Effect of type and timing of oil supplements to sows during pregnancy on the growth performance and endocrine profile of low and normal birth weight offspring

John Laws, Jennie C. Litten, Alison Laws, Ian J. Lean, Peter F. Dodds and Lynne Clarke*

Department of Agricultural Sciences, Faculty of Natural Sciences, Imperial College London, Wye, Ashford, Kent TN25 5AH, UK

(Received 24 September 2007 – Revised 1 April 2008 – Accepted 7 April 2008 – First published online 2 September 2008)

Eighty-eight multiparous sows were used to evaluate whether type and timing of oil supplementation during gestation influences the incidence of low birth weight (LBW). Sows were allocated (eight per treatment) commercial sow pellets (3 kg/d; control diet) or an experimental diet consisting of control diet plus 10% extra energy in the form of excess pellets, palm oil, olive oil (OO), sunflower oil (SO) or fish oil; experimental diets were fed during either the first half (G1) or second half (G2) of gestation. Growth performance and endocrine profile of LBW (<1.09 kg) and normal birth weight (NBW; 1.46–1.64 kg) offspring were compared. Maternal dietary supplementation altered the distribution curve for piglet birth weight. SOG1 sows had a greater proportion of LBW piglets (P<0.05), whilst it was reduced in the OOG1 group (P<0.05). Growth rate of LBW piglets was lower compared with their NBW siblings (P<0.05) when dietary supplementation was offered in G2 but were similar for G1. At birth, LBW offspring of supplemented animals possessed more fat compared with the control group (P<0.05); LBW offspring of control animals exhibited a more rapid decline in fat free mass/kg prior to weaning. Plasma metabolites and insulin concentrations were influenced by maternal diet and birth weight. In conclusion, maternal dietary supplementation altered the distribution of piglet birth weights and improved the energy status of LBW piglets. Supplementation with MUFA during G1 reduced the incidence of LBW, whereas PUFA had the reverse effect.

Birth weight: Piglet growth: Body composition: Oil

In the UK, mortality of live born piglets is approximately 11%1 with over 50% of deaths occurring within 3 d of birth2–3. Many factors influence piglet mortality4, but one of the main causes of mortality is cold stress due to lack of energy reserves at birth4–7.

There is a plethora of data demonstrating that fat supplementation of sow diets during late gestation increases liver glycogen and body fat reserves in their offspring at birth8–10. Furthermore, it is well established that fat supplementation of sow diets during late gestation improves the growth performance and survival of their offspring11–13, this improvement is most pronounced in low birth weight (LBW) piglets14–17. There is also some evidence to suggest that the fatty acid profile of the supplement is of importance; Jean & Chiang18, for example, observed that medium-chain TAG and coconut oil were more effective than soya oil at reducing piglet morbidity and mortality.

Substantial evidence now exists to support Barker’s fetal origins hypothesis, which suggests that fetal undernutrition is associated with an altered fetal and placental endocrine environment, thereby linking fetal undernutrition with abnormal organ structure, function and disease in later life19,20. Fetal nutrition is dependent upon the transfer of nutrients across the placenta. Manipulation of placental development by maternal nutrition, during early–mid-gestation, is therefore likely to have direct consequences for fetal nutrition and development. Our previous work21 suggests that energy supplementation and the fatty acid profile of the maternal diet during the first half of gestation influence the growth and development of piglets both in utero and during postnatal life. Since LBW has previously been linked to placental insufficiency22 and placental development occurs during the first half of gestation23,24, it seems probable that the maternal diet during early gestation will also have some influence upon the incidence of LBW. It was the aim of the present study to determine the consequences of altering the fatty acid profile of a 10% dietary supplement, offered either during the first half or second half of pregnancy on the incidence of LBW piglets and their subsequent growth and endocrine profiles.

Methods

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.

Abbreviations: DE, digestible energy; FFM, fat-free mass; G1 and G2, first and second half of gestation, respectively, used for the experimental diets; IGF, insulin-like growth factor; LBW, low birth weight; NBW, normal birth weight.

* Corresponding author: Lynne Clarke, fax +44 20 7594 2640, email lynne.clark@imperial.ac.uk
and were licensed by the Home Office (UK). At all stages of life, animals were kept within the guidelines set out by the Department for Environment, Food and Rural Affairs (25).

Initially birth weight data for the litters of a control population of fifty multiparous sows, of the same genetic line as those used in this experiment, were pooled and a probability plot of their weight constructed in MINITAB (Fig. 1). Piglets with a birth weight under the 10th percentile for the population were considered to be LBW, while those between the 40th and 60th percentiles were considered to be of normal birth weight (NBW). In the control population, the LBW animals weighed less than 1.09 kg and NBW animals were between 1.46 kg and 1.64 kg. These weight boundaries were used to classify LBW and NBW piglets within the litters of experimental animals at birth.

