncture. Tubes were immediately placed onto ice before centrifuging at 3200 × *g* for 10 min to recover plasma. Every 28 d

thereafter, cattle were individually weighed, unconsumed feed was weighed, and blood samples were collected.

Feed was offered *ad libitum* and delivered to individual fenceline feed bunks once daily at approximately 1300 h. Feed refusals were recorded every 28 d or as needed when excess residual feed accumulated in feed bunks. Water from a municipal supply was offered *ad libitum* using automatic water fountains shared between adjacent pens.

Harvest Data Collection and Sampling Procedures

The heavy weight block was harvested on d 89, and the light weight block was harvested on d 119. Final BW (gross BW × 0.96) was determined before cattle were transported to a commercial abattoir in Holcomb, KS. Incidence and severity of liver abscesses and HCW were recorded the d of harvest. Incidence and severity of liver abscesses were scored according to the scoring system (Brink et al., 1990): 0 = no abscesses; A^- = 1 or 2 small abscesses or abscess scars; A^0 = 2 to 4 small, well-organized abscesses; and A^+ = 1 or more large or active abscesses with or without adhesions. Carcass data were collected after a 24-h chill for the heavy block and after a 72-h chill for the light block. Boneless ribs from the 6^{th} through the 12^{th} rib section were collected from the left side of each carcass. Marbling scores, KPH, 12^{th} rib fat thickness, LM area, USDA yield grades, and USDA quality grades were determined. Actual BW × 0.96 was used to determine dressing percent.

Dry matter intakes were calculated from the as-fed deliveries using actual feedstuff DM values, less the amount of unconsumed DM. Daily gain was calculated as kg of gain on a shrunk basis. Feed efficiency was calculated as kg of gain per kg of dry matter consumed.

Analyses of Plasma Fatty Acids

Blood was collected from steers using heparinized tubes (BD, Franklin Lakes, NJ) 18-h postfeeding on d 1 and at 28-d intervals thereafter, and immediately placed onto ice before centrifuging at 3200 × g for 10 min to recover plasma. Plasma was freeze dried (500 *u*l) and combined with 1 mL benzene containing internal standard (1000 *u*g/mL methyl-C:13) and 4 mL BF_3-Methanol reagent (Supelco B1252). Tubes were incubated at 60°C for 60 minutes and then cooled to room temperature. Hexane (1 mL) and H_20 (4 mL) were added and tubes were vortexed. Tubes were then centrifuged at 1000 × g for 5 min. The organic solvent layer (1 to 2 mL) was then analyzed via gas chromatography (Schimadzu model 17A**,** Palo Alto, CA) equipped with a Supelco SP-2560 capillary column (100 m × 0.25 mm × 0.20*u* film) using He as the carrier gas at a flow rate of 1.1 mL/min. Initial temperature was 140 C for 4 min, followed by an increase of 4 C/min to a final temperature of 240 C.

Muscle Sample Analyses

At collection, boneless ribs were placed into multipurpose plastic bags, transported to the Kansas State University Meats Laboratory, and refrigerated overnight at 0 ± 2°C. Rib sections were weighed and vacuum packaged in Nylon/PE Multivac bags **(**Ultravac Solutions, Kansas City, MO) using a Multivac vacuum packager (Multivac C500, Sepp Hagenmüller GnbH & Co, Germany). Vacuum was checked using a Kennedy Gauge (Kennedy Gauge, Kennedy Enterprises, Lincoln, NE) with an average vacuum reading of 0.7774 Bar. Vacuum packaged ribs were stored for an additional 16 d at 0 ± 2 C. Starting at the cranial end of the ribs, a steak (2.54 cm thick) was removed for analysis of long-chain fatty acid (LCFA) composition. Longissimus muscle LCFA profiles were analyzed following the procedures of Sukhija and Palmquist (1988).

Briefly, 50 to 500 mg of samples were mixed with 2 mL internal standard in benzene and 3 mL methanolic-HCl before being flushed with nitrogen. Tubes were then capped and vortexed, heated for 2.25 h at 70 C, and vortexed every 45 min during heating. Tubes were cooled to room temperature and mixed with 5 mL 6% K_2CO_3 and 2 mL benzene while being vortexed. Tubes were centrifuged at 500 × g for 5 min. The organic solvent layer was then analyzed using a Schimadzu gas chromatography (model 17A**;** Schimadzu Corp., Palo Alto, CA) equipped with a Supelco SP-2560 capillary column (100 m × 0.25 mm × 0.20 *u*m; Supelco Inc., Bellefonte, PA) using He as the carrier gas at a flow rate of 1.1 mL/min. Initial temperature was 140 C for 4 min, and was increased by 4 C/min to a final temperature of 240 C.

Statistical Analyses

Plasma LCFA, muscle LCFA, growth performance, and carcass characteristics were analyzed using the MIXED procedure of SAS (version 9.0, SAS Inst. Inc., Cary, NC). Animal was the experimental unit, diet was the fixed effect, and animal was the random effect for analysis of plasma LCFA, muscle LCFA, growth performance, and carcass characteristics. The statistical analysis of plasma LCFA also included diet × day and day as fixed effects. USDA quality grade and liver abscesses were calculated and analyzed using Monte Carlo's Chi Square analysis (Higgins, 2004) with animal as the experimental unit and block as the random effect. Plasma served as the experimental unit for plasma LCFA analyses. Ribs served as the experimental unit for muscle LCFA analyses. Mean comparisons were determined following an *F*-test with $P \leq 0.05$. Means and differences were considered different at *P*-value ≤ 0.05, with a *P*-value of ≤ 0.10 considered as a tendency.

Results and Discussion

Performance

Performance results are reported in Table 3. In the present study inclusion of 10% flaxseed or 10% UFCP flaxseed did not affect ($P > 0.58$) performance, consistent with results from studies feeding 5% flaxseed (Drouillard et al., 2004) and 10% flaxseed (LaBrune et al., 2008).

Several studies have reported that feeding flaxseed has no effect on DMI, (Drouillard et al., 2004; Maddock et al., 2006; LaBrune et al., 2008), yet Good (2004) reported a linear effect of 5, 10, and 15% flaxseed inclusion on DMI. Studies feeding flaxseed at 5% (Drouillard et al., 2004), 10% (LaBrune et al., 2008), and 5, 10 and 15% diet DM (Good, 2004) reported no effect of flaxseed level on ADG or G:F. In contrast, Maddock et al. (2006) observed that feeding flaxseed at 8% of diet DM increased ADG and G:F.

Carcass Characteristics

Results for carcass characteristics are reported in Table 3. The effects of feeding flaxseed on carcass quality are inconsistent between previous studies. Good (2004) observed a quadratic effect of various levels of flaxseed (0, 5, 10, and 15%) on 12th rib fat thickness. Feeding flaxseed also increased KPH and USDA yield grade, agreeing with the observations of Maddock et al. (2006). Contrasting Good (2004), Maddock et al. (2006) reported increases in HCW, marbling scores, and percentage carcasses grading premium Choice when cattle were fed 8% flaxseed.

Fatty Acid Profiles

Plasma and muscle LCFA composition results are reported as absolute values (Tables 4 and 6) and percent of total FA (Tables 5 and 7).

Previous research which have reported that flaxseed supplementation does not affect muscle content of MUFA (Maddock et al., 2006; Kronberg et al., 2006), PUFA (Scollan et al., 2001; Raes et al., 2003; Maddock et al., 2006), or SFA (Kronberg et al., 2006). It was hypothesized UFCP would covalently-bond with lipids, protecting FA against ruminal BH. If BH were decreased, unsaturated FA would increase in cattle fed UFCP Flaxseed compared to cattle fed Flaxseed. Unsaturated FA were measured across several categories including monounsaturated FA (MUFA), polyunsaturated FA (PUFA), and the ratio of PUFA relative to saturated FA (PUFA:SFA). Excluding LM percent PUFA, plasma and LM unsaturated FA categories were not affected ($P > 0.05$) in cattle fed UFCP Flaxseed compared to cattle fed Flaxseed, suggesting UFCP was not effective in protecting flaxseed FA from ruminal BH.

The high levels of omega-3 in flaxseed have been observed to alter n-3 FA composition in plasma and muscle from cattle fed flaxseed (Maddock et al., 2006; Kronberg et al., 2006; LaBrune et al., 2008). Flaxseed diets of the current study affected n-3 FA composition so that steers fed Flaxseed and UFCP Flaxseed had increased absolute and percent values of ALA ($P < 0.01$), omega-3 ($P < 0.01$), and decreased n-6:n-3 ($P < 0.01$) in plasma and muscle tissues compared to control cattle.

The changes in plasma n-3 FA occurred quickly. On d 28, cattle fed flaxseed products had greater ($P > 0.05$) plasma omega-3 FA concentration than control cattle, maintaining elevated plasma omega-3 throughout the trial. There were no differences ($P > 0.05$) in plasma omega-3 between cattle fed Flaxseed and UFCP Flaxseed. Increases in plasma concentrations of omega-3 FA subsequently altered the ratio of n-6 and n-3 FA, so that plasma n-6:n-3 generally followed changes in plasma omega-3. Although cattle

fed UFCP Flaxseed had different ($P < 0.05$) d 1 plasma n-6:n-3 than other treatments, cattle fed flaxseed products had lower ($P < 0.05$) n-6:n-3 than control cattle by d 28 and maintained these reduced levels throughout the trial. Similarly, Good (2004) reported that feeding flaxseed increased the omega-3 ALA in plasma and decreased plasma n-6:n-3 by d 30 when compared to control cattle, and maintained these altered levels throughout the remainder of the trial.

Duration of feeding affected plasma LCFA composition (Figure 1 through 6). The interaction between diet and day affected ($P < 0.01$) plasma omega-3. In cattle fed UFCP flaxseed, levels of plasma omega-3 were not different on d 28, 56, 84, and 112. In contrast, cattle fed Flaxseed had greater ($P < 0.01$) plasma omega-3 on d 56 and 112 than d 28 and 84. Similarly, Drouillard et al. (2004) reported that duration of feeding ground flaxseed affected n-3 FA concentrations in edible tissues. There was also an interaction between diet and day for plasma n-6:n-3 ($P < 0.01$); however, for the flaxseed diets, levels of plasma n-6:n-3 were not affected ($P > 0.05$) by days on feed.

In the current study, plasma collected 5 d prior to harvest had similar LCFA composition compared to muscle LCFA composition. Similarly, LaBrune et al. (2008) reported plasma collected 14 d prior to harvest to have comparable LCFA composition to muscle LCFA composition. Results from flaxseed studies of Good (2004) also suggest increases in plasma n-3 FA generally reflect increases in muscle n-3 FA composition. Therefore, changes in plasma LCFA over time may indicate the necessary duration of feeding required to effectively alter muscle LCFA composition.

Results from this study suggest application of UFCP does not affect ruminal BH or FA deposition. Feeding flaxseed products, with or without UFCP, can increase omega-3 FA in beef products without compromising finishing performance or carcass quality.

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Table 1. Composition of treatment diets reported on a DM basis.[§]

	Treatments		
Item, % DM	**Soy Oil**	**Flax**	**UFCP**
Ingredients			
Dry rolled corn	70.87	68.81	68.69
Corn silage	9.99	10.00	10.00
Ground flaxseed	-	10.00	-
UFCP flaxseed	-	-	10.12
Corn steep liquor	3.00	3.00	3.00
Molasses	3.00	3.00	3.00
Soybean meal	4.05	-	
Feed additive[b]	1.96	1.96	1.96
Limestone	1.67	1.64	1.64
Urea, 46% N	1.01	1.01	1.01
Salt	0.30	0.30	0.30
Vitamin premix	0.22	0.22	0.22
Trace mineral premix	0.06	0.06	0.06
Nutrients, %			
DM	73.19	74.60	74.65
CP	12.67	12.91	13.41
P	0.46	0.48	0.48
Ca	0.72	0.74	0.74
Ether extract	7.61	6.35	7.04
NDF	10.43	11.23	11.22

[§]SO = Soy Oil, Flax = Flaxseed, UFCP = Flaxseed treated with urea formaldehyde condensation polymer.

[a]Formulated to provide 0.3% salt, 2650 IU vitamin A, 22 IU vitamin E, 0.10 mg Co, 10 mg Cu, 0.5 mg I, 0.25 mg Se, 50 mg Mn, and 50 mg Zn per kg diet DM.

[b]Provided 300 mg monensin and 90 mg tylosin (Elanco Animal Health; Greenfield, IN) in a ground corn carrier.

Table 2. Ingredient fatty acid profiles reported on an as-is basis.[§]

Fatty acid, % of sample[¥]	Ingredient		
	Soy Oil	Flax	UFCP
No. of sample	3	3	3
C10:0	0.007	0	0
C11:0	0.002	0.003	0
C12:0	0.001	0.006	0.002
C14:0	0.018	0.005	0.002
C14:1	0.006	0.005	0
C15:0	0.009	0.006	0
C15:1	0.088	0.002	0
C16:0	4.309	2.006	2.170
C16:1	0.003	0.005	0.013
C17:0	0.003	0.001	0.033
C17:1	0.028	0.004	0.023
C18:0	1.706	1.462	1.747
C18:1nt9	0.207	0.012	0.011
C18:1nc9	7.272	6.782	8.630
C18:1n7	0.648	0.303	0.333
CLA c9, t11	0.005	0.003	0.001
CLA t10, c12	0.004	0	0.001
C18:2nt6	0.004	0.003	0.001
C18:2nc6	20.690	5.670	7.379
C18:3n3	3.059	19.665	20.450
C18:3n6	0.010	0.093	0.099
C20:0	0.138	0.069	0.082
C20:1	0.083	0.039	0.048
C20:2	0.042	0.001	0.018
C20:3n6	0.001	0.003	0.008
C20:4n6	0.033	0.008	0.012
C20:5n3	0.002	0.009	0.002
C21:0	0.030	0	0
C22:0	0.19	0.056	0.055
C22:5n3	0.006	0	0.004
C22:6n3	0	0	0.001
C24:0	0.090	0.040	0.048
C24:1	0.032	0.012	0.009
omega-3[a]	3.067	19.674	20.457
omega-6[b]	20.792	5.780	7.502
SFA[c]	6.502	3.653	4.139
MUFA[d]	0.241	0.066	0.094
PUFA[e]	23.901	25.456	27.977

[§]SO = Soy Oil, Flax = Flaxseed, UFCP = Flaxseed treated with urea formaldehyde condensation polymer.

[¥]Number after C denotes number of Carbons; n denotes the double bond location from the omega position; c and t denote the *cis* or *trans* configuration.

[a]omega-3= C18:3n3 + C20:5n3 + C22:5n3 + C22:6n3
[b]omega-6 = C18:2nt6 + C18:2nc6 + CLAc9t11 + CLAt10c12 + C18:3n6 + C20:3n6 + C20:4n6
[c]SFA = C14:0 + C16:0 + C17:0 + C18:0 + C20:0 + C21:0 + C22:0 + C24:0
[d]MUFA = C14:1 + C16:1 + C17:1 + C18:1nt9 + C18:1n11 + C18:1nc9 + C18:1n7 + C20:1 + C24:1
[e]PUFA = C18:2n6t + C18:2n6c + CLAc9t11 + CLAt10c12 + C18:3n6 + C18:3n3 + C20:2 + C20:3n6 + C20:4n6 + C20:5n3 + C22:5n3 + C22:6n3

Table 3. Effect of diet on performance and carcass characteristics.[§]

	Treatments				
Item	**Soy Oil**	**Flax**	**UFCP**	**SEM**	***P*-value[£]**
Initial BW, kg	497.7	497.2	498.8	16.2	0.89
Final BW[†], kg	621.2	615.5	612.7	13.7	0.88
DMI, kg/d	12.58	12.60	12.03	0.56	0.58
ADG, kg/d	1.16	1.11	1.07	0.11	0.82
G:F	0.091	0.088	0.088	0.007	0.89
HCW, kg	391.8	381.9	378.8	8.04	0.48
LM area, cm^2	79.6	72.0	79.8	3.22	0.16
Fat thickness, cm	0.54	0.45	0.50	0.08	0.76
KPH, %	2.77	2.68	2.73	0.14	0.91
Liver abcesses, %	0.0	0.0	0.0	-	1.00
Dressing percent	63.0	62.0	61.9	0.005	0.27
Average YG	2.63	3.00	2.50	0.16	0.06
Marbling score[‡]	560	573	498	30	0.15
Select, %	0.0	0.0	10.0	-	1.00
Choice, %	75.0	88.9	90.0	-	0.64
Prime, %	25.0	11.1	0.0	-	0.17

[§]SO = Soy Oil, Flax = Flaxseed, UFCP = Flaxseed treated with urea formaldehyde condensation polymer.

[†]Calculated as full BW × 0.96

[‡]300 to 399 = Slight, 400 to 499 = Small, 500 to 599 = Modest, 600 to 699 = Moderate

[¥]Monte Carlo Exact Fit Chi Square analysis (Higgins, 2004)

[a,b,c]Means within a row without common superscripts are different ($P \leq 0.05$)

[£]Effect of diet *P*-value protected by an overall *F*-test ≤ 0.05

Table 4. Effect of diet on plasma concentrations of fatty acid profiles reported on an as-is basis.[§]

Fatty acid, mg/L[‡]	d 0			d 28			d 56			d 84				P-value[£]		
	SO	Flax	UFCP	SO	Flax	UFCP	SO	Flax	UFCP	SO	Flax	UFCP	SEM	Diet	DOF[†]	D×DOF[¥]
C12:0	1.73	3.21	2.05	1.31	2.13	1.01	1.80	1.43	1.24	1.22	1.67	1.79	0.62	0.29	0.25	0.74
C14:0	2.39	1.30	1.40	2.27	1.89	3.42	1.92	3.85	5.03	1.40	2.52	2.10	1.11	0.54	0.12	0.52
C14:1	2.36	2.54	3.31	2.28	1.63	3.44	2.74	3.34	1.72	1.80	2.36	4.34	0.87	0.32	0.95	0.27
C16:0	153.6	134.4	130.0	162.7	131.3	136.1	139.4	132.5	116.8	140.0	117.5	118.7	9.4	0.02	0.02	0.80
C16:1	21.08	20.48	19.55	12.64	19.32	16.43	4.16	13.70	10.20	3.43	2.53	2.53	2.41	0.11	<0.01	0.28
C17:0	10.03	10.56	8.08	4.71	5.54	4.08	1.98	1.24	5.60	3.07	9.16	6.25	1.89	0.45	<0.01	0.24
C17:1	5.43	5.89	4.90	3.64	2.68	3.21	3.29	2.45	2.30	3.50	3.64	1.73	0.78	0.21	<0.01	0.75
C18:0	235.6	218.5	222.2	295.6	242.2	255.3	241.6	240.2	222.7	248.0	195.6	224.2	20.0	0.30	<0.01	0.56
C18:n1t9	26.28	24.86	29.20	31.49	14.78	20.87	19.36	13.99	12.72	13.94	10.45	12.03	3.59	0.12	<0.01	0.19
C18:n1c9	96.98	85.01	76.01	86.83	88.89	85.04	84.71	96.35	80.36	68.90	83.59	68.66	6.81	0.22	0.01	0.25
C18:1n7	12.98	9.79	9.46	13.53	8.97	7.26	9.87	6.96	6.03	4.92	2.68	2.06	1.72	0.04	<0.01	0.94
CLA c9, t11	3.16^{a}	0.95^{d}	2.11^{abcd}	2.05^{abcd}	0.98^{d}	1.12^{cd}	0.89^{d}	2.58^{abc}	1.23^{cd}	2.25^{abcd}	1.86^{abcd}	2.73^{ab}	0.58	0.62	0.12	0.02
CLA t10, c12	3.14	1.73	0.78	0.90	1.84	0.55	0.89	0.93	0.46	1.38	0.64	1.54	0.67	0.27	0.21	0.29
C18:2nt6	4.37	3.58	3.27	4.88	4.93	6.94	1.25	1.46	1.80	1.83	1.78	2.44	1.23	0.70	<0.01	0.92
C18:2nc6	595.4^{abc}	579.9^{a}	585.0^{ab}	906.1^{l}	654.9^{abcdef}	717.1^{defghij}	745.3^{defghijk}	692.7^{bcdefgh}	661.0^{abcdefg}	703.2^{defghi}	604.8^{abcd}	624.5^{abcde}	57.6	0.27	<0.01	0.03
C18:3n3	24.00^{abcde}	13.77^{ab}	11.98^{a}	27.97^{abcdef}	135.12^{gh}	164.18^{ghi}	18.67^{abcd}	168.73^{i}	158.20^{ghi}	15.53^{abc}	129.54^{g}	159.21^{ghi}	13.03	<0.01	<0.01	<0.01
C18:3n6	19.77	21.62	17.20	13.57	11.32	9.92	20.45	10.63	8.45	11.52	8.83	5.79	2.56	0.02	<0.01	0.27
C20:0	2.76	1.91	1.40	2.54	1.42	2.06	1.73	2.47	3.11	2.56	1.96	1.22	0.70	0.64	0.76	0.34
C20:1	2.55	2.42	2.01	1.43	1.37	1.14	1.39	1.47	1.96	1.05	2.59	3.08	0.75	0.67	0.27	0.67
C20:2	7.47	5.47	5.57	2.33	2.41	3.65	2.91	4.17	3.42	3.36	4.32	5.11	0.97	0.84	<0.01	0.35
C20:3n6	42.94	38.59	33.73	31.29	18.72	14.75	33.87	15.02	14.52	26.88	18.25	11.93	3.40	<0.01	<0.01	0.26
C20:4n6	0.986	0.247	0.233	0.401	0.737	0.070	0.188	0.991	0.637	0.955	3.259	3.11	0.64	0.37	<0.01	0.22
C20:5n3	5.16	8.62	5.12	6.57	12.74	13.08	4.77	12.64	10.32	5.24	12.14	9.81	1.47	<0.01	<0.01	0.25
C21:0	3.37	1.63	0.99	1.88	0.55	0.20	1.57	1.78	1.14	1.22	1.45	0.71	0.62	0.04	0.12	0.52
C22:0	3.71	2.68	3.81	2.07	1.55	2.18	2.30	2.88	2.44	2.69	1.45	2.86	0.82	0.46	0.16	0.92
C22:5n3	2.95	2.12	3.11	3.66	1.23	3.05	1.22	2.27	1.35	3.33	4.22	3.81	0.80	0.77	0.01	0.32
C22:6n3	6.50	6.22	5.53	3.72	4.05	2.64	4.18	2.81	2.48	2.00	3.24	1.91	0.96	0.30	<0.01	0.90
C24:0	1.46	3.91	2.46	3.01	1.76	2.31	2.05	1.69	1.88	1.72	1.95	1.33	0.79	0.89	0.64	0.40
C24:1	2.49	1.63	1.10	1.72	1.46	1.72	1.35	1.03	1.58	2.03	2.38	2.31	0.58	0.78	0.28	0.76
Total	1307	1217	1202	1644	1381	1489	1359	1442	1345	1238	1238	1290	102	0.78	<0.01	0.25

[§]SO = Soy Oil, Flax = Flaxseed, UFCP = Flaxseed treated with urea formaldehyde condensation polymer.
[¥] Interaction between diet and days on feed.
[†]DOF = Days on feed
[£]Effect of diet, days on feed, and interaction between diet and days on feed *P*-values protected by an overall *F*-test ≤ 0.05
[‡]Number after C denotes number of Carbons; n denotes the double bond location from the omega position; c and t denote the *cis* or *trans* configuration.
[a,b,c]Means within a row without common superscripts are different ($P \leq 0.05$).

