Use of α-Tocopherol Acetate to Improve Fresh Pig Meat Quality of Full-Fat Rapeseed-Fed Pigs

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ABSTRACT: Thirty-two pigs were allocated to one of four diets, FFRD0 and FFRD200, containing full-fat rapeseed (FFR), 150 g/kg [25–50 kg liveweight (LW)], and 250 g/kg (50–90 kg LW), or CD0 and CD200, containing equivalent quantities of rapeseed meal and 34 g/kg (25–50 kg LW) or 59.2 g/kg (50–90 kg LW) coconut oil and lard (0.5:0.5, w/w). Diets FFRD200 and CD200 were supplemented with 200 mg/kg α-tocopherol acetate (ATA). ATA supplementation significantly ($P < 0.001$) reduced muscle drip loss. The melting point (°C) of subcutaneous fat was significantly lowered by FFR ($P < 0.001$) but increased by ATA supplementation ($P < 0.05$). Tissue α-tocopherol (AT) concentrations were significantly increased by ATA supplementation. *Longissimus dorsi* AT concentration was positively correlated with AT concentration in subcutaneous fat ($r = 0.86$) and in plasma at 35 ($r = 0.65$) and 77 ($r = 0.85$) days of feeding ($P < 0.001$). In both *L. dorsi* and subcutaneous adipose tissue lipids, FFRD caused a significant ($P < 0.001$) decrease in the ratio of n-6 to n-3 fatty acids and a significant ($P < 0.001$) increase in the ratio of polyunsaturated to saturated fatty acids. AT supplementation significantly reduced the susceptibility of *L. dorsi* and subcutaneous fat to lipid oxidation during storage at 4°C for up to 16 d. For all dietary treatments and storage times, lipid oxidation [mg malondialdehyde (MDA)/kg muscle] was greater in the surface layer (0–2.5 mm) of *L. dorsi* than below the surface (2.5–5 mm). Oxidative stability of *L. dorsi* lipids to iron-induced lipid peroxidation was significantly improved ($P < 0.001$) by AT supplementation. Meat from pigs fed FFRD diets was significantly less stable to iron-induced oxidation (nmoles MDA/mg protein) at the longer incubation periods (100 and 200 min). The susceptibility of *L. dorsi* to iron-induced lipid oxidation decreased as the ratio of the tissue concentration of AT to unsaturated fatty acid increased.

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KEY WORDS: Fatty acid composition, full-fat rapeseed, lipid oxidation, pig meat, α-tocopherol.

There is now a considerable body of knowledge that shows the importance of unsaturated fatty acids, especially n-6 and n-3 polyunsaturated fatty acids (PUFA), as components of a healthy human diet (1). Pigs and poultry are able to incorporate dietary fatty acids directly into adipose and muscle tissue lipids, and thus in these species it is possible to modify the fatty acid profile of muscle tissue by the strategic use of unsaturated dietary fat sources (2,3). Rapeseed oil is highly unsaturated and may contain as much as 7% saturated fatty acids, 61% monounsaturated fatty acids (predominantly C18:1n-9), and 32% PUFA (mainly C18:2n-6) and therefore has considerable potential as an unsaturated fat source in monogastric diets. However, the melting point of fatty acids decreases and their susceptibility to oxidative deterioration increases as the degree of unsaturation increases (4,5). Thus, when diets with high levels of unsaturated fatty acids are fed to pigs, adipose tissue firmness may be reduced and meat storage and processing properties may be adversely affected owing to accelerated peroxidation of tissue lipids. A decrease in the firmness of meat-associated adipose tissue may cause the fat tissue to separate from the muscle tissue during cooking and reduce the sliceability of cooked joints. Increased peroxidation of muscle membrane lipids may be reflected in excessive and unsightly fluid loss from the uncooked joints of meat (6,7) and can lead to the development of unacceptable flavors and smells. These aspects of the use of unsaturated fats in pig diets must be balanced against the nutritional benefits to the consumer of increased PUFA intake.

Peroxidative changes in muscle lipids can be reduced by increasing the tissue content of protective antioxidants, particularly α-tocopherol (AT), by dietary supplementation (8). The present study was carried out to examine the effect of feeding diets that contained full-fat rapeseed (FFR), with or without supplemental α-tocopherol acetate (ATA), on porcine adipose and muscle tissue fatty acid composition and AT content, melting point of subcutaneous (s/c) adipose tissue fat, muscle drip loss, and muscle lipid peroxidation.

EXPERIMENTAL PROCEDURES

Animals and diets. Thirty-two pigs (16 boars and 16 gilts), from a pure-bred Large White boar and F1 hybrid gilts (Large White × Landrace), with initial weights of 20 kg were individually fed the experimental diets from 25 to 90 kg liveweight (LW) (grower diet, 25–50 kg; finisher diet, 50–90 kg). A 2 × 2 factorial experimental design was employed in which the diets (CD with rapeseed meal and FFRD with

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TABLE 1
Composition of Experimental Diets

	Grower diets		Finisher diets	
Ingredients (g/kg)	Control (CD0 and CD200)	Full-fat rapeseed (FFRD0 and FFRD200)	Control (CD0 and CD200)	Full-fat rapeseed (FFRD0 and FFRD200)
Wheat	300.00	285.10	274.50	318.90
Barley	300.00	285.10	274.50	183.20
Full-fat rapeseed	—	150.00	—	250.00
Ext. rapeseed meal	90.00	—	150.00	—
Fat-filled whey[a]	67.80	—	118.40	—
Dried whey	—	33.90	—	59.20
Soybean meal 48% (HiPro)	173.30	177.10	159.90	165.80
White fish meal	50.00	50.00	—	—
Limestone	7.70	7.55	10.20	9.80
Dicalcium phosphate	3.75	3.70	5.40	5.60
Salt	4.14	4.40	4.00	4.61
Lysine hydrochloride	.81	.65	.60	.39
Minerals and vitamins[b]	2.50	2.50	2.50	2.50
Total	1000.00	1000.00	1000.00	1000.00

