

## Linseed oil in the maternal diet increases long chain-PUFA status of the foetus and the newborn during the suckling period in pigs

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Linseed oil, being rich in 18:3n-3, represents an alternative source of n-3 PUFA in the maternal diet. However, little is known about the effect of this oil on the long chain n-3 PUFA composition of offspring, which are required for normal growth and maturation of numerous organs. The main objective of the experiment was therefore to investigate fatty acid composition of tissues from sows at the end of gestation and from piglets during the first week of postnatal life in response to maternal dietary linseed oil intake. Sows received either a lard (LAR)-based diet or a linseed oil (LSO)-based diet during gestation and lactation. Fatty acid composition was evaluated in sow plasma, placenta and milk, and in different tissues of piglets on days 0, 3, 7, 21 and 32. The LSO diet increased the proportions of n-3 PUFA and especially 22:6n-3 in the placenta. The carcass of LSO piglets at birth contained greater proportions of 20:5n-3, 22:5n-3 and 22:6n-3. The LSO sow milk exhibited greater proportions of 18:3n-3 compared with the LAR sow milk. The piglets suckling LSO sows had greater proportions of 18:3n-3, 20:5n-3 and 22:5n-3 in plasma and carcass. The proportions of 22:5n-3 and 22:6n-3 were greater in the brain of LSO piglets than in that of LAR piglets during the suckling period. In conclusion, LSO in the maternal diet during gestation and lactation increases 22:6n-3 concentrations in the placenta and in the foetus carcass, and it maintains 22:6n-3 concentrations in the brain during the first week of postnatal life.

**n-3 PUFA: Maternal diet: Neonate: Fatty acid composition: Linseed**

The PUFA composition of the maternal diet during gestation and lactation can affect the composition of the foetus and the neonate (through the maternal milk)<sup>(1,2)</sup> and, consequently, modulate the development of physiological functions. Indeed, PUFA, including n-6 and n-3 PUFA, are essential components in the cells of all tissues, and are required for normal growth and maturation of numerous organ systems in newborns.

$\alpha$ -Linolenic acid (18:3n-3) and linoleic acid (18:2n-6) are the precursors of the n-3 and n-6 PUFA families, respectively. The metabolism of 18:3n-3 and 18:2n-6 by desaturation and elongation leads to a series of long-chain PUFA (LC-PUFA) including eicosapentaenoic acid (20:5n-3), docosapentaenoic acid (22:5n-3) and DHA (22:6n-3) of the n-3 family, and arachidonic acid (20:4n-6) of the n-6 family<sup>(3,4)</sup>. These precursors cannot be synthesised by animals. Precursors and/or LC-PUFA must therefore be supplied in the diet or through placental transport during foetal life. The n-6 to n-3 PUFA ratio in the diet is crucial because of enzymatic competition between 18:2n-6 and 18:3n-3 for elongation and desaturation into LC-PUFA. An inadequate n-6 to n-3 PUFA ratio may lead to an imbalance in the concentrations of n-6 LC-PUFA and n-3 LC-PUFA, and it may have consequences for the development of physiological functions. Neuringer *et al.*<sup>(5)</sup>

showed that rhesus monkeys deprived of dietary n-3 PUFA during the prenatal and postnatal periods developed visual impairment. Moreover, an epidemiological study suggested that a high 18:2n-6 status of newborns at high risk of atopy was linked to allergy sensitivity<sup>(6)</sup>.

In Western populations, over the last 40 years, pregnant and lactating women have been consuming high levels of n-6 PUFA, resulting in a high n-6 to n-3 PUFA ratio in their milk<sup>(7)</sup>. Actual recommendations are to balance the n-6 to n-3 PUFA ratio in the maternal diet by decreasing the consumption of n-6 PUFA and/or by increasing that of n-3 PUFA. For example, the Agence Française de Sécurité Sanitaire des Aliments suggests dietary intakes of 2 g/d of 18:3n-3 and 10 g/d of 18:2n-6 for pregnant and lactating women. The main sources of n-3 PUFA currently consumed are marine sources such as mackerel rich in 20:5n-3 (1.8 g/100 g of fish flesh) and 22:6n-3 (1.2 g/100 g of fish flesh). Some vegetable oils, such as linseed oil (LSO) or walnut oil, are rich in 18:3n-3 (56–71 and 9–15% of total fatty acids, respectively), and they could be used to diversify n-3 PUFA dietary sources. However, the capacity of adults to convert 18:3n-3 into 20:5n-3 or 22:6n-3 is very limited<sup>(8,9)</sup>, although some data suggest that this capacity increases during

**Abbreviations:** LAR, lard; LC-PUFA, long-chain PUFA; LSO, linseed oil.

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gestation<sup>(10,11)</sup>. Some studies have already investigated the effect of a maternal 18:3n-3-rich diet on newborn fatty acid composition. For example, Bazinet *et al.*<sup>(12)</sup> showed that piglets suckling sows consuming an 18:3n-3-rich diet had 54% more 22:6n-3 in their liver and 24% more 22:6n-3 in their brain than control piglets at 14 d of age. Farmer & Petit<sup>(13)</sup> also demonstrated that a maternal 18:3n-3-rich diet increased the percentage of 22:6n-3 (1.2 times more than control) in piglet brain at 1 d of age. Moreover, supplementation of the maternal diet with LSO slightly increased the proportions of 20:5n-3 and 22:6n-3 in piglet plasma at birth<sup>(14)</sup>. In parallel, in human subjects, maternal 18:3n-3 supplementation during pregnancy increased the proportion of 20:5n-3 by twofold in newborn plasma at birth<sup>(15)</sup>. These studies clearly confirm that 18:3n-3 supplementation can increase LC-PUFA status in the newborn. However, only one time point has been studied after birth. The main objective of our experiment was to investigate, in sows and piglets, the effect of maternal LSO intake on the fatty acid composition of the sow plasma and placenta and on the fatty acid composition of different tissues of the newborn before any suckling. The effect of maternal LSO intake on the evolution of the fatty acid composition of sow milk and piglet tissues at different time points during the suckling period was also established.

## Materials and methods

### Animals and diets

Experiments were conducted under the guidelines of the French Ministry of Agriculture for Animal Research.

Twenty sows (Large White × Landrace, 237 (SEM 3) kg) and their piglets ((Large White × Landrace) × Pietrain) from the experimental herd of INRA (St Gilles, France) were used.