Table 5. Effect of diet on plasma concentrations of fatty acids expressed as a percent of total plasma fatty acids reported on an as-is basis.[§]

Fatty acid,	d 0			d 28			d 56			d 84				P-value[£]		
% of total[‡]	SO	Flax	UFCP	SO	Flax	UFCP	SO	Flax	UFCP	SO	Flax	UFCP	SEM	Diet	DOF[†]	D×DOF[¥]
C12:0	0.131	0.249	0.183	0.082	0.153	0.078	0.142	0.119	0.091	0.119	0.130	0.124	0.051	0.37	0.18	0.85
C14:0	0.173	0.110	0.139	0.142	0.140	0.202	0.137	0.258	0.389	0.107	0.228	0.197	0.081	0.33	0.25	0.60
C14:1	0.164	0.193	0.286	0.136	0.122	0.232	0.211	0.241	0.138	0.142	0.178	0.340	0.065	0.15	0.69	0.33
C16:0	11.86	11.24	11.14	9.91	9.59	9.16	10.28	9.30	8.82	11.04	9.75	9.64	0.58	0.12	<0.01	0.96
C16:1	1.596	1.661	1.77	0.764	1.396	1.16	0.311	0.929	0.765	0.242	0.202	0.217	0.181	0.04	<0.01	0.41
C17:0	0.738	0.836	0.650	0.228	0.426	0.270	0.145	0.086	0.433	0.231	0.737	0.452	0.138	0.21	<0.01	0.18
C17:1	0.430	0.493	0.454	0.229	0.184	0.228	0.245	0.179	0.179	0.293	0.341	0.151	0.074	0.58	<0.01	0.69
C18:0	18.09^{abc}	18.07^{abc}	18.99^{ab}	17.97^{abcd}	17.57^{abcdef}	17.00^{cdefg}	17.80^{abcde}	16.65^{cdefg}	16.67^{cdefg}	19.34^{a}	15.39^{g}	17.26^{abcdefg}	0.76	0.17	<0.01	0.04
C18:n1t9	2.01	1.96	2.29	1.88	1.08	1.40	1.43	0.97	0.95	1.10	0.84	0.92	0.22	0.11	<0.01	0.39
C18:n1c9	7.56	7.19	6.67	5.26	6.44	5.80	6.21	6.70	6.09	5.41	6.85	5.27	0.41	0.02	<0.01	0.27
C18:1n7	0.987	0.856	0.805	0.815	0.638	0.488	0.721	0.474	0.450	0.387	0.247	0.172	0.123	0.11	<0.01	0.98
CLA c9, t11	0.243^{a}	0.085^{cde}	0.165^{abcde}	0.127^{abcde}	0.071^{de}	0.078^{cde}	0.063^{e}	0.190^{abc}	0.095^{cde}	0.180^{abcd}	0.163^{abcde}	0.228^{ab}	0.044	0.78	0.02	0.04
CLA t10,	0.259	0.166	0.096	0.056	0.175	0.041	0.066	0.071	0.036	0.100	0.064	0.111	0.058	0.49	0.06	0.35
C18:2nt6	0.332	0.271	0.258	0.298	0.439	0.451	0.096	0.102	0.138	0.098	0.161	0.215	0.098	0.82	<0.01	0.94
C18:2nc6	45.30^{a}	47.28^{abcd}	47.24^{abc}	55.22^{j}	47.15^{ab}	48.15^{abcdef}	54.75^{j}	47.94^{abcde}	48.84^{abcdefghi}	54.80^{j}	48.63^{abcdefgh}	48.30^{abcdefg}	1.43	<0.01	<0.01	<0.01
C18:3n3	1.80^{a}	1.08^{a}	0.97^{a}	1.71^{a}	9.71^{abcd}	10.91^{def}	1.38^{a}	11.58^{fg}	11.50^{efg}	1.20^{a}	10.46^{cd}	12.05^{g}	0.36	<0.01	<0.01	<0.01
C18:3n6	1.478^{abcd}	1.759^{a}	1.524^{abc}	0.811^{e}	0.834^{e}	0.690^{e}	1.538^{ab}	0.737^{e}	0.661^{e}	0.910^{e}	0.715^{e}	0.449^{e}	0.184	0.10	<0.01	0.03
C20:0	0.204	0.193	0.115	0.154	0.123	0.128	0.133	0.183	0.236	0.203	0.186	0.101	0.055	0.81	0.62	0.34
C20:1	0.173	0.209	0.186	0.087	0.112	0.086	0.109	0.096	0.160	0.089	0.205	0.228	0.057	0.40	0.14	0.83
C20:2	0.598	0.435	0.477	0.146	0.168	0.242	0.223	0.300	0.250	0.275	0.375	0.378	0.077	0.90	<0.01	0.51
C20:3n6	3.33^{a}	3.14^{ab}	2.88^{abc}	1.90^{ef}	1.33^{fg}	0.98^{g}	2.48^{cd}	0.97^{g}	1.11^{g}	2.09^{de}	1.42^{fg}	0.98^{g}	0.22	<0.01	<0.01	0.04
C20:4n6	0.073	0.019	0.020	0.022	0.056	0.007	0.012	0.071	0.054	0.080	0.293	0.208	0.054	0.32	<0.01	0.29
C20:5n3	0.392	0.700	0.431	0.395	0.935	0.894	0.356	0.878	0.790	0.427	1.067	0.778	0.118	<0.01	0.04	0.45
C21:0	0.257	0.130	0.093	0.119	0.044	0.013	0.115	0.098	0.084	0.093	0.116	0.063	0.048	0.09	0.05	0.63
C22:0	0.288	0.222	0.320	0.127	0.128	0.148	0.174	0.223	0.207	0.216	0.108	0.225	0.065	0.48	0.06	0.90
C22:5n3	0.217	0.164	0.242	0.226	0.093	0.198	0.090	0.189	0.100	0.276	0.345	0.339	0.063	0.86	<0.01	0.51
C22:6n3	0.512	0.540	0.500	0.223	0.302	0.190	0.315	0.206	0.185	0.149	0.281	0.165	0.079	0.53	<0.01	0.79
C24:0	0.112	0.324	0.203	0.180	0.139	0.176	0.144	0.108	0.161	0.143	0.134	0.159	0.056	0.35	0.31	0.38
C24:1	0.182	0.120	0.091	0.102	0.106	0.116	0.096	0.069	0.123	0.146	0.213	0.173	0.044	0.98	0.11	0.63

[§]SO = Soy Oil, Flax = Flaxseed, UFCP = Flaxseed treated with urea formaldehyde condensation polymer.
[£]Effect of diet, days on feed, and interaction between diet and days on feed *P*-values protected by an overall *F*-test ≤ 0.05
[¥] Interaction between diet and day.
[†]DOF = Days on feed
[‡]Number after C denotes number of Carbons; n denotes the double bond location from the omega position; c and t denote the *cis* or *trans* configuration.
[a,b,c]Means within a row without common superscripts are different ($P \leq 0.05$).

Table 6. Effect of diet on longissimus muscle concentrations of fatty acids reported on an as-is basis.[§]

Fatty acid, mg/kg[‡]	Treatments			SEM	*P*-value[£]
	Soy Oil	Flax	UFCP		
C14:0	0.307	0.260	0.213	0.037	0.08
C14:1	0.046	0.040	0.170	0.010	0.10
C16:0	2.13	1.93	1.67	0.23	0.25
C16:1	0.346[a]	0.288[ab]	0.206[b]	0.040	0.02
C17:0	0.093	0.096	0.093	0.003	0.75
C17:1	0.068	0.064	0.049	0.009	0.24
C18:0	1.11	1.12	1.07	0.12	0.96
C18:n1t9	0.269	0.181	0.221	0.026	0.07
C18:n1c9	2.77	2.69	2.34	0.29	0.49
C18:1n7	0.136	0.132	0.119	0.016	0.72
CLA c9, t11	0.0034[a]	0.0003[b]	0.0019[a]	0.0007	0.01
CLA t10, c12	0.0006	0.0002	0.0011	0.0003	0.09
C18:2nt6	0.017[a]	0.029[b]	0.023[ab]	0.003	0.02
C18:2nc6	0.385	0.327	0.346	0.024	0.09
C18:3n3	0.024[a]	0.083[b]	0.085[b]	0.075	<0.01
C18:3n6	0.003	0.003	0.002	0.0008	0.44
C20:0	0.005	0.009	0.010	0.002	0.07
C20:1	0.018	0.016	0.015	0.002	0.63
C20:2	0.006	0.005	0.005	0.0007	0.22
C20:3n6	0.012	0.011	0.007	0.002	0.08
C20:4n6	0.002[a]	0.003[b]	0.004[b]	0.0005	<0.01
C20:5n3	0.002	0.003	0.002	0.0008	0.42
C21:0	0.021	0.014	0.018	0.003	0.36
C22:0	0.003	0.004	0.004	0.0007	0.86
C22:5n3	0.004	0.002	0.002	0.0008	0.07
C22:6n3	0.003	0.006	0.001	0.002	0.08
C24:0	0.003	0.003	0.002	0.0005	0.12
C24:1	0.0009	0.0011	0.0008	0.0003	0.72
Total	7.63	7.16	6.36	0.74	0.41
omega-3[d]	0.031[a]	0.092[b]	0.089[b]	0.008	<0.01
omega-6[e]	0.424	0.374	0.385	0.027	0.22
n-6:n-3[f]	17.69[a]	3.97[b]	4.18[b]	1.22	<0.01
SFA[g]	3.70	3.45	3.10	0.39	0.44
MUFA[h]	3.62	3.39	2.94	0.35	0.35
PUFA[i]	0.463	0.473	0.480	0.033	0.89
PUFA:SFA[j]	0.116	0.128	0.146	0.009	0.06

[§]SO = Soy Oil, Flax = Flaxseed, UFCP = Flaxseed treated with urea formaldehyde condensation polymer.
[£]Effect of diet *P*-value protected by an overall *F*-test ≤ 0.05.
[a,b,c] Means within a row without common superscripts are different ($P \leq 0.05$).
[‡]Number after C denotes number of Carbons; n denotes the double bond location from the omega position; c and t denote the *cis* or *trans* configuration.
[d]omega-3= C18:3n3 + C20:5n3 + C22:5n3 + C22:6n3

[e]omega-6 = C18:2nt6 + C18:2nc6 + CLAc9t11 + CLAt10c12 + C18:3n6 + C20:3n6 + C20:4n6
[f]n-6:n-3 = as omega-6 / omega-3
[g]SFA = C14:0 + C16:0 + C17:0 + C18:0 + C20:0 + C21:0 + C22:0 + C24:0
[h]MUFA = C14:1 + C16:1 + C17:1 + C18:1nt9 + C18:1n11 + C18:1nc9 + C18:1n7 + C20:1 + C24:1
[i]PUFA = C18:2n6t + C18:2n6c + CLAc9t11 + CLAt10c12 + C18:3n6 + C18:3n3 + C20:2 + C20:3n6 + C20:4n6 + C20:5n3 + C22:5n3 + C22:6n3
[j]PUFA:SFA = PUFA / SFA

Table 7. Effect of diet on longissimus muscle concentrations of fatty acids expressed as a percent of total fatty acids reported on an as-is basis.[§]

Fatty acid, % of total[‡]	Treatments			SEM	*P*-value[£]
	Soy Oil	Flax	UFCP		
C14:0	3.78[a]	3.47[ab]	3.11[b]	0.20	0.02
C14:1	0.558	0.561	0.250	0.126	0.11
C16:0	27.39[a]	26.48[ab]	25.50[b]	0.46	<0.01
C16:1	4.12[a]	3.83[ab]	3.03[b]	0.31	0.04
C17:0	1.22	1.29	1.46	0.16	0.39
C17:1	0.874	0.900	0.760	0.065	0.23
C18:0	14.82	15.49	17.05	0.67	0.06
C18:n1t9	3.60[a]	2.52[b]	3.62[a]	0.33	0.03
C18:n1c9	35.63	36.90	35.67	0.92	0.35
C18:1n7	1.51	1.55	1.56	0.20	0.97
CLA c9, t11	0.040[a]	0.016[b]	0.030[a]	0.008	<0.01
CLA t10, c12	0.009	0.003	0.020	0.006	0.10
C18:2nt6	0.195[a]	0.383[b]	0.342[b]	0.046	<0.01
C18:2nc6	4.63	4.11	4.94	0.31	0.09
C18:3n3	0.317[a]	1.164[b]	1.358[b]	0.102	<0.01
C18:3n6	0.043	0.037	0.032	0.013	0.81
C20:0	0.059[a]	0.124[b]	0.147[b]	0.022	0.02
C20:1	0.233	0.213	0.226	0.023	0.77
C20:2	0.083	0.069	0.078	0.011	0.68
C20:3n6	0.172	0.157	0.109	0.028	0.23
C20:4n6	0.024[a]	0.039[ab]	0.058[b]	0.007	<0.01
C20:5n3	0.023	0.037	0.034	0.013	0.70
C21:0	0.265	0.196	0.273	0.032	0.16
C22:0	0.049	0.058	0.059	0.013	0.83
C22:5n3	0.045	0.021	0.022	0.010	0.21
C22:6n3	0.051	0.095	0.024	0.026	0.14
C24:0	0.043	0.039	0.028	0.009	0.47
C24:1	0.010	0.014	0.009	0.005	0.76
omega-3[d]	0.400[a]	1.282[b]	1.40[b]	0.108	<0.01
omega-6[e]	5.11	4.72	5.52	0.344	0.15
SFA[f]	47.48	47.00	47.48	0.96	0.87
MUFA[g]	46.59	46.53	45.19	0.98	0.31
PUFA[h]	5.62[a]	6.10[a]	7.04[b]	0.44	0.03

[§]SO = Soy Oil, Flax = Flaxseed, UFCP = Flaxseed treated with urea formaldehyde condensation polymer.

[£]Effect of diet *P*-value protected by an overall *F*-test ≤ 0.05.

[a,b,c]Means within a row without common superscripts are different ($P \leq 0.05$).

[‡]Number after C denotes number of Carbons; n denotes the double bond location from the omega position; c and t denote the *cis* or *trans* configuration.

[d]omega-3= C18:3n3 + C20:5n3 + C22:5n3 + C22:6n3

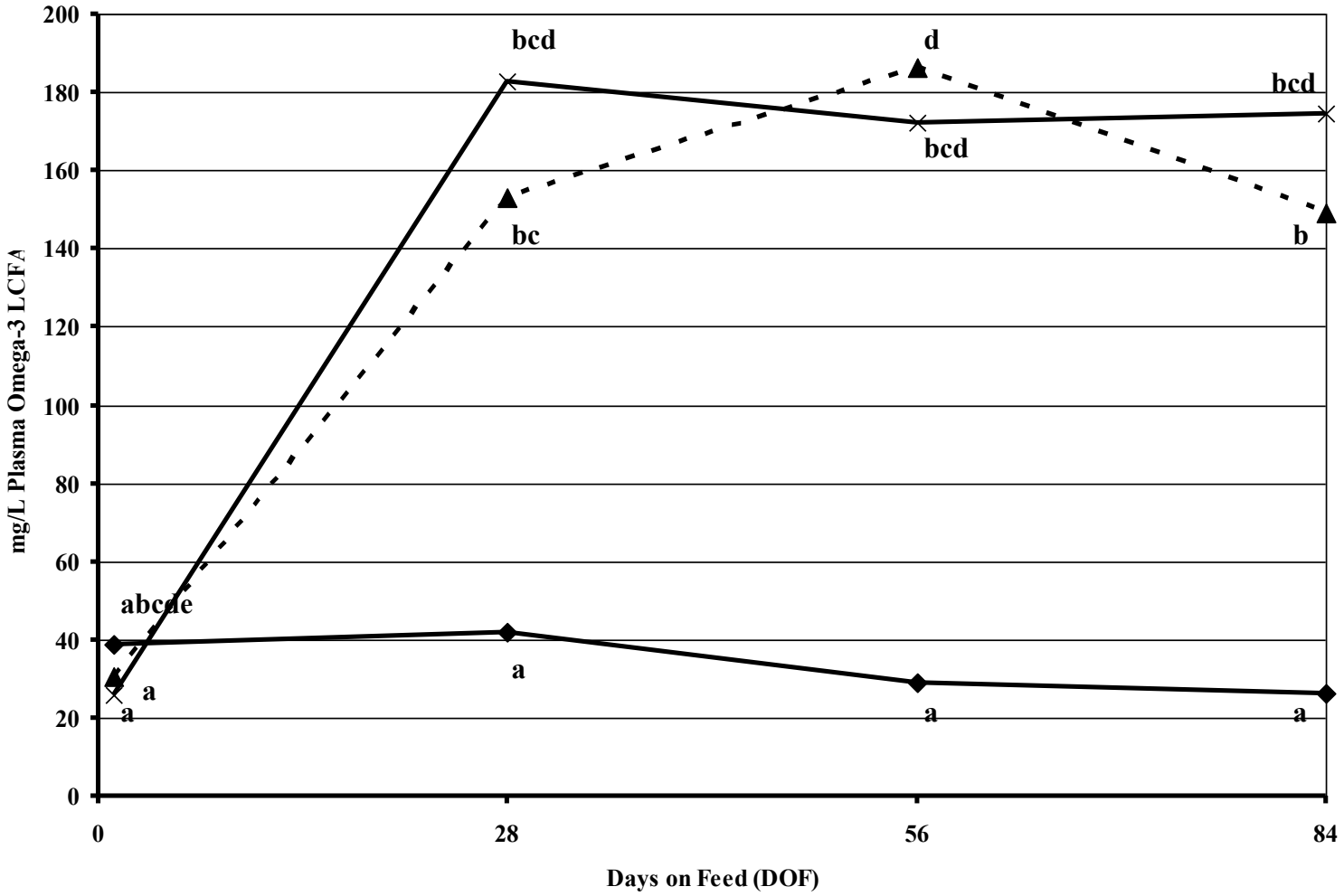
[e]omega-6 = C18:2nt6 + C18:2nc6 + CLAc9t11 + CLAt10c12 + C18:3n6 + C20:3n6 + C20:4n6

[f]SFA = C14:0 + C15:0 + C16:0 + C17:0 + C18:0 + C20:0 + C21:0 + C22:0 + C24:0

[g]MUFA = C14:1 + C16:1 + C17:1 + C18:1nt9 + C18:1n11 + C18:1nc9 + C18:1n7 + C20:1 + C24:1

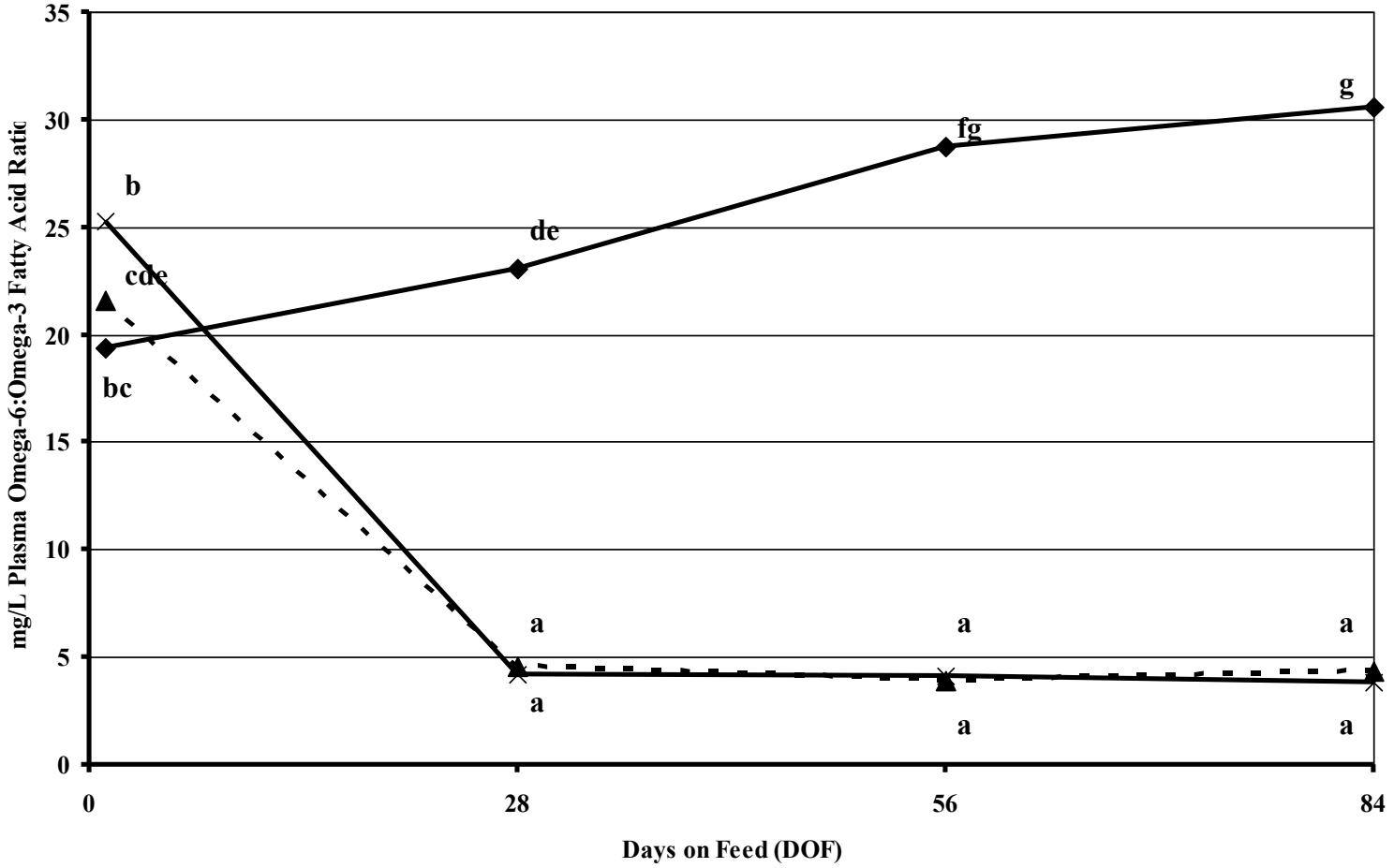
[h]PUFA = C18:2n6t + C18:2n6c + CLAc9t11 + CLAt10c12 + C18:3n6 + C18:3n3 + C20:2 + C20:3n6 + C20:4n6 + C20:5n3 + C22:5n3 + C22:6

A.



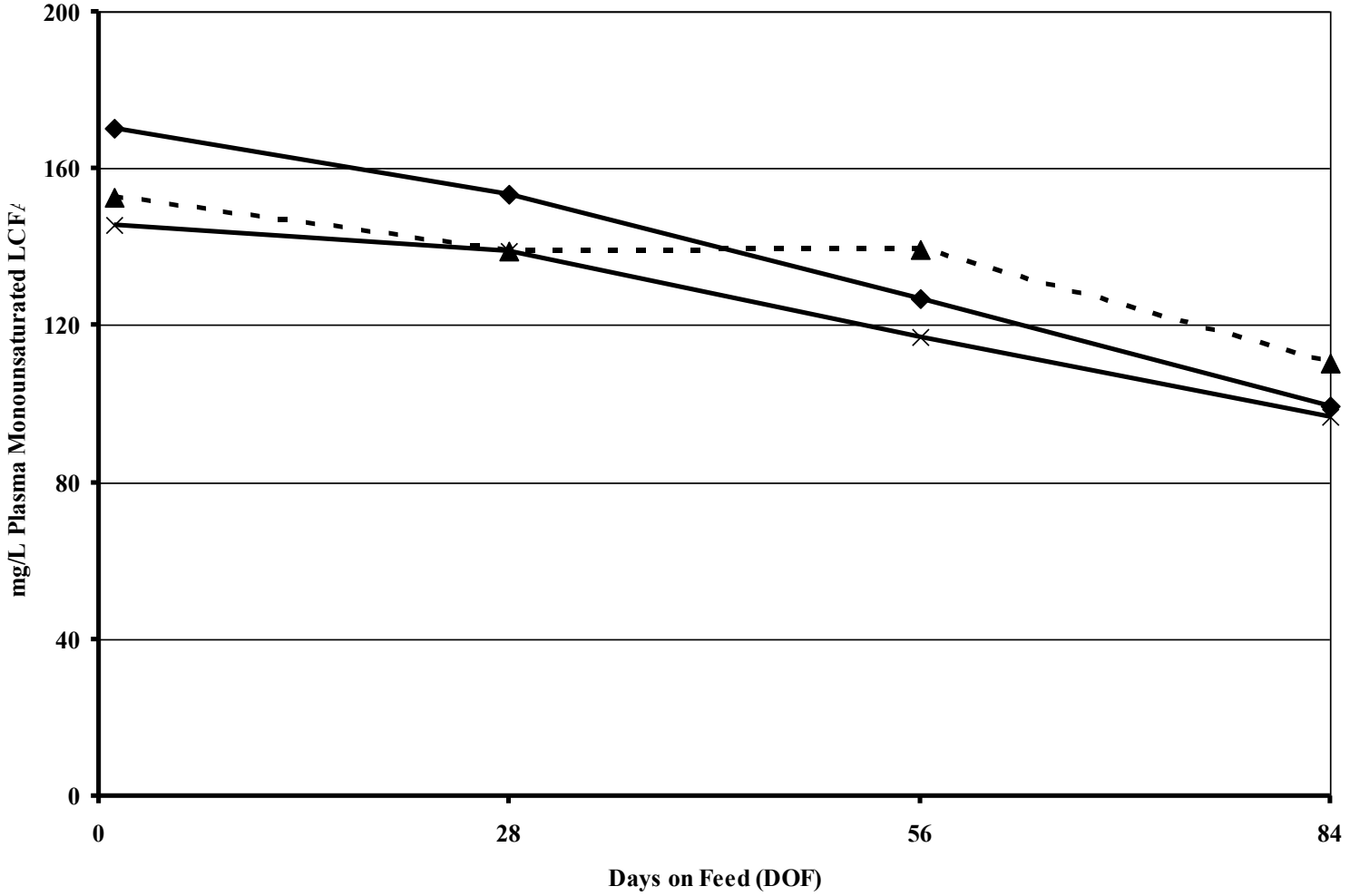
SEM = 13.50 Diet×DOF, $P < 0.01$ Diet, $P < 0.01$ DOF, $P < 0.01$

B.