[a]Contains 25% coconut oil, 25% lard, and 50% whey.
[b]α-Tocopherol acetate at 200 g/ton added to diets CD200 and FFRD200.

whole FFR) were supplemented with two levels of ATA (0 and 200 mg/kg diet). The composition of the diets fed is shown in Tables 1 and 2. The control diets (CD) were formulated to contain a quantity of rapeseed meal equivalent to the rapeseed in the full-fat rapeseed diets (FFRD).

The feed intake of all pigs was restricted to 90% of their daily voluntary food intake, calculated as digestible energy (MJ) = $50 \times (1 - e^{-0.0204W})$ (9), on the basis of LW measured at the beginning of each week. Pigs were fed twice daily between 9–10 A.M. and 3–4 P.M. All pigs were weighed weekly. Heparinized blood samples (10 mL) were taken twice throughout the feeding experiment, at about the time of change-over from grower diet to finisher diet (35 d of feeding) and prior to slaughter (77 d of feeding). The blood plasma was prepared from whole blood by centrifugation at 1500 rpm for 10 min and was stored at −20°C prior to analysis.

TABLE 2
Calculated and Measured Constituents (g/kg) and α-Tocopherol (AT) Content (mg/kg) of Experimental Diets

	Grower diets		Finisher diets	
	Control (CD0 and CD200)	Full-fat rapeseed (FFRD0 and FFRD200)	Control (CD0 and CD200)	Full-fat rapeseed (FFRD0 and FFRD200)
Calculated composition				
Crude protein (CP)	220.00	220.00	200.00	200.00
Total lysine	12.70	12.70	11.00	11.00
Calcium	8.50	8.50	8.00	8.00
Phosphorus	6.50	6.50	6.00	6.00
Sodium	2.50	2.50	2.50	2.50
Oil	51.00	75.50	72.70	110.90
Digestible energy (DE) (MJ/kg)	14.32	14.32	14.72	14.72
Measured composition				
Dry matter (DM)	883.02	883.75	889.93	891.07
CP	211.97	209.78	195.91	200.16
Oil (ether extract)	52.86	69.41	74.98	94.14
Neutral detergent fiber (NDF)	147.13	150.71	154.10	156.23
Ash	54.55	55.89	56.61	55.66
DE[a]				
MJ/kg DM	15.93	16.10	16.01	16.39
MJ/kg	14.07	14.23	14.25	14.61

	CD0	CD200	FFRD0	FFRD200	CD0	CD200	FFRD0	FFRD200
AT (mg/kg)[b]	16	190	36	162	16	176	41	192

[a]Estimated from proximate constituents by the following regression equation (Ministry of Agriculture, Fisheries and Food, 1993): DE (MJ/kg DM) = 17.47 + (0.079 × % CP) + (0.158 × % oil) − (0.331 × % ash) − (0.140 × % NDF).
[b]Added to the diets as α-tocopherol acetate.

Pigs were individually selected for slaughter at about 90 kg LW. Hot-carcass weight and backfat thickness (P^2) were measured immediately after slaughter. Shoulder, midback, and rump fat thickness measurements were taken on the slaughter line in the cold room, and the chump joints were marked for cutting. The chump joints (taken 10 cm anterior to the symphysis pubis at right angles to the longitudinal axis of the body) were cut and delivered to the laboratory cold room within 24 h of slaughter. *Longissimus dorsi* and *Semimembranosus* muscles and s/c fat were separated within 48 h post mortem, and samples were prepared for the various analyses.

Drip loss and storage of tissues. Drip loss from *S. membranosus* of the chump joint was measured as described by Warriss (10). The melting point of the s/c fat was determined as outlined by Cocks and van Rede (11). For measurement of lipid oxidation, the *L. dorsi* of the chump joints were cut into four separate meat chops. One of the chops was put into a self-sealing plastic bag, immediately dipped into liquid nitrogen, and stored thereafter at −20°C until required for analysis. The remaining three chops were placed in uncovered petri dishes and refrigerated at 4°C for storage periods of 4, 8, and 16 d. After the appropriate storage period, two slices of meat, 2.5 mm thick, were removed from each chop to measure lipid oxidation in meat between 0–2.5 mm, and 2.5–5.0 mm below the surface of the sample. The s/c fat (together with the skin) of the chump joint was separated from the muscle within 48 h post mortem and cut into blocks of approximately 3.0 cm long by 3.0 cm wide. From two of the blocks, the fat (2.5 mm thick) was separated from the skin with a Kenwood food slicer, put into self-sealing plastic bags, frozen in liquid nitrogen, and thereafter stored at −20°C. The remaining blocks were placed in uncovered petri dishes and refrigerated in a cold room at 4°C for storage periods of 4, 8, and 16 d, after which time the fat was separated from the skin, placed into self-sealing bags, frozen in liquid nitrogen, and stored at −20°C until the lipid was extracted for thiobarbituric acid (TBA) analysis by the procedure described by Kates (12) as modified by Pikul *et al.* (13).

Oxidative stability of tissues. Noninduced lipid peroxidation in muscle samples was measured by the modified extraction TBA method of Salih *et al*. (14). Peroxidation of lipids extracted from the s/c fat was determined according to Pikul *et al.* (13).

