Livestock Science 123 (2009) 88–96

Contents lists available at ScienceDirect

Livestock Science

journal homepage: www.elsevier.com/locate/livsci

Supplementation of sow diets with oil during gestation: Sow body condition, milk yield and milk composition ☆

J. Laws [a], E. Amusquivar [b], A. Laws [a], E. Herrera [b], I.J. Lean [a], P.F. Dodds [a], L. Clarke [a,*]

[a] Department of Agricultural Sciences, Imperial College, London, TN25 5AH UK
[b] Facultad de Farmacia, Universidad San Pablo-CEU, Boadilla del Monte/Madrid, Spain

ARTICLE INFO

Article history:
Received 16 October 2006
Received in revised form 4 September 2008
Accepted 21 October 2008

Keywords:
Sow
Oil
Milk yield
Milk composition

ABSTRACT

This study aimed to determine the consequences of altering the fatty acid profile of sow diets during gestation. 88 multiparous sows were used to evaluate the effects of fat supplementation during either the first (G1) or second (G2) half of gestation. Sows were allocated to either 3 kg/day of commercial sow pellets (control; C), or an experimental diet consisting of C diet with 10% extra energy in the form of excess pellets (E), palm oil (P), olive oil (O), sunflower oil (S) or fish oil (F). Experimental diets were fed during either the first 60 days of gestation, or from day 61 of gestation until term. All sows were given 3 kg/day of sow pellets as for the C group, during the non-supplemented period. The provision of extra energy resulted in increased fat deposition over the period of supplementation. E G1 and S G1 groups continued to deposit fat at elevated rates during G2. Fat accretion occurred at a much lesser extent in E G2 and P G2 compared to OG2, S G2 and F G2 animals. E G1, S G1 and F G2 mothers mobilized more fat over the lactation period compared to all other groups, except F G1 who mobilized a greater proportion of their fat reserves during lactation than they had accumulated during gestation, resulting in a net loss in back fat depth over the whole production cycle. The timing of supplementation influenced milk yield, and the percentages of fat and protein in the milk but not milk lactose. Milk fatty acid profile reflected the fatty acid profile of the maternal diet during gestation; this effect was most pronounced in the lacteal secretions of sows receiving the F diet. The concentration of immunoglobulins was increased in the colostrum of sows that had received the P and S diets during G1. In conclusion the type and timing of maternal dietary supplementation influences maternal fat deposition and mobilization as well as the fatty acid profile and immunoglobulin concentration of the milk.

1. Introduction

Sow nutrition during gestation focuses on preparation for parturition and lactation; the majority of the research aimed at improving sow performance has targeted nutrition during late gestation or lactation, or both. Coffey et al. (1982) reported that fat supplementation of sows during late gestation resulted in increased milk yields. Other studies suggest that an increased plane of nutrition during late gestation may have a negative effect on sow performance during lactation. In gilts increased energy intake during late gestation inhibited the development of mammary secretory tissue (Weldon et al., 1991), and increased the loss of body condition during lactation as a result of reduced feed intake (Weldon et al., 1994). It is widely accepted that fetal energy demands are greatly increased during late gestation, and that catabolism of maternal reserves occurs if dietary energy supply is insufficient to meet requirements at this time (Close et al., 1985; Boyd et al., 2002); increasing the energy intake of sows during the anabolic phase of gestation (early–mid) is likely to increase the amount of fat available for

☆ The authors acknowledge the financial support of the Commission of the European Communities (QLK1-2001-00138). PERILIP. This work does not necessarily reflect the views of the Commission and in no way anticipates its future policy in this area. The authors thank United Fish Industries for providing the salmon oil and the staff at The Pig Research and Development Unit for their care of animals throughout the study. We would also like to thank Dr Darren Juniper for completing the additional statistical analyses.

* Corresponding author.
E-mail address: lynne.clark@imperial.ac.uk (L. Clarke).

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doi:10.1016/j.livsci.2008.10.012

mobilization during late pregnancy (Revell et al., 1998), and may result in enhanced sow performance during lactation.

The fatty acid composition of body fat reserves in pigs reflects that of the diet (Huo et al., 2003). In rodent studies it has been shown that fat accumulates during early pregnancy (López-Luna et al., 1986), as a consequence of enhanced insulin sensitivity (Ramos et al., 2003) resulting in enhanced adipose tissue lipoprotein lipase activity (Knopp et al., 1973; Martin-Hidalgo et al., 1994). These changes allow maternal fat reserves to be laid down during early gestation, being later mobilized during late pregnancy and lactation (Mullan and Williams, 1990). Thus, it seems probable that the fatty acid composition of maternal fat reserves will influence milk composition (Hartmann and Holmes, 1989; Rooke et al., 2001a,b), which in turn may have consequences for piglet growth and survival (Cieslak et al., 1983; Rooke et al., 2001b). It is well established that milk yield is influenced by the number size and vigor of piglets being suckled (King, 2000). An improved milk fatty acid profile, as a result of maternal fat supplementation, may result in increased piglet vigor; leading to enhanced milk yield, which acts synergistically to improve piglet growth and development, further enhancing milk yield.

The role of dietary fat during early pregnancy on milk composition and yield in sows has not yet been fully established. To enable the importance of type and timing of oil supplementation to be evaluated this study aimed to determine the consequences of altering the fatty acid profile of sow diets during either the first or second half of gestation; oils of different fatty acid composition were chosen as energy supplements to provide diets with different fatty acid profiles. Consideration was given to sow condition, milk yield and milk composition.

2. Materials and methods

2.1. Animals and diets

All animals used in these studies were maintained at the Pig Research and Development Unit, Imperial College, London. Experimental procedures were carried out according to the regulations of the Animals (Scientific Procedures) Act, 1986 and were licensed by the Home Office (UK). At all stages of life, animals were kept according to the guidelines set out by the Department for Environment Food and Rural Affairs (DEFRA, 2003).