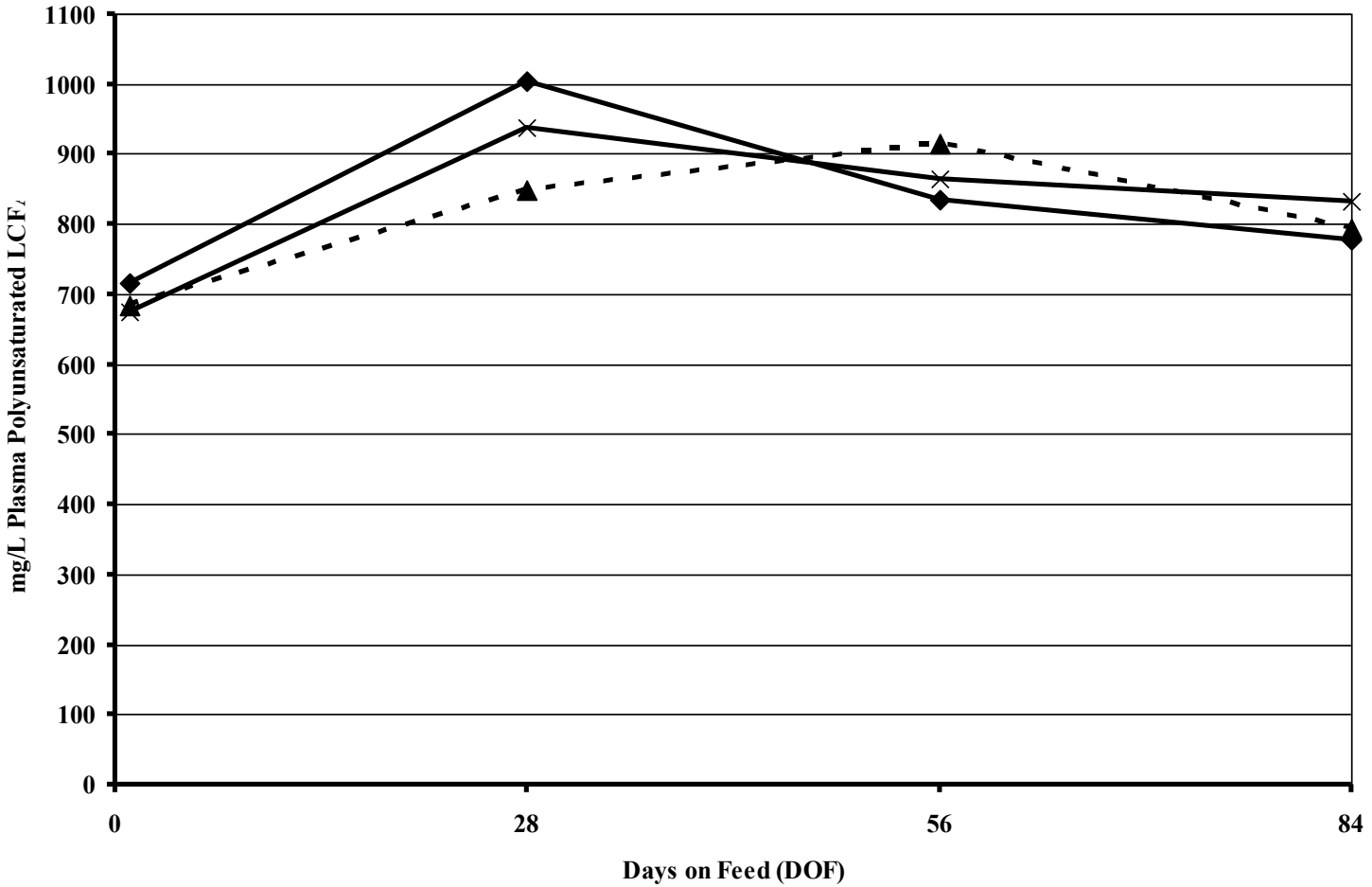
SEM = 1.39 Diet×DOF, P < 0.01 Diet, P < 0.01 DOF, P < 0.01

C.



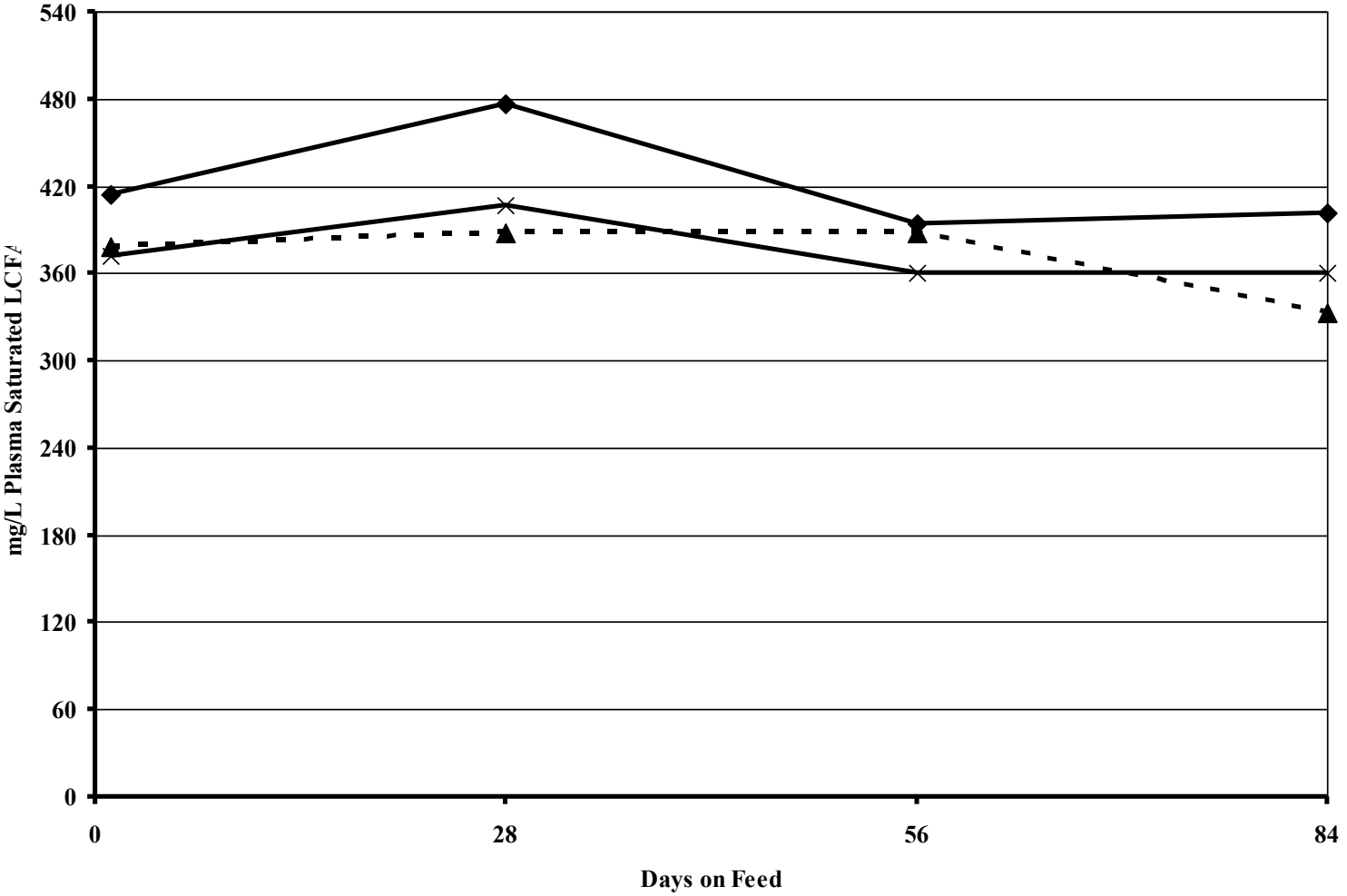
SEM = 10.73 Diet×DO, P = 0.52 Diet, P = 0.39 DOF, P < 0.01

D.



SEM = 70.04 Diet×DOF, P = 0.16 Diet, P = 0.96 DOF, P <0.01

E.



SEM = 28.55 Diet×DOF, P = 0.65 Diet, P = 0.17 DOF, P = 0.01

Figure 1. Effect of diet, day, and the interaction between diet and day on plasma concentrations of fatty acids reported on an as-is basis.

Soy Oil —♦—; Flaxseed - ▲ -; UFCP Flaxseed —×—

A. Effect of diet, day, and the interaction between diet and day on plasma concentrations of Omega-3 fatty acids.
B. Effect of diet, day, and the interaction between diet and day on plasma n-6:n-3 fatty acid ratio.
C. Effect of diet, day, and the interaction between diet and day on plasma concentrations of monounsaturated fatty acids.
D. Effect of diet, day, and the interaction between diet and day on plasma concentrations of polyunsaturated fatty acids.
E. Effect of diet, day, and the interaction between diet and day on plasma concentrations of saturated fatty acids.

CHAPTER 3: Novel extrusion of flaxseed (LinPro) increases omega-3 fatty acids in plasma and longissimus muscle tissues from Holstein steers fed feedlot diets

ABSTRACT

Holstein steers (n = 30; initial BW = 499 ± 46 kg) were used in a finishing study to evaluate the effects of feeding linseed oil and a new extrusion of flaxseed and field peas on plasma and LM fatty acid (FA) profiles. Experimental treatments included corn-based diets containing soy oil (SO), linseed oil (LO), or a blend of flaxseed and field peas that were mixed and extruded (LinPro). These treatments were replicated across 10 individually-fed steers per treatment. Residual feed, plasma FA profiles, and BW were determined every 28-d period. Steers were harvested after 89 and 119 d on feed (heavy and light blocks, respectively). Body weights were recorded prior to shipmen to the abattoir. Following a 48-h chill, full rib primals were collected and lean tissue was analyzed for FA profiles. Finishing performance and carcass quality were not affected by diet ($P > 0.07$). There was an interaction between diet and days on feed; plasma α-linolenic acid (ALA) and total omega-3 FA increased ($P < 0.01$), and n-6:n-3 decreased ($P < 0.01$) after 28 DOF in response to feeding products containing flaxseed and plateaued thereafter. Cattle fed flaxseed products had increased LM ALA and total omega-3 FA, and decreased n-6:n-3 than cattle fed SO ($P < 0.01$). Cattle fed LinPro had greater increases in LM ALA and total omega-3 than cattle fed LO ($P < 0.05$). Including flaxseed products in finishing diets increases desirable FA in both plasma and LM. Changes in plasma ALA, total omega-3 FA and n-6:n-3 transpire quickly, occurring by d 28 and throughout the trial. The novel feedstuff LinPro resulted in greatest levels of C18:3n3 and total omega-3 FA in LM.

Key words: beef, fatty acid, finishing, linseed meal, linseed oil

Introduction

Many Americans don't consume the recommended amounts of n-3 fatty acids (Kris-Etherton et al., 2000). Americans eat a great deal more beef (81.6 g/d per capita; USDA-ERS, 2005) than fish (4.58 g/d per capita; EPA, 2002). Consequently, n-3 enriched beef could be useful as an alternative source of omega-3 fatty acids (FA) in Western diets.

Previous studies have investigated the use of forage diets to increase n-3 FA in beef (Lorenz et al., 2002). Most cattle in the U.S. are finished on grain-based diets, yielding beef products with organoleptic properties deemed desirable by U.S. consumers (Medeiros et al. 1987). Beef produced with conventional high grain diets generally contains relatively low levels of n-3 FA. Previous research has investigated feeding strategies aimed at altering FA composition of beef using fats with high n-3 content (Clinquart et al., 1991; Scollan et al., 2001). However, these fats often are altered by rumen microbes through biohydrogenation (BH), limiting n-3 FA assimilation and tissue deposition.

Flaxseed is rich in the n-3 FA α-linolenic acid (ALA). Feeding flaxseed can increase concentrations of n-3 FA in lean tissues (Maddock et al., 2006; La Brune et al., 2008), suggesting some FA escape BH. Similarly, Montgomery et al. (2008) also reported 22% of flax oil FA escapes BH. Protection of these FA against ruminal BH could increase FA that are available for deposition into edible tissues.

Ruminal bypass of flaxseed FA has been achieved through extrusion (Raes et al., 2004; Barton et al., 2007; Dawson et al., 2010). LinPro is a feedstuff composed of an

extruded mixture of flaxseed and field peas. The effects of LinPro on beef cattle performance and carcass traits have not been determined. Therefore the objectives of this study were to evaluate LinPro as a dietary method of increasing ruminal bypass of flaxseed FA and the subsequent effects on plasma FA profiles and muscle deposition of omega-3 FA in salable red meat.

Materials and Methods

This study was conducted in accordance with procedures approved by the Kansas State University Institutional Animal Care and Use Committee.

Production of LinPro

LinPro is an extruded product produced using an approximated 50:50 combination of full-fat flaxseed and split field peas (Oleet Processing Ltd., Regina, SK). The flaxseed and peas were extruded using an Instapro extruder (Instapro Inc., Des Moines, IA) for 20 to 25 s at a temperature of 125 to 130ºC.

Animals and Diets

Experimental treatments included corn-based diets containing soy oil at 4% (SO), linseed oil at 4% (LO), or LinPro at 16% of the diet DM (Table 1). Compositions of diets are summarized in Table 1. Table 2 reports the ingredient FA profiles. The ingredients soy oil and linseed oil were first mixed into an emulsion with salt, molasses, and xanthum gum before being added to the total mixed ration.

To minimize differences in gastrointestinal tract fill, steers were fed a common diet based on dry-rolled corn for several d before initiation of the experiment. Cattle (n = 30; initial BW = 499 ± 46 kg) were stratified by BW and randomly allocated to 1 of 4 dietary treatments within each of the ten strata. The heaviest strata were allocated to the

first heaviest group. Steers were housed within barns consisting of 20 individual partially-covered concrete pens per barn; each pen measured 1.5 m × 6 m. On d 1, steers were implanted (Revalor XS, 200 mg of trenbolone acetate, 40 mg of estradiol; Intervet Inc., Millsboro, DE), dewormed (Safeguard; Intervet, Inc., Millsboro, DE), individually weighed and identified with uniquely numbered ear tags. Baseline blood samples were collected in heparinized vacuum tubes (BD; Franklin Lakes, NJ) via jugular puncture. Tubes were immediately placed onto ice before centrifuging at 3200 × *g* for 10 min to recover plasma. Every 28 d thereafter, cattle were weighed individually, unconsumed feed was weighed, and blood samples were collected for analyses of FA.

Feed was offered *ad libitum* and delivered to individual fenceline feed bunks once daily at approximately 1300 h. Feed refusals were recorded every 28 d or as needed when excess residual feed accumulated in feed bunks. Dry matter intakes were calculated from the as-fed deliveries using actual feedstuff DM values, less the amount of unconsumed DM. Daily gain was calculated as kg of gain on a shrunk basis. Feed efficiency was calculated as kg of gain on a shrunk basis per kg of DM consumed. Water was offered *ad libitum* via automatic water fountains shared between adjacent pens.

Harvest Data Collection and Sampling Procedures

Steers were harvested on 2 d. The heavier weight block was slaughtered on d 89, and the lighter initial weight block was slaughtered on d 119. Final BW (gross BW × 0.96) was determined before cattle were transported to a commercial abattoir (Holcomb, KS). Incidence and severity of liver abscesses and HCW were recorded the d of harvest. Incidence and severity of liver abscesses were scored according to the scoring system (Elanco, Greenfield, IN): 0 = no abscesses; A^- = 1 or 2 small abscesses or abscess scars;

A^0 = 2 to 4 small, well-organized abscesses; and A^+ = 1 or more large or active abscesses with or without adhesions. Carcass data were collected after a 24-h chill for the heavy block and after a 72-h chill for the light block. The boneless ribs from the 6th through the 12th rib section were collected from the left side of each carcass. KPH, 12th rib fat thickness, LM area, marbling scores, USDA yield grades, and USDA quality grades were determined.

Analyses of Plasma Fatty Acids

Blood was collected from steers using heparinized tubes (BD, Franklin Lakes, NJ) 18-h postfeeding on d 1 and at 28-d intervals thereafter. Tubes were immediately placed onto ice before centrifuging at 3,200 × *g* for 10 min to recover plasma. Plasma was freeze dried (500 *u*l) and combined with 1 mL benzene containing internal standard (1,000 *u*g/mL methyl-C:13) and 4 mL BF_3-Methanol reagent (Supelco B1252). Tubes were incubated at 60°C for 60 minutes and then cooled to room temperature. Hexane (1 mL) and ddH_2O (4 mL) were added and tubes were vortexed. Tubes were then centrifuged at 1000 × g for 5 min. The organic solvent layer (1 to 2 mL) was then analyzed via gas chromatography (Schimadzu model 17A**,** Palo Alto, CA) equipped with a Supelco SP-2560 capillary (100m × 0.25 mm × 0.20*u* film) using He as the carrier gas at a flow rate of 1.1 mL/min. Initial temperature was 140°C for 4 min, followed by an increase of 4°C/min to a final temperature of 240°C.

Muscle Sample Analyses

At collection, boneless ribs were placed into multipurpose plastic bags, transported to the Kansas State University Meats Laboratory, and refrigerated overnight at 0 ± 2°C. Rib sections were weighed and vacuum packaged in Nylon/PE Multivac bags

(Ultravac Solutions, Kansas City, MO) by using a Multivac vacuum packager (Multivac C500, Sepp Hagenmüller GnbH & Co, Germany). Vacuum was checked using a Kennedy Gauge (Kennedy Gauge, Kennedy Enterprises, Lincoln, NE) with an average vacuum reading of 0.7774 Bar. Vacuum packaged ribs were stored for an additional 16 d at 0 ± 2°C. Starting at the cranial end of the ribs, a steak (2.54 cm thick) was removed for analysis of long-chain fatty acid (LCFA) composition. Longissimus muscle LCFA profiles were analyzed following the procedures of Sukhija and Palmquist (1988). Briefly, 50 to 500 mg of samples were mixed with 2 mL internal standard in benzene and 3 mL methabolic-HCl before being dried with nitrogen. Tubes were then capped and vortexed, heated for 2.25 hours at 80ºC, and vortexed every 45 min during heating. Tubes were cooled to room temperature and mixed with 5 mL 6% K_2CO_3 and 2 mL benzene while being vortexed. Tubes were centrifuged at 500 × g for 5 min. The organic solvent layer was then analyzed using a Schimadzu gas chromatography (model 17A**;** Schimadzu Corp., Palo Alto, CA) equipped with a Supelco SP-2560 capillary (100 m × 0.25 mm × 0.20 *u*m; Supelco Inc., Bellefonte, PA) using He as the carrier gas at a flow rate of 1.1 mL/min. Initial temperature was 140°C for 4 min, and was increased by 4°C/min to a final temperature of 240°C.

Statistical Analyses

Plasma LCFA, LM LCFA, growth performance, and carcass characteristics were analyzed using the MIXED procedure of SAS (version 9.0, SAS Inst. Inc., Cary, NC). Animal was the experimental unit, diet was the fixed effect, and block was the random effect for analysis of plasma LCFA, LM LCFA, growth performance, and carcass characteristics. The statistical analysis of plasma LCFA also included diet × day and day

as fixed effects. USDA quality grade and liver abscesses were calculated and analyzed using Monte Carlo's Chi Square analysis (Higgins, 2004) with animal as the experimental unit and block as the random effect. Ribs (n = 30; 10 per treatment) served as the experimental unit for LM LCFA analyses. Mean comparisons were determined following an *F*-test with $P \leq 0.05$. Means were considered different at *P*-value ≤ 0.05, with a *P*-value of ≤ 0.10 considered as a tendency.

Results & Discussion

Performance and Carcass Characteristics

Figure 2 demonstrates BW was not affected by diet throughout the trial. Finishing performance and carcass characteristic results are reported in Table 3. Performance variables and carcass quality were not affected ($P \geq 0.07$) by treatment in the present study. This was expected as the diets were formulated with similar energy density and fat content. This agrees with several studies where extruded flaxseed (Raes et al., 2004; Barton et al., 2007; Dawson et al., 2010) did not affect performance or carcass traits.

Fatty Acid Profiles

Plasma and LM FA compositions are reported as absolute values (Tables 3 and 5) and percent of the total FA (Tables 4 and 6).

There was an interaction between the effects of diet and day for plasma ALA and total omega-3 ($P < 0.01$). By d 28, flaxseed products induced greater ($P < 0.05$) plasma ALA and maintained the elevated levels throughout the study when compared to control cattle. When expressed as a percent of total FA, plasma ALA was further increased ($P < 0.05$) in cattle fed LinPro compared to cattle fed LO or control diets on d 28 and throughout the trial. Feeding flaxseed products also increased plasma total omega-3 at d

28 and throughout the trial; however of the flaxseed products LinPro produced higher ($P < 0.05$) absolute levels of plasma total omega-3 on d 28, 56 and 84, and higher ($P < 0.05$) percent values of plasma total omega-3 on d 28, 56, 84, and 112 than LO. Steers fed flaxseed products had greater ($P < 0.05$) ALA and total omega-3 than control steers**.** This was expected as the flaxseed products provided more n-3 FA than the control diets (Table 2). The extruded product LinPro induced greater ($P < 0.05$) levels of ALA and total omega-3 in LM tissues than cattle fed LO or control diets. These observations are consistent with previous studies where extruded flaxseed increased LM ALA and total omega-3 compared to control animals (Raes et al., 2004; Barton et al., 2007; Dawson et al., 2010). The current study's observations demonstrate extrusion protects n-3 FA against BH and causes greater LM ALA and total omega-3 depositions than feeding unprotected n-3 FA.

Plasma total omega-6 FA were affected by the interaction of diet and day when values were expressed as a percent of total FA, but not as absolute values. By d 28 and throughout the trial, flaxseed products produced lower ($P < 0.01$) levels of percent plasma total omega-6 than control cattle. The decrease in percent plasma total omega-6 was even greater ($P < 0.05$) on d 56 in cattle fed LinPro compared to LO. Feeding flaxseed products decreased ($P < 0.01$) percent of plasma total omega-6 compared to control cattle. Conversely, diet did not affect ($P \geq 0.13$) levels of LM omega-6. These observations agree with Dawson et al. (2010) and Barton et al. (2007), but contrasts with results of Raes et al. (2004) where a decrease in LM omega-6 was observed when feeding extruded flaxseed. The current observations may be explained by the results of Brenner (1989), where it was shown that n-3 metabolism is preferred over n-6 metabolism.

Therefore, the presence of high n-3 FA, such as in flaxseed, could prevent the use of n-6 FA as a substrate, subsequently preventing any decrease in n-6 FA.

