Chapter 2

Effect of increasing intakes of α-linolenic acid on growth performance, essential fatty acid status and plasma lipids in weanling piglets

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Abstract

Weanling piglets (n = 360) were fed diets with different levels of α-linolenic acid (ALA, C18:3 n-3), the levels being 0.22, 0.47, 0.77 and 1.13 % of metabolizable energy. The experimental diets were formulated by the addition of various amounts of linseed oil at the expense of corn oil. The experimental diets were fed for two weeks followed by a three-week period during which all piglets received the same commercial diet. Intakes of ALA above 0.22 energy % tended to increase growth during the first two weeks post weaning and tended to reduce feed conversion during the first week. The average increase in weight gain was 9% and the decrease in feed conversion was 14%. The diet with 1.13 energy % ALA produced a significantly better body condition after two weeks than did the diet with 0.22 energy % ALA. The good condition persisted after the pigs had been transferred to the commercial diet. Increasing amounts of ALA in the diets stimulated the desaturation and elongation into eicosapentaenoic (EPA, C20:5 n-3) and docosahexaenoic acid (DHA, C22:6 n-3) and the incorporation of these fatty acids into erythrocyte membranes. The piglets showed a post-weaning decrease in total cholesterol, HDL cholesterol and phospholipids, but the intake of various amounts of linseed oil did not influence the concentration of plasma lipids. The requirement of ALA by weanling piglets to display maximum growth is not known, but this study indicates that it may be above 0.22 energy %.

Introduction

Weanling piglets are prone to the development of the so-called post-weaning syndrome which is associated with atrophy of the villi, inflammation of the gut (Cera et al., 1988; Hall et al., 1989; Hampson, 1986; Kenworthy, 1976; Nabuurs, 1991) and depressed performance (Jahn and Uecker, E., 1987; Svedsen et al., 1974; Svensmark et al., 1989). There is evidence that dietary n-3 polyunsaturated fatty acids (PUFAs) may antagonize atrophy of villi and have anti-inflammatory activity. In growing chicks, the intake of extra n-3 PUFAs has been shown to improve performance and decrease the inflammatory response to LPS from *S. typhimurium* and *S. aureus* (Korver and Klasing, 1997). In young mice with hypoxia-induced bowel necrosis, supplementation with n-3 PUFAs reduced the degree of necrosis (Akisu et al., 1998). Mucosal damage in food-sensitive enteropathy in mice was prevented by supplementation with n-3 PUFAs (Ohtsuka et al., 1997).

The beneficial effects of n-3 PUFAs probably relate to their conversion into eicosanoids which have a variety of biological functions, including stimulation of immunity (Wu and Meydani, 1998; Fritsche et al., 1993). The parent compound of the eicosanoids is α-linolenic acid (ALA, C18:3 n-3), which can be converted by piglets (Clandinin et al., 1985) into the direct precursor, eicosapentaenoic acid (EPA, C20:5 n-3). The piglet is also able to further desaturate and elongate EPA into docosahexaenoic acid (DHA, C22:6 n-3) which is abundant in brain and retina (Bourre et al., 1993; Ward et al., 1998; Clandinin, 1999; Arbuckle et al., 1994; Arbuckle and Innis, 1992). The requirement of ALA by piglets is not exactly known (Innis, 1993), but it could be suggested that extra intake of n-3 PUFAs is beneficial. Diets for weanling piglets typically contain 0.20 – 0.30 % (w/w) n-3 PUFAs in the form of the sum of ALA, EPA and DHA (unpublished results).

In this study, weanling piglets were fed experimental diets with increasing contents of linseed oil which is rich in ALA. Three levels of linseed oil were added to the diets at the expense of the corn oil component which is rich in linoleic acid (LA, C18:2 n-6). The control diet without linseed oil had a nutrient composition similar to that of common diets for weanling piglets. The following three questions were addressed. (i) Does the consumption of increasing amounts of linseed oil affect weight gain, feed conversion, body condition and consistency of the faeces? In the light of the above-mentioned, it was anticipated that extra ALA in the form of linseed oil would have beneficial effects. (ii) Does linseed oil ingestion raise the status of n-3 PUFAs as mirrored by the fatty acid composition of erythrocyte membranes? The intake of PUFAs is generally reflected by the fatty acid composition of erythrocytes (Alessandri et al., 1996; Arbuckle and Innis, 1993; Goustard-Langelier et al., 1999; Rooke et al., 1998; Ward et al., 1998), and it was expected that this study would add to knowledge of quantitative relationships. (iii)

Does the intake of linseed oil influence the concentrations of plasma lipids? In rats, diets containing linseed oil instead of corn oil have been shown to lower plasma triglyceride and cholesterol concentrations (Herman and Beynen, 1989). It was expected that the answer to the first question would provide information as to the optimum level of ALA in diets for weanling piglets. Answers to the second and third question would give more insight in fundamental aspects of fatty acid and lipid metabolism in weanling piglets.

