

INFORMATION TO USERS

This manuscript has been reproduced from the microfilm master. UMI films the text directly from the original or copy submitted. Thus, some thesis and dissertation copies are in typewriter face, while others may be from any type of computer printer.

The quality of this reproduction is dependent upon the quality of the copy submitted. Broken or indistinct print, colored or poor quality illustrations and photographs, print bleedthrough, substandard margins, and improper alignment can adversely affect reproduction.

In the unlikely event that the author did not send UMI a complete manuscript and there are missing pages, these will be noted. Also, if unauthorized copyright material had to be removed, a note will indicate the deletion.

Oversize materials (e.g., maps, drawings, charts) are reproduced by sectioning the original, beginning at the upper left-hand corner and continuing from left to right in equal sections with small overlaps.

Photographs included in the original manuscript have been reproduced xerographically in this copy. Higher quality 6" x 9" black and white photographic prints are available for any photographs or illustrations appearing in this copy for an additional charge. Contact UMI directly to order.

**Bell & Howell Information and Learning
300 North Zeeb Road, Ann Arbor, MI 48106-1346 USA
800-521-0600**

UMI[®]

**Effects of conjugated linoleic acid (CLA) on growth and carcass
characteristics when fed for differing amounts of time and when fed with high
oil corn (HOC) to growing-finishing swine**

by

Joseph Christopher Sparks

**A dissertation submitted to the graduate college
in partial fulfillment of the requirements for a degree of
DOCTOR OF PHILOSOPHY**

Major: Animal Nutrition

Major Professor: D. R. Zimmerman

Iowa State University

Ames, Iowa

2000

UMI Number: 9962848

UMI[®]

UMI Microform 9962848

Copyright 2000 by Bell & Howell Information and Learning Company.

**All rights reserved. This microform edition is protected against
unauthorized copying under Title 17, United States Code.**

**Bell & Howell Information and Learning Company
300 North Zeeb Road
P.O. Box 1346
Ann Arbor, MI 48106-1346**

Graduate College
Iowa State University

This is to certify that the Doctoral dissertation of

Joseph Christopher Sparks

has met the dissertation requirement of Iowa State University

Signature was redacted for privacy.

Major Professor

Signature was redacted for privacy.

For the Major Program

Signature was redacted for privacy.

For the Graduate College

TABLE OF CONTENTS

CHAPTER 1: GENERAL INTRODUCTION.....	1
Dissertation Organization	2
CHAPTER 2: LITERATURE REVIEW	3
Conjugated Linoleic Acid (CLA) Chemistry and History	3
Conjugated linoleic acid in foods	4
CLA in dairy products	7
CLA in beef products.....	8
CLA and cancer prevention	9
CLA in animal performance and body composition.....	11
Mechanisms of CLA.....	15
Responses To Energy Intake In The Growing Pig	18
Effects of added fat.....	19
Effect of fat on carcass quality	21
Literature Cited	26
CHAPTER 3: PERFORMANCE, COMPOSITION AND QUALITY CHARACTERISTICS OF FINISHING BARROWS SUPPLEMENTED WITH CONJUGATED LINOLEIC ACID.....	33
Introduction.....	34
Materials and Methods	35
Animals.....	35
Meat Laboratory Procedures and Analysis.....	36
Statistical analysis.....	39
Results and Discussion	39
Implications	43
Literature Cited.....	44
CHAPTER 4: EFFECTS OF CONJUGATED LINOLEIC ACID AND HIGH OIL CORN ON GROWTH PERFORMANCE AND PORK QUALITY IN FINISHING PIGS	51
Introduction.....	52
Materials and Methods	53
Animals.....	53
Carcass measurements	54
Meat Laboratory Procedures and Analysis.....	55
Fatty Acid Profile	56
Statistical analysis.....	58
Results.....	58
Growth performance data	58
Ultrasound data.....	59

Carcass characteristics	59
Discussion	62
Implications	63
Literature Cited	63
CHAPTER 5: GENERAL SUMMARY	72
Literature Cited	75

CHAPTER 1: GENERAL INTRODUCTION

A large amount of research has been done on the health effects of conjugated linoleic acid (CLA). The research has shown that CLA can be beneficial toward prevention and curing some diseases such as cancer and atherosclerosis. In growing animals, feeding CLA increases percent lean and decreases fat in the body. Most of this work, until recently, has been in mice and rats. The research conducted for this dissertation was designed to determine the applicability of CLA into swine production by determining the amount of time or weight gain in finishing pigs that was necessary to achieve optimal results from including CLA in swine diets, and to determine if CLA has beneficial effects on carcasses of swine fed diets that contain high oil corn (HOC) that is high of unsaturated fats. HOC has been shown to increase unsaturated fatty acids which has detrimental effects on fat quality of the carcass. Unsaturated fats lower the melting point causing bellies to be soft and raise Iodine Values increasing the oxidation potential and potentially reducing the shelf life of the pork products. Animals fed CLA have been shown to have increased saturated fat which is the opposite of animals fed HOC, therefore CLA should correct quality problems that occur from feeding HOC.

This research is important to the swine industry. First, research needs to be carried out to confirm growth performance and secondly to evaluate effects of CLA and HOC and in combination on pork quality. Also, with the inevitable loss of antibiotics as a growth promotant, natural occurring growth promotants like CLA will need to be studied and further understood.

Dissertation Organization

This dissertation contains two papers that have been prepared in the style appropriate for submission to the *Journal of Animal Science*. The papers are preceded by a literature review and followed by a general summary. The first paper includes dissertation research by J. C. Sparks and B. R. Wiegand. J. C. Sparks was responsible for the growth performance, carcass composition, and fatty acid profile measurements. B. R. Wiegand was responsible for the pork quality and sensory evaluation of loin chops.

CHAPTER 2: LITERATURE REVIEW

Conjugated Linoleic Acid (CLA) Chemistry and History

Conjugated linoleic acid (CLA) is the acronym for the collective combinations of positional and geometric isomers of octadecadienoic acid (18:2) in which the constituent double bonds are separated by a single carbon-carbon bond. It is produced in the rumen of ruminant animals via biohydrogenation of polyunsaturated fatty acids, as well as during the mechanical processing of dairy products (Chin et al. 1992; Gurr 1987; Kepler and Tove 1967; Viviani 1970). The *cis*-9, *trans*-11 isomer is particularly enriched in the phospholipid fraction of cell membranes of rats fed CLA (Ha et al. 1989, Huang et al. 1994, Kramer et al. 1998, Sebedio 1997). Belury and Kempa-Steczko (1997) found that CLA is incorporated into hepatic phospholipids at the expense of linoleic acid.

Within the rumen, microorganisms form CLA. This formation is accomplished by hydrolyzing linoleic acid. The initial step in the transformation of dietary acyl lipids entering the rumen is the hydrolysis of ester linkages by microbial lipolytic enzymes; this step is a prerequisite for the biohydrogenation of unsaturated fatty acids (Harfoot and Hazlewood, 1997).

Girard and Hawke (1978), using suspensions of washed holotrich protozoa, showed that, although 1-¹⁴C linoleic acid was rapidly incorporated into the phospholipids of these organisms, very little biohydrogenation occurred. However, bacterial suspensions from the same animals rapidly hydrogenated linoleic acid to *trans*-11-octadecenoic acid and stearic acids, suggesting that it is bacteria that biohydrogenate linoleic acid and not protozoa.

Similar results were found by Singh and Hawke (1979). They also suggested that any hydrolyzed lipids found in protozoa originated from bacteria that had been digested by the protozoa.

Incubation of linolenic or linoleic acids with rumen contents *in vivo* and *in vitro*, or with pure cultures of biohydrogenating bacteria, yields a variety of fatty acids in different proportions. A major problem of understanding biohydrogenation pathways is determining which of these fatty acids are intermediates which are end products of metabolism and are not further metabolized. The present consensus is that the biohydrogenation of α -linolenic acid proceeds according to the scheme shown in Figure 2.1, and the corresponding scheme for biohydrogenation of linoleic acid is shown in Figure 2.2 (Harfoot and Hazlewood, 1997).

In formation of the *cis,trans* conjugated diene Kepler et al., 1966 observed that the first detectable intermediate was a *cis*-9,*trans*-11 conjugated diene. Later, Kepler et al., (1970) isolated a delta 12-*cis*, delta 11-*trans* isomerase from *Butyrivibrio fibrisolvens* and tested the isomerase from this bacterium and the substrate specificity of the enzyme against different positional and configurational isomers of CLA. They demonstrated an absolute requirement for the *cis*-9, *cis*-12 diene configuration, along with a free -COOH group at carbon position 1 (Kepler et al., 1971).

Conjugated linoleic acid in foods

The total CLA content of foods varies widely. With a few exceptions, the c9,t11 isomer is the predominant form, this is particularly true in meat, cheese and other dairy products. Meat from ruminants generally contains more CLA than meat from non-

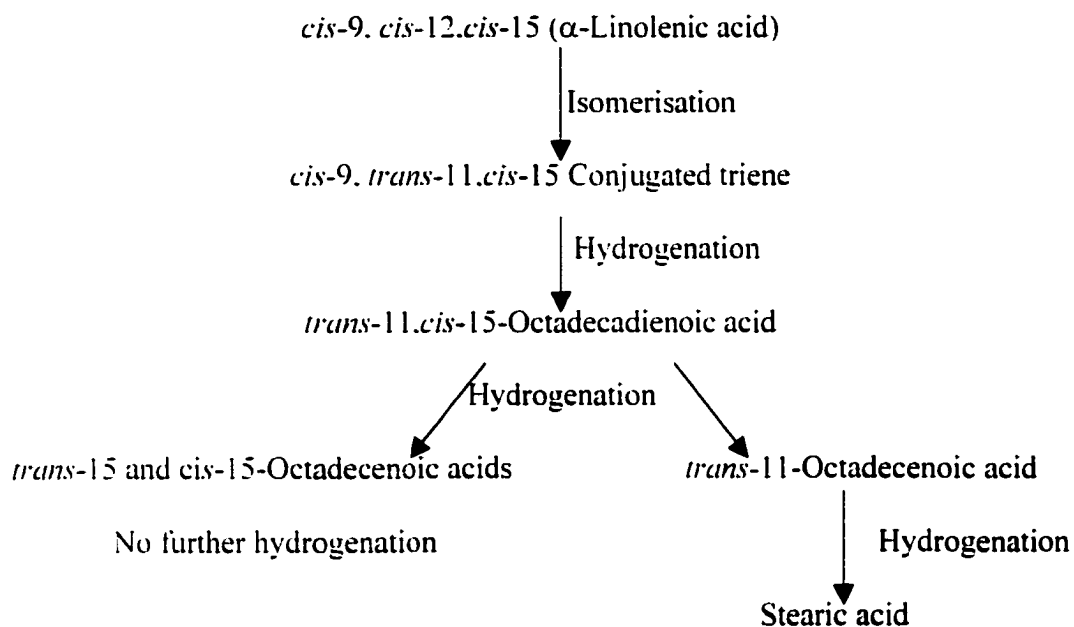


Figure 2.1. Scheme for the biohydrogenation of α -linolenic acid.

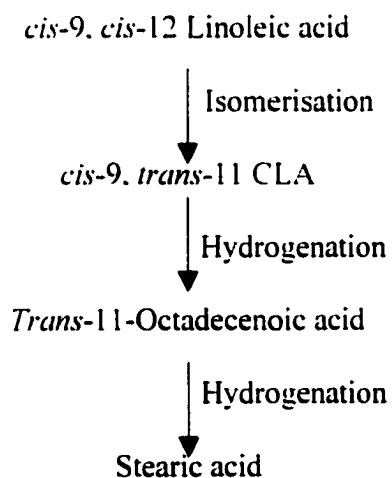


Figure 2.2. Scheme for the biohydrogenation of α -linoleic acid.

ruminants. Cheese and other dairy products are also good sources of CLA, while seafood and vegetable oils are not. It is of interest to note that the $c9, t11$ -isomer accounts for less than 50% of the total CLA in vegetable oils, in contrast to the 80-90% range found in meat and dairy products.

The applied research of CLA is still in its infancy. Some research has been done in the areas of dairy product content and fortification, feed efficiency, fat deposition and body weight gain in laboratory animals, and carcinogenesis and atherogenesis. The indications from this early research suggest that further study, especially in non-ruminants may provide useful information on potential effects of CLA in humans.

Researchers have demonstrated that the concentration of CLA in milk fat is dependent on the diet and lactation number (age) of cows. The presence of fatty acids in milk fat with conjugated unsaturation was established first by Booth et al. (1935), who reported that when cows were turned out to pasture after winter, the fatty acids of milk fat showed greatly increased absorption in the ultraviolet region at 230 nm. Additional research suggested that the UV absorbency could be increased by feeding cows polyunsaturated oils which exhibit little or no UV absorbency at 230 nm (Dann et.al. 1935; Houston et al. 1939). In 1955, Shorland et al. established that CLA, which absorbs strongly at 230-234 nm, was produced from polyunsaturated oils by microorganisms in the rumen. Bartlett and Chapman (1961) reported that CLA was an intermediate in the microbial biohydrogenation of linoleic acid to oleic acid. The $c9, t11$ isomer is formed in the rumen as a first intermediate in the biohydrogenation of linoleic acid by a linoleic acid isomerase from the anaerobic bacterium *Butyrivibrio fibrisolvens* (Kepler et al. 1966). Lal

and Narayanan (1984) found that cows with seven or more lactations produced significantly more CLA in their milk fat than cows with six or less lactations

CLA in dairy products

As cheese is the richest dietary source of CLA, this product has received the most attention. Ha et al. (1989) determined the CLA content of cheese and milk fat. In general, they found that the fat in cheese usually had greater concentrations of CLA than milk fat. These authors considered that the increased CLA content may be due to heating during processing, free-radical type oxidation of linoleic acid affected by aging and protein quantity of the cheese. The positive relationship between protein quantity and CLA content suggested that higher protein content results in higher CLA concentrations during processing, because proteins, acting as hydrogen donors, facilitate CLA formation. The results of Shantha et al. (1992) indicated that different starter cultures, processing conditions and aging periods had negligible effects on the total CLA content but did alter significantly, although not to a great extent, the CLA isomer distribution in the cheese studied.

The influence of processing conditions on the composition of the dairy products and activity of microbial metabolic reactions may be related to the differences in the CLA content of these products (Lin et al. 1995). The mechanism by which CLA increased during the processing of nonfat yogurt and butter is not understood (Shantha et al. 1995). Oxidative reactions have been postulated to promote CLA formation by causing formation of linoleate radicals resulting in a shift in double bonds to form a conjugated system (Ha et al. 1989). Factors such as air incorporation could increase oxidative reactions and,

therefore, CLA formation in butter. However, air is also incorporated into ice cream during processing and no increases in CLA were observed (Shantha et al. 1995).

Additional factors such as changes in lipid distribution (i.e., phospholipids), removal of water-soluble antioxidants (Colbert and Decker, 1991), differences in processing temperatures and/or solid-to-liquid fat ratios may cause such increases. Also differences in physical state of the fat (the fat globule is intact in ice cream and broken in butter) could increase CLA formation in butter vs. ice cream. The CLA concentrations are primarily dependent on the CLA content of the unprocessed raw material and the final fat content (Shantha et al. 1995).

CLA in beef products

Ha et al. (1987) described the isolation and identification of CLA from fried ground beef. This study suggested that CLA isomers isolated from cooked ground beef can be differentiated from hydroperoxy- or hydroxy-derivatives because they exhibit molecular weights of 280 or 294. Hence, the CLA isomers isolated from fried ground beef are not induced by oxygen but rather by some other factor, probably heat from cooking.

CLA concentrations in raw and cooked beef steaks (ribeye, T-bone, sirloin, and round) were studied by Shantha et al. (1994). They concluded that different cooking methods, including frying, broiling, baking, and microwaving, do not produce any major changes in the CLA content of ground beef when concentrations are compared on a milligrams of CLA per gram of fat basis. However, cooking method and degree of doneness do affect the concentration of CLA from beef products because cooking methods influence both fat content and amount of edible portion (i.e. yield). In addition to cooking

methods. oxidative reactions also do not alter CLA. suggesting that CLA concentrations will not change during processing and storage. However. wide variations in CLA concentration were observed in steaks originating from different animals.

CLA and cancer prevention

Conjugated linoleic acid has received considerable attention as a chemopreventive agent in the past few years since being shown to inhibit rat mammary tumorigenesis. mouse forestomach neoplasia. and mouse skin carcinogenesis (Ha et al. 1987. Ip et al. 1994. Ip et al. 1991. Ha et al. 1990). Conjugated linoleic acid has been found in triglycerides. lipoproteins. and cell membrane phospholipids in several tissues of rodents. rabbits. and humans (Ha et al. 1990. Chin et al. 1992. Huang et al. 1994. Harrison et al. 1985. Lee et al. 1994).

The anticarcinogenic activity of ground beef was investigated by using a partially purified extract (Pariza and Hargraves. 1985). In a mouse skin multistage carcinogenesis model the topical application of the partially purified extract from ground beef 5 minutes before 7,12-dimethylbenz[a]anthracene (DMBA) treatment reduced the number of papillomas per mouse. as well as the number of mice with papillomas. The anticarcinogenic factor was isolated and identified 2 years later (Ha et al. 1987) as a mixture of conjugated dienoic derivatives of linoleic acid. The analysis involved characterization by gas chromatography-mass spectrometry. and by ultraviolet and proton nuclear magnetic resonance spectroscopy. Once the characterization was complete. pure CLA was synthesized and tested for its inhibitory activity in the same model (DMBA-induced mouse epidermal carcinogenesis). Conjugated linoleic acid was applied topically

at 7 days (at a dose of 20 mg/mouse), 3 days (20 mg), and 5 minutes (10 mg) before DMBA treatment. Control mice were administered with linoleic acid topically before DMBA. The results indicated that CLA reduced the number of papillomas by one-half compared with the linoleic acid treated controls. A follow-up study showed that the synthetic CLA mixture also inhibited benzo(a)pyrene (BP)-induced forestomach tumors in mice (Ha et al. 1990). Ha et al. (1987) also determined that the mechanism for CLA inhibition of the initiation of epidermal tumors by DMBA was not yet known. However, it is likely that CLA acts in part through direct inhibition of microsomal cytochrome P-450 enzyme activity. Verma et al. (1980) suggested that CLA could act in part by competing with linoleic acid in the biosynthesis of arachidonic acid, because in mouse skin, prostaglandin synthesis inhibitors also inhibit tumor promotion. It is also possible that CLA may induce detoxification enzymes.