Eighty-eight multiparous sows of the same commercial genotype (25% Meishan; 12.5% Duroc; 62.5% Large White × Landrace) were selected for study after the previous litter had been weaned and prior to insemination. Sows were categorized by parity before being randomly assigned to one of six dietary treatment groups, to ensure that parity was balanced across treatments. All sows were artificially inseminated with pooled Large White semen (P17 2006, JSR Genetics). The control (C: $n=8$) diet consisted of 3 kg/day of the standard diet (ABN HE sow pellets ABN, Peterborough, UK). Dietary treatments consisted of 3 kg/day of the C ration plus 10% extra energy derived from either: i) extra pellets (E: $n=16$); ii) palm oil (P: $n=16$); iii) olive oil (O: $n=16$); iv) sunflower oil (S: $n=16$) or fish oil (F: $n=16$). Our analyses of the experimental diets and their fatty acid profiles can be seen in Table 1. The experimental diets were fed during either the first (G1: day 1 of gestation (assuming day of service to be day 0) until day 60 of gestation: $n=40$) or second half (G2: day 60 of gestation until term (≈day 115): $n=40$). All diets, with the exception of the C ration, were isocaloric. All sows were offered 3 kg/day of the standard diet (as for the C group) outside the experimental period. Between farrowing and weaning (21–28 days post partum) sows were offered 6–9 kg of a standard lactation ration (ABN supreme lactation pellets; ABN, Peterborough, UK).

Table 1
Composition of diets.

	Control/ excess diet	Palm oil diet	Olive oil diet	Sunflower oil diet	Fish oil diet	Lactation diet
Crude protein (%)	13.1	12.7	12.5	12.7	12.5	18.2
Ash (%)	4.6	4.5	4.4	4.3	4.5	4.9
Crude fibre (%)	4.4	5.05	4.4	3.9	5.1	4.1
ME (MJ/kg DM)	13.3	13.7	13.8	13.8	13.8	14.2
Oil (%)	5.2	6.6	6.8	6.7	6.8	7.1
14:0*	0.54	0.77	0.28	0.31	2.40	0.85
16:0*	16.92	31.76	13.79	12.91	15.84	20.45
16:1 n−7*	0.37	0.72	1.05	0.22	4.18	0.24
18:0*	2.61	3.56	2.57	3.39	2.74	5.22
18:1 n−9*	19.76	26.68	47.30	20.09	19.92	32.81
18:2 n−6*	53.13	32.82	31.14	58.51	27.96	34.83
18:3 n−3*	5.25	2.76	2.77	3.13	3.16	3.97
20:1 n−9*	0.46	0.36	0.42	0.38	5.26	0.36
20:5 n−3*	0.17	0.08	0.07	0.13	4.16	0.30
22:0*	0.31	0.20	0.285	0.55	0.22	ND
22:1 n−9*	0.11	0.04	0.03	0.08	5.40	ND
22:3 n−3*	ND	ND	0.03	ND	0.08	ND
22:5 n−3*	ND	0.03	0.03	0.03	1.63	ND
22:6 n−3*	0.03	0.02	0.01	0.02	6.13	0.08
∑S*	22.65	32.9	20.43	16.17	22.09	27.31
∑M*	21.42	30.42	48.41	22.57	31.35	33.48
∑P*	55.94	36.69	31.025	61.27	46.55	39.21
∑ n−6*	50.58	33.54	27.205	57.97	32.56	34.84
∑n−3*	5.36	3.15	3.82	3.3	13.99	4.36

* = g/100 g fatty acids; S = saturated fatty acids; M = monounsaturated fatty acids; P = Poly-unsaturated fatty acids; ND = none detected. Values presented are mean percentages of total lipid fraction. Values presented are mean percentages from 2 determinations of total lipid fraction extracted from samples of diet.

2.2. Sow weight and body condition

On days 0, 35, 56, 84 and 109 of gestation and at weaning sows were restrained in a weigh crate (UHL Products, UK) whilst their weight and back fat thickness, using ultrasound (Aloka-echo camera 550–500, Aloka Ltd. Japan), were measured. Back fat thickness was measured, level with the head of the last rib, at the P1 (45 mm from the midline) and P3 (80 mm from the midline) positions. The average of these two values was then calculated to give the P2 value.

2.3. Milk yield and composition

Milk yield was assessed using an adaptation of the weigh–suckle–weigh method, described by Sinclair et al. (1999). Milk yield was assessed on days 3, 7, 14 and 21 of lactation. Natural suckling was allowed, and the inter-suckling interval recorded. Piglets were observed throughout, and weighed before and after, four consecutive sucklings. Urination and defecation by piglets during this period was also recorded. The following equation was used to estimate milk yield per suckling:

$$\text{Milk yield (kg)} = W + U + D + M$$

where W = litter weight gain (kg); U = weight loss due to urination; D = weight loss due to defecation; M = metabolic weight loss.

Weight loss due to urination was calculated using the equation described by Klaver et al. (1981):

$$\text{Weight loss} = \left[U \times \left(2.9 \times W^{0.75} + 18.7\right)\right]$$

where: U = number of urinations; $W^{0.75}$ = piglet metabolic weight.

A 10 g loss was allowed per defecation (Sinclair et al., 1999). An estimate of metabolic loss was calculated using the equation described by Noblet and Etienne (1986):

$$\text{Weight loss (mg)} = 60 \text{ per kg live weight per min.}$$

Colostrum samples were collected on the day of parturition, within 4 h of the first piglet being born. Milk samples were collected on day 3, day 7, day 14 and day 21 of lactation following intra-muscular administration of 2 mL oxytocin (10 IU/mL; NVS, UK). A 20 mL aliquot of each milk sample was stored in azide coated sample pots at 4 °C prior to analysis for milk composition by an automated infrared filtration system; these analyses were carried out by National Milk Records (Harrogate, UK). Estimated total milk energy was then calculated using an adaptation of the equation described by Klaver et al. (1981):

$$\begin{aligned}\text{Total energy (MJ/kg)} = 0.0042 \times [&(92.2 \times \text{fat\% w/w}) \\ &+ (61.3 \times \text{protein\% w/w}) \\ &+ (35.6 \times \text{lactose\% w/w})].\end{aligned}$$

A further two 1.5 mL aliquots of each sample were frozen at −80 °C, in tubes containing 2 μL Na_2 EDTA (0.5 M: Sigma-Aldrich, Germany), prior to analysis of fatty acid profile and immunoglobulin content.