Iron-induced peroxidation of muscle lipids was determined in muscle homogenates by using the modified procedure described by Kornbrust and Mavis (15) as supplied by Dr. Martin Frigg of F. Hoffmann-La Roche (Basel, Switzerland). The incubation times selected for the oxidative study were 0, 50, 100, and 200 min.

The protein content of tissue homogenates was determined as described by Lowry *et al.* (16).

AT contents of plasma and tissues. Plasma AT was isolated by a single-step hexane extraction in which 400 µL of plasma was mixed with 400 µL ethanol and 400 µL hexane. The mixture was vortexed thoroughly and centrifuged, and the hexane layer was removed for analysis.

AT in samples of *L. dorsi* and diet was extracted by the method of Taylor *et al.* (17), modified by the inclusion of an internal standard (2,2,5,7,8-pentamethylchroman, PMC). Extraction of AT in s/c fat samples was based on the method of Rottenmaier and Schuep (18) with PMC incorporated as internal standard.

AT was measured by high-performance liquid chromatography (HPLC) with a Varian HPLC system (Palo Alto, CA) and a Shimadzu RF-535 fluorescence detector (Tokyo, Japan) set at an excitation wavelength of 296 nm and an emission wavelength of 326 nm. Separation was achieved in a Partisil Si (250 mm × 4.6 mm) column (Alltech, Carnforth, United Kingdom). The mobile phase was *n*-hexane and 1,4-dioxane, programmed to change linearly from 95:5 (vol/vol) to 75:25 (vol/vol) over the 10-min run time.

Fatty acid analysis. Lipids present in samples of diets, *L. dorsi* and s/c fat were extracted by the method of Folch *et al.* (19) as described by Christie (20). Antioxidant (butylated hydroxytoluene at 0.01%) was added to all solvents used for homogenization. Fatty acid methyl esters (FAME) of the extracted lipids were prepared with 0.5 M sodium methoxide as described by Christie (21). The FAME were dissolved in 1 mL hexane and analyzed in a Philips Pye Unicam PU 4500 gas chromatograph (Cambridge, United Kingdom) (fitted with PU 4700 autoinjector) with a Supelco SP 2330 fused-silica capillary column (30 m × 0.75 mm i.d., 0.20 µm film thickness) (Bellefonte, PA). The fatty acids were identified by comparison with pure fatty acid reference mixtures and cod liver oil FAME (Sigma Chemical Co., Poole Dorset, United Kingdom).

Statistical analyses. Data were subjected to analysis of variance (ANOVA). Main effects of treatment were partitioned into those due to dietary fat source, those due to ATA supplementation, and the interaction between fat source and ATA supplementation. The relationships between AT concentrations in plasma, adipose tissue and muscle, and the muscle TBA-reactive substances (TBARS) content and AT/unsaturated fatty acid ratio were assessed by linear regression analysis.

RESULTS

Growth performance and carcass measurements. Final LW was significantly higher ($P > 0.05$) for pigs fed supplemental ATA compared to those given no additional ATA (97.4 vs. 93.9 kg). The feed conversion ratio (FCR) was significantly lower ($P > 0.05$) for those pigs fed CD diets. There were no other significant effects of diet or supplemental ATA on growth performance (Table 3) or measured carcass characteristics (Table 4) of the pigs.

Drip loss and subcutaneous fat melting point. Supplemental ATA significantly reduced ($P < 0.001$) drip loss of fresh pork strips. There was no significant effect of diet or interaction between diet and level of inclusion of ATA. The melting point of s/c fat from pigs given the control diets was significantly higher ($P < 0.001$) than that from pigs fed the FFRD diets. ATA supplementation significantly ($P < 0.05$) increased fat melting point. There was no signifi-

TABLE 3
Growth Performance and Feed Intake[a]

Diet	ATA suppl. (mg/kg feed)	*n*	Inital LW (kg)	Final LW (kg)	Time taken (d)	Feed intake (g/d)	LW gain (g/d)	FCR (g feed/g gain)
CD	0	8	23.9	93.1	91.3	1828.7	764.6	2.41
	200	8	24.3	98.4	88.1	1896.8	844.8	2.26
FFRD	0	8	23.8	94.6	93.0	1876.5	763.1	2.47
	200	8	23.9	96.3	93.4	1913.8	774.9	2.47
SED			1.00	2.27	3.38	48.82	33.19	0.10
Statistical significance								
Diet			NS	NS	NS	NS	NS	*
ATA			NS	*	NS	NS	NS	NS
Diet × ATA			NS	NS	NS	NS	NS	NS

[a]ATA = α-tocopherol acetate; LW, liveweight; FCR, feed conversion ratio; SED = standard error of the difference; NS = not significant ($P > 0.05$); * = $P < 0.05$; LW, liveweight. See Table 1 for other abbreviations.

cant interaction between diet and level of inclusion of ATA (Table 5).

Plasma and tissue AT concentrations. AT concentration in blood plasma, *L. dorsi* and s/c fat are presented in Table 6. Supplementary dietary ATA significantly increased ($P < 0.001$) adipose and muscle tissue and plasma AT concentrations. Between days 35 and 77, plasma AT concentration increased two- to threefold. The significant diet × ATA interaction in plasma at 77 d and in muscle and adipose tissue at slaughter indicates that AT concentrations were higher in pigs fed diet CD200 than in those fed diet FFRD200. *Longissimus dorsi* AT concentration was positively correlated with AT concentration in s/c fat ($r = 0.86$) and in plasma taken on days 35 ($r = 0.65$) and 77 ($r = 0.85$) of the feeding trial (Fig. 1).