Ip et al. (1991) and Ip et al. (1994) found CLA included in the diet was able to prevent mammary cancer in rodents. Three levels of CLA feeding (0.5, 1 and 1.5% of diet by weight) were started 2 weeks before DMBA administration and continued until the end of the experiment (6 months after DMBA). The total number of mammary tumors in the 0.5, 1 and 1.5% CLA groups was reduced by 32, 56 and 60%, respectively (Ip et al. 1991). There seemed to be a dose-dependent protection at levels of 1% CLA and below, but no additional beneficial effect was evident at levels above 1%. A pathologist evaluated 15 different tissues at the end of the experiment and found no evidence of histomorphological abnormality.

To expand the CLA efficacy curve below 0.5%, rats were fed CLA at inclusion rates of 0.05, 0.1, 0.25 and 0.5% of the diet starting 2 weeks before DMBA and continuing for 9 months (Ip et al. 1994). Results indicated a dose-dependent effect of CLA on mammary cancer inhibition in the range between 0.05% and 0.5%. Total mammary tumor yield was reduced by 22, 36, 50 and 58% in the 0.05, 0.1, 0.25 and 0.5% CLA diets, respectively. Inter-group comparison showed that as little as 0.1% CLA was sufficient to cause a significant reduction in the total number of tumors.

CLA in animal performance and body composition

Positive effects on growth performance and feed conversion were first seen by Chen et al. (1994) in a study that utilized female Fischer rats that were fed 0.25% or 0.5% CLA during gestation and/or lactation. Feeding CLA to the dams significantly increased the postnatal body weight gain of pups. Pups given CLA post weaning also had significantly greater body weight gain and improved feed efficiency compared with control pups. Male and female ICR mice, female Sprague-Dawley rats, and Leghorn chicks were allowed diets *ad libitum* that contained 0.5% CLA for 4 to 8 weeks (Pariza et al. 1996). The CLA-fed mice, rats and chicks exhibited significant body fat reductions of 57 to 70%, 23% and 22%, respectively. These animals also showed significant increases in lean body mass and carcass water of 5 to 14%, 3%, and 4%, respectively. Mice, fed a 20% tallow diet and supplemented with additional synthetic CLA (6-fold increase), gained weight and ate food comparably to mice fed the 20% tallow diet unsupplemented with additional CLA. However, the CLA-supplemented mice exhibited significantly reduced body fat accumulation (-46%) and increased lean body mass (+9%). The effects of the removal of

dietary CLA on changes in body composition and CLA content were investigated in mice by Park et al. (1997). This study determined that CLA content of fat pad and muscle of mice fed CLA returns to levels found in control fed mice within 2 weeks to 4 weeks following dietary withdrawal. The rate of rise in body fat and decline in body water of mice previously fed the CLA diets was similar to the rate of mice fed the control diets; however, the percent fat in mice fed CLA remained 4 weeks behind percent fat in control fed mice.

Dugan et al. (1997) fed 54 barrows and 54 gilts either 2% CLA or 2% sunflower oil. The results suggested that pigs fed CLA tended to have reduced feed intakes and improved feed conversion. The pigs fed CLA also had reduced subcutaneous fat and more lean than pigs fed sunflower oil. Dugan et al. (1999) reported on the subsequent effects of CLA on pork quality. They saw no difference in glycogen utilization, lactate accumulation, or pH decline in the longissimus thoracis. However, pigs fed CLA had slightly higher longissimus thoracis temperatures at 3 h postmortem, but subsequent test on longissimus thoracis shear force, drip loss and soluble protein levels were unaffected. Diet did not affect subjective color or structure for the longissimus, however, objective values indicated longissimus from pigs fed CLA had slightly greater chroma values. There was also an increase in marbling of the longissimus of pigs fed CLA and increased petroleum-ether-extractable intramuscular fat. But, diet did not affect any palatability characteristics. Dunshea et al. (1998) and Ostrowska et al. (1999) fed 0, 1.25, 2.5, 5.0, 7.5, and 10.0 g/kg of CLA to 30 and 66 gilts, respectively. CLA was substituted for soy oil in the diets. Dunshea et al. (1998) reported that CLA had no effect on feed intake, however, average

daily gain and feed efficiency tended to increase as CLA supplementation increased. Pigs fed CLA also tended to have greater final body weights. CLA also gave a linear response in reducing back fat thickness. Ostrowska et al. (1999) reported similar results for ADG, ADFI, GF, and back fat. Ostrowska et al. (1999) also reported a linear increase in percentage water, protein and lean of the carcass, and quadratic response for carcass ash. They also showed a linear decrease in fat:protein. Cook et al. (1998) fed 24 pigs diets containing 0, 4.8 or 9.5 g of CLA- 60/kg of diet. Feed intake was decreased over the first 49 days of CLA supplementation, however, feed intake recovered for the remainder of the trial. Pigs fed CLA had reduced tenth rib back fat thickness of 24% compared with pigs fed CLA diet. No differences were seen for final body weight or loin muscle area. Loin dissection data showed less fat for both intramuscular and subcutaneous in pigs fed CLA-60 compared with the pigs fed the control diet. Theil et al. (1998) used 40 barrows in a dose titration trial where CLA 60 replaced corn. Therefore, these diets were not isocaloric. However, the added energy did not affect feed intake in this trial, and ADG and feed efficiency was improved linearly. Loin muscle area also increased linearly while 10th rib back fat decreased linearly. In most of the trials, an initial drop in feed intake has been noted or there has been a significant decrease in feed intake. Eggert et al. (1999) tried to determine if the decrease in feed intake was the cause of the decrease in backfat and increase in percent lean. In their trial they used three dietary treatments: CLA, sunflower oil (SFO) or a restricted fed sunflower oil (RSFO). In this experiment pigs fed CLA had a higher ADG than RSFO but not different than SFO. There also was no difference in feed efficiency between treatment groups.

Modified tall oil (MTO) is a byproduct of kraft (sulfate) paper production. It is found in the nonaqueous layer of rosin acids and fatty acids. In the process of making kraft paper, linoleic acid is transformed into CLA. Modified tall oil has a total CLA composition of 69% compared to the commercially available CLA-60 that is only 60% CLA. O'Quinn et al. (1998a) compared pigs fed CLA, pigs fed MTO and pigs fed a control (soy oil) diet. In the O'Quinn et al. (1998a) study, soy oil was replaced with either MTO or CLA-60 on an equal weight bases. MTO showed an advantage over both CLA and the control for ADG, ADFI, and F/G ratio; while CLA-60 showed a disadvantage compared with the control for ADG, ADFI, and F/G ratio. No differences were seen in carcass composition or quality. In a second study done by O'Quinn et al. (1998b), a dose titration study of MTO (0.0, 0.25, 0.50, and 1.00% MTO) showed no differences in ADG, ADFI, or F/G. However, there was a quadratic response for decreasing all back fat depth measurements, increasing loin muscle area, and percent lean with increasing concentration of MTO. There also was an increase in percent drip loss of carcasses from pigs fed MTO.

These studies suggest that the differences in the amount of CLA and ratio of CLA isomers have effects on the performance of the animals fed CLA. Park et al. (1999a) investigated the effects of CLA preparations, which were enriched for the *cis*-9, *trans*-11 CLA isomer or the *trans*-10, *cis*-12 CLA isomer, on body composition in mice. Reduced body fat, and enhanced body water, body protein, and body ash were associated with feeding the *trans*-10, *cis*-12 CLA isomer. Although effects of the *cis*-9, *trans*-11 CLA isomer were not of the same magnitude as that of the *trans*-10, *cis*-12 CLA isomer, there was a significant decrease in body fat compared with the control-fed mice. Park et al.

(1999b). also. found that *trans*-10, *cis*-12 CLA isomer was removed from the body faster than the *cis*-9, *trans*-11 CLA isomer when CLA was withdrawn from the diet of mice.

Although, CLA is a natural occurring fatty acid in the ruminant, and increasing the production of CLA by the ruminant does not affect milk production Looor and Herbein (1998) and Chouinard et al. (1999) observed a reduction in milk fat in dairy cows that received an abomasal infusion of a commercial CLA (CLA-60). The main differences between the commercial CLA and natural CLA is that the natural CLA is made up of only the *cis*-9 *trans*-11 isomer, and the commercial CLA has numerous isomers. Therefore, it is thought the *cis*-9 *trans*-11 isomer is not the isomer responsible for the reduction in milk fat production. Griinari et al., (1998) has shown that a milk fat depression is correlated with *trans*-10 18:1 fatty acid. Therefore, *trans*-10 *cis*-12 or *cis*-8 *trans*-10 CLA isomers or, *trans*-10 18:1 within the commercial CLA may be the specific isomer that causes milk fat depression observed by the abomasal infusion of the CLA 60. Furthermore, it is surprising that CLA would cause milk fat depression because studies in which lactating rats were fed a CLA supplement at 0.25 and 0.50% of diet dry matter showed no indication of reduction in milk fat secretion as indicated by growth rates of the nursing pups (Chin et al., 1994).

Mechanisms of CLA

Linoleic acid (18:2 ω 6) is the precursor for the formation of arachidonic acid which is metabolized to proinflammatory mediators such as prostaglandins (PGE₂). Other polyunsaturated fatty acids such as α -linolenic acid (18:3 ω 3) and γ -linolenic acid (18:3 ω 6) are precursors for the formation of eicosapentaenoic acid (EPA: 20:5 ω 3) and dihomo- γ -linolenic acid (DGLA: 20:3 ω 6) respectively, both of which form less inflammatory

mediators than arachidonic acid, can also displace arachidonic acid (20:4 ω 6). The displacement reduces the production of PGE₂. Thus, dietary fats rich in γ -linolenic acid, α -linolenic acid or EPA have been employed to modulate some of the inflammatory responses in experimental animals models (Carrick et al., 1994 and Yacoob et al., 1995) and in clinical trials (Espersen et al., 1992 and Engler et al., 1992). Similarly, CLA has been found to reduce PGE₂ (Liu et al., 1998). Banni et al. (1999) found that CLA was converted into conjugated diene arachidonic acid, and that the conjugated diene arachidonic acid was incorporated into the tissue as phospholipids. This finding suggests that conjugated arachidonic acid may compete with arachidonic acid in the biosynthesis of eicosanoids. CLA also accomplishes the reduction of PGE₂ by inhibiting linoleic acid from being further unsaturated by desaturase enzymes (Liu et al., 1998). Desaturase enzymes make linoleic acid into arachidonic acid that is the precursor for prostaglandins. The reduction in arachidonic acid production caused by CLA gives a similar effect on the immune system as increasing the ω 3 fatty acid and decreasing the ω 6 in the diet.

Conjugated linoleic acid displays a number of biological activities that may be regarded as beneficial. CLA reduces the catabolic response induced by immune stimulation in mice, rats and chickens without adversely affecting immune function (Cook et al. 1993, Miller et al. 1994). This response is cytokine-mediated and regulated by prostaglandin E₂ synthesis. It is possible that these seemingly disparate effects of CLA are mechanistically related through effects on prostaglandin E₂ metabolism and signal transduction pathways (Chin et al. 1994). The observation that CLA exhibits antioxidant

activity *in vitro* and *in vivo*, especially in tissues (e.g. mammary gland) in which it is also an anticarcinogen (Ha et al. 1990, Ip et al. 1991) may also be mechanistically important.

The catabolism of skeletal muscle that follows immune stimulation partitions energy and protein into immune functions and away from other biological processes. Reducing the catabolism of skeletal muscle enhances growth and improves feed efficiency (Benson et al. 1993, Klasing and Austic 1984, Klasing et al. 1987). Fish oil reduces the catabolic process induced by immune stimulation (Meydani 1992), but in mice and chickens, CLA seems much more effective than fish oil (Cook et al. 1993, Miller et al. 1994).

Park et al. (1997) established that mice fed CLA-supplemented diets exhibited significant reductions in body fat composition relative to controls. This study was conducted with tissues from control and CLA-fed mice. The findings are consistent with the interpretation that feeding mice a diet supplemented with CLA enhances fatty acid β -oxidation in skeletal muscle and fat pad, but not liver. These data also indicate that CLA treatment reduced lipoprotein lipase (LPL) activity while apparently enhancing lipolysis by increased hormone-sensitive lipase activity and enhanced norepinephrine-induced lipolysis. If these *in vitro* findings are representative of *in vivo* physiological changes, then the reduction of LPL and enhanced lipolysis seem to provide a framework for partially understanding the reduction in body fat that is evident in several studies.

A possible explanation for the apparent enhancement of lean body mass may also be explained by this mechanism, in that the increased lipolysis may allow the lean tissue more access to energy. The enhanced lean body mass may also be related to the

protections against the catabolic effects of immune stimulation (Cook et al., 1993; Miller et al., 1994), which is modulated by PGE₂ and mediated by interleukin-1 and tumor necrosis factor- α , both of which are linked to obesity (Hotamisligil et al., 1993, 1995, and 1996; Bunout et al., 1996).

There seems to be parallels between the effects of CLA and the effects of dietary fish oil. Like CLA, fish oil is reported to reduce fat pad size (Parrish et al., 1990; Awad et al., 1990), prevent cachexia and body weight loss after immune stimulation (Beck et al., 1991; Hellerstein et al., 1989) and reduce tissue arachidonic acid levels (Leslie et al., 1985; Lokesh et al., 1986). The omega-3 fatty acids of fish oil modulate numerous biological and physiological effects that are linked to tumor necrosis factor- α , interleukin-1, and eicosanoid pathways (Hellerstein et al., 1989; Endres et al., 1989), indicating that the effects of CLA may also involve eicosanoid mechanisms.

Responses To Energy Intake In The Growing Pig

Protein deposition is limited by an inadequate supply of either protein or energy. The response curve of protein deposition has two phases, an initial, protein-dependent phase, that is followed by one in which deposition can only be further enhanced by an increase in energy intake (energy dependent phase). Under conditions of protein inadequacy, protein deposition responds linearly to protein intake and is unaffected by increases in energy intake. Once increment of protein fails to elicit further response, protein deposition can only be raised by increasing energy intake, whereupon further protein inputs lead to a resumption of the initial, linear response until, once again, energy intake becomes the limiting factor.

Whittemore and Fawcett (1976) suggested that under conditions of protein adequacy, the relationship between energy and protein deposition is basically of linear-plateau form. In young pigs, limited ingestive capacity usually prevents full expression of potential for protein deposition, even when high energy diets are offered (Dunkin 1990). Consequently, only the linear portion of the curve applies. As growth proceeds, the potential for protein accretion declines relative to appetite, and once capacity for energy intake exceeds that for protein growth the full linear-plateau form of the relationship is seen (Dunkin et al. 1986). The stage of growth at which the plateau occurs depends upon the sex and genotype of the pig. This relationship between energy intake and protein accretion is important to understand because it is closely related to feed efficiency and composition of gain and is affected by sex, genotype and stage of growth.

Effects of added fat

Addition of fat to the diet increases energy density, however, DE intake remains constant because of a reduction in feed intake (Ewan 1991). Pettigrew and Moser (1991) suggest that the ME intake of the pig is increased even though feed intake is decreased. As fatty acids are digested and absorbed, they are generally deposited as fat and increase the efficiency of energy utilization. Added fat also has a lower heat increment than carbohydrate and can be beneficial at high environmental temperatures by maintaining energy intake to support maximal growth rate (Stahly and Cromwell 1979). With reduced feed intake and increased energy intake the dietary protein to energy intake becomes important in formulating diets. If protein is not increased proportionally with the increase in energy intake the added fat will be directly deposited in the adipose tissue and will result

in a fatter carcass. Therefore, it is importance to adjust diets to the appropriate protein:energy ratio which is influenced by the age of the pig.

In pigs weaned at 3 weeks of age, additional fat is not well utilized for 10 to 14 days after weaning and can result in depression of growth rate (Cera et al. 1988). Therefore, fat sources should be used sparingly in weanling diets. However, Pettigrew et al. (1986) found that oral doses of corn oil during the first 2 days after birth resulted in a delay of mortality but there was no effect on preweaning survival. Furthermore, research by Chiang et al. (1987) has shown that newborn piglets have ample capacity to digest and absorb a large oral dose of fish oil. Lepin et al. (1986) and Odle et al. (1987) found that oral doses of fats containing medium-chain fatty acids increased glucose and nonesterified fatty acid concentrations and decreased urinary nitrogen excretion, supporting the notion that piglets can utilize these fats effectively.

Supplemental fat in starting pigs (5 to 20 kg BW) has centered on improved gain and feed efficiency and improved palatability. Pettigrew and Moser (1991) reviewed published reports to determine the effects of supplemental fat on average daily gain, average daily feed intake, and gain:feed ratio of starting pigs. They counted the number of positive and negative responses, without regard to statistical significance within the experiment. They found that average daily gain and average daily feed intake of starting pigs was reduced by the addition of supplemental fat. However the reduction in average daily gain was small and the total number of positive responses was similar to the number of negative responses. Also, adjusting to a constant protein:energy ratio resulted in zero response for average daily gain, and the number of positive responses were higher than the

number of negative responses. The addition of fat to the diet improved gain:feed ratio, with greater improvement in diets adjusted to a constant protein:energy ratio.

The addition of fat to growing-finishing diets is mainly concerned with improved gain, feed efficiency and effects on carcass composition. Moser (1977) and Pettigrew and Moser (1991) reviewed published reports to determine the effects of supplemental fat on average daily gain, average daily feed intake, gain:feed ratio and backfat measurements of growing-finishing pigs. In their reviews, they found an increase in average daily gain and a greater number of positive responses than negative responses for average daily gain. Average daily gain was only slightly increased when a constant protein:energy ratio was used. Average daily feed intake was reduced and there was a greater number of negative responses than positive response. Gain:feed response followed that of the average daily gain with an overall increased response for added fat and a slight further increase when a constant protein:energy ratio was used. Moser (1977) reported little effect on backfat if low levels of supplemental fat ($\leq 3\%$) were used. Whereas, Pettigrew and Moser (1991) showed an increase in backfat with each incremental increase of supplemental fat.