Lipids from diet aliquots were extracted and purified in chloroform-methanol (Folch et al., 1957), and aliquots of either diet lipid extracts or milk were saponified and the fatty acids methylated following the method of Lepage and Roy (1984, 1986). Fatty acid methyl esters were separated on a 30 m × 0.25 mm Omegawax capillary column (Supelco, Bellefonte PA, USA) and quantified using a Perkin-Elmer gas chromatograph (Autosystem; Norwalk, Conn.) with a hydrogen flame ionization detector. Nitrogen was used as a carrier gas, and the fatty acid methyl esters were compared with purified standards (Sigma Chemical Co., St Louis, MO). Concentrations of immunoglobulins were determined by the use of commercially available radial immunodiffusion kits (RID; Pig IgG, IgA and IgM VET-RID kits; Bethyl Laboratories, Texas, USA).

2.4. Statistical analyses

One of the C sows became unwell shortly after farrowing, and its piglets had to be weaned early. All data from this animal were excluded prior to analyses. Statistical differences between the main effects of timing (2 *df*) and type (5 *df*) of dietary treatment were determined by ANOVA using the mixed model procedure of SAS version 9.1.3 (SAS Institute Inc., Cary, NC, USA). Sources of variation within the model included dietary intervention (i.e. type or timing) and sample point where individual animals formed the repeated subject and sample point the repeated measure. Statistical tests were undertaken for main effects and their first order interaction (i.e. time by treatment). Where appropriate, start weight or start backfat thickness, parity, litter size and number of pigs reared were used as covariate terms within the model. Results are presented as least square means with standard error and *P* value. Tukey's simultaneous tests were used to establish statistical differences between individual dietary interventions.

3. Results

3.1. Sow weight and body composition

Despite being balanced for parity (3.1 ± 0.5; mean ± SEM), sow weights at the start of the study varied ($P = 0.09$) between treatments (Table 2) and thus were analyzed as a co-variate in the statistical analysis of sow weights. Parity was

Table 2
Mean effects of the type and timing of dietary supplementation on sow body weight during gestation and lactation.

		C* (*n*=7)	E G1 (*n*=8)	P G1 (*n*=8)	O G1 (*n*=8)	S G1 (*n*=8)	F G1 (*n*=8)	E G2 (*n*=8)	P G2 (*n*=8)	O G2 (*n*=8)	S G2 (*n*=8)	F G2 (*n*=8)	Pooled SEM	Statistical difference		
														Diet	Timing (G1 vs G2)	Diet*timing
Start (day 0)		202	213	197	202	207	200	207	201	213	204	208	6.7			
Sow weight (kg)	1	218	217	216	218	216	220	211	211	212	210	208	3.2	$P=0.682$	$P=0.002$	$P=0.655$
at sample point†	2	229	230	234	233	234	234	223	224	222	221	222	3.2			
	3	245	243	245	244	245	253	242	240	241	240	241	3.2			
	4	256	260	261	263	261	265	264	262	260	260	258	3.2			
	5	234	224	220	226	220	215	218	228	230	225	223	3.2			
Sample point		$P<0.001$														
Net change (kg)‡	G1	26.3	28.7	23.0	25.2	20.0	14.2	19.7	23.4	28.2	24.7	20.9	3.7	$P=0.598$	$P=0.737$	$P=0.577$
	G2	23.1[a]	33.1	29.3	26.1[a]	33.9	27.5[a]	40.0[b]	31.7	37.5	43.7[b]	30.9	3.1	$P=0.263$	$P<0.01$	$P=0.858$
	G	56.5	61.1	61.0	61.2	60.8	61.9	61.8	60.6	32.3	61.8	55.8	3.1	$P=0.971$	$P=0.787$	$P=0.892$
	L	−24.7[a]	−32.5[a]	−39.1	−31.9[a]	−43.4	−47.1[b]	−38.0	−34.9	−33.6	−36.9	−34.2	4.1	$P=0.718$	$P=0.387$	$P=0.523$
	W	26.3	28.7	23.0	25.2	20.0	14.3	19.7	23.4	28.2	24.7	20.9	3.7	$P=0.598$	$P=0.737$	$P=0.577$

*C = control; E = excess; P = palm oil; O = olive oil; S = sunflower oil; F = fish oil; G1 = first half of gestation (days 1–60 of gestation); G2 = second half of gestation (days 61–≈115 of gestation).
†1 = day 35 gestation; 2 = day 56 gestation; 3 = day 84 gestation; 4 = day 109 gestation; 5 = weaning (i.e. 21–28 days post partum).
‡G = gestation; G1 = 1st half of gestation (0–56 days); G2 = 2nd half of gestation (56–109 days); L = lactation (day 109 gestation–weaning) (corrected for litter weight); W = whole reproductive cycle (i.e. day 0 gestation–weaning).
Data presented are least square means ± pooled SEM. Within a row, means with different superscripts differ significantly ($P<0.05$).

Table 3
Mean effects of the type and timing of dietary supplementation on sow backfat thickness (mm at the P2 position) during gestation and lactation.