Animals, materials and methods

Animals, feed and housing

Three hundred and sixty weanling pigs (F2 cross-bred: GY x [Finnish X Dutch Landrace]), weighing on average 8.5 kg and aged 21 days, were used. They were housed in pens containing 10 piglets each. According to a randomised complete block design the pens were allocated to one of the four dietary treatments on the basis of weight, gender and ancestry of the piglets. The experiment was carried out in the form of three cohorts of 120 piglets each. The pens (2.60 x 1.20 m) were climate controlled and had a combination of a slatted and concrete (1.10 x 1.20 m) floor. The piglets had ad libitum access to feed and water. Each pen was equipped with a single-space self-feeder and a water nipple. The room temperature was 26 °C on the first day after weaning, gradually declining to 23 °C after 35 days. Daylight could enter the pens. During the experiment one piglet died due to an internal bleeding caused by vene puncture. No medicines were used.

There were 4 experimental diets with an increasing amount of linseed oil. Table 1 shows the ingredients and analysed composition of the experimental diets. With increasing contents of linseed oil the amount of ALA was higher and that of LA lower, but the analysed macronutrient levels of all four diets were similar. The diets, which were in meal form, were formulated to meet the requirements of growing pigs as set by the National Research Council (1998). The diets were fed for two weeks. This two-week period was followed by a three-week period during which all piglets received the same commercial diet (Standard pig pellet “315”, Cehave, Veghel, The Netherlands). The declared composition of the commercial diet was 174 g crude protein/kg, 40 g crude fat/kg, 45 g crude fiber/kg, 62 g ash/kg and 105 g moisture/kg.

Data collection and analyses

The piglets were weighed on days 0, 7, 14 and 35 post weaning. Amounts of feed offered were recorded and left-overs were weighed to calculate feed intake. Feed samples were taken for chemical analyses. Crude fat concentration and fatty acid composition of the diets were determined according to the methods of Folch et

Table 1. Ingredients, chemical composition and fatty acid composition of the experimental diets.

	α-linolenic acid (% of ME)[1]			
	0.22	0.47	0.77	1.13
Ingredients (g/kg)				
Corn oil	50	46	41	36
Linseed oil	0	4	9	14
Constant components[2]	950	950	950	950
Chemical analysis				
Dry matter (g/kg)	906	905	907	906
Crude protein (g/kg dm[3])	191	193	187	194
Crude fat (g/kg dm)	54	55	55	55
Crude fiber (g/kg dm)	27	26	27	27
Ash (g/kg dm)	42	43	47	47
Analysed fatty acids (g/100 g methylesters)[4]				
C16:0	12.87	12.90	12.94	12.71
C18:0	2.30	2.38	2.53	2.83
C18:1 n-9	24.95	25.00	24.67	24.63
C18:2 n-6	51.92	48.11	44.26	41.17
C18:3 n-3	1.66	3.37	5.59	8.17
n-3:n-6 ratio	0.03	0.07	0.13	0.20

[1] ME = metabolizable energy. The ME content of the diets was calculated on the basis of the ingredient composition and feed tables (National Research Council, 1998).
[2] Constant components: 505 g barley, 231.5 g dextrose, 152.5 g casein, 20 g molasses, 15 g rukanaphos, 12.5 g $CaCO_3$, 5 g vitamin-mineral premix (Cehave, Veghel, The Netherlands), 5 g tryptophan (purity 5%), 1.75 g threonine (purity 10%) and 1.75 g methionine (purity 50%). FAME: fatty acid methyl esters
[3] dm = dry matter
[4] C20:3 n-6, C20:4 n-6, 20:5 n-3, 22:4 n-6, 22:5 n-3 and 22:6 n-3 were not detectable.

al. (1957) and Metcalfe et al. (1966), respectively. Crude protein, crude fiber and ash were determined by the Weende analysis.