Effect of fat on carcass quality

Fat quality is largely defined in terms of physical and nutritive characteristics, both of which are closely related to the fatty acid composition of the fat depot. Because monogastric animals are very susceptible to tissue fatty acid alteration through dietary modification (Villiegas et al., 1973; Skelley et al., 1975; St. John et al., 1987; Romans et al., 1995a and 1995b), it is practical to alter the fatty acid profile to both create and solve problems in carcass quality by changing dietary fat sources. In monogastrics, many of the

fatty acids in the diet are absorbed intact across the intestine and directly deposited into the fat. Therefore, the composition of the fat depots, in terms of fatty acid profile, are closely related to the fatty acid profile of the dietary fat. If monogastrics are fed a diet with no added fats, they synthesize and deposit saturated fatty acids and mono-unsaturated fatty acids (Metz and Dekker, 1981). Because the pig and many other monogastrics cannot synthesize linoleic acid, which is used to synthesize arachidonic acid, there is a dietary requirement for linoleic acid of 0.10% (NRC, 1998).

The major issues relating to fat quality are soft fat, oxidative rancidity, and the impact of the composition of pork fat on human health. These issues are receiving attention in the US pork industry because of the significant changes in production practices and consumer preferences that have occurred over recent years. Soft fat is of major concern to the meat processor because it can cause problems during cutting, grinding and slicing operations and can result in lower processing yields and, therefore, reduce the value of the carcass (Shackelford et al., 1990). An area that has received relatively little attention is the relationship between the composition of pig fat and the eating quality of pork. Specifically, odor and flavor of pork can be affected by the fatty acid composition of the fat depots. Historically, the major problems in this respect were experienced with feeding fish oils or fish meals with a relatively high oil content and the associated development of fishy taints in the meat.

The economics of competitiveness of certain fats relative to corn on a cost per unit of energy basis and also use of fat to suppress dust levels within swine buildings has increased the inclusions of fats in corn-soy diets. High oil corn is one ingredient that is

coming to the forefront as a cost effective and convenient way to increase the energy density of the diet and get a complete mix of the fat in the diet. High oil corn also has the benefit of having a higher crude protein level and a more favorable amino acid balance in that it has a higher lysine concentration than conventional corn. All of which helps in its economic competitiveness. However, the oil in high oil corn is rich in unsaturated fatty acids, mainly in linoleic. Linoleic acid is of major concern. This concern is because linoleic acid is directly deposited by the pig into its fat depot. Also, most conventional feedstuffs and fat sources have relatively high concentrations of linoleic acid. The consumption of linoleic acid from the various feedstuffs adds up to a large percentage of the fatty acids being polyunsaturated. The absorption and deposition of the PUFA causes the problem of soft fat and potential for increased fat oxidation.

A measure of the degree of unsaturation of fats, both dietary and within the body, is the Iodine Value. Higher values indicate a greater proportion of unsaturated fat. Boyd (1997) investigated the relationships between dietary fatty acid profile and the fatty acid profile and Iodine Value of backfat. The relationship between dietary linoleic content and the Iodine Value of the backfat was linear, with Iodine Values increasing from approximately 65 to 76 for diets containing 1.3 and 3.5% of linoleic acid, respectively.

The Danish Meat Research Institute has set a fairly rigid standard of a maximum body fat Iodine Value of 70 (Barton-Gade, 1987). Boyd (1997) suggested that some pigs fed a corn-soy diet with no added fat would exceed this threshold. Therefore, the US pork industry threshold for Iodine Values for body fat composition has not been clearly established and this continues to perpetuate the soft fat problems. Boyd (1997) has

suggested a less stringent Iodine Value of 74 for US conditions and dietary linoleic acid maximum of 2.1% to be under this threshold. In Europe, to prevent problems from occurring, dietary specifications specify a maximum inclusion for linoleic acid of 1.6% of the diet for finisher diets.

St. John et al. (1987) demonstrated a significant increase in oleic acid content of subcutaneous fat from pigs fed 20% canola oil. The carcasses were significantly more oily than pigs fed normal corn control diet, and, therefore, were unacceptable for bacon production. Shackelford et al. (1990) fed corn-soy diets with 0 (control) or 10% of either beef tallow, safflower oil, sunflower oil, or canola oil. They showed that slicing yields and sensory scores for overall palatability and flavor quality were lower for bacon produced from swine fed canola oil. The softness of fat is directly proportional to the amount of unsaturated fatty acids in the fat depot. The changes in the genetics of pigs and in feed ingredients used to formulate swine diets have brought the problems of soft fat to the attention of the packer. Soft fat problems are relatively greater in leaner pigs which have a greater proportion of the fatty acids in the carcass fat derived from the diet and a smaller proportion from *de novo* synthesis of fatty acids by the animal than pigs that are genetically fatter (Wood et al., 1989). Wood et al. (1989) compared the composition of the backfat in pigs with different backfat depths and showed that leaner pigs had a higher proportion of polyunsaturated fatty acids (i.e. C18:2 and C18:3).

Cameron and Enser (1991) investigated the relationship between fatty acid composition of intramuscular fat and the palatability of pork. In this study the data showed the correlations between the concentration of specific fatty acids and eating quality traits

were generally weak. However, correlations involving polyunsaturated fatty acids and palatability scores were generally negative and those for the saturated fatty acids were generally positive. These results suggest that the higher the degree of unsaturation in the intramuscular fat, the greater the incidence of abnormal flavors. The increase potential of oxidation and development of rancidity of the unsaturated fatty acid is the most likely explanation of these results.

The idea of making pork a "heart healthy" food has lead to the inclusion of feedstuffs that are high in omega-3 fatty acids in swine diets. Omega-3 fatty acids have been associated with a beneficial effect on the cardiovascular diseases. Feed sources that are rich in omega-3 fatty acids include: fish oils and certain vegetable oils such as linseed. Including these in the diet of the pigs increases the concentration of omega-3 fatty acids in the fat depots. With increased omega-3 fatty acids in pork, pork is then called a "healthier" food for human consumption. Omega-3 fatty acids were increased by Irie and Sakimoto, (1992) by feeding pigs 0, 2, 4, or 6% fish oil. However, they found a linear increase in Iodine Value and a linear decrease in hardness of backfat of pigs fed increasing amount of fish oil, suggesting that there is a potential for increased rancidity and off flavor of pork from pigs fed fish oil. Romans et al. (1995a and 1995b) also increased omega-3 fatty acids by feeding flaxseed. They also found an increase in fat softness, but they reported that there were no processing difficulties caused by the softer fat. However, a trained taste panel could detect off flavors from bacon of pigs fed 10 or 15% flaxseed. Also, TBA levels were increased in rendered lard, suggesting an increase in oxidation of the polyunsaturated fatty acids.

Literature Cited

- Awad, A. B., L. L. Bernardis, and C. S. Fink. 1990 Failure to demonstrate an effect of dietary fatty acid composition on body weight, body composition and parameters of lipid metabolism in mature rats. *J. Nutr.* 120: 1277-1282.
- Banni S. E. Angioni, V. Casu, M. P. Melis, G. Carta, F. P. Corongiu, H. Thompson, and C. Ip. 1999. Decrease in linoleic acid metabolites as a potential mechanism in cancer risk reduction by conjugated linoleic acid. *Carcinogenesis* 20:1019-1024.
- Bartlett, J. C. and D. G. Chapman. 1961. Detection of hydrogenated fats in butter fat by measurement of cis-trans conjugated unsaturation. *J. Agric. Food Chem.* 9:50-53.
- Barton-Gade, P. A. 1987. Meat and fat quality in boars, castrates and gilts. *Livestock Prod. Sci.* 16:187.
- Beck, S., K. L. Smith, and M. J. Tisdale. 1991. Anticachectic and antitumor effect of eicosapentaenoic acid and its effect on protein turnover. *Cancer Res.* 51:6089-6093.
- Belury M. A. and A. Kempasteczko. 1997. Conjugated linoleic acid modulates hepatic lipid composition in mice. *Lipids* 32:199-204.
- Benson, B. N., C. C. Calvert, E. Roura, and K. C. Klasing. 1993. Dietary energy source and density modulate the expression of immunologic stress in chicks. *J. Nutr.* 123:1714-1723.
- Booth, R.G., S. K. Kon, W. J. Dann, and T. Moore. 1935. A study of seasonal variation in butter fats. II. A seasonal spectroscopic variation in the fatty acid fraction. *Biochem. J.* 29:133-137.
- Boyd, R. D. 1997. Relationship between dietary fatty acid profile and body fat composition in growing pig. PIC USA T & D Technical Memo 153. Pig Improvement Company, USA Franklin, Kentucky.
- Bunout, D., C. Munoz, M. Lopez, M. P. de la Maza, L. Schlesinger, S. Hirsch, and M. Pettermann. 1996. Interleukin 1 and tumor necrosis factor in obese alcoholics compared with normal-weight patients. *Am. J. Clin. Nutr.* 63: 373-376.
- Cameron, N. D. and M. B. Enser. 1991. Fatty acid composition of lipid in longissimus dorsi muscle of Duroc and British Landrace pigs and its relationship with eating quality. *Meat Sci.* 26:295-307.
- Carrick J. R. Schnellmann, and J. Moore. 1994. Dietary source of ω -3 fatty acids affects endotoxins-induced peritoneal macrophage tumor necrosis factor and eicosanoid synthesis. *Shock* 2:421-426.
- Cera, K. R., D. C. Mahan, and G. A. Reinhart. 1988. Weekly digestibilities of diets supplemented with corn oil, lard or tallow by weanling swine. *J. Anim. Sci.* 66:1430-1437.
- Chiang, S. H., J. E. Pettigrew, S. D. Clarke, S. G. Cornelius, and R. L. Moser. 1987. Fat utilization in newborn piglets. *J. Anim. Sci.* 65(Suppl. 1):307.

- Chin, S. F., W. Liu, J. M. Storkson, Y. L. Ha and M. W. Pariza. 1992. Dietary sources of conjugated dienoic isomers of linoleic acid, a newly recognizes class of anticarcinogens. *J. Food Comp. Anal.* 5:185-197.
- Chin, S. F., J. M. Storkson, K. J. Albright, M. E. Cook, and M. W. Pariza. 1994. Conjugated linoleic acid is a growth factor for rats as shown by enhanced weight gain and improved feed efficiency. *J. Nutr.* 124:2344-2349.
- Chouinard P. Y., L. Corneau, D. M. Barbano, L. E. Metzger, and D. E. Bauman. 1999. Conjugated linoleic acids alter milk fatty acid composition and inhibit milk fat secretion in dairy cows. *J. Nutr.* 129:1579-1584.
- Colbert, L. B. and E. A. Decker. 1991. Antioxidant activity of an ultrafiltration permeate from acid whey. *J. Food Sci.* 56:1248-1250.
- Cook, M. E., D. L. Jerome, T. D. Crenshaw, D. R. Buege, M. W. Pariza, K. J. Albright, S. P. Schmidt, J. A. Scimeca, P. A. Lofgren, and E. J. Hentges. 1998. Feeding conjugated linoleic acid improves feed efficiency and reduces whole body fat in pigs. *FASEB J.* 12, A836.
- Cook, M. E., C. C. Miller, Y. Park, and M. W. Pariza. 1993. Immune modulation by altered nutrient metabolism: nutritional control of immune-induced growth depression. *Poult. Sci.* 72:1301-1305.
- Dugan M. E. R., J. L. Aalhus, A. L. Schaefer, and J. K. G. Kramer. 1997 The effect of conjugated linoleic acid on fat to lean repartitioning and feed conversion in pigs. *Can. J. Anim. Sci.* 77:723-725.
- Dugan M. E. R., J. L. Aalhus, L. E. Jeremiah, J. K. G. Kramer, and A. L. Schaefer. 1999. The effects of feeding conjugated linoleic acid on subsequent pork quality. *Can. J. Anim. Sci.* 79:45-51
- Dunkin, A. C. 1990. Responses to energy intake in the growing pig. *Pig News Info.* 11:2 p 159-162.
- Dunkin, A. C., J. L. Black, and J. L. James. 1986. Nitrogen balance in relation to energy intake in entire male pigs weighting 75 kg. *Brit. J. of Nutr.* 55, 201-207.
- Dunshea, F. R., E. Ostrowska, M. Muralitharan, R. Cross, D. E. Bauman, M W. Pariza, and C. Skarie. 1998. Dietary conjugated linoleic acid decreases back fat in finisher gilts. *J. Anim. Sci.* 76 (Suppl. 1) 131.
- Eggert, J. M., M. A. Belury, and A. P. Schinckel. 1999. The effects of conjugated linoleic acid (CLA) and feed intake on lean pig growth and carcass composition. *Purdue University Swine Day.* p. 21-25.
- Endres, S., R. Ghorbani, V. E. Kelley, K. Georgilis, G. Lonnemann, J. W. M. van der Meer, J. G. Cannon, T. S. Rogers, M. S. Klempner, P. C. Weber, E. J. Schaefer, S. M. Wolff, and C. A. Dinarello. 1989. The effect of dietary supplementation with n-3 polyunsaturated fatty acids on the sythesis of interleukin-1 and tumor necrosis factor by mononuclear cells. *New Engl. J. Med.* 320: 265-271.
- Engler M., M. Engler, S. Erinckson, and S. Paul. 1992. Dietary gamma-linolenic acid lowers blood pressure and alter aortic reactivity and cholesterol metabolism in hypertension. *J. Hypertens.* 10: 1197-1204.

- Esperson G., N. Grunnet, and H. Lervang. 1992. Decreased interleukin-1 beta levels in plasma from rheumatoid arthritis patients after dietary supplementation with n-3 polyunsaturated fatty acids. *Clin Rheumatol.* 11:393-395.
- Ewan, R. C. 1991. Energy utilization in swine nutrition. In: E. R. Miller, D. E. Ullrey, and A. J. Lewis.(Ed.) *Swine Nutrition*. P. 121. Butterworth-Heinemann, Stoneham, MA.
- Girard, V. and J. C. Hawke. 1978. The role of holotrichs in the metabolism of dietary linoleic acid in the rumen. *Biochim. Biophys. Acta.* 528 17-27.
- Griinari, J. M., P. Y. Chouinard, D. E. Bouman, D. L. Palmquist, and K. V. V. Nurmela. 1998. *Trans*-Octadecenoic acids and milk fat depression in lactating dairy cows. *J. Dairy Sci.* 81: 1251-1261.
- Gurr, I. G. 1987. Isomeric fatty acids. *Biochem. Soc. Trans.* 15:336-339.
- Ha, Y. L., N. K. Grimm, and M. W. Pariza. 1987. Anticarcinogens from fried ground beef: heat-altered derivatives of linoleic acid. *Carcinogenesis.* 8:1881-1887.
- Ha, Y. L., J. M. Storkson, and M. W. Pariza. 1990. Inhibition of benzo[α]pyrene-induced mouse forestomach neoplasia by conjugated dienoic derivatives of linoleic acid. *Cancer Res.* 50:1097-1101.
- Ha, Y.L., N. K. Grimm, and M. W. Pariza. 1989. Newly recognized anticarcinogenic fatty acids: identification and quantification in natural and processed cheeses. *J. Agric. Food Chem.* 37:75-81.
- Harfoot, C. G. and G. P. Hazlewood. 1997. Lipid metabolism in the rumen. In: *The rumen microbial ecosystem*, p 382-426. P. N. Hobson and C. S. Stewart, eds. Blackie Academic and Professional, London.
- Harrison, K., K. Cawood, A. Iverson, and T. Dormandy. 1985. Diene conjugation patterns in normal human serum. *Life Chem. Rep.* 3:41-44.
- Hellerstein, M. K., S. N. Meydani, M. Meydani, K. Wu, and C. A. Dinarello. 1989. Interleukin-1-induced anorexia in the Rat. *J. Clin. Invest.* 84:228-235.
- Hotamisligil, G. S., N. S. Shargill, and B. M. Spiegelman. 1993. Adipose expression of tumor necrosis factor- α : Direct role in obesity-linked insulin resistance. *Science* 259, 87-91.
- Hotamisligil, G. S., P. Arner, J. F. Caro, R. L. Atkinson, and B. M. Spiegelman. 1995. Increased adipose tissue expression of TNF- α in human obesity ad insulin resistance. *J. Clin. Invest.* 95, 2409-2415.
- Hotamisligil, G. S., P. Peraldi, A. Budavari, R. L. Atkinson, and B. M. Spiegelman. 1996. IRS-1-mediated inhibition of insulin receptor tyrosine kinase activity in TNF- α and obesity-induced insulin resistance. *Science* 271, 665-668.
- Huang, Y. C., L. O. Lueddecke, And T. D. Shultz. 1994. Effect of cheddar cheese consumption on plasma conjugated linoleic acid concentrations in men. *Nutr. Res.* 14:373-386.
- Ip, C., Chin, S. F., Scimeca, J. A. and M. W. Pariza. 1991. Mammary cancer prevention by conjugated dienoic derivative of linoleic acid. *Cancer Res.* 51:6118-6124.
- Ip, C., M. Singh, H. J. Thompson, and J. A. Scimeca. 1994. Conjugated linoleic acid suppresses mammary carcinogenesis and proliferative activity of the mammary gland in the rat. *Cancer Res.* 54:1212-1215.