		C* (n=7)	E G1 (n=8)	P G1 (n=8)	O G1 (n=8)	S G1 (n=8)	F G1 (n=8)	E G2 (n=8)	P G2 (n=8)	O G2 (n=8)	S G2 (n=8)	F G2 (n=8)	Pooled SEM	Statistical difference		
														Diet	Timing (G1 vs G2)	Diet*timing
Start (day 0)		14.2	17.4	14.4	14.3	19.1	14.1	15.7	16.3	16.8	17.9	16.4	0.60			
Backfat thickness (mm) at sample point†	1	18.9	20.0[a]	20.9[a]	20.4[a]	21.2[a]	20.1[a]	18.3	16.9[b]	17.8[b]	18.1	18.4	0.64	$P=0.12$	$P<0.001$	$P<0.001$
	2	18.5[a]	21.6[b]	21.7 [b]	21.3 [b]	23.6[b]	22.1 [b]	19.3[a]	19.3[a]	18.3[a]	18.4[a]	19.9[a]	0.68			
	3	21.5 [a]	22.6 [a]	21.5 [a]	22.3 [a]	26.2 [b]	21.7 [a]	21.5 [a]	21.5 [a]	21.0 [a]	21.1 [a]	23.7 [a]	0.74			
	4	21.7 [a]	24.4 [b]	23.1	22.7	26.1 [b]	23.2	22.8	23.0	22.7	23.1	25.2 [b]	0.68			
	5	19.6[b]	18.9	18.6	18.8	19.7[b]	16.6[a]	16.9 [a]	18.0	18.9	18.4	19.2[b]	0.64			
Sample point		$P<0.001$														
Net change (mm)‡	G1	2.1[a]	4.1 [b]	4.8 [b]	3.9 [b]	5.9 [b]	5.3 [b]	2.6 [a]	2.2 [a]	0.5[a]	0.5[a]	3.0[a]	0.23	$P=0.324$	$P<0.001$	$P=0.326$
	G2	1.0[a]	4.3 [b]	0.7[a]	0.9[a]	4.1 [b]	−0.5[c]	1.6[a]	2.8[b]	5.3 [b]	4.5 [b]	4.1 [b]	0.21	$P=0.076$	$P<0.01$	$P<0.01$
	G	4.2	7.8	5.0	4.6	9.4	5.6	5.1	5.6	5.7	5.3	8.3	0.23	$P=0.079$	$P=0.461$	$P<0.01$
	L	−3.2	−6.5	−4.9	−2.1	−7.2	−6.5	−5.6	−4.2	−4.5	−3.0	−6.1	0.29	$P=0.112$	$P=0.346$	$P=0.166$
	W	2.0	1.6	0.8	2.0	2.4	−1.0	−0.7	0.3	2.0	2.1	1.3	0.30	$P=0.328$	$P=0.809$	$P=0.456$

*C = control; E = excess; P = palm oil; O = olive oil; S = sunflower oil; F = fish oil; G1 = first half of gestation (days 1–60 of gestation); G2 = second half of gestation (days 61–≈115 of gestation).
†1 = day 35 gestation; 2 = day 56 gestation; 3 = day 84 gestation; 4 = day 109 gestation; 5 = weaning (i.e. 21–28 days post partum).
‡G = gestation; G1 = 1st half of gestation (0–56 days); G2 = 2nd half of gestation (56–109 days); L = lactation (day 109 gestation–weaning); W = whole reproductive cycle (i.e. day 0 gestation–weaning).
Data presented are least square means ± pooled SEM. Within a row means with different superscripts differ significantly ($P<0.05$).

shown to have no effect on either sow weight *per se* or changes in sow weight throughout the duration of the study and so was removed from the model. As expected litter size ($P<0.001$) and number of piglets reared ($P<0.001$) influenced sow weight throughout the various sample points. Sows gained weight throughout gestation ($P<0.001$) and lost weight between farrowing and lactation ($P<0.001$; Table 2). The timing of dietary supplementation had a pronounced effect on sow body weight ($P=0.002$). In contrast, there was no influence of dietary type on weight, hence the body weight of all G1 or G2 were similar throughout the various stages of the experimental period.

Initial sow body weight influenced their weight change over the whole experimental period ($P<0.001$). However, this effect was predominantly during G1 ($P<0.05$) rather than G2 and over the lactation period. Litter size but not number of piglets reared influenced weight changes observed in G1 ($P<0.05$) but not G2. However, both litter size and number of piglets reared had an effect on weight gain over the whole of gestation ($P<0.05$), whereas only the number of piglets

Table 4
Mean effects of the type and timing of dietary supplementation on milk yield, milk composition and milk energy yield.

	Stage of lactation (days)	C[a] (n=7)	E G1 (n=8)	P G1 (n=8)	O G1 (n=8)	S G1 (n=8)	F G1 (n=8)	E G2 (n=8)	P G2 (n=8)	O G2 (n=8)	S G2 (n=8)	F G2 (n=8)	Pooled SEM	Statistical difference		
														Diet	Timing (G1 vs G2)	Diet*timing
Estimated milk yield (kg/day)	3	7.35	7.50	7.06	7.77	7.36	7.30	6.00	4.66	5.97	5.32	5.48	0.76	$P=0.311$	$P<0.001$	$P<0.05$
	7	9.14	8.04	8.25	7.71	9.47	8.37	5.99	6.00	6.97	6.31	7.65	0.75			
	14	7.65	7.71	7.86	7.83	8.55	6.88	7.27	9.28	5.63	8.46	8.09	0.74			
	21	7.94	7.26	7.88	7.81	8.55	8.30	8.58	9.36	6.72	7.71	9.88	0.76			
Milk fat (%)	3	5.43	4.97	4.66	4.74	5.62	4.75	4.58	4.13	5.05	4.67	4.70	0.22	$P=0.411$	$P<0.001$	$P=0.610$
	7	4.52	4.77	4.49	4.37	4.74	4.52	5.22	4.78	5.23	5.36	5.08	0.22			
	14	5.28	4.41	4.51	4.47	4.37	4.43	5.49	5.45	5.10	5.28	5.55	0.20			
	21	4.97	4.65	4.74	4.59	4.50	4.59	5.43	5.42	5.39	5.44	5.45	0.24			
Milk protein (%)	3	5.18	4.75	4.43	4.49	5.38	4.49	5.08	6.00	4.75	5.13	5.28	0.47	$P=0.241$	$P<0.01$	$P=0.208$
	7	4.40	4.64	4.35	4.23	4.68	4.37	4.85	4.84	4.70	4.71	4.59	0.32			
	14	4.71	4.50	4.59	5.54	4.47	4.62	4.65	4.66	4.87	4.46	4.58	0.26			
	21	5.15	4.93	5.02	4.87	5.02	4.95	5.00	5.06	4.50	4.94	4.87	0.55			
Milk lactose (%)	3	5.31	5.69	5.31	5.51	5.03	5.69	5.22	4.84	5.73	5.38	5.40	0.39	$P=0.336$	$P=0.669$	$P=0.175$
	7	5.35	5.51	5.64	4.42	5.61	5.69	5.60	5.21	5.63	5.70	5.42	0.27			
	14	5.21	4.90	5.08	5.17	5.29	5.17	5.26	5.26	4.89	4.99	5.29	0.22			
	21	4.38	4.77	4.71	4.70	4.72	4.82	4.63	4.63	4.58	4.62	4.61	0.45			
Milk energy (MJ/kg)	3	5.53	5.24	5.14	4.90	5.90	5.03	5.86	5.93	5.13	4.97	5.71	0.29	$P=0.800$	$P=0.145$	$P=0.273$
	7	5.04	5.36	4.87	4.32	5.44	5.17	5.70	5.60	5.30	5.29	5.23	0.27			
	14	4.89	5.32	5.29	5.20	5.16	5.21	5.15	5.16	5.06	5.14	5.11	0.27			
	21	5.15	5.14	5.00	5.14	5.15	5.15	5.01	5.34	4.81	5.29	4.95	0.29			