The consistency of faeces was scored weekly on a scale ranging from 0 to 3 (0 = normal, solid faeces, 1 = soft, looser than normal stools, 2 = diarrhoea and 3=liquid, severe diarrheal faeces). The condition of the pigs was scored weekly, the scores being based on an integration of color and gloss of the skin, hair length and meat cover (0 = good, 3 = bad condition). Both faeces and body condition were scored by the same experienced person who was blinded to treatment modality.

Blood samples were collected by vena cava puncture on days 0, 7 and 14 post weaning from one pig choosen at random out of each pen. For the analysis of the fatty acid composition of erythrocyte membranes, blood was collected in EDTA-containing tubes. Erythrocytes were separated by centrifugation for 5 min at 3000 rpm. The erythrocytes were haemolysed and stored at - 80 °C until fatty acid analysis. From the erythrocyte membranes fatty acids were extracted, methylated (Metcalfe et al., 1966) and determined by gas chromatography (Nelson, 1975; Angelico et al., 1983; Popp-Snijders, 1985). Fatty acid methyl esters were isolated on a Chrompack 9002 gas chromatograph equipped with a CP-FFAP CB 25 m x 0.32 mm column (Chrompack, Bergen op Zoom, The Netherlands) and a flame ionization detector. To analyse plasma lipids and lipoproteins, blood was taken in heparinized tubes. Plasma triglycerides, phospholipids, total cholesterol and HDL cholesterol were measured enzymatically using test combinations (Boehringer-Mannheim GmbH, Mannheim, Germany). Lipoproteins were isolated according to Terpstra et al (1982).

Statistical analyses

Results are presented as means ± SEM. Data were tested for normal distribution with the Kolmogorov-Smirnov test. Diet effects were evaluated for statistically significant differences with ANOVA and Bonferroni test. The body-condition and faeces-consistency scores were subjected to the Chi-square-test. The correlation between body condition and either the α-linolenic acid to linoleic acid (18:2 n-6) or the ratio of eicosapentaenoic acid to arachidonic acid (C20:4 n-6) was calculated. The correlations were based on group-mean values which can be considered the best estimates for each diet. For daily gain and feed intake, pen was the experimental unit and for the data on the fatty acid composition of the erythrocyte membranes the experimental unit was pen as well, because one animal per pen was sampled. The model was y = mean + treatment effect + error. For all statistical analyses, the SPSS program (SPSS Inc., Chicago, IL, USA) was used. The level of statistical significance was pre-set at $p<0.05$.

Results

There were no significant differences in average daily gain (ADG) and feed conversion ratio for the four experimental diets (Table 2). ADG during weeks 1 + 2 rose with increasing intakes of ALA and during weeks 1 and 2 it was on average 9% higher for the diets containing linseed oil than for the control diet without linseed oil. The overall average daily feed intake (ADFI) during weeks 1 + 2 was 246 g and there were no significant differences for the four diets. Feed conversion during the first week was consistently lower for the diets with linseed oil, the lowering being 14%, which failed to reach statistical significance. After

Table 2. Growth performance of weanling piglets fed increasing amounts of linseed oil.

	α-linolenic acid (% of ME)				Pooled	
Item	0.22	0.47	0.77	1.13	SEM	P value
Weight, day 0 (g)	8521	8429	8424	8502	473	0.998
ADG, week 1 (g)	138	172	155	172	93	0.230
ADG, week 2 (g)	272	271	292	279	116	0.795
ADG, week 1 + 2 (g)	205	221	224	226	185	0.679
ADFI, week 1 (g)	256	266	253	276	93	0.614
ADFI, week 2 (g)	421	440	444	452	158	0.814
ADFI, week 1 + 2 (g)	339	353	348	364	236	0.768
Feed conversion, week 1	1.93	1.59	1.70	1.71	0.12	0.272
Feed conversion, week 2	1.56	1.66	1.52	1.66	0.08	0.485
Feed conversion, week 1 + 2	1.68	1.63	1.57	1.67	0.08	0.771

Results are means for 9 pens, containing 10 piglets each, per dietary treatment.

two weeks on the experimental diets, all piglets were switched to the same commercial diet. There was no significant carry-over effect of ALA intake. After three weeks on the commercial diet, body weights were 22.61 ± 0.84, 23.17 ± 0.54, 22.84 ± 0.75 and 23.09 ± 0.89 kg (mean ± SE, n = 90) for the piglets with increasing post-weaning intakes of ALA.