Eighty-eight sows of a commercial genotype (25 % Meishan; 12.5 % Duroc; 62.5 % Large White × Landrace) were entered into the study after weaning and prior to service. Sows were categorized by parity before being randomly assigned to one of eleven 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, Southburn, Driffield, East Yorkshire, UK). The control diet consisted of the standard diet (3 kg/d) (ABN HE sow pellets; 13.1 MJ/kg metabolizable energy; 12.7 % protein; 6.5 % fat; ABN, Peterborough, Cambridgeshire, UK), and was fed throughout gestation. Experimental diets were as the control ration plus 10% (3.93 MJ/d) extra energy derived from either: (i) excess pellets (E); (ii) palm oil (PO); (iii) olive oil (OO); (iv) sunflower oil (SO); (v) fish (salmon) oil (FO). Experimental diets were fed during either the first half (G1; from days 0 to 60 of gestation) or second half (G2; from day 60 of gestation until term at approximately day 115 of gestation). During the period when supplemented diets were not received, experimental sows were offered the standard diet (3 kg/d) as for the control group. Fatty acid profiles of the diets were determined by GLC and can be seen in Table 1. All diets, with the exception of the control ration, were isoenergetic. Between farrowing and weaning (21–28 d post partum) sows were offered a standard lactation ration (6–9 kg) (ABN supreme lactation pellets; 14.1 MJ/kg metabolizable energy; 18 % protein; 7.2 % oil; ABN). Piglets had ad libitum access to creep feed (Primary Select; 17.35 MJ/kg digestible energy (DE), 23.5 % protein; Primary Diets Ltd., Melmerby, Ripon, North Yorkshire, UK) from day 14 of life.

All piglets were weaned at 21–28 d, provided they weighed more than 6.5 kg. Piglets that failed to reach 6.5 kg by this age were fostered on to another sow for a further 1–2 weeks. Post-weaning management of all pigs was according to the standard procedures of the College Pig Unit. Maximum group sizes were thirty animals per pen (total area 5.45 m²; minimum area per pig 0.18 m²); animals were grouped according to sex and weight and food was available ad libitum throughout post weaning (Primary Benefit; 15.6 MJ/kg DE, 22.8 % protein; Primary Diets Ltd.), growing (Deltagrow 10 salocin pellets; 15 MJ/kg DE; 20.9 % protein; ABN) and fattening (Deltagrow 25 salocin pellets; 14 MJ/kg DE; 17.8 % protein; ABN) periods.

**Piglet growth and composition**

Piglet growth performance was observed throughout the neonatal period; body weight, crown to rump length and body composition using a total body electrical conductivity analysing system (TOBEC, Model-SA3000 EMSCAN/TOBEC, SA-3203; EM-Scan Inc., Springfield, IL, USA) were recorded on days 0, 7, 14 and 21 of life. Piglet growth rate was calculated by regression analysis of piglet weight against time. Piglet fat free mass (FFM) per kg was calculated using the equation,

\[
\text{FFM(}\text{arbitrary units}) = \sqrt[4]{\frac{\text{TOBEC reading} \times \text{crowntorump length}}{\text{Piglet weight}}}\]

At commercial end-point (80 (SE 5) kg) back fat and eye muscle depth were measured, at the P2 position, using ultrasound (Aloka-echo camera 550-500; Aloka Ltd., Osaka, Japan).

**Milk intake**

Individual piglet milk intake was assessed using an adaptation of the weigh-suckle-weigh method, described by Sinclair et al. (26). Intake was assessed on days 3, 7, 14 and 21 of lactation. The first recording of milk intake was conducted on day 3 to ensure that we were not measuring colostral yield. Natural suckling was allowed, and the inter-suckling interval recorded.
Piglets were observed throughout and weighed individually 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 intake per suckling:

\[
\text{Milk yield (kg)} = W + U + D + M,
\]

where \(W\), piglet 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. (27):

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

where \(U\), number of urinations; \(W^{0.75}\), piglet metabolic weight.

A 10 g loss was allowed per defecation (26). An estimate of metabolic loss was calculated using the equation described by Noblet & Etienne (28):

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

**Statistical analysis**

One of the control sows became unwell shortly after farrowing and her piglets had to be weaned early. All data from this animal and her offspring were removed prior to analysis. Data were analysed with general linear model ANOVA using MINITAB version 13.1 computer software (Minitab Ltd., Coventry, UK). The main effect included in the model was dietary treatment; parity was included as a covariate. Data were adjusted for litter size or number of piglets reared as appropriate. When treatment effects were significantly different, pair-wise comparisons of means were conducted using Tukey’s test. Probability values of less than 0.05 were considered to be statistically significant.