Four diets were formulated according to the nutrient and energy needs of pregnant and lactating sows<sup>(16)</sup>. They included a standard gestation or lactation diet (Cooperl, Lamballe, France) supplied with 40 mg/kg  $\alpha$ -tocopherol and either lard (LAR; Charcuterie Industrielle, Mordelles, France) or LSO (Valorex, Dunkerque, France; Table 1). Sows were fed a standard gestation diet (Cooperl) until the 28th day of gestation. They were then separated into two groups: the first group received the LAR diet throughout gestation and lactation and the second group received the LSO diet throughout gestation and lactation. They were given 2.8 kg/d of feed during gestation corresponding to 42 g/d of LAR or linseed and 7 kg/d of feed during lactation corresponding to 385 g/d of LAR or linseed. This amount of feed is slightly under the usual feeding practice for pregnant and lactating sows. The transition from 2.8 to 7 kg/d was gradual within the first week of lactation. Sows were not fed *ad libitum*, but were slightly restricted to minimise feed refusals. Indeed, sows consumed all the feed that they were offered daily.

Sows were weighed every 3 weeks from day 28 of gestation until farrowing. Parturitions were not induced. Forty-eight hours after parturition, the number of piglets was adjusted to 10–12 by removing the extra piglets from the litter. Piglets were weaned at 28 (SEM 2) d. They were weighed immediately at birth, and then weekly until weaning, and weighing was done 4 d after weaning for those that were killed on day 32. At weaning, piglets that had been selected for killing on

**Table 1.** Composition of diets

Ingredients	LAR (g/kg)		LSO (g/kg)	
	Gestation	Lactation	Gestation	Lactation
Wheat	221.0	227.0	221.0	227.0
Maize	100.0	120.0	100.0	120.0
Barley	337.0	254.0	337.0	254.0
Wheat bran	155.0	100.0	155.0	100.0
Soyabean meal	90.0	210.0	90.0	210.0
Sugar beet pulp	50.0	–	50.0	–
Calcium carbonate	19.0	11.0	19.0	11.0
Dicalcium phosphate	3.0	13.0	3.0	13.0
NaCl	5.0	5.0	5.0	5.0
Trace element and vitamin mix*	5.0	5.0	5.0	5.0
LSO	0.0	0.0	15.0	55.0
LAR	15.0	55.0	0.0	0.0
Fatty acid composition (g/100 g fatty acid)				
SFA	30.0	42.0	19.1	19.5
MUFA	30.0	32.0	20.3	21.7
PUFA	40.0	26.0	60.6	58.8
<i>n</i> -6 PUFA	36.0	22.1	40.7	29.2
18:2 <i>n</i> -6	35.9	21.8	40.7	29.2
<i>n</i> -3 PUFA	4.0	2.5	19.5	29.6
18:3 <i>n</i> -3	3.8	2.4	19.0	29.3
20:5 <i>n</i> -3	0.2	0.1	0.3	0.1
<i>n</i> -6 PUFA/ <i>n</i> -3 PUFA	8.6	8.8	2.1	1.0

LAR, lard-based diet; LSO, linseed oil-based diet.

\* This premix (Cooperl) supplied per kg of diet: retinol 3 mg, cholecalciferol 37.5  $\mu$ g,  $\alpha$ -tocopherol 40 mg, menadione 2 mg, thiamine 2 mg, riboflavin 4 mg, niacin 20 mg, D-pantothenic acid 10 mg, pyridoxine 3 mg, D-biotin 0.2 mg, folic acid 3 mg, vitamin B<sub>12</sub> 20  $\mu$ g, choline 500 mg, Fe 80 mg as ferrous carbonate, Cu 10 mg as copper sulphate, Mn 40 mg as manganese oxide, Zn 100 mg as zinc oxide, Co 0.1 mg as cobalt sulphate, iodine 0.6 mg as calcium iodate and Se 0.15 mg as sodium selenite.

day 32 were placed in individual cages and offered a standard weaning diet (net energy 10.6 MJ/kg and 195 g crude protein/kg of DM; Cooperl). The amount of feed offered was adjusted to their body weight (15 g/kg body weight/d).

#### Tissue collection

**Sows.** At 92 d of gestation, blood samples were obtained by puncture of a jugular vein from each sow in heparinised tubes. After centrifugation (4000g, 10 min, 4°C), plasma was stored at -20°C. Just after expulsion of the placenta, three pieces (around 150 g each) were randomly sampled from the entire placenta. The three pieces were homogenised together using a meat grinder with a 5 mm diameter screen (Hobart, Paris, France). The homogenates were stored at -20°C. For half of the sows ( $n = 5$ ), colostrum samples were collected on day 0 (within 12 h after the onset of parturition), and milk samples were collected on days 7, 14, 21 and 28 (following the same sows throughout lactation) after oxytocin injection (1 ml of a 20 µg/ml solution) from four randomly selected teats (4–5 ml per teats). Samples from the different teats were pooled and kept at -20°C.

**Piglets.** One piglet per litter was killed at birth before suckling (day 0). Since the piglets were killed just at birth before any suckling, we could not wait for the entire litter to be born to calculate the average weight of the piglets within the litter. Therefore, we selected piglets weighing between 1.2 and 1.5 kg, corresponding to the average weight of newborn piglets in our herd over the past 10 years. Other piglets from the same litter (with birth weights and weight gain until killing close to the average weight and weight gain of the litter) were killed at days 3, 7, 21 and 32 (4 d after weaning) of age. Piglets were separated from the sow (days 3, 7 and 21), or had no access to feed (day 32) 1 h before killing. Each piglet was killed by electronarcosis and exsanguination. At exsanguination, blood samples were collected in heparinised tubes (5–10 ml) and centrifuged (4000g, 10 min, 4°C) within 15 min, and plasma was stored at -20°C. Carcasses were frozen (without the head, the tail and the internal organs). Frozen carcasses were then minced and homogenised using a meat grinder with a 5 mm diameter screen (Hobart). Samples were stored at -20°C. Brains were dissected from the head at the time of killing, and were immediately frozen at -20°C. Frozen brains were then homogenised in a meat grinder with 1 mm diameter screen (Hobart), and the samples were stored at -20°C.

#### Determination of fatty acid composition

Lipids were extracted from the tissues of piglets from four litters by dietary group (litters randomly selected) and from five randomly selected sows by dietary group. Extractions of lipids from the placenta, brain and carcass were performed using chloroform–methanol (2:1, v/v) as described by Folch *et al.*<sup>(17)</sup>. Lipids from colostrum and milk were extracted using ethyl ether–petroleum ether–ammonium (25:25:2, v/v/v) as described by Jensen & Clark<sup>(18)</sup>. The lipid content of the different tissues was determined gravimetrically. Extraction of lipids from plasma was performed using the hexane–isopropanol method (3:2, v/v)<sup>(19)</sup>. The lipid content

of the plasma was not determined because the gravimetric method is not sensitive enough for such low values.