All diets induced solid faeces and there was no diet effect on faeces-consistency scores. The median of the scores for days 7 and 14 was 0.5 and 0.0 respectively. There were diet-related differences in body-condition of the pigs. The diet with 1.13 % ALA had produced a significantly better condition after two weeks than had the diet with 0.22 energy % ALA. The improved condition persisted after the pigs had been switched to the commercial diet. After 21, 28 and 35 days, the body-condition scores for the pigs earlier fed the diet with 1.13 energy % ALA were 0.6, 0.9 and 1.3, respectively, whereas for the pigs weaned onto the control diet the scores were 0.8, 1.1 and 1.4. Such a carry-over effect on body condition was also seen for the diet containing 0.47 energy % ALA, but it lasted only until day 28, the score at that time point being 0.9.

The increasing amounts of ALA in the diets were reflected by the amounts of this fatty acid in the erythrocyte membranes (Table 3). The percentage of EPA in the erythrocytes was highest for the diet with the highest amount of ALA. At two weeks after weaning only the level of DHA was elevated in piglets fed the diet with the highest amount of ALA. The amounts of LA, dihomo-γ-linolenic acid

Table 3. Fatty acid composition of erythrocyte membranes in piglets fed the experimental diets for 14 days as from weaning

	α-linolenic acid (% of ME)				Pooled	
	0.22	0.47	0.77	1.13	SEM	P value
Analysed fatty acids (g/100 g methylesters)						
C16:0	22.00	22.31	21.89	21.81	0.222	0.579
C18:0	11.46	11.57	11.70	12.00	0.231	0.234
C18:1 n-9	30.37	29.26	29.20	29.39	0.549	0.705
C18:2 n-6	12.50	13.72	13.17	12.98	0.501	0.633
C18:3 n-3	0.17	0.23	0.35	0.46	0.129	0.000
C20:3 n-6	0.27	0.26	0.29	0.27	0.013	0.414
C20:4 n-6	4.39	4.36	4.38	4.13	0.123	0.367
C20:5 n-3	0.07	0.06	0.10	0.19	0.059	0.001
C22:4 n-6	0.44	0.44	0.40	0.36	0.041	0.012
C22:5 n-3	ND[1]	ND	ND	ND		
C22:6 n-3	1.74	1.65	1.83	2.04	0.170	0.002
n-3	1.98	1.93	2.29	2.69	0.363	0.001
n-6	17.61	18.77	18.24	17.74	0.535	0.651
n-3:n-6 ratio	0.11	0.10	0.13	0.15	0.023	0.000

Results are means for 9 pigs per dietary treatment.
[1] ND = non detectable

(DGLA, C18:3 n-6) and AA in the erythrocyte membranes were similar for all four diets. There was significantly more adrenic acid (C22:4 n-6) in the erythrocyte membranes of piglets fed on the diet with 0.22 energy % ALA when compared to that seen after feeding the diet with 1.13 energy % ALA. The changes in fatty acid composition of erythrocyte membranes resulted in significantly different ratios of ALA to LA for all four diets, the ratio increasing with increasing amounts of ALA (Fig. 1). A similar pattern was seen for the ratio of EPA to AA in erythrocyte membranes (Fig. 2).

There was a linear relation between the group-mean fatty acid composition of erythrocyte membranes and group-mean body condition scores. The regression line between the body condition score on day 14 and the ALA to LA ratio can be described by $y = -18.29x + 1.6552$ with $R^2 = 0.8686$ (Fig. 3). The regression between the body condition on day 14 and the EPA to AA ratio can be described by $y = -13.373x + 1.5584$ with $R^2 = 0.9533$ (Fig. 4). Thus, high amounts of both the parent compound and direct eicosanoid precursor of the n-3 family of PUFAs