Fatty acid composition. There were large differences in the fatty acid composition of the control and FFR diets (Table 7). In comparison with CD diets, FFRD contained markedly lower proportions of C12:0, C14:0, C16:0, and C18:0 and greater proportions of C18:1n-9, C18:2n-6, and C18:3n-3. These differences are reflected in the total saturated, monounsaturated, and PUFA concentrations and polyunsaturated/saturated (P/S) fatty acid ratios. For both, the grower and finisher diets FFRD had a lower n-6/n-3 ratio.

Differences in dietary fatty acid composition are clearly seen in the composition of the tissue fatty acids (Tables 8 and 9). Both *L. dorsi* and s/c adipose tissue from pigs fed CD diets contained significantly higher concentrations of C12:0 and C14:0, reflecting the inclusion of coconut oil-filled whey in these diets. Similarly, the higher concentration of C16:0 and C18:0 in CD diets is reflected in significantly higher concentrations of these fatty acids in the muscle and adipose tissue lipids of CD-fed pigs. The concentrations of C18:1n-9, C18:2n-6, and C18:3n-3 were significantly higher in tissues from FFRD-fed pigs. Inclusion of whole FFR in the diet significantly reduced the n-6/n-3 ratio of both muscle and adipose tissue lipids, largely due to the increased C18:3n-6 content of the tissue. P/S ratios were significantly greater in tissues from FFRD-fed pigs owing to the combined increase in their C18:2n-6 and C18:3n-3 content.

Oxidation of tissue lipid. Data on the oxidative stability of *L. dorsi* samples, stored at 4°C for up to 16 d, are shown in Table 10. There was no significant effect ($P > 0.05$) of treatments on muscle lipid oxidation at less than 48 h after slaughter. After 4 d of storage, lipid oxidation in muscle samples remained low. Malondialdehyde (MDA) content in the surface layer of muscle tissue (0–2.5 mm deep) (SF) from pigs fed FFRD diets was significantly ($P < 0.001$) greater than that from pigs fed control diets. ATA supplementation signifi-

TABLE 4
Carcass Measurements[a]

Diet	ATA suppl. (mg/kg feed)	*n*	Hot-carcass weight (kg)	Dressing percentage	Shoulder fat (mm)	Midback fat (mm)	Average rump fat (mm)	P^2 (mm)
CD	0	8	70.2	75.4	32.8	14.4	18.2	11.3
	200	8	74.3	75.4	32.1	14.6	18.9	12.3
FFRD	0	8	72.4	76.7	33.8	15.2	20.9	11.3
	200	8	71.9	74.8	29.9	13.2	17.5	10.9
SED			1.92	1.64	2.27	1.45	1.81	1.00
Statistical significance								
Diet			NS	NS	NS	NS	NS	NS
ATA			NS	NS	NS	NS	NS	NS
Diet × ATA			NS	NS	NS	NS	NS	NS

[a]NS = Not significant ($P > 0.05$). See Tables 1 and 3 for other abbreviations.

TABLE 5
Muscle Drip Loss and Subcutaneous Fat Melting Point[a]

Diet	ATA suppl. (mg/kg feed)	Muscle drip loss (% fresh tissue wt)	Subcutaneous fat melting point (°C)
CD	0	6.64	28.67
	200	5.28	29.04
FFRD	0	6.73	25.21
	200	5.96	26.88
SED		0.35	0.56
Statistical significance			
Diet		NS	***
ATA		***	*
Diet × ATA		NS	NS
n		5	6

[a]Average of duplicate determinations; NS = not significant ($P > 0.05$); * = $P < 0.05$; *** = $P < 0.001$; see Tables 1 and 3 for other abbreviations.

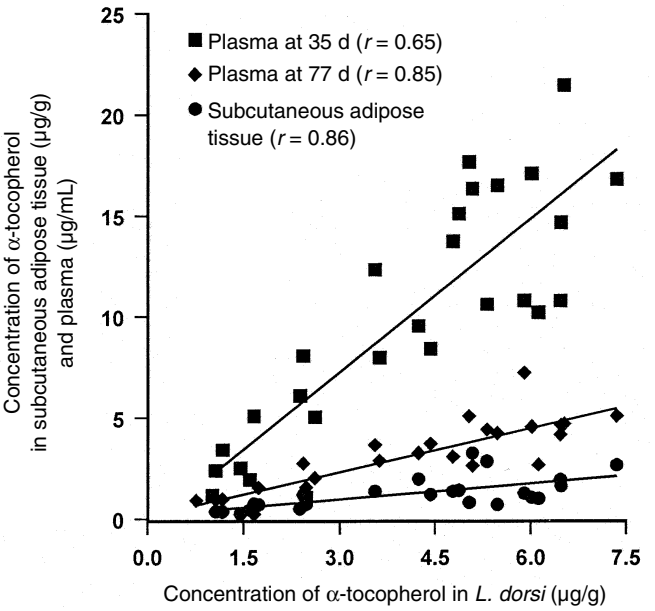

FIG. 1. Relationship between α-tocopherol concentration in *Longissimus dorsi,* subcutaneous adipose tissue, and plasma.