Fatty acid composition was determined after transmethylation of lipids with boron trifluoride–methanol solution<sup>(20)</sup>, and was analysed on a Perkin Elmer Autosystem XL gas chromatograph equipped with a fused silica gel capillary column (0.25 mm × 30 m; Agilent Technologies, Massy, France), with an 80 % biscyanopropyl and 20 % cyanopropyl stationary phase, using margaric acid (17:0) as an internal standard for fatty acid composition analysis of tissues. For analysis of fatty acid composition in milk, a mixed standard with margaric acid and pelargonic acid (9:0) was used. The chromatography conditions were as follows: temperature programme 45°C for 2 min, increased to 195°C over 7.5 min, held for 7 min, then raised to 220°C for 2 min and finally increased to 240°C for 2 min. The injector and detector temperatures were maintained at 220 and 280°C, respectively. Retention times and peak areas were determined using chromatography software (Nelson Analytical, Inc., Cupertino, CA, USA). Results were expressed as proportions of the total fatty acid composition in comparison to the internal standards.

#### Statistical analysis

Data are expressed as mean values with their standard errors. Due to the large number of data, we concentrated the analysis on SFA, MUFA, PUFA, *n*-3 PUFA and *n*-6 PUFA proportions in the different tissues but not on the other classes of fatty acids. Data were analysed by analysis of variance using the general linear model procedure of Statistical Analysis Systems (1989). For zootechnical data, the fatty acid composition of sow plasma and placenta, ANOVA tested the maternal diet effect. For the other parameters (milk and piglet tissues from days 0 to 32), the model included the effect of litter, day of lactation (for milk) or piglet age (for piglet tissues), maternal diet and the day of lactation or piglet age × maternal diet interaction. Differences were considered significant at  $P \leq 0.05$ .

## Results

### Gestation outcomes and piglet growth

The maternal diet had no effect on the weight gain of sows throughout gestation (0.69 (SEM 0.05) *v.* 0.68 (SEM 0.05) g/d). Similarly, duration of gestation was not influenced by the diet (113.9 (SEM 0.3) *v.* 114.0 (SEM 0.4) d).

There were no differences in the litter size or mean birth weight of live piglets between the LAR and LSO groups (14.8 (SEM 1.3) *v.* 13.6 (SEM 1.4) piglets per litter and 1.65 (SEM 0.1) *v.* 1.61 (SEM 0.09) kg). Average daily weight gain of piglets from birth to 7 d of age (0.18 (SEM 0.01) *v.* 0.19 (SEM 0.01) g/d) and from birth to 28 d of age (0.27 (SEM 0.01) *v.* 0.25 (SEM 0.01) g/d) was not different between the two groups.

### Sow plasma

Although the proportions of both *n*-3 and *n*-6 PUFA were increased in the plasma of the LSO sows ( $P < 0.001$  and  $P = 0.01$ , respectively), the 18:2*n*-6 to 18:3*n*-3 ratio was

**Table 2.** Total lipid content and fatty acid composition of sow plasma at 92 d of gestation and placenta at parturition  
(Mean values with their standard errors for five sows)

	Plasma				Placenta		
	LAR	LSO	SEM		LAR	LSO	SEM
Total lipid content (g/100 g of tissue)	ND	ND		0.6	0.5	0.1	
Fatty acid composition (g/100 g of total fatty acids)							
SFA	26.0	20.3	0.6	*	32.8	33.7	0.9
MUFA	30.8	22.3	0.7	*	54.2	50.5	0.9
PUFA	43.2	57.4	0.9	*	13.0	15.8	0.5
<i>n</i> -6 PUFA	40.1	45.3	1.0	*	10.2	11.3	0.2
18:2 <i>n</i> -6	34.5	41.9	1.3	*	5.6	6.8	0.3
20:4 <i>n</i> -6	5.6	3.4	0.6	*	4.5	4.5	0.2
<i>n</i> -3 PUFA	2.9	12.0	0.5	*	1.6	3.3	0.2
18:3 <i>n</i> -3	1.4	9.3	0.7	*	0.1	0.4	0.1
20:5 <i>n</i> -3	0.3	1.2	0.2	*	0.4	0.7	0.2
22:5 <i>n</i> -3	0.8	1.0	0.1		0.4	0.8	0.1
22:6 <i>n</i> -3	0.3	0.3	0.1		0.8	1.4	0.2
<i>n</i> -6 PUFA/ <i>n</i> -3 PUFA	13.7	3.8	0.5	*	6.8	3.7	0.4
18:2 <i>n</i> -6/18:3 <i>n</i> -3	24.6	4.6	2.0	*	44.0	16.4	4.5

LAR, lard-based diet; LSO, linseed oil-based diet; ND, not determined.

\* Mean values were significantly different between the two groups ( $P < 0.05$ ).

dramatically lower in the LSO group than in the LAR group ( $P < 0.001$ ; Table 2).

Within the *n*-3 PUFA family, the LSO diet increased the proportions of 18:3*n*-3 ( $P < 0.001$ ) and 20:5*n*-3 ( $P = 0.002$ ), but it had no effect on 22:5*n*-3 and 22:6*n*-3 proportions (Table 2). Within the *n*-6 PUFA family, the proportion of 18:2*n*-6 was greater ( $P = 0.006$ ), while the proportion of 20:4*n*-6 was lesser ( $P = 0.03$ ) in the LSO group than in the LAR group (Table 2).

#### Placenta

The proportion of *n*-3 PUFA was greater in the placenta from LSO litters ( $P = 0.002$ ), with no difference for *n*-6 PUFA (Table 2). As a result, the 18:2*n*-6 to 18:3*n*-3 ratio was lower in the LSO group ( $P = 0.006$ ; Table 2).

Within the *n*-3 PUFA family, the dietary supplementation with LSO increased all the proportions of *n*-3 PUFA (18:3*n*-3,  $P = 0.004$ ; 20:5*n*-3,  $P = 0.03$ ; 22:5*n*-3,  $P = 0.001$  and 22:6*n*-3,  $P = 0.01$ ) in the placenta (Table 2). The proportion of 18:2*n*-6 was greater in the placenta of the LSO sows ( $P = 0.02$ ), but there was no treatment effect on the proportion of 20:4*n*-6 (Table 2).

#### Piglet tissues at birth

In the plasma, the proportions of *n*-3 and *n*-6 PUFA were greater in the LSO group ( $P = 0.04$  and  $0.05$ , respectively; Table 3). There was no difference in the 18:2*n*-6 to 18:3*n*-3 ratio between the two groups (Table 3). Proportions of 20:5*n*-3 and 22:6*n*-3 were greater in the plasma of the LSO piglets than in that of the LAR piglets ( $P = 0.02$  and  $0.001$ , respectively; Table 3). Within the *n*-6 PUFA family, the LSO diet increased the proportion of 20:4*n*-6 ( $P = 0.02$ ), but it did not change that of 18:2*n*-6 in piglet plasma (Table 3).