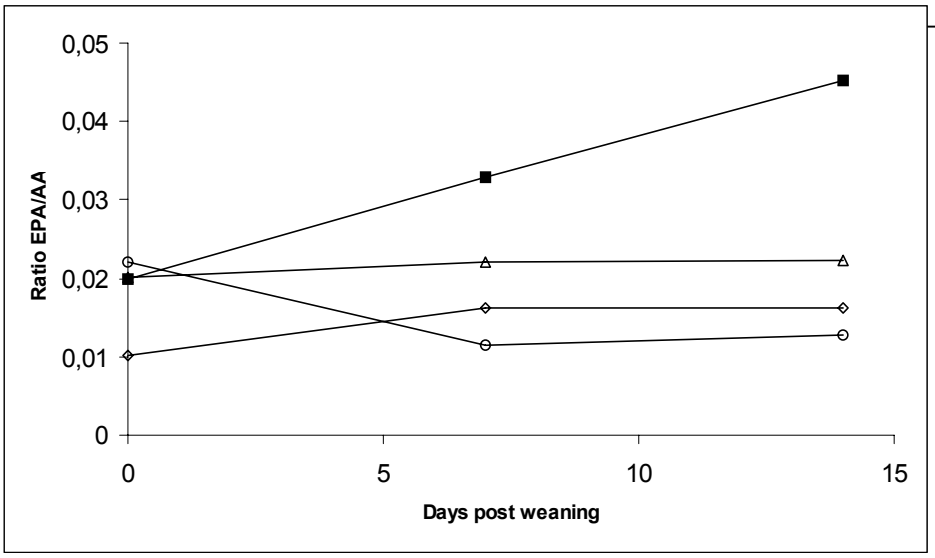

fig. 1. Time course of the ratio of α-linolenic acid (ALA) to linoleic acid (LA) in erythrocyte membranes from weanling piglets fed the experimental diets. Symbols: ○, 0.22 energy % ALA; ●, 0.47 energy % ALA; □, 0.77 energy % ALA; ■, 1.13 energy % ALA.

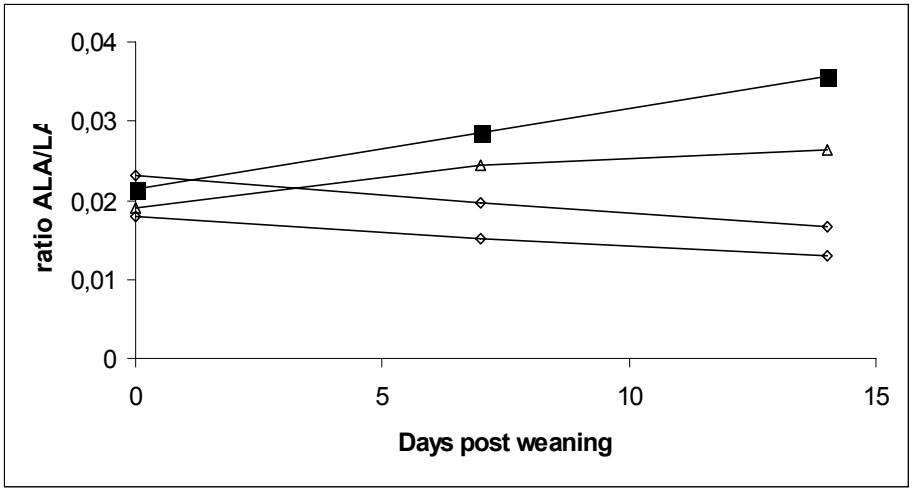

Fig. 2. Time course of the ratio of eicosapentaenoic acid (EPA) to arachidonic acid (AA) in erythrocyte membranes from weanling piglets fed the experimental diets. Symbols: ○, 0.22 energy % ALA; ●, 0.47 energy % ALA; □, 0.77 energy % ALA; ■, 1.13 energy % ALA.

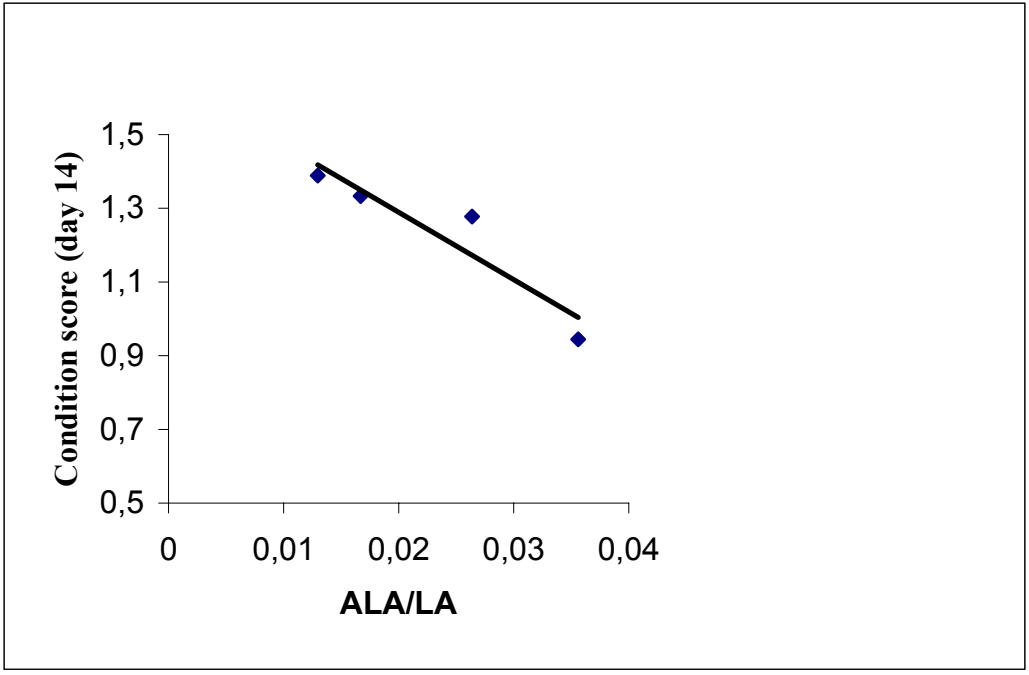
Condition score (day 14)
1,5
1,3
1,1
0,9
0,7
0,5
0
0,01
0,02
0,03
0,04
ALA/LA