As a result of changes in omega-3 levels, the ratio between n-3 and n-6 FA was affected. Diet interacted with day ($P < 0.01$) so that n-6:n-3 decreased ($P < 0.01$) on d 28 and throughout the trial in cattle fed flaxseed products compared to cattle fed the control diet. Both plasma and LM n-6:n-3 were decreased ($P < 0.01$) in steers fed flaxseed products compared to cattle fed the control diet. This is in agreement with Dawson et al. (2010) and Barton et al. (2007) where feeding extruded flaxseed was reported to decrease ($P < 0.001$) LM n-6:n-3.

Fatty acid saturation (SFA, MUFA, PUFA, PUFA:SFA) was not affected ($P \geq 0.08$) by the interaction between diet and day. Diet did not affect ($P \geq 0.41$) levels of SFA in LM tissues. Previous research has also shown that feeding extruded flaxseed does not affect muscle SFA compared control animals (Raes et al., 2004; Barton et al., 2007). Agreeing with previous research (Raes et al., 2004; Barton et al., 2007; Dawson et al., 2010), diet did not affect ($P \geq 0.19$) plasma or LM MUFA values. Percent of LM PUFA was affected ($P = 0.02$) by diet, although plasma PUFA and LM PUFA absolute values were not affected ($P > 0.05$). These results are in contrast to studies where extruded flaxseed inclusion did not affect muscle PUFA levels (Raes et al., 2004; Barton et al., 2007). A diet effect on LM PUFA was not expected since concentrations of muscle PUFA are primarily impacted by genetics and are only minimally influenced by nutrition (De Smet et al., 2003).

In plasma and LM tissues, PUFA:SFA was greater ($P < 0.05$) in steers fed flaxseed products than those fed SO. These results agree with Dawson et al. (2010), but

contrast Barton et al. (2007). Dawson et al. (2010) used double-muscled Belgium Blue bulls whereas Barton et al. (2007) used Limousin and Charolais steers, which may affected the degree of saturation of the FA profile. Even though LinPro increases n-3 FA in plasma and LM tissues, the extrusion process does not greatly affect biohydrogenation or the saturation of FA.

Including flaxseed products in finishing diets can increase desirable FA in both plasma and LM. Changes in plasma ALA, omega-3 and n-6:n-3 occur quickly, as early as d 28 and remain affected throughout the trial. Feeding LinPro resulted in the greatest concentrations of ALA and total omega-3 FA in LM tissues.

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Table 1. Composition of treatment diets on a DM basis.[a]

	Treatments		
Item, % DM	**Soy Oil**	**Linseed Oil**	**LinPro**
Ingredients			
Dry rolled corn	70.87	70.87	62.75
Corn silage	9.99	9.99	10.00
LinPro	-	-	16.05
Soy oil	4.00	-	-
Linseed oil	-	4.00	-
Corn steep liquor	3.00	3.00	3.00
Molasses	3.00	-	3.00
Soybean meal	4.05	4.05	-
Feed additive	1.96	1.96	1.96
Limestone	1.67	1.67	1.64
Urea, 46% N	1.01	1.01	1.01
Salt	0.30	0.30	0.30
Trace mineral premix	0.06	0.06	0.06
Vitamin premix	0.28	0.28	0.28
Nutrients, %			
DM	73.19	73.52	74.65
CP	12.67	13.15	13.41
P	0.46	0.49	0.48
Ca	0.72	0.72	0.74
Ether extract	7.61	6.17	7.04
NDF	10.43	10.43	11.22

[a]Formulated to provide provide 300 mg/d monensin, 90 mg/d tylosin (Elanco Animal Health, Greenfield, IN), 0.3% salt, 2650 IU vitamin A, 22 IU vitamin E, 0.10 mg Co, 10 mg Cu, 0.5 mg I, 0.25 mg Se, 50 mg Mn, and 50 mg Zn per kg diet DM.

Table 2. Ingredient fatty acid profiles reported on an as-is basis.

Fatty acid, % of sample[‡]	Ingredient		
	Soy Oil	Linseed Oil	LinPro
No. of samples	3	3	3
C10:0	0.007	0.018	0.003
C11:0	0.002	0.007	0.004
C12:0	0.001	0	0.001
C14:0	0.018	0.002	0.002
C14:1	0.006	0	0.002
C15:0	0.009	0.028	0.002
C15:1	0.088	0.006	0.002
C16:0	4.309	5.030	1.459
C16:1	0.003	0.021	0.005
C17:0	0.003	0.026	0.002
C17:1	0.028	0.120	0.012
C18:0	1.706	3.544	0.813
C18:1nt9	0.207	0.028	0.010
C18:1nc9	7.272	17.879	3.978
C18:1n7	0.648	0.696	0.216
CLA c9, t11	0.005	0.353	0.009
CLA t10, c12	0.004	0	0.004
C18:2nt6	0.004	0.004	0
C18:2nc6	20.690	15.819	4.123
C18:3n3	3.059	54.688	12.762
C18:3n6	0.010	0.234	0.079
C20:0	0.138	0.195	0.045
C20:1	0.083	0.122	0.031
C20:2	0.042	0.042	0.011
C20:3n6	0.001	0.001	0.003
C20:4n6	0.033	0.019	0.014
C20:5n3	0.002	0.002	0.022
C21:0	0.030	0	0
C22:0	0.19	0.118	0.034
C22:5n3	0.006	0.003	0.003
C22:6n3	0	0	0.001
C24:0	0.090	0.111	0.047
C24:1	0.032	0.022	0.015
omega-3[a]	3.067	54.694	12.788
omega-6[b]	20.792	16.431	4.235
SFA[c]	6.502	9.079	2.410
MUFA[d]	0.241	0.291	0.067
PUFA[e]	23.901	71.167	17.034

[‡]Number after C denotes number of Carbons; n denotes the double bond location from the omega position; c and t denote the *cis* or *trans* configuration.

[a]omega-3= C18:3n3 + C20:5n3 + C22:5n3 + C22:6n3

[b]omega-6 = C18:2nt6 + C18:2nc6 + CLAc9t11 + CLAt10c12 + C18:3n6 + C20:3n6 + C20:4n6
[c]n-6:n-3 = omega-6 / omega-3
[d]SFA = C14:0 + C15:0 + C16:0 + C17:0 + C18:0 + C20:0 + C21:0 + C22:0 + C24:0
[e]MUFA = C14:1 + C16:1 + C17:1 + C18:1nt9 + C18:1n11 + C18:1nc9 + C18:1n7 + C20:1 + C24:1

Table 3. Effect of diet on performance and carcass characteristics.

	Treatments				
Item	**Soy Oil**	**Linseed Oil**	**LinPro**	**SEM**	***P*-value[§]**
Initial BW, kg	498.0	497.1	497.9	16.0	0.97
Final BW[†], kg	624.5	612.7	626.4	13.1	0.62
DMI, kg/d	12.70	12.03	12.10	0.57	0.59
ADG, kg/d	1.17	1.11	1.20	0.11	0.76
G:F	0.091	0.093	0.100	0.007	0.65
HCW, kg	393.7	389.5	392.5	7.9	0.34
LM area, cm^2	79.5	76.7	79.8	3.8	0.78
Fat thickness, cm	0.55	0.47	0.50	0.08	0.68
KPH, %	2.81	2.93	2.88	0.27	0.95
Dressing percent	63.04	62.13	62.68	0.005	0.07
Liver abcesses, %[¥]	0.0	10.0	0.0	-	1.00
USDA yield grade	2.6	2.5	2.4	0.2	0.59
Marbling score[‡]	557	468	510	47	0.18
Select, %[¥]	0.0	20.0	10.0	-	0.75
Choice, %[¥]	75.0	80.0	70.0	-	1.00
Upper Choice, %[¥]	37.5	40.0	20.0	-	0.68
Prime, %[¥]	25.0	0.0	20.0	-	0.42

[†]Calculated as full BW × 0.96

[‡]300 to 399 = Slight, 400 to 499 = Small, 500 to 599 = Modest, 600 to 699= Moderate

[¥]Monte Carlo Exact Fit Chi Square Analysis (Higgins, 2004)

[a,b,c] Means within a row without common superscripts are different ($P \leq 0.05$)

[§] Effect of diet *P*-value protected by an overall *F*-test ≤ 0.05

Table 4. Effect of diet on plasma concentrations of fatty acids reported on an as-is basis.[§]

Fatty acid,	d 0			d 28			d 56			d 84				P-value[£]		
mg/L[‡]	SO	LO	LP	SO	LO	LP	SO	LO	LP	SO	LO	LP	SEM	Diet	DOF[†]	D×DOF[¥]
C12:0	1.73	3.10	1.31	1.31	1.77	1.53	1.81	1.68	1.42	1.22	1.78	1.41	0.61	0.22	0.63	0.79
C14:0	2.39	2.24	1.32	2.27	2.53	4.98	1.92	3.13	5.21	1.40	2.25	0.98	0.57	0.44	0.03	0.10
C14:1	2.36	1.94	2.07	2.28	3.33	3.44	2.74	3.49	2.82	1.80	4.70	2.43	0.81	0.21	0.39	0.41
C16:0	153.6	147.0	147.1	162.7	141.1	149.4	139.4	118.0	120.4	140.0	128.7	102.9	7.76	0.02	<0.01	0.09
C16:1	21.07	20.17	23.58	12.64	19.87	21.93	4.16	13.12	9.62	3.43	3.52	0.67	1.26	2.33	<0.01	0.06
C17:0	10.02	7.62	6.52	4.71	6.96	6.97	1.98	5.57	2.61	3.07	9.64	6.82	1.89	0.16	0.02	0.28
C17:1	5.43	8.60	8.05	3.64	3.78	5.06	3.29	4.24	3.26	3.50	3.88	2.51	1.14	0.34	<0.01	0.58
C18:0	235.6	241.3	228.7	295.6	264.3	267.6	241.6	229.1	223.9	248.0	217.9	196.1	15.01	0.22	<0.01	0.45
C18:n1t9	26.28	28.48	24.73	31.49	24.18	21.07	19.36	10.68	16.43	13.94	13.71	11.98	3.28	0.25	<0.01	0.30
C18:n1c9	96.98	89.88	94.60	86.83	108.42	104.35	84.71	89.49	85.76	68.90	81.48	68.47	6.25	0.40	<0.01	0.11
C18:1n7	12.98	11.12	10.32	13.53	11.43	10.09	9.87	5.15	4.39	4.92	4.95	4.51	1.79	0.16	<0.01	0.75
CLA c9, t11	3.16	1.86	0.96	2.05	0.21	0.76	0.89	2.05	0.92	2.25	1.40	1.75	0.29	0.56	0.08	0.06
CLA t10, c12	3.14[a]	0.28[b]	1.24[b]	0.90[b]	0.68[b]	0.70[b]	0.89[b]	1.05[b]	0.66[b]	1.38[b]	1.23[b]	0.88[b]	0.33	0.50	0.17	0.04
C18:2nt6	4.37	4.95	3.95	4.88	9.92	6.47	1.25	2.50	0.85	1.83	1.76	2.32	0.59	0.11	<0.01	0.40
C18:2nc6	595.4	631.6	576.0	906.1	792.3	758.5	745.3	707.6	662.2	703.2	686.6	610.6	42.2	0.20	<0.01	0.18
C18:3n3	24.01[abcde]	19.11[abc]	22.15[abcd]	27.97[abcdef]	132.74[ghi]	167.01[j]	18.66[ab]	117.85[g]	168.04[j]	15.53[a]	123.69[gh]	157.24[ij]	10.20	<0.01	<0.01	<0.01
C18:3n6	19.77[abc]	18.28[abcd]	21.52[a]	13.57[cde]	12.53[defg]	12.99[def]	20.45[ab]	7.45[efgh]	7.35[efgh]	11.52[efgh]	6.43[h]	5.22[h]	2.37	0.01	<0.01	0.02
C20:0	2.76	2.28	2.68	2.54	1.21	1.58	1.73	1.76	1.67	2.56	2.69	1.14	0.64	0.57	0.18	0.40
C20:1	2.55	1.74	1.84	1.43	1.39	1.25	1.39	1.72	0.91	1.05	3.53	1.32	0.84	0.45	0.56	0.52
C20:2	7.47	5.51	4.69	2.33	4.25	4.35	2.91	4.08	4.91	3.36	3.24	4.84	1.00	0.67	0.01	0.12
C20:3n6	42.94[a]	42.01[ab]	39.54[abc]	31.29[cde]	18.66[fgh]	20.43[fg]	33.87[bcd]	15.21[gh]	15.49[gh]	26.88[def]	17.43[gh]	12.47[h]	3.06	<0.01	<0.01	0.05
C20:4n6	0.986	1.443	0.666	0.401	0.409	0.644	0.188	1.201	0.851	0.955	1.447	2.705	0.35	0.58	0.06	0.47
C20:5n3	5.16	8.60	7.61	6.57	9.30	13.03	4.77	9.30	12.96	5.24	10.86	12.80	1.39	<0.01	0.03	0.16
C21:0	3.37	2.73	1.95	1.88	0.47	0.94	1.57	1.78	0.86	1.22	1.63	1.39	0.68	0.30	0.02	0.74
C22:0	3.71	3.52	2.15	2.07	2.04	1.26	2.30	0.92	1.72	2.69	1.82	1.67	0.38	0.74	0.06	0.82
C22:5n3	2.95	3.69	2.65	3.66	2.56	0.98	1.22	2.52	1.50	3.33	3.55	3.07	0.75	0.12	0.04	0.48
C22:6n3	6.50	6.41	6.92	3.72	3.93	5.54	4.18	2.19	2.86	2.00	2.44	2.01	0.91	0.68	<0.01	0.56
C24:0	1.46	3.00	3.42	3.01	2.96	3.02	2.05	2.65	1.56	1.72	4.26	2.23	0.82	0.17	0.53	0.41
C24:1	2.49	1.99	1.61	1.72	2.02	1.68	1.35	2.00	1.30	2.03	2.12	1.73	0.55	0.45	0.69	0.96
Total	1307	1325	1253	1644	1589	1600	1359	1368	1363	1280	1349	1227	72	0.82	<0.01	0.74

[§]SO = Soy Oil, LO = Linseed Oil, LP = LinPro.
[£]Effect of diet, days on feed, and interaction between diet and days on feed *P*-values protected by an overall *F*-test ≤ 0.05
[†]DOF = Days on feed
[¥] Interaction between diet and days on feed
[‡]Number after C denotes number of Carbons; n denotes the double bond location from the omega position; c and t denote the *cis* or *trans* configuration.
[a,b,c]Means within a row without common superscripts are different ($P \leq 0.05$).

Table 5. Effect of diet on plasma concentrations of fatty acids expressed as a percent of total plasma fatty acids reported on an as-is basis.[§]

Fatty acid, % of total[‡]	d 0			d 28			d 56			d 84			SEM	P-value[£]		
	SO	LO	LP	SO	LO	LP	SO	LO	LP	SO	LO	LP		Diet	DOF	D×DOF[¥]
C12:0	0.131	0.236	0.103	0.082	0.119	0.089	0.142	0.116	0.107	0.119	0.126	0.105	0.046	0.30	0.42	0.74
C14:0	0.173	0.176	0.102	0.142	0.166	0.315	0.137	0.232	0.365	0.107	0.172	0.080	0.071	0.49	0.06	0.10
C14:1	0.164	0.154	0.161	0.136	0.209	0.214	0.211	0.273	0.195	0.142	0.363	0.195	0.060	0.20	0.30	0.39
C16:0	11.86^{a}	11.11^{abc}	11.72^{ab}	9.91^{def}	8.93^{fgh}	9.32^{efgh}	10.28^{cde}	8.69^{gh}	8.90^{fgh}	11.04^{abcd}	9.60^{efg}	8.31^{h}	0.41	<0.01	<0.01	0.04
C16:1	1.596^{ab}	1.528^{abc}	1.872^{a}	0.764^{fg}	1.245^{bcde}	1.390^{bcd}	0.311^{hi}	0.974^{def}	0.683^{fgh}	0.241^{i}	0.317^{hi}	0.057^{i}	0.160	0.02	<0.01	0.04
C17:0	0.738	0.570	0.523	0.278	0.481	0.451	0.145	0.426	0.206	0.231	0.715	0.538	0.135	0.16	<0.01	0.24
C17:1	0.430	0.708	0.621	0.229	0.233	0.316	0.245	0.307	0.257	0.293	0.324	0.208	0.097	0.38	<0.01	0.65
C18:0	18.09^{abcd}	18.29^{ab}	18.23^{abc}	17.97^{bcde}	16.52^{efg}	16.63^{defg}	17.80^{bcdef}	16.75^{cdefg}	16.49^{efg}	19.34^{a}	16.27^{fg}	16.01^{g}	0.57	<0.01	0.01	0.03
C18:n1t9	2.01	2.11	2.01	1.88	1.48	1.31	1.43	0.77	1.20	1.10	1.00	0.99	0.20	0.16	<0.01	0.35
C18:n1c9	7.56^{ab}	6.85^{abc}	7.63^{a}	5.26^{g}	6.77^{abcd}	6.57^{bcde}	6.21^{cdefg}	6.57^{bcdef}	6.34^{cdefg}	5.41^{g}	6.13^{cdefg}	5.63^{defg}	0.40	0.37	<0.01	0.05
C18:1n7	0.987	0.834	0.830	0.815	0.712	0.633	0.721	0.358	0.346	0.387	0.376	0.378	0.12	0.21	<0.01	0.69
CLA c9, t11	0.243	0.139	0.086	0.127	0.013	0.051	0.063	0.152	0.070	0.180	0.109	0.153	0.04	0.17	0.02	0.10
CLA t10, c12	0.259^{a}	0.021^{b}	0.110^{b}	0.056^{b}	0.047^{b}	0.048^{b}	0.066^{b}	0.078^{b}	0.047^{b}	0.010^{b}	0.096^{b}	0.079^{b}	0.040	0.10	0.05	0.03
C18:2nt6	0.332	0.365	0.314	0.298	0.632	0.422	0.096	0.182	0.069	0.165	0.123	0.193	0.088	0.20	<0.01	0.37
C18:2nc6	45.30^{a}	47.25^{abc}	45.87^{ab}	55.22^{j}	50.01^{defg}	47.46^{abcd}	54.75^{ij}	51.64^{efghi}	48.39^{abcde}	54.79^{ij}	50.60^{defgh}	49.60^{cdef}	1.22	<0.01	<0.01	<0.01
C18:3n3	1.80^{abcdef}	1.42^{abc}	1.77^{abcde}	1.71^{abcd}	8.32^{g}	10.42^{j}	1.38^{ab}	8.58^{gh}	12.30^{k}	1.20^{a}	9.00^{ghi}	12.94^{k}	0.50	<0.01	<0.01	<0.01
C18:3n6	1.478^{abc}	1.452^{abcd}	1.721^{a}	0.811^{ef}	0.795^{ef}	0.809^{ef}	1.538^{ab}	0.559^{ef}	0.547^{ef}	0.910^{e}	0.482^{ef}	0.426^{f}	0.17	0.02	<0.01	<0.01
C20:0	0.204	0.182	0.210	0.154	0.080	0.097	0.133	0.131	0.125	0.203	0.192	0.088	0.047	0.61	0.04	0.53
C20:1	0.173	0.131	0.145	0.087	0.085	0.074	0.109	0.123	0.061	0.089	0.258	0.114	0.062	0.52	0.33	0.64
C20:2	0.598	0.424	0.379	0.146	0.265	0.268	0.223	0.289	0.377	0.275	0.251	0.395	0.081	0.70	<0.01	0.15
C20:3n6	3.33^{a}	3.20^{ab}	3.15^{abc}	1.90^{ef}	1.14^{g}	1.26^{g}	2.48^{d}	1.13^{g}	1.16^{g}	2.09^{de}	1.27^{g}	1.00^{g}	0.20	<0.01	<0.01	0.04
C20:4n6	0.073	0.099	0.053	0.022	0.029	0.035	0.012	0.078	0.053	0.080	0.106	0.225	0.042	0.29	<0.01	0.34
C20:5n3	0.392	0.685	0.615	0.395	0.612	0.815	0.356	0.670	0.968	0.427	0.888	1.042	0.113	<0.01	0.04	0.29
C21:0	0.257	0.209	0.161	0.119	0.032	0.057	0.115	0.127	0.071	0.093	0.123	0.122	0.052	0.49	<0.01	0.83
C22:0	0.289	0.305	0.177	0.127	0.132	0.083	0.174	0.070	0.127	0.216	0.127	0.129	0.061	0.24	0.02	0.78
C22:5n3	0.217	0.291	0.220	0.226	0.166	0.065	0.090	0.190	0.113	0.276	0.270	0.259	0.061	0.28	0.01	0.68
C22:6n3	0.512	0.509	0.554	0.229	0.238	0.344	0.315	0.163	0.205	0.149	0.175	0.167	0.068	0.60	<0.01	0.66
C24:0	0.112	0.233	0.292	0.180	0.194	0.201	0.144	0.195	0.119	0.143	0.331	0.201	0.065	0.20	0.46	0.43
C24:1	0.182	0.155	0.134	0.102	0.125	0.100	0.096	0.146	0.097	0.146	0.150	0.150	0.038	0.65	0.28	0.96

[§]SO = Soy Oil, LO = Linseed Oil, LP = LinPro.

[£]Effect of diet, days on feed, and interaction between diet and days on feed *P*-values protected by an overall *F*-test ≤ 0.05

[†]DOF = Days on feed

[¥] Interaction between diet and days on feed

[‡]Number after C denotes number of Carbons; n denotes the double bond location from the omega position; c and t denote the *cis* or *trans* configuration.

[a,b,c] Means within a row without common superscripts are different ($P \leq 0.05$).

Table 6. Effect of diet on longissimus muscle concentrations of fatty acids reported on an as-is basis.

Fatty acid, mg/kg[‡]	Treatments			SEM	*P*-value[§]
	Soy Oil	Linseed Oil	LinPro		
C14:0	0.284	0.219	0.213	0.038	0.27
C14:1	0.046	0.031	0.028	0.012	0.49
C16:0	2.107	1.747	1.793	0.244	0.45
C16:1	0.357	0.269	0.291	0.044	0.20
C17:0	0.093	0.100	0.097	0.005	0.50
C17:1	0.069	0.054	0.048	0.009	0.15
C18:0	1.18	1.11	1.16	0.136	0.90
C18:1nt9	0.274[a]	0.185[b]	0.204[ab]	0.028	0.04
C18:1nc9	2.82	2.37	2.44	0.29	0.43
C18:1n7	0.135	0.098	0.110	0.015	0.21
CLA c9, t11	0.0034[a]	0.0001[b]	0.0007[b]	0.0004	<0.01
CLA t10, c12	0.0006	0.0002	0.0005	0.0002	0.35
C18:2nt6	0.014[a]	0.025[b]	0.028[b]	0.003	<0.01
C18:2nc6	0.339	0.301	0.293	0.024	0.31
C18:3n3	0.015[a]	0.057[b]	0.089[c]	0.010	<0.01
C18:3n6	0.003	0.002	0.003	0.0007	0.37
C20:0	0.005[a]	0.011[b]	0.011[b]	0.002	<0.01
C20:1	0.018	0.013	0.015	0.002	0.08
C20:2	0.006[a]	0.004[b]	0.006[a]	0.0008	0.04
C20:3n6	0.012	0.009	0.010	0.002	0.45
C20:4n6	0.001[a]	0.002[b]	0.003[c]	0.0004	<0.01
C20:5n3	0.002	0.003	0.001	0.0008	0.17
C21:0	0.021[a]	0.009[b]	0.019[a]	0.003	<0.01
C22:0	0.003	0.003	0.004	0.0006	0.24
C22:5n3	0.003	0.003	0.002	0.0008	0.33
C22:6n3	0.003	0.005	0.006	0.002	0.53
C24:0	0.0030[a]	0.0024[ab]	0.0019[b]	0.0003	0.05
C24:1	0.009	0.009	0.001	0.0004	0.96
Total	7.83	6.65	6.90	0.81	0.47
omega-3[d]	0.023[a]	0.067[b]	0.099[c]	0.010	<0.01
omega-6[e]	0.374	0.340	0.340	0.026	0.53
n-6:n-3[f]	17.69[a]	5.55[b]	3.64[b]	1.25	<0.01
SFA[g]	3.70	3.21	3.31	0.42	0.59
MUFA[h]	3.71	3.01	3.13	0.37	0.30
PUFA[i]	0.398	0.406	0.438	0.033	0.63
PUFA:SFA[j]	0.109[a]	0.130[ab]	0.138[b]	0.010	0.04

[§] Effect of diet *P*-value protected by an overall *F*-test ≤ 0.05.