cantly reduced ($P < 0.05$) lipid oxidation in this layer on day 4. Little lipid oxidation occurred in muscle sampled below the surface (2.5–5 mm deep) (SS), and there were no significant effects of the dietary treatments. After 8 and 16 d of storage, levels of lipid oxidation in muscle samples were markedly higher than observed after 4 d of storage. On day 8, the MDA concentration was significantly higher in samples from pigs fed the FFRD diets. ATA supplementation significantly reduced the MDA contents of samples, and the significant diet × ATA interaction suggests that ATA supplementation was more effective in reducing lipid oxidation in muscle from pigs fed the control diets, presumably owing to the lower unsaturated fatty acid content of this tissue. A similar trend was also seen in subsurface muscle tissue, although the level of oxidation was approximately 50% lower than that in the surface layer. On day 16, there was no significant difference between the MDA concentration in samples taken between 0–2.5 mm from the SF in pigs fed control and FFRD diets. However, in samples taken between 2.5–5 mm SS of pigs fed the control diet, the MDA concentration was significantly lower than in similar samples taken from pigs fed FFRD. ATA supplementation significantly reduced lipid oxidation in these samples. Oxidation of the s/c fat, stored at 4°C for up to 16 d (Table 11), increased with duration of storage and was significantly greater in samples from FFRD-fed pigs but significantly lower in samples from pigs fed ATA-supplemented diets.

Iron-induced oxidation of muscle lipids. Oxidative stability of *L. dorsi* to iron-induced lipid oxidation (Table 12) was significantly improved ($P < 0.001$) by ATA supplementation at 0 and 50 min of incubation. However, there was no significant effect of diet on MDA concentration at these incubation periods. After 100 and 200 min of incubation, oxidation was significantly greater in samples from pigs fed FFRD diets compared to that in samples from pigs fed the control diets. Lipid oxidation in muscle from pigs, fed either the control or FFRD diets, was significantly reduced by dietary ATA supplementation when samples were subjected to induced oxida-

TABLE 6
Concentration of AT in Blood Plasma, *Longissimus dorsi* and Subcutaneous (s/c) Fat[a]

Diet	ATA suppl. (mg/kg feed)	Blood plasma (μg/mL)		*L. dorsi* (μg/g)	S/c fat (μg/g)
		Bled at 35 d	Bled at 77 d		
CD	0	0.49 ± 0.08 (6)	0.96 ± 0.17 (6)	1.31 ± 0.13 (8)	2.76 ± 0.55 (6)
	200	1.73 ± 0.30 (7)	5.04 ± 0.39 (7)	5.94 ± 0.30 (8)	15.07 ± 1.41 (8)
FFRD	0	0.67 ± 0.12 (6)	1.98 ± 0.29 (8)	2.71 ± 0.23 (5)	6.38 ± 1.02 (7)
	200	1.57 ± 0.28 (8)	3.50 ± 0.25 (8)	5.02 ± 0.35 (8)	12.74 ± 1.10 (8)
Statistical significance					
Diet		NS	NS	NS	NS
ATA		***	***	***	***
Diet × ATA		NS	***	***	*
r		0.65	0.85	—	0.86

[a]Mean ± SE. Figures in parentheses indicate number of samples analyzed; NS = not significant ($P > 0.05$); * = $P < 0.05$; *** = $P < 0.001$; *r* = correlation with *L. dorsi* AT content. See Tables 1–3 for abbreviations.

TABLE 7
Fatty Acid Composition (g/kg total fatty acid) of Experimental Diets and Major Fat-Containing Foodstuffs[a]

	Grower diets				Finisher diets						
	Control (CD1)		Full-fat rapeseed (FFRD1)		Control (CD2)		Full-fat rapeseed (FFRD2)				
	ATA supplement (mg/kg feed)				ATA supplement (mg/kg feed)				Foodstuffs		
Fatty acids	0	200	0	200	0	200	0	200	CFW	LFW	FFR
C12:0	154.7	157.9	4.0	—	214.9	224.1	4.7	3.1	537.7	12.1	—
C14:0	71.7	73.3	5.4	5.2	84.5	86.3	2.6	2.4	211.3	20.6	—
C16:0	173.9	174.3	73.4	71.7	174.6	173.4	54.4	54.3	110.2	250.4	40.7
C16:1n-7	15.2	14.8	6.2	6.6	9.5	12.6	5.9	3.2	—	27.9	3.1
C18:0	71.8	75.3	18.0	18.3	86.8	88.2	17.2	18.4	33.2	157.9	15.4
C18:1n-9	217.4	226.7	486.4	488.3	242.0	234.7	531.3	530.1	85.4	393.4	570.9
C18:2n-6	194.8	185.7	260.2	255.6	156.0	144.1	244.2	240.6	22.3	105.6	213.9
C18:3n-3	27.9	27.0	108.1	108.9	20.2	19.6	119.1	113.7	—	13.9	125.1
C20:0	12.6	13.0	14.5	15.6	5.7	6.0	17.8	19.2	—	8.2	19.9
C20:1n-9	4.0	3.4	3.1	3.3	2.4	1.7	7.2	3.9	—	3.6	1.1
C20:2n-6	1.8	2.3	5.8	6.9	1.4	1.3	—	—	—	—	3.0
C20:4n-6	8.6	10.5	4.3	4.9	—	—	—	—	—	2.7	2.5
C20:5n-3	7.4	7.3	2.4	2.1	—	—	—	—	—	—	1.5
C22:5n-3	2.0	2.5	—	3.6	—	—	—	—	—	—	1.6
C22:6n-3	14.1	14.2	8.1	8.9	—	—	—	—	—	—	1.2
SFA	484.7	493.8	115.3	114.4	566.5	578.0	96.7	97.4	892.4	449.2	76.0
MUFA	236.6	244.9	495.7	498.2	253.9	249.0	544.4	537.2	85.4	510.3	575.1
PUFA	256.6	249.5	388.9	387.3	177.6	165.0	363.3	354.3	22.3	122.2	348.8
P/S ratio	0.53	0.51	3.37	3.39	0.31	0.29	3.76	3.64	0.03	0.27	4.59
n-6/n-3 ratio	4.00	3.85	2.27	2.20	7.69	7.14	2.04	2.13	—	7.69	1.70

[a]SFA = saturated fatty acids; MUFA = monounsaturated fatty acids; PUFA = polyunsaturated fatty acids; P/S = polyunsaturated/saturated fatty acid; CFW = coconut oil-filled whey; LFW = lard-filled whey; FFR = full-fat rapeseed. See Tables 1–3 for other abbreviations.

