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Feeding linseed to increase the *n*-3 PUFA of pork: fatty acid composition of muscle, adipose tissue, liver and sausages

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Abstract

Eighty pigs, male and female littermate pairs, were fed a control or a test diet from 25 to 95 kg live weight. The diets, as fed, contained 15.5 g/kg linoleic acid (18:2) and 1.9 g/kg α-linolenic acid (18:3) (control) or 10 g/kg linoleic acid and 4 g/kg α-linolenic acid (test). The test diet, with added linseed, was, therefore, high in the main *n*-3 polyunsaturated fatty acid (PUFA) 18:3 and low in the main *n*-6 PUFA 18:2. Making this relatively small change led to a 56% increase in the content of 18:3 in muscle and major increases in the contents of the beneficial longer chain PUFAs EPA (20:5*n*-3) (100% increase) and DHA (22:6*n*-3) (35% increase) which are synthesised from 18:3*n*-3. Levels of EPA and DHA in pigmeat adipose tissue were also increased by the test diet. In liver, the test diet resulted in an 18:3 level 4× higher than in muscle, with 10× more EPA and 20× more DHA. Sausages, analysed after 6 months frozen storage also had high *n*-3 PUFA levels, due to the contribution of these fatty acids from both muscle and adipose tissue. From a health perspective these results confirm the potential of pigmeat to supply valuable *n*-3 PUFA to the human diet. The test diet produced a PUFA:saturated FA ratio in muscle of 0.4, close to the minimum recommended value for the diet as a whole and an *n*-6:*n*-3 ratio of 5, a significant improvement on the current average for pigmeat (7). It is estimated that the test diet would provide 12 g of long chain *n*-3 PUFA to the human diet per annum at current pigmeat consumption levels in the UK, about a third of that from oily fish.

1. Introduction

Fat is an important component of the human diet but current levels of intake in the UK are considered too high and the overall fatty acid composition is unbalanced (Department of Health and Social Security, 1994). There is an excessive intake of saturated fatty acids (S) relative to polyunsaturated fatty acids (PUFA, P), expressed usually as the P:S ratio, and the consumption of *n*-3 PUFA is too low relative to *n*-6 PUFA. Meat, both muscle and adipose tissue, is a major source of fat in the human diet and there is interest in modifying the composition of meat by dietary means, to improve its nutritional value. This is possible in all meat species but easier in non-ruminants (dietary fatty acids being absorbed unchanged from the intestine) than in ruminants, where dietary fatty acids are hydrogenated in the gut (Wood, Enser, Fisher, Nute, Richardson & Sheard, 1999). Some fatty acids respond more readily to dietary change than others. For example, in pigs, saturated and monounsaturated fatty acids are synthesised in vivo and, as a result, are less readily influenced by diet than the polyunsaturated fatty acids, linoleic (18:2, *n*-6) and α-linolenic (18:3, *n*-3), which cannot be synthesised and, therefore, reflect dietary change. The longer chain (C20-22) fatty acids of the *n*-6 and *n*-3 series can also be synthesised from their dietary precursors, linoleic and α-linolenic acid, respectively. A potential benefit, therefore, of feeding diets rich in α-linolenic acid is that its increased deposition could also lead to higher levels of the *n*-3 fatty acids involved in

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decreasing the thrombogenicity of blood, especially eicosapentaenoic acid (EPA, 20:5) and docosahexaenoic acid (DHA, 22:6). Previous studies aimed at increasing EPA and DHA levels via the diet have used either fish oils (Irie & Sakimoto, 1992; Ishida, Konno, Suzuki, Ogawa & Abe, 1996; Leskanich, Matthews, Warkup, Noble & Hazzledine, 1997; Morgan, Noble, Cocchi & McCartney, 1992), or dietary sources of 18:3 such as linseed (flaxseed) (Ahn, Lutz & Sim, 1996; Cherian & Sim, 1995; Cunnane, Stitt, Ganguli & Armstrong, 1990; Riley, Enser, Hallet, Hewett, Wood & Atkinson, 1998a,b; Romans, Johnson, Wulf, Libal & Costello, 1995a; Romans, Wulf, Johnson, Libal & Costello, 1995b; Spect-Overholt et al., 1997) or canola oil (from rapeseed) (Rhee, Ziprin, Ordonez & Bohac, 1988). However, the use of fish oil may be considered to be environmentally unsound. Studies with linseed have demonstrated the potential for increased deposition of EPA and DHA in addition to α-linolenic acid in pigs but some have been in young animals (Cunnane et al., 1990) or over a short feeding period (Riley et al., 1998a,b; Romans et al., 1995a,b). Other, longer term studies, have failed to exploit the potential for increased synthesis of EPA and DHA from α-linolenic acid through lowering the concentration of linoleic acid in the feed to decrease competition for the Δ6 desaturase enzymes (Brenner, 1974).

The overall aim of this experiment was to improve the nutritional value of pigmeat using a linseed-rich diet high in α-linolenic but low in linoleic acid to obtain the maximum increase in EPA and DHA through endogenous synthesis and to decrease the ratio of *n*-6 to *n*-3 PUFA from 10–14 (Enser, Hallett, Hewitt, Fursey & Wood, 1996) to 5 or less. By replacing some of the dietary linoleic acid by α-linolenic acid and by using a relatively low level of α-linolenic acid we hoped to avoid deterioration in meat quality, particularly increased susceptibility to lipid peroxidation (Romans, et al., 1995b) or an increased incidence of soft fat. This paper reports tissue fatty compositions and the meat quality results are presented in the accompanying paper (Sheard, Enser, Wood, Nute, Gill & Richardson, in press).

2. Materials and methods

2.1. Production

Eighty pigs (equal numbers of entire males and females), with a target slaughter live weight of 95 kg, were drawn from a pool of 108 pigs (full sibling pairs) reared from 25 kg at Stotfold Pig Development Unit and fed ad libitum on control and test diets. The quantity of feed consumed by a sibling pair was recorded weekly; individual pigs were also weighed weekly.

The two diets, prepared by Farm Nutrition Ltd. (Bungay, Suffolk, UK), consisted of crushed whole linseed, soyabean meal, soya oil, palm oil, tallow, rapeseed, oleic acid oil, with wheat, barley, sugar beet feed and ethoxyquin. They had the same energy (14 MJDE), protein (200 g), total oil (45 g), and oleic acid content (15 g) per kg but different target levels of α-linolenic acid (18:3) (1.5 g control, 4.5 g test diet), linoleic acid (18:2) (16 g control and 10 g test diet) and palmitic acid (16:0) (5 g control and 10 g test diet) contents per kg. A composite sample of each diet (initial and final) was analysed for total lipid and fatty acid composition. Vitamin E was included in both diets at a level of 100 mg/kg.

2.2. Slaughter

The pigs were delivered to the University of Bristol abattoir for slaughter, in groups ranging in size from 11 to 17. The mean hot carcass weight was 82 kg (range 56–112 kg), with a mean backfat thickness (P_2) value of 10.8 mm (range 6–18 mm), and mean longissimus pH of 6.57 and 5.67 at 45 min and 24 h post-slaughter, respectively. None of these parameters was different between the diets. Carcasses were chilled overnight at 1°C. The average weight loss during chilling was 2.59%.