[‡]Number after C denotes number of Carbons; n denotes the double bond location from the omega position; c and t denote the *cis* or *trans* configuration.

[a,b,c] Means within a row without common superscripts are different ($P \leq 0.05$).

[d]omega-3= C18:3n3 + C20:5n3 + C22:5n3 + C22:6n3

[e]omega-6 = C18:2nt6 + C18:2nc6 + CLAc9t11 + CLAt10c12 + C18:3n6 + C20:3n6 + C20:4n6

[f]n-6:n-3 = omega-6 / omega-3
[g]SFA = C14:0 + C15:0 + C16:0 + C17:0 + C18:0 + C20:0 + C21:0 + C22:0 + C24:0
[h]MUFA = C14:1 + C16:1 + C17:1 + C18:1nt9 + C18:1n11 + C18:1nc9 + C18:1n7 + C20:1 + C24:1
[i]PUFA = C18:2n6t + C18:2n6c + CLAc9t11 + CLAt10c12 + C18:3n6 + C18:3n3 + C20:2 + C20:3n6 + C20:4n6 + C20:5n3 + C22:5n3 + C22:6n3
[j]PUFA:SFA = PUFA / SFA

Table 7. Effect of diet on longissimus muscle fatty acids expressed as a percentage of total fatty acids reported on an as-is basis.

Fatty acid, % of total[‡]	Treatments			SEM	*P*-value[§]
	Soy Oil	Linseed Oil	LinPro		
C14:0	3.45	3.18	2.89	0.19	0.12
C14:1	0.558	0.475	0.364	0.136	0.57
C16:0	26.66	26.07	25.62	0.56	0.40
C16:1	4.16	3.71	3.75	0.28	0.39
C17:0	1.19	1.52	1.38	0.18	0.29
C17:1	0.874	0.836	0.668	0.080	0.13
C18:0	14.82	16.03	16.30	0.68	0.25
C18:1nt9	3.60	2.86	3.00	0.25	0.09
C18:1nc9	36.56	36.23	36.29	0.66	0.93
C18:1n7	1.74	1.53	1.64	0.25	0.78
CLA c9, t11	0.044[a]	0.003[b]	0.011[b]	0.005	<0.01
CLA t10, c12	0.009	0.004	0.009	0.004	0.62
C18:2nt6	0.179[a]	0.402[b]	0.442[b]	0.044	<0.01
C18:2nc6	4.36	4.64	4.37	0.32	0.68
C18:3n3	0.198[a]	0.878[b]	1.382[c]	0.098	<0.01
C18:3n6	0.034	0.022	0.023	0.013	0.66
C20:0	0.037[a]	0.162[b]	0.138[b]	0.029	<0.01
C20:1	0.242	0.199	0.229	0.019	0.23
C20:2	0.083[ab]	0.055[a]	0.099[b]	0.014	0.05
C20:3n6	0.172	0.134	0.169	0.032	0.61
C20:4n6	0.012[a]	0.032[b]	0.047[c]	0.007	<0.01
C20:5n3	0.023	0.053	0.022	0.015	0.20
C21:0	0.265[a]	0.139[b]	0.295[a]	0.030	<0.01
C22:0	0.040	0.045	0.064	0.015	0.37
C22:5n3	0.034	0.035	0.007	0.015	0.17
C22:6n3	0.051	0.073	0.098	0.028	0.46
C24:0	0.039	0.036	0.026	0.007	0.32
C24:1	0.013	0.013	0.015	0.005	0.91
omega-3[d]	0.314[a]	1.050[b]	1.525[c]	0.105	<0.01
omega-6[e]	4.82	5.25	5.08	0.35	0.59
SFA[f]	46.68	47.39	46.97	0.89	0.84
MUFA[g]	47.81	45.91	46.03	0.97	0.30
PUFA[h]	5.18[a]	6.32[b]	6.66[b]	0.42	0.02

[§] Effect of diet *P*-value protected by an overall *F*-test ≤ 0.05.

[‡]Number after C denotes number of Carbons; n denotes the double bond location from the omega position; c and t denote the *cis* or *trans* configuration.

[a,b,c]Means within a row without common superscripts are different ($P \leq 0.05$).

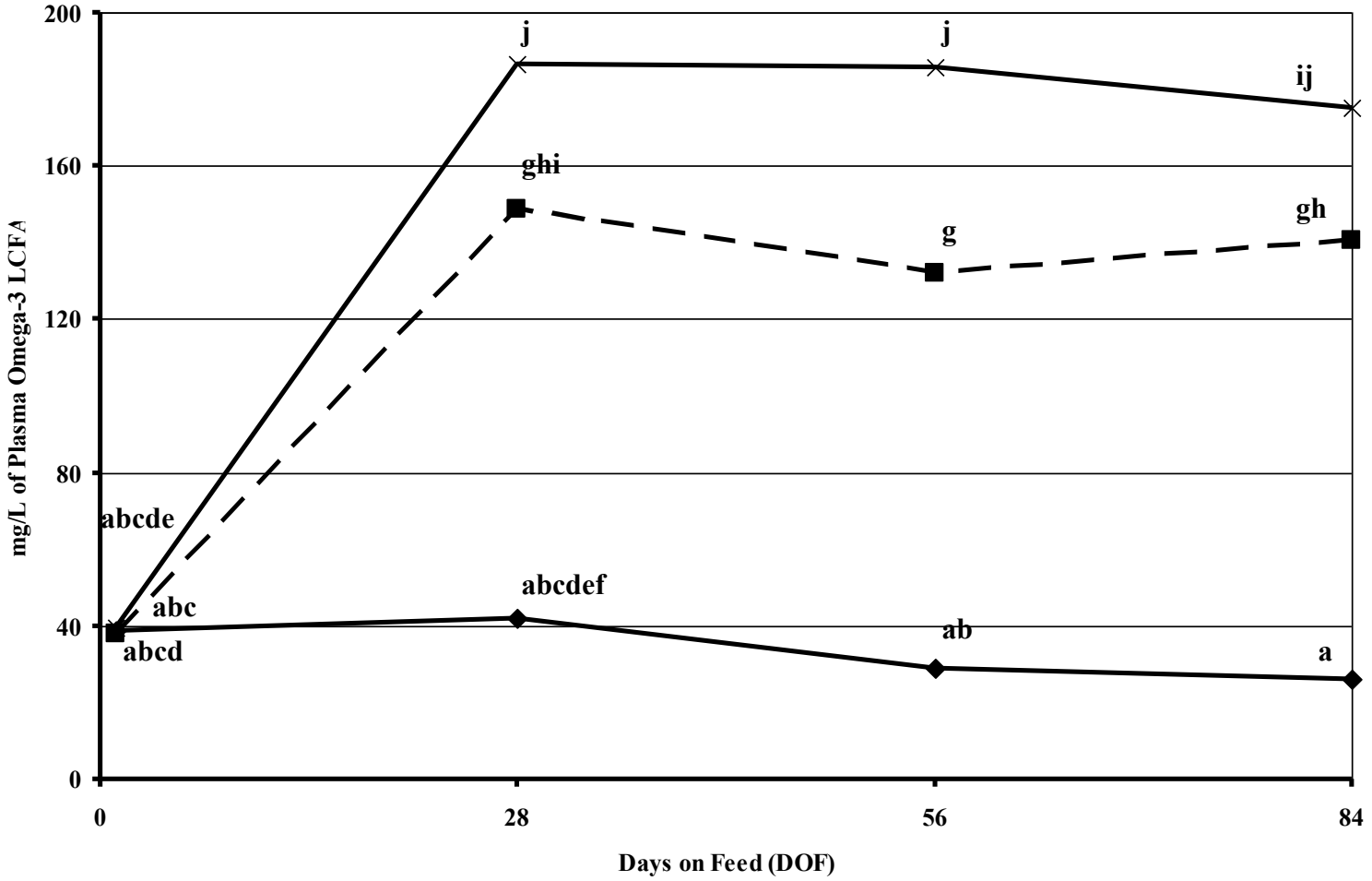
[d]omega-3= C18:3n3 + C20:5n3 + C22:5n3 + C22:6n3

[e]omega-6 = C18:2nt6 + C18:2nc6 + CLAc9t11 + CLAt10c12 + C18:3n6 + C20:3n6 + C20:4n6

[f]SFA = C14:0 + C16:0 + C17:0 + C18:0 + C20:0 + C21:0 + C22:0 + C24:0

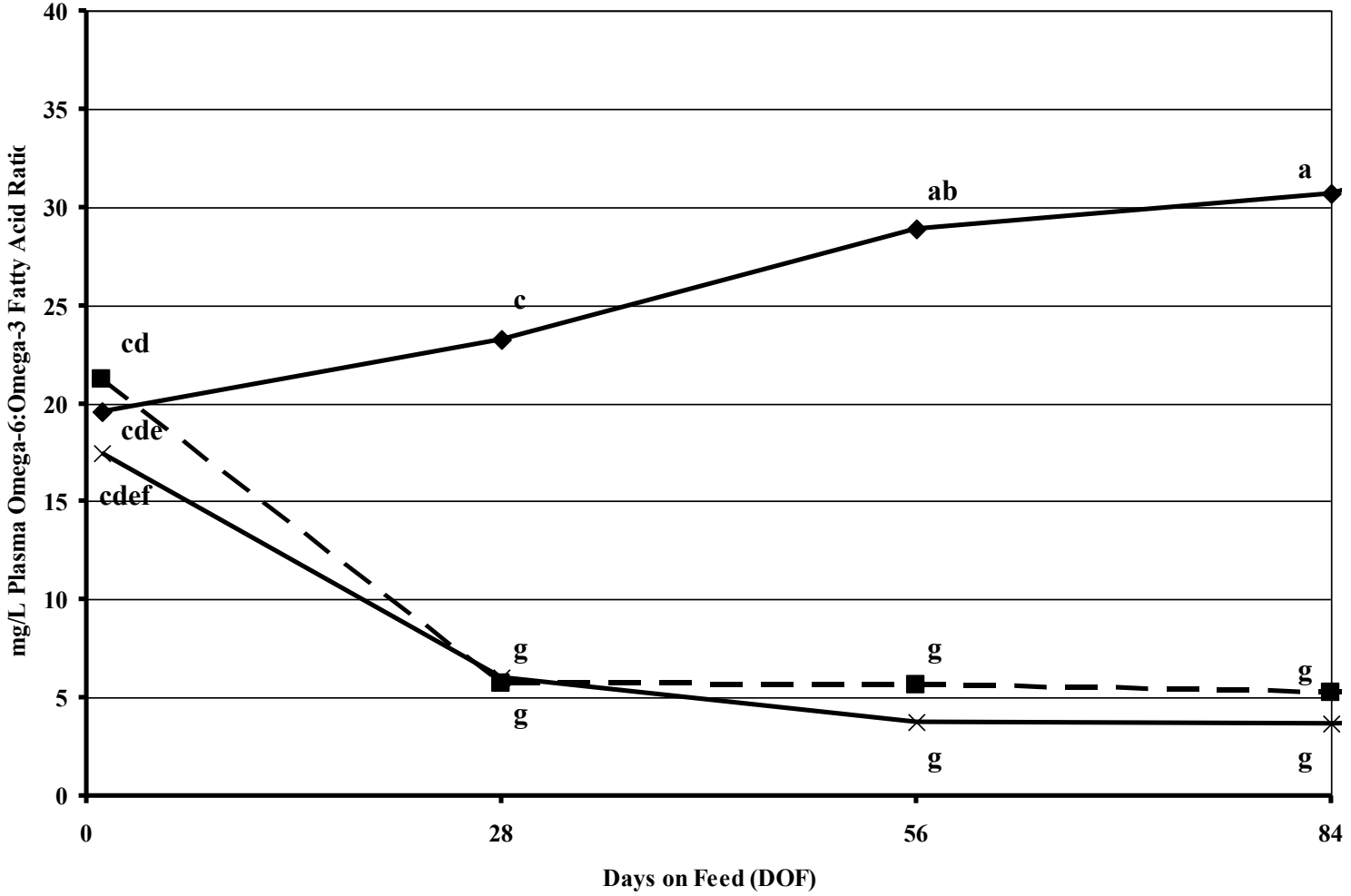
[g]MUFA = C14:1 + C15:1 + C16:1 + C17:1 + C18:1nt9 + C18:1n11 + C18:1nc9 + C18:1n7 + C20:1 + C24:1 [i]PUFA = C18:2n6t + C18:2n6c + CLAc9t11 + CLAt10c12 + C18:3n6 + C18:3n3 + C20:2 + C20:3n6 + C20:4n6 + C20:5n3 + C22:5n3 + C22:6n3

A.



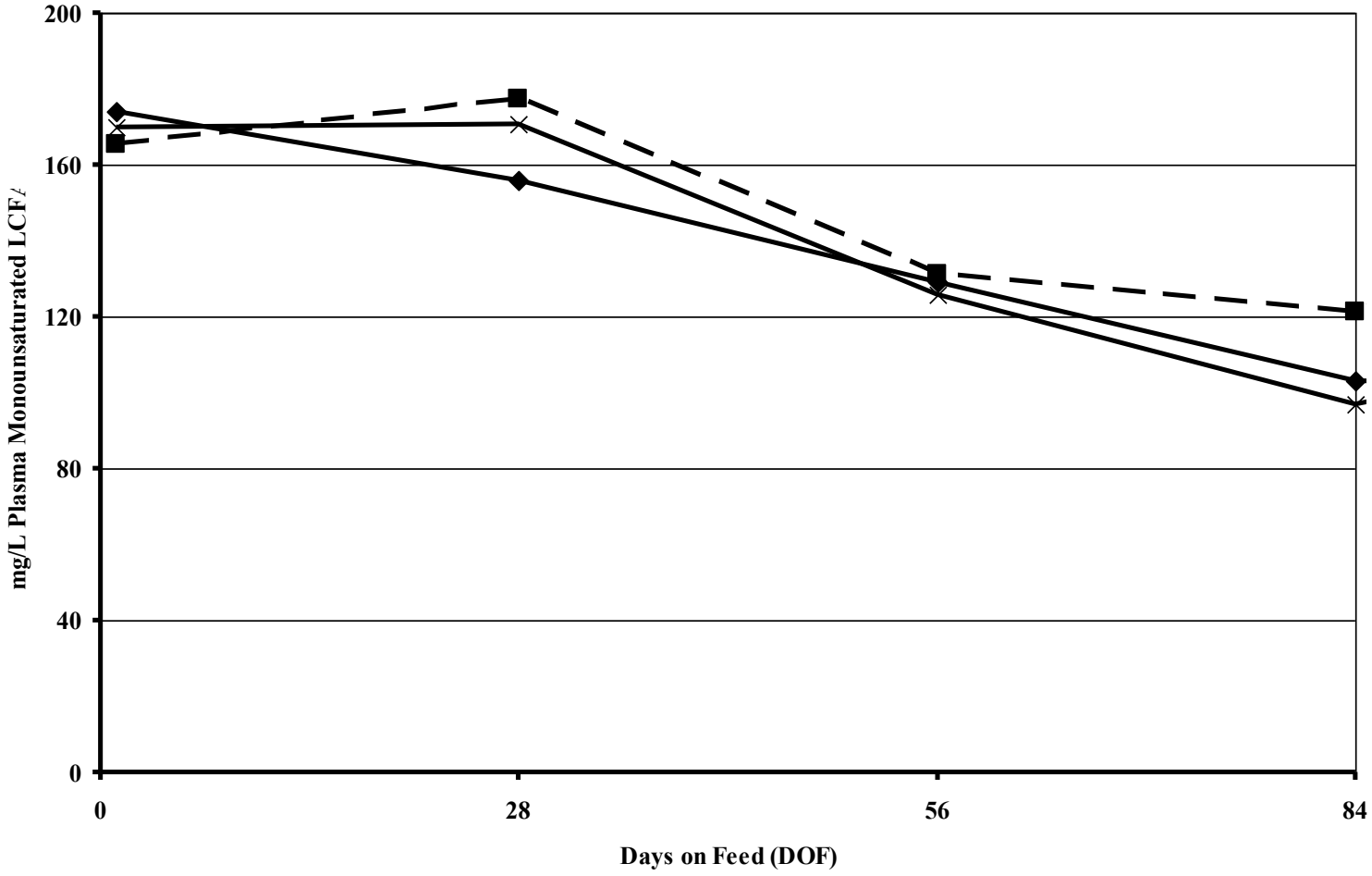
SEM = 10.5 Diet×DOF, P < 0.01 Diet, P < 0.01 DOF, P < 0.01

B.



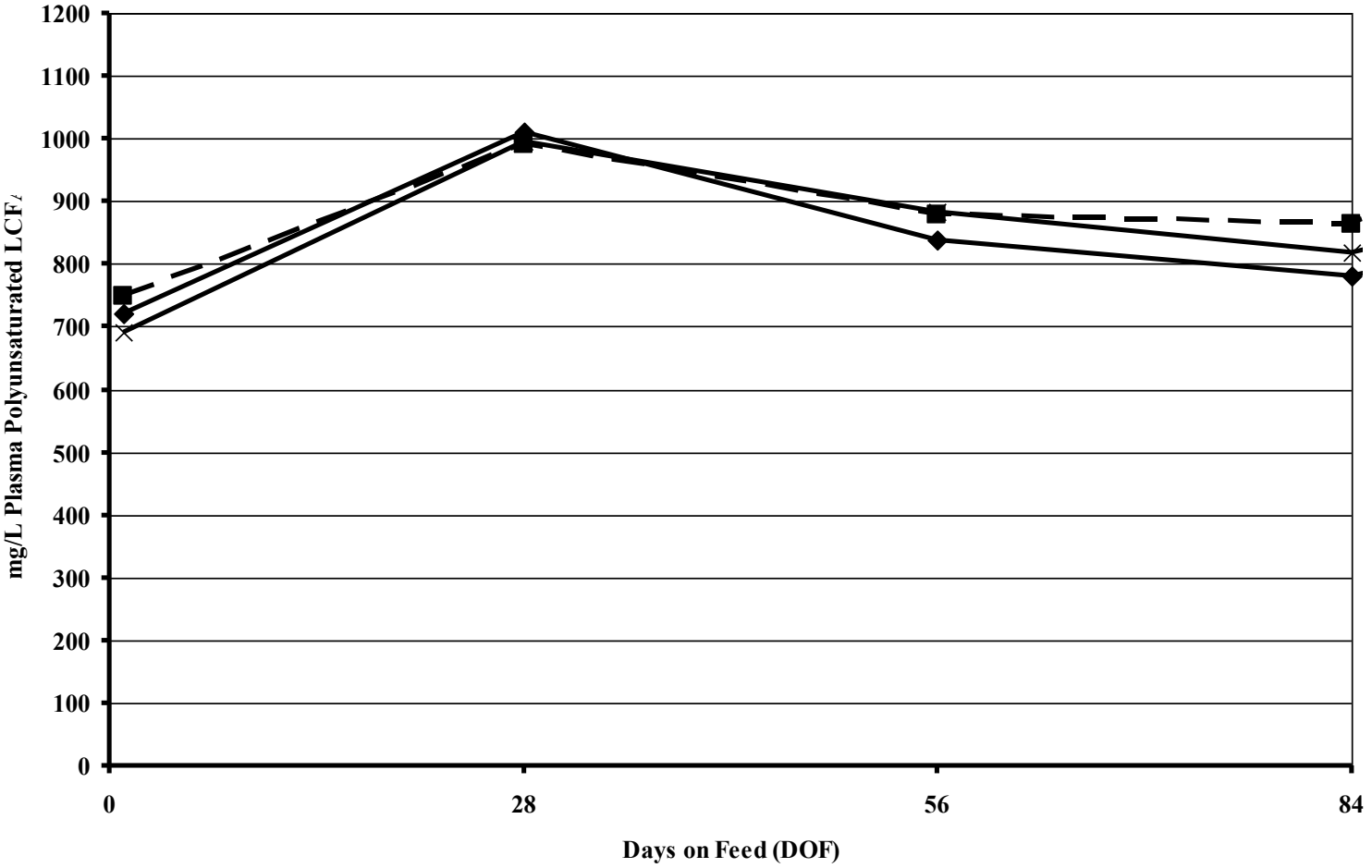
SEM = 1.7 Diet×DOF, $P < 0.01$ Diet, $P < 0.01$ DOF, $P < 0.01$

C.



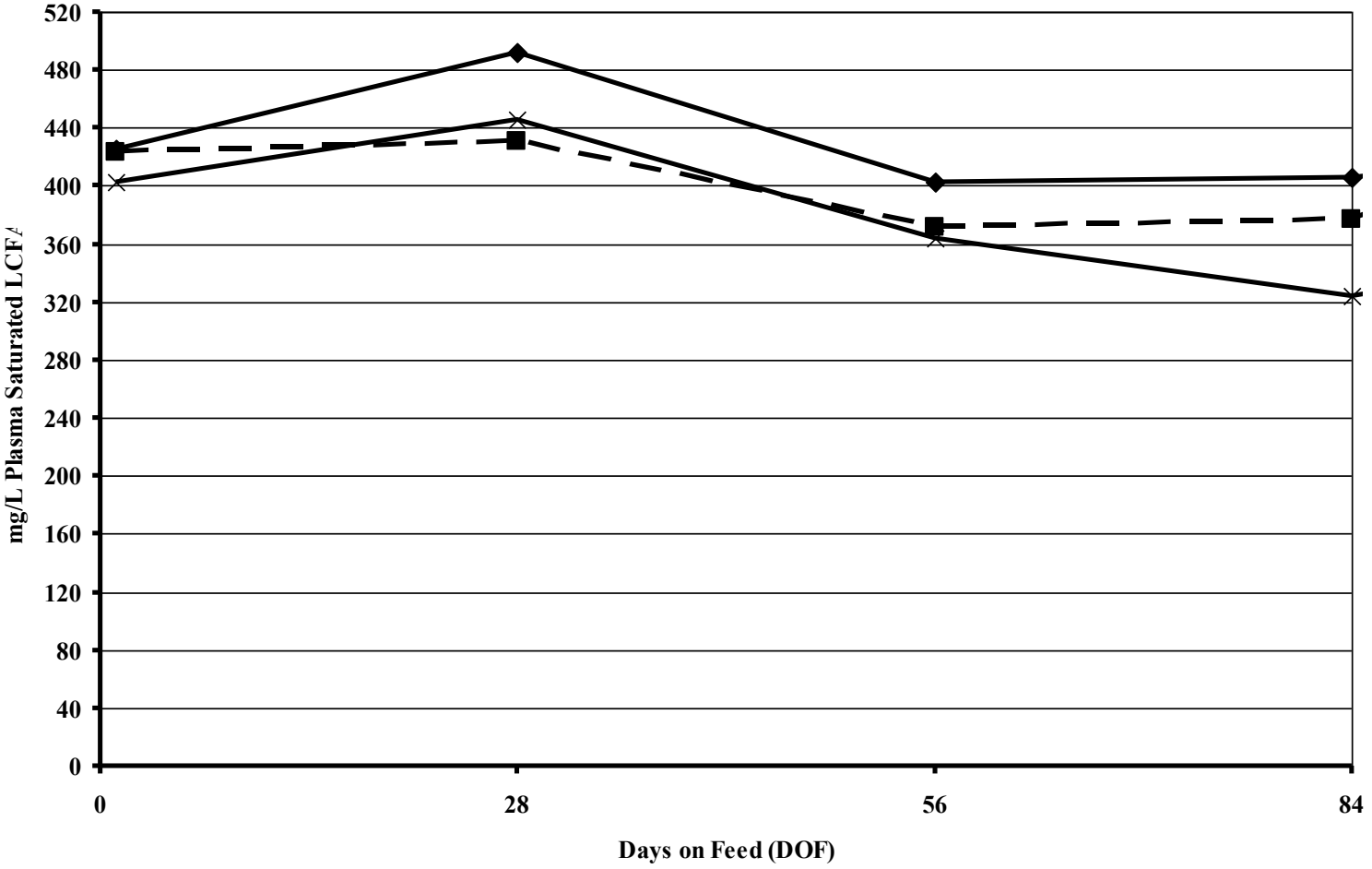
SEM = 10.75 Diet×DOF, P = 0.51 Diet, P = 0.62 DOF, P < 0.01

D.