- Irie, M., and M. Sakimoto. 1992. Fat characteristics of pigs fed fish oil containing eicosapentaenoic and docosahexaenoic acids. *J. Anim. Sci.* 70:470.
- Kepler, C. R., W. P. Tucker, and S. B. Tove. 1970. Biohydrogenation of unsaturated fatty acids. IV. Substrate specificity and inhibition of linoleate Δ^{12} -*cis* Δ^{11} -*trans* isomerase from *Butyrivibrio fibrisolvens*. *J. Biol. Chem.* 245:3612-3620.
- Kepler, C. R., W. P. Tucker, and S. B. Tove. 1971. Biohydrogenation of unsaturated fatty acids. V. Stereospecificity of proton addition and mechanism of action of linoleic acid Δ^{12} -*cis* Δ^{11} -*trans* isomerase from *Butyrivibrio fibrisolvens*. *J. Biol. Chem.* 246: 2765-2771.
- Kepler, C. R., K. P. Hiron, J. J. McNeill, and S. B. Tove. 1966. Intermediates and products of the biohydrogenation of linoleic acid by *Butyrivibrio fibrisolvens*. *J. Biol. Chem.* 241:1350-1354.
- Kepler, C. R. and S. B. Tove. 1967. Biohydrogenation of unsaturated fatty acids. *J. Biol. Chem.* 242:5686-5696.
- Klasing, K. C. and R. E. Austic. 1984. Changes in protein degradation in chickens due to an inflammatory challenge. *Proc. Soc. Exp. Biol. Med.* 176:292-296.
- Klasing, K. C., D. E. Laurin, R. K. Peng, and D. M. Fry. 1987. Immunologically mediated growth depression in chicks: influence of feed intake, corticosterone and interleukin-1. *J. Nutr.* 117:1629-1637.
- Kramer, J. K. G., N. Sehat, M. E. R. Dugan, M. M. Mossoba, M. P. Yurawecz, J. A. G. Roach, K. Eulitz, J. L. Aalhus, A. L. Schaefer, and Y. Ku. 1998. Distributions of conjugated linoleic acid (CLA) isomers in tissue lipid classes of pigs fed a commercial CLA mixture determined by gas chromatography and silver ion high-performance liquid chromatography. *Lipids* 33:549-558.
- Lee, K. N., D. Kritchevsky, and M. W. Pariza. 1994. Conjugated linoleic acid and atherosclerosis in rabbits. *Atherosclerosis*. 108:19-25.
- Lepine, A. J., R. D. Boyd, and J. A. Welch. 1986. Effect of colostrum or MCT supplementation on the pattern of plasma glucagon, FFA and Glucose in newborn pigs. *J. Anim. Sci.* 63(Suppl. 1):275.
- Leslie, C. A., W. A. Gonnerman, M. D. Ullman, K. C. Hayes, C. Franzblau, and E. S. Cathcart. 1985. Dietary fish oil modulates macrophage fatty acids and decreases arthritis susceptibility in mice. *J. Exp. Med.* 162:1336-1349.
- Lin, H., T. D. Boylston, M. J. Chang, L. O. Lueddecke, and T. D. Shultz. 1995. Survey of the conjugated linoleic acid contents of dairy products. *J. Dairy Sci.* 78:2358-2365.
- Liu, K. and M. A. Belury. 1998. Conjugated linoleic acid reduces arachidonic acid content and PGE₂ synthesis in murine keratinocytes. *Cancer Lett.* 127, 15-22.
- Lokesh, B. R., H. L. Hsieh, and J. E. Kinsella. 1986. Peritoneal macrophages from mice fed dietary (n-3) polyunsaturated fatty acids secrete low levels of prostaglandins. *J. Nutr.* 116: 2547-2552.
- Lor J. J. and J. H. Herbein. 1998. Exogenous conjugated linoleic acid isomers reduce bovine milk fat concentration and yield by inhibiting de novo fatty acid synthesis. *J. Nutr.* 128:2411-2419.

- Metz, S. H. M. and R. A. Dekker. 1981. The contribution of fat mobilization to the regulation of fat deposition in growing Large White and Pietrain pigs. *Anim. Prod.* 33: 149-157.
- Meydani, S. N. 1992. Modulation of cytokine production by dietary polyunsaturated fatty acids. *Proc. Soc. Exp. Biol. Med.* 200:189-193.
- Miller, C. C., Y. Park, M. W. Pariza and M. E. Cook. 1994. Feeding conjugated linoleic acid to animals partially overcomes catabolic response due to endotoxin injection. *Biochem. Biophys. Res. Commun.* 198:1107-1112.
- Moser, B. D. 1977. Fat in Swine Nutrition. *Feedstuffs* 49:20.
- NCR. 1998. Nutrient Requirements of Swine (10th ed.). National Academy Press, Washington, DC.
- O'Quinn, P. R., J. W. Smith, II, J. L. Nelssen, M. D. Tokach, R. D. Goodband, and J. S. Smith. 1998a. Effects of modified tall oil versus conjugated linoleic acid on finishing pig growth performance and carcass characteristics. *Kansas State University Swine Day 1998*. 157-161.
- O'Quinn, P. R., J. W. Smith, II, J. L. Nelssen, M. D. Tokach, R. D. Goodband, J. C. Woodworth, and J. A. Unruh. 1998b. Effects of level of modified tall oil on finishing pig growth performance and carcass characteristics. *Kansas State University Swine Day 1998*. 162-165.
- Odle, J., N. J. Benevenga, and T. D. Crenshaw. 1987. Evaluation of medium and long chain triglycerides as energy sources in newborn pigs. *J. Anim. Sci.* 65(Suppl. 1):307-308.
- Ostrowska E., M. Muralitharan, R. F. Cross, D. E. Bauman, and F. R. Dunshea. 1999. Dietary conjugated linoleic acids increase lean tissue and decrease fat deposition in growing pigs. *J. Nutr.* 129:2037-2042.
- Pariza, M. W. and Hargraves, W. A. 1985. A beef-derived mutagenesis modulator inhibits initiation of mouse epidermal tumors by 7,12-dimethylbenz[a]anthracene. *Carcinogenesis*. 6:591-593.
- Pariza, M. W., Y. Park, M. Cook, K. Albright, and W. Liu. 1996. Conjugated linoleic acid (CLA) reduces body fat. *FASEB J.* 10:A560.
- Park Y. J. M. Storkson, K. J. Albright, K. J. Liu, and M. W. Pariza. 1999a. Evidence that the trans-10,cis-12 isomer of conjugated linoleic acid induces body composition changes in mice. *Lipids* 34:235-241.
- Park Y. J. M. Storkson, K. J. Albright, K. J. Liu, and M. W. Pariza. 1999b. Changes in body composition in mice during feeding and withdrawal of conjugated linoleic acid. *Lipids* 34:243-248.
- Park Y. K. L. Albright, W. Liu, J. M. Storkson, M. E. Cook, and M. W. Pariza. 1997. Effect of conjugated linoleic acid on body composition in mice. *Lipids* 32:853-858.
- Parrish, C. C., D. A. Pathy, and A. Angel. 1990. Dietary fish oils limit adipose tissue hypertrophy in rats. *Metabolism* 39:217-219.
- Pettigrew J. E. and R. L. Moser. 1991 Fat in Swine Nutrition. In: E. R. Miller, D. E. Ullrey, and A. J. Lewis.(ed.) *Swine Nutrition*. p. 133. Butterworth-Heinemann, Stoneham, MA.

- Pettigrew, J. E., S. G. Cornelius, R. L. Moser, T. R. Heeg, H. E. Hanke, K. P. Miller and C. D. Hagen. 1986. Effects of oral doses of corn oil and other factors on preweaning survival and growth of piglets. *J. Anim. Sci.* 62:601-612.
- Romans, J. R., R. C. Johnson, E. M. Wulf, G. W. Libal, and W. J. Costello. 1995a. Effects of ground flaxseed in swine diets on pig performance and on physical and on physical and sensory characteristics and omega-3 fatty acids content of pork: I. Dietary level of flaxseed. *J. Anim. Sci.* 73:1982-1986.
- Romans, J. R., R. C. Johnson, E. M. Wulf, G. W. Libal, and W. J. Costello. 1995b. Effects of ground flaxseed in swine diets on pig performance and on physical and on physical and sensory characteristics and omega-3 fatty acids content of pork: II. Duration of 15% dietary flaxseed. *J. Anim. Sci.* 73:1987-1999.
- Sebedio J. L., P. Juaneda, G. Dobson, I. Ramilison, J. D. Martin, and J. M. Chardigny. 1997. Metabolites of conjugated isomers of linoleic acid (CLA) in the rat. *Biochim. Biophys. Acta Lipids & Lipid Metabolism* 1345:5-10.
- Shackelford, S. D., M. F. Miller, K. D. Haydon, N. V. Lovegreen, C. E. Lyon, and J. O. Reagan. 1990. Acceptability of bacon as influenced by the feeding of elevated levels of monounsaturated fats to growing-finishing swine. *J. Food Sci.* 55:621-624.
- Shantha, N. C., Crum, A. D. and Decker, E. A. 1994. Evaluation of conjugated linoleic acid concentrations in cooked beef. *J. Agric. Food Chem.* 42:1757-1760.
- Shantha, N. C., Decker, E. A. and Ustunol, Z. 1992. Conjugated linoleic acid concentration in processed cheese. *J. Am. Oil Chem. Soc.* 69:425-428.
- Shantha, N. C., Ram, L. N., O'Leary, J., Hicks, C. L. and Decker, E. A. 1995. Conjugated linoleic acid concentrations in dairy products as affected by processing and storage. *J. Food Sci.* 60:695-697.
- Singh, S. and J. C. Hawke. 1979. The *in vitro* lipolysis and biohydrogenation of monogalactosyldiglyceride by whole rumen contents and its fractions. *J. Sci. Food Agric.* 30: 603-612.
- Skelley, G. C., R. F. Borgman, D. L. Handlin, J. C. Acton, J. C. McConnell, F. B. Wardlaw, and E. J. Evans. 1975. Influence of diet on quality, fatty acids, and acceptability of pork. *J. Anim. Sci.* 41:1298.
- St. John, L. C., C. R. Yung, D. A. Knabe, L. D. Thompson, G. T. Schelling, S. M. Grundy, and S. B. Smith. 1987. Fatty acid profile and sensory and carcass traits of tissues from steers and swine fed an elevated monounsaturated fat diet. *J. Anim. Sci.* 64:1441.
- Stahly, T. S. and G. L. Cromwell. 1979. Effect of environmental temperature and dietary fat supplementation the performance and carcass characteristics of growing and finishing swine. *J. Anim. Sci.* 49: 1478-1488.
- Thiel, R. L., J. C. Sparks, B. R. Wiegand, F. C. Parrish, Jr., and R. C. Ewan. 1998. Conjugated linoleic acid improves performance and body composition in swine. *J. Anim. Sci.* 76(Suppl. 1):61.
- Verma, A. K., C. L. Ashendel, and R. K. Boutwell. 1980. Inhibition by prostaglandin synthesis inhibitors of the induction of epidermal ornithine decarboxylase activity,

- the accumulation of prostaglandins, and tumor promotion caused by 12-O-tetradecanoylphorbol-13-acetate. *Cancer Res.* 40:308-315.
- Villegas, F. J., H. B. Hedrick, T. L. Veum, K. L. McFate, and M. E. Bailey. 1973. Effect of diet and breed on fatty acid composition of porcine adipose tissue. *J. Anim. Sci.* 36: 663.
- Viviani, R. 1970. Metabolism of long-chain fatty acids in the rumen. *Adv. Lipid Res.* 8:267-274.
- Whittemore, C. T. and R. H. Fawcett. 1976. Theoretical aspects of a flexible model to simulate protein and lipid growth in pigs. *Anim. Prod.* 22, 87-96.
- Wood, J. D., M. Enser, F. M. Whittington, C. B. Moncrieff, and A. J. Kempster. 1989. Backfat composition in pigs: differences between fat thickness groups and sexes. *Livestock Prod. Sci.* 22:351-362.
- Yacoob P., and P. Calder. 1995. Effects of dietary lipid manipulation upon inflammatory mediator production by murine macrophages. *Cell Immunol.* 163:120-128.

CHAPTER 3: PERFORMANCE, COMPOSITION AND QUALITY CHARACTERISTICS OF FINISHING BARROWS SUPPLEMENTED WITH CONJUGATED LINOLEIC ACID

A paper to be submitted to the Journal of Animal Science

J. C. Sparks, B. R. Wiegand, F. C. Parrish, Jr., and D. R. Zimmerman

Department of Animal Science, Iowa State University, Ames

Abstract: Conjugated linoleic acid (CLA), a collective term for geometric and positional isomers of linoleic acid, was supplemented to growing-finishing barrows ($n = 92$) at 0.75% of the diet. Growth, carcass, meat quality, physical, chemical, and sensory data were collected and analyzed. Treatments were defined by the amount of weight gain before slaughter while fed a CLA diet and included 0, 28, 57, and 86 kg of gain (T1, T2, T3 and T4, respectively). Average daily gain and feed intake were not affected by CLA supplementation. Gain:feed exhibited a quadratic response ($P < 0.05$) with T2 and T3 having the highest gain:feed. Loin muscle area increased ($P < 0.01$) linearly with increasing gain while fed CLA, and tenth rib, first rib and last rib fat depth decreased ($P < 0.02$) linearly. Subjective quality measures on loin muscles increased linearly for marbling ($P < 0.03$) and firmness ($P < 0.07$) with increasing weight gain on CLA. Objective Hunter color scores were not different for L^* or a^* values, but were higher ($P < 0.05$) for b^* values with CLA supplementation. Lipid oxidation values on loin muscle tissue were lower ($P < 0.05$) for CLA-supplemented pigs (T1 vs. T4). Fatty acid analysis showed a linear increase ($P < 0.0001$) for saturated fatty acids and CLA isomers in loin tissue and a linear increase ($P < 0.01$) for saturated fatty acids and CLA isomers in subcutaneous fat

tissue as weight gain on CLA increased. Sensory panel characteristics of loin chops were not changed by CLA supplementation. Increased gain:feed, increased loin muscle area, decreased fat depth, and improvements in marbling and firmness with CLA supplementation could result in more profitable pork production systems.

Keywords: Conjugated linoleic acid, Swine growth, Pork quality

Introduction

Current research is focusing on maintaining the genetic growth potential of pigs without compromising pork quality. One approach to improving pork quality is the supplementation of naturally occurring feed additives, such as conjugated linoleic acid (CLA), in the growing-finishing diet. Conjugated linoleic acid consists of positional and geometric isomers of linoleic acid, an 18:2 fatty acid. The c-9, t-11 isomer was thought to be the biologically active form of CLA because it is the only isomer found in the phospholipid portion of tissue (Ha et al., 1990). More recently, Park et al. (1999) reported increased muscle and decreased fat in rats with supplementation of the t-10, c-12 isomers. Conjugated linoleic acid consists has been shown to improve growth rates of rats, as well as serve as an anticarcinogenic compound when tumors were introduced into rats (Ip et al., 1991). Additionally, CLA has been may have an antioxidant effect (Ha et al., 1990). The autooxidation of CLA forms furan fatty acids that can protect cells against peroxide attack (Yurawecz, 1996). However, other research has disputed the antioxidant capabilities of CLA (Banni et al., 1998). The possibility of an antioxidant effect with CLA prompted us to examine the effects CLA might have on fatty acid oxidation and color stability

parameters of pork loins. The supplementation of CLA in growing-finishing barrows has been shown to improve growth and carcass characteristics (Cook et al., 1999; Thiel et al., 1998). The hypothesis tested in this study was that CLA at a constant level (0.75%) in the growing-finishing diet would improve feed efficiency, decrease subcutaneous carcass fat, increase color, flavor, and shelf life and decrease lipid oxidation in pork chops. Thus, our objectives were to investigate the changes in growth, carcass and meat quality characteristics caused by feeding CLA to growing-finishing pigs for varying body weight gains before slaughter.

Materials and Methods

Animals

This project was carried out in accordance with Iowa State University Animal Care and Use Committee Guidelines. Ninety-two Yorkshire x Landrace x Duroc x Hampshire barrows were randomly assigned from littermate groups to four treatment with five replications per treatment. Three replication had five pigs per pen and two replications had four pigs per pen. Pigs were housed in a total confinement, slatted floor facility in 20 adjacent pens. Treatment groups included a control diet at 0 kg (T1) and CLA supplementation for 28, 57, and 86 kg of body weight gain before slaughter (T2, T3, and T4, respectively). CLA-60 (Conlinco, Inc., Detroit Lakes, MN) was included at 1.25% and replaced soy oil. The CLA isomers, *cis*-9, *trans*-11 / *trans* - 9, *cis*- 11 and *trans*-10, *cis*-12 / *cis*-10, *trans*-12 made up 27% and 20% of the CLA, respectively. Diet composition is presented in Table 3.1. Average daily gain, average daily feed intake and gain:feed were calculated at the conclusion of the feeding trial. All pigs remained on their respective diets

until 115 kg of body weight, at which time they were humanely slaughtered at Hormel, Inc., in Austin, MN. Due to different growth rates, pigs were slaughtered in two groups, 30 d apart. Carcasses were chilled for 24 h postmortem before carcass measurements were taken. The left side of each carcass was ribbed between the 10th and 11th rib, and loin muscle area, 10th rib, 1st rib and last rib fat depth were measured. Percent lean was calculated using the 10th rib and loin muscle area according to the National Swine Improvement Federation. (1997).

Meat Laboratory Procedures and Analysis

At 24 h postmortem, whole bone-in loins were removed from the left side of each carcass. Loins (IMPS 410) were weighed, boxed, and shipped under refrigerated conditions to the Iowa State University Meat Laboratory. At 48 h postmortem, whole loins were subjectively evaluated for color, marbling, and firmness between the 10th and 11th rib face according to a five-point descriptive scale (NPPC, 1991). All T1 and T4 loins were cut into 2.54-cm chops. Chops were deboned and trimmed to 0.64 cm of subcutaneous fat. Chops were paired and placed in Viskase® vacuum bags. The vacuum packages were assigned to 1, 14, or 28 d of cold (2° C) storage. Three 1.27-cm chops were also cut from each loin for measurement of pH, water holding capacity (WHC), lipid oxidation (TBA), and proximate analysis. These chops were stored in vacuum under the same conditions as the 2.54-cm chops.

Lipid content was determined by hexane extraction using a Soxhlet method (AOAC, 1990). Moisture was determined as the weight difference in samples after 18 h in an 80° C drying oven.