2.3. Butchery

After overnight chilling, one chop was removed from the lumbar end of the right *longissimus thoracis et lumborum* (LTL), vacuum packed, blast frozen and stored at −18°C for fatty acid analysis of the muscle and adipose tissue. The remainder of the LTL was used in the subsequent study (Sheard et al., in press) to assess eating quality and the susceptibility of the tissues to oxidation.

Liver samples for fatty acid analysis were vacuum packed, blast frozen and stored at −18°C until required.

Shoulders were removed from the right side of the first 40 pigs slaughtered and the skin removed. The subcutaneous fat was removed from the skin, and the muscles from the hand joint were deboned, both fat and muscle being used for sausage production. Batches of about 5 kg were made using the following ingredients (g/kg): 450 lean muscle, 200 fat, 125 rusk, 200 water/ice and 25 Flavourburst TTD seasoning (Lucas Ingredients, Bristol, UK). Lean and fat were minced through a Butcher Boy mincer (Baker & Nixson, Norwich, UK) with a 5 mm plate and then placed in a Stalberg mixer (Team Equipment, Oxted, Surrey). Seasoning was added during the first 30 s of mixing together with half the ice/water, and mixed for a further 3.5 min. Rusk was added, together with the remaining water/ice and mixing continued for a total of 6 min. The mix was formed into 56 g (2 oz) sausages using synthetic sausage casings

(Devro Teepak, Moodiesburn, Scotland) and a Handtmann sausage stuffer (Handtmann Ltd, Luton, UK) fitted with a 15 mm stuffing horn. Sausages were left overnight in sealed plastic bags, for the rusk to hydrate, and then blast frozen and stored at −18°C until required.

2.4. Fatty acid analysis

Fatty acid analyses were carried out on the following: sausages (40 samples), adipose tissue, LTL muscle, liver and the control and test diets. Samples were extracted from liver and LTL muscle, after blending separately in a food processor, by the chloroform: methanol procedure of Folch, Lees and Stanley (1957). Liver lipid extracts were hydrolysed directly for fatty acid analysis. The LTL muscle lipids were separated into the neutral lipid and phospholipid fractions on silicic acid columns (Isolute, 500 mg, Jones Chromatography, Mid Glamorgan, Wales) using 10 ml of chloroform and 20 ml of methanol to elute the respective fractions. The fatty acid composition was then determined on each fraction.

Sausages and adipose tissue (from the inner layer of loin backfat) were blended separately in a small food processor. Fatty acids were prepared from the extracted lipids or directly from tissue homogenates by alkaline hydrolysis in the presence of methyl heneicosanoate (21:0) as an internal standard. After acidification and extraction, the fatty acids were converted to methyl esters using diazomethane in diethyl ether. The methyl esters and long-chain aldehydes were analysed by gas-liquid chromatography on a CP Sil 88 column, 50 m × 0.25 mm ID (Chrompack, UK, Ltd, London) and quantified using the internal standard (Enser, Hallett, Hewett, Fursey, Wood & Harrington, 1998). Peaks were identified using standards where available (Sigma Chemical Co. Ltd, Poole).

Fatty acid results are presented as mg/100g of tissue and as g/100 g fatty acids (% by weight). The latter is most commonly used by other authors but the former allows nutritional value to be assessed. Nutritional quality is described by the P:S ratio expressed as: 18:2*n*-6 + 18:3*n*-3/12:0 + 14:0 + 16:0 + 18:0. Although we recognise that this is not an ideal indication of atherogenicity or thrombogenicity (Ulbricht & Southgate, 1991) it has been widely used. The *n*-6:*n*-3 ratio has been calculated as 18:2*n*-6/18:3*n*-3 which is relevant to the competition for synthesis of longer-chain PUFA between each series. Since this ratio ignores the existence of pre-formed longer-chain PUFA in the meat the results are also expressed as Σn-6:Σn-3.

2.5. Statistics

The data were analysed using a one way factorial analysis of variance, or a generalised linear model.

3. Results

3.1. Feed fatty acids

The control diet contained 15.5 g/kg linoleic acid and 1.9 g/kg α-linolenic acid and the test diet 10 g/kg linoleic acid and 4 g/kg α-linolenic acid. These values are very close to the target values, giving 18:2 to 18:3 ratios of 8.2 and 2.5 in the control and test diets, respectively. The major feed fatty acids (g per 100 g total fatty acids) were as follows (results for the test diet in parantheses): 14:0–1.1 (2.1), 16:0–17.8 (20.0), 16:1–1.0 (0.9), 17:0–0.4 (0.8), 18:0–10.1 (23.7), 18:1–25.6 (15.4), 18:2–35.7 (22.1), and 18:3–4.2 (9.4).

The daily liveweight gain was approximately 0.8 kg for pigs on both diets, and slightly higher for entire male pigs. The feed conversion ratio, for both diets (littermate pairs), was 2.6 kg feed/kg gain.

Making these relatively small dietary changes led to significant increases in the content of 18:3 and the beneficial long chain PUFAs EPA (20:5) and DHA (22:6) for muscle, adipose tissue, sausages and liver (Tables 1–11). Most *n*-6 PUFA were lower but in general tissue levels of 18:2 decreased less and 18:3 increased less than in the test diet.

3.2. Muscle fatty acids

In comparison with the control diet, the test diet resulted in significantly higher amounts of all the *n*-3 PUFA in the *m. longissimus lumborum* of both boars and gilts (Table 1). The increases were: EPA 100%, 18:3 55%, DHA 35% and DPA 29% averaged over both sexes. Of the *n*-6 PUFA, 18:2, arachidonic acid (20:4) and 22:4 were lower by 14, 16 and 35% but 20:3*n*-6 (dihomogammalinolenic acid) was unchanged. There were no significant effects of diet on the quantities of any of the saturated or monounsaturated fatty acids. Since the intramuscular fatty acid content did not differ between sexes or diet, expressing the results as percentage composition of total fatty acids gave similar results (Table 2) except that 16:1 (palmitoleic acid) was significantly higher in pigs fed the test diet and 18:1 trans significantly lower.

The test diet produced a P:S ratio of 0.4, close to the minimum recommended value (0.45) for the diet as a whole. The *n*-6:*n*-3 ratio was 5, a significant improvement over the control diet (8–9). Total *n*-3 fatty acid content was 3.6% of total fatty acids (Table 1).