Proportions of *n*-3 and *n*-6 PUFA and the 18:2*n*-6 to 18:3*n*-3 ratio were similar in the carcasses of piglets from both the groups (Table 4). Within the *n*-3 PUFA family, the LSO diet increased the proportion of 20:5*n*-3 ( $P < 0.001$ ), 22:5*n*-3 ( $P < 0.001$ ) and 22:6*n*-3 ( $P = 0.001$ ) in the carcass (Table 4). The proportions of *n*-6 PUFA did not differ between the two groups (Table 4).

In the brain, the proportions of *n*-6 PUFA were similar between the LSO and LAR piglets at birth (Table 5). The proportion of *n*-3 PUFA was greater in the LSO group ( $P = 0.05$ ; Table 5). Within the *n*-3 PUFA family, there were no differences between the two groups for 18:3*n*-3, 20:5*n*-3 and 22:5*n*-3. However, the proportion of 22:6*n*-3 tended to be greater in the LSO group than in the LAR group ( $P = 0.07$ ; Table 5).

#### Sow colostrum and milk

The colostrum/milk of LSO sows contained more *n*-3 PUFA than that of LAR sows (diet effect  $P < 0.001$ ; Table 6, data online). Dietary supplementation of the sow diet with LSO increased the proportions of 18:3*n*-3 (diet effect  $P < 0.001$ ), 20:5*n*-3 (diet effect  $P < 0.001$ ) and 22:5*n*-3 (diet effect  $P < 0.001$ ) in the colostrum/milk, but it had no influence on the proportion of 22:6*n*-3 (Table 6, data online).

#### Piglet plasma after birth

The proportion of *n*-6 PUFA increased with time (age effect  $P < 0.001$ ), but it was not different between the two groups (Table 3). While the proportion of *n*-3 PUFA did not vary with time in the LAR group, it increased in the plasma of the LSO piglets from day 7 and was greater in the LSO piglets than in the LAR piglets from day 3 (diet  $\times$  age interaction  $P < 0.001$ ; Table 3). While the 18:2*n*-6 to 18:3*n*-3 ratio increased from day 0 in the plasma of the LAR piglets, it decreased from day 3 in the plasma of the LSO piglets (diet  $\times$  age interaction  $P < 0.001$ ; Table 3).

**Table 3.** Fatty acid composition of piglet plasma at 0, 3, 7, 21 and 32 d of age in the linseed oil-based diet (LSO) and lard-based diet (LAR) groups (Mean values with their standard errors for four piglets)

	LAR (g/100 g of total fatty acids)					LSO (g/100 g of total fatty acids)					P			
	d0	d3	d7	d21	d32	d0	d3	d7	d21	d32	SEM	Diet	Age	Diet × age
SFA	37.2	28.7	27.4	30.2	29.3	40.2	37.5	29.2	30.5	24.0	3.9	NS	0.03†	NS
MUFA	44.0	35.7	40.1	33.6	31.7	33.3	22.5	31.3	18.6	27.5	6.6	0.03	NS	NS
PUFA	18.8	35.6	32.5	36.2	39.0	26.5	40.0	39.5	50.9	48.5	3.5	<0.001	<0.001†	NS
n-6 PUFA	13.3	22.0	29.5	33.5	35.1	18.2	31.8	32.0	33.0	31.6	8.5	NS	<0.001†	NS
18:2n-6	7.7	26.4	25.3	28.7	26.3	9.0	28.1	29.0	30.0	28.7	2.2	NS	<0.001†	NS
20:4n-6	5.6 <sup>a</sup>	5.6 <sup>a</sup>	4.2 <sup>a</sup>	4.8 <sup>a</sup>	8.8 <sup>b</sup>	9.2 <sup>a</sup>	3.7 <sup>a</sup>	3.0 <sup>a</sup>	3.0 <sup>a</sup>	2.9 <sup>a</sup>	0.1	NS	0.003	0.002
n-3 PUFA	4.5	3.2	2.6	2.3	3.7	7.5 <sup>a</sup>	7.6 <sup>a</sup>	7.1 <sup>a</sup>	17.7 <sup>a</sup>	17.0 <sup>a</sup>	8.2	<0.001	<0.001	<0.001
18:3n-3	0.6	1.0	1.0	1.2	1.2	0.7 <sup>a</sup>	4.7 <sup>a</sup>	5.0 <sup>a</sup>	13.2 <sup>a</sup>	10.4 <sup>a</sup>	0.6	<0.001	<0.001	<0.001
20:5n-3	0.5	0.4	0.2	0.3	0.4	1.2 <sup>a</sup>	0.7 <sup>a</sup>	0.8 <sup>a</sup>	2.3 <sup>a</sup>	3.6 <sup>a</sup>	0.2	<0.001	<0.001	<0.001
22:5n-3	0.5	0.6	0.7	0.4	0.8	0.8 <sup>a</sup>	0.8 <sup>a</sup>	0.5 <sup>a</sup>	1.1 <sup>a</sup>	1.8 <sup>a</sup>	0.1	<0.001	<0.001	0.003
22:6n-3	2.4 <sup>a</sup>	0.8 <sup>b</sup>	0.5 <sup>b</sup>	0.2 <sup>c</sup>	1.2 <sup>b</sup>	4.0 <sup>a</sup>	0.9 <sup>a</sup>	0.6 <sup>a</sup>	0.9 <sup>a</sup>	1.1 <sup>a</sup>	0.2	0.01	<0.001	0.001
n-6 PUFA/n-3 PUFA	2.7 <sup>a</sup>	10.1 <sup>b</sup>	10.8 <sup>b</sup>	14.3 <sup>c</sup>	10.0 <sup>b</sup>	2.6 <sup>a</sup>	5.2 <sup>a</sup>	5.5 <sup>a</sup>	2.7 <sup>a</sup>	3.1 <sup>a</sup>	1.7	<0.001	<0.001	<0.001
18:2n-6/18:3n-3	11.8 <sup>a</sup>	26.5 <sup>b</sup>	23.6 <sup>b</sup>	24.1 <sup>b</sup>	22.1 <sup>b</sup>	13.6 <sup>a</sup>	9.0 <sup>a</sup>	5.9 <sup>a</sup>	2.4 <sup>a</sup>	2.9 <sup>a</sup>	9.3	<0.001	NS	<0.001

d, Day.

<sup>a,b,c</sup> Mean values for the LAR group with unlike superscript letters (for LSO group) were significantly different within a dietary group ( $P < 0.05$ ).