TABLE 8
Fatty Acid Composition (g/kg total fatty acid) of *Longissimus dorsi* Lipid[a]

	Diets							
	CD		FFRD					
	ATA supplement (mg/kg feed)		ATA supplement (mg/kg feed)		Statistical significance			
Fatty acids	0	200	0	200	SED	Diet	ATA	Diet × ATA
C12:0	14.2	7.2	2.9	n.d.	2.71	***	*	NS
C14:0	38.2	28.5	15.4	14.8	4.67	***	NS	NS
C16:0	230.2	229.8	198.8	196.2	6.88	***	NS	NS
C16:1n-7	33.3	34.4	27.1	29.8	2.82	*	NS	NS
C18:0	123.1	129.2	96.9	103.5	7.11	***	NS	NS
C18:1n-9	365.6	399.1	427.4	415.4	17.78	**	NS	NS
C18:2n-6	128.8	112.4	149.6	154.3	12.51	**	NS	NS
C18:3n-3	10.9	9.1	37.4	26.1	3.17	***	*	NS
C20:0	9.3	9.2	12.2	11.2	1.15	*	NS	NS
C20:1n-9	3.2	3.4	5.0	4.5	0.74	*	NS	NS
C20:2n-6	2.9	3.2	1.9	2.2	0.69	NS	NS	NS
C20:4n-6	18.2	17.8	14.0	17.6	3.36	NS	NS	NS
C20:5n-3	2.8	1.7	2.7	3.8	0.82	NS	NS	NS
C22:5n-3	5.2	4.9	4.3	2.5	1.33	NS	NS	NS
C22:6n-3	4.5	2.7	1.5	3.7	1.07	NS	NS	*
SFA	414.9	404.0	325.7	325.7	15.67	***	NS	NS
MUFA	402.1	436.8	459.5	449.7	19.33	*	NS	NS
PUFA	173.3	151.9	211.3	210.3	17.22	**	NS	NS
P/S ratio	0.42	0.38	0.65	0.65	0.06	***	NS	NS
n-6/n-3 ratio	6.39	7.31	3.63	4.99	0.54	***	*	NS

[a]CD = control; FFRD = full-fat rapeseed; n.d. = not detected; NS = not significant ($P > 0.05$); * = $P < 0.05$; ** = $P < 0.01$; *** = $P < 0.001$; four tissue samples analyzed per treatment. See Tables 1, 3, 4, and 7 for other abbreviations.

TABLE 9
Fatty Acid Composition (g/kg total fatty acid) of Subcutaneous Fat Lipid[a]

	Diets							
	CD		FFRD					
	ATA supplement (mg/kg feed)		ATA supplement (mg/kg feed)		Statistical significance			
Fatty acids	0	200	0	200	SED	Diet	ATA	Diet × ATA
C12:0	16.5	14.9	2.3	1.4	2.06	***	NS	NS
C14:0	44.0	42.5	11.8	12.0	2.93	***	NS	NS
C16:0	228.0	227.0	158.0	170.4	8.76	***	NS	NS
C16:1n-7	32.4	32.5	18.5	20.2	1.77	***	NS	NS
C18:0	120.6	120.5	80.9	91.4	7.07	***	NS	NS
C18:1n-9	401.9	410.1	456.6	445.8	14.68	**	NS	NS
C18:2n-6	107.2	105.3	174.2	167.6	9.78	***	NS	NS
C18:3n-3	11.8	11.7	51.3	48.2	1.66	***	NS	NS
C20:0	12.3	11.4	16.7	15.7	1.42	***	NS	NS
C20:1n-9	4.7	4.4	7.3	6.5	0.63	***	NS	NS
C20:2n-6	0.9	0.9	0.9	0.8	0.23	NS	NS	NS
C20:4n-6	3.7	3.6	8.4	6.8	0.96	***	NS	NS
C20:5n-3	0.4	0.7	0.9	0.8	0.28	NS	NS	NS
C22:5n-3	2.6	2.6	2.6	3.2	0.65	NS	NS	NS
C22:6n-3	2.5	2.3	1.5	1.8	0.50	NS	NS	NS
SFA	421.4	416.3	269.7	290.8	12.85	***	NS	NS
MUFA	439.0	447.0	482.4	472.5	13.88	**	NS	NS
PUFA	129.0	127.0	239.7	229.2	11.81	***	NS	NS
P/S ratio	0.31	0.31	0.89	0.80	0.06	***	NS	NS
n-6/n-3 ratio	6.47	6.37	3.27	3.24	0.20	***	NS	NS

[a]NS = Not significant ($P > 0$ 05); * = $P < 0.05$; ** = $P < 0.01$; *** = $P < 0.001$; four tissue samples analyzed per treatment. See Tables 1, 3, 5, and 7 for other abbreviations.

tion for 100 and 200 min. There was no interaction between diet and ATA supplementation levels.