3.3. Muscle neutral lipids and phospholipids

The neutral and phospholipid fractions accounted for 67% (826 mg) and 33% (400 mg), respectively of the total fatty acids (1225 mg) in the *m. longissimus lumborum* (Tables 3 and 5), with proportionally more long

Table 1
Fatty acid content of *Longissimus lumborum* muscle (mg per 100 g muscle) from pigs raised on a test or control diet[a]

Fatty acid	Control diet		Test diet			
	Female	Male	Female	Male	Sed	Significance[e]
12:0 (lauric)	0.73	0.99	0.86	1.01	0.18	ns
14:0 (myristic)	11.5	14.6	13.6	15.4	2.5	ns
16:0 (palmitic)	239	282	267	295	40	ns
16:1	29.3	35.3	35.1	38.4	6.0	ns
18:0 (stearic)	129	154	145	165	21	ns
18:1 *trans*	1.9	2.6	1.4	1.8	0.5	ns
18:1 oleic	330	414	395	427	70	ns
18:1 *cis* (vaccenic)	37.0	44.2	42.6	46.5	6.5	ns
18:2*n*-6 (linoleic)	185a,b	208b	165a	174a	15	*
20:1	5.4	7.5	6.2	7.3	1.2	ns
18:3*n*-3 (α-linolenic)	9.0a	11.9a	15.7b	16.9b	1.7	***
20:3*n*-6	5.9	6.4	5.9	6.4	0.3	ns
20:4*n*-6 (arachidonic)	43.1b	43.1b	35.5a	26.5a	1.6	***
20:5*n*-3 (eicosapentaenoic)	4.4a	3.9a	8.3b	8.3b	0.3	***
22:4*n*-6	4.5b	5.0b	3.0a	3.2a	0.3	***
22:5*n*-3 (docosapentaenoic)	9.9a	9.6a	12.1b	13.0b	0.5	***
22:6*n*-3 (docosahexaenoic)	4.4a	3.7a	5.3b	5.6b	0.4	***
Total	1093	1295	1204	1309	167	ns
P:S ratio[b]	0.51	0.49	0.42	0.40		
n-6:*n*-3 ratio[c]	20.5	17.5	10.5	10.2		
n-6:*n*-3 ratio[d]	8.61	9.02	5.06	5.03		

[a] Means in the same row with the same letter do not differ significantly at the 0.05 level of probability.
[b] P:S ratio defined as $\frac{18:2n\text{-}6 + 18:3n\text{-}3}{12:0 + 14:0 + 16:0 + 18:0}$.
[c] Ratio of 18:2*n*-6 to 18:3*n*-3.
[d] Ratio of Σ*n*-6 to Σ*n*-3.
[e] ns, not significant; $*P < 0.05$; $**P < 0.01$; $***P < 0.001$; Sed is strandard error of difference between means.

Table 2
Fatty acid composition of *Longissimus lumborum* muscle (g per 100 g total fatty acids) from pigs raised on a test or control diet[a]

	Control diet		Test diet			
Fatty acid	Female	Male	Female	Male	Sed	Significance[b]
12:0 (lauric)	0.06	0.08	0.07	0.08	0.005	ns
14:0 (myristic)	1.03	1.08	1.11	1.15	0.05	ns
16:0 (palmitic)	21.7	21.5	22.0	22.2	0.4	ns
16:1	2.6a	2.6a	2.9b	2.9b	0.1	*
18:0 (stearic)	11.7	12.0	11.9	12.5	0.3	ns
18:1 *trans*	0.17b	0.18b	0.11a	0.14a	0.02	*
18:1 oleic	29.6	30.1	32.2	31.6	1.1	ns
18:1 *cis* (vaccenic)	3.3	3.3	3.5	3.5	0.1	ns
18:2*n*-6 (linoleic)	17.5b	17.3b	14.1a	14.1a	0.9	***
20:1	0.49	0.54	0.50	0.55	0.03	ns
18:3*n*-3 (α-linolenic)	0.84a	0.91a	1.32b	1.32b	0.06	***
20:3*n*-6	0.56	0.56	0.52	0.54	0.05	ns
20:4*n*-6 (arachidonic)	4.1b	3.9b	3.1a	3.1a	0.3	**
20:5*n*-3 (eicosapentaenoic)	0.42a	0.36a	0.73b	0.69b	0.05	***
22:4*n*-6	0.43b	0.43b	0.26a	0.28a	0.03	***
22:5*n*-3 (docosapentaenoic)	0.95a,b	0.85a	1.06b	1.08b	0.08	***
22:6*n*-3 (docosahexaenoic)	0.43a,b	0.34a	0.47b	0.43a,b	0.05	*

[a] Means in the same row with the same letter do not differ significantly at the 0.05 level of probability.
[b] See Table 1, footnote e.

Table 3
Neutral lipid fatty acid content of *Longissimus lumborum* muscle (mg per 100 g muscle) from pigs raised on a test or control diet[a]

	Control diet		Test diet			
Fatty acid	Female	Male	Female	Male	Sed	Significance[e]
12:0 (lauric)	0.70	0.96	0.84	0.96	0.18	ns
14:0 (myristic)	10.4	13.5	12.3	13.9	2.4	ns
16:0 (palmitic)	169	214	196	225	38.2	ns
16:2	24.4	30.2	28.8	31.5	5.7	ns
18:0 (stearic)	84	108	101	119	20	ns
18:1 *trans*	1.3	2.1	1.0	1.3	0.5	ns
18:1 oleic	282	363	340	368	68	ns
18:1 *cis* (vaccenic)	27.2	34.1	32.3	35.6	6.2	ns
18:*n*-6 (linoleic)	65a	93b	56a	67a	12.6	*
20:1	4.7	6.7	5.4	6.4	1.2	ns
18:3*n*-3 (α-linolenic)	5.4	8.3	8.4	9.5	1.5	ns
20:3*n*-6	0.9	1.4	0.9	0.9	0.2	ns
20:4*n*-6 (arachidonic)	4.5	5.2	3.3	4.0	0.8	ns
20:5*n*-3 (eicosapentaenoic)	0.3	0.2	0.5	0.5	0.2	ns
22:4*n*-6	0.7	1.1	0.5	0.6	0.2	ns
22:5*n*-3 (docosapentaenoic)	1.8	2.3	2.1	2.5	0.4	ns
22:6*n*-3 (docosahexaenoic)	0.5	0.5	0.5	0.8	0.2	ns
Total	695	901	804	903	157	ns
P:S ratio[b]	0.27	0.30	0.21	0.21		
n-6:*n*-3 ratio[c]	12.0	11.2	6.7	7.1		
n-6:*n*-3 ratio[d]	8.88	8.91	5.28	5.18		

[a] Means in the same row with the same letter do not differ significantly at the 0.05 level of probability.

[b] P:S ratio defined as $\frac{18:2n\text{-}6 + 18:3n\text{-}3}{12:0 + 14:0 + 16:0 + 18:0}$.

[c] Ratio of 18:2-*n*6 to 18:3*n*-3.

[d] Ratio of Σ*n*-6 to Σ*n*-3.

[e] See Table 1, footnote e.