At the appropriate day of storage (1, 14, or 28), chops were removed from vacuum packages, placed on styrofoam trays and overwrapped with oxygen-permeable polyvinyl chloride film. Chops were placed in a retail self-service case for 24 h after being repackaged. After the 24-h period, objective colors were measured with the Hunter Lab Color system (Hunter Associates Laboratory, Inc., Reston, VA) with a 2.54-cm objective. The L*, a*, and b* values were measured for the 1-, 14-, and 28-d storage. The WHC was measured by using the Carver Press Method (Kauffman et al., 1986) on 1, 14, and 28 d of storage. This method utilizes a 0.3-g sample, which is pressed at 3000 psi for 3 min on 125 mm diameter filter paper. The areas of the pressed sample and expressed moisture were traced on the filter paper. The areas of these tracings were determined with a planometer (Model K & E 4236, Keuffel-Esser Co., Germany). A ratio of water to meat areas was calculated giving a measure of WHC. The pH measurements were also made on each package of chops after 1, 14, and 28 d of storage with a Fisher Accumet 925 pH meter. The pH method utilized 10 g of homogenized muscle in 90 mL of distilled water. Duplicate pH samples were used for each chop.

Lipid oxidation was measured at 1, 14, and 28 d of storage by using thiobarbituric acid (TBA) and a distillation apparatus (Tarladgis et al., 1960). Malonaldehyde, a product of lipid oxidation, was measured with a Beckman DU 640 spectrophotometer (Fullerton, CA) at a wavelength of 532 nm.

Fatty acid profile of subcutaneous fat and loin muscle samples from the 10th and 11th rib junction was determined with gas chromatography. Lipids were extracted from the respective liquid nitrogen pulverized samples using the Folch extraction method. This

method utilized a 2 g and 0.5 g sample for loin tissue and adipose tissue, respectively, which was homogenized with a Polytron homogenizer in 10 mL chloroform:methanol at 2:1 (Folch 1) and 25 µl BHT (Aldrich Chemical Co.). The homogenized sample was placed in 50 mL tubes with teflon-lined caps and incubated for 2 h at 4° C. Samples were then filtered through Whatman #1 paper into a 100 mL graduated cylinder. The sample tube and paper were rinsed with Folch 1 solution. Twenty-five percent volume (of sample solution) of 0.88% NaCl was added to each cylinder and shaken 10 times to mix. The mixture was allowed to phase separate (2 to 4 h) and the lipid layer (bottom) was removed after the methanol/water (top layer) was removed by suction. Samples (10 mL) were transferred by pipet to scintillation vials. Vials were placed on a 50° C hot plate to evaporate the chloroform under a stream of nitrogen gas. The concentrated sample and vial were weighed.

Fatty acids were prepared for gas chromatography determination by using fatty acid methylation with sodium methoxide. One mL of hexane was added to each of the scintillation vials from the Folch extraction. Then 2 mL of sodium methoxide was added to each vial and the vials were vortexed at low speed for 30 sec. Vials were incubated in a heat block at 50° C for 10 min. Five mL of deionized water and 0.1 mL of glacial acetic acid were added to each vial. Lipids were extracted with two successive washings of 3 mL of hexane per vial. A small amount of anhydrous sodium sulfate was added to each vial to remove any residual water. One mL of the fatty acid methyl esters (FAME) was transferred to gas chromatography vials and stored at 4° C until analysis. All FAME were analyzed with a Varian 3350, Varian Chromatography Systems, Walnut Creek, CA. All

FAME were identified by comparison to their retention times with authentic standards (Nu-Chek-Prep, Elysian, MN). The GC was fitted with a 8200cx autosampler and a 60 m x 0.317 mm column (J & W Scientific, Folsom, CA). One μL of sample was injected onto the column with an injector temperature of 250° C and a detector temperature of 220° C.

Sensory characteristics were evaluated for the T1 and T4 chops. Chops were cooked in a broiler (General Electric Model CN02, Chicago Heights, IL) set at 176° C to 71° C internally and cut into 2.54-cm cubes for sensory evaluation. A panel consisting of 10 human subjects evaluated 28 d chops for tenderness, flavor, juiciness, off-flavor, and overall acceptability according to an eight-point descriptive scale (AMSA, 1995).

Statistical analysis

Statistical analysis was performed with the GLM procedure of SAS (1990). The statistical model included fixed effects of treatment and day of storage time when appropriate. The model for sensory analysis included fixed effects of treatment, and panelist. Pen was the experimental unit. Contrasts were used to determine linear and quadratic relationships for means with regard to treatment group. Data are presented as least squares means. Means were considered statistically different at $P < 0.05$.

Results and Discussion

Average daily gain and feed intake were not affected for any of the feeding groups. However, gain:feed exhibited a quadratic response ($P < 0.05$) with T2 and T3 groups having the highest values (Table 3.2). These results are in contrast to reports by Cook et al. (1999), where there was decreased feed intake in the first 49 d of feeding and a

compensatory gain from 49 d to 84 d for pigs consuming a CLA diet (4.8 g/kg or 9.5 g/kg CLA in the diet). Dugan et al.(1997) and Thiel et al. (1998) reported no differences for feed intake with CLA. However, Thiel et al. (1998) reported linear increases for average daily gain and feed efficiency with increasing levels of CLA at 0.12% to 1.0% in the diet.

Loin muscle area increased ($P < 0.01$) linearly with increasing time on a CLA diet. Additionally, 10th rib ($P < 0.01$), 1st rib ($P < 0.01$) and last rib ($P < 0.02$) fat thickness decreased linearly with increasing time on CLA. The calculated percent lean also, increased ($P < 0.01$) linearly with increasing time on a CLA diet. Cook et al. (1999) reported no differences in loin muscle area, but did observe decreases in backfat with increasing levels of CLA in the diet of pigs. A decrease in 10th rib fat but no differences in loin muscle area was also reported by Thiel et al. (1998).

Least squares means for subjective quality measures of color, marbling, and firmness are shown in Table 3.3. Linear treatment differences were observed for marbling ($P < 0.03$), where marbling scores increased with longer feeding time on CLA. Proximate analysis verified these marbling scores, with hexane-extractable lipid increasing ($P < .05$) with increasing time on CLA. Additionally, a linear trend ($P < 0.07$) was observed for firmness values, where firmness tended to increase with longer feeding time on CLA. In general, fat from CLA- supplemented pigs was firmer than fat from pigs fed control diet as determined by visual observation. This difference could be due to an increase in saturated fat in the CLA-supplemented pigs (Eggert et al., 1999). These increases in fat firmness and total intramuscular fat are thought to be an explanation for the increased subjective firmness scores. To test this hypothesis, the correlation between marbling and firmness

scores was determined. The correlation was high (0.93), confirming the previous hypothesis. No differences were observed for subjective color values ($P = 0.95$) when T1 and T4 chops were compared. These results are in contrast to those of Cook et al., (1999) who reported increases in subjective color scores and no difference for marbling or firmness scores.

Subjective values for color were objectively measured with the Hunter Color system (Table 3.4). Due to processing logistics, only loin chops from treatments 1 (control) and 4 (longest fed) were further measured for self-service shelf stability. When day of retail storage was included in the model, no statistical differences were observed between treatments at each day (1, 14, and 28). If day was removed from the model, and the T1 and T4 groups were compared, chops from CLA-supplemented pigs had higher b^* values ($P < 0.05$) compared with chops of pigs fed control diet. This higher b^* value corresponds to a more yellow product, which is a deviation from the desirable grayish pink or reddish pink color of pork. Additionally, CLA chops tended to have higher L^* and a^* values compared with control chops. These values correspond to lighter and redder products, respectively. Thiel et al., (1998) previously reported increases in a^* values with increasing levels of CLA, from 0.12% to 1.0% in the diet, suggesting that dietary CLA may protect meat color.

Values for pH, WHC and lipid oxidation (TBA) are presented in Table 3.5. Least squares means for pH were not different for treatment groups at 1, 14, and 28 d of retail storage. Means ranged from 5.70 and 5.80 indicating acceptable pork quality was observed for control and treated groups. These pH values were consistent with normal

pork pH values. of to high pork quality as reported in the literature (Bendall and Swatland, 1989). WHC, the ability of meat to bind water, is related to juiciness and cooking attributes of pork (Hamm, 1960). WHC ratio values, a smaller ratio indicating a higher WHC, did not differ between treatments, but tended to decrease over time of retail storage indicating and improvement in WHC over storage time. The reason for improved WHC over time of storage is yet unresolved.

Results shown in Table 3.5 indicate that lipid oxidation, based on TBA values, was lower ($P < 0.05$) for 1-d chops from CLA-supplemented pigs compared with chops from pigs fed control diet. TBA values were not different for 14-d and 28-d chops when T1 (control) and T4 (longest fed) diets were compared. These TBA values were well below 0.5 to 1.0 mg malonaldehyde/kg of tissue, a value that might indicate an unacceptable product (Tarladgis et al., 1960; Green and Cumuze, 1981). The low TBA values in loin samples from CLA-fed pigs at 1 d may be because of a decrease in arachadonic acid and an increase in saturated fatty acids in the loin tissue samples from CLA-fed pigs (Table 3.6). This is significant because arachadonic acid (20:4) is more prone to lipid oxidation compared with saturated fatty acids because the double bonds in its structure are subject to free radical attack. Thus, the TBA test only detected small levels of volatiles from lipid oxidation.

Fatty acid profile of loin muscle tissue between the 10th and 11th rib and subcutaneous fat samples are shown in Tables 3.6 and 3.7. The GC analysis of fatty acids showed a linear decrease ($P < 0.0008$) in polyunsaturated fatty acids (PUFA), a linear increase ($P < 0.0001$) in saturated fatty acids (SFA), quadratic decrease in oleic acid ($P <$

0.004) and a linear increase ($P < 0.01$) in total CLA isomers. These data verify previous reports with regard to fatty acid profile changes in loin muscle because of CLA supplementation (Eggert et al., 1998). Fatty acid analysis of subcutaneous fat samples from the 10th and 11th rib junction showed a linear increase ($P < 0.0001$) in SFA, quadratic decrease in oleic acid ($P < 0.03$) and a linear increase ($P < 0.0001$) in total CLA isomers. One would likely expect an increase of CLA in pork tissue when dietary CLA is provided, because monogastrics tend to absorb fatty acids in the form in which they are consumed. The incorporation of CLA isomers into muscle and fat tissue may prove positive in terms of making pork a “functional food” with respect to CLA intake and potential decreases in cancer and heart disease.

Sensory panel results are summarized in Figure 1. No significant differences were observed between control and CLA loin chops, based on an eight-point descriptive scale. All samples were acceptable for tenderness, juiciness, flavor intensity, and overall flavor. These results indicate that supplementation of CLA to pigs does not affect sensory characteristics of pork loin chops.

Implications

Improved feed efficiency, increased loin muscle area, and decreased fat thickness as a result of feeding CLA should help pork producer to be more efficient. Improvements in marbling and firmness scores might result in increased premiums or decreased price deductions for producers selling in a value-based system. Other research should be done to identify mechanistic control of CLA responses in growing-finishing pigs.

Literature Cited

- AMSA. 1995. Research Guidelines for Cookery, Sensory Evaluation and Instrumental Tenderness Measurements of Fresh Meat. National Livestock and Meat Board. Chicago, IL.
- AOAC. 1990. Official Methods of Analysis (15th ed.). Association of Official Analytical Chemists, Arlington, VA.
- Banni, S., E. Angioni, M. S. Contini, G. Carta, V. Casu, G. A. Iengo, M. P. Melis, M. Deiana, M. A. Dessi, and F. P. Corongiu. 1998. Conjugated linoleic acid and oxidative stress. *J. Am. Oil Chem. Soc.* 75:261-267.
- Bendall, J. R. and H. J. Swatland. 1989. A review of the relationship of pH with physical aspects of pork quality. *Meat Sci.* 40:85-126.
- Cook, M. E., D. L. Jerome, T. D. Crenshaw, D. R. Buege, M. W. Pariza, K. J. Albright, S. P. Schmidt, J. A. Scimeca, P. A. Lofgren, and E. J. Hentges. 2000. Feeding conjugated linoleic acid reduces carcass fat in pigs. *J. Lipids* (submitted).
- Dugan, M. E. R., J. L. Aalhus, A. L. Shaefer, and J. K. G. Kramer. 1997. The effect of conjugated linoleic acid on fat to lean repartitioning and feed conversion in pigs. *Can. J. Anim. Sci.* 77:1045-1050.
- Eggert, J. M., M. A. Belury, A. Kempa-Steczko and A. P. Schinckel. 1999. Effects of conjugated linoleic acid (CLA) on growth and composition of lean gilts. *J. Anim. Sci.* 77:53.
- Green, B. E., and T. H. Cumuze. 1981. Relationship between TBA numbers and inexperienced panelists' assessments of oxidized flavor in cooked beef. *J. Food Sci.* 47:52-54.
- Ha, Y. L., J. Storkson, and M. W. Pariza. 1990. Inhibition of benzo(a)pyrene-induced mouse forestomach neoplasia by conjugated dienoic derivatives of linoleic acid. *Cancer Res.* 50:1097-1101.
- Hamm, R. 1960. Biochemistry of meat hydration. *Adv. in Food Res.* 10:355-443.
- Ip, C., S. F. Chin, J. A. Scimeca, and M. W. Pariza. 1991. Mammary cancer prevention by conjugated dienoic derivatives of linoleic acid. *Cancer Res.* 51:6118-6124.
- Kauffman, R. G., G. Eikelenboom, P. G. van der Wal, B. Engel, and M. Zaar. 1986. A comparison of methods to estimate water-holding capacity in post-rigor porcine muscle. *Meat Sci.* 18:307-322.
- National Swine Improvement Federation. 1997. Guidelines for uniform swine improvement programs. p. 24.
- NPPC. 1991. Procedures to Evaluate Market Hogs. 3rd Ed. National Pork Producers Council, Des Moines, IA.
- Park, Y., J. M. Storkson, K. J. Albright, W. Liu and M. W. Pariza. 1999. Evidence that the trans-10, cis-12 isomer of conjugated linoleic acid induces body composition changes in mice. *Lipids* 34:235-241.
- SAS. 1990. SAS User's Guide: Statistics (Version 6 Ed.) SAS Inst. Inc., Cary, NC.

- Tarladgis. B. G., B. M. Watts, M. T. Younthan, and L. R. Dugan, Jr. 1960. J. Am. Oil Chem. Soc. 37:44.
- Thiel, R. L., J. C. Sparks, B. R. Wiegand, F. C. Parrish, Jr., and R. C. Ewan. 1998. Conjugated linoleic acid improves performance and body composition in swine. J. Anim. Sci. 76:57 (Suppl. 2).
- Yurawecz, M. P. 1996. Conjugated linoleic acid: Researcher examining isomer's metabolic effects. INFORM 7(2):152-159.

Table 3.1. Diet composition and calculated analysis

Ingredient	Body weight range, kg		
	28 to 57	57 to 86	86 to 115
Corn	68.76	83.47	85.01
Soybean meal	27.38	12.99	11.76
Dicalcium phosphate	1.24	0.82	0.57
Calcium carbonate	0.82	0.77	0.77
Salt	0.25	0.25	0.25
Vitamin premix ^a	0.20	0.20	0.20
Trace mineral premix ^b	0.05	0.05	0.05
Tylosin premix ^d	0.05	0.05	0.05
Lysine·HCl	0.00	0.15	0.09
Oil ^c	1.25	1.25	1.25
Total	100.00	100.00	100.00
Calculated analysis:			
ME, kcal/kg	3369	3382	3395
Lysine, ‰	1.00	0.73	0.65
Calcium, ‰	0.70	0.58	0.50
Phosphorus, ‰	0.60	0.47	0.42

^a At 0.2‰ of diet contributes per kilogram of diet: 4,400 IU vitamin A; 22 IU vitamin E; 1,100 IU vitamin D₃; 6.6 mg riboflavin; 17.6 mg pantothenic acid; 33 mg niacin; 22 µg vitamin B₁₂.

^b At 0.05‰ of the diet contributes in ppm: 75 Zn, 870.5 Fe, 30 Mn, 8.75 Cu, 0.1 I.

^c Soybean oil or CLA oil in their respective treatments.

^d Supplied tylosin at 40 mg/kg of diet.

Table 3.2. Effects of conjugated linoleic acid on growing-finishing pig

Item	Weight(kg)	Final weight gain while fed CLA. kg				P-value		SEM
		0 (T1)	29 (T2)	58 (T3)	87 (T4)	Linear	Quadratic	
ADG. g								
	28 to 57	855	834	869	828	0.60	0.61	18.4
	57 to 86	942	958	1002	968	0.30	0.32	25.1
	86 to 115	980	1003	966	950	0.45	0.61	36.6
	28 to 86	894	890	929	890	0.71	0.36	17.9
	28 to 115	924	929	942	912	0.77	0.33	17.3
ADFI. g								
	28 to 57	2126	2024	2117	2055	0.46	0.57	34.9
	57 to 86	3007	2967	2917	2876	0.06	0.98	47.4
	86 to 115	3306	3287	3297	3292	0.95	0.95	117.0
	28 to 86	2521	2447	2476	2424	0.08	0.73	31.1
	28 to 115	2791	2737	2759	2724	0.47	0.86	53.9
G:F. g/kg								
	28 to 57	402	413	411	404	0.92	0.30	7.6
	57 to 86	314	323	345	336	0.05	0.30	8.8
	86 to 115	296	306	293	288	0.20	0.29	6.4
	28 to 86	355	364	376	367	0.20	0.55	5.5
	28 to 115	331	340	342	335	0.44	0.05	3.5
Hot carcass weight. kg ^a		84.4	84.4	85.3	83.5	0.93	0.95	3.4
Backfat. mm								
	10th rib	26.2	22.4	22.4	20.8	0.01	0.15	0.42
	1st rib	47.0	43.2	42.9	41.7	0.05	0.28	0.06
	Last rib	24.6	22.1	21.6	21.6	0.02	0.08	0.04
Loin muscle area. cm ²		39.1	41.7	43.4	42.7	0.01	0.17	0.15
Percent lean		50.0	52.5	53.2	53.7	0.01	0.17	0.69

^a Hot carcass weight was used as a covariant for all backfat depth and loin muscle area measurements.

Table 3.3. Least squares means^a and standard errors for subjective scores^b of loin color, marbling, firmness and percentage lipid at differing weight gain on CLA

Treatment	Color	Marbling	% Lipid	Firmness
T1 (0 kg)	2.43	2.04 ^c	4.02 ^c	2.36
T2 (29 kg)	2.31	2.18 ^d	4.40 ^d	2.27
T3 (58 kg)	2.47	2.35 ^c	4.76 ^c	2.45
T4 (87kg)	2.38	2.31 ^c	5.06 ^c	2.49
SEM	0.10	0.10	0.09	0.06

^a Values within a column with different superscripts are significant at $P < 0.05$.