Fig. 3. Relationship between group mean values on day 14 post weaning for the ratio of α-linolenic acid (ALA) to linoleic acid (LA) in erythrocyte membranes and body condition scores. Symbols: ○, 0.22 energy % ALA; ●, 0.47 energy % ALA; □, 0.77 energy % ALA; ■, 1.13 energy % ALA.

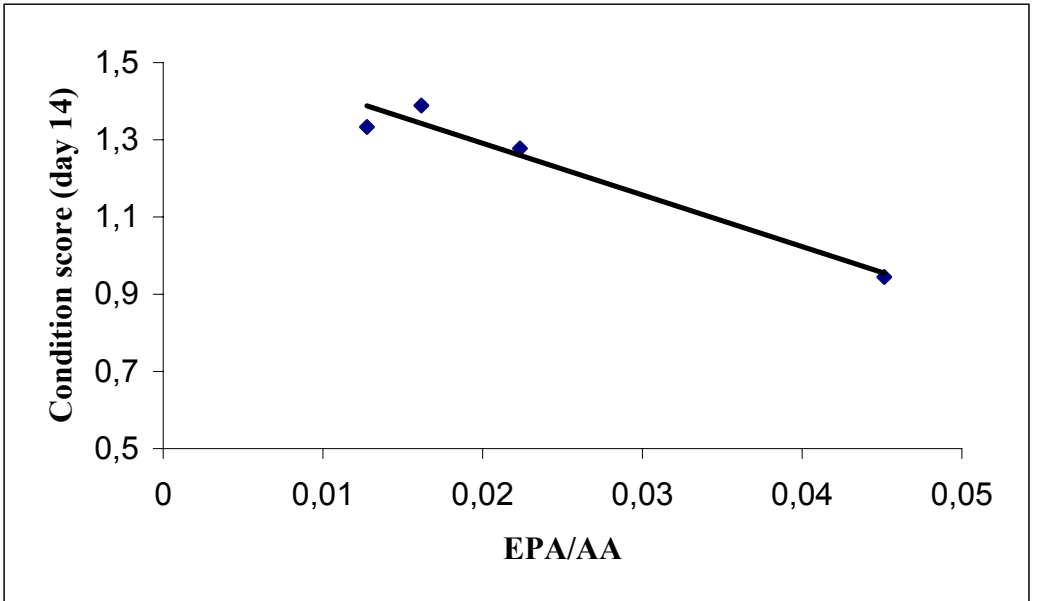
Condition score (day 14)
1,5
1,3
1,1
0,9
0,7
0,5
0
0,01
0,02
0,03
0,04
0,05
EPA/AA

Fig. 4. Relationship between group mean values on day 14 post weaning for the ratio of eicosapentaenoic acid (EPA) to arachidonic acid (AA) in erythrocyte membranes and body condition scores. Symbols: ○, 0.22 energy % ALA; ●, 0.47 energy % ALA; □, 0.77 energy % ALA; ■, 1.13 energy % ALA.