[a]C = control; E = excess; P = palm oil; O = olive oil; S = sunflower oil; F = fish oil; G1 = first half of gestation (days 1–60 of gestation); G2 = second half of gestation (days 61–≈115 of gestation).
Data presented are least square means ± pooled SEM.

reared influenced weight loss during lactation ($P<0.05$). Irrespective of the timing of supplementation, the gain in body weight over G1 was similar between the groups but weight gain during the second half of gestation was greater in animals receiving extra energy during this period compared to in C or G1 animals ($P<0.01$; Tables 2 and 3). E G2 and S G2 sows gained considerably more weight than those animals in the C, OG1 and FG1 groups ($P<0.05$; Table 2). After parturition, weight loss was lowest in C, EG1 and OG1 mothers, and greatest in the FG1 group ($P<0.05$). When the weight gain over either the entire gestational period or the whole of the reproductive cycle were examined no differences were observed.

As with body weight, there was also variation (not significant) in back fat depth at the start of the study (Table 3), and therefore this was used as a co-variate in the analysis of sow back fat depth. Parity was shown to have little influence on either backfat depth *per se* or changes in backfat thickness throughout study and so was removed from the model. Likewise, neither litter size nor number of piglets reared influenced maternal backfat depth at the various sample points. Back fat depth increased ($P<0.001$) throughout gestation and decreased during lactation ($P<0.001$; Table 3). Supplementation during the first half of gestation resulted in a net increase in fat deposition at the P2 position, compared to C and G2 animals during this period ($P<0.05$; Table 3); this effect was most pronounced in S G1 sows and these animals remained the fattest, although the difference became less apparent towards the end of gestation. Approximately 1 week prior to parturition, E G1 and F G2 groups also possessed considerably more fat than C mothers ($P<0.05$). At the end of lactation E G2 and F G1 had

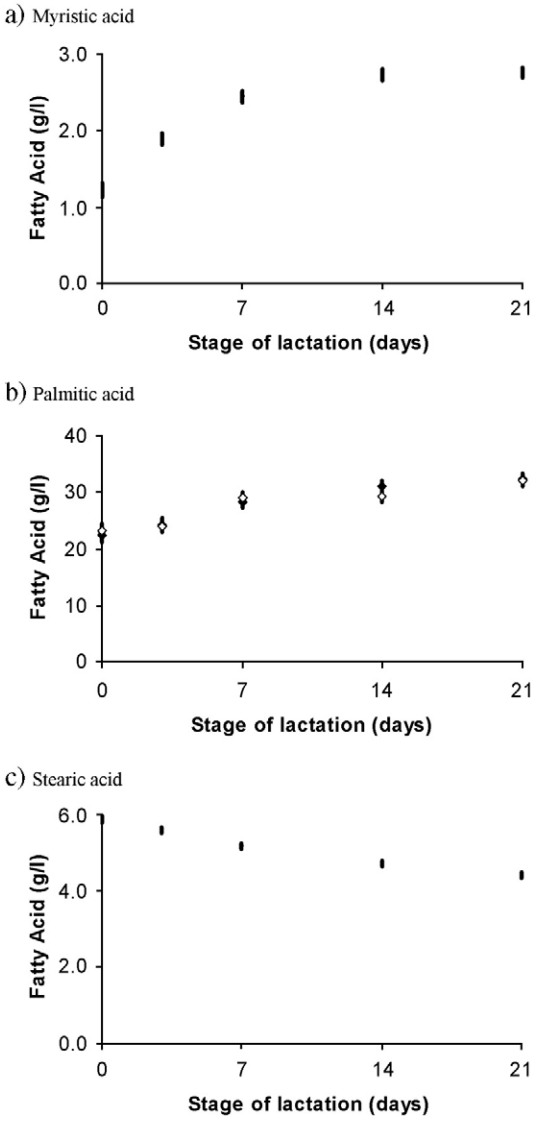

Fig. 1. Mean concentration of key fatty acids in the milk fat of sows given experimental diets during gestation. Mean value of all the treatments at each time point = –. Specific mean values are only given for diets for which the fatty acid shown is typical; thus ♦ = P G1; ◊ = P G2; ● = O G1; ○ = O G2; ▲ = S G1; Δ = S G2; ■ = F G1; □ = F G2; Values are presented as adjusted least squares means.

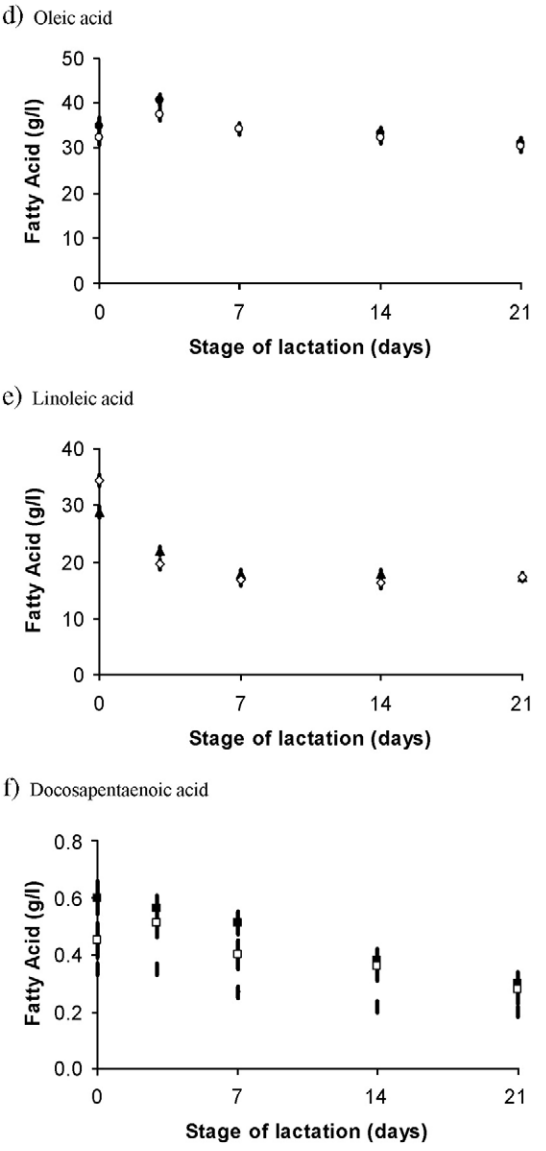

Fig. 1 (*continued*).

less fat at the P2 position compared to C, S G1 and F G2 groups ($P<0.05$).