^b Based on National Pork Producer Council 5-point scale.

Table 3.4. Least squares means^a for Hunter L*, a* and b* values of overwrapped loin chops independent of storage day

Treatment ^b	L*	a*	b*
T1	51.53	6.16	11.76 ^c
T4	52.18	6.53	12.15 ^d
SEM	0.34	0.17	0.09

^a Values within columns with different superscripts significant at $P < 0.05$.

^b T1 = 0 kg gain on CLA. T4 = 86 kg gain on CLA.

Table 3.5. Least squares means and standard errors for pH, water holding capacity (WHC) and TBA values of loin chops at 1, 14 and 28 days of self-service case storage

Treatment ^a	Day	pH	WHC	TBA ^b
T1	1	5.70	3.10 ^c	0.098 ^c
T4	1	5.70	3.03 ^c	0.081 ^d
T1	14	5.80	2.77 ^{cd}	0.098 ^c
T4	14	5.79	2.92 ^{cd}	0.158 ^c
T1	28	5.77	2.59 ^d	0.187 ^f
T4	28	5.76	2.39 ^d	0.132 ^e
SEM		0.02	0.13	0.003

^a T1 = 0 kg gain on CLA. T4 = 86 kg weight gain on CLA.

^b TBA values expressed as mg malonaldehyde/kg of sample.

^{c-e} Means with different letter within a column are significant at $P < 0.05$.

Table 3.6. Fatty acid profile (%)^a of loin tissue from 10th and 11th rib junction

Fatty Acid	Final weight gain while fed CLA, kg				P-value		SEM
	0	29	58	87	Linear	Quadratic	
14:0	1.53	1.88	2.03	2.03	0.001	0.07	0.09
16:0	30.39	33.18	35.49	36.70	0.0001	0.24	0.63
18:0	13.67	14.70	15.02	14.27	0.11	0.007	0.27
18:1	44.88	40.27	38.82	38.99	0.0001	0.004	0.68
18:2	8.22	8.74	7.78	6.52	0.002	0.03	0.36
18:3	0.27	0.23	0.16	0.12	0.001	0.89	0.03
20:4	1.03	0.41	0.34	0.66	0.03	0.001	0.11
CLA ^b	0.00	0.58	0.35	0.71	0.01	0.48	0.15
SFA ^c	45.60	49.76	52.54	53.00	0.0001	0.03	0.77
PUFA ^d	9.53	9.39	8.29	7.30	0.0008	0.29	0.39

^a Expressed as a ratio of shown fatty acids of sample injected on gas chromatograph column.^b Conjugated linoleic acid isomers.^c Saturated fatty acids is sum of 14:0, 16:0, and 18:0.^d Polyunsaturated fatty acids is sum of 18:1, 18:2, 18:3, and 20:4.**Table 3.7. Fatty acid profile (%)^a of subcutaneous fat tissue from 10th and 11th rib junction**

Fatty Acid	Final weight gain while fed CLA, kg				P-value		SEM
	0	29	58	87	Linear	Quadratic	
14:0	1.67	2.29	2.77	3.00	0.0001	0.08	0.11
16:0	27.84	31.35	30.34	32.51	0.01	0.52	1.00
18:0	13.98	15.56	17.16	17.82	0.003	0.58	0.82
18:1	39.17	31.81	30.36	27.53	0.0001	0.03	0.94
18:2	15.55	15.12	14.63	14.74	0.28	0.65	0.58
18:3	0.93	0.79	0.59	0.53	0.0001	0.56	0.06
20:4	0.37	0.63	0.53	0.26	0.64	0.22	0.21
CLA ^b	0.50	2.45	3.61	3.62	0.0001	0.008	0.30
SFA ^c	43.49	49.19	50.27	53.33	0.0001	0.23	1.07
PUFA ^d	16.84	16.54	15.76	15.52	0.08	0.95	0.56

^a Expressed as a ratio of shown fatty acids of sample injected on gas chromatograph column.^b Conjugated linoleic acid isomers.^c Saturated fatty acids is sum of 14:0, 16:0, and 18:0.^d Polyunsaturated fatty acids is sum of 18:1, 18:2, 18:3, and 20:4.

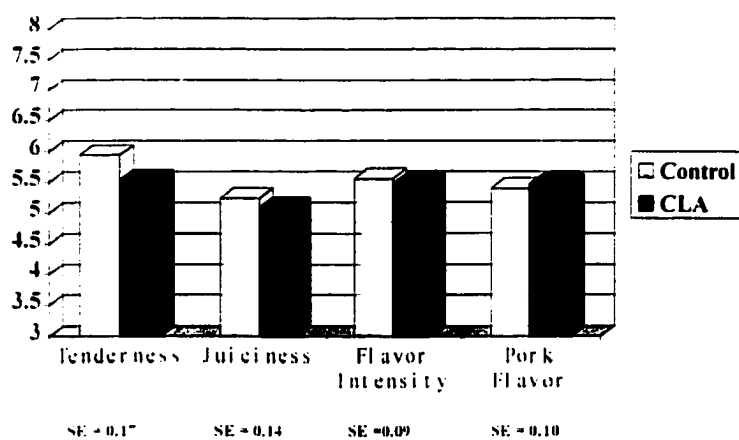


Fig 3.1. Mean sensory attributes of CLA and control pork loins at 1 d shelf storage.

CHAPTER 4: EFFECTS OF CONJUGATED LINOLEIC ACID AND HIGH OIL CORN ON GROWTH PERFORMANCE AND PORK QUALITY IN FINISHING PIGS

A paper to be submitted to the Journal of Animal Science

**J. C. Sparks, B. R. Wiegand, J. E. Swan, S. T. Larsen, F. C. Parrish,
and D. R. Zimmerman**

Department of Animal Science, Iowa State University, Ames

Abstract: Forty-eight individually-penned barrows weighing 55 kg were fed diets containing normal corn (NC), NC and conjugated linoleic acid (CLA) (NC+CLA), high oil corn (HOC), HOC and CLA (HOC+CLA), NC and choice white grease (CWG) (NC+CWG) and NC, CWG and CLA (NC+CWG+CLA). CLA-60 (60% CLA) was included at 1.25% and replaced soy oil. NC and NC + CLA diets (low energy, LE) contained 3.3 Mcal ME/kg, whereas all other diets (high energy, HE) contained 3.5 Mcal ME/kg. Diets had equal calculated lysine:ME ratios. Ultrasound measurements of backfat thickness (BF) and loin muscle area (LMA) were determined at body weights of 55, 90 and 113 kg. Pigs were slaughtered at 113 kg. Pigs fed CLA had lower ADG ($P < 0.05$) for the first 28 d. However, by the end of the trial there was no effect ($P < 0.28$) of CLA. For the first 42 d, pigs fed HE diets had greater ($P < 0.05$) gain/feed (GF) than pigs fed LE diets. For the overall trial there was no difference in GF between pigs fed diets containing HOC and pigs fed LE. However, pigs fed diets containing CWG had a greater ($P < 0.05$) GF than pigs fed diets containing HOC. For the overall trial, pigs fed CLA had reduced ($P < 0.05$) GF. CLA improved GF of pigs fed HOC but it did not improve GF of pigs fed diets containing CWG (interaction, $P < 0.01$). Pigs fed the LE diets had less ($P < 0.05$) 10th

rib BF and greater LMA as measured by ultrasound at 90 kg ($P < 0.05$) and just before slaughter ($P < 0.03$) than those fed HE diets, resulting in a greater ($P < 0.001$) percent lean at 90 kg and slaughter. Carcasses of pigs fed LE diets had less ($P < 0.04$) BF at the last lumbar and 10th rib than those of pigs fed HE. Bellies from pigs fed CLA were firmer ($P < 0.001$) than bellies from those fed other treatments. Bellies of pigs fed the **HOC** were softer ($P < 0.03$) compared with bellies of pigs fed the **NC**, but the pigs fed **HOC+CLA** had bellies that were slightly firmer ($P < 0.13$) than those from pigs fed **NC** diet, but less ($P < 0.03$) firm than those from pigs fed **NC+CLA**. Fatty acid analysis of the loin tissue showed a increase ($P < 0.0001$) in saturated fatty acids and a decrease ($P < 0.0001$) in polyunsaturated fatty acids for pigs fed CLA. In summary, CLA had no effect on ADG, ADFI or GF, but did increased belly firmness, thus correcting the negative effect on firmness caused by feeding a high level of polyunsaturated fatty acids found in high oil corn.

Keywords: pig, conjugated linoleic acid, high oil corn

Introduction

Incorporation of high oil corn (HOC) in the diets of market pigs is beneficial to swine producers because feed efficiency (Bowers et al., 2000) and dust control are improved and handling and mixing of oil are user friendly. Because of the high energy density of diets containing HOC, feeding HOC is particularly beneficial for young pigs, for lactating sows and for all pigs during heat stress (Stahly and Cromwell, 1979). A potentially detrimental result of feeding HOC, however, is that of producing soft carcass fat and soft bellies because of the high proportion of unsaturated fatty acids in HOC diets

(Skelly et al., 1975). Unsaturated fats are susceptible to oxidation (Meynier et al., 1999), and as a consequence, there is also potential for shorter shelf life of pork products from pigs fed HOC. Soft pork bellies result in reduced sliceability and yield of bacon (Shackelford et al., 1990). Reduced shelf life manifested in undesirable lean color, aroma, and flavor of retail cuts of pork, and decreased sliceability and yield of bacon because of soft fat could cause significant economic loss to the pork industry. Supplementation of the diet with conjugated linoleic acid (CLA) is a possible method of improving shelf life and belly yield of pigs fed HOC. Earlier results have demonstrated that bellies from pigs fed CLA are firmer than those from pigs fed control diets (Theil et al., 1998). These results indicate that CLA might be a valuable addition to pig diets that contain HOC. Therefore, pork quality benefits could be derived from the supplementation of CLA in HOC diets by making pork bellies firmer, and pork cuts more consumer acceptable. The purpose of the work reported here was to test the hypothesis that the inclusion of CLA in a finisher pig diet containing HOC would improve pork quality and growth performance.

Materials and Methods

Animals

Forty-eight individually-penned PIC Cambrough 22 barrows weighing 55 kg were randomly allotted within outcome groups based on weight to dietary treatments containing normal corn (NC), NC and conjugated linoleic acid (CLA) (NC+CLA), high oil corn (HOC), HOC and CLA (HOC+CLA), NC and choice white grease (CWG) (NC+CWG) and NC, CWG and CLA (NC+CWG+CLA). CLA-60 (Conlinco, Inc., Detroit Lakes, MN) was included at 1.25% and replaced soy oil. The CLA isomers, *cis*-9, *trans*-11 /

trans-9, *cis*-11 and *trans*-10, *cis*-12 / *cis*-10, *trans*-12 made up 27% and 20% of the CLA, respectively. NC and NC+CLA diets (low energy, LE) contained 3.3 Mcal ME/kg, whereas all other diets (high energy, HE) contained 3.5 Mcal ME/kg. Experimental diets were formulated to have equal ratios of metabolizable energy to lysine between the LE diets and HE diets. Calculated and analyzed composition of the dietary treatments are shown in Tables 4.1 and 4.2.

Feed intake and weight gain were measured every 2 weeks. Ultrasound measurements of backfat depth and loin muscle area between the 10th and 11th rib were determined at initiation of the experiment, on d 28 and just before slaughter. Carcass lean was calculated from ultrasound loin muscle area and back fat measurements according to National Swine Improvement Federation, (1997). Pigs were slaughtered at 113 kg.

Carcass measurements

Animals were slaughtered at the Iowa State Meat Laboratory. At slaughter, carcasses and leaf fat were weighed. At 24-h post mortem, carcasses were fabricated into primal cuts, including ham, loin, belly, picnic shoulder and Boston butt. Carcasses were cut between the 10th and 11th ribs and loin muscle area and back fat depth at the 10th rib, first rib, last rib, and last lumbar vertebra were measured. Subjective scores for color, marbling, and firmness were taken on each loin at the 10th rib. Loins were boned and cut into chops. Starting at the 10th and 11th rib junction, chops were cut in alternating thickness for samples for specific pork quality measures. The first chop was 0.63 cm in thickness followed by two 2.54-cm chops. This alternating method was used for all the loin to get representative samples of the entire loin. The 2.54-cm chops were paired and

wrapped on styrofoam trays with oxygen permeable polyvinyl overwrap. All chops were held at 2°C for 1, 7, 14, and 21 d. At each representative day, a package of chops was measured for lean color and pH. Hams were mechanically skinned and physically dissected by knife into lean, separable fat, and bone. Bellies were removed and measured for firmness by measuring the distance between ends of a belly suspended over a horizontal bar were measured with both the fat and the lean side of the belly in the up position. An average of the left and right side bellies were used for analysis.

Meat Laboratory Procedures and Analysis

At 24 h postmortem, whole bone-in loins were removed from the left side of each carcass. Chops were deboned and trimmed to 0.64 cm of subcutaneous fat. Chops were paired and placed in Viskase® vacuum bags. The vacuum packages were assigned to 0, 1, or 2 d of cold (2°C) storage. Three 1.27-cm chops were also cut from each loin for measurement of pH and proximate analysis. These chops were stored in vacuum under the same conditions as the 2.54-cm chops.

Lipid content was determined by hexane extraction using a Soxhlet method (AOAC, 1990). Moisture was determined as the weight difference in samples after 18 h in an 80°C drying oven.

At the appropriate day of storage (0, 1, or 2), chops were removed from vacuum packages and placed on styrofoam trays and overwrapped with oxygen-permeable polyvinyl chloride film. Chops were placed in the retail self-service case for 24 h after being repackaged. After the 24-h period, objective colors were measured with the Hunter

Lab Color system (Hunter Associates Laboratory, Inc., Reston, VA) with a 2.54-cm objective. L^* , a^* , and b^* values were measured for the 0-, 1-, and 2-d storage times.

A pH measurement was also made on each package of chops at all 3 d (0, 1, and 2) of storage with a pH Star 5000 fitted with a glass probe (SFK Technology, Inc. Cedar Rapids, IA).

Myoglobin concentration of loin muscle samples from the 10th and 11th rib junction was determined with spectrophotometer. Three grams of pulverized raw sample was blended in Waring blender with 30 mL of cold buffer (40 mM potassium phosphate, pH 6.8) and held at 4° C for 1 h in a centrifuge bottle, then centrifuged for 15 min at 25000 x g at 4° C. The supernatant was poured off and syringe filtered through Whatman No. 1 filter paper into a 2 mL cuvette. Filtered supernatant was scanned in a scanning spectrophotometer at 418 nm absorbency. Myoglobin was calculated by taking the absorbance of the soret peak (416 to 418nm) (Abs_{418}) and the following formulas: $Abs_{418} / e * b =$ molar concentration of myoglobin, where $e = 128,000$ g for oxymyoglobin b and b = length of the cuvette. Molar concentration of myoglobin x molecular weight of myoglobin = g / L of myoglobin. Gram / L of myoglobin x number of L of buffer added to pulverized sample x 1000 / g of sample blended = mg of myoglobin / g of tissue.

Fatty Acid Profile

Fatty acid profile of loin muscle samples from the 10th and 11th rib junction was determined with gas chromatography. Lipids were extracted from the respective samples using the Folch extraction method. This method utilized 2 g of tissue which was homogenized with a Polytron homogenizer in 10 mL chloroform:methanol at 2:1 (Folch 1)

and 25 μ l BHT (Aldrich Chemical Co.). The homogenized samples were placed in 50 mL tubes with teflon-lined caps and incubated for 2 h at 4° C. Samples were then filtered through Whatman #1 paper into 100 mL graduated cylinders. The sample tube and paper were rinsed with Folch 1. Twenty-five percent volume (of sample solution) of 0.88% NaCl was added to each cylinder and shaken 10 times to mix. The mixture was allowed to phase separate (2-4 h) and the lipid layer (bottom) was removed after the methanol/water (top layer) was suctioned off. Samples (10 mL) were transferred by pipet to scintillation vials. Vials were placed on a 50° C hot plate to evaporate the chloroform under a stream of nitrogen gas. The concentrated sample and vial were weighed.

Fatty acids were prepared for gas chromatography determination by using fatty acid methylation with sodium methoxide. One mL of hexane was added to each of the scintillation vials from the Folch extraction. Then 2 mL of sodium methoxide was added to each vial and the vials were vortexed at low speed. Vials were incubated in a heat block at 50° C for 10 min. Five mL of deionized water and 0.1 mL of glacial acetic acid were added to each vial. Lipids were extracted with two successive washings of 3 mL of hexane per vial. A small amount of anhydrous sodium sulfate was added to each vial to remove any residual water. One mL of the fatty acid methyl esters (FAME) was transferred to gas chromatography vials and stored at 4° C until loading on the gas chromatograph (GC) (HP 6890, autosampler 6890: Hewlett-Packard Co., Avondale, PA) equipped with an HP 19091J-413 column (30 m, 320 μ m film thickness: Hewlett – Packard Co., Avondale PA) and operated at 180° C for 0.5 min. (temperature programmed 2° C / min to 230° C and held for 4.5 min). The injector and flame-ionization detector

temperatures were both set at 300 °C. All FAME were identified by comparison of their retention times with an authentic standard (UC-59 MX, Nu-Check-Prep, Elysian, MN), and were verified with mass spectrophotometry. Fat extractions were done in duplicates and averaged. All FAME and CLA results are presented as a ratio of fatty acids analyzed by the GC.

Statistical analysis

Data were analyzed using SAS GLM procedures (1990) as a randomized complete block design with initial weight as the blocking variable. The individual pig served as the experimental unit and treatment differences were separated by appropriate single degree of freedom contrast statements. Hot carcass weight was used as a covariant for all wholesale cut weights, backfat depth, and loin muscle area.