<sup>x,y,z</sup> Mean values for the LSO group with unlike superscript letters were significantly different within a dietary group ( $P < 0.05$ ).

\* Mean values were significantly different between the two groups at the same age ( $P < 0.05$ ).

† Significant difference between day 0 and days 7–32.

The plasma of the LSO piglets had greater proportions of 18:3n-3, 20:5n-3 and 22:5n-3 than that of the LAR piglets (diet effect  $P < 0.001$  for 18:3n-3, 20:5n-3 and 22:5n-3), and these proportions increased with age in the LSO group (diet × age interaction  $P < 0.001$  for 18:3n-3 and 20:5n-3 and  $P = 0.003$  for 22:5n-3; Table 3). The proportion of 22:6n-3 was greater over the whole period in the plasma of the LSO piglets than in that of the LAR piglets (diet effect  $P < 0.001$ ), and decreased between days 0 and 7 in both the groups although more sharply in the LSO group (diet × age interaction  $P = 0.001$ ; Table 3). Within the n-6 PUFA family, the proportion of 18:2n-6 in piglet plasma was similar between the two groups, and this proportion increased between days 0 and 3 in both the groups (age effect  $P < 0.001$ ; Table 3). The proportion of 20:4n-6 differed between the two groups on days 0 and 32. It decreased with age in the LSO piglets, whereas it increased between days 21 and 32 in the LAR piglets (diet × age interaction  $P = 0.002$ ; Table 3).

*Piglet carcasses after birth*

The proportion of n-6 PUFA in the carcasses increased with time in both the groups (age effect  $P < 0.001$ ), and it was greater over the whole period in the carcass of the LSO piglets than in that of the LAR piglets (diet effect  $P < 0.001$ ; Table 4). The proportion of n-3 PUFA in the carcasses increased from day 3 in the LSO group, but it was not affected by time in the LAR group (diet × age interaction  $P = 0.004$ ; Table 4). As a result, the 18:2n-6 to 18:3n-3 ratio decreased between days 0 and 3 in both the groups (age effect  $P < 0.001$ ), but it was lower in the carcass of the LSO piglets (diet effect  $P < 0.001$ ; Table 4).

The proportions of 18:3n-3, 20:5n-3 and 22:5n-3 in piglet carcass were greater in the LSO group than in the LAR group (diet effect  $P < 0.001$ ; Table 4). The proportion of 18:3n-3 increased between birth and day 3 in the LSO group, while it was not influenced by age in the LAR group (diet × age interaction  $P < 0.001$  for the LSO group; Table 4). Moreover, the proportions of 20:5n-3 and 22:5n-3 decreased between birth and day 7 in the LAR group, and between birth and day 3 and then between days 7 and 21 in the LSO group (age effect  $P < 0.001$ ; Table 4). The carcass of LSO piglets contained a greater proportion of 22:6n-3 over the whole period (diet effect  $P = 0.01$ ; Table 4). This proportion decreased with time in both the groups (age effect  $P < 0.001$ ), but the decrease tended to be sharper due to greater proportions of 22:6n-3 at birth in the LSO group (diet × age interaction  $P = 0.08$ ; Table 4). Within the n-6 PUFA family, the proportion of 18:2n-6 increased between days 0 and 3 in both the groups (age effect  $P < 0.001$ ), and it was greater in the carcass of the LSO piglets on days 7, 21 and 32 than in the carcass of the LAR piglets (diet effect  $P < 0.001$ ; Table 4). There was no difference between the two groups for the proportion of 20:4n-6 (Table 4).

*Piglet brain after birth*

No difference was observed in the proportion of n-6 PUFA between the two groups. It increased from days 0 to 7 and decreased thereafter in both the groups (age effect  $P < 0.001$ ;

**Table 4.** Total lipid content and fatty acid composition of piglet carcass at 0, 3, 7, 21 and 32 d of age in the linseed oil-based diet (LSO) and lard-based diet (LAR) groups (Mean values with their standard errors for four piglets)

	LAR					LSO					SEM	P value		
	d0	d3	d7	d21	d32	d0	d3	d7	d21	d32		Diet	Age	Diet × age
Total lipid content (g/100 g of tissue)	1.1	3.3	9.5	14.4	12.6	1.5	4.3	6.3	13.1	12.0	0.8	NS	< 0.001†	NS
Fatty acid composition (g/100 g of total fatty acids)														
SFA	49.0	37.0	34.3	35.6	36.8	46.7	35.0	33.7	35.7	36.8	1.1	NS	< 0.001‡	NS
MUFA	40.9 <sup>a</sup>	43.3 <sup>a</sup>	49.5 <sup>b</sup>	50.8 <sup>b</sup>	49.2 <sup>b</sup>	41.7	39.0*	39.6*	40.0*	39.7*	1.1	< 0.001	0.002	< 0.001
PUFA	10.1 <sup>a</sup>	19.7 <sup>b</sup>	16.2	13.6 <sup>a</sup>	14.0 <sup>a</sup>	11.6 <sup>x</sup>	26.0 <sup>y*</sup>	26.7 <sup>y*</sup>	24.3 <sup>y*</sup>	23.7 <sup>y*</sup>	1.3	< 0.001	< 0.001	0.01
<i>n</i> -6 PUFA	7.2	17.1	14.1	12.0	12.3	7.5	18.4	17.4	14.7	14.7	0.7	< 0.001	< 0.001‡	NS
18:2 <i>n</i> -6	7.0	17.0	14.0	11.9	12.2	7.4	18.3	17.3	14.7	14.6	0.7	< 0.001	< 0.001‡	NS
20:4 <i>n</i> -6	0.2	0.1	0.1	0.0	0.1	0.1	0.1	0.1	0.0	0.0	0.1	NS	NS	NS
<i>n</i> -3 PUFA	2.4	2.0	1.4	1.0	1.1	3.7 <sup>x</sup>	7.0 <sup>y*</sup>	8.3 <sup>y*</sup>	9.0 <sup>y*</sup>	8.3 <sup>y*</sup>	0.8	< 0.001	NS	< 0.001
18:3 <i>n</i> -3	0.3	1.0	1.0	0.8	0.8	0.4 <sup>x</sup>	5.7 <sup>y*</sup>	7.2 <sup>z*</sup>	8.3 <sup>z*</sup>	7.7 <sup>z*</sup>	0.7	< 0.001	< 0.001	< 0.001
20:5 <i>n</i> -3	0.2 <sup>a</sup>	0.1 <sup>b</sup>	0.0 <sup>c</sup>	0.1 <sup>c</sup>	0.1 <sup>c</sup>	0.5 <sup>x*</sup>	0.3 <sup>y*</sup>	0.2 <sup>y*</sup>	0.2 <sup>z*</sup>	0.2 <sup>z*</sup>	0.1	< 0.001	< 0.001	< 0.001
22:5 <i>n</i> -3	0.6 <sup>a</sup>	0.5 <sup>b</sup>	0.3 <sup>c</sup>	0.2 <sup>c</sup>	0.2 <sup>c</sup>	1.0 <sup>x*</sup>	0.6 <sup>y</sup>	0.6 <sup>y*</sup>	0.4 <sup>z*</sup>	0.4 <sup>z</sup>	0.1	< 0.001	< 0.001	0.04
22:6 <i>n</i> -3	1.3	0.3	0.1	0.1	0.1	1.8*	0.4	0.2	0.1	0.1	0.1	0.01	< 0.001	0.08
<i>n</i> -6 PUFA/ <i>n</i> -3 PUFA	3.5 <sup>a</sup>	9.5 <sup>b</sup>	10.4 <sup>b</sup>	11.0 <sup>b</sup>	7.4 <sup>b</sup>	2.0	2.6*	2.2*	1.7*	1.8*	1.1	< 0.001	0.01	0.02
18:2 <i>n</i> -6/18:3 <i>n</i> -3	19.0	16.1	9.6	14.4	15.2	17.5	3.3	2.5	1.8	1.9	1.4	< 0.001	< 0.001	0.07