SEM = 49.3 Diet×DOF, P = 0.68 Diet, P = 0.83 DOF, P < 0.01

E.



SEM = 22.2 Diet×DOF, P = 0.31 Diet, P = 0.12 DOF, P < 0.01

Figure 1. Effect of diet, day, and the interaction between diet and day on plasma concentrations of fatty acids reported on an as-is basis.
Soy Oil —♦—; Linseed Oil — ■ —; LinPro —×—

A. Effect of diet, day, and the interaction between diet and day on plasma concentrations of omega-3 fatty acids.
B. Effect of diet, day, and the interaction between diet and day on plasma n-6:n-3 fatty acid ratio.
C. Effect of diet, day, and the interaction between diet and day on plasma concentrations of monounsaturated fatty acids.
D. Effect of diet, day, and the interaction between diet and day on plasma concentrations of polyunsaturated fatty acids.
E. Effect of diet, day, and the interaction between diet and day on plasma concentrations of saturated fatty acids.

CHAPTER 4: Flaxseed products do not interact with exogenous steroids in finishing cattle

ABSTRACT

The effects of flaxseed products and exogenous steroids on performance, carcass quality, and plasma and LM fatty acid (FA) profiles were evaluated. In study 1, steers were fed diets with tallow (Con) or flaxseed (Flaxseed), implanted with Revalor S (Imp; Revalor S, 120 mg of trenbolone acetate, 24 mg of estradiol**;** Intervet Inc., Millsboro, DE) or not implanted (No Imp), resulting in four treatments: Con-Imp, Con-No Imp, Flaxseed-Imp, and Flaxseed-No Imp. In study 2, steers were fed diets including soy oil (Con) or flaxseed (Flaxseed), implanted with Revalor XS (Imp; Revalor XS, 200 mg of trenbolone acetate, 40 mg of estradiol; Intervet Inc., Millsboro, DE) or not implanted (No Imp), resulting in four treatments: Con-Imp, Con-No Imp, Flaxseed-Imp, and Flaxseed-No Imp. Plasma samples were collected every 28 d. After a 48-h chill, full rib primals were collected. Plasma and lean tissues were analyzed for FA profiles. In study 3, heifers were fed diets with or without linseed meal (LSM) and management regimes with or without an estradiol/trenbolone acetate implant (Rev IH, 80 mg trenbolone acetate and 8 mg estradiol; Intervet Inc, Millsboro, DE) and melengestrol acetate (MGA; Pfizer Animal Health; New York, NY). This resulted in four treatments, a conventional program consisting of Rev IH and MGA with no added LSM (Con-Conv); Rev IH and MGA with LSM (LSM-Conv); a nonhormone treated cattle (NHTC) program with no implant or MGA with no added LSM (Con-NHTC); and no implant or MGA with LSM (LSM-NHTC). In all studies residual feed and BW were determined every 28 d period and prior to transport for harvest, and carcass quality was determined for each animal following a 48-h chill. Interactions between diet and exogenous hormones occurred in study 1

tending to affect KPH ($P = 0.08$) and in study 2 affecting yield grade (YG; $P = 0.03$) and marbling scores ($P = 0.04$); however there were no interactions ($P \geq 0.12$) in study 3. Feeding flaxseed improved YG ($P < 0.01$) and decreased BF ($P < 0.01$) compared to steers fed control diets in study 2. Exogenous hormones consistently improved ($P \leq 0.03$) DMI, ADG, feed efficiency, and HCW; but also exerted inconsistent effects on YG, dressing percentage and KPH. The LM of cattle fed flaxseed had increased ($P < 0.01$) concentrations of α-linolenic acid and total omega-3 FA, and decreased ($P < 0.01$) n-6:n-3 compared to control cattle. Contrary to our hypothesis, flaxseed products did not act as natural growth promoters in NHTC cattle.

Key words: finishing, flaxseed, linseed meal, steroidal implant, MGA, fatty acids

Introduction

Flaxseed is rich in secoisolariciresinol diglycoside (SDG) and omega-3 fatty acids (FA) and is fed in many forms to livestock. There is evidence indicating gastrointestinal bacteria can convert SDG to estrogen-like compounds that are metabolically active in mammals (Clavel et al., 2005 and Thompson et al., 1991). Research shows this metabolism also occurs in goats (Zhou et al., 2009a). These estrogen-like metabolites affect lipid metabolism in diet-induced obese rats (Fukumitsu et al., 2008); increase skeletal muscle growth in rats (Zhou et al., 2009b); and interact with estradiol-17β implants to affect estrogen receptors and mass of digestive organs in sheep (O'Neil et al., 2009), and alter liver mass and proteins (O'Neil et al., 2006). One study (Dunn et al., 2003) reported that implanted steers fed without flaxseed had greater ADG than implanted steers fed flaxseed, whereas the non-implanted steers fed flaxseed had

numerically greater gains than the non-implanted steers without flaxseed, suggesting a potential hormonal effect of the flaxseed.

Flaxseed is rich in α-linolenic acid (ALA) and has been recorded to increase concentrations of n-3 FA in beef (Maddock et al., 2006; La Brune et al., 2008). Beef enriched with n-3 FA could be a useful source of the essential FA to U.S. consumers given that many Americans do not consume the recommended amounts of n-3 FA (Kris-Etherton et al., 2000).

To our knowledge previous experiments have not studied the estrogenic effects of SDG, and how it may interact with exogenous hormones to affect growth in cattle. We assessed the interactions between ground flaxseed and implants on performance and carcass quality in studies 1 and 2. In study 2, we also evaluated the interactions between ground flaxseed and an implant on plasma and LM FA profiles. In study 3, LSM was fed to remove the effects of flaxseed oil so as to study only SDG and its interactions with exogenous steroids and the effects on performance and carcass quality.

Materials and Methods

This study was conducted in accordance with procedures approved by the Kansas State University Institutional Animal Care and Use Committee.

Experiment 1

Yearling steers were used in a 2 × 2 factorial arrangement to test the effects of ground flaxseed and estradiol/trenbolone acetate implants on feedlot performance and carcass quality. Steers (n = 246; initial BW = 396 kg) were housed in concrete-surfaced pens (36 m^2) with 6 to 7 animals each. Pens provided overhead shade covering the fenceline bunk and half of each pen. Pens included automatic water fountains and 3.2 m

of bunk space. Starting d -14, cattle were fed transition diets to reduce variation in gut fill. Steers were blocked by previous treatment (once vs twice per d feeding) and assigned randomly to 1 of 4 finishing diet treatments: no implant with beef tallow (Control NI), no implant with 10% ground flaxseed (Flaxseed NI), Revalor-S implant with beef tallow (Control RevS), and Revalor-S implant with 10% ground flaxseed (Flaxseed RevS). Composition of diets is summarized in Table 1.

On d 1, steers were weighed and implanted (Revalor S, 120 mg of trenbolone acetate, 24 mg of estradiol**;** Intervet Inc., Millsboro, DE) according to treatment. Diets (Table 1) were formulated to be isocaloric and isonitrogenous and were fed *ad libitum* once daily for 80 d. Water was offered *ad libitum* provided by an automatic water fountains. Weights of fresh feed were recorded daily. Dry matter composition of each diet was determined weekly to allow for determination of total feed consumption for each pen. Feed refusals were recorded every 28 d or when in excess. These amounts on a DM basis were subtracted from the total feed offered to determine actual feed intake for each pen of cattle.

At the end of the finishing period, pen weights were recorded and cattle were transported to a commercial abattoir. At harvest carcass weights were recorded. After a 24-h chill, subcutaneous fat thickness over the 12th rib, LM area, KPH, USDA quality grade, and USDA yield grade were determined.

Experiment 2

Holstein steers (n = 40; initial BW = 499 ± 46 kg) were used in a randomized complete block experiment with a 2 × 2 factorial treatment arrangement to evaluate the effects of ground flaxseed and steroidal implants on feedlot performance, carcass quality

and fatty acid composition in plasma and LM. Experimental treatments included diets of dry rolled corn with soy oil or 10% ground flaxseed, with or without an estradiol/trenbolone acetate implant. This resulted in four treatments: soy oil with implant (Control Imp), soy oil without implant (Control No Imp), flaxseed diet with implant (Flaxseed Imp), and flaxseed diet without implant (Flaxseed No Imp). Composition of diets is summarized in Table 2.

To minimize differences in gastrointestinal tract fill, steers were fed a common diet based on dry rolled corn for several d before initiation of the experiment. On d 1, steers were dewormed (Safeguard, Intervet Inc., Millsboro, DE), individually weighed, and identified with individually numbered ear tags on d 1. Baseline blood samples were collected in heparinized vacuum tubes (BD, Franklin Lakes, NJ) via jugular vein. Tubes were immediately placed onto ice before centrifuging at 3200 × *g* for 10 min to recover plasma. Every 28-d period cattle were weighed individually, residual feed collected, and blood samples collected. Cattle were blocked by individual BW and assigned randomly to 1 of 4 treatments. Steers were implanted (Revalor XS, 200 mg of trenbolone acetate, 40 mg of estradiol; Intervet Inc., Millsboro, DE) according to treatment and then placed in 1 of 4 barns consisting of 20 individual partially-covered concrete pens per barn; each pen measuring 1.5 m × 6 m.

Feed was offered *ad libitum* and delivered to individual fenceline feed bunks once daily. Weights of fresh feed were recorded daily. Orts were recorded every 28 d or as needed before the scheduled orts recording. Dry matter intakes were calculated from the as-fed deliveries using actual feedstuff DM values, less the amount of unconsumed DM.

Daily gain was calculated as pounds of gain on a shrunk basis. Feed efficiency was calculated as pounds of gain on a shrunk basis per pound of DM consumed.

Water was offered *ad libitum* provided by automatic water fountains shared between two pens.

The heavier initial weight blocks were slaughtered on d 89, and the lighter initial weight blocks were slaughtered on d 119. Final BW (gross BW × 0.96) were determined before cattle were transported to a commercial abattoir (Holcomb, KS). Incidence and severity of liver abscesses and HCW were recorded at harvest. Incidence and severity of liver abscesses were scored according to the scoring system (Brink et al., 1990): 0 = no abscesses, A^- = 1 or 2 small abscesses or abscess scars; A^0 = 2 to 4 small; well-organized abscesses; and A^+ = 1 or more large or active abscesses with or without adhesions. Carcass data were collected after a 24-h chill for the first shipment and after a 72-h chill for the second shipment. Boneless sections of the 6th through 12th ribs were collected from the left side of each carcass. Marbling scores, 12th rib fat thickness, KPH, LM area, USDA yield grades, and USDA quality grades were determined. Actual BW (shrunk) was used to determine dressing percentage.

Blood was collected from steers 18-h postfeeding on d 1 and every subsequent 28-d period. Blood was collected into heparinized vacuum tubes (BD, Franklin Lakes, NJ) and immediately placed onto ice before centrifuging at 3200 × g for 10 min to recover plasma. To analyze for plasma long chain FA (LCFA) composition, plasma was freeze dried (500 ul) and combined with 1 mL benzene containing internal standard (1000ug/mL methyl-C:13) and 4 mL BF_3-Methanol reagent (Supelco B1252). Tubes were incubated at 60°C for 60 min and then cooled to room temperature. Hexane (1 mL)

and ddH_20 (4 mL) were added and vortexed. Tubes were centrifuged at 1000 × g for 5 min. The organic solvent layer (1-2 mL) was then analyzed via gas chromatograph (Schimadzu model 17A**,** Palo Alto, CA) equipped with a Supelco SP-2560 capillary (100m × 0.25 mm × 0.20u film) using He as the carrier gas at a flow rate of 1.1 mL/min. Initial temperature was 140°C for 4 min. This was followed by an increase of 4°C/min to a final temperature of 240°C.

Boneless ribs were placed into multipurpose plastic bags at collection, transported to the Kansas State University Meats Laboratory, and refrigerated overnight at 0 ± 2°C. Rib sections were weighed and vacuum packaged in Nylon/PE Multivac bags **(**Ultravac Solutions, Kansas City, MO) using a Multivac vacuum packager (Multivac C500, Sepp Hagenmüller GnbH & Co, Germany). Vacuum was checked using a Kennedy Gauge (Kennedy Gauge, Kennedy Enterprises, Lincoln, NE) with an average vacuum reading of 0.7774 Bar. Vacuum packaged ribs were stored for an additional 16 d at 0 ± 2°C. Starting at the cranial end of the ribs, a steak (2.54 cm thick) was removed for analysis of long-chain fatty acid (LCFA) composition. Muscle LCFA profiles were analyzed following the procedures of Sukhija and Palmquist (1988). Briefly, 50 to 500 mg of samples were mixed with 2 mL internal standard in benzene and 3 mL methanolic-HCl before being flushed with nitrogen. Tubes were then capped and vortexed, heated for 2.25 h at 70ºC, and vortexed every 45 min during heating. Tubes were cooled to room temperature and mixed with 5 mL 6% K_2CO_3 and 2 mL benzene while being vortexed. Tubes were centrifuged at 500 × g for 5 min. The organic solvent layer was then analyzed using a Schimadzu gas chromatograph (model 17A**;** Schimadzu Corp., Palo Alto, CA) equipped with a Supelco SP-2560 capillary (100 m × 0.25 mm × 0.20 um; Supelco Inc., Bellefonte,

PA) using He as the carrier gas at a flow rate of 1.1 mL/min. Initial temperature was 140°C for 4 min, and was increased by 4°C/min to a final temperature of 240°C.

Experiment 3

Heifers were arranged in a randomized complete block experiment with a 2 × 2 factorial treatment arrangement to evaluate the effects of LSM and different management regimes on feedlot performance and carcass quality. Heifers (n = 366; initial BW = 374 ± 0.6 kg) were housed in 48 concrete-surfaced pens (36 m^2) containing 6 to 8 animals each. Pens provided overhead shade covering the fenceline bunk and half of each pen. Pens included automatic water fountains and 3.2 m of bunk space. Prior to this finishing trial, these heifers were in a growing trial consisting of three treatments. Heifers were blocked by the previous growing treatment and assigned randomly to 1 of 4 finishing diet treatments.

Experimental treatments included diets based on dry rolled corn with or without solvent-extracted LSM, and management regimes with or without an estradiol/trenbolone acetate (ET) implant and melengestrol acetate (MGA). This resulted in four treatments, a conventional program consisting of Rev IH and MGA with no added LSM (Con Conv), Rev IH and MGA with LSM (LSM Conv), a non-hormone treated cattle (NHTC) program consisting of no implant or MGA with no added LSM (Con NHTC), and a NHTC program with LSM (LSM NHTC). Composition of diets is summarized in Table 3.

On d 1, heifers were weighed and implanted (Revalor IH, 80 mg of trenbolone acetate, 8 mg of estradiol**;** Intervet Inc., Millsboro, DE) according to treatment. Diets (Table 1) were formulated to be isocaloric and isonitrogenous and were fed once daily

such that animals had *ad libitum* access. Access to water was provided *ad libitum* with automatic water fountains. Weights of fresh feed were recorded daily. Feed refusals were recorded every 28 d or when in excess. These amounts on a DM basis were subtracted from the total feed offered to determine actual feed intake for each pen of cattle.

Heifers were harvested across 2 d. Half of the weight blocks were harvested on d 119 and the other on d 120. Final BW (gross BW × 0.96) was determined before cattle were transported to a commercial abattoir in Holcomb, KS. Incidence and severity of liver abscesses and HCW were recorded the d of harvest. Incidence and severity of liver abscesses were scored according to the scoring system (Brink et al., 1990): 0 = no abscesses, A^- = 1 or 2 small abscesses or abscess scars; A^0 = 2 to 4 small, well-organized abscesses; and A^+ = 1 or more large or active abscesses with or without adhesions. Carcass data were collected after a 48-h chill. Marbling scores, 12th rib fat thickness, LM area, KPH, USDA yield grades, and USDA quality grades were determined. Dressing percentage was calculated as HCW divided by shrunk BW.

Dry matter intakes were calculated from the as-fed deliveries using actual feedstuff DM values, less the amount of unconsumed DM. Daily gain was calculated as kg of gain on a shrunk basis. Feed efficiency was calculated as kg of gain on a shrunk basis per kg of dry matter consumed.

Statistical Analyses

For all experiments, growth performance and carcass characteristics were analyzed using the MIXED procedure of SAS (version 9.0, SAS Inst. Inc., Cary, NC). Experimental unit was pen in experiments 1 and 3, and animal in experiment 2; diet and implant status served as fixed effects; and random effect was block in experiments 1 and

3, and animal in experiment 2. In experiments 1 and 3, USDA quality grade and liver abscesses were calculated and analyzed using the PROC GLIMMIX procedure of SAS (version 9.0, SAS Inst. Inc., Cary, NC).

In experiment 2, USDA quality grade and liver abscesses were calculated and analyzed using Monte Carlo's Chi Square analysis (Higgins, 2004) with animal as the experimental unit and the random effect. Ribs (n = 40; 10 per treatment) served as the experimental unit for muscle long-chain LCFA analyses. Plasma LCFA and muscle LCFA were analyzed using the MIXED procedure of SAS version 9.0, SAS Inst. Inc., Cary, NC). Animal was the experimental unit, diet and implant status were the fixed effects, and the random effect was animal for analysis of LCFA composition. The statistical analysis of plasma LCFA also included day, interactions between diet and day, and interaction between implant and day as fixed effects.

Across all studies, mean comparisons were determined following an *F*-test with $P \leq 0.05$. Means and differences were considered different at *P*-value ≤ 0.05, and *P*-value of ≤ 0.10 was considered as a tendency.

Results

Experiment 1

Performance and carcass quality results from experiment 1 are shown in Table 4. There was no effect ($P \geq 0.36$) from interaction between diet and implant or diet on performance, contrary to the hypothesis that flaxseed lignans would promote growth naturally. But as expected, the ET implant improved rate of gain ($P < 0.01$), feed intake ($P < 0.03$), and feed efficiency ($P < 0.01$).

There were an interaction between diet and implant status that neared significance for a carcass trait. The interaction between diet and implant status tended to affect ($P = 0.08$) KPH, so that control/implanted cattle had more KPH than flaxseed/implanted cattle, and control/no implant cattle had less KPH than flaxseed/no implant cattle. Diet affected some carcass traits. Feeding flaxseed decreased back fat thickness ($P < 0.01$) and USDA yield grade ($P = 0.01$) compared to cattle fed the control diet. Some carcass traits were affected by implant status. Carcass weights were improved ($P < 0.01$) when cattle were implanted, producing heavier carcasses by 9 kg or greater. Implanted cattle also produced more valuable carcasses, with increased LM area ($P < 0.01$) and decreased fat thickness ($P = 0.02$), resulting in lower USDA yield grade ($P = 0.01$).

Experiment 2

Performance and carcass characteristic results from experiment 2 are shown in Table 5. There was no effect ($P \geq 0.22$) of diet or the interaction between diet and implant status on finishing performance, yet implants improved DMI ($P = 0.02$), ADG ($P < 0.01$) and feed efficiency ($P < 0.01$).

Interactions between diet and implant status affected some carcass traits. The yield grades of the cattle fed flaxseed were greatly affected by the interaction of diet and implant status ($P = 0.03$), recording the extremes amongst treatments. When implanted, cattle fed flaxseed had the highest yield grade of all treatments, though this difference was only significant compared to flaxseed/no implant cattle. When cattle fed flaxseed were not implanted, they had the lowest yield grad of all treatments, though this difference was only significant compared to flaxseed/implant cattle. Why the yield grade of flaxseed changed greatly depending on implant status is not clear. A similar effect

from the interaction between diet and implant was observed for marbling scores ($P = 0.04$). Cattle fed flaxseed reported the extremes of the marbling scores, depending on implant status. Flaxseed/no implant cattle had the lowest marbling scores of all treatments, but being significantly decreased only to soy oil/no implant and flaxseed/implant cattle. Flaxseed/implant cattle had one of the greatest marbling scores amongst treatments; however were only significantly increased compared to soy oil/implant and flaxseed/no implant cattle. Implant status affect few carcass traits. Hot carcass weights increased ($P < 0.01$) and dressing percentage decreased ($P = 0.04$) when cattle were implanted. There was no effect of diet ($P \geq 0.11$) on carcass quality.

Results of fatty acid profiles are reported in Tables 6 and 7. With the exception of plasma C18:1n7, interactions between diet and implant status did not affect ($P > 0.05$) FA composition of plasma or LM tissues. Plasma total omega-3 increased ($P < 0.01$) when cattle were fed flaxseed. Subsequently, plasma n-6:n-3 fatty ratio also decreased ($P < 0.01$) when cattle were fed flaxseed. Plasma SFA decreased ($P < 0.01$) when cattle were fed flaxseed. Many of the changes in plasma FA profiles were reflected in concentrations of LM FA. Longissimus muscle total omega-3 fatty acids increased ($P < 0.01$) with flaxseed feeding, subsequently decreasing ($P < 0.01$) n-6:n-3.

Experiment 3

Finishing performance was not affected ($P \geq 0.12$) by the interaction between diet and exogenous hormones or the effect of diet, although exogenous hormones greatly affected performance. The use of anabolic steroids and estrous suppressants increased DMI ($P = 0.02$), ADG ($P < 0.01$), and improved feed efficiency ($P < 0.01$) in conventional cattle compared to NHTC. As in finishing performance, carcass quality was

not affected ($P \geq 0.10$) by the interaction of diet and exogenous hormones or the effect of diet. Conventional cattle had increased HCW ($P < 0.01$), but greater KPH ($P = 0.05$) than NHTC.

Discussion

Performance

Dunn et al. (2003) observed an interaction between ground flaxseed and implants ($P < 0.05$). Feeding LSM has been shown to interact with estradiol-17β in ovariectomized ewes, affecting estrogen-related jejunum growth and jejunum gene expression (O'Neil et al., 2009), and increasing liver weight and liver protein production with increased days fed LSM (O'Neil et al., 2006). From these results, we anticipated feeding flaxseed products to affect growth differently based on the exposure to exogenous hormones. In contrast, finishing performance results from the present three experiments indicate flaxseed products did not interact with the levels of exogenous hormones; however the interaction between diet and exogenous hormones neared tendency ($P = 0.12$) for ADG in experiment 3. Though the interaction effect was not significant for performance traits ($P \geq 0.12$), it is interesting to note numeric differences. The inclusion of exogenous hormones affected growth of cattle fed control diets more so than the growth of cattle fed flaxseed diets. This suggests feeding flaxseed products may replace some of the growth losses associated with the absence of exogenous hormones. Nevertheless, this effect is modest and only numeric. Feeding flaxseed products does not replace the growth effects of exogenous hormone entirely, but may promote modest growth improvements numerically.

There are differences in study designs between the previous and current experiments that may explain the differing results. O'Neil et al. (2006) and O'Neil et al. (2009) fed LSM and implanted exogenous hormones similar to experiment 3, but those authors studied the effects on digestive tissues of sheep rather than the effects on muscle tissues of cattle as in present study. Perhaps the different tissues and species altered the effect of flaxseed SDG between the studies.

In experiments 1 and 2 feeding ground flaxseed did not affect ($P \geq 0.32$) finishing performance, consistent with previous research. Concerning ground flaxseed, Maddock et al. (2006) and LaBrune et al. (2008) observed no effect on DMI, contrasting the findings of Drouillard et al. (2004), where ground flaxseed increased DMI. Studies feeding ground flaxseed at 5% diet DM (Drouillard et al., 2004) and 10% diet DM (LaBrune et al., 2008) also have reported no effect of flaxseed inclusion on ADG or G:F. In one instance ground flaxseed at an intermediate level of 8% diet DM has been shown to increase ADG and G:F (Maddock et al., 2006), but likely resulted from increased energy density in the flaxseed diet.