Initial backfat thickness, litter size and number of piglets reared did not influence the changes in fat depth either during G1, G2, the entire gestation period, the lactation period or the whole of the experimental period. The provision of extra energy during G1 resulted in increased fat deposition during G1 ($P<0.001$; Table 3). Interestingly, sows in the E G1 and S G1 groups continue to deposit fat at elevated rates during G2. Generally, dietary supplementation during G2 resulted in the accretion of more fat but to a much lesser extent in E G2 and P G2 compared to OG2, S G2 and F G2 animals ($P<0.05$). Those animals exhibiting higher levels of fat deposition during gestation, namely, E G1, S G1 and F G2 mothers, mobilized more fat over the lactation period compared to all other groups, except F G1 who mobilized a greater proportion of their fat reserves during lactation than they had accumulated during gestation, resulting in a net loss in back fat depth over the whole production cycle (from service-weaning).

3.2. Milk yield and composition

There was a trend for both parity ($P=0.092$) and number of piglets reared to influence milk yield ($P=0.079$) but not milk composition or milk energy content. Actual milk yields increased between day 3 and day 21 of lactation ($P<0.05$; Table 4). The type of dietary supplementation had little effect on milk yield and composition (Table 4). However, the timing of dietary supplementation appeared to have a significant influence on milk yield ($P<0.001$) as well as the percentages of fat ($P<0.001$) and protein ($P<0.01$) in milk; percentage milk lactose and milk energy were similar between C, G1 and G2 groups. On day 3 of lactation milk yield and the fat content of milk were all higher in C and G1 mothers compared to G2 sows, whereas the protein content of milk was lower in the C and G1 groups. Milk yield was still reduced in G2 animals on day 7; this effect continued in O G2 and S G2 throughout the remainder of gestation. Conversely, milk yield increased in mothers receiving E, P and F diets by day 21 of lactation. In contrast, milk fat had increased in G2 mothers by day 7 of lactation, whereas the reverse situation occurred in C and G1 sows. The protein content of milk decreased after day 3 of lactation in G2 pigs and by day 7 they had reached similar levels to the other treatment groups.

The results of the fatty acid analysis showed that, irrespective of the maternal diet, the concentration of myristic (14:0) and palmitic (16:0) acid increased with advancing lactation (Fig. 1a and b). The concentration of stearic (18:0), oleic (18:1) and linoleic (18:2 n−6) acids decreased over the course of lactation, independently of maternal diet (Fig. 1c–e). The fatty acid profile of the maternal diet during gestation appeared to influence the fatty acid profile of milk during lactation. The most dramatic differences in milk fatty acid profile were seen in sows that had received either sunflower oil or fish oil diets during gestation; with change in milk fatty acid profile reflecting the fatty acid composition of the diet (Table 5). The concentration of linoleic acid was greater throughout lactation in the milk of S sows (Fig. 1e). The concentration of both docasapentaenoic (22:5 n−3) and docosahexaenoic (22:6 n−3) acid was greater throughout lactation in the milk of F sows, irrespective of the timing of supplementation; the concentration of these long chain n−3 PUFA was highest during early lactation and decreased over the course of lactation, although concentrations were not as high in the colostrum off F G1 supplemented sows (Table 5; Fig. 1f–g).

Irrespective of the type or timing of maternal dietary supplementation, the concentration of immunoglobulins were greater in colostrums compared to milk, and there was a dramatic decline in concentration between day 0 and day 3 of lactation, after which their concentrations remained relatively stable. Maternal dietary supplementation during the first half of gestation resulted in a greater ($P<0.01$) concentration of IgG in colostrum; this effect was exacerbated in P G1 and S G1 sows ($P<0.01$; Table 6). Although the timing of the maternal supplement did not influence the concentrations of either IgA ($P<0.01$) or IgM ($P<0.05$), the type of diet the mother received altered their concentrations. P G1, F G1, S

Table 5
Mean effects of sow diet supplementation during either the first (G1) or second half of gestation (G2) on the fatty acid composition of milk (g/100 g of fat) during lactation.

Fatty acid	C* ($n=7$)	E G1 ($n=8$)	P G1 ($n=8$)	O G1 ($n=8$)	SG1 ($n=8$)	F G1 ($n=8$)	E G2 ($n=8$)	P G2 ($n=8$)	O G2 ($n=8$)	SG2 ($n=8$)	F G2 ($n=8$)	Pooled SEM	Statistical difference		
													Diet	Timing (G1 vs G2)	Diet*timing
14:0	2.37	2.29	2.36	2.20	2.00	2.17	2.24	2.12	2.25	2.14	1.98	0.17	$P=0.617$	$P=0.588$	$P=0.771$
16:0	29.50	27.75	27.68	26.80	26.91	27.56	27.85	27.52	27.80	27.22	27.06	0.58	$P=0.731$	$P=0.713$	P0.800
18:0	5.08	4.91	5.13	5.18	5.29	5.45	5.01	5.15	4.92	5.13	5.30	0.20	$P=0.273$	$P=0.469$	$P=0.900$
18:1(n−9)	32.77	32.54	33.58	34.84	32.66	32.26	32.20	33.55	33.82	31.54	34.09	0.80	$P=0.115$	$P=0.938$	$P=0.222$
18:2(n−6)	17.07 [a]	18.01[a]	19.07[a]	18.7 4[a]	20.77[b]	19.69	17.66[a]	18.86[a]	18.74[a]	20.82 [b]	18.38[a]	0.51	$P<0.001$	$P=0.256$	$P=0.656$
22:6(n−3)	0.12[a]	0.18[a]	0.10[a]	0.10[a]	0.11[a]	0.49[b]	0.35[b]	0.12[a]	0.14[a]	0.14[a]	0.42 [b]	0.05	$P<0.001$	$P=0.201$	$P=0.197$
∑S	37.23	35.33	35.42	34.63	34.40	35.58	35.35	35.14	35.23	34.77	34.51	0.65	$P=0.578$	$P=0.937$	$P=0.814$
∑M	41.60	40.66	40.65	41.96	39.49	39.52	40.63	40.78	40.55	38.81	41.33	0.74	$P=0.059$	$P=0.938$	$P=0.268$
∑P	20.87[a]	23.82	23.72	23.17	25.55[b]	24.92[b]	22.34	23.79	23.74	25.62b[a]	23.04	0.61	$P<0.01$	$P=0.171$	$P=0.196$
∑n−3	2.39[a]	3.87[b]	2.86	2.79	2.78	3.52[b]	3.04	3.34[b]	2.86	2.97	3.52	0.21	$P<0.01$	$P=0.892$	$P<0.05$
∑ n−6	18.61[a]	20.08	20.95	20.48	22.87 [b]	21.90	19.64[a]	20.56	21.03	22.78[b]	19.93	0.59	$P<0.01$	$P=0.210$	$P=0.304$
P:S	0.62 [a]	0.70	0.70	0.69	0.77[b]	0.74[b]	0.65[a]	0.70	0.70	0.80[b]	0.69	0.02	$P<0.001$	$P=0.514$	$P=0.334$
n−6 :n−3	8.73[a]	6.82	7.89	7.70	8.74[a]	6.45	7.27	6.93	7.90	8.35 [a]	5.94[b]	0.42	$P<0.001$	$P=0.360$	$P=0.455$

*C = control; E = excess; P = palm oil; O = olive oil; S = sunflower oil; F = fish oil; G1 = first half of gestation (days 1–60 of gestation); G2 = second half of gestation (days 61–≈115).
S = saturated fatty acids; M = monounsaturated fatty acids; P = poly-unsaturated fatty acids.
Within a row, means with different superscripts differ significantly ($P<0.05$).

Table 6
Mean effects of the type and timing of dietary supplementation on the immunoglobulin concentration of lacteal secretions.

	Stage of lactation (days)	C* ($n=7$)	E G1 ($n=8$)	P G1 ($n=8$)	O G1 ($n=8$)	S G1 ($n=8$)	F G1 ($n=8$)	E G2 ($n=8$)	P G2 ($n=8$)	O G2 ($n=8$)	S G2 ($n=8$)	F G2 ($n=8$)	Pooled SEM	Statistical difference		
														Diet	Timing (G1 vs G2)	Diet*timing
IgG (mg/mL)	0	3103	4131	5880	4675	6039	4631	3661	3823	3042	3558	2250	289	$P=0.460$	$P<0.01$	$P=0.352$
	3	310	275	304	388	311	307	269	424	229	275	327	254			
	7	277	238	254	287	253	256	272	259	313	246	262	253			
	14	283	236	277	259	259	240	275	254	245	229	258	254			
	21	337	245	352	270	262	259	270	246	234	251	270	255			
IgA (mg/mL)	0	501[a]	561[a]	1101[b]	776	703	980[b]	614	764	463	823[b]	1010[b]	83	$P<0.01$	$P=0.182$	$P=0.053$
	3	172	167	202	181	214	195	184	219	131	192	196	72			
	7	134	137	172	201	155	138	189	172	192	157	163	72			
	14	152	136	220	185	204	166	193	151	147	175	191	72			
	21	249	187	384	184	223	195	211	165	162	186	217	72			
IgM (mg/mL)	0	334	247[a]	240[a]	251[a]	616[b]	374	266[a]	456[b]	330	282	216[a]	30	$P<0.05$	$P=0.273$	$P<0.001$
	3	65	49	40	42	71	54	35	53	52	45	52	26			
	7	42	42	44	48	57	49	37	49	59	40	53	26			
	14	56	41	43	48	51	47	45	55	67	47	60	26			
	21	61	43	109	41	51	54	45	47	47	78	57	26			

*C = control; E = excess; P = palm oil; O = olive oil; S = sunflower oil; F = fish oil; G1 = first half of gestation (days 1–60 of gestation); G2 = second half of gestation (days 61–≈115.
Values presented are adjusted least squares means ± SEM. Within a row, means with common superscripts differ significantly ($P<0.05$).

G2 and F G2 groups exhibited secreted more IgA in their colostrums compared to the other groups. ($P<0.05$; Table 6), whereas S G1 and P G2 sows possessed more IgM in their colostrum ($P<0.05$; Table 6).

4. Discussion

4.1. Sow weight and body composition

There is a plethora of data demonstrating that sow weight gain during gestation increases with increasing dietary energy intake (e.g. Baker et al., 1968; Cromwell et al., 1989; Dourmad, 1991; Dourmad et al., 1996; Wladyslaw, 1991; Averette Gatlin et al., 2002). In the present study sow weight gain was generally increased during the period of supplementation compared to the other groups. Interestingly weight gain did not appear to be influenced by the type of dietary supplement during G1. In contrast the E and S diets appeared to increase sow weight gain when they were received during G2. It is speculated that this may be due to an over supply of linoleic acid (18:2 n−6), since the basal diet also contained some sunflower oil.

During lactation it is normal for sows to loose weight as their body reserves are mobilized for milk production (Trottier and Johnston, 2001). In the present study, weight loss during lactation was lower in the C sows compared to those receiving supplements. Moreover, weigh loss was similar for all experimental groups, with the exception of F G1 and, to a lesser extent, S G1 sows. These sows lost considerably more weight during lactation, which may, in part, be due to the fact that they put on less weight over the first part of gestation compared to the other groups. Previously Averette et al. (1999) observed no difference in weight loss between control and fat supplemented animals, but our results indicate that the type of fat supplement may have a role to play when supplements are provided, particularly during G1. Further work is required to fully understand the relationship between energy partitioning during lactation and the fatty acid profile of the diet.

Reserves of body fat in sows given higher energy diets have been observed to increase during gestation, compared to controls (Dourmad, 1991; Dourmad et al., 1996; Mullan and Williams, 1989). Similarly, in the present study, the accumulation of body fat reserves during gestation (as indicated by the change in back fat depth) was increased by supplementation with extra energy, whatever the source. In particular the addition of sunflower oil to the maternal diet during both G 1 and G 2 had a beneficial effect on fat accumulation. It is of interest to note that both E G1 and S G1 continued to maintain high rates of fat accretion throughout G2 when the supplement was no longer being given. These observations suggest that the extra dietary energy received by these animals was being stored in maternal fat depots rather than being used for maintenance or growth of the products of conception (i.e. placenta as well as fetus). Again, it is likely that the higher concentration of dietary linoleic acid in the E and S diets may mediate this effect.

P G1 and F G1 also exhibited greater rates of fat accumulation during G1 but unlike the E G1 and S G1 groups, the net change in backfat depth was reduced to basal levels when the supplement was no longer supplied. Whilst the F G1 mothers showed increased fat deposition during G1, they appeared to be unable to sustain fat deposition during G2 when their energy intake was returned to normal. This may be due to selective mobilization of long chain polyunsaturated fatty acids (PUFAs) (Raclot, 2003) leading to changes in the ration of n−6 to n−3 fatty acids affecting blood hormone concentrations (Steele et al., 1985; Hornstra and Stegan, 1989), resulting in increased catabolism of maternal reserves during late gestation. O G2 exhibited the greatest change in backfat depth during G2 and was almost twice that observed in E G2 and P G2 sows.

During lactation, the nutritional requirements for milk production are met from both dietary sources and from maternal body reserves. At weaning F G1 sows were the leanest, together with the data for weight loss during lactation this suggests that F sows (albeit from a lower starting point) mobilized a higher proportion of their body reserves than those receiving other supplements (Hulten et al., 1993; Clowes et al., 2003).

4.2. Milk yield and composition

Milk yield during early lactation was unaffected by treatment *per se* but milk yield was much lower over the first week of lactation in sows receiving supplements during G 2. However, by the end of lactation milk yield tended to be higher in E G2, P G2 and F G2 mothers, and is in agreement with the findings of others (Boyd et al., 1982; Noblet and Etienne, 1986; Shurson et al., 1986). Supplementation of sow diets with fat during late pregnancy and lactation has been demonstrated to increase the concentration of fat in colostrum and milk (Boyd et al., 1982; Coffey et al., 1982). However, we failed to show any differences in percentage milk fat, or indeed protein and lactose with respect to diet but those animals supplemented during G2 tended to have higher percentages of fat and protein in their milk; this was only true for the first week of lactation.

The fatty acid profile of milk is known to alter with fat supplementation of sow diets (Seerley et al., 1974; Coffey et al., 1982; Wladyslaw, 1991; Newcomb et al., 1991; Rooke et al., 2001b). The fatty acid profiles of the lipid fraction of the milk, in the current study, differed both with stage of lactation and between treatment groups. Independently of the diet received, the concentration of myristic acid (14:0) and palmitic acid (16:0) increased with advancing lactation as fatty acid synthesis by the mammary gland increases (Bazer et al., 2001). In contrast, stearic and (18:0) and oleic (18:1) acid decrease as lactation advances indicating that fatty acid synthesis becomes increasingly important as lactation progresses, relative to the importation of fatty acids from the maternal circulation.

In the milk of sows that had received the F diet (irrespective of timing), the concentration of docosapentaenoic (22:5 n−3) and docosahexaenoic (22:6 n−3) acid was much higher than in that of other animals, supporting the work of Rooke et al. (2001b) and Lauridsen and Danielsen (2004). In the case of F G1 sows long chain (n−3) polyunsaturated fatty acids were not available in the diet given during G2 or in lactation, so their appearance in milk must result from the fish oil given between 2 and 4 months earlier; the probable site of their storage is maternal adipose tissue.

The particularly high content of long chain n–3 polyunsaturated fatty acids, in the colostrum of F sows, suggests that there may be a mechanism by which they are selectively stored in adipose tissue for release during lactation. Preferential storage of n–3 fatty acids has previously been observed in rats and rabbits (Lin and Connor, 1990; Raclot and Groscolas, 1994; Raclot, 2003).

The concentration of IgG was highest in the colostrum and rapidly decreased as lactation progressed; this is consistent with previous reports (Klobasa et al., 1987), and reflects the changing ability of the piglet to utilize these antibodies. IgG concentrations were higher in the colostrum of P G2 and S G1 sows, in the present study. The mechanism behind these changes is not fully understood but observed differences may be due to increased maternal intakes of β-carotene (via palm oil; Lietz et al., 2001; Edem, 2002) and vitamin E (via sunflower; Sheppard and Pennington, 1993; Edem, 2002; Duran, 2002). In the current study IgM concentrations were also higher in the colostrum of S G1 sows, whilst IgA concentrations were observed to be higher in the milk of P G1, F G1 and F G2 animals but the mechanisms behind this remains to be fully elucidated.

5. Conclusions

The fatty acid profile of sow diets, during either the first or second half of gestation, appears to be of more importance than the energy content of the diet *per se*. The type and timing of dietary supplementation had a pronounced influence on the backfat deposition during pregnancy and fat mobilization during the lactation period. The provision of extra energy during G1, and to a lesser extent in G2, resulted in increased fat deposition during the period of supplementation. As with the E G1 diet, the inclusion of S oil during G1 improved sow condition throughout the whole of pregnancy, whilst the F oil promoted greater mobilization of body reserves during lactation. E G2 and P G2 diets resulted in reduced body condition compared to OG2, S G2 and F G2 diets, suggesting that diets with longer chain fatty acids may be more beneficial during G2. Maternal diet during gestation influenced the fatty acid profile or milk; in particular the proportions of long chain n−3 polyunsaturated fatty acids were greater in the lacteal secretions of F sows. This demonstrates the important role of maternal adipose tissue as a store of biologically important fatty acids, for mobilization during late gestation and lactation when they will be of most benefit to their offspring. Oil supplements during G1 also had a profound effect on immunoglobulin secretion during lactation. Further work is required to examine the combined effects of dietary supplementation with oils during G1 and G2 to ascertain optimal nutrition for the reproducing pig.

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