Results

Growth performance data

Animals fed CLA showed a decline in ADG ($P < 0.05$) for the first 28 days, however, by the end of the trial there was no effect ($P > 0.28$) of CLA (Table 4.3). For the period of 49 to 56 days, when only half of the pigs remained on test, the pigs fed the lower energy diets had a greater ADG ($P < 0.02$) and ADFI ($P < 0.03$), (Table 4.4) than those fed the higher energy diets. During the second 2-week period, pigs fed diets containing CWG had a lower ADFI than pigs fed other treatments, which resulted in pigs fed diets containing CWG having a higher GF ratio ($P < 0.05$), (Table 4.5). Also, for the first 42 days, pigs fed diets with higher energy had a greater GF ratio ($P < 0.05$) than pigs fed

lower energy diets. The advantage of a higher GF ratio for pigs fed diets containing HOC over pigs fed LE diets was not seen in the overall trial. Also, pigs fed diets containing CWG had a greater ($P < 0.05$) GF than did pigs fed diets containing HOC diets. A main effect of CLA was a reduction ($P < 0.05$) in GF, which is in contrast to past experiments in which GF was increased by CLA (Thiel et al., 1998; Wiegand et al., 2000). Also, an interaction ($P < 0.01$) in overall GF was seen between corn source and CLA for pigs fed HE diets. Pigs fed diets containing HOC showed a benefit in GF from the addition of CLA whereas pigs fed diets containing CWG did not show the same effect from the addition of CLA.

Ultrasound data

Initial measurements (55 kg body weight) showed that pigs assigned to the lower-energy treatments had greater LMA ($P < 0.03$) but were not different for 10th rib (Table 4.6). Pigs fed the LE treatments had a lower 10th rib back fat ($P < 0.05$) and greater LMA at day 28 ($P < 0.05$) and just before slaughter ($P < 0.03$). The reduced backfat and greater LMA resulted in pigs fed the LE diets having the greatest ($P < 0.001$) percent lean (NSIF, 1997) at day 28 and at slaughter.

Carcass characteristics

Pigs fed LE diets had less backfat at the last lumbar ($P < 0.04$) and 10th rib ($P < 0.04$) than pigs fed other treatments (Table 4.7). An interaction ($P < 0.04$) was seen between corn source and CLA for leaf fat weight. The interaction seems to have been caused by an unusual decrease in leaf fat for pigs fed the NC+CWG diet. The decrease in

backfat of pigs fed NC is explained by lower energy intake. The decrease in leaf fat was not expected because it would be expected that an increase in energy intake would increase leaf fat. Therefore, the decrease of leaf fat of pigs fed the NC+CWG cannot be explained. Loins from pigs fed the LE diets had higher subjective color scores ($P < 0.05$) at 24-h post mortem than loins from pigs fed other treatments.

A trend ($P > 0.11$) for pigs fed CLA to have lighter hot carcasses was seen when weighing intact hot carcasses (Table 4.7). This trend was found to be significant ($P < 0.05$) for the cold halved carcasses (Table 4.8). Pigs fed CLA also had lighter ($P < 0.05$) shoulder weights. Pigs fed CLA had heavier loin weights ($P < 0.05$) when fed the HE diet, whereas there was no effect of CLA for pigs fed the LE diets. An interaction ($P < 0.05$) between CLA and energy density was seen for belly weights, with pigs fed LE having a greater belly weight when fed CLA.

Ham weights were not affected by any treatment, but skin weights of the ham were decreased ($P < 0.01$) and separable fat weights were increased ($P < 0.01$) in pigs fed CLA (Table 4.9). These effects may be explained by the harder fat of pigs fed CLA, resulting in more efficient skin-fat separation by the skinning machine. When fat and skin weights were added together, there were no effects of CLA.

Bellies from pigs fed CLA were firmer ($P < 0.001$) than those from pigs fed other treatments, as indicated by the belly bar test (Table 4.10). The firmness of the bellies is the one measure that has been most consistently affected by feeding CLA. Averages of left and right side bellies of pigs fed the HOC diet were softer ($P < 0.03$) compared with bellies of pigs fed the NC diet, but the HOC+CLA-fed pigs had bellies that were slightly firmer

than those fed the NC diet but less firm than those fed the NC+CLA diet. These results suggest that CLA improves belly quality (firmness) of pigs fed high oil corn.

The fatty acid composition data confirm the belly bar test data. The fatty acid composition of the loin tissue of pigs fed CLA (Table 4.11) had an increase ($P < 0.0001$) in percent saturated fatty acids (SFA) and a decrease in percent oleic acid ($P < 0.0001$), and in ($P < 0.0001$) polyunsaturated fatty acids (PUFA). The increase in SFA for pigs fed CLA was consistent among all SFA. There was a decrease ($P < 0.004$) in linoleic acid in loin tissue for pigs fed the LE compared with all other treatments. For SFA, there was an interaction ($P = 0.01$) between loin tissue of pigs fed LE diets and loin tissue of pigs fed diets containing HOC, with loin tissue from pigs fed the HOC having a larger increase in SFA when fed CLA. Loin tissue of pigs fed diets containing HOC had a decrease in linolenic acid compared with loin tissue of pigs fed LE ($P < 0.0001$) and loin tissue of pigs fed diets containing CWG ($P < 0.0001$). Loin tissue from pigs fed LE diets had an increase ($P < 0.03$) in oleic acid compared to all other treatments. There also was an increase ($P < 0.0001$) percent CLA in loin tissue of pigs fed CLA.

Loins from pigs fed CLA had greater L^* values for loin on day 0 ($P < 0.05$), 1 ($P < 0.05$), and 2 ($P < 0.05$), and their b^* values were lower for days 0 ($P < 0.05$) and 1 ($P < 0.05$) for pigs fed CLA than for pigs fed other treatments. No differences between treatment groups were seen in a^* values, and this was confirmed by the myoglobin values (Table 4.12).

Discussion

All growth rates were exceptionally good and it is difficult to explain why pigs fed the NC+CWG diet grew at a faster rate throughout the trial than other treatment groups. The lack of a higher GF response for pigs fed a high energy density diet such as the HOC diet over a lower energy diet is difficult to explain, however, this result is not unprecedented (Pettigrew and Moser, 1991), especially when the animals are not heat stressed (Stahly and Cromwell 1979). Therefore, had the rooms been kept at a temperature that resulted in a heat stress, the pigs fed HE diets may have performed better than the pigs fed LE diets. However, the increase in backfat of pigs fed HE does agree with the findings in the review by Pettigrew and Moser (1991).

In this experiment there were no differences in marbling or firmness of LMA as a result of CLA supplementation. In previous experiments (Thiel et al., 1998 and Wiegand et al., 2000), there were increases in marbling and firmness as a result of feeding CLA, and these two variables were highly correlated. The main change in protocol between those studies and the one reported here has been a change in genetics of the pigs. Therefore, the most likely reason for the differences in results for marbling and firmness is this change in genetics. The change in genetics may also explain the difference in magnitude of back fat change in response to feeding CLA. In the previous experiments there was a more dramatic effect of CLA on back fat depth than was seen in this experiment. Previous experiments here at Iowa State University utilized a moderately lean pig and the current experiment utilized a leaner genetic strain.

The most consistent effect of feeding CLA is its effect on belly firmness. In general, fat from CLA- supplemented pigs was firmer than fat from pigs fed control diets as determined by visual observation. This effect seems to be the result of an increase in saturated and a decrease in unsaturated fatty acids caused by feeding CLA. In this experiment, the inclusion of HOC in the diet resulted in softer bellies, but feeding CLA to pigs fed HOC increased all SFA and decreased all unsaturated fatty acids resulting in firmer bellies and decreased potential for fatty acid oxidation.

Implications

Feeding CLA corrects the subjective fat quality problem that occurs from feeding HOC. The fatty acid profile from pigs fed CLA is more likely to be less subject to oxidation and, therefore, feeding CLA could potentially increase shelf life of pork products. However, CLA does not consistently act as a growth promotant or consistently decrease fat in the carcass of pigs.

Literature Cited

- Bowers, K. A., D. C. Kendall, and B. T. Richert. 2000 Evaluation of high oil corn for grow-finish pigs. *J. Anim. Sci.* (78 Suppl. 1):50.
- Eggert, J. M., M. A. Belury, A. Kempa-Steczko and A. P. Schinckel. 1999. Effects of conjugated linoleic acid (CLA) on growth and composition of lean gilts. *J. Anim. Sci.* 77:53.
- Meynier, A., C. Genot, G. Gandemer. 1999. Oxidation of muscle phospholipids in relation to their fatty acid composition with emphasis on volatile compounds. *J. Sci. Food Agric.* 79:6 p. 797-804.
- National Swine Improvement Federation. 1997. Guidelines for uniform swine improvement programs. p. 24.
- Pettigrew J. E. and R. L. Moser. 1991 Fat in Swine Nutrition. In: E. R. Miller, D. E. Ullrey, and A. J. Lewis.(ed.) *Swine Nutrition*. p. 133. Butterworth-Heinemann, Stoneham, MA.
- SAS. 1990. *SAS User's Guide: Statistics (Version 6 Ed.)* SAS Inst. Inc., Cary, NC.

- Shackelford, S. D., M. F. Miller, K. D. Haydon, N. V. Lovegreen, C. E. Lyon, and J. O. Reagan. 1990. Acceptability of bacon as influenced by the feeding of elevated levels of monounsaturated fats to growing-finishing swine. *J. Food Sci.* 55:3 p 621-624.
- Skelley, G. C., R. F. Borgman, D. L. Handlin, J. C. Acton, J. C. McConnell, F. B. Wardlaw, and E. J. Evans. 1975. Influence of diet on quality, fatty acids, and acceptability of pork. *J. Anim. Sci.* 41:1298-1309.
- Stahly, T. S. and G. L. Cromwell. 1979. Effect of environmental temperature and dietary fat supplementation on the performance and carcass characteristics of growing and finishing swine. *J. Anim. Sci.* 49: 1478-1488.
- Thiel, R. L., J. C. Sparks, B. R. Wiegand, F. C. Parrish, Jr., and R. C. Ewan. 1998. Conjugated linoleic acid improves performance and body composition in swine. *J. Anim. Sci.* 76(Suppl. 1):61.
- Wiegand, B. R., J. C. Sparks, F. C. Parrish, Jr., and D. R. Zimmerman. 2000. Performance, composition and quality characteristics of finishing barrows supplemented with conjugated linoleic acid. Submitted to *J. Anim. Sci.*

Table 4.1. Composition of experimental diets for days 1 through 28

Ingredient	Treatments*					
	NC	NC+ CLA	HOC	HOC+ CLA	NC+ CWG	NC+CWG +CLA
Corn	76.6	76.6	-	-	72.4	72.4
HOC	-	-	76.9	76.9	-	-
SBM	19.6	19.6	19.5	19.5	21.1	21.1
CWG	-	-	-	-	3.0	3.0
Soy oil	1.25	-	1.25	-	1.25	-
CLA	-	-1.25	-	1.25	-	1.25
Dicalcium phosphate	0.71	0.71	0.71	0.71	0.70	0.70
Calcium carbonate	1.33	1.33	1.18	1.18	0.72	0.72
Salt	0.25	0.25	0.25	0.25	0.25	0.25
Vitamin premix ^a	0.2	0.2	0.2	0.2	0.2	0.2
Trace mineral premix ^b	0.05	0.05	0.05	0.05	0.05	0.05
Tylosin premix ^c	0.05	0.05	0.05	0.05	0.05	0.05
Calculated analysis						
ME kcal/kg	3318	3318	3468	3468	3468	3468
Crude protein, %	15.0	15.0	15.6	15.6	15.6	15.6
Lysine, %	0.80	0.80	0.84	0.84	0.84	0.84
Meth. + cys., %	0.54	0.54	0.58	0.58	0.55	0.55
Analyzed, %						
Lysine	0.73	0.72	0.75	0.73	0.75	0.73

* NC-normal corn; CLA-conjugated linoleic acid; HOC-high oil corn; CWG-Choice white grease

^a At 0.2% of diet contributes per kilogram of diet: 4,400 IU vitamin A; 22 IU vitamin E; 1,100 IU vitamin D; 6.6 mg riboflavin; 17.6 mg pantothenic acid; 33 mg niacin; 22 µg vitamin B₁₂.

^b At 0.05% of the diet contributes in ppm: 75 Zn, 870.5 Fe, 30 Mn, 8.75 Cu, 0.1 I.

^c Supplied tylosin at 40 mg/kg of diet.

Table 4.2. Composition of experimental diets for days 29 through 56

Ingredient	Treatments*					
	NC	NC+ CLA	HOC	HOC+ CLA	NC+ CWG	NC+CWG +CLA
Corn	87.95	87.95	-	-	82.46	82.46
HOC	-	-	87.88	87.88	-	-
SBM	10.75	10.75	8.86	8.86	10.7	10.7
CWG	-	-	-	-	3.59	3.59
Soy oil	1.25	-	1.25	-	1.25	-
CLA	-	1.25	-	1.25	-	1.25
Dicalcium phosphate	0.63	0.63	0.63	0.63	0.62	0.62
Calcium carbonate	0.67	0.67	0.7	0.7	0.69	0.69
Salt	0.25	0.25	0.25	0.25	0.25	0.25
Vitamin premix ^a	0.2	0.2	0.2	0.2	0.2	0.2
Trace mineral premix ^b	0.05	0.05	0.05	0.05	0.05	0.05
Tylosin premix ^c	0.05	0.05	0.05	0.05	0.05	0.05
Lysine-HCL	0.14	0.14	0.13	0.13	0.13	0.13
Calculated analysis						
ME kcal/kg	3333	3333	3499	3499	3499	3499
Crude protein, %	10.6	10.6	11.4	11.4	11.1	11.1
Lysine, %	0.60	0.60	0.64	0.64	0.64	0.64
Meth. + cys., %	0.46	0.46	0.48	0.48	0.44	0.44
Analyzed, %						
Lysine	0.58	0.54	0.62	0.60	0.59	0.59

* NC-normal corn; CLA-conjugated linoleic acid; HOC-high oil corn; CWG-Choice white grease

^a At 0.2% of diet contributes per kilogram of diet: 4,400 IU vitamin A; 22 IU vitamin E; 1,100 IU vitamin D; 6.6 mg riboflavin; 17.6 mg pantothenic acid; 33 mg niacin; 22 µg vitamin B₁₂.

^b At 0.05% of the diet contributes in ppm: 75 Zn, 870.5 Fe, 30 Mn, 8.75 Cu, 0.1 I.

^c Supplied tylosin at 40 mg/kg of diet.

Table 4.3. Average daily gain of pigs fed conjugated linoleic acid and high oil corn

Table 1. Average daily gain of piglets on very good, moderate and high on corn								
Item	Period, d	Treatments						SEM
		NC	NC+CLA	HOC	HOC-CLA	NC+CWG	NC+CWG+CLA	
ADG, kg								
	1-14 ^a	1.19	1.14	1.25	1.16	1.34	1.23	0.05
	15-28	1.30	1.21	1.23	1.24	1.29	1.25	0.05
	28-42	1.07	1.03	0.97	1.05	1.02	0.98	0.06
	42-49	1.10	1.16	1.10	1.25	1.25	1.16	0.13
	49-56 ^{b,c}	0.98	0.83	0.66	0.57	0.84	0.71	0.08
Cumulative								
	1-28 ^a	1.25	1.18	1.24	1.20	1.32	1.24	0.04
	1-42	1.19	1.13	1.15	1.15	1.22	1.14	0.04
	1-49	1.18	1.13	1.15	1.16	1.22	1.15	0.04
	Over all ^d	1.17	1.10	1.11	1.13	1.20	1.12	0.05

^a CLA main effect $P < 0.05$.^b Energy main effect $P < 0.02$.^c Diets containing HOC vs. LE diets $P < 0.01$.^d Over all was calculated by subtracting the initial body weight from the final body weight and dividing by total number of days the individual pig was on test.**Table 4.4. Average daily feed intake of pigs fed conjugated linoleic acid and high oil corn**

		Treatments						
Item	Period, d	NC	NC+ CLA	HOC	HOC+ CLA	NC+ CWG	NC+CWG +CLA	SEM
ADFI, kg								
	1-14	2.94	2.82	2.94	2.80	2.91	2.91	0.08
	15-28 ^a	3.19	3.11	3.30	3.14	2.90	3.01	0.11
	28-42	3.24	3.26	3.14	3.14	2.95	3.16	0.11
	42-49	3.45	3.29	3.23	3.60	3.29	3.10	0.15
	49-56 ^b	3.10	3.00	2.80	2.62	2.93	2.64	0.13
Cumulative								
	1-28	3.07	2.96	3.12	2.97	2.91	2.96	0.08
	1-42	3.12	3.06	3.13	3.03	2.92	3.02	0.08
	1-49	3.16	3.08	3.14	3.09	2.96	3.03	0.08
	Over all ^d	3.15	3.08	3.14	3.08	2.98	3.02	0.08

^a Diets containing HOC vs. diets containing CWG $P < 0.01$.^b Energy main effect $P < 0.03$.^d Over all was calculated by taking the total feed consumed and dividing by the total number of days the individual pig was on test.

Table 4.5. Gain:feed of pigs fed conjugated linoleic acid and high oil corn

Item	Period, d	Treatments					SEM	
		NC	NC+CLA	HOC	HOC+CLA	NC+CWG		NC+CWG+CLA
GF, g/kg								
	1-14 ^{a,b,c}	405	404	427	413	462	421	9.9
	15-28 ^{b,c,d}	408	392	372	396	445	415	12.2
	28-42	330	314	308	335	344	311	13.3
	42-49 ^d	310	347	337	348	379	375	29.5
	49-56	319	273	233	219	284	267	25.5
Cumulative								
	1-28 ^{a,b,c,d}	407	397	397	404	454	418	7.3
	1-42 ^{a,c,d}	380	368	367	380	417	379	7.1
	1-49 ^{b,c}	373	366	364	376	413	378	7.1
	Over all ^{a,b,c}	370	357	352	367	402	371	8.2

^a CLA main effect $P < 0.05$.^b Energy main effect $P < 0.05$.^c Diets containing HOC vs. diets containing CWG $P < 0.05$.^d CLA interaction for diets containing HOC and CWG $P < 0.01$.^e Over all was calculated by dividing the over all ADFI into the overall ADG.**Table 4.6. Ultrasound back fat and loin muscle measurements of pigs fed conjugated linoleic acid and high oil corn**

Item	Treatments						SEM
	NC	NC+CLA	HOC	HOC+CLA	NC+CWG	NC+CWG+CLA	
10th rib BF, cm							
Initial	1.02	0.99	1.04	0.98	0.96	1.02	0.05
Day 28 ^a	1.49	1.53	1.61	1.73	1.54	1.70	0.08
Slaughter ^{b,c}	2.11	1.98	2.22	2.21	2.03	2.42	0.10
LMA, cm ²							
Initial ^b	22.51	22.99	20.90	22.05	21.44	21.35	0.61
Day 28 ^{a,b}	38.34	38.56	35.04	35.60	34.14	37.33	1.2
Slaughter ^{b,d}	40.36	40.86	38.13	38.69	36.59	40.46	1.0
% Lean							
Day 28 ^e	55.50	55.42	53.66	53.44	53.56	54.29	0.46
Slaughter ^e	52.73	53.43	51.37	51.63	51.46	51.28	0.51

^a LE diets vs. diets containing HOC $P < 0.05$.^b Energy main effect $P < 0.03$.^c CLA interaction for diets containing HOC and CWG $P < 0.04$.^d CLA main effect $P < 0.06$.^e Energy main effect $P < 0.001$.