d, Day.

<sup>a,b,c</sup> Mean values for the LAR group with unlike superscript letters (for LSO group) were significantly different within a dietary group ( $P < 0.05$ ).

<sup>x,y,z</sup> Mean values for the LSO group with unlike superscript letters were significantly different within a dietary group ( $P < 0.05$ ).

\* Mean values were significantly different between the two groups at the same age ( $P < 0.05$ ).

† Significant difference between days 0 and 3 and between day 7 and days 21–32.

‡ Significant difference between day 0 and days 3–32.

**Table 5.** Total lipid content and fatty acid composition of piglet brain at 0, 3, 7, 21 and 32 d of age in the linseed oil-based diet (LSO) and lard-based diet (LAR) groups (Mean values with their standard errors for four piglets)

	LAR					LSO					SEM	P		
	d0	d3	d7	d21	d32	d0	d3	d7	d21	d32		Diet	Age	Diet × age
Total lipid content (g/100 g of tissue)	5.4	5.2	6.2	6.8	7.6	5.0	5.5	6.3	7.3	7.7	1.3	NS	<0.001†	NS
Fatty acid composition (g/100 g of total fatty acids)														
SFA	48.3	48.0	47.5	46.9	44.7	47.7	47.2	46.4	45.0	45.0	2.2	NS	<0.001‡	NS
MUFA	26.0	25.2	26.3	28.4	31.4	25.6	25.5	25.3	28.2	28.5	2.9	NS	<0.001§	NS
PUFA	25.7	26.8	26.2	24.7	23.9	26.7	27.3	28.3	26.8	26.5	1.8	<0.001	<0.001	NS
n-6 PUFA	12.7	13.9	14.0	13.4	12.2	12.8	13.6	14.2	12.8	12.5	1	NS	<0.001¶	NS
18:2n-6	0.6	1.6	1.7	2.0	1.6	0.5	1.7	2.0	2.4	2.2	0.7	0.02	<0.001**	NS
20:4n-6	12.1	12.2	12.3	11.5	10.7	12.3	11.9	12.1	10.5	10.4	1.1	NS	<0.001†	NS
n-3 PUFA	12.6 <sup>a</sup>	12.7 <sup>a</sup>	11.7 <sup>b</sup>	10.8 <sup>b</sup>	11.1 <sup>b</sup>	13.7	13.6	13.7*	13.6*	13.5*	1.4	<0.001	0.009	0.02
18:3n-3	0.7 <sup>a</sup>	0.8 <sup>a</sup>	0.9 <sup>a</sup>	1.0	1.5 <sup>b</sup>	0.8	0.8	0.8	1.1	0.9*	0.3	0.05	<0.001	0.005
20:5n-3	0.5	0.5	0.4	0.4	0.4	0.5	0.5	0.4	0.4	0.4	0.1	NS	NS	NS
22:5n-3	0.4	0.5	0.5	0.5	0.5	0.5 <sup>x</sup>	0.7 <sup>x</sup>	1.1 <sup>y*</sup>	1.3 <sup>z*</sup>	1.6 <sup>z*</sup>	0.4	<0.001	<0.001	<0.001
22:6n-3	11.0	11.0	10.0	8.7	8.8	11.9	11.6	11.3	10.9	10.7	1.3	<0.001	<0.001†	NS
n-6 PUFA/n-3 PUFA	1.0 <sup>a</sup>	1.1 <sup>a</sup>	1.2 <sup>b</sup>	1.2 <sup>b</sup>	1.1 <sup>a</sup>	0.9 <sup>x</sup>	1.0 <sup>y</sup>	1.0 <sup>y*</sup>	0.9 <sup>x*</sup>	0.9 <sup>x*</sup>	0.1	<0.001	0.003	0.02
18:2n-6/18:3n-3	0.8	2.0	1.9	1.9	1.1	0.7	2.1	2.7	2.3	3.2	1.3	0.03	0.01	0.08

d, Day.

<sup>a,b,c</sup> Mean values for the LAR group with unlike superscript letters (for LSO group) were significantly different within a dietary group ( $P < 0.05$ ).

<sup>x,y,z</sup> Mean values for the LSO group with unlike superscript letters were significantly different within a dietary group ( $P < 0.05$ ).

\* Mean values were significantly different between the two groups at the same age ( $P < 0.05$ ).

† Significant difference between days 0 and 3 and day 7 and days 21–32.

‡ Significant difference between days 0 and 3 and between days 7–21 and day 32.

§ Significant difference between days 0–7 and days 21–32.

|| Significant difference between days 0–7 and day 21 and day 32.

¶ Significant difference between day 0 and days 3–21 and day 32.

\*\* Significant difference between day 0 and days 3–32.

**Table 6.** Total lipid content and fatty acid composition of sow colostrum (day 0) and milk at 7, 14, 21 and 28 d after parturition in the linseed oil (LSO) and lard (LAR) groups (Mean values with their standard errors (*n* 4))

	LAR					LSO					SEM	<i>P</i>		
	d0	d7	d14	d21	d28	d0	d7	d14	d21	d28		Diet	Day	Diet × day
Total lipid content (g/100 g of tissue)	5.0	9.0	7.5	7.7	7.7	5.8	8.5	8.6	9.1	7.9	1.7	NS	0.02†	NS
Fatty acid composition (g/100 g of total fatty acids)														
SFA	31.5	37.3	50.0	40.0	42.5	27.5	35.9	40.6	34.3	39.2	8.6	NS	NS	NS
MUFA	39.0	45.2	36.0	43.0	42.3	28.3	39.9	34.0	39.2	33.3	7.3	0.01	NS	NS
PUFA	29.5	17.5	14.0	17.0	15.2	44.2	24.2	25.4	26.5	27.5	8.7	<0.001	<0.001†	NS
<i>n</i> -6 PUFA	25.7	15.1	12.1	14.8	13.2	31.5	15.1	14.3	16.2	14.9	5.6	0.04	<0.001†	NS
18:2 <i>n</i> -6	24.3	14.4	11.7	14.2	12.7	30.8	14.7	14.1	15.9	14.7	5.5	0.01	<0.001†	NS
20:4 <i>n</i> -6	1.4	0.7	0.4	0.6	0.6	0.7	0.4	0.2	0.3	0.2	0.3	<0.001	<0.001†	NS
<i>n</i> -3 PUFA	3.3	2.0	1.5	1.8	1.6	12.4	8.9	10.8	9.9	12.3	4.9	<0.001	NS	NS
18:3 <i>n</i> -3	2.1	1.3	1.0	1.3	1.2	10.7	8.0	10.1	9.2	11.6	4.7	<0.001	NS	NS
20:5 <i>n</i> -3	0.2	0.1	0.1	0.1	0.1	0.4	0.3	0.3	0.2	0.3	0.1	<0.001	NS	NS
22:5 <i>n</i> -3	0.5 <sup>a</sup>	0.3 <sup>b</sup>	0.2 <sup>b</sup>	0.2 <sup>b</sup>	0.2 <sup>b</sup>	0.8 <sup>x*</sup>	0.3 <sup>y</sup>	0.2 <sup>y</sup>	0.3 <sup>y</sup>	0.2 <sup>y</sup>	0.2	<0.001	<0.001	0.01
22:6 <i>n</i> -3	0.1	0.1	0.1	0.1	0.1	0.2	0.1	0.1	0.1	0.1	0.1	NS	0.02†	NS
<i>n</i> -6 PUFA/ <i>n</i> -3 PUFA	7.9	9.6	9.6	8.2	8.0	2.6	1.7	1.4	1.7	1.2	4.0	<0.001	NS	NS
18:2 <i>n</i> -6/18:3 <i>n</i> -3	11.5	11.0	11.0	11.2	10.7	2.9	1.8	1.4	1.7	1.3	6.2	<0.001	NS	NS

d, Day.

<sup>a,b,c</sup> Mean values for the LAR group with unlike superscript letters (for LSO group) were significantly different within a dietary group (*P*<0.05).

<sup>x,y,z</sup> Mean values for the LSO group with unlike superscript letters were significantly different within a dietary group (*P*<0.05).

\* Mean values were significantly different between the two groups for the same day (*P*<0.05).

† Significantly different between day 0 and days 7–28.



Table 5). There was a greater proportion of *n*-3 PUFA after day 7 in the LSO group than in the LAR group, with a decrease occurring from day 7 in the brain of the LAR piglets only (diet  $\times$  age interaction  $P=0.02$ ; Table 5). The 18:2*n*-6 to 18:3*n*-3 ratio was greater in the brain of the LSO piglets than in that of the LAR piglets over the whole period (diet effect  $P=0.03$ ; Table 5).

The proportion of 18:3*n*-3 increased between days 21 and 32 in the brain of the LAR piglets but not in that of the LSO piglets (diet  $\times$  age interaction  $P=0.005$ ; Table 5). There was no treatment effect on the proportion of 20:5*n*-3 in the brain (Table 5). The proportion of 22:5*n*-3 increased as of day 7 in the LSO group, and was greater in this group than in the LAR group from that age onwards (diet  $\times$  age interaction  $P<0.001$ ; Table 5). The proportion of 22:6*n*-3 was greater in the brain of the LSO piglets than in that of the LAR piglets (diet effect  $P<0.001$ ; Table 5). Although the diet  $\times$  age interaction was NS ( $P=0.14$ ), 22:6*n*-3 proportion decreased in the LAR group from day 3, while it remained constant over the whole period in the LSO group (age effect  $P<0.001$ ; Table 5). The proportion of 18:2*n*-6 increased between days 0 and 3 in both the groups (age effect  $P<0.001$ ), and it was greater overall in the LSO group (diet effect  $P=0.02$ ; Table 5). The proportion of 20:4*n*-6 decreased between days 7 and 21 in both the groups (age effect  $P<0.001$ ; Table 5).

## Discussion

The high contents of *n*-3 LC-PUFA in the placenta suggest that sows are capable of synthesising *n*-3 LC-PUFA from dietary 18:3*n*-3 during gestation, although the site of synthesis is not known. The capacity of adults to convert 18:3*n*-3 to 20:5*n*-3 or 22:6*n*-3 is very limited<sup>(8,9)</sup>, but this synthesis seems to increase during gestation<sup>(10,11)</sup>. Indeed, Brazle *et al.*<sup>(21)</sup> reported an increase in *n*-3 LC-PUFA in the placenta of sows fed 18:3*n*-3-rich diet during gestation, suggesting the synthesis of *n*-3 LC-PUFA from dietary 18:3*n*-3 also. Moreover, Otto *et al.*<sup>(10)</sup> showed that the percentage of 22:6*n*-3 in plasma phospholipids from women increased in early pregnancy (2.98% at 4 weeks of pregnancy *v.* 3.93% at 10 weeks of pregnancy) without modification in the dietary intake. In the present study, LSO in the maternal diet increased LC-PUFA accumulation in the placenta. Placental transfer of certain fatty acids (octanoic acid, palmitic acid and linoleic acid) has been reported to be quite limited in sows<sup>(22,23)</sup> whose placenta is epitheliochorial<sup>(22)</sup>. However, Rooke *et al.*<sup>(14,24)</sup> observed that supplementation of the sow diet with *n*-3 LC-PUFA during gestation increased the percentages of 20:5*n*-3 and 22:6*n*-3 in piglet cord plasma at birth. Although specific measurements of the placenta were not performed, it can be suggested that *n*-3 LC-PUFA cross the sow placenta during gestation, similar to what is observed in human subjects, where *n*-3 LC-PUFA are transferred from the mother to the foetus by a specific transporter called plasma membrane fatty acid-binding protein<sup>(25,26)</sup>. In addition, a recent study has demonstrated that supplementing the sow diet with 18:3*n*-3 from vegetable sources or with *n*-3 LC-PUFA from marine sources during early gestation affects the fatty acid composition of the placenta and the foetus at 40 d of gestation. This is consistent with the idea

of placental transport of *n*-3 LC-PUFA between the sow and foetuses<sup>(21)</sup>. Whether this increase in LC-PUFA in placental tissue in the present study is related to an upregulation of fatty acid transporters in this tissue needs further investigations.