Table 4
Neutral lipid fatty acid composition *Longissimus lumborum* muscle (g per 100 g total fatty acids) from pigs raised on a test or control diet[a]

	Control diet		Test diet			
Fatty acid	Female	Male	Female	Male	Sed	Significance[b]
12:0 (lauric)	0.10	0.11	0.11	0.11	0.007	ns
14:0 (myristic)	1.48	1.50	1.53	1.54	0.05	ns
16:0 (palmitic)	24.0	23.8	24.4	24.7	0.4	ns
16:1	3.4	3.3	3.6	3.5	0.1	ns
18:0 (stearic)	12.0	12.2	11.9	12.4	0.4	ns
18:1 *trans*	0.19b	0.20b	0.13a	0.15a,b	0.03	*
18:1 oleic	40.1a	39.3a	42.2b	40.0a	0.9	**
18:1 *cis* (vaccenic)	3.9	3.7	4.1	3.9	0.1	ns
18:2*n*-6 (linoleic)	10.1b	10.9b	7.2a	8.2a	0.8	***
20:1	0.67	0.71	0.67	0.71	0.03	ns
18:3*n*-3 (α-linolenic)	0.81a	0.91a,b	1.04b,c	1.10c	0.08	**
20:3*n*-6	0.15	0.18	0.12	0.16	0.04	ns
20:4*n*-6 (arachidonic)	0.76	0.73	0.46	0.62	0.18	ns
20:5*n*-3 (eicosapentaenoic)	0.05	0.04	0.07	0.09	0.03	ns
22:4*n*-6	0.13	0.13	0.07	0.09	0.04	ns
22:5*n*-3 (docosapentaenoic)	0.31	0.30	0.29	0.36	0.08	ns
22:6*n*-3 (docosahexaenoic)	0.10	0.06	0.07	0.13	0.04	ns

[a] Means in the same row with the same letter do not differ significantly at the 0.05 level of probability.

[b] See Table 1, footnote e.

Table 5
Phospholipid fatty acid content of *Longissimus lumborum* muscle (mg per 100 g muscle) from pigs raised on a test or control diet[a]

	Control diet		Test diet			
Fatty acid	Female	Male	Female	Male	Sed	Significance[e]
12:0 (lauric)	0.03	0.03	0.02	0.05	0.02	ns
14:0 (myristic)	1.1	1.2	1.3	1.5	0.14	ns
16:0 (palmitic)	70.1	68.2	70.3	70.1	3.6	ns
16:1	4.9a	5.0a	6.4b	6.9b	0.6	***
18:0 (stearic)	44.4	45.5	43.4	46.4	1.9	ns
18:1 *trans*	0.8b	0.8b	0.5a	0.9b	0.11	*
18:1 oleic	48.4	51.4	54.8	58.3	3.7	ns
18:1 *cis* (vaccenic)	9.8	10.1	10.4	10.9	0.6	ns
18:2*n*-6 (linoleic)	120	115	108	107	6.2	ns
20:1	0.7	0.8	0.8	0.9	0.08	ns
18:3*n*-3 (α-linolenic)	3.6a	3.7a	7.3b	7.4b	0.3	***
20:3*n*-6	5.0	5.0	5.1	5.3	0.3	ns
20:4*n*-6 (arachidonic)	38.7b	37.9b	32.2a	32.5a	1.8	***
20:5*n*-3 (eicosapentaenoic)	4.1a	3.7a	7.9b	7.7b	0.3	***
22:4*n*-6	3.8b	3.9b	2.5a	2.7a	0.2	***
22:5*n*-3 (docosapentaenoic)	8.1a	7.5a	10.0b	10.5b	0.5	***
22:6*n*-3 (docosahexaenoic)	3.9a	3.2a	4.8b	4.7b	0.3	***
Total	399	394	400	406	19	ns
P:S ratio[b]	1.07	1.03	0.98	0.97		
n-6:*n*-3 ratio[c]	33.3	31.1	14.8	14.5		
n-6:*n*-3 ratio[d]	8.50	8.94	4.93	4.87		

[a] Means in the same row with the same letter do not differ significantly at the 0.05 level of probability.
[b] P:S ratio defined as $\frac{18:2n\text{-}6 + 18:3n\text{-}3}{12:0 + 14:0 + 16:0 + 18:0}$.
[c] Ratio of 18:2*n*-6 to 18:3*n*-3.
[d] Ratio of Σ*n*-6 to Σ*n*-3.
[e] See Table 1, footnote e.

Table 6
Phospholipid fatty acid composition of *Longissimus lumborum* muscle (g per 100 g total fatty acids) from pigs raised on a test or control diet[a]

	Control diet		Test diet			
Fatty acid	Female	Male	Female	Male	Sed	Significance[b]
12:0 (lauric)	0.006	0.007	0.004	0.012	0.005	ns
14:0 (myristic)	0.29a	0.30a	0.33a,b	0.38b	0.03	*
16:0 (palmitic)	17.6	17.3	17.6	17.3	0.3	ns
16:1	1.2a	1.3a	1.6b	1.7b	0.1	***
18:0 (stearic)	11.2a,b	11.6b	10.9a	11.5b	0.2	***
18:1 *trans*	0.20b	0.20b	0.13a	0.20b	0.02	**
18:1 oleic	12.1a	13.0a,b	13.6b,c	14.4c	0.5	***
18:1 *cis* (vaccenic)	2.5	2.6	2.6	2.7	0.08	ns
18:2*n*-6 (linoleic)	30.2c	29.0b	27.0a	26.3a	0.5	***
20:1	0.18	0.20	0.67	0.19	0.01	ns
18:3*n*-3 (α-linolenic)	0.90a	0.93a	1.84b	1.83b	0.06	***
20:3*n*-6	1.2	1.3	1.3	1.3	0.03	ns
20:4*n*-6 (arachidonic)	9.7b	9.7b	8.1a	8.0a	0.3	***
20:5*n*-3 (eicosapentaenoic)	1.0a	0.9a	2.0b	1.9b	0.05	***
22:4*n*-6	0.94b	1.00b	0.62a	0.66a	0.03	***
22:5*n*-3 (docosapentaenoic)	2.0a	1.9a	2.5b	2.6b	0.07	***
22:6*n*-3 (docosahexaenoic)	0.96a	0.83a	1.21b	1.15b	0.07	***

[a] Means in the same row with the same letter do not differ significantly at the 0.05 level of probability.
[b] See Table 1, footnote e.

chain PUFAs in the phospholipid fraction. Effects of the diet on the neutral lipid fatty acids were small and only significant for 18:2 because of the variation in neutral lipid content between animals (Table 3). When expressed as % composition (Table 4) this effect was removed and 18:2 was significantly lower and 18:3 significantly higher in animals fed the test diet. The content of phospholipid fatty acids in the muscle revealed the same overall effects of diet on the total muscle fatty acids with significantly more *n*-3 PUFA and less *n*-6 PUFA, except for 20:3, in animals fed the test diet (Table 5). The decrease in 18:2 was not significant on a wet tissue basis but became so when expressed as % of total phospholipid fatty acids (Table 6). The partition of 18:2 and 18:3 between the neutral and phospholipids differed markedly. Whereas 18:2 was concentrated in the phospholipids, 27.0–30.2% compared with 7.2–10.9% of neutral lipid fatty acids, 18:3 was more evenly distributed, constituting 0.9–1.84% of the phospholipid fatty acids and 0.8–1.1% of the neutral lipid fatty acids. Therefore, more than half of the 18:3 in the muscle was present in the neutral lipid fraction in contrast to the other PUFA.