Table 4.7. Objective and subjective measurements of carcasses from pigs fed conjugated linoleic acid and high oil corn

Item	Treatments						SEM
	NC	NC+CLA	HOC	HOC+CLA	NC+CWG	NC+CWG+CLA	
Hot carcass wt., kg	82.8	82.4	82.3	80.8	84.7	82.2	1.10
Back fat, cm							
1st rib	3.78	3.81	4.01	4.11	3.68	3.86	0.06
Last rib	2.69	2.44	2.92	2.69	2.67	2.64	0.06
Last lumbar ^a	2.21	2.03	2.49	2.44	1.98	2.44	0.06
10th rib ^a	2.31	2.16	2.51	2.59	2.08	2.57	0.05
LMA, cm ²	40.58	42.32	39.42	38.58	39.94	44.32	0.23
Leaf fat, kg ^b	1.16	1.17	1.41	1.34	1.10	1.54	0.12
Loin quality							
Color ^c	2.63	2.38	2.00	2.13	2.38	2.16	0.19
Marbling	1.88	2.13	1.88	1.88	1.88	1.81	0.22
Firmness	2.75	2.25	2.50	2.25	2.38	2.03	0.28
45 min pH	6.68	6.54	6.52	6.50	6.55	6.56	0.10
120 min pH	6.71	6.43	6.39	6.54	6.42	6.41	0.13
24 h pH	5.78	5.75	5.74	5.73	5.77	5.70	0.05

^a LE diets vs. diets containing HOC P < 0.04.^b CLA interaction for diets containing HOC and CWG P < 0.04.^c Energy main effect P = 0.05.**Table 4.8. Whole carcass and left side primal weights of pigs fed conjugated linoleic acid and high oil corn, kg**

Item	Treatments						SEM
	NC	NC+CLA	HOC	HOC+CLA	NC+CWG	NC+CWG+CLA	
Right side ^a	39.5	39.3	39.1	37.8	40.2	38.8	0.56
Left side ^b	39.9	39.6	39.7	38.7	41.0	39.5	0.56
Picnic ^c	4.99	5.00	4.98	4.72	5.14	4.83	0.10
Boston ^c	4.90	4.82	4.92	4.93	4.86	4.57	0.09
Shoulder ^c	9.90	9.82	9.90	9.65	10.00	9.40	0.14
Loin ^c	10.97	10.62	10.78	11.33	10.58	11.19	0.17
Ham ^d	9.70	9.74	9.74	9.84	9.85	9.71	0.12
Belly ^d	7.64	8.04	7.81	7.47	7.90	8.08	0.18

^a CLA main effect P = 0.05.^b CLA main effect P = 0.07.^c Diets containing HOC vs. diets containing CWG P < 0.03.^d CLA interaction for diets containing HOC and LE diets P < 0.05.

Table 4.9. Ham dissection weights from pigs fed conjugated linoleic acid and high oil corn

Item	Treatments						SEM
	NC	NC+CLA	HOC	HOC+CLA	NC+CWG	NC+CWG+CLA	
Ham, kg	9.63	9.64	9.66	9.52	10.04	9.57	0.39
Lean, kg	6.60	6.70	6.65	6.77	6.66	6.57	0.17
Bone, kg	1.35	1.30	1.31	1.17	1.31	1.23	0.14
Skin, kg ^a	0.69	0.53	0.68	0.51	0.73	0.58	0.11
Fat, kg ^a	0.97	1.15	1.03	1.27	1.00	1.21	0.20
Fat + skin, kg	1.70	1.68	1.70	1.78	1.74	1.79	0.19

^a CLA main effect $P < 0.01$.**Table 4.10. Belly bar test from pigs fed conjugated linoleic acid and high oil corn**

Item	Treatments						SEM
	NC	NC+CLA	HOC	HOC+CLA	NC+CWG	NC+CWG+CLA	
Belly bar test, cm							
Lean down ^a	12.3	18.8	11.1	16.0	10.1	18.8	1.5
Lean up ^{a,b}	18.4	28.7	12.8	22.4	12.6	29.3	1.9

^a CLA main effect $P < 0.001$.^b CLA interaction for diets containing HOC and LE diets $P < 0.03$.**Table 4.11. Fatty acid profile (%)^a of loin tissue from 10th and 11th rib junction of pigs fed conjugated linoleic acid and high oil corn**

Item	Treatments						SEM
	NC	NC+CLA	HOC	HOC+CLA	NC+CWG	NC+CWG+CLA	
14:0 ^{b,c}	1.24	1.64	1.26	1.83	1.26	1.93	0.055
16:0 ^b	25.7	29.5	25.1	29.6	24.8	30.7	0.32
18:0 ^b	14.54	14.92	13.65	15.59	13.88	15.19	0.26
18:1 ^{b,c,d}	43.5	39.4	43.2	36.9	43.2	38.0	0.57
18:2 ^{b,c}	9.35	7.82	11.73	9.90	11.22	7.48	0.55
18:3 ^{c,d,e}	5.40	5.53	4.70	4.89	5.26	5.42	0.12
20:4	0.237	0.190	0.200	0.189	0.189	0.180	0.019
CLA ^{f,b,c}	0.05	0.91	0.08	1.13	0.19	1.14	0.053
SFA ^{g,b,h}	41.5	46.1	40.1	47.0	39.9	47.8	0.44
PUFA ^{i,b,c,d}	15.0	13.5	16.6	15.0	16.7	13.1	0.58

^a Expressed as a ratio of shown fatty acids of sample injected on gas chromatograph column.^b CLA main effect $P < 0.01$.^c Energy main effect $P < 0.05$.^d LE diets vs. diets containing HOC $P < 0.01$.^e Diets containing HOC vs. diets containing CWG $P < 0.0001$.^f Conjugated linoleic acid isomers.^g Saturated fatty acids is sum of 14:0, 16:0, and 18:0.^h CLA interaction between diets containing HOC and LE diets $P < 0.02$.ⁱ Polyunsaturated fatty acids is sum of 18:1, 18:2, 18:3, and 20:4.

Table 4.12. Loin composition quality measurements from pigs fed conjugated linoleic acid and high oil corn

Item	Treatments						SEM
	NC	NC+ CLA	HOC	HOC+ CLA	NC+ CWG	NC+CWG +CLA	
Loin							
% Moisture	73.0	72.9	72.8	72.7	72.8	72.2	0.28
% Fat	2.60	2.67	2.78	2.75	3.00	2.78	0.25
Myoglobin, mg/g	0.660	0.677	0.739	0.620	0.685	0.666	0.05
Hunter values							
Day 0							
L ^{a,b}	42.6	45.7	44.3	47.1	44.5	45.6	1.12
A [*]	5.8	5.7	5.8	6.4	6.6	6.7	0.51
B [*]	10.3	11.2	10.6	11.4	11.2	11.4	0.32
Day 1							
L ^{a,b}	42.7	45.7	44.0	46.6	44.4	45.8	1.24
a [*]	5.7	6.0	5.9	5.6	6.1	6.2	0.48
b ^{a,b}	10.2	11.0	10.7	11.2	11.0	11.4	0.35
Day 2							
L ^{a,b}	43.3	45.1	43.9	47.7	44.7	46.2	1.02
a [*]	6.6	6.3	6.1	6.3	6.4	6.9	0.45
b [*]	10.2	10.6	10.4	10.9	10.6	10.8	0.24

^a CLA main effect $P = 0.05$.

CHAPTER 5: GENERAL SUMMARY

Two experiments were conducted to evaluate the use of conjugated linoleic acid (CLA) on growth performance, carcass quality and pork quality.

In experiment 1, 92 four-way cross barrows were used to determine the optimal time at which to start including CLA in the diet for pigs slaughtered at 113 kg body weight. Experiment 2 was conducted to determine the value of feeding CLA to reverse the expected negative effects of feeding high oil corn (HOC) on pork quality.

Results of the first experiment indicated that the optimal weight at which to start the feeding of CLA was between 58 and 87 kg of body weight. This study was conducted with moderate lean pigs that were genetically selected to be slaughtered at 115 kg of body weight before slaughter. Currently pigs of high lean gain genetics are being fed to a heavier body weight. Therefore, the optimal starting weight for these newer genetics is probably going to be somewhat heavier than 58 to 87 kg.

In the second experiment, pigs fed HOC did not have increased GF expected as a result of the increased diet energy density. This lack of response may be because HOC did not have the lysine concentration that was predicted and, therefore, the HOC diets were not equal in ME:lysine ratio to the normal corn diets (lower energy).

The expected positive effects of CLA on growth performance were lacking in this experiment compared with those observed in previous experiments conducted. The lack of responses are difficult to explain, however, it is noteworthy that the pigs in experiment 2 grew rapidly. They were brought into a segregated early weaning unit that had not housed any pigs

for over a month and then into a room that had not housed pigs for over 3 months. The rate of gain of the pigs in this experiment was nearly 1.4 times that of pigs fed in a typical environment. It is important to note that CLA did improve fat quality of the bellies from pigs fed NC, HOC and CWG. Therefore, this benefit of feeding CLA was evident in these rapid growing pigs.

Results of these two studies are indicative of the mixed results seen in previous studies conducted by Dugan et al. (1997, 1999), Dunshea et al. (1998), Ostowska et al. (1999), Cook et al. (1999), Theil et al. (1998), Eggert and Schinkel (1999), and O'Quinn et al. (1998). The results of the CLA studies reported up to this time have shown very little effect on ADG and a slight decrease in ADFI. These slight changes in ADG and ADFI, however, result in a more consistent increase in GF. Growth performance in the first experiment reported in this dissertation agrees with the consensus of the studies previous reported. However, our second study revealed no effect on ADG or ADFI and a decrease on GF due to feeding CLA. Furthermore, this study indicated that the higher energy density of HOC diets compared with that of diets containing normal corn were not effective in increasing GF. If the fat from HOC is well digested, one would expect the HOC-fed animals to have had the same GF as the pigs fed CWG diets (Pettigrew and Moser 1991).

Reported effects of feeding CLA on pig carcasses have consistently shown an increase in loin muscle area and a decrease in back fat, resulting in increased percent lean in the carcass. Results of our first experiment agrees with these previous reports. In our second experiment CLA increased LMA, but did not decrease BF at the 10th rib, and, consequently, the percent

lean was not increased. However, the study did show the expected increase in percent lean associated with feeding lower energy diets.

Fat quality changes are easily made by feeding a high concentration of a particular type of fatty acid (Villiegas et al., 1973; Skelley et al., 1975; St. John et al., 1987; Romans et al., 1995a, 1995b; Shackelford et al., 1990). All CLA studies that have measured the effects on fat have found, either a higher degree of saturation in fat tissue or an increase in belly firmness (Dugan et al., 1997, 1999; Dunshea et al., 1998; Cook et al., 1999; Theil et al., 1998; and O'Quinn et al., 1998). Feeding CLA or a fat source high in saturated fats will increase carcass fatty acid saturation and belly firmness, whereas feeding a fat source high in unsaturated fatty acids will decrease fatty acid saturation and belly firmness (Villiegas et al., 1973; Skelley et al., 1975; St. John et al., 1987; Romans et al., 1995a, 1995b; Shackelford et al., 1990). Both of our experiments have shown fat composition and belly firmness results that are consistent with experiments that have been previously reported. The fatty acid composition of carcass fat of the first experiment increased in saturated fatty acids as the amount of weight gain while fed CLA increased. In the second experiment, feeding CLA increased belly firmness and feeding HOC (unsaturated fatty acids) decreased belly firmness. The increase in belly firmness was confirmed by change in the fatty acid profile. Pigs fed CLA had an increase in saturated fatty acids and a decrease in unsaturated fatty acids. The bellies of pigs fed HOC were softer than bellies of pigs fed low energy diets. The fatty acid profile suggests that this effect was largely caused by an increase in linoleic acid. Also, the overall saturation of fatty acids in pigs fed HOC was decreased compared with that of pigs fed the low energy diet. In the second experiment, the fatty acid profile of the loin tissue had an unusually high percent of linolenic

acid. Although, this result can not be explained, it is believed to be caused by a lack of complete separation from either oleic acid or some contaminant during gas chromatography analysis.

There is potential for increased shelf life and consumer preference as a result of feeding CLA. Villiegas et al., (1973); Skelley et al., (1975); St. John et al., (1987); Romans et al., (1995a and 1995b); and Shackelford et al., (1990) have shown that feeding PUFA decreased consumer preferences and increased fatty acid oxidation causing a decrease in shelf life of the pork products.

With the lack of a strong effect of CLA as a growth promotant and only a moderate effect on feed efficiency, the major benefits of feeding CLA are the increase in carcass lean and the improvements in pork quality. It remains to be seen whether these benefits are of sufficient economic value to justify application of this technology.

Literature Cited

- Cook, M. E., D. L. Jerome, T. D. Crenshaw, D. R. Buege, M. W. Pariza, K. J. Albright, S. P. Schmidt, J. A. Scimeca, P. A. Lofgren, and E. J. Hentges. 1998. Feeding conjugated linoleic acid improves feed efficiency and reduces whole body fat in pigs. *FASEB J.* 12, A836.
- Dugan M. E. R., J. L. Aalhus, A. L. Schaefer, and J. K. G. Kramer. 1997 The effect of conjugated linoleic acid on fat to lean repartitioning and feed conversion in pigs. *Can. J. Anim. Sci.* 77:723–725.
- Dugan M. E. R., J. L. Aalhus, L. E. Jeremiah, J. K. G. Kramer, and A. L. Schaefer. 1999 The effects of feeding conjugated linoleic acid on subsequent pork quality. *Can. J. Anim. Sci.* 79:45–51
- Dunshea, F. R., E. Ostrowska, M. Muralitharan, R. Cross, D. E. Bauman, M W. Pariza, and C. Skarie. 1998. Dietary conjugated linoleic acid decreases back fat in finisher gilts. *J. Anim. Sci.* 76 (Suppl. 1) 131.

- Eggert, J. M., M. A. Belury, and A. P. Schinckel. 1999. The effects of conjugated linoleic acid (CLA) and feed intake on lean pig growth and carcass composition. *Purdue University Swine Day*. p. 21-25.
- O'Quinn, P. R., J. W. Smith, II, J. L. Nelssen, M. D. Tokach, R. D. Goodband, and J. S. Smith. 1998. Effects of modified tall oil versus conjugated linoleic acid on finishing pig growth performance and carcass characteristics. *Kansas State University Swine Day* 1998. 157-161.
- Ostrowska E., M. Muralitharan, R. F. Cross, D. E. Bauman, and F. R. Dunshea. 1999. Dietary conjugated linoleic acids increase lean tissue and decrease fat deposition in growing pigs. *J. Nutr.* 129:2037-2042.
- Pettigrew J. E. and R. L. Moser. 1991 *Fat in Swine Nutrition*. In: E. R. Miller, D. E. Ullrey, and A. J. Lewis.(ed.) *Swine Nutrition*. p. 133. Butterworth-Heinemann, Stoneham, MA.
- Romans, J. R., R. C. Johnson, E. M. Wulf, G. W. Libal, and W. J. Costello. 1995a. Effects of ground flaxseed in swine diets on pig performance and on physical and on physical and sensory characteristics and omega-3 fatty acids content of pork: I. Dietary level of flaxseed. *J. Anim Sci.* 73:1982-1986.
- Romans, J. R., R. C. Johnson, E. M. Wulf, G. W. Libal, and W. J. Costello. 1995b. Effects of ground flaxseed in swine diets on pig performance and on physical and on physical and sensory characteristics and omega-3 fatty acids content of pork: II. Duration of 15% dietary flaxseed. *J. Anim Sci.* 73:1987-1999.
- Shackelford, S. D., M. F. Miller, K. D. Haydon, N. V. Lovegreen, C. E. Lyon, and J. O. Reagan. 1990. Acceptability of bacon as influenced by the feeding of elevated levels of monounsaturated fats to growing-finishing swine. *J. Food Sci.* 55:3 p 621-624.
- Skelley, G. C., R. F. Borgman, D. L. Handlin, J. C. Acton, J. C. McConnell, F. B. Wardlaw, and E. J. Evans. 1975. Influence of diet on quality, fatty acids, and acceptability of pork. *J. Anim. Sci.* 41:1298.
- St. John, L. C., C. R. Yung, D. A. Knabe, L. D. Thompson, G. T. Schelling, S. M. Grundy, and S. B. Smith. 1987. Fatty acid profile and sensory and carcass traits of tissues from steers and swine fed an elevated monounsaturated fat diet. *J. Anim. Sci.* 64(5):1441.
- Thiel, R. L., J. C. Sparks, B. R. Wiegand, F. C. Parrish, Jr., and R. C. Ewan. 1998. Conjugated linoleic acid improves performance and body composition in swine. *J. Anim. Sci.* 76(Suppl. 1) p. 61.
- Villegas, F. J., H. B. Hedrick, T. L. Veum, K. L. McFate, and M. E. Bailey. 1973. Effect of diet and breed on fatty acid composition of porcine adipose tissue. *J. Anim. Sci.* 36: 663.