**Blood sampling**

Blood samples were obtained from the piglets on days 3 and 21 of life. Blood was put into an EDTA tube and immediately placed on ice and after centrifugation at 1600 g the plasma was removed and stored at −20°C until further analysis. Plasma concentrations of leptin (Biogenesis XL-85; Biogenesis Ltd., Poole, Dorset, UK), insulin (ICN 07-260102; ICN Pharmaceuticals, New York, USA), insulin-like growth factor (IGF)-1; DSL-2800; Diagnostic Systems Laboratories Inc., Webster, Tx, USA) and thyroid hormones (triiodothyronine (ICN 07B74102; ICN Pharmaceuticals) and thyroxine (ICN 06B256447; ICN Pharmaceuticals) were determined by RIA using commercially available kits.

Glucose (GL 366; Randox Laboratories Ltd., Crumlin, Antrim, UK), TAG (TR 148; Randox Laboratories Ltd.) and NEFA (944-75 409; Wako Chemicals GmbH, Neuss, Germany) were measured enzymatically. A reference sample was included in every assay and the intra- and inter-assay CV were below 5 and 10% respectively.

**Results**

After adjustment for both parity and litter size, the type and timing of maternal supplementation influenced the incidence of LBW piglets (\(P<0.05\); Table 2). The percentage of piglets born alive that could be categorized as LBW was highest in the
litters of SOG1 sows, and lowest in those of OOG1 animals (P<0.05; Table 2). The incidence of NBW piglets was similar irrespective of sow parity, litter size and type or timing of maternal supplementation (Table 2).

Comparison of the distribution curve for piglet birth weights, within each treatment, gives some insight into the effect of the different dietary supplements (Fig. 2). The OOG1 diet resulted in a shift of the distribution curve to the right (i.e. an increase in the average piglet birth weight, while deviation from the mean was similar to that observed in other groups), meaning that fewer than 10% of the OOG1 piglets fell below the 10th percentile weight (1.09 kg) derived from the control population. In contrast, the SOG1 diet resulted in a shift of the distribution curve to the left and a slight skewing towards the left (i.e. a slight decrease in average birth weight, with a tendency for an increased proportion of LBW piglets), meaning that more than 10% of piglets fell below the 10th percentile weight (1.09 kg) for the control population. Interestingly, the POG1 diet appeared to result in a shift of the distribution curve to the left and to result in a wider shallower curve (i.e. a slight decrease in average birth weight and a greater proportion of piglets at either end of the weight range); these differences appear to be largely attributable to the increased litter size observed on this treatment.

G2 supplementation with OO or SO diets resulted in a shift of the distribution curve for piglet birth weight to the left (i.e. a decrease in the average birth weight). In contrast supplementation with the E diet during G2 resulted in a shift of the peak to the right as a result of a skewing towards heavier piglets (i.e. an increase in the average birth weight of piglets, with a more gradual decline in frequency at the heavier end of the range).

By 21 d of age, after correction for maternal parity and litter size, the surviving LBW offspring of FOG1 and FOG2 sows were still lighter than their NBW siblings (P<0.05; Table 3). A similar pattern was observed in the weights of the offspring of SOG2 sows (P<0.05; Table 3) on day 21 of life. At 21 d of age the offspring of SOG2 sows were lighter than those of FOG1 or EG2 animals (P<0.001 and P<0.05 respectively; Table 3).

### Table 2. Effect of timing and type of maternal supplement on the incidence of low birth weight (LBW; <1.09 kg) and normal birth weight (NBW; 1.46–1.64 kg) piglets†

<table>
<thead>
<tr>
<th></th>
<th>Total LBW</th>
<th>Total NBW</th>
<th>Total piglets</th>
<th>% LBW</th>
<th>% NBW</th>
<th>Mean ± SEM</th>
<th>Adjusted % LBW‡</th>
<th>Adjusted % NBW‡</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Control</strong></td>
<td>6</td>
<td>33</td>
<td>72</td>
<td>8</td>
<td>46</td>
<td>9 ± 4</td>
<td>28 ± 5</td>
<td>5 ± 4</td>
</tr>
<tr>
<td>G1 (0–60 d)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>E</td>
<td>12</td>
<td>44</td>
<td>107</td>
<td>11</td>
<td>41</td>
<td>11 ± 3</td>
<td>20 ± 4</td>
<td>4 ± 2</td>
</tr>
<tr>
<td>PO</td>
<td>19</td>
<td>40</td>
<td>120</td>
<td>16</td>
<td>33</td>
<td>13 ± 3</td>
<td>23 ± 4</td>
<td>4 ± 2</td>
</tr>
<tr>
<td>OO</td>
<td>4</td>
<td>39</td>
<td>91</td>
<td>4</td>
<td>43</td>
<td>5 ± 3</td>
<td>23 ± 4</td>
<td>4 ± 2</td>
</tr>
<tr>
<td>SO</td>
<td>8</td>
<td>32</td>
<td>95</td>
<td>8</td>
<td>34</td>
<td>20 ± 4</td>
<td>16 ± 4</td>
<td>4 ± 2</td>
</tr>
<tr>
<td>FO</td>
<td>18</td>
<td>45</td>
<td>102</td>
<td>12</td>
<td>36</td>
<td>9 ± 3</td>
<td>21 ± 4</td>
<td>4 ± 2</td>
</tr>
<tr>
<td>G2 (60 d–term)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>E</td>
<td>12</td>
<td>37</td>
<td>102</td>
<td>12</td>
<td>36</td>
<td>9 ± 3</td>
<td>21 ± 4</td>
<td>4 ± 2</td>
</tr>
<tr>
<td>PO</td>
<td>10</td>
<td>47</td>
<td>93</td>
<td>11</td>
<td>51</td>
<td>10 ± 3</td>
<td>25 ± 4</td>
<td>4 ± 2</td>
</tr>
<tr>
<td>OO</td>
<td>9</td>
<td>40</td>
<td>87</td>
<td>10</td>
<td>46</td>
<td>17 ± 3</td>
<td>27 ± 5</td>
<td>4 ± 2</td>
</tr>
<tr>
<td>SO</td>
<td>13</td>
<td>35</td>
<td>95</td>
<td>14</td>
<td>37</td>
<td>16 ± 3</td>
<td>22 ± 4</td>
<td>4 ± 2</td>
</tr>
<tr>
<td>FO</td>
<td>15</td>
<td>48</td>
<td>96</td>
<td>16</td>
<td>50</td>
<td>10 ± 4</td>
<td>20 ± 4</td>
<td>4 ± 2</td>
</tr>
</tbody>
</table>