Table 4. Plasma lipid concentrations in weanling piglets fed increasing amount of linseed oil

		α-linolenic acid (% of ME)				Pooled	
	Day	0.22	0.47	0.77	1.13	SEM	P value
Total cholesterol	0	5.80	5.82	5.48	5.79	0.72	0.957
(mmol/l)	7	1.73	1.69	1.65	1.92	0.15	0.648
	14	2.06	2.20	2.23	1.97	0.23	0.848
HDL cholesterol	0	2.36	1.73.	1.67	1.74	0.25	0.640
(mmol/l)	7	0.79	0.77	0.73	0.68	0.05	0.505
	14	0.90	1.25	0.96	0.89	0.17	0.446
Triacylglycerols	0	0.32	0.36	0.34	0.33	0.04	0.999
(mmol/l)	7	0.33	0.30	0.29	0.27	0.03	0.592
	14	0.39	0.41	0.31	0.41	0.04	0.374
Phospholipids	0	2.74	2.58	2.53	2.68	0.22	0.993
(mmol/l)	7	1.10	1.06	1.03	1.04	0.07	0.915
	14	1.06	1.42	1.24	1.25	0.11	0.662

Resultes are means for 9 pigs per dietary treatment.

Table 5. Lipoprotein cholesterol concentrations in weanling piglets fed increasing amounts of linseed oil

Lipoprotein		α-linolenic acid (% of ME)				Pooled	
cholesterol	Day	0.22	0.47	0.77	1.13	SEM	P value
VLDL (mmol/l)	0	0.21	0.22	0.21	0.16	0.44	0.808
	7	0.05	0.03	0.05	0.05	0.05	0.502
	14	0.06	0.06	0.05	0.07	0.07	0.838
LDL (mmol/l)	0	4.13	4.29	3.65	4.24	4.57	0.528
	7	0.97	1.07	1.02	1.62	0.88	0.171
	14	1.17	1.23	1.51	1.06	1.18	0.539
HDL (mmol/l)	0	1.92	1.64	1.81	1.66	1.10	0.789
	7	0.71	0.67	0.76	0.65	0.25	0.150
	14	0.87	0.84	0.97	0.81	0.52	0.652

Results are means for 9 pigs per dietary treatment.

relative to those of the n-6 family of PUFAs were associated with low body-condition scores, i.e. good appearance.

The levels of total cholesterol, HDL cholesterol and phospholipids in plasma showed a decrease during the first week after weaning and generally rose again during the second week without reaching the levels at weaning (Table 4). Plasma concentrations of triacylglycerols were generally higher at 14 days after weaning than at weaning. There was no sytematic effect of the dietary ALA concentration on plasma lipids. Lipoprotein-cholesterol concentrations were not significantly affected by the amount of ALA in the diet (Table 5). The recovery of lipoprotein cholesterol was on average 116% of total plasma cholesterol. At weaning, the VLDL, LDL and HDL fractions carried 3, 65 and 28% of plasma total cholesterol, respectively. After 14 days, the proportion of total cholesterol in the VLDL, LDL and HDL fractions was 3, 51 and 36%. Lipoprotein-cholesterol concentrations at weaning were higher than those seen on day 14.

Discussion

In this study with weanling piglets, experimental diets were fed in which the corn-oil component was replaced by increasing amounts of linseed oil. Thus, diets were obtained with increasing levels of ALA at the expense of LA. The dietary concentrations of ALA ranged between 0.22 and 1.13 % of ME. The experimental diets produced no significant differences in weight gain and feed conversion. However, intakes of ALA higher than 0.22 energy % increased group-mean ADG during the first two weeks post weaning in a dose-dependent fashion. In addition, extra ALA in the diet, when compared with the linseed-oil free diet, reduced FCR during the first week. There were statistically significant diet effects on body condition of the pigs. The pigs fed on the diet with the highest amount of ALA had a significantly better condition than those given the diet with the lowest amount of ALA. Thus, high intake of ALA had a positive effect on the integrated measure of color and gloss of the skin, hair length and meat cover. For group means, high ratios of ALA to LA and EPA to AA in erythrocyte membranes were associated with low body-condition scores, i.e. good condition. It appears that high intakes of ALA, which is reflected by high concentrations of n-3 PUFAs in erythrocyte membranes, tended to have positive effects on ADG, FCR and body condition.