DISCUSSION

Pigs fed diet CD200 showed the highest final LW, and the significant effect of diet on FCR was largely due to the contribution made by this diet to the overall CD effect. Raj *et al.* (22) recently reported that, as the proportion of rapeseed substituting for barley in pig diets increased, average daily gain decreased. However, in the present study, there was no evidence of any effect of whole rapeseed on daily gain, although values for pigs fed diet CD200 were noticeably, but not significantly, higher than the other treatments. There are conflicting reports of the effects of vitamin E supplementation on growth performance. Rafai *et al*. (23) and Bonnette *et al*. (24) observed no effect of supplementation in pigs up to 30 kg LW. However, Asghar *et al*. (25) reported that daily gain and feed

TABLE 10
Surface and Subsurface Oxidation of *Longissimus dorsi* (mg MDA/kg muscle)[a]

			Storage length at 4°C (d)						
				4		8		16	
Diet	ATA suppl. (mg/kg feed)	*n*	>48 h	SF	SS	SF	SS	SF	SS
CD	0	6	0.11	0.13	0.12	0.75	0.32	1.49	1.10
	200	6	0.10	0.12	0.11	0.45	0.20	1.50	1.05
FFRD	0	6	0.12	0.14	0.12	0.85	0.33	1.50	1.14
	200	6	0.11	0.14	0.12	0.73	0.29	1.50	1.12
SED			0.005	0.004	0.006	0.030	0.008	0.027	0.015
Statistical significance									
Diet			NS	***	NS	***	***	NS	***
ATA			NS	**	NS	***	***	NS	**
Diet × ATA			NS	NS	NS	***	***	NS	NS

[a]SF = surface (0–2.5 mm deep); SS = subsurface (2.5–5 mm deep); MDA, malondialdehyde; * = $P < 0.05$; ** = $P < 0.01$; *** = $P < 0.001$. See Tables 1 and 3 for other abbreviations.

TABLE 11
Lipid Oxidation in Subcutaneous Fat (μg MDA/g of extracted fat)[a]

Diet	ATA suppl. (mg/kg feed)	*n*	Storage length at 4°C (d) >48 h	4	8	16
CD	0	4	13.2	16.7	33.1	48.1
	200	4	12.9	13.2	27.7	32.1
FFRD	0	4	47.3	54.5	63.4	68.2
	200	4	30.3	33.8	44.5	53.3
SED			5.33	3.64	5.13	4.77
Statistical significance						
Diet			***	***	***	***
ATA			*	***	**	***
Diet × ATA			*	**	NS	NS

[a]See Tables 1, 3, and 10 for abbreviations. * = $P < 0.05$; ** = $P < 0.01$; *** = $P < 0.001$.

efficiency were significantly improved in the early stages (weeks 0–2) of a growth trial in which pigs were reared from 29 to 100 kg LW. Although not shown in the tables, our data suggest that ATA supplementation had no effect on growth up to 50 kg LW. Previous studies in poultry have indicated that dietary supplementation with ATA at concentrations up to 150 mg/kg diet has no effect on production traits (26,27). Diet had no effect on carcass measurements, although daily LW gain was noticeably higher and FCR lower in pigs fed diet CD200.

The significant decrease in drip loss due to ATA supplementation can be attributed to a reduction in lipid oxidation of phospholipids in the sacroplasmic membranes. However, because the bulk of the fluid loss from the muscle samples occurred in the first 48 h of storage, the changes in membrane integrity, which lead to loss of intracellular fluid, must precede hydroperoxide cleavage to yield MDA, which accumulated in tissue after 8 d of storage. Membrane lipid oxidation is associated with a decrease in membrane fluidity (8), and Monahan *et al.* (28) used 1,6-diphenyl-1,3,5-hexatriene as a fluorescent probe to demonstrate that fluorescence anisotropy, a measure of membrane fluidity, was positively correlated with TBARS in muscle microsome preparations. Early changes in postmortem membrane permeability may result from a loss of integrity due to fatty acid peroxidation, which could modify the function/activity of membrane proteins and enzymes before TBARS accumulate in the tissue. Buckley *et al.* (8) have suggested that measurement of fatty acid hydroperoxides would provide a better measure of early stages of membrane lipid oxidation.

The inclusion of FFR significantly reduced the melting point of s/c fat. Rapeseed is often cited as an excellent source of C18:1n-9 that can be used to increase the concentration of this fatty acid in tissue lipids, Although C18:1n-9 was the major fatty acid (570.9 g/kg total fatty acids) of rapeseed oil, it also contained considerable amounts of C18:2n-6 and C18:3n-3 (213.9 and 125.1 g/kg total fatty acids, respectively, in the variety of rapeseed used in this study). It is most likely that the decrease in melting point observed in fat from FFRD-fed pigs was mainly due to the combined effects of a reduction in the total saturated fatty acid content and an increase in the C18:2n-6 and C18:3n-3 content of the adipose tissue lipids. ATA supplementation appeared to increase the fat melting point. It is difficult to explain this effect because there was no significant effect of ATA supplementation on fatty

TABLE 12
Oxidative Stability of *Longissimus dorsi* Measured by the Iron-Induced Lipid Peroxidation Technique (nmoles MDA/mg protein)[a]

Diet	ATA suppl. (mg/kg feed)	*n*	Incubation time (min) 0	50	100	200
CD	0	6	0.36	0.57	0.94	2.24
	200	6	0.26	0.35	0.51	1.12
FFRD	0	6	0.40	0.55	1.12	3.11
	200	6	0.30	0.42	0.73	1.70
SED			0.04	0.05	0.07	0.14
Statistical significance						
Diet			NS	NS	***	***
ATA			***	***	***	***
Diet × ATA			NS	NS	NS	NS

[a]Average of duplicate determinations. *** = $P < 0.01$. See Tables 1, 3, and 10 for other abbreviations.

acid composition, and even though dietary supplementation markedly increased the AT content of adipose tissue, the presence of a higher concentration of AT *per se* should not influence fat melting point.