Feeding LSM did not affect ($P \geq 0.25$) finishing performance in experiment 3. Similarly, early studies have reported feeding LSM does not affect gain or efficiency when fed in confined beef systems (Matsushima et al., 1956; Webb et al., 1958) or grazing systems (Smith et al., 1958). Contrasting early studies and experiment 3, Kolari et al. (1960) combined data of 2 studies reporting that Hereford steers and heifers supplemented with LSM had 5% greater ADG compared to control cattle fed isonitrogenous diets, but reported no effects on DMI or feed efficiency. These early works demonstrate LSM supplementation generally does not affect performance in beef

systems. Recent dairy experiments show various levels of LSM supplementation does not affect ($P > 0.05$) DMI compared to isonitrogenous and isoenergetic control diets (Petit and Gagnon, 2009; Petit et al. 2009).

Experiments 1 and 2 using ET implants, and experiment 3 using ET implants with MGA found exogenous hormones to increase DMI ($P \geq 0.02$), ADG ($P < 0.01$), and feed efficiency ($P < 0.01$) when compared to cattle without exogenous hormones. This agrees with previous studies administering ET implants (Herschler et al. 1995) and ET implants with MGA (Kreikemeier and Mader, 2004).

Carcass Quality

Currently there are no known previous studies examining the effect of the interaction between flaxseed products and exogenous hormones on carcass characteristics. The interaction between diet and exogenous hormones affected KPH in experiment 1 ($P = 0.08$) and USDA yield grade in experiment 2 ($P = 0.03$), but exerted no effects ($P \geq 0.22$) on carcass quality in experiment 3. Though these interaction effects were not consistent amongst the three experiments, it is interesting to note that the interaction affected carcass traits relating to fat. Fukumitsu et al. (2008) demonstrated the SDG of flaxseed to affect fat metabolism in diet-induce obese rats. Perhaps the interaction of exogenous hormones would influence the effect of flaxseed SDG on fat in ruminants.

Feeding ground flaxseed decreased USDA yield grade ($P = 0.01$) and 12^{th} rib fat thickness ($P = 0.01$) in experiment 1, but did not affect ($P \geq 0.11$) carcass quality in experiment 2. Similarly, previous studies have reported feeding ground flaxseed does not affect LM area, 12^{th} rib fat thickness, or incidence of liver abcesses (Maddock et al.,

2006; LaBrune et al., 2008). As in experiments 1 and 2, LaBrune et al. (2008) reported HCW were not affected by ground flaxseed supplementation, but Maddock et al. (2006) had greater HCW in steers fed ground flaxseed than control steers.

In experiment 3 feeding LSM did not affect ($P \geq 0.10$) carcass characteristics, in agreement with results of Kolari et al. (1960). No other carcass characteristics were reported. The effects of LSM SDG on carcass traits have been studied more extensively in rat models. Flaxseed lignans have been shown to interact with estrogen receptors to promote skeletal muscle growth (Zhou et al., 2009) and decrease visceral fat in diet-induced obese rats (Fukumitsu et al. 2008). Because of these findings, we anticipated similar responses with increased skeletal muscle measured as LM area and decreased visceral fat measured as KPH; however, carcass traits were not affected by diet in experiment 3. The lack of effect is possibly due to the differences in myogenesis and lipogenesis between rats and cattle. In agreement with an early beef study (Kolari et al. 1960), but in contrast to recent rat experiments (Fukumitsu et al. 2008), feeding LSM did not affect LM area, KPH, or other carcass characteristics in experiment 3. The inclusion of flaxseed products affected few carcass traits and results were not consistent amongst the experiments. Overall, results suggest flaxseed products do not affect carcass quality.

Exogenous hormones consistently increased ($P < 0.01$) HCW across all three experiments. This agrees with previous research studying the effects of ET implants (Herschler et al., 1995) and ET implants with MGA (Kreikemeier and Mader, 2004). In spite of consistent increases in HCW, other carcass traits were variably affected by exogenous hormones in the present studies. Both experiments 1 and 2 used implants with similar potency, yet only experiment 1 had increased LM area ($P < 0.01$), decreased 12th

rib fat thickness (P = 0.01), and lower USDA yield grade (P = 0.01); whereas only experiment 2 had decreased dressing percentage (P = 0.04). This contrasts the results of Herschler et al. (1995) where two experiments studying similar ET implants found HCW was the only carcass trait affected by implant status. The NHTC of experiment 3 had decreased HCW (P < 0.01) and KPH (P = 0.05) compared to conventionally managed cattle receiving ET implants and MGA. Similarly, Kreikemeier and Mader (2004) reported ET implants administered with MGA to increase HCW; however no other carcass traits were affected, including KPH. The increases in KPH and BF were not anticipated as earlier authors have reported no effect of implants and MGA on KPH or BF in heifers (Kreikemeier and Mader, 2000; Schneider et al., 2007); however, Wagner et al. (2007) reported that use of MGA increases carcass fatness regardless of implant type used. Though KPH and BF increased, these changes were not great enough to affect USDA yield grade (P = 0.91) in the present study.

Feeding flaxseed increased (P < 0.01) α-linolenic acid and total omega-3 fatty acid, and decreased (P < 0.01) the n-6:n-3 fatty acid ratio in plasma and LM when cattle were fed flaxseed, agreeing with previous research (Drouillard et al. 2002; Drouillard et al., 2004; Good, 2004; Maddock et al., 2006; Kronberg et al., 2006; LaBrune et al., 2008). Experiment 2 is the first study to report implants do interact (P > 0.05) with flaxseed to alter plasma or LM FA composition.

The interaction between flaxseed products and exogenous hormones did not significantly affect finishing performance, though numeric differences were noted. Contrasting carcass results between experiments warrant further investigation of the interaction between flaxseed products and exogenous hormones. Feeding flaxseed may

be fed without negatively impacting performance or carcass quality. Using exogenous hormones like that found in conventional regimes improved DMI, ADG, feed efficiency, and HCW when compared to NHTC regimes. Contrary to the hypothesis, flaxseed SDG did not act as a natural growth promoter. However, ground flaxseed did increase desirable FA in saleable meat without compromising feedlot performance.

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Table 1. Composition of experiment 1 diets reported on a DM basis.

Item, % of DM	Control	Flaxseed
Steam-flaked corn	74.25	69.72
Ground alfalfa hay	8.00	8.00
Fancy bleachable tallow	3.82	-
Ground flaxseed	-	10.00
Corn steep liquor	7.50	7.50
Soybean meal	1.19	0.23
Urea	1.00	0.45
Limestone	1.46	1.40
Potassium chloride	0.15	0.07
Salt	0.30	0.30
Vitamin and mineral premix[a]	0.12	0.12
Feed additive premix[b]	2.21	2.21
Crude protein, %	14.00	14.00
Crude fat, %	7.30	7.30

[a]Provided (DM basis) 2650 IU/kg vitamin A, 22 IU/kg vitamin E, 0.1 ppm Co, 10 ppm Cu, 0.5 ppm I, 60 ppm Mn, 0.25 ppm Se, 60 ppm Zn.
[b]Provided 300 mg monensin and 90 mg tylosin **(**Elanco Animal Health, Greenfield, IN**)** per hd/d.

Table 2. Composition of experiment 2 diets reported on a DM basis.[¥]

Item, % of DM	Control	Flaxseed
Ingredients		
Dry rolled corn	70.87	68.81
Corn silage	9.99	10.00
Ground flaxseed	-	10.00
Corn steep liquor	3.00	3.00
Molasses	3.00	3.00
Soybean meal	4.05	-
RT premix	1.96	1.96
Limestone	1.67	1.64
Urea, 46% N	1.01	1.01
Salt	0.30	0.30
Vitamin premix	0.22	0.22
Trace mineral mix	0.06	0.06
Nutrients, %		
DM	73.19	74.60
CP	12.67	12.91
P	0.46	0.48
Ca	0.72	0.74
Ether extract	7.61	6.35
NDF	10.43	11.23

[¥]Formulated to provide provide 300 mg/d monensin, 90 mg/d tylosin (Elanco Animal Health, Greenfield, IN), 0.3% salt, 2650 IU vitamin A, 22 IU vitamin E, 0.10 mg Co, 10 mg Cu, 0.5 mg I, 0.25 mg Se, 50 mg Mn, and 50 mg Zn per kg diet DM.

Table 3. Composition of experiment 3 diets on a DM basis.[¥]

Item, % of DM	Control	Linseed Meal
Ingredients		
Dry rolled corn	54.3	49.7
Corn gluten	30.0	30.0
Corn silage	10.0	10.0
Linseed meal	-	5.0
Feed additive[a]	2.16	2.16
Limestone	1.69	1.69
Potassium chloride	1.08	1.08
Urea, 46% N	0.36	0.36
Salt	0.30	0.30
Vitamin premix	0.06	0.06
Trace mineral mix	0.06	0.06
Nutrients, %		
DM	70.65	70.67
CP	14.04	14.20
P	0.48	0.51
Ca	0.58	0.55
Ether extract	3.59	3.42
NDF	24.20	25.01

[¥]Formulated to provide 0.3% salt; 2650 IU vitamin A; 22 IU vitamin E; 0.09 mg Co; 10 mg Cu; 0.5 mg I; 0.25 mg Se; 5 mg Mn; and 5 mg Zn per kg diet DM.
[a]Provided 300 mg monensin (Elanco Animal Health; Greenfield, IN), 90 mg tylosin, and 0 or 0.5 mg melengestrol acetate (Pfizer Animal Health; New York, NY) in a ground corn carrier.

Table 4. Experiment 1 effect of diet and implant status on performance and carcass characteristics.*

	Control		Flaxseed			*P*-values[§]		
Item	**Implant**	**No Implant**	**Implant**	**No Implant**	**SEM**	**Diet**	**Implant**	**DxI[¥]**
Initial BW, kg	396.0	397.8	394.2	396.0	4.4	0.68	0.65	0.97
DMI, kg	8.98	8.68	8.70	8.42	0.15	0.59	0.03	0.60
ADG, kg	1.65	1.39	1.66	1.34	0.05	0.64	<0.01	0.57
G:F	0.186	0.163	0.192	0.162	0.008	0.55	<0.01	0.36
HCW, kg	327.9	318.9	325.7	313.0	3.9	0.29	<0.01	0.68
LM area, cm^2	81.3	76.8	81.3	76.8	1.10	0.83	<0.01	0.93
Fat thickness, cm	1.10	1.20	1.00	1.10	0.008	<0.01	0.02	0.88
KPH, %	2.15	2.13	2.06	2.22	0.05	0.94	0.17	0.08
Choice & prime, %	65.6	63.9	65.1	63.9	6.2	0.97	0.82	0.97
Select, %	31.1	32.8	31.7	36.1	6.1	0.75	0.62	0.82
Standard, %	3.3	1.6	3.1	0.0	1.8	0.63	0.18	0.67
USDA yield grade	2.66	2.73	2.30	2.66	0.088	0.01	0.01	0.12

[*]Implant = cattle implanted with Revalor S (Intervet Inc., Millsboro, DE)
[§]Effects of diet, implant status, interaction between diet and implant status *P*-values protected by an overall *F*-test ≤ 0.05
[¥]DxI = interaction between diet and implant status

Table 5. Experiment 2 effect of diet and implant status on performance and carcass characteristics.[†]

	Control		Flaxseed			*P*-value[§]		
Item	**Implant**	**No Implant**	**Implant**	**No Implant**	**SEM**	**Diet**	**Implant**	**D×I[*]**
Initial BW, kg	498.2	498.5	497.7	496.9	15.2	0.69	0.94	0.83
DMI, kg/d	12.63	11.62	12.53	12.50	0.54	0.32	0.02	0.22
ADG, kg/d	1.16	0.83	1.12	0.90	0.09	0.87	<0.01	0.45
G:F	0.091	0.071	0.088	0.071	0.005	0.81	<0.01	0.74
HCW, kg	390	360	383	359	9.4	0.49	<0.01	0.60
LM area, cm^2	79.42	73.37	72.00	74.94	3.30	0.33	0.61	0.14
Backfat, cm	0.54	0.46	0.45	0.47	0.06	0.53	0.60	0.40
KPH, %	2.78	2.68	2.70	2.75	0.14	0.99	0.81	0.54
Dressing percent	63.0	61.5	62.0	60.8	0.6	0.17	0.04	0.84
USDA yield grade	2.63[ab]	2.80[ab]	3.00[a]	2.50[b]	0.15	0.79	0.26	0.03
Marbling score[‡]	546[ab]	593[a]	563[a]	476[b]	38	0.11	0.52	0.04
Select, %[¥]	0.0	0.0	0.0	10.0	-	1.00	1.00	1.00
Choice, %[¥]	75.00	70.00	88.89	80.00	-	0.44	0.71	0.87
Prime, %[¥]	25.00	30.00	11.11	10.00	-	0.24	1.00	0.66

[†]Implant = implanted with Revalor XS (Intervet Inc., Millsboro, DE)

[§] Effects of diet, implant status, interaction between diet and implant status *P*-values protected by an overall *F*-test ≤ 0.05

[*]D×I = interaction between diet and implant status

[‡]300 to 399 = Slight, 400 to 499 = Small, 500 to 599 = Modest, 600 to 699 = Moderate

[¥]Monte Carlo Exact Fit Chi Square Analysis (Higgins, 2004)

[a,b,c]Means within a row without common superscripts are different ($P \leq 0.05$)

Table 6. Experiment 2 effect of diet and implant status on plasma concentrations of fatty acids reported on an as-is basis.[§]

Fatty Acid, mg/L[†]	d 0				d 28				d 56				d 84					
	Control		Flaxseed		Control		Flaxseed		Control		Flaxseed		Control		Flaxseed			
	Imp	No Imp	Imp	No Imp	Imp	No Imp	Imp	No Imp	Imp	No Imp	Imp	No Imp	Imp	No Imp	Imp	No Imp	SEM	*P*-values*
C12:0	1.73	1.76	3.21	2.11	1.31	1.75	2.13	2.30	1.80	2.16	1.43	1.03	1.22	2.45	1.67	2.60	0.66	
C14:0	2.39	1.85	1.30	0.84	2.27	1.62	1.89	1.45	1.92	5.80	3.85	3.86	1.40	1.29	2.52	1.40	0.96	T
C14:1	2.35	1.74	2.54	3.31	2.28	3.93	1.64	2.48	2.74	2.31	3.34	5.29	1.80	2.70	2.36	4.92	0.93	I
C16:0	153.6	140.7	134.4	148.8	162.7	154.9	131.3	130.4	139.4	143.5	132.5	132.6	140.0	129.2	117.5	123.5	7.87	D, T
C16:1	21.07	23.75	20.48	23.49	12.64	11.02	19.32	20.75	4.16	11.58	13.70	17.50	3.43	2.30	2.53	5.50	2.33	D, T, X
C17:0	10.03	5.58	10.56	7.94	4.71	5.23	5.54	3.00	1.98	2.60	1.24	1.72	3.07	9.44	9.16	7.68	1.76	T
C17:1	5.43	5.56	5.89	4.31	3.64	5.27	2.68	1.91	3.29	3.16	2.45	1.82	3.50	4.13	3.64	2.40	0.90	D, T
C18:0	235.6	238.6	218.5	234.9	295.6	271.7	242.2	218.8	241.6	245.2	240.2	227.2	248.0	217.2	195.6	220.3	16.2	D, T, X
C18:n1t9	26.28	26.44	24.86	24.42	31.49	19.08	14.78	13.47	19.36	16.62	13.99	11.30	13.94	12.17	10.45	11.09	2.95	D, T
C18:n1c9	96.98	93.91	85.01	87.75	86.83	81.19	88.89	84.96	84.71	85.38	96.35	86.80	68.90	68.69	83.59	78.76	6.39	T
C18:1n7	12.98	10.23	9.79	12.01	13.53	7.71	8.97	9.90	9.87	6.55	6.96	5.45	4.92	2.58	2.68	3.22	1.78	T, W
CLA c9, t11	3.16	2.75	0.95	0.87	2.05	1.33	0.98	0.72	0.89	1.69	2.58	1.15	2.25	2.26	1.86	1.53	0.63	D, X
CLA t10, c12	3.14	1.73	1.73	2.26	0.90	1.18	1.84	2.49	0.90	1.10	0.93	1.54	1.38	1.68	0.64	0.50	0.71	T
C18:2nt6	4.37	4.34	3.58	4.43	4.88	5.05	4.93	3.49	1.25	2.14	1.46	1.64	1.83	2.42	1.78	1.77	1.13	T
C18:2nc6	595.4	579.3	579.9	584.7	906.1	823.9	654.9	693.9	745.3	743.2	692.7	660.5	703.2	641.5	604.8	649.6	47.5	D, T, X
C18:3n3	24.00	20.04	13.77	24.15	27.97	42.68	135.12	133.27	18.66	22.05	168.73	151.26	15.53	14.68	129.54	150.30	9.22	D, T, X
C18:3n6	19.77	16.08	21.62	22.87	13.57	21.33	11.32	13.21	20.45	16.01	10.63	11.31	11.52	14.33	8.83	8.84	2.90	T, X
C20:0	2.76	1.25	1.91	1.62	2.54	2.62	1.42	1.46	1.73	1.33	2.47	2.02	2.56	2.46	1.96	2.06	0.64	
C20:1	2.55	1.43	2.42	2.90	1.43	2.22	1.37	1.31	1.39	0.89	1.47	1.62	1.05	1.18	2.59	3.08	0.68	
C20:2	7.47	5.12	5.47	5.60	2.33	3.23	2.41	4.40	2.91	3.14	4.17	5.67	3.36	3.81	4.32	2.98	0.87	T
C20:3n6	42.94	41.03	38.59	36.05	31.29	31.76	18.72	24.26	33.87	37.14	15.02	16.77	26.88	26.89	18.25	14.48	3.40	D, T, X
C20:4n6	0.99	0.53	0.25	1.09	0.40	0.53	0.74	1.91	0.19	1.09	0.99	0.88	0.96	2.52	3.26	0.83	0.60	T, Z
C20:5n3	5.16	5.66	8.62	7.12	6.58	7.31	12.74	12.72	4.77	4.37	12.64	12.59	5.24	7.65	12.14	16.52	1.48	D, T, X
C21:0	3.37	2.58	1.63	1.73	1.88	0.40	0.55	0.69	1.57	2.49	1.36	2.24	1.22	1.38	1.45	0.75	0.63	T
C22:0	3.71	3.96	2.68	2.48	2.07	1.32	1.55	2.21	2.30	1.82	2.88	1.95	2.69	3.45	1.45	1.13	0.78	
C22:5n3	2.95	2.69	2.12	3.95	3.66	2.63	1.23	1.81	1.22	2.53	2.27	2.36	3.33	2.71	4.22	3.01	0.81	
C22:6n3	6.50	5.32	6.22	5.81	3.72	3.86	4.05	4.89	4.18	2.18	2.81	1.73	2.00	2.00	3.24	2.49	0.92	T
C24:0	1.46	3.70	3.91	3.12	3.01	2.43	1.76	4.41	2.05	2.44	1.69	2.26	1.72	4.22	1.95	3.12	0.89	I
C24:1	2.49	1.48	1.63	3.20	1.72	1.79	1.46	1.77	1.35	1.19	1.03	1.86	2.03	2.72	2.38	1.46	0.66	
Total	1293	1241	1208	1258	1630	1513	1372	1393	1347	1360	1431	1362	1270	1180	1228	1318	78	T, X

[§]Imp = implanted with Revalor XS (Intervet Inc., Millsboro, DE); No Imp = not implanted.

*Denotes significance at $P < 0.05$. Effects represented as D = diet; I = implant status; T = sample day; W = two-way interaction between diet and implant status; X = two-way interaction between diet and sample day; Y = two-way interaction between implant status and sample day; Z = three-way interaction between diet, sampling day and implant status.

[a,b,c]Means within a row without common superscripts are different ($P \leq 0.05$).

[†]Number after C denotes number of Carbons; n denotes the double bond location from the omega position; c and t denote the cis or trans configuration.

Table 7. Experiment 2 effect of diet and implant status on longissimus muscle concentrations of fatty acids reported on an as-is basis.[†]

Fatty acid, mg/kg[¥]	Control		Flaxseed			*P*-value[§]		
	Implant	No Implant	Implant	No Implant	SEM	Diet	Implant	D×I[‡]
C14:0	0.271	0.291	0.224	0.219	0.031	0.05	0.81	0.67
C14:1	0.046	0.048	0.040	0.045	0.013	0.70	0.78	0.89
C16:0	2.04	2.27	1.84	1.84	0.19	0.09	0.54	0.52
C16:1	0.323	0.258	0.266	0.273	0.038	0.56	0.43	0.32
C17:0	0.093	0.086	0.096	0.095	0.006	0.29	0.53	0.60
C17:1	0.068	0.076	0.064	0.070	0.008	0.55	0.36	0.85
C18:0	1.11	1.34	1.11	1.12	0.110	0.29	0.26	0.28
C18:n1t9	0.269	0.313	0.181	0.200	0.027	<0.01	0.22	0.62
C18:n1c9	2.77	3.26	2.69	2.86	0.27	0.36	0.20	0.53
C18:1n7	0.135	0.167	0.130	0.142	0.014	0.26	0.10	0.45
CLA c9, t11	0.0034	0.0026	0.0003	0.0016	0.0007	<0.01	0.72	0.15
CLA t10, c12	0.0006	0.0008	0.0002	0.0009	0.0005	0.73	0.33	0.56
C18:2nt6	0.014	0.015	0.026	0.026	0.002	<0.01	0.71	0.86
C18:2nc6	0.335	0.340	0.278	0.300	0.025	0.04	0.56	0.71
C18:3n3	0.015	0.017	0.073	0.077	0.005	<0.01	0.61	0.84
C18:3n6	0.003	0.004	0.003	0.003	0.001	0.39	0.40	0.55
C20:0	0.005	0.006	0.009	0.010	0.001	<0.01	0.15	0.83
C20:1	0.018	0.018	0.016	0.018	0.002	0.48	0.51	0.65
C20:2	0.006	0.006	0.005	0.007	0.001	0.70	0.40	0.40
C20:3n6	0.012	0.015	0.011	0.008	0.002	0.07	0.87	0.12
C20:4n6	0.001	0.001	0.003	0.003	0.0003	<0.01	0.70	0.24
C20:5n3	0.002	0.002	0.003	0.003	0.0008	0.16	0.34	0.86
C21:0	0.021	0.021	0.014	0.019	0.004	0.22	0.52	0.59
C22:0	0.003	0.003	0.004	0.004	0.001	0.38	0.97	0.99
C22:5n3	0.003	0.002	0.002	0.002	0.0006	0.21	0.57	0.12
C22:6n3	0.003	0.005	0.006	0.006	0.002	0.29	0.73	0.60
C24:0	0.0030	0.0015	0.0026	0.0021	0.0003	0.79	<0.01	0.08
C24:1	0.0009	0.0011	0.0011	0.0011	0.0004	0.73	0.75	0.73
Total	7.58	8.58	7.11	7.36	0.67	0.19	0.33	0.55
omega-3[d]	0.023	0.026	0.084	0.089	0.006	<0.01	0.49	0.84
omega-6[e]	0.371	0.378	0.319	0.342	0.026	0.05	0.47	0.72
n-6:n-3[f]	17.69	16.80	3.97	3.93	1.50	<0.01	0.74	0.76
SFA[g]	3.55	4.02	3.30	3.30	0.33	0.13	0.45	0.44
MUFA[h]	3.62	4.14	3.39	3.61	0.33	0.23	0.24	0.64
PUFA[i]	0.398	0.411	0.409	0.438	0.032	0.52	0.50	0.79