The fatty acid composition of piglet tissues before suckling was influenced by the maternal diet. At birth, the carcasses of piglets from the LSO sows had greater proportions of *n*-3 LC-PUFA than the carcasses of piglets from the LAR sows, whereas in the plasma, the proportions of 20:5*n*-3 and 22:6*n*-3, but not of 22:5*n*-3, were greater with the LSO diet. Although the elongation and desaturation enzymes for LC-PUFA synthesis are present in the fetal liver of piglets<sup>(27,28)</sup>, their activity appears to be low before birth than in the postnatal period<sup>(27,28)</sup>, suggesting that the *n*-3 LC-PUFA accumulated by the foetus *in utero* are derived predominantly from placental transfer. The dietary maternal 18:3*n*-3 content seems to be important for the deposition of *n*-3 LC-PUFA in fetal tissues. Indeed, Rooke *et al.*<sup>(14)</sup> demonstrated that inclusion of 26 g/d of LSO in the diet of sows during gestation increased the proportion of 20:5*n*-3 (1.4 times more than control) and 22:6*n*-3 (1.2 times more than control) in the plasma of piglets at birth compared with piglets born to sows without dietary LSO supplementation. In the present study, sows were fed 42 g/d of LSO, which increased the proportion of 20:5*n*-3 by 2.4 times and of 22:6*n*-3 by 1.7 times in the plasma of the LSO piglets compared with the LAR piglets at birth. However, we have recently observed that supplementation of 105 g/d of LSO in the diet of sows during gestation increased the proportion of 20:5*n*-3 (5.2 times more than control) and 22:6*n*-3 (1.35 times more than control) in erythrocytes of piglets at birth compared with piglets from sows receiving no LSO (Boudry G, personal communication). In the last study, the lower synthesis of 22:6*n*-3 compared with the present study might be due to the enzymatic competition between *n*-3 PUFA for the  $\Delta$ -6 desaturase. Indeed, this enzyme is involved in two steps during the *n*-3 LC-PUFA synthesis pathway, namely in the first step to convert 18:3*n*-3 into 20:5*n*-3, and in the second step to convert 22:5*n*-3 into 22:6*n*-3. An excessive level of 18:3*n*-3 in the liver could therefore inhibit the synthesis of 22:6*n*-3 from 22:5*n*-3, suggesting that the maternal diet has to be supplemented with an optimal content of 18:3*n*-3 to allow the synthesis of all *n*-3 LC-PUFA which will be transferred to the foetus. In the piglet brain at birth, the proportion of 22:6*n*-3 tended to be greater with the LSO diet. This high level of 22:6*n*-3 in the LAR piglet brain despite the relatively low level of 18:3*n*-3 in the sow diet during gestation is consistent with the fact that during foetal development, the brain is a preferential tissue to accumulate 22:6*n*-3. Therefore, increasing the dietary level of *n*-3 PUFA has only little impact on brain 22:6*n*-3 proportion.

Our results on milk and fatty acid composition are consistent with those reported by Farmer & Petit<sup>(13)</sup> who showed that 18:3*n*-3-rich diet during gestation and lactation increased the percentages of 20:5*n*-3 and 22:5*n*-3 in sow milk on days 3 and 20 of lactation with no difference for 22:6*n*-3. In the present study, the milk of the LSO sows had greater proportions of 18:3*n*-3 compared with the other *n*-3 LC-PUFA. The relative contents of *n*-3 PUFA in milk reflect the nutritional status of mothers during gestation and lactation.

Similar results were reported for several species, including human subjects<sup>(29,30)</sup>, rats<sup>(31)</sup> and sows<sup>(1,12,32)</sup>. Moreover, Rodriguez-Cruz *et al.*<sup>(33)</sup> demonstrated that the  $\Delta$ -6 desaturase was present in lactating mammary gland of rats, which could explain, if similar in sows, why sows in the present study had milk that was richer in LC-PUFA.

The maternal LSO diet influenced the fatty acid composition of piglet tissues during the postnatal life. Piglets suckling the LSO sows consumed high levels of 18:3n-3 through the milk. This resulted in the increased plasma and carcass proportions of 18:3n-3, 20:5n-3 and 22:5n-3, with the proportion of 18:3n-3 in the plasma and carcasses of LSO piglets increasing with time. Assuming that the carcass weight to live weight of piglets and the fatty acid content to total lipid content ratio are constant during the postnatal period, we estimated that the total amount of fatty acid deposit in the carcasses of piglets increases from birth to day 32 (0.1–2.3 g for 20:5n-3; 0.3–4.1 g for 22:5n-3 and 0.5–1.4 g for 22:6n-3). This suggests that piglets efficiently absorbed 18:3n-3 derived from the maternal milk, and were capable of transforming it to n-3 LC-PUFA during the postnatal period.

The evolution of fatty acid composition in piglet brain was different from that of fatty acid composition in piglet plasma and carcasses. Although not different during the first week after birth, the proportion of 22:5n-3 and 22:6n-3 were greater from day 7 in the brain of the LSO piglets than in that of the LAR piglets, with no difference for 18:3n-3 and 20:5n-3. The 22:6n-3 could, in part, be transported via the circulation from the carcass to the brain. Furthermore, the neonatal piglets might be capable of synthesising 22:6n-3 from 22:5n-3 in the brain during the postnatal life. Indeed, Clandinin *et al.*<sup>(28)</sup> demonstrated that the enzymatic activities that are necessary to synthesise LC-PUFA in the brain of piglets double between the foetal and the postnatal period.

In conclusion, LSO in the maternal diet during gestation and lactation increases LC-PUFA content in the placenta and 22:6n-3 deposition in the carcasses and brain of piglets at birth. Furthermore, this diet maintains the proportion of 22:6n-3 in the brain of the LSO piglets compared with the LAR piglets during the postnatal period. However, although LSO could be an interesting alternative source of n-3 PUFA for women, caution should be exercised while using LSO in diets. This oil is easily oxidised and, consequently, must be supplemented with antioxidants and/or an adequate protein level in the diet<sup>(34)</sup>. Moreover, the present work raises questions on the optimal dose of LSO, which seems to be a key component to modulate sow and piglet fatty acid metabolism and tissue composition. Further work is also needed to understand the dietary regulation of fatty acid transporters in the placenta, and to explore the capacity of neonatal piglets to transform maternal 18:3n-3 in n-3 LC-PUFA, especially in the brain.

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