Plasma AT concentrations were significantly higher in animals supplemented with ATA and increased more than twofold between day 35 and day 77 of the feeding trial to values similar to those previously reported (25,29) in pigs fed 200 mg ATA/kg feed for 125 d. The main storage depots of AT are adipose tissue, liver, and muscle (30). Adipose tissue and muscle AT concentrations were similar to those recently reported by Pfalzgraf *et al*. (31) in pigs fed diets with 20 and 200 mg AT/kg diet. For each of the treatment diets, concentrations of AT in *L. dorsi* were similar to those found in plasma. The s/c adipose tissue AT concentrations were approximately two- to threefold greater than found in *L. dorsi*. When examined by regression analysis, there were clear relationships between AT concentration in *L. dorsi* and that in plasma at 35 d ($r = 0.65$) and 77 d ($r = 0.85$) or s/c adipose tissue ($r = 0.86$) (Fig. 1).

It is not surprising that the fatty acid composition of both *L. dorsi* and s/c adipose tissues was significantly affected by dietary fat source because fat digestion and absorption in monogastrics involve little structural modification to ingested fatty acids, and there are numerous similar reports in the literature (2,3,31). The presence of elevated concentrations of C12:0 and C14:0 in tissues from CD-fed animals is clearly caused by the inclusion of coconut fat-filled whey in these diets, and the reduced concentrations of C16:0 and C18:0 in FFRD-fed animals are attributed to the much lower concentrations of these fatty acids in FFRD diets. The concentrations of C18:1n-9, C18:2n-6, and C18:3n-3 were significantly higher in tissues from FFRD-fed pigs. However, comparison of the ratios of the concentration of each one of these fatty acids in tissues from CD- and FFRD-fed pigs with their ratios in their respective diets (means for CD or FFRD for both grower and finisher diets) shows that, although C18:2n-6 and C18:3n-3 are present in tissues at approximately the same ratio as that in the diets (18:2n-6—dietary CD/FFRD ratio 0.68, liver CD/FFRD ratio 0.79, adipose tissue CD/FFRD ratio 0.62; C18:3n-3—dietary CD/FFRD ratio 0.21, liver CD/FFRD ratio 0.31, adipose tissue CD/FFRD ratio 0.24), this was not so for C18:1n-9 (dietary CD/FFRD ratio 0.45, liver CD/FFRD ratio 0.91, adipose tissue CD/FFRD ratio 0.90). The higher concentration of C16:0 and C18:0 in tissues of both CD- and FFRD-fed pigs, compared to the diets, suggests ongoing *de novo* synthesis of C16:0 and elongation to C18:0 in both groups of animals. The relatively high concentration of C18:1n-9 in the tissues from CD-fed pigs, compared to that in the diets, suggests the presence of an active Δ^9 desaturase responsible for the conversion of C18:0 to C18:1n-9.

The pattern of lipid oxidation in the surface layer of *L. dorsi,* stored for up to 8 d at 4°C, was similar to that reported by Monahan *et al*. (28) in pork chops from pigs fed a control diet and a diet supplemented with a similar level of ATA. MDA concentration rose markedly between 4 and 8 d of storage, and ATA supplementation was more effective in reducing oxidation in tissues from pigs fed the control diets than those fed FFRD. After 16 d of storage, MDA concentrations were approximately double those after 8 d of storage. Development of oxidation in the subsurface layer of *L. dorsi* was delayed, compared to the surface layer. This probably reflects the more limited exposure of tissue lipids to oxygen deeper within the muscle mass and the delayed onset of the propagation phase of lipid peroxidation.

The protective effects of elevated levels of tissue AT were more apparent from the measurement of iron-induced lipid peroxidation in homogenates of *L. dorsi*. Similar effects, over shorter incubation times (0–90 min), have been shown in pork by Monahan *et al.* (28) who also demonstrated the relationship between accumulation of tissue MDA and the production of free radicals as measured by electron spin resonance. The effects of the different fat sources on iron-induced MDA production were not apparent at the shortest incubation period (50 min). However, after 100 and 200 min, oxidation in tissue from FFRD-fed pigs was significantly greater than that in the control-fed animals. Figure 1 clearly shows that, as the ratio of the concentrations of AT/total unsaturated fatty acids increased, the susceptibility of *L. dorsi* to iron-induced lipid oxidation decreased [$r = -0.63$ ($P < 0.05$), -0.70 ($P < 0.01$), -0.83 ($P < 0.001$), and -0.79 ($P < 0.001$) for 0, 50, 100, and 200 min of incubation].

Changes in muscle and adipose tissue lipid fatty acid compositions can clearly be achieved by the inclusion of whole FFR in pig diets. To prevent unacceptable oxidation of tissue lipids, supplemental dietary ATA is required to counteract the increased susceptibility of tissue lipids to oxidation due to the elevated PUFA content. The increased tissue content of PUFA and AT may be beneficial to the processor and consumer but must be balanced against the reduced melting point of s/c adipose tissue, which may adversely affect meat processing properties and consumer preference. The effects of modifying the fatty acid and AT content of muscle on organoleptic properties of the cooked product were not assessed and require further investigation to ensure customer acceptability at the point of consumption.

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