[†]Imp = implanted with Revalor XS (Intervet Inc., Millsboro, DE); No Imp = not implanted.
[§] Effect of diet *P*-value protected by an overall F-test ≤ 0.05.
[¥]Number after C = number of Carbons; n = double bond location from the omega position; c and t = the cis or trans configuration.
[‡]D×I = interaction between diet and implant.
[a,b,c]Means within a row without common superscripts are different (P ≤ 0.05).
[d]omega-3 = C18:3n3 + C20:5n3 + C22:5n3 + C22:6n3
[e]omega-6 = C18:2nt6 + C18:2nc6 + CLAc9t11 + CLAt10c12 + C18:3n6 + C20:3n6 + C20:4n6
[f]n-6:n-3 = omega-6 / omega-3
[g]SFA = C14:0 + C16:0 + C17:0 + C18:0 + C20:0 + C21:0 + C22:0 + C24:0
[h]MUFA = C14:1 + C16:1 + C17:1 + C18:1nt9 + C18:1n11 + C18:1nc9 + C18:1n7 + C20:1 + C24:1
[i]PUFA = C18:2n6t + C18:2n6c + CLAc9t11 + CLAt10c12 + C18:3n6 + C18:3n3 + C20:2 + C20:3n6 + C20:4n6 + C20:5n3 + C22:5n3 + C22:6n3

Table 8. Experiment 3 effects of diet and feeding regime on performance and carcass characteristics.*

	Control		Linseed meal			*P*-values[§]		
Item	**Conventional**	**NHTC**	**Conventional**	**NHTC**	**SEM**	**Diet**	**Regime**	**D×R[¥]**
Initial BW, kg	372.5	376.7	370.9	376.5	4.4	0.84	0.27	0.87
DMI, kg	9.99	9.55	10.05	9.83	0.15	0.25	0.02	0.45
ADG, kg	1.29	1.09	1.23	1.12	0.03	0.66	<0.01	0.12
G:F	0.129	0.114	0.123	0.114	0.002	0.20	<0.01	0.24
HCW, kg	323.8	311.2	321.9	314.0	3.4	0.89	<0.01	0.49
LM area, cm^2	81.3	79.4	82.0	80.2	1.13	0.53	0.11	0.95
Fat thickness, cm	1.64	1.49	1.61	1.52	0.06	0.98	0.04	0.67
KPH, %	2.36	2.32	2.44	2.31	0.04	0.36	0.05	0.33
Dressing percent	61.51	61.42	62.09	61.56	0.002	0.10	0.16	0.30
Average YG	2.78	2.63	2.65	2.78	0.11	0.94	0.91	0.22
Liver abcesses, %	15.76	16.00	13.05	16.52	3.86	0.77	0.62	0.67
Marbling score[‡]	474	488	482	483	9.87	0.85	0.42	0.52
Select, %	17.86	15.08	16.52	16.30	4.00	0.99	0.74	0.70
Choice, %	77.90	75.77	77.28	76.92	4.54	0.95	0.78	0.84
Prime, %	4.24	9.15	6.56	6.42	2.65	0.94	0.33	0.36

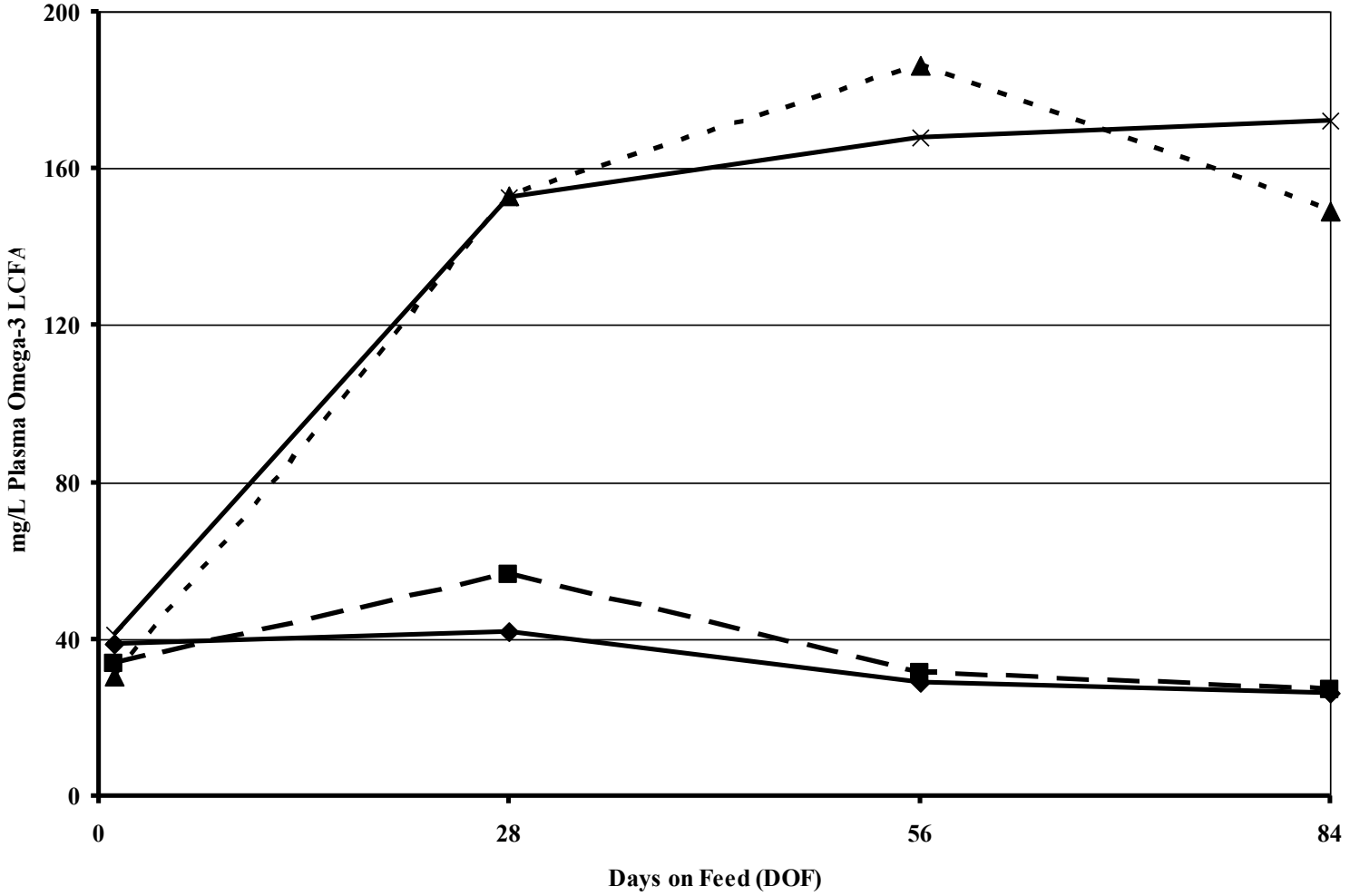
[*]NHTC = non-hormone treated cattle, Conventional = implanted with Revalor IH (Intervet Inc., Millsboro, DE) and fed melengestrol acetate (Pfizer Animal Health; New York, NY).

[§]Effects of diet, management regime, and the interaction between diet and management regime *P*-values protected by an overall *F*-test ≤ 0.05

[¥]D×R represents the interaction between diet and management regime

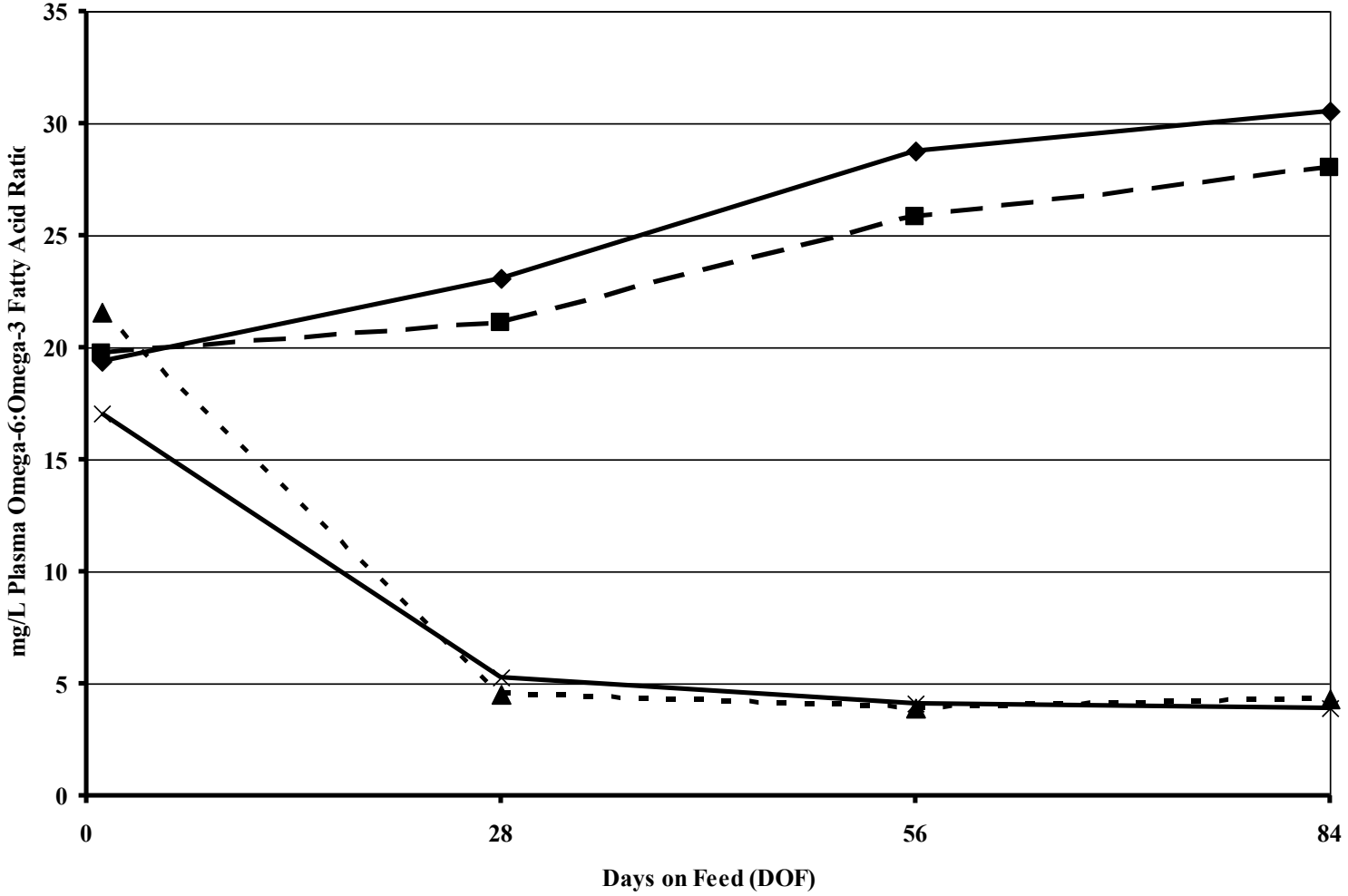
[‡]300 to 399 = Slight, 400 to 499 = Small, 500 to 599 = Modest, 600 to 699 = Moderate

A.



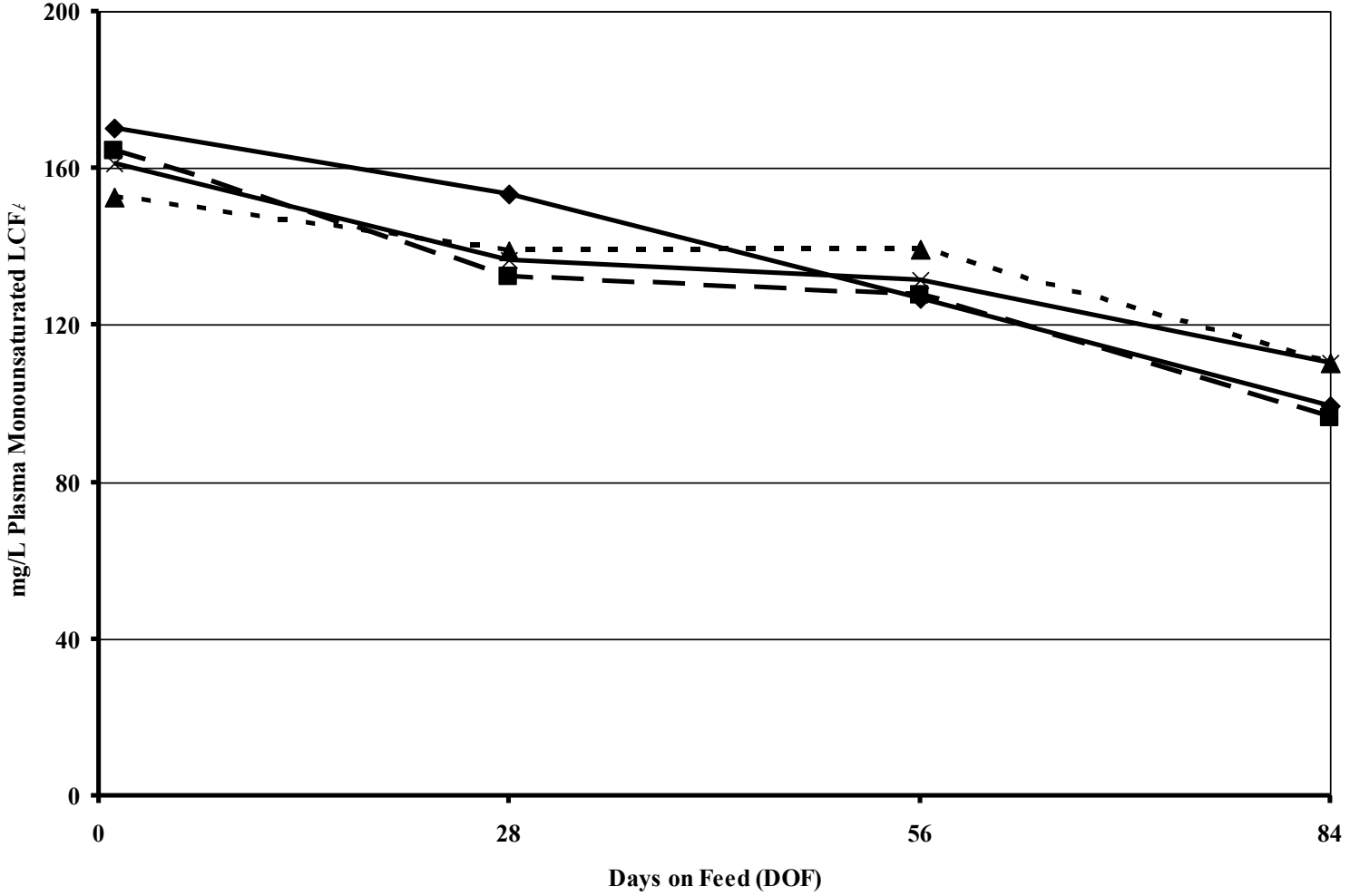
SEM = 9.64 Diet×implant×DOF, P = 0.17 Diet×implant, P = 0.97 Diet×DOF, P < 0.01
Implant×DOF, P = 0.36 Diet, P < 0.01 Implant, P = 0.58 DOF, P < 0.01

B.



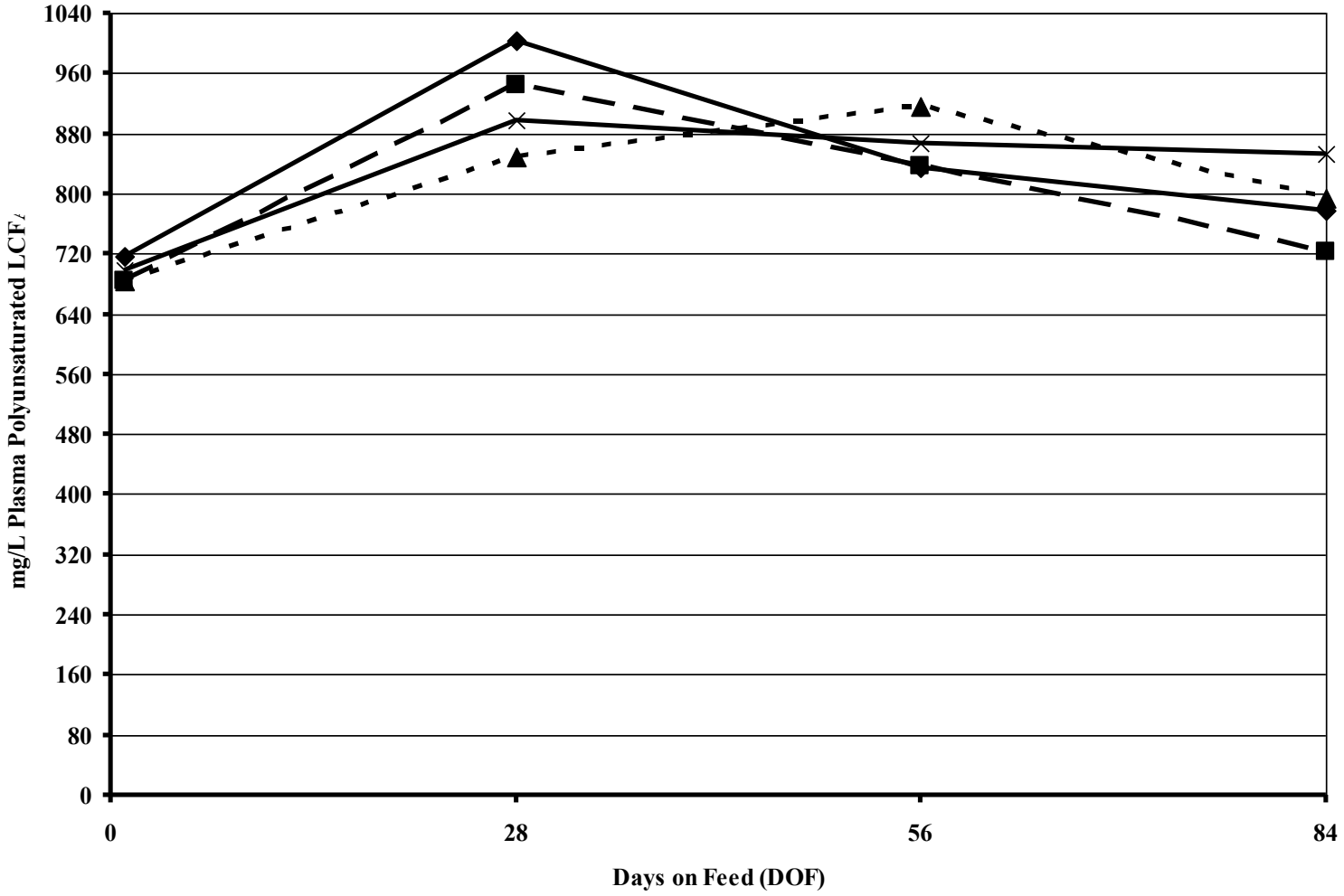
SEM = 1.7 Diet×implant×DOF, P = 0.25 Diet×implant, P = 0.64 Diet×DOF, P < 0.01
Implant×DOF, P = 0.93 Diet, P < 0.01 Implant, P = 0.10 DOF, P < 0.01

C.



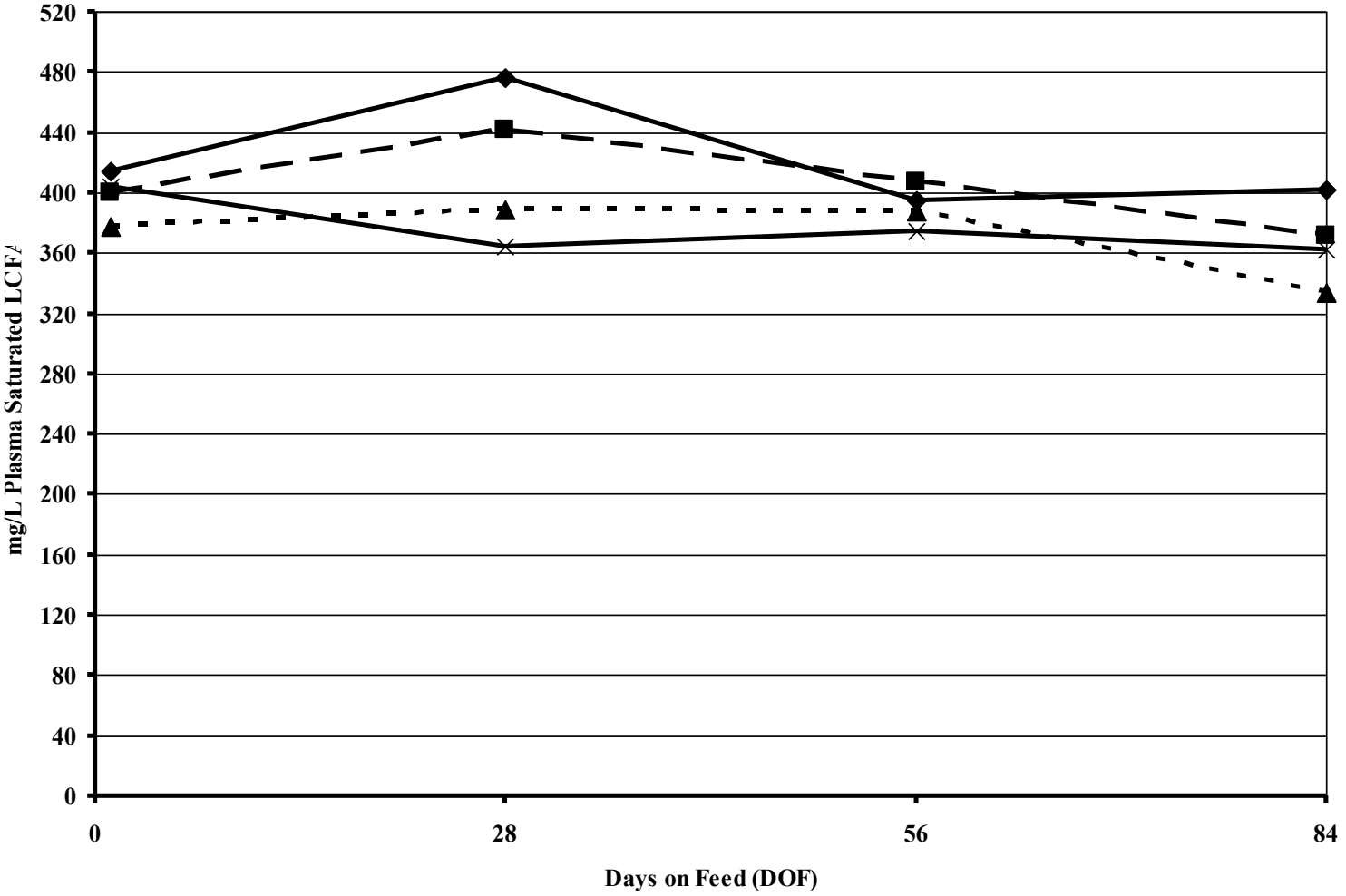
SEM = 10.0 Diet×implant×DOF, P = 0.71 Diet×implant, P = 0.56 Diet×DOF, P = 0.23
Implant×DOF, P = 0.74 Diet, P = 0.83 Implant, P = 0.52 DOF, P < 0.01

D.



SEM = 54.8 Diet×implant×DOF, P = 0.38 Diet×implant, P = 0.52 Diet×DOF, P < 0.01
Implant×DOF, P = 0.97 Diet, P = 0.92 Implant, P = 0.85 DOF, P < 0.01

E.



SEM = 23.2 Diet×implant×DOF, P = 0.42 Diet×implant, P = 0.49 Diet×DOF, P = 0.06
Implant×DOF, P = 0.58 Diet, P = 0.01 Implant, P = 0.70 DOF, P < 0.01

Figure 1. Effect of diet, implant, day, and the interactions between the factors on plasma concentrations of fatty acids reported on an as-is basis.

Control Implant —♦—; Control No Implant — ■ —; Flaxseed Implant - ▲ -; Flaxseed No Implant —×—

A. Effect of Diet and Day on Plasma Concentrations of Omega-3 Fatty Acids
B. Effect of Diet and Day on Plasma n-6:n-3 Fatty Acid Ratio
C. Effect of Diet and Day on Plasma Concentrations of Monounsaturated Fatty Acids
D. Effect of Diet and Day on Plasma Concentrations of Polyunsaturated Fatty Acids
E. Effect of Diet and Day on Plasma Concentrations of Saturated Fatty Acids