E, excess; PO, palm oil; OO, olive oil; SO, sunflower oil; FO, fish oil; G1, first half of gestation; G2, second half of gestation.

† For details of diets and procedures, see Methods.

‡ Data for number of piglets born alive were analysed in Minitab using ANOVA general linear model (parity (P<0.05) and litter size (P<0.001) were analysed as covariates).

---

By 21 d of age, after correction for maternal parity and litter size, the surviving LBW offspring of FOG1 and FOG2 sows were still lighter than their NBW siblings (P<0.05; Table 2). The incidence of NBW piglets was similar irrespective of sow parity, litter size and type or timing of maternal supplementation (Table 2).

Comparison of the distribution curve for piglet birth weights, within each treatment, gives some insight into the effect of the different dietary supplements (Fig. 2). The OOG1 diet resulted in a shift of the distribution curve to the right (i.e. an increase in the average piglet birth weight, while deviation from the mean was similar to that observed in other groups), meaning that fewer than 10% of the OOG1 piglets fell below the 10th percentile weight (1.09 kg) derived from the control population. In contrast, the SOG1 diet resulted in a shift of the distribution curve to the left and a slight skewing towards the left (i.e. a slight decrease in average birth weight, with a tendency for an increased proportion of LBW piglets), meaning that more than 10% of piglets fell below the 10th percentile weight (1.09 kg) for the control population. Interestingly, the POG1 diet appeared to result in a shift of the distribution curve to the left and to result in a wider shallower curve (i.e. a slight decrease in average birth weight and a greater proportion of piglets at either end of the weight range); these differences appear to be largely attributable to the increased litter size observed on this treatment.

G2 supplementation with OO or SO diets resulted in a shift of the distribution curve for piglet birth weight to the left (i.e. a decrease in the average birth weight). In contrast supplementation with the E diet during G2 resulted in a shift of the peak to the right as a result of a skewing towards heavier piglets (i.e. an increase in the average birth weight of piglets, with a more gradual decline in frequency at the heavier end of the range).

By 21 d of age, after correction for maternal parity and litter size, the surviving LBW offspring of FOG1 and FOG2 sows were still lighter than their NBW siblings (P<0.05; Table 3). A similar pattern was observed in the weights of the offspring of SOG2 sows (P<0.05; Table 3) on day 21 of life. At 21 d of age the offspring of SOG2 sows were lighter than those of FOG1 or EG2 animals (P<0.001 and P<0.05 respectively; Table 3).

---

![Fig. 2. Effect of type of maternal energy supplement on the distribution of piglet birth weights when supplementation occurs during (a) the first 60 d of gestation (G1) or (b) from 60 d of gestation until term (approximately 115 d) (G2).](https://www.cambridge.org/core/terms)

**Downloaded from** <https://www.cambridge.org/core> **IP address:** 54.191.40.80, on 08 Sep 2017 at 14:54:08, subject to the Cambridge Core terms of use, available at <https://www.cambridge.org/core/terms>
Table 3. Effect of timing and type of maternal supplement and piglet birth weight category on weight change during the first 21 d of life†

(Least squares means with their standard errors)

<table>
<thead>
<tr>
<th>Birth weight (kg)</th>
<th>Weight at 21 d of age (kg)</th>
<th>Neonatal growth rate (kg/d)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>LBW</td>
<td>NBW</td>
</tr>
<tr>
<td></td>
<td>Mean SEM</td>
<td>Mean SEM</td>
</tr>
<tr>
<td>G1 (0–60 d)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Con</td>
<td>0.93a 0.05 1.61a 0.02</td>
<td>5.76 0.56 7.17 0.24</td>
</tr>
<tr>
<td>E</td>
<td>0.94a 0.04 1.65a 0.02</td>
<td>4.61b 0.51 7.28b 0.20</td>
</tr>
<tr>
<td>PO</td>
<td>0.92a 0.03 1.62a 0.02</td>
<td>5.56 0.04 5.56 0.36</td>
</tr>
<tr>
<td>OO</td>
<td>1.02a 0.03 1.64a 0.02</td>
<td>6.37 0.62 7.11 0.02</td>
</tr>
<tr>
<td>SO</td>
<td>0.97a 0.03 1.65a 0.02</td>
<td>5.44 0.35 5.44 0.35</td>
</tr>
<tr>
<td>FO</td>
<td>0.95a 0.03 1.63a 0.02</td>
<td>5.13b 0.47 7.85Ab 0.21</td>
</tr>
<tr>
<td>G2 (60 d–term)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>E</td>
<td>0.96a 0.04 1.62a 0.02</td>
<td>5.23b 0.50 7.76bb 0.22</td>
</tr>
<tr>
<td>PO</td>
<td>0.98a 0.04 1.64a 0.02</td>
<td>5.83 0.51 7.25 0.20</td>
</tr>
<tr>
<td>OO</td>
<td>0.94a 0.03 1.61a 0.02</td>
<td>5.91 0.51 7.26 0.21</td>
</tr>
<tr>
<td>SO</td>
<td>0.94a 0.03 1.62a 0.02</td>
<td>5.19b 0.34 5.78Ab 0.22</td>
</tr>
<tr>
<td>FO</td>
<td>0.90a 0.04 1.67a 0.02</td>
<td>5.05ab 0.51 6.94b 0.19</td>
</tr>
</tbody>
</table>

LBW, low birth weight; NBW, normal birth weight; G1, first half of gestation; G2, second half of gestation; Con, control; E, excess; PO, palm oil; OO, olive oil; SO, sunflower oil; FO, fish oil.

a,b Mean values within a row with unlike superscript letters were significantly different (P<0.05).

Data were analysed in Minitab using ANOVA general linear model (parity, sex and litter size) were analysed as covariates.

† For details of diets and procedures, see Methods.

Table 4. Effect of timing and type of maternal supplement and piglet birth weight category on change in fat free mass (FFM) per kg body weight, during the first 21 d of life†

(Least squares means with their standard errors)

<table>
<thead>
<tr>
<th>FFM/kg at birth (arbitrary units)</th>
<th>FFM/kg at 21 d of age (arbitrary units)</th>
<th>Decline in FFM/kg per d</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>LBW</td>
<td>NBW</td>
</tr>
<tr>
<td></td>
<td>Mean SEM</td>
<td>Mean SEM</td>
</tr>
<tr>
<td>G1 (0–60 d)</td>
<td>Con 111.60Ab 3.3 78.25a 1.82</td>
<td>62.04 2.92 61.63 1.55</td>
</tr>
<tr>
<td>E</td>
<td>88.57a 2.41 80.46 1.74</td>
<td>63.16 2.54 58.78 1.37</td>
</tr>
<tr>
<td>PO</td>
<td>81.89a 3.05 82.70 2.29</td>
<td>59.00 1.66 58.81 1.22</td>
</tr>
<tr>
<td>OO</td>
<td>84.83a 3.97 81.83 1.75</td>
<td>62.56 2.84 59.84 1.39</td>
</tr>
<tr>
<td>SO</td>
<td>89.34a 2.12 78.28 2.06</td>
<td>59.74 1.74 60.45 1.68</td>
</tr>
<tr>
<td>FO</td>
<td>84.28a 2.52 80.84 1.70</td>
<td>63.96 2.17 56.17 1.46b</td>
</tr>
<tr>
<td>G2 (60 d–term)</td>
<td>Con 91.25a 2.96 78.42 1.90</td>
<td>61.21 2.83 57.80 1.39b</td>
</tr>
<tr>
<td>E</td>
<td>81.96a 2.65 82.50 1.64</td>
<td>62.05 2.34 58.70 1.26</td>
</tr>
<tr>
<td>PO</td>
<td>84.46a 2.31 85.52 1.77</td>
<td>63.43 2.02 59.41 1.38</td>
</tr>
<tr>
<td>SO</td>
<td>82.48a 2.11 78.92 1.73</td>
<td>64.06 1.64 65.20bc 1.34</td>
</tr>
<tr>
<td>FO</td>
<td>81.90a 2.66 81.99 1.89</td>
<td>61.68 2.14 60.74 2.92</td>
</tr>
</tbody>
</table>

G1, first half of gestation; G2, second half of gestation; LBW, low birth weight; NBW, normal birth weight; Con, control; E, excess; PO, palm oil; OO, olive oil; SO, sunflower oil; FO, fish oil.

Mean values within a row with unlike superscript letters were significantly different (P<0.05).

For details of diets and procedures, see Methods.
did not reach significance, however, due to the wide variation in milk intake between LBW piglets.

At commercial end-point (determined by weight), there were no significant differences in pig age, weight or body composition irrespective of birth weight category or maternal diet. Similarly, there was no difference in pig growth rate from birth to slaughter (Table 6).

There was a trend \((P<0.1)\) for plasma glucose concentration to be lower on day 3 of life in LBW piglets \((6.12 (SE 0.66) \text{ mM})\) compared with NBW siblings \((8.12 (SE 0.52))\) and LBW animals in the other treatment groups on both days 3 and 21 of postnatal life. Plasma glucose concentrations tended to be higher on day 3 in those offspring in the G2 group \((P<0.05)\) compared with G1 piglets. In G2, LBW piglets \((6.12 (SE 0.66) \text{ mM})\) born to SOG1 sows had lower plasma glucose \((P<0.05)\) than their NBW siblings \((8.12 (SE 0.52))\) and LBW animals in the other groups on both days 3 and 21 of postnatal life.

On day 3 of age, plasma TAG and NEFA were higher \((P<0.001)\) in low \((\text{TAG } 2.96 (SE 0.32) \text{ mM}; \text{NEFA } 3.04 (SE 0.19) \text{ mM})\) compared with normal \((\text{TAG } 1.69 (SE 0.21) \text{ mM}; \text{NEFA } 0.90 (SE 0.11) \text{ mM})\) birth weight piglets born to PO-supplemented sows in G1 and all other treatment groups. These differences were no longer apparent by day 21 as concentrations had decreased \((P<0.001)\) over the neonatal period. Plasma TAG and NEFA were generally higher in LBW offspring born in G1 compared with G2 \((P<0.001)\) on day 3 but this observation was less marked with regard to the NBW group.

NBW \((18.15 (SE 2.05) \text{ \(\mu\)g/l/U/ml})\) piglets born to EO sows exhibited elevated \((P<0.001)\) concentrations of plasma insulin compared with their LBW siblings \((8.42 (SE 2.29) \text{ \(\mu\)g/l/U/ml})\) and all other treatment groups; these differences were not apparent by day 21 of age. LBW FO animals \((1.25 (SE 0.22) \text{ \(\mu\)g/l/U/ml})\) had significantly higher \((P<0.05)\) circulating insulin compared with NBW piglets \((0.87 (SE 0.14) \text{ \(\mu\)g/l/U/ml})\) and those born to other fat-supplemented mothers in G2 \((P<0.001)\) but by day 21 plasma insulin was similar between groups. There was no effect of type or timing of maternal dietary supplementation in gestation or birth weight group on plasma leptin, IGF-1, thyroxine or triiodothyronine at any sampling point.

Discussion
It is well established that supplementation of the maternal diet during late gestation improves the growth performance and survival of LBW piglets\(^{14-17}\). This is particularly true with the addition of medium-chain TAG to the sow diet\(^{18,29}\). Furthermore Bee\(^{30}\) found that maternal energy consumption during early gestation determined the ratio of primary to secondary muscle fibres, while birth weight affected muscle fibre area. Both the ratio of primary to secondary muscle fibres and muscle fibre area are known to have consequences for growth performance and carcass composition\(^{31-33}\). In the present study, both the timing and type of energy supplementation were found to be of importance, with regard to the incidence of LBW piglets, although the body composition of LBW piglets appeared to be improved irrespective of the type or timing of energy supplementation.

The greatest incidence of LBW piglets was observed in the litters of SOG1 sows, in contrast with those of OOG1 animals, which appeared to have a reduced incidence of LBW piglets. The differences observed between these two supplements are likely to have been due to the fatty acid profile of the diet, since parity, total litter size, number of piglets born alive and gestation length were similar for both groups\(^{34}\). A possible explanation for the differences observed is that gestational gain in back fat thickness at the P2 position was greater in the SOG1 animals, than in OOG1 sows\(^{34}\). This suggests that a greater proportion of the supplement was partitioned towards maternal reserves rather than to the growth of the products of conception in SOG1 sows. During the first half of gestation, energy directed towards growth of the products of conception could be expected to improve placental development\(^{24}\) and may provide an explanation for the decreased occurrence of LBW piglets in the litters of OOG1 animals. It is known...
Table 6. Effect of timing and type of maternal supplement and piglet birth weight category on performance at commercial end point†

<table>
<thead>
<tr>
<th></th>
<th>Age (d)</th>
<th>Weight (kg)</th>
<th>P2 back fat depth (mm)</th>
<th>P2 eye muscle thickness (mm)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>LBW</td>
<td>NBW</td>
<td>LBW</td>
<td>NBW</td>
</tr>
<tr>
<td>Con</td>
<td>Mean</td>
<td>SEM</td>
<td>Mean</td>
<td>SEM</td>
</tr>
<tr>
<td>G1 (0–60 d)</td>
<td>Mean</td>
<td>SEM</td>
<td>Mean</td>
<td>SEM</td>
</tr>
<tr>
<td>E</td>
<td>Mean</td>
<td>SEM</td>
<td>Mean</td>
<td>SEM</td>
</tr>
<tr>
<td>PO</td>
<td>Mean</td>
<td>SEM</td>
<td>Mean</td>
<td>SEM</td>
</tr>
<tr>
<td>OO</td>
<td>Mean</td>
<td>SEM</td>
<td>Mean</td>
<td>SEM</td>
</tr>
<tr>
<td>FO</td>
<td>Mean</td>
<td>SEM</td>
<td>Mean</td>
<td>SEM</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Con</td>
<td>Mean</td>
<td>SEM</td>
<td>Mean</td>
<td>SEM</td>
</tr>
<tr>
<td>E</td>
<td>Mean</td>
<td>SEM</td>
<td>Mean</td>
<td>SEM</td>
</tr>
<tr>
<td>PO</td>
<td>Mean</td>
<td>SEM</td>
<td>Mean</td>
<td>SEM</td>
</tr>
<tr>
<td>OO</td>
<td>Mean</td>
<td>SEM</td>
<td>Mean</td>
<td>SEM</td>
</tr>
<tr>
<td>FO</td>
<td>Mean</td>
<td>SEM</td>
<td>Mean</td>
<td>SEM</td>
</tr>
</tbody>
</table>

|                      | Con           | Mean           | SEM           | Mean          | SEM           |
|                      | E             | Mean           | SEM           | Mean          | SEM           |
|                      | PO            | Mean           | SEM           | Mean          | SEM           |
|                      | OO            | Mean           | SEM           | Mean          | SEM           |
|                      | FO            | Mean           | SEM           | Mean          | SEM           |

- LBW: low birth weight piglet; NBW: normal birth weight piglet; G1: first half of gestation; G2: second half of gestation; Con: control; E: excess; PO: palm oil; OO: olive oil; FO: fish oil.

* Data were analysed in Minitab using ANOVA general linear model (parity, sex and litter size were analysed as covariates).

† For details of diets and procedures, see Methods.

That fetal growth is directly dependent on the supply of nutrients across the placenta and will therefore be influenced by placental development and efficiency. Analysis of distribution curves of piglet birth weights for the different treatment groups revealed that the mean birth weight of OOG1 piglets is increased. Standard deviation from the mean was similar for OOG1 to that calculated for other supplements, suggesting that more nutrients were available in utero to the offspring of OOG1 sows. It is speculated that improved nutrient availability may be as a result of improved placental development during G1.

It is well established that the growth of small piglets during the pre-weaning period may be improved as a consequence of fat supplementation of their dams, suggesting that they can compete more successfully with their litter mates. In the present series of experiments, the growth rate of LBW piglets was similar, irrespective of type or timing of maternal energy supplementation. This is surprising since the average growth rate, from whole litter data, was influenced by the type of maternal energy supplementation during G1. It is speculated that the low numbers of LBW piglets, compared with those available from the whole litter, together with other factors affecting piglet growth (litter size, thermal stress, etc.) may have masked the effects of maternal supplementation.

The growth of LBW piglets is known to be slower than that of heavier piglets from the same litter. In the current experiment, comparison of growth rates of LBW and NBW piglets within treatment groups revealed less of a difference in growth rate in the offspring of sows supplemented with extra energy during G1; this is interesting because placental development occurs during the first 60 d of gestation. It is probable that the reduced growth performance of LBW piglets, born to sows supplemented during G2, may be primarily due to compromised fetal development as a result of placental insufficiency. Other studies have also linked reduced fetal growth to placental insufficiency. It is well established that the growth of small piglets during pregnancy is compromised due to placental insufficiency. In the current study, it was observed that the LBW piglets of control sows had reduced body fat (as indicated by their higher FFM per kg), compared with their NBW siblings and that the addition of extra energy to the maternal diet resulted in increased body fat of LBW piglets, irrespective of the type or timing of supplementation. The present results may provide an explanation for the increased survival of LBW piglets reported in previous studies with maternal supplementation, since nutritional status...
and energy balance during the immediate neonatal period is known to be of importance with regard to survival\(^{(47,48)}\).

The LBW offspring of control sows had an increased rate of fat deposition during the pre-weaning period, compared with those of supplemented sows and their NBW siblings. By 21 d of age, surviving LBW offspring of control sows had a similar quantity of FFM per kg body weight to that exhibited by low and NBW piglets in other groups; their body weight was also similar to that for LBW piglets from other treatment groups. Rapid fat deposition also suggests that growth was less efficient than in either NBW siblings or LBW offspring from other groups\(^{(35,39)}\). Since the weight gain of all LBW piglets was similar during the first 21 d of life, but the fat deposition of those born to control sows was greater, their energy intake would be expected to be greater over this period. In the current study, milk energy yield per kg was similar throughout lactation irrespective of type or timing of maternal energy supplementation\(^{(34)}\). Similarly, the milk consumption of LBW piglets was similar irrespective of type or timing of energy supplementation, although there was more variation in milk consumption of control animals than those in other groups. An alternative explanation for the rapid fat deposition observed in LBW offspring of control sows is that they may have consumed more creep feed than those from other groups, although this seems unlikely since the consumption of creep feed during the first 3 weeks of life is usually very low\(^{(49)}\).

Previously, LBW piglets have been observed to reach a lower mature body weight, when compared with heavier litter mates\(^{(40)}\). Consequently, there is an inverse relationship between birth weight and time taken to reach commercial slaughter weight\(^{(41)}\). Similarly, average daily gain has been observed to be lower in LBW pigs prior to weaning and from weaning to 70 d of age\(^{(32)}\). In the present study, there was no significant difference in age of pigs at slaughter, irrespective of type or timing of maternal supplementation or piglet birth weight. Although not significant, age at slaughter was generally slightly higher for LBW animals, but the reverse was true in the case of those animals whose dams had received EG2, FOG2 or POG1 diets; it is not clear whether these results demonstrate improved performance of some LBW piglets as a result of catch-up growth\(^{(50)}\).

Previously it has been found that LBW piglets have lower feed conversion efficiency, due to their lower potential for lean deposition, compared with NBW pigs\(^{(32,39)}\). In the current studies, no difference was observed in the back fat depth or eye muscle thickness at the P2 position at slaughter. This is in agreement with the findings of England\(^{(51)}\) and Hegarty & Allen\(^{(52)}\), who found no difference in the carcass composition of low and NBW animals at similar slaughter weights. Recently, it has been suggested that the feed conversion efficiency of small pigs (0.9–1.3 kg) is similar to that observed for larger pigs, but that very small pigs (800–900 g) have a 30% lower feed conversion efficiency\(^{(41)}\); this has previously been hinted at by Powell & Aberle\(^{(59)}\). In the present study, average birth weight of LBW piglets was above 0.9 kg, irrespective of maternal diet, and may provide an explanation for the lack of difference observed between birth weight groups.

In addition to those already discussed, there are a number of other factors that were not measured that influence pig growth. It is well established that social stressors influence pig performance\(^{(53–55)}\). On the College Unit, piglets are grouped by sex and weight at weaning into pens of thirty animals. By dividing pigs into groups in this way, it becomes possible for a LBW pig to become more dominant than would be possible if whole litters were transferred together. Similarly, a dominant animal, which gets grouped with the largest pigs weaned, may end up in a subordinate position. It is not well documented as to how such social situations would affect the feed consumption and efficiency of thermoregulation and growth in pigs from different birth weight groups.

A previous study by Boyd et al.\(^{(10)}\) demonstrated that the addition of fat to the maternal diet did not influence piglet plasma glucose at birth. However, other studies have reported that the addition of medium-chain TAG or saturated fat to the sows’ diet during late gestation cause an increase in glucose concentration of up to 16%\(^{(14,18)}\). The present data support these findings and are further modified by the timing of maternal supplementation as plasma glucose was higher in G1 compared with G2 offspring. Moreover, this mirrors the differences in maternal glucose towards the end of pregnancy. It is proposed that exposure to high concentrations of glucose \textit{in utero} (as a cause of glucose-sparing) alters the hypothalamic set-point of the offspring and thus they strive to maintain this elevated set-point after birth. By day 21 the differences in plasma glucose are no longer apparent, suggesting that the glucose set-point may be readjusted as the offspring adapts to postnatal life.

Several studies have reported that fat in the late gestation diet of sows has no effect on piglet plasma NEFA\(^{(10,56,57)}\) and TAG\(^{(56)}\) concentration. With the exception of the POG1 offspring, a similar scenario was demonstrated in the current investigation. The reasons why pigs born to POG1 sows exhibited elevated concentrations of NEFA and TAG remains to be fully established.

NBW piglets born to either G1 or G2 sows exhibited higher concentrations than their LBW counterparts and all offspring born to fat-supplemented mothers. The higher concentrations of insulin on day 3 of age may also provide one explanation for the lower plasma glucose observed in these pigs. There is great variation in the insulin results obtained in the current study, which may be due to the inability to starve the pigs prior to blood sampling as they had access to feed \textit{ad libitum}. Insulin concentrations are usually low before a feed and much higher after an intake of food. Since blood samples were not necessarily taken from the pigs at the same time interval after feeding, there will be a high degree of variation in the results. It is interesting to note that there was no difