Influence of extruded linseed on growth, carcass composition, and meat quality of slaughtered pigs at one hundred ten and one hundred sixty kilograms of liveweight

C. Corino, M. Musella and J. Mourot

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Influence of extruded linseed on growth, carcass composition, and meat quality of slaughtered pigs at one hundred ten and one hundred sixty kilograms of liveweight[1]

C. Corino,*[2] M. Musella,* and J. Mourot†

*Department of Veterinary Sciences and Technologies for Food Safety, University of Milan, Via Celoria 10, 20133 Milan, Italy; and †l'Institut National de la Recherche Agronomique, UMR 1079, Systèmes d'Elevage, Nutrition Animale et Humaine, F-35590 St-Gilles, France

ABSTRACT: The Western diet is characterized by a high intake of SFA relative to PUFA, and the consumption of n-3 PUFA is decreased relative to n-6 PUFA. Therefore, there has been much interest in recent years in ways to manipulate the fatty acid composition of meat. The objective of this work was to determine the effects of dietary extruded linseed, an n-3 PUFA source, on growth performance, carcass composition, meat quality, and oxidative stability of pigs slaughtered at 111.0 (±4.8) kg of BW and 160.0 (±9.2) kg of BW. The association of these factors with BW at slaughter was also examined. Forty barrows, 78.1 (±1.75) kg of initial BW, were fed a control diet (2.5% sunflower oil) or a linseed diet containing 5% of whole extruded linseed. Both diets contained 170 mg of vitamin E and 250 μg of selenium. Eight pigs from each dietary treatment were slaughtered at 110 kg of BW and the others at 160 kg of BW. There was no dietary effect ($P > 0.05$) on growth, carcass characteristics, meat quality, or the activity of malic enzyme in LM and backfat. Inclusion of linseed increased ($P < 0.05$) n-3 PUFA content in both LM and backfat and decreased the n-6:n-3 PUFA ratio from 12 to 4.5 in LM, and from 11 to 3 in backfat. Liveweight at slaughter significantly influenced carcass characteristics, meat quality, total lipid and oxidative stability of LM, malic enzyme activity in adipose tissue, and fatty acid content of LM and backfat. This study shows that the inclusion of linseed in swine diets may improve the fatty acid profile of pork without deleteriously affecting oxidation or color stability. Thus, such a feeding practice may improve human health based on the n-6:n-3 PUFA ratio recommended for the human diet.

Key words: linseed, liveweight, meat quality, n-3 polyunsaturated fatty acid, pig

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INTRODUCTION

In recent years, nutritionists have expressed concern that the typical Western diet provides too much n-6 and not enough n-3 PUFA. Some of the most important international organizations for nutrition, such as the joint Food and Agriculture Organization/World Health Organization committee, advised nutritional recommendations concerning the n-6:n-3 PUFA ratio, suggesting a value between 5:1 and 10:1, as reported in the International Society for the Study of Fatty Acids and Lipids (ISSFAL, 2004).

The n-6:n-3 PUFA ratio may be as high as 17:1 in the overall Western diet (Simopoulos, 2001), so consumption of n-3 PUFA is too low relative to n-6 PUFA. The current high level of n-6 PUFA in the food supply is a concern because these acids can interfere with the conversion of α-linolenic acid (**ALA**) to eicosapentaenoic acid (**EPA**) and docosahexanoic acid (**DHA**). Furthermore, diets high in n-6 PUFA lead to increased concentrations of arachidonic acid (**ARA**) in membrane phospholipids, which results in an overproduction of eicosanoids that may contribute to hardening of the arteries and other chronic conditions (Simopoulos, 2002). Moreover, eating less n-6 and more n-3 PUFA may help decrease the risk of heart disease and cancer (Simopoulos, 2002). Meat could be a source of n-3 PUFA if the animals are fed diets supplemented with sources of n-3 PUFA. The most important alimentary source of n-3 PUFA is fish and fish products, which are rich in long-chain n-3 PUFA, but supplementation of fish and fish products can be costly and increases the risk of

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[2]Corresponding author: carlo.corino@unimi.it

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Table 1. Composition of experimental diets (as-fed basis)

Item	Diet	
	Control	Linseed
Ingredient, %		
Barley	24.50	24.00
Wheat	24.00	24.00
Corn	15.00	15.00
Soybean meal	23.00	21.00
Wheat bran	5.00	5.00
Extruded linseed	—	5.00
Sunflower oil	2.50	—
Molasses, cane	3.00	3.00
Calcium carbonate	1.30	1.30
Dicalcium phosphate	0.51	0.51
Sodium chloride	0.45	0.45
Mineral and vitamin premix[1]	0.74	0.74
Calculated nutrient composition[2]		
DE, Mcal/kg	3.20	3.20
CP, %	17.02	17.02
Crude fat, %	4.48	4.47
Crude fiber, %	3.87	4.19
Lysine, %	0.84	0.83
Methionine + Cysteine, %	0.59	0.57
Tryptophan, %	0.21	0.22
Threonine, %	0.61	0.62
Ca, %	0.78	0.79
P, %	0.48	0.50

[1]The vitamin and mineral per kilogram of the diet provided by premix: vitamin A, 7,400 UI; vitamin D_3, 1,500 UI; vitamin E, 170.0 mg; vitamin K_3, 3.0 mg; vitamin B_1, 3.0 mg; vitamin B_2, 6.0 mg; calcium D-pantothenate, 15.0 mg; niacin, 22.0 mg; vitamin B_{12}, 0.03 mg; vitamin B_6, 1.5 mg; folic acid, 1.5 mg; biotin, 0.3 mg; choline chloride, 740.0 mg; Zn (ZnO), 150.0 mg; Cu ($CuSO_4$), 15.0 mg; Mn (MnO), 59.0 mg; Fe ($FeCO_3$), 120.0 mg; I ($Ca(IO_3)_2$), 300.0 µg; Co ($CoSO_4$), 30.0 µg; Se (as Na_2SeO_3, 170.0 µg, and as Se-methionine 80 µg), 250 µg.

[2]Calculated based on INRA (1989).

Table 2. Fatty acid composition (g/100 g of fatty acids) of diets supplemented with 2.5% sunflower oil (control diet) or 5% extruded linseed (linseed diet)

Fatty acid	Diet	
	Control	Linseed
C14:0	0.17	0.16
C14:1	0.01	0.01
C16:0	14.51	14.22
C16:1	0.39	0.36
C18:0	3.33	3.15
C18:1n-9	25.64	20.91
C18:2n-6	51.34	39.61
C20:0	0.02	0.02
C18:3n-3	2.58	19.96
C20:1n-9	0.43	0.44
C20:2n-6	0.28	0.27
C20:3n-3	0.65	0.34
C20:4n-6	0.03	0.04
C22:1n-9	0.11	0.15
C20:5n-3	0.12	0.07
C24:0	0.22	0.10
C24:1	0.07	0.06
C22:5n-3	0.01	0.02
C22:6n-3	0.09	0.11
Total	100	100
SFA	18.25	17.65
MUFA	26.65	21.93
PUFA	55.10	60.42
n-6 PUFA	51.65	39.92
n-3 PUFA	3.45	20.50
n-6:n-3 PUFA	14.97	1.95

flavor taints and rancidity in meat (Wood et al., 1999). An alternative source is linseed and its by-products, which are rich in ALA, and are precursors for EPA and DHA.

The aim of this study was to investigate in pigs slaughtered at different weights (110 kg and 160 kg of BW) and the effect of extruded linseed on 1) the accumulation of ALA, EPA, and DHA fatty acids in muscle and in adipose tissue; and 2) the growth performance, carcass characteristics, and meat quality.

MATERIALS AND METHODS

Animals and Diets

All experimental procedures were carried out in accordance with the guidelines of the French Institut National de la Recherche Agronomique (**INRA**) for the care and use of laboratory animals.

A total of 40 castrated crossbred pigs [Large White × (Landrace × Pietrain)] of 78.1 (±1.7) kg of BW, were chosen on the basis of their BW and were assigned to 1 of 2 dietary treatments. Eight pigs per treatment, randomly selected, were slaughtered at 111.0 (±4.8) kg of BW, followed by 12 pigs per treatment at 160.0 (±9.2) kg of BW. The diets, formulated to be isoenergetic and isolipidic, differed in lipid source and in fatty acid composition: 2.5% sunflower oil was present in the control diet, and 5% extruded linseed was included in the linseed diet. Diets and fatty acids composition of diets are shown in Table 1 and in Table 2, respectively. Both diets contained 170 mg of vitamin E and 250 µg of selenium (170 µg as selenite, and 80 µg as Se-methionine). The animals were kept in individual cages with a totally slotted floor, in total confinement, in an environmentally controlled building at the experimental station of the INRA center of Saint Gilles (France), with ad libitum access to feed and water until slaughter. The BW of each pig was recorded at the beginning of the trial and weekly until slaughter at 110 kg of BW or 160 kg of BW. Feed intake was measured for each pig by weighing all feed offered and subtracting what was removed from the trough daily. Average daily feed intake, ADG, and G:F were calculated for each pig.

Carcass Measurements

All animals were slaughtered at the abattoir of INRA of Saint-Gilles (France) at 111.0 (±4.8) kg of BW and 160.0 (±9.2) kg of BW after fasting for 12 h. Pigs were electrically stunned, and following exsanguination, the carcasses were scalded, dehaired, and eviscerated.

Liveweight at slaughter and hot carcass weight were recorded. Dressing percentage (hot carcass weight/liveweight at slaughter) was calculated. Backfat and LM thickness were measured with a Fat-O-Meater optical probe (Sydel, Lorient, France). Backfat thickness, including the rind, was evaluated between the third and fourth from the last lumbar vertebrae and the third and fourth from the last ribs, at 8 cm and 6 cm from median line, respectively. Longissimus muscle was evaluated between the third and fourth from the last ribs at 6 cm from median line. The lean meat content of all pig carcasses was calculated according to the following formula (Daumas et al., 1998):

$$Y = 58.15 - (0.198 \times G1) - (0.570 \times G2) + (0.255 \times M2),$$

where Y = the estimated percentage of lean meat in the carcass, G1 = the thickness of backfat (including rind) in millimeters, measured 8 cm off the midline of the split carcass between the third and fourth from the last lumbar vertebrae, G2 = the thickness of backfat (including rind) in millimeters, measured 6 cm off the midline of the split carcass between the third and fourth from the last ribs, and M2 = the thickness of the LM in millimeters, measured 6 cm off the midline of the split carcass between the third and fourth from the last ribs.

Meat and Fat Quality

All chemicals used in this study were reagent grade. Unless otherwise indicated, reagents were purchased from Sigma Chemical Co. (St. Louis, MO).

Immediately after slaughter, samples of LM and backfat from all pigs were taken from the region of the last rib of the right side of each carcass, to evaluate meat quality parameters and malic enzyme activity. At 24 h postmortem, samples of LM and backfat at the last lumbar vertebra were taken, vacuum-packed, frozen and stored at –20°C pending analyses of total lipids and thiobarbituric acid-reactive substances (**TBARS**; of LM only).

Color and pH were measured for all 40 pigs at 24 h postmortem on the LM (at the last lumbar vertebra). The pH was determined (HI 9023 microcomputer, Hanna Instruments, Amorim-Póvoa de Varzim, Portugal) by insertion of electrodes. Tri-stimulus color coordinates (L*, a*, b*) were recorded using a Chroma Meter CR-300 (Minolta Cameras, Osaka, Japan). The instrument was calibrated using the white calibration plate (Calibration Plate CR-A43, Minolta Cameras, Osaka, Japan) at the beginning of the session. The Chroma Meter has a measuring area 8 mm in diameter and illuminates the sample area with diffuse illumination from a pulsed Xenon arc lamp at 0° viewing angle (illuminant C). Reflectance measurements were obtained at a viewing angle of 0° and the spectral component was included. Color was measured with reference to lightness (L*: 0 = black, 100 = white) and 2 color coordinates, a* for redness and b* for yellowness, with the extreme colors of a* equal to red (positive) and green (negative) and of b* equal to yellow (positive) and blue (negative).

Malic enzyme was analyzed in the LM and in backfat. To measure the activity of malic enzyme (EC 1.1.1.40), samples of muscle (1.5 g) and fat (0.7 g) were homogenized in 0.25 mol/L sucrose buffer and centrifuged at 30,000 × *g* for 40 min at 4°C. Supernatants were analyzed for malic enzyme using a modified (Gandemer et al., 1983) method of Hsu and Lardy (1969). Malic enzyme was assayed through measurement of NADPH formation at 37°C by absorbance at 340 nm. The activity of malic enzyme was expressed as nanomoles of NADPH formed per minute per milligram of protein.

Lipid extraction was done using chloroform/methanol (2:1) according to the method of Folch et al. (1957). Lipids were extracted from 10 g of each sample of LM and from 1.5 g of each sample of backfat. The extracts were dried under vacuum on a rotary evaporator (Laborota 4000, Heidolph Instruments, Milan, Italy).

Fatty acid composition was measured after methylation of samples. Fatty acid methyl esters were prepared with boron trifluoride methanol according to Morrison and Smith (1964) and analyzed on a Di 200 (Delsi) gas chromatograph equipped with fused silica gel capillary column (0.25 mm i.d. × 30 m), filled with stationary phase (80% biscyanopropyl and 20% cyanopropylphenyl), using margaric acid (C17) as the internal standard. The chromatography conditions used were: temperature program from 45 to 240°C at 20 to 35°C/min. The injector and detector temperatures were maintained at 220 and 280°C, respectively. Retention times and peak areas were determined using chromatography software (Nelson Analytical, Manchester, NH). The identities of the peaks were verified by comparison with the retention time of standard fatty acid methyl esters. Results were expressed as the percentage of the total fatty acid composition of feed, LM, and backfat.

Iron-induced lipid oxidation of LM was determined using a TBARS assay and was carried out as a modification of the method described by Monahan et al. (1992). Moreover, the iron-induced lipid oxidation of LM was assessed by determining the production of TBARS, mostly malondialdehyde (MDA), after incubation with ferrous sulfate as a promoter of peroxidation. Briefly, 1 g of tissue was homogenized (Polytron homogenizer; Kinematica, Kùens-Lucerne, Switzerland) with 9 mL of 1.15% (w/v) KCl for 45 s. From the solution, 100 µL of homogenate was immediately submitted to iron-induced lipid oxidation by incubation for 60, 120, 200, or 300 min at 37°C in 80 m*M* Tris maleate buffer (pH 7.4) in the presence of 5 m*M* ferrous sulfate (to catalyze lipid peroxidation) in a total volume of 1 mL. At fixed times (0, 60, 120, 200, and 300 min), aliquots were removed and immediately analyzed for their MDA content, adding 2 mL of stock thiobarbituric acid-trichloracetic acid-HCl reagent and mixing thoroughly. The

solution was heated for 15 min in boiling water. After cooling, the precipitate was removed by centrifugation at 715 × *g* for 15 min. The absorbance of the samples was measured using a spectrophotometer (Genesys 5-Milton Roy, Rochester, NY) at a wavelength of 535 nm against a blank sample that contained all the reagents but not the test sample. The TBARS values are expressed as nanomoles of MDA/mg of protein during the time period of induced oxidation. Protein content was determined by the method of Lowry et al. (1951) with bovine serum albumin as the standard.

Statistical Analysis

The statistical model for performance used only the diet (control and linseed) as the main effect, analyzed separately for animals at 110 and 160 kg of BW. The statistical model for carcass characteristics, meat quality, malic enzyme activity, total lipid content, and fatty acid composition included the following effects: diet (control and linseed), weight at slaughter (110 and 160 kg of BW), and their interaction term. Statistical analysis of the data was performed by ANOVA (SPSS/PC Statistics, SPSS Inc., Chicago, IL). When the interaction between the dietary treatments and liveweight was significant, the effect of the treatments was assessed by comparing the least-squares means using a Student's *t*-test. The development of MDA in tissues during iron-induced lipid oxidation was analyzed by a repeated measures ANOVA in SPSS separately for animals killed at 110 kg and 160 kg of BW; a fixed-effects model was used to assess the main effects of dietary treatment (2 levels: control and linseed) and time of iron-induced lipid oxidation (5 levels: 0, 60, 120, 200, 300), and the interaction between the 2 factors. Individual pigs were considered as the experimental unit. Differences were considered significant if $P \leq 0.05$.

RESULTS

Fatty Acid Composition of Diet and Growth Performance

The control diet, supplemented with sunflower oil, was characterized by a high content of C18:1n-9 (oleic acid), C18:2n-6 (linoleic acid, **LA**), and of monounsaturated fatty acid generally, with an n-6:n-3 ratio of about 15 (Table 2). Alternatively, the treatment diet, supplemented with extruded linseed, was characterized by a high content of C18:3n-3 (ALA), with a n-6:n-3 PUFA ratio of less than 2. Dietary treatment did not affect final weight, ADFI, ADG or G:F, either in 110 kg of BW pigs or in 160 kg of BW pigs (Table 3). However, greater homogeneity of final BW was observed for pigs fed the linseed diet at 110 and 160 kg of BW. Liveweight at slaughter did not affect ADG (110 kg = 1.18 kg vs. 160 kg = 1.04 kg; $P > 0.05$), but significantly affected G:F (110 kg = 319 g/kg vs. 160 kg = 242 g/kg; $P < 0.001$) and ADFI (110 kg = 3.69 kg vs. 160 kg = 4.09 kg; $P < 0.001$).

Table 3. Effects of diet (control diet: 2.5% sunflower oil; linseed diet: 5% extruded linseed) on performance of 110 and 160-kg liveweight pigs (least-squares means)[1]

Item	Control diet	Linseed diet	SE
Phase I, 78 to 110 kg of BW			
No.[2]	20	20	
Initial weight, kg	78.23	77.97	0.846
Final weight, kg	111.46	110.52	2.435
ADFI, kg	3.70	3.67	0.169
ADG, kg	1.18	1.17	0.078
G:F, g of liveweight gain/kg of feed	319	320	17.070
Phase II, 110 to 160 kg of BW			
No.[2]	12	12	
Initial weight, kg	113.78	111.88	1.733
Final weight, kg	161.48	158.76	3.924
ADFI, kg	4.13	4.05	0.210
ADG, kg	1.06	1.01	0.081
G:F, g of liveweight gain/kg of feed	245	240	5.607

[1]The data were analyzed separately for animals at 110 kg and 160 kg of BW.
[2]Number of replicate pens per diet. Pigs were housed 1 per pen.

Carcass Characteristics and Meat Quality

Dietary treatment did not affect carcass characteristics (Table 4). Muscle thickness did not differ by weight at slaughter. However, all other variables were different in pigs slaughtered at 110 kg of BW compared with pigs slaughtered at 160 kg of BW. Obviously, warm carcass weight and dressing yield percentage were greater ($P < 0.05$) in 160 kg slaughtered pigs. Heavy pigs, as expected, were fatter than pigs of 110 kg of BW, as shown by greater values of backfat thickness ($P < 0.05$) and by lower values of lean percentage ($P < 0.05$). Loin weight was not affected by dietary treatment, but was greater ($P < 0.05$) in pigs slaughtered at 160 kg of BW than in pigs slaughtered at 110 kg of BW. The loin weight/carcass weight ratio was greater ($P < 0.05$) in pigs fed the linseed diet, slaughtered at both 110 and 160 kg of BW.

Body weight and dietary treatment did not affect pH at 24 h, or the coordinates L* and b* of the color at 24 h. The a* values were statistically different between pigs of 110 kg of BW and 160 kg of BW, with redder LM in pigs of 160 kg of BW ($P < 0.05$; Table 4).

Lipogenic Enzyme Activities and Total Lipids

There was no effect of dietary treatment on the activities of this lipogenic enzyme ($P > 0.05$) in LM or in backfat (Table 5). Malic enzyme activity was greater ($P < 0.05$) in backfat of heavy pigs, but not in LM ($P > 0.05$).

Total lipid content in LM and in backfat was not affected by dietary treatment, as shown in Table 5. The slaughter weight of animals affected total lipid content

Table 4. Effects of diet (control diet: 2.5% sunflower oil; linseed diet: 5% extruded linseed) and liveweight at slaughter, 110 kg of BW (BW_{110}) or 160 kg of BW (BW_{160}), on carcass characteristics and meat quality (least-squares means)

	BW_{110}		BW_{160}			Significance[1]		
Item	Control diet	Linseed diet	Control diet	Linseed diet	SE	D	BW	D × BW
No. of animals	8	8	12	12				
Warm carcass weight, kg	87.57	86.50	132.87	130.41	4.435	NS	*	NS
Dressing yield, %	80.60	80.12	82.28	82.15	0.749	NS	*	NS
G1,[2] mm	18.78	18.29	27.91	28.39	1.696	NS	*	NS
G2,[3] mm	15.67	16.14	25.36	24.77	1.495	NS	*	NS
M2,[4] mm	61.44	65.71	62.36	64.00	4.029	NS	NS	NS
Lean, %	61.17	62.08	53.13	51.98	3.163	NS	*	NS
Loin, kg	11.00	11.25	16.46	16.51	0.703	NS	*	NS
Loin weight/carcass weight, %	12.84	13.30	12.66	12.94	0.310	*	NS	NS
LM								
pH 24 h	5.47	5.58	5.59	5.59	0.098	NS	NS	NS
Color 24 h								
L*	57.05	56.80	57.20	56.15	2.418	NS	NS	NS
a*	8.35	7.73	9.66	8.51	0.941	NS	*	NS
b*	5.11	4.89	6.06	5.20	0.864	NS	NS	NS

[1]D = diet effect; BW = BW at slaughter effect; D × BW = diet × liveweight at slaughter effect.
[2]G1 = the thickness of backfat (including rind) in millimeters, measured 8 cm off the midline of the split carcass between the third and fourth from the last lumbar vertebrae.
[3]G2 = the thickness of backfat (including rind) in millimeters, measured 6 cm off the midline of the split carcass between the third and fourth from the last ribs.
[4]M2 = the thickness of LM in millimeters, measured 6 cm off the midline of the split carcass between the third and fourth from the last ribs.
*$P < 0.05$; NS = not significant.

of LM, which was 24% greater in LM of 160-kg slaughtered pigs but did not affect the lipid content of backfat ($P > 0.05$; Table 5).

Longissimus Muscle and Backfat Fatty Acid Composition

The dietary treatment caused some significant changes in the fatty acid composition of LM (Table 6). Longissimus muscles of pigs fed the linseed diet showed a greater content of total n-3 PUFA ($P < 0.001$) and particularly of ALA (C18:3n-3; $P < 0.001$), EPA (C20:5n-3; $P < 0.001$), docosapentaenoic acid (**DPA**, C22:5n-3; $P < 0.001$), and DHA (C22:6n-3; $P < 0.001$). The percentage of ALA increased 3 fold, EPA increased 4-fold, and those of DPA and DHA were nearly doubled in linseed-fed pigs compared with the control pigs. Longissimus muscle composition of pigs fed the control diet did not show a greater content of n-6 PUFA, than LM of pigs fed the linseed diet. As a consequence of these changes in the fatty acid composition of the LM, the n-6:n-3 PUFA ratio was markedly reduced in the

Table 5. Effects of diet (control diet: 2.5% sunflower oil; linseed diet: 5% extruded linseed) and liveweight at slaughter, 110 kg of BW (BW_{110}) or 160 kg of BW (BW_{160}), on malic enzyme activity and total lipid content (%) of LM and backfat (least squares means)

	BW_{110}		BW_{160}			Significance[1]		
Item	Control diet	Linseed diet	Control diet	Linseed diet	SE	D	BW	D × BW
No. of animals	8	8	12	12				
Malic enzyme activity[2]								
LM	1.13	1.23	1.02	1.01	0.233	NS	NS	NS
Subcutaneous adipose tissue	0.49	0.50	0.79	0.71	0.211	NS	*	NS
Total lipids,[3] %								
LM	1.89	1.79	2.46	2.11	0.435	NS	*	NS
Subcutaneous adipose tissue	68.66	68.77	70.79	69.49	3.182	NS	NS	NS

[1]D = diet effect; BW = BW at slaughter effect; D × BW = diet × liveweight at slaughter effect.
[2]Malic enzyme activity: nmol of NADPH formed per min per mg of protein.
[3]Data expressed as percentage of wet weight.
*$P < 0.05$; NS = not significant.

Table 6. Effects of diet (control diet: 2.5% sunflower oil; linseed diet: 5% extruded linseed) and liveweight at slaughter, 110 kg of BW (BW_{110}), or 160 kg of BW (BW_{160}) on fatty acid composition (g/100 g of fatty acids) of LM (least squares means)

	BW_{110}		BW_{160}			Significance[1]		
Fatty acid	Control diet	Linseed diet	Control diet	Linseed diet	SE	D	BW	D × BW
No. of animals	8	8	12	12				
C14:0	1.22	1.25	1.30	1.32	0.088	NS	NS	NS
C14:1	0.07	0.07	0.05	0.05	0.010	NS	***	NS
C16:0	24.59	24.31	24.98	24.98	0.675	NS	NS	NS
C16:1	3.40	3.41	3.46	3.48	0.249	NS	NS	NS
C18:0	11.86	12.02	12.18	12.36	0.616	NS	NS	NS
C18:1n9	40.54	39.93	43.74	42.28	1.295	NS	***	NS
C18:2n6	13.24	12.33	10.10	9.61	1.364	NS	***	NS
C18:3n3	0.48	1.48	0.35	1.49	0.105	***	NS	NS
C20:0	0.15	0.17	0.18	0.16	0.039	NS	NS	NS
C20:1	0.66	0.68	0.73	0.68	0.068	NS	NS	NS
C20:2n6	0.39	0.35	0.30	0.30	0.039	NS	***	NS
C20:3n3	0.30	0.29	0.25	0.27	0.051	NS	NS	NS
C20:4n6	2.44	2.42	1.91	1.82	0.417	NS	**	NS
C20:5n3	0.12	0.37	0.07	0.39	0.057	***	NS	NS
C22:1n9	0.04	0.10	0.03	0.08	0.020	***	NS	NS
C22:5n3	0.35	0.59	0.25	0.57	0.094	***	NS	NS
C22:6n3	0.07	0.14	0.06	0.09	0.025	***	*	NS
C24:1	0.08	0.09	0.06	0.07	0.014	NS	**	NS
SFA	37.82	37.75	38.64	38.82	1.182	NS	NS	NS
MUFA	44.79	44.28	48.07	46.64	1.403	NS	***	NS
PUFA	17.39	17.97	13.29	14.54	2.007	NS	***	NS
n-6 PUFA	16.07	15.10	12.31	11.73	1.752	NS	***	NS
n-3 PUFA	1.32	2.87	0.98	2.81	0.265	***	NS	NS
n-6:n-3 PUFA	12.17[c]	5.26[b]	12.56[c]	4.17[a]	0.473	***	NS	***
Total fatty acids[2]	1,278	1,268	1,956	1,668	358.603	NS	**	NS

[a–c]Means in the same row without common superscripts differ ($P < 0.05$).
[1]D = diet effect; BW = BW at slaughter effect; D × BW = diet × liveweight at slaughter effect.
[2]Expressed as mg/100 g of muscle.
*$P < 0.05$; **$P < 0.01$; ***$P < 0.001$; NS = not significant.

muscle of pigs fed linseed diets ($P < 0.001$): 43 and 33% for 110- and 160-kg slaughtered pigs, respectively, vs. control values.

The dietary treatment produced significant changes in the fatty acid composition of backfat (Table 7). Backfat of pigs fed the linseed diet showed a high content of total n-3 PUFA ($P < 0.001$); ALA (C18:3n-3; $P < 0.001$), eicosatrienoic acid (C20:3n-3; $P < 0.001$), EPA (C20:5n-3; $P < 0.001$), DPA (C22:5n-3; $P < 0.001$), and DHA (C22:6n-3; $P < 0.05$). The percentage of ALA in the pigs fed the linseed diet increased 3 fold in 110-kg pigs and nearly increased 4-fold in 160-kg pigs, DPA increased 3-fold in 110-kg pigs and doubled in 160-kg pigs, eicosatrienoic acid nearly doubled in 160-kg pigs, EPA nearly doubled in 110-kg pigs and increased 3-fold in 160-kg pigs, and DHA increased 3-fold in 110-kg pigs and nearly doubled in 160-kg pigs, compared with the control. The backfat composition of pigs fed a control diet showed a higher content of n-6 PUFA, and particularly of LA (C18:2n-6), than the backfat of pigs fed the linseed diet. The ARA (C20:4n-6) content was greater in the backfat of pigs fed the linseed diet than in pigs fed the control diet. As a consequence of these changes in the fatty acid composition of backfat, the n-6:n-3 PUFA ratio was markedly reduced in the backfat of pigs fed the linseed diet ($P < 0.001$; 30 and 22% for 110- and 160-kg slaughtered pigs, respectively, vs. control values). No differences in the content of SFA, MUFA, and PUFA were observed in LM or in backfat as a result of the different diets. The linseed diet in LM produced a n-6:n-3 ratio of about 5 in 110-kg pigs and of about 4 in 160-kg pigs ($P < 0.05$), and in backfat a n-6:n-3 ratio of about 3.5 in 110-kg pigs and of about 2 in 160-kg pigs ($P < 0.05$).

Fatty acid content of LM was also affected by the weight of the animals. In particular, the proportions of MUFA and total fatty acids content were significantly greater, and total PUFA (particularly n-6 PUFA, LA, and ARA) was decreased in LM of 160-kg pigs than 110-kg pigs. The fatty acid content of backfat, like that of LM, was also affected by the weight of the animals. Linoleic acid, total PUFA, and n-6 PUFA were significantly decreased, and ALA, MUFA, n-3 PUFA, and the total fatty acids content were greater in the backfat of 160-kg pigs compared with 110-kg pigs.

Some interactions BW × diet were observed in backfat fatty acids composition for C14:1 (myristoleic acid), C18:2n-6 (LA), C18:3n-3 (ALA), C20:3n-3 (eicosatrienoic acid), n-6 PUFA, n-3 PUFA, and n-6:n-3 PUFA ratio. In linseed fed pigs C14:1 (myristoleic acid), C18:2n-6

Table 7. Effects of diet (control diet: 2.5% sunflower oil; linseed diet: 5% extruded linseed) and liveweight at slaughter, 110 kg of BW (BW_{110}) or 160 kg of BW (BW_{160}), on fatty acid composition (g/100 g of fatty acids) of backfat (least-squares means)

	BW_{110}		BW_{160}			Significance[1]		
Fatty acid	Control diet	Linseed diet	Control diet	Linseed diet	SE	D	BW	D × BW
Number of animals	8	8	12	12				
C14:0	1.18	1.20	1.18	1.15	0.050	NS	NS	NS
C14:1	0.06[bc]	0.06[c]	0.05[b]	0.04[a]	0.005	NS	***	*
C16:0	24.96	24.91	24.50	24.90	0.664	NS	NS	NS
C16:1	1.99	2.04	1.77	1.74	0.142	NS	***	NS
C18:0	14.38	14.68	14.46	15.12	0.885	NS	NS	NS
C18:1n9	39.27	38.46	40.27	39.86	0.821	NS	**	NS
C18:2n6	15.00[c]	12.74[b]	14.52[c]	10.45[a]	0.790	***	***	*
C18:3n3	0.90[a]	3.29[b]	0.78[a]	3.82[c]	0.156	***	*	***
C20:0	0.03	0.05	0.13	0.16	0.052	NS	***	NS
C20:1	0.98	0.98	1.01	1.06	0.096	NS	NS	NS
C20:2n6	0.67	0.60	0.74	0.56	0.069	**	NS	NS
C20:3n3	0.34[a]	0.58[b]	0.36[a]	0.80[c]	0.030	***	***	***
C20:4n6	0.04	0.05	0.03	0.03	0.005	***	NS	NS
C20:5n3	0.02	0.01	0.03	0.04	0.007	***	†	NS
C22:1n9	0.02[a]	0.03[b]	0.01[a]	0.04[c]	0.005	***	NS	*
C22:5n3	0.04	0.11	0.0	0.12	0.023	***	NS	NS
C22:6n3	0.02	0.06	0.03	0.04	0.017	*	NS	NS
C24:0	0.07	0.05	0.11	0.04	0.039	*	NS	NS
C24:1	0.03	0.03	0.03	0.03	0.010	NS	NS	NS
SFA	40.62	41.03	40.24	41.37	1.321	NS	NS	NS
MUFA	42.35	41.61	43.13	42.77	0.924	NS	*	NS
PUFA	17.03	17.36	16.63	15.86	0.937	NS	*	NS
n-6 PUFA	15.71[c]	13.53[b]	15.15[c]	11.04[a]	0.790	***	***	*
n-3 PUFA	1.32[a]	3.83[b]	1.48[a]	4.82[c]	0.167	***	**	***
n-6:n-3 PUFA	11.90[c]	3.53[b]	10.24[c]	2.29[a]	0.541	***	NS	*
Total fatty acids[2]	52.22	51.47	62.77	61.26	3.554	NS	***	NS

[a–c]Means in the same row without common superscripts differ ($P < 0.05$).
[1]D = diet effect; BW = BW at slaughter effect; D × BW = diet × liveweight at slaughter effect.
[2]Expressed as g/100 g of tissue.
†$P < 0.10$; *$P < 0.05$; **$P < 0.01$; ***$P < 0.001$; NS = not significant.

(LA) and n-6 PUFA content and n-6:n-3 PUFA ratios were decreased in heavy pigs compared with light pigs and C18:3n-3 (ALA), C20:3n-3 (eicosatrienoic acid) and n-3 PUFA content were greater in heavy compared with light pigs. Only one interaction was observed in the LM fatty acids composition for n-6:n-3 PUFA ratio of pigs fed linseed, which was decreased in heavy compared with light pigs.

Iron-Induced Lipid Oxidation

Time of iron-induced lipid oxidation significantly affected TBARS values ($P < 0.001$) in pigs slaughtered at 110 kg of BW, but there was no significant interaction between time and treatment (Figure 1). Time of iron-induced lipid oxidation and dietary treatment significantly affected TBARS values ($P < 0.001$ and $P < 0.05$, respectively), and there was a significant interaction between time and treatment in pigs slaughtered at 160 kg of BW (Figure 2). In LM of control heavy pigs, however, TBARS values were always less than 1 nmol of MDA/mg of protein (Figure 2), in contrast to TBARS values of LM in light pigs (Figure 1). In the LM of heavy pigs fed on the linseed diet, iron-induced lipid oxidation increased at 200 min, whereas in LM of light pigs fed on the linseed diet, iron-induced lipid oxidation increased at 120 min.

DISCUSSION

The Effects of Fatty Acid Composition of Diet and Slaughtering Weight on Growth Performance

This study was designed to investigate the use of extruded linseed to increase the n-3 PUFA content of the LM and adipose tissue of pigs slaughtered at 2 different weights, 1 of which is characteristic in Italy (160 kg of BW) and the other in other European nations (110 kg of BW). Extruded linseed did not affect growth parameters in pigs slaughtered at 110 or 160 kg of BW (ADFI, ADG, or G:F) similar to the studies of Romans et al. (1995a) and Riley et al. (2000) who used ground flaxseed and extruded linseed as sources of n-3 PUFA.

Consistent with the results of Cisneros et al. (1996), slaughtering weight did not affect ADG in pigs from 100 to 120 or 160 kg, respectively. In contrast, Ellis et al. (1996) showed that ADG decreased with increasing

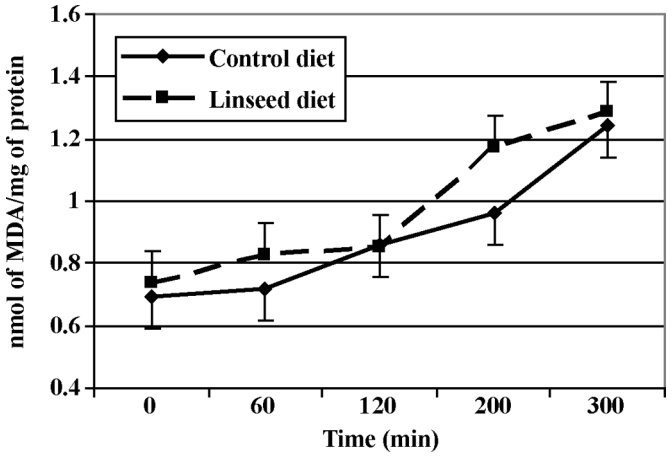

Figure 1. Effect of diet (control diet: 2.5% sunflower oil; linseed diet: 5% extruded linseed) on iron-induced lipid oxidation, expressed as nanomoles of malondialdehyde (MDA)/milligram of protein, of LM from pigs slaughtered at 110 kg of BW. This was assessed by determining the production of thiobarbituric acid-reactive substances (TBARS), mostly MDA, after incubation with ferrous sulfate as a promoter of peroxidation. Values are means ± SEM, 8 animals per group. Significant effect ($P < 0.001$) of time of iron-induced lipid oxidation.

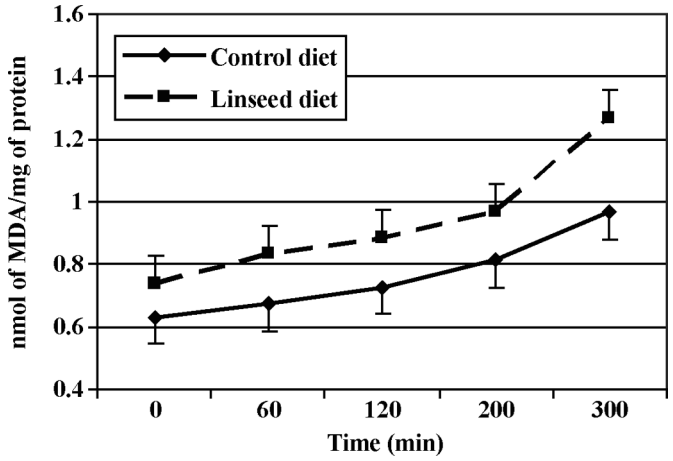

Figure 2. Effect of diet (control diet: 2.5% sunflower oil; linseed diet: 5% extruded linseed) on iron-induced lipid oxidation, expressed as nanomoles of malondialdehyde (MDA)/mg of protein, of LM from pigs slaughtered at 160 kg of BW. This was assessed by determining the production of thiobarbituric acid-reactive substances (TBARS), mostly MDA, after incubation with ferrous sulfate as a promoter of peroxidation. Values are means ± SEM, 12 animals per group. Significant effects of dietary treatment ($P < 0.05$), of time of iron-induced lipid oxidation ($P < 0.001$), and interaction of time of iron-induced lipid oxidation × dietary treatment ($P < 0.05$).

BW. These conflicting results may be explained by the genetic propensity for growth rate of pigs used in these studies. In regards to ADFI, our results are consistent with those of other studies (Cisneros et al., 1996) that showed that ADFI increased linearly with age and liveweight, due to greater BW. Also, our results are in agreement with the results of Latorre et al. (2004), who observed a linear decrease of G:F with increasing liveweight.

Dietary and Weight Effect on Carcass Characteristics and Meat Quality

The absence of a dietary effect on carcass traits of light and heavy pigs is consistent with previous observations using heavy pigs, of Corino et al. (2002), who used dietary rapeseed oil, and, results using light pigs (Riley et al., 2000). These authors showed no effects of various dietary linseed additions on warm carcass weight, dressing yield, muscle and fat thickness, lean percentage, or cold loin weight. As observed by other researchers (Correa et al., 2006), there were significant increases in hot carcass weight and dressing yield as slaughtering weight increased. In addition, backfat increased linearly with age as reported by Ellis et al. (1996) and Cisneros et al. (1996). According to Correa et al. (2006), slaughtering weight did not affect loin weight as a percentage of carcass weight. Our results concerning meat quality traits are also consistent with results in heavy pigs of Corino et al. (2002), and with those in light pigs in Riley et al. (2000), who did not find any dietary effect on pH at 24 h or on color coordinates (L*, a*, and b*). However, Van Oeckel et al. (1996) observed a significant difference in color coordinate L* in light pigs fed linseed. Previous studies showed little or no effect of slaughter weight on technological meat quality traits (Cisneros et al., 1996; Latorre et al., 2004; Correa et al., 2006). Our results are in agreement with those of Candek-Potokar et al. (1998), who reported no effect of age on pH. The increase of the a* value of the meat with age is in agreement with results reported by Cannata et al. (2007).

Dietary and Weight Effects on Activities of Lipogenic Enzymes and on Total Lipids

In the pig, malic enzyme, with glucose-6-phosphate dehydrogenase, is a major enzyme involved in supplying NADPH for the reductive biosynthesis of fatty acids (Young et al., 1964). Our results are in agreement with those obtained by Mourot et al. (1994) showing the absence of a significant effect of diets rich in LA on pig muscle lipogenic enzyme activities. Our results in adipose tissue differ from previous results obtained in pigs (Allee et al., 1972; Kouba and Mourot, 1999), which showed a significant effect of diets with different fatty acid compositions on the lipogenic enzyme activities of pig adipose tissue. Malic enzyme activity is not weight-related in LM, although malic enzyme activity is decreased in heavy pigs compared with light

pigs. This result is in contrast with the observations of Mourot and Kouba (1998), who studied muscle growth in (20 to 100 kg of BW) Large White and Meishan pigs. In backfat, malic enzyme activity is weight-related in accordance with previous observations of Mersmann et al. (1976) on the extramuscular adipose tissue of young pigs. However, our results differ from those previously reported that showed a decrease in malic enzyme activity in the extramuscular adipose tissue of older and heavier pigs (Allee et al., 1971; Mourot et al., 1995). Whether malic enzyme is actually involved in the regulation of the deposition of intramuscular lipids deserves further investigation.

In the present study, the total lipid content of LM and backfat were not affected by dietary treatment, which is in agreement with other studies showing the affects of different fats in light pigs (Romans et al., 1995a,b; Riley et al., 2000). Pig weight at slaughter did not affect the total lipid content of backfat. However, animal weight at slaughter did affect the total lipid content of LM, and the values were notably greater in the LM of heavy pigs compared with light pigs.

Dietary Effects on Fatty Acid Composition

Diet is known to affect the fatty acid composition of pig adipose tissue, particularly back and perirenal fat (Fontanillas et al., 1997). Leszczynski et al. (1992) showed that LM intramuscular fat was less affected by diet than subcutaneous fat. This may be due to weak deposition of absorbed fat in muscle tissue (Vernon, 1992), or the greater amount of membrane lipids in intramuscular fat containing high quantities of PUFA, which are less sensitive to dietary variations. However, in the present study, there were significant changes in the fatty acid compositions of LM. In particular, dietary linseed increased C18:3n-3 (ALA) concentrations and decreased the n-6:n-3 PUFA ratio in LM, and allowed C18:3n-3 (ALA) to compete more effectively with C18:2n-6 (LA) for the pathways responsible for producing longer-chain fatty acid products. Our results are in agreement with those reported previously in light pigs by Enser et al. (2000), who observed significant increases in all n-3 PUFA in muscle with linseed feeding. Specifically, the increases were: EPA 100%, ALA 55%, DHA 35%, and DPA 29%. A significant increase limited only to some n-3 PUFA was observed by Ahn et al. (1996), who showed that increasing the content of α-linolenic acid in the diet to 35 g/kg by the inclusion of linseed resulted in an increase in the C18:3n-3 (ALA), C20:5n-3 (EPA), and C22:5n-3 (DPA) content in the loin muscle, but not in C22:6n-3 (DHA). These results confirmed by Hoz et al. (2003) and by Riley et al. (2000). For heavy pigs our results contrast with those previously reported by Corino et al. (2002), who showed significant differences in the LM composition of animals fed diets supplemented with 2.5% rapeseed oil during the finishing phase [only for C18:3n-3 (ALA), but not for any other n-3 fatty acid]. This can be explained by the observation that rapeseed oil has a very low content of n-3 PUFA compared with extruded linseed and the different dietary vitamin E supplementation used.