Possibly, the conditions of the present study were not suitable to demonstrate clear effects of increasing intakes of ALA on growth performance. Dietary ALA can be converted into EPA which is the direct substrate for the synthesis of eicosanoids. The status of EPA, as based on its concentration in the erythrocyte membranes, did not fall with time in the linseed-oil free, control group and was only slightly affected by the intake of ALA. It could be suggested that in

all piglets the status of EPA was optimal so that no significant effects of ALA intake on growth performance could be shown. This suggestion would imply that the initial fatty acid status of weanling piglets may affect their sensitivity to the fatty acid composition of the diet onto which they are weaned. Moreover, it cannot be excluded that the diets used in this study had ALA levels well above the requirement of weanling piglets. If this were the case, no effect of ALA would be expected. The diet with the lowest amount contained 1.10 g ALA/kg which is equivalent to 0.22 energy %. The requirement of n-3 PUFAs by piglets is not known. Recommended intakes for humans are 0.2 to 0.4 energy % (Bjerve et al., 1987; Bjerve, 1989), but a value of 1 energy % had also been put forward (Bjerve, 1989). For growing rats, 0.4 energy % had been suggested (Bourre et al., 1989b) and for adult rats an ALA intake of 0.26 energy % has been recommended (Bourre et al., 1993, Bourre et al, 1996). Although different criteria were used to arrive at the recommended intakes for the different species the range of ALA intakes in this study may be considerd appropriate to demonstrate effects, if any, on growth performance. There is evidence that apart from the absolute amount of ALA in the diet the ratio of n-3:n-6 PUFAs is important (Innis, 1991). The two types of PUFA have inhibitory effects on each others conversion into eicosanoids, whereas the eicosanoids produced from the n-6 and n-3 families of PUFAs have antagonistic activities (Calder ,1996). In this study the dietary concentration of LA was relatively high. It cannot be excluded that the high concentration of LA in the diet had influenced the observed effects on weight gain and body condition.

As expected, ingestion of increasing amounts of linseed oil did increase the status of n-3 PUFAs. The levels of EPA and DHA in erythrocyte membranes were increased in piglets fed the diets with high levels of ALA. Thus, the intake of extra ALA stimulates its desaturation and elongation. It is unlikely that ALA inhibits the catabolism of EPA and DHA. Although the intake of LA decreased with increasing intakes of linseed oil, there was no change in the LA content of erythrocyte membranes. This could indicate that LA intake with all four experimental diets was close to or above the requirement. Indeed, the LA requirement of piglets is 0.3 energy % (National Research Council, 1998), while the diet with the lowest level contained 0.22 energy %. A surprising finding emerged in that the diets with lowest amounts of ALA, and thus the highest amounts of LA, produced a somewhat higher content of adrenic acid in erythrocyte membranes, whereas the content of AA remained stable. Adrenic acid is formed by elongation of AA. Possibly, a low intake of n-3 PUFAs leads to a diminished transformation of adrenic acid. Rats fed a diet with 6 instead of 130 mg ALA/100 g showed an increase in the amount of docosapentaenoic acid (C22:5 n-6) and adrenic acid in liver, while the content of AA remained stable (Bourre et al., 1993).

During the first week after weaning, plasma levels of total cholesterol, HDL cholesterol and phospholipids decreased. This observation can be explained by the change from milk as sole source of nutrition to dry feed. Sow milk is rich in fat, where as the dry feed was lower in fat and richer in carbohydrates. It is well-known that a decrease in fat intake in favour of carbohydrates leads to a fall in plasma total cholesterol and HDL cholesterol (Herman and Beynen, 1989; Geelen et al, 1995; Salter et al, 1998; Roche and Gibney, 2000). However, the invariably associated rise of plasma triacylglycerol concentrations (Herman and Beynen, 1989) was not seen in the piglets. The intake of various amounts of linseed oil did not influence the concentrations of plasma lipids. The type of fat in the diet of rats can affect plasma lipid concentrations (Geelen et al., 1995; Van Lith et al., 1992). It seems that in this study the variation in fatty acid composition was not sufficiently extreme to elicit plasma lipid responses or that the background composition of the diet had masked any effects.

In conclusion, this study shows that a diet containing 0.22 energy % of ALA sustains growth in weanling piglets and that extra ALA had only a minor impact. This study does present suggestive evidence that extra ALA in the diet may have positive effects on ADG, FCR and body condition of weanling piglets. Further studies on the intake of n-3 PUFAs and growth performance of weanling piglets appear to be relevant. At least three important questions can be raised since the requirement of n-3 PUFAs by weanling piglets is not known. Do dietary levels lower than 0.22 energy % of ALA depress growth performance? Will higher levels be beneficial in weanling piglets kept under stressful conditions such as infectious pressure? Is the feeding of the product of ALA transformation, EPA, more effective in stimulating growth performance than is the feeding of ALA feeding?

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