3.4. Adipose tissue fatty acids

The test diet led to a significant ($P < 0.001$) increase in the percentage of 18:3 (1.8 and 2.3% for the control and test diet, respectively) and a significant decrease ($P < 0.05$) in 18:2 (19.3 and 14.7% for the control and test diet, respectively), with significant increases ($P < 0.001$) in the contents of the beneficial long chain PUFAs: EPA (20:5) and DHA (20:6), and significantly lower contents ($P < 0.001$) of the *n*-6 PUFA, 20:4 (Table 7). Both 18:2 and 18:3 were at higher levels in adipose tissue lipid than in muscle neutral lipid with EPA and DHA at similar levels in the two tissues. The percentages of 18:2 and 18:3 were higher in the adipose tissue of boars than of gilts on both diets whereas oleic acid percentages were lower but only significantly so for the test diet.

3.5. Sausage fatty acids

The test diet led to a significant ($P < 0.001$) increase in 18:3 (222 and 307 mg for the control and test diet, respectively) and a significant decrease ($P < 0.01$) in 18:2 (2208 and 1827 mg for the control and test diet, respectively), with significant increases ($P < 0.01$) in the contents of 20:4 *n*-3, EPA (20:5) and DPA (22:5) and a significantly lower content ($P < 0.001$) of 22:4*n*-6 (Table 8). Although the mean values for arachidonic acid were also lower the differences were not significant. All sausages contained about 12% fat.

The test diet produced a P:S ratio of 0.48, close to the minimum recommended value (0.45) for the diet as a

Table 7
Fatty acid composition of adipose tissue (g per 100 g total fatty acid) from pigs raised on a test or control diet[a]

	Control diet		Test diet			
Fatty acid	Female	Male	Female	Male	Sed	Significance[e]
12:0 (lauric)	0.09a	0.09a	0.11b	0.13c	0.005	***
14:0 (myristic)	1.34a,b	1.28a	1.41b	1.44b	0.04	***
16:0 (palmitic)	22.4a,b	21.7a	23.5b,c	23.7c	0.6	**
16:1	2.0	1.9	2.1	2.1	0.07	ns
17:0	0.47a	0.54b	0.46a	0.54b	0.03	*
18:0 (stearic)	13.6a	13.2a	15.0b	15.4b	0.6	***
18:1 *trans*	0.47b	0.50b	0.39a	0.43a,b	0.04	*
18:1 oleic	33.3a,b	32.5a	34.6b	32.3a	0.6	**
18:1 *cis* (vaccenic)	2.27	2.22	2.33	2.26	0.05	ns
18:2*n*-6 (linoleic)	18.4c	20.2d	13.8a	15.5b	0.8	***
20:1	0.73a	0.78a	0.88b	0.80a,b	0.05	*
18:3*n*-3 (α-linolenic)	1.74a	1.90a	2.43b	2.80c	0.10	***
20:4*n*-6 (arachidonic)	0.23b	0.24b	0.16a	0.18a	0.02	***
20:4*n*-3	0.03a	0.03a	0.04b	0.04b	0.004	***
20:5*n*-3 (eicosapentaenoic)	0.05a	0.05a	0.05a	0.07b	0.008	**
22:5*n*-3 (docosapentaenoic)	0.19a	0.19a	0.24b	0.25b	0.01	***
22:6*n*-3 (docosahexaenoic)	0.08a	0.08a	0.12b	0.12b	0.009	***
P:S ratio[b]	0.54	0.61	0.41	0.45		
n-6:*n*-3 ratio[c]	10.52	10.63	5.68	5.54		
n-6:*n*-3 ratio[d]	8.91	9.08	4.85	5.58		

[a] Means in the same row with the same letter do not differ significantly at the 0.05 level of probability.

[b] P:S ratio defined as $\frac{18:2n\text{-}6 + 18:3n\text{-}3}{12:0 + 14:0 + 16:0 + 18:0}$.

[c] Ratio of 18:2*n*-6 to 18:3*n*-3.

[d] Ratio of Σ*n*-6 to Σ*n*-3.

[e] See Table 1, footnote e.

Table 8
Fatty acid content of pork sausages (mg per 100 g sausage) from pigs raised on a test or control diet[a]

	Control diet		Test diet			
Fatty acid	Female	Male	Female	Male	Sed	Significance[e]
12:0 (lauric)	12.6a	11.5a	14.3a,b	16.0b	1.56	*
14:0 (myristic)	188	165	188	198	18.9	ns
16:0 (palmitic)	2840	2456	2738	2843	274	ns
16:1	325	275	319	313	34.1	ns
18:0 (stearic)	1468	1305	1440	1552	149	ns
18:1 *trans*	54.0	40.3	41.8	51.5	6.3	ns
18:1 oleic	4528	3802	4357	4291	394	ns
18:1 *cis* (vaccenic)	344	282	339	335	30.9	ns
18:2*n*-6 (linoleic)	2304c	2112b,c	1729a	1925a,b	163	**
20:1	89.1	79.5	85.7	87.7	8.3	ns
18:3*n*-3 (α-linolenic)	232a	212a	290b	324b	21.7	***
20:3*n*-6	16.5	15.8	12.8	13.4	1.8	ns
20:4*n*-6 (arachidonic)	51.4	53.4	43.7	46.9	5.2	ns
20:4*n*-3	0.6a	0.9a	3.9b	5.3b	1.2	***
20:5*n*-3 (eicosapentaenoic)	8.5a	10.0a,b	12.7b,c	13.9c	1.4	**
22:4*n*-6	12.2c	10.7b,c	8.1a	10.0b	0.9	***
22:5*n*-3 (docosapentaenoic)	31.8a	31.3a,b	37.7b,c	41.9c	3.2	**
22:6*n*-3 (docosahexaenoic)	15.3	14.7	18.3	19.5	2.5	ns
Total	12,839	11,172	11,987	12,426	1075	ns
P:S ratio[b]	0.56	0.59	0.46	0.49		
n-6:*n*-3 ratio[c]	9.93	9.96	5.96	5.94		
n-6:*n*-3 ratio[d]	8.13	8.05	4.91	5.21		

[a] Means in the same row with the same letter do not differ significantly at the 0.05 level of probability.

[b] P:S ratio defined as $\frac{18:2n\text{-}6 + 18:3n\text{-}3}{12:0 + 14:0 + 16:0 + 18:0}$.

[c] Ratio of 18:2*n*-6 to 18:3*n*-3.

[d] Ratio of Σ*n*-6 to Σ*n*-3.