For backfat of pigs fed the linseed diet, the content of n-3 PUFA was greater than in pigs fed the control diet. In fact, in backfat of pigs fed the control diet, the n-6:n-3 PUFA ratio value was 11, as in LM, but the n-6:n-3 ratio of backfat of pigs fed the linseed diet was about 3, which was decreased compared with LM. In agreement with our results, Enser et al. (2000) observed significant increases in DHA in adipose tissue with long-term linseed feeding. However, in several studies DHA content was not affected in adipose tissue by a greater amount of ALA in the diet (Cunnane et al., 1990; Ahn et al., 1996). These authors showed that dietary linseed supplementation increased concentrations of ALA and EPA, but not of DHA, in adipose tissue. In contrast, Romans et al. (1995b) fed pigs linseed at 150 g/kg, for different lengths of time, and observed an increase in the quantities of ALA, EPA, and DHA in the inner and outer layers of backfat. Rossi and Corino (2002) observed significant differences in the backfat composition of heavy pigs fed diets supplemented with 2.5% rapeseed oil during their growing-finishing phase only for C18:3n-3 (ALA). As observed for LM, this can be explained by the different content of n-3 PUFA of rapeseed oil compared with that of extruded linseed.

Slaughtering weight affected the fatty acid composition for both adipose and muscle tissue. In particular, a decreased PUFA and n-6 PUFA content in LM and in backfat, with a simultaneous increase in MUFA was observed in 160-kg pigs. Animal weight at slaughter affected total fatty acids content of LM and backfat, and was greater in tissues from heavy compared with light pigs. With increasing age and weight, the partition of ingested energy turns from muscular to adipose tissue growth, which increases the ratio between de novo synthesized fatty acids (mainly C18:1, oleic acid; Enser, 1991) and those derived from dietary fatty acids (C18:2n-6, LA, and C18:3n-3, ALA, in our diets). In adipose tissue, our results are in agreement with Virgili et al. (2003), who showed a reduction of total PUFA in ham subcutaneous adipose tissue, particularly in LA and ALA, and an increase in MUFA. Nevertheless, subcutaneous fat from pigs slaughtered at 160 kg, characterized by decreased percentages of PUFA, appears more suitable for long-curing pork production.

It should be noted that PUFA of the n-3 family have different modes of incorporation in tissues. Our results are in agreement with those of Nguyen et al. (2003), who observed that EPA is preferentially stored in the organs or muscle rather than in adipose tissue, and DHA is more efficiently incorporated into adipose tissue than EPA. The authors explain the difference between the 2 fatty acids by the observation that EPA, but not DHA, can be converted into eicosanoids. However, in our study, there were greater quantities of EPA, DPA, and DHA in LM than in adipose tissue. Øverland et al. (1996) argue that greater concentrations of EPA,

DPA, and DHA in the lipid of muscle tissue compared with subcutaneous adipose tissue were probably due to greater contents of phospholipids in intramuscular fat compared with subcutaneous fat and that PUFA is preferentially incorporated into phospholipids rather than into triacylglycerol.

Our results for LA are in agreement with those of Nguyen et al. (2003); in fact, LA is stored preferentially in adipose tissue rather than in muscle. Likely, the intake of LA is much greater than the amount required for membrane synthesis, so a high proportion of the intake is channeled into a storage site (adipose tissue). However, in contrast with Nguyen et al. (2003), in our study, ALA was stored much more in adipose tissue than in muscle. This difference could be due to a different quantity of ALA in the diets, different sources of n-3 PUFA in the diets, or the length of feeding.

From our results, and those of other authors (Enser et al., 2000), it appears possible that the pig may use ALA for elongation and desaturation, which explains the moderate effect of dietary treatment observed on long chain n-3 PUFA. However, the reason for a greater or decreased deposition of n-3 PUFA is unclear. Some authors, such as Romans et al. (1995a,b) and Enser et al. (2000), reported that n-3 PUFA content could be dependent on the time of administration of the source of n-3 PUFA, and by several factors including the rate of synthesis, rate of conversion to other fatty acids, rate of loss (i.e., oxidation or conversion to other metabolites), specificity of acylating enzymes, and the amount and type of lipids into which they may be incorporated.

Effects of Diet and Weight on Iron-Induced Lipid Oxidation of Longissimus Muscle

The n-3 fatty acids are particularly susceptible to lipid oxidation, and small increases in the concentration of these fatty acids in the phospholipid fraction of fat may be significant in the progress of oxidation (Lopez-Bote et al., 1997). Lipid oxidation in food is also a problem for the sensory characteristics of pork and a risk to human health. In fact, some molecules produced by lipid oxidation, particularly oxides of cholesterol, could be atherogenic agents and may be mutagenic, carcinogenic, or cytotoxic (Kubow, 1990). The oxidative stability of the LM from heavy pigs fed the linseed diet was lower than for controls, but the oxidative stability was not affected by dietary treatment in light pigs.

Values of TBARS of LM were greater in light pigs than in heavy pigs that were fed the control diet. Different liveweights influence iron-induced lipid oxidation of LM, as assessed by the TBARS test; this is due to a greater content in MUFA in the LM of heavy pigs than of light pigs, and to a greater content in PUFA, particularly n-6 PUFA, in the LM of light pigs than heavy pigs. The decreased value of TBARS was probably due to inclusion in the diets of a greater concentration of antioxidants, which increased the oxidative stability of the raw LM, and by the time of dietary supplementation (i.e., total vitamin E intake) in heavy pigs vs. light pigs. This observation is consistent with the results of Corino et al. (1999) and Hoz et al. (2003), who showed greater oxidative stability in the muscle of pigs fed with diets containing high concentration of antioxidants.

This study confirmed that it is possible to improve the nutritional quality of pork through feeding of live pigs. In particular, dietary enrichment with raw materials rich in linolenic acid increased not only linolenic acid content, but also that of its derivates EPA, DPA, and DHA. Quality problems that might have been expected as a result of this type of tissue manipulation, concerning oxidation resistance, appear to have been controlled by dietary supplementation with vitamin E and selenium in pigs slaughtered at 110 kg of BW. Dietary extruded linseed reduces the n-6:n-3 PUFA ratio and a value of 5 was obtained as recommended by ISSFAL (2004).

An increase in slaughter weight of pigs from 110 to 160 kg impaired live pig performance and carcass characteristics important for meat intended for immediate consumption. However, an increased slaughter weight resulted in greater intramuscular fat content and greater SFA and MUFA content in backfat that positively affected the technological quality and oxidative stability of meat.

It may be possible to improve the nutritional quality of fresh meat and of raw material for transforming products, but there is a need to further evaluate the impact of extruded linseed on long-cured meat products (e. g., Parma ham) and to redefine antioxidant needs related to the supply of n-3 PUFA in pig feeding.

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