Table 9
Fatty acid composition of pork sausages (g per 100 g total fatty acids) from pigs raised on a test or control diet[a]

	Control diet		Test diet			
Fatty acid	Female	Male	Female	Male	Sed	Significance[b]
12:0 (lauric)	0.10a	0.10a	0.12b	0.13b	0.006	***
14:0 (myristic)	1.46	1.48	1.57	1.59	0.06	ns
16:0 (palmitic)	22.0	22.0	22.7	22.8	0.57	ns
16:1	2.53	2.46	2.66	2.46	0.14	ns
18:0 (stearic)	11.4	11.7	11.9	12.5	0.5	ns
18:1 *trans*	0.43	0.36	0.35	0.42	0.05	ns
18:1 (oleic)	35.2b	34.0a	36.4c	34.5a,b	0.6	**
18:1 *cis* (vaccenic)	2.68b	2.52a	2.84c	2.68b	0.08	**
18:2*n*-6 (linoleic)	18.1b	18.9b	14.6a	15.6a	0.7	***
20:1	0.70	0.71	0.71	0.70	0.03	ns
18:3*n*-3 (α-linolenic)	1.83	1.90a	2.45b	2.63b	0.10	***
20:3*n*-6	0.13b	0.14b	0.11a	0.11a	0.01	**
20:4*n*-6 (arachidonic)	0.41	0.48	0.37	0.38	0.05	ns
20:4*n*-3	0.003a	0.007a	0.03b	0.04b	0.009	***
20:5*n*-3 (eicosapentaenoic)	0.07a	0.09b	0.11c	0.11c	0.01	***
22:4*n*-6	0.10b	0.10b	0.07a	0.08a	0.007	***
22:5*n*-3 (docosapentaenoic)	0.25a	0.28a	0.32b	0.34b	0.02	***
22:6*n*-3 (docosahexaenoic)	0.12	0.13	0.15	0.16	0.02	ns

[a] Means in the same row with the same letter do not differ significantly at the 0.05 level of probability.

[b] See Table 1, footnote e.

Table 10
Fatty acid content of liver (mg per 100 g liver) from pigs raised on a test or control diet[a]

	Control Diet		Test diet			
Fatty acid	Female	Male	Female	Male	Sed	Significance[e]
12:0 (lauric)	1.1a	1.8b	1.3a	1.1a	0.23	*
14:0 (myristic)	21.6	17.2	20.0	21.0	2.8	ns
16:0 (palmitic)	677	590	628	677	46	ns
16:1	56.7	48.7	54.7	56.2	6.1	ns
18:0 (stearic)	905	813	872	866	45.0	ns
18:1 *trans*	14.8	12.1	10.7	11.0	1.8	ns
18:1 (oleic)	630	543	565	570	56.5	ns
18:2 *cis-vaccenic*	65.0	58.6	60.2	62.4	4.8	ns
18:2*n*-6 (linoleic)	805b	725a,b	631a	701a	48.1	**
20:1	7.6	7.0	6.6	6.8	0.6	ns
18:3*n*-3 (α-linolenic)	47.7a,b	40.3a	59.8b,c	68.8c	6.6	***
20:3*n*-6	30.1	30.3	31.1	34.1	3.2	ns
20:4*n*2-6 (arachidonic)	590c	521b	472a	454a	23.5	***
20:5*n*-3 (eicosapentaenoic)	33.1a	26.1a	83.9b	82.5b	5.5	***
22:4*n*-6	30.4c	25.0b	19.5a	16.4a	1.7	***
22:5*n*-3 (docosapentaenoic)	120b	102a	139c	131b,c	6.4	***
22:6*n*-3 (docosahexaenoic)	90b	73a	121c	112c	5.4	***
Total	4,290	3,775	3,933	3,982	243	ns
P:S ratio[b]	0.53	0.54	0.45	0.49		
n-6:*n*-3 ratio[c]	16.9	18.0	10.6	10.2		
n-6*n*-3 ratio[d]	5.00	5.39	2.86	3.06		

[a] Means in the same row with the same letter do not differ significantly at the 0.05 level of probability.
[b] P:S ratio defined as $\frac{18:2n\text{-}6 + 18:3n\text{-}3}{12:0 + 14:0 + 16:0 + 18:0}$.
[c] Ratio of 18:2*n*-6 to 18:3*n*-3.
[d] Ratio of Σ*n*-6 to Σ*n*-3.
[e] See Table 1, footnote e.

Table 11
Fatty acid content of liver (g per 100 g total fatty acids) from pigs raised on a test or control diet[a]

	Control diet		Test diet			
Fatty acid	Female	Male	Female	Male	Sed	Significance[b]
12:0 (lauric)	0.02a	0.04b	0.03a	0.02a	0.004	**
14:0 (myristic)	0.5	0.4	0.5	0.5	0.04	ns
16:0 (palmitic)	15.7	15.5	15.9	15.9	0.4	ns
16:1	1.3	1.3	1.4	1.4	0.08	ns
18:0 (stearic)	21.2	21.7	22.4	22.0	0.6	ns
18:1 *trans*	0.36b	0.33a,b	0.27a	0.27a	0.03	**
18:1 (oleic)	14.5	14.2	14.1	14.0	0.6	ns
18:1 *cis-vaccenic*	1.51	1.55	1.52	1.56	0.05	ns
18:2*n*-6 (linoleic)	18.7c	19.2c	16.1a	17.6b	0.38	***
20:1	0.18	0.18	0.17	0.17	0.01	ns
18:3*n*-3 (α-linolenic)	1.08a	1.04a	1.51b	1.68b	0.11	***
20:3*n*-6	0.70	0.80	0.80	0.89	0.07	ns
20:4*n*-6 (arachidonic)	14.0b	14.0b	12.2a	11.5a	0.5	***
20:5*n*-3 (eicosapentaenoic)	0.81a	0.75a	2.13b	2.09b	0.11	***
22:4*n*-6	0.72b	0.67b	0.51a	0.42a	0.04	***
22:5*n*-3 (docosapentaenoic)	2.8a	2.8a	3.6b	3.3b	0.13	***
22:6*n*-3 (docosahexaenoic)	2.1a	1.9a	3.1b	2.9b	0.13	***

[a] Means in the same row with the same letter do not differ significantly at the 0.05 level of probability.
[b] See Table 1, footnote e.

Table 12
Estimates of C20 and C22 *n*-3 PUFA intake (g per person per annum) from pork based on per capita consumption of 21.2 kg

	18:3*n*-3	20:4*n*-3	20:5*n*-3 (EPA)	22:5*n*-3	22:6*n*-3 (DHA)	Total 20–22 *n*-3
20% fat						
Control						
Muscle	1.8	ND[a]	0.7	1.6	0.7	3.0
Adipose tissue	54.0	0.9	1.5	5.6	2.4	10.4
Total	55.8	0.9	2.2	7.2	3.1	13.4
Test						
Muscle	2.8	ND	1.4	2.1	0.9	4.4
Adipose tissue	77.6	1.2	1.8	7.3	3.6	13.9
Total	80.4	1.2	3.3	9.4	4.5	18.3
10% fat						
Control						
Muscle	2.0	ND	0.8	1.8	0.8	3.4
Adipose tissue	27.0	0.5	0.7	2.8	1.2	5.2
Total	29.0	0.5	1.5	4.6	2.0	8.6
Test						
Muscle	3.1	ND	1.6	2.4	1.0	5.0
Adipose tissue	38.8	0.6	0.9	3.6	1.8	6.9
Total	41.9	0.6	2.5	6.0	2.8	11.9

[a] ND, not detected.

whole. The *n*-6:*n*-3 ratio was 5.1, a significant improvement over the control diet (8.1).

The total *n*-3 fatty acid concentration for the control and test diets were 2.3 and 3.1% of total fatty acids, respectively (Table 9). The contributions of individual PUFA to total fatty acids (g/100 g) were similar to those in adipose tissue (Table 7).

3.6. Liver fatty acids

The concentrations of PUFA in liver (Table 10) were much higher than in muscle (Table 1) even allowing for the fourfold greater fatty acid content of liver. However, in general, the effect of the test diet was similar in both tissues with highly significant increases in 18:3 and the longer-chain *n*-3 PUFA and decreases in 18:2 and the *n*-6 PUFA except for 20:3*n*-6. Whilst the decreases in 18:2, 20:4 and 22:4 of 13, 16 and 35% were almost identical to muscle, the test diet produced much greater increases in EPA and DHA, 280 and 43% in liver compared with 100 and 35% in muscle although, the increase in 22:5 was less, 21 compared to 35%. Comparison of the fatty acid compositions of liver and muscle (Tables 2 and 11) showed a higher proportion of 18:0 and a lower proportion of 16:0 and 18:1 in liver. The percentages of 18:2 and 18:3 were similar in the muscle and liver fatty acids whereas the percentages of the longer-chain PUFA (except for 20:3*n*-6) were 3–7 fold larger in liver. Despite this, the P:S ratios, all of which were above 0.45, did not differ greatly from those of muscle (Tables 1 and 10) and the *n*-6:*n*-3 ratio remained high and similar to that in muscle: 17.5 for the combined pigs fed the control diet, falling to 10.4 for the pigs on the test diet. However, the high content of longer-chain *n*-3 PUFA resulted in Σ*n*-6:Σ*n*-3 ratios of 5.2 and 3.0 for the control and test diets, respectively.

4. Discussion and conclusions

Our strategy of decreasing dietary 18:2 whilst increasing 18:3 was highly successful in raising the quantities of 18:3 and the longer-chain *n*-3 PUFA in pork. The extent of the increases varied between fatty acids and tissues. EPA showed the greatest change, increasing 2.8 fold in liver and doubling in muscle but with only a 1.4 fold increase in adipose tissue from entire males. Increases in 18:3 were somewhat lower, about 50%, and slightly greater in muscle than liver and adipose tissue. DHA increased 50% in adipose tissue, followed by 43% in liver and 35% in muscle whereas 22:5*n*-3 increased by 29% in adipose tissue and muscle and only 21% in liver.

Despite these changes the levels of PUFA in muscle were somewhat lower than we have reported previously (Enser et al., 1996). The lower levels of 18:2 and 18:3 in the present study (Table 1) result from the extremely lean pigs with very little marbling fat, 1.2% total fatty acids compared with 2.2%. In Enser et al. (1996), 18:2 was 302 mg per 100 g muscle. However, 18:2 expressed as g/100 g fatty acids was more similar between the studies. In Enser et al. (1996) 18:2 was 14.2 and 18:3 was 0.95 g/100g. Longer chain PUFA levels (C20 and C22) reported by Enser et al. (1996), were also higher than those in Table 1, which is partly due to the difference in fat content but probably also due to the (formerly) widespread use of fish meal in pig diets.

The effects on tissue fatty acids of feeding linseed resemble those reported by Romans et al. (1995b) after feeding 35 g/kg 18:3 for 28 days before slaughter for adipose tissue although they obtained greater increases in 18:3 and EPA in *longissimus thoracis* and liver. We observed significant increases in DHA after feeding linseed although other short and long-term feeding trials with linseed have failed to increase tissue DHA levels consistently (Riley et al., 1998a,b; Ahn et al., 1996; Cherian & Sim, 1995; Specht-Overholt et al., 1997). The reason for this low deposition is unclear but in the long-term feeding trials reported, dietary levels of 18:3 have ranged from 15 to 35 g/kg, much more than in the present study. This has resulted in much higher tissue concentrations of 18:3 and EPA and may indicate competitive exclusion of DHA from tissue lipids, particularly from phospholipids which normally contain only small amounts of 18:3.

The concentration of PUFA in any tissue results from several factors which include 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 amount and type of lipids into which

they may be incorporated. If it is assumed that changes in liver C20-22 PUFA as a result of feeding different levels of 18:2 and 18:3 indicate their availability in the tissue then it is constructive to look at the relative changes in different *n*-3 PUFA. The ratio of 22:6 to its precursor 22:5 increased from 0.73 in the control diet to 0.86 in the test diet whereas the ratio of 22:5–20:5 fell from 3.75 to 1.62. This indicates that the availability of 22:5 can drive the synthesis of 22:6 and that the limiting process is conversion of 20:5 to 22:5, contrary to the usual view that desaturases rather than elongases control PUFA synthesis (Brenner, 1989).

It is unlikely that lowering dietary 18:2 alone caused the increase in DHA that we found since tissue 18:2 levels were similar to ours in studies in which DHA did not increase. This analysis tends to confirm the idea that competitive exclusion of DHA from phospholipid synthesis explains the failure to obtain higher concentrations when much larger amounts of linseed were fed (Ahn et al., 1996; Cherian & Sim, 1995; Romans et al., 1995a,b). It also indicates that feeding more 18:3 than in the present study could raise tissue DHA further before the 6-fold higher rate of EPA deposition excluded it.

4.1. Nutritional ratios

Concerns about excess saturated fat and a defi-ciency of *n*-3 fatty acids in the human diet has led to recommendations that the ratio of polyunsaturated fatty acids to saturated fatty acids (P:S ratio) be increased to 0.4 or higher and that the ratio of *n*-6 to *n*-3 fatty acids in the diet be lowered to between 1 and 4 (Department of Health and Social Security, 1984 and 1994). Pig meat normally has a high 18:2 content, producing a high P:S ratio, but an unfavourable *n*-6:*n*-3 ratio. A major aim of the work, therefore, was to improve the *n*-6:*n*-3 ratio, whilst maintaining a beneficially high P:S ratio. The test diet produced a P:S ratio in muscle of 0.4, close to the minimum recommended value for the human diet as a whole. Although we achieved *n*-6:*n*-3 ratios of nearly 5 in the adipose tissue, the change is particularly significant since pork is the major red meat source of 18:2 (Enser et al., 1996). If the level of intramuscular fat were higher, a desirable feature for eating quality, the *n*-6:*n*-3 ratio in the muscle would be lower overall as a result of the lower ratio in muscle triacylglycerols compared to the phospholipids. Given the high intake of 18:2 from other dietary sources the effect of meat 18:3 on endogenous synthesis of long-chain PUFA in man may be small relative to the supply of these fatty acids by meats in the diet. The muscle Σn-6:Σn-3 ratio of 5 is a significant improvement on the current average for pigmeat (about 7). Improvements in the *n*-6: *n*-3 ratio due to the test diet were also evident in adipose tissue (reduced from about 9.0 for the control to 4.8 for the test diet), liver (reduced from 5.2 to 2.9) and sausages (reduced from 8.1 to 5.1), whilst still maintaining beneficially high P:S ratios.

4.2. Contribution to the diet

The COMA report on the nutritional aspects of cardiovascular disease (Department of Health and Social Security, 1994) recommended that the intake of C20 and C22 *n*-3 PUFA should be increased from the current population average of about 100 mg per day to 200 mg. Based on the data given in Tables 8 and 9, a 100 g serving of pork sausages with a 650 g/kg meat content (test diet) would provide 75–80 mg of C20 and C22 *n*-3 PUFA (about 75% of the current population daily average intake), whilst a 100 g serving of liver (test diet) would provide approximately 335 mg, almost 25% of the weekly recommended intake, including 200 mg of pre-formed EPA and DHA.

Estimates of C20 and C22 *n*-3 PUFA intake from pork, assuming recent consumption figures, are given in Table 12. At 100 g/kg fat, it is estimated that the test diet would provide 11.9 g of long chain *n*-3 PUFA to the human diet per annum, and 18.3 g at 200 g/kg fat (predominantly from adipose tissue rather than muscle), about a quarter of the recommended annual requirement (Department of Health and Social Security, 1994). The quantity of *n*-3 PUFA supplied by pigmeat is highly dependent upon the quantity of adipose tissue, since adipose tissue from pigs contains appreciable amounts of C20-22 *n*-3 PUFA (Table 7), unlike ruminant adipose tissue where the longer chain PUFA are not detectable (Enser et al., 1996). Compared to beef and lamb, which contribute about 3.6 and 2.4 g, respectively, of C20-22 *n*-3 PUFAs to the human diet at current consumption levels (Enser et al., 1996), pork is a better source of these nutritionally important fatty acids and may be more readily manipulated to increase the concentrations. However, the limiting factor in raising *n*-3 PUFA in pork is the increase in susceptibility to oxidative rancidity. The level of supplementation we chose was designed to avoid such problems, particularly fishy taints in bacon, which is an oxidatively unstable product, observed by Romans et al. (1995b). The meat quality studies are reported elsewhere (Sheard et al., in press).

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References

Ahn, D. H., Lutz, S., & Sim, J. S. (1996). Effects of dietary α-linolenic acid on the fatty acid composition, storage stability and sensory characteristics of pork loin. *Meat Science, 43*, 291–299.

Brenner, R. R. (1974). The oxidative desaturation of unsaturated fatty acids in animals. *Molecular and Cellular Biochemistry, 3*, 41–52.

Brenner, R. R. (1989). Factors influencing fatty acid chain elongation and desaturation. In A. J. Vergroesen, & M. Crawford, *The role of fat in human nutrition* (2nd ed.) (pp. 45–79). London: Academic Press Ltd.

Cherian, G., & Sim, J. S. (1995). Dietary α-linolenic acid alters the fatty acid composition of lipid classes in swine tissues. *Journal of Agricultural and Food Chemistry, 43*, 2911–2916.

Cunnane, S. C., Stitt, P. A., Ganguli, S., & Armstrong, J. K. (1990). Raised omega-3 fatty acid levels in pigs fed flax. *Canadian Journal of Animal Science, 70*, 251–254.

Department of Health and Social Security (1984). Report on Health and Social Subjects No. 28. *Nutritional Aspects of Cardiovascular Disease*. London: HMSO.

Department of Health and Social Security (1994). Report on Health and Social Subjects No. 46. *Nutritional Aspects of Cardiovascular Disease*. London: HMSO.

Enser, M., Hallett, K., Hewitt, B., Fursey, G. A. J., & Wood, J. D. (1996). Fatty acid content and composition of English beef, lamb and pork at retail. *Meat Science, 42*, 443–456.

Enser, M., Hallett, K. G., Hewett, B., Fursey, G. A. J., Wood, J. D., & Harrington, G. (1998). The polyunsaturated fatty acid composition of beef and lamb liver. *Meat Science, 49*, 321–327.

Folch, J., Lees, M., & Stanley, G. H. S. (1957). A simple method for the isolation and purification of total lipids from animal tissues. *Journal of Biological Chemistry, 226*, 497–509.

Irie, M., & Sakimoto, M. (1992). Fat characteristics of pigs fed fish oil containing eicosapentaenoic and docosahexaenoic acids. *Journal of Animal Science, 70*, 470–477.

Ishida, M., Konno, Y., Suzuki, K., Ogawa, Y., & Abe, H. (1996). The effects of fish oil-enriched with *n*-3 polyunsaturated fatty acids on lipids and tasty compounds of pork loin. *Nippon Shokuhin Kagaku Kogaku Kaishi, 43*, 1219–1226.

Leskanich, C. O., Matthews, K. R., Warkup, C. C., Noble, R. C., & Hazzledine, M. (1997). The effect of dietary oil containing (*n*-3) fatty acids on the fatty acid, physiochemical, and organoleptic characteristics of pig meat and fat. *Journal of Animal Science, 75*, 673–683.

Morgan, C. A., Noble, R. C., Cocchi, M., & McCartney, R. (1992). Manipulation of the fatty acid composition of pig meat lipids by dietary means. *Journal of the Science of Food and Agriculture, 58*, 357–368.

Rhee, K. S., Ziprin, Y. A., Ordonez, G., & Bohac, C. E. (1988). Fatty acid profiles of the total lipids and lipid oxidation in pork muscles as affected by canola oil in the animal diet and muscle location. *Meat Science, 23*, 201–210.

Riley, P., Enser, M., Hallet, K., Hewett, B., Wood, J. & Atkinson, J. (1998a). Long-term feeding of low levels of linseed before slaughter to manipulate tissue fatty acid composition and improve pork nutritional value. *Proceedings of the 15th IPVS Congress, Birmingham, England*, 5–9 July. p. 16.

Riley, P., Enser, M., Hallet, K., Hewett, B., Wood, J. & Atkinson, J. (1998b). Short-term feeding of a high level of linseed before slaughter to rapidly alter tissue fatty acid composition and improve pork nutritional value. *Proceedings of the 15th IPVS Congress, Birmingham, England*, 5–9 July. p. 28.

Romans, J. R., Johnson, R. C., Wulf, D. M., Libal, G. W., & Costello, W. J. (1995a). Effects of ground flaxseed in swine diets on pig performance and on physical and sensory characteristics of ω-3 fatty acid content of pork. I. Dietary level of flaxseed. *Journal of Animal Science, 73*, 1982–1986.

Romans, J. R., Wulf, D. M., Johnson, R. C., Libal, G. W., & Costello, W. J. (1995b). Effects of ground flaxseed in swine diets on pig performance and on physical and sensory characteristics of ω-3 fatty acid content of pork. II. Duration of 15% dietary flaxseed. *Journal of Animal Science, 73*, 1987–1999.

Sheard, P. R., Enser, M., Wood, J. D., Nute, G. R., Gill, B. P. & Richardson, R.I. (in press). Shelf life and quality of pork and pork products with raised *n*-3 PUFA. *Meat Science*.

Specht-Overholt, S., Romans, J. R., Marchello, M. J., Izard, R. S., Crews, M. G., Simon, D. M., Costello, W. J., & Evenson, P. D. (1997). Fatty acid composition of commercially manufactured omega-3 enriched pork products, haddock and mackerel. *Journal of Animal Science, 75*, 2335–2343.

Ulbricht, T. L. V., & Southgate, D. A. T. (1991). Coronary heart disease: seven dietary factors. *Lancet, 338*, 985–992.

Wood, J. D., Enser, M., Fisher, A. V., Nute, G. R., Richardson, R. I., & Sheard, P. R. (1999). Manipulating meat quality and composition. *Proceedings of the Nutrition Society, 58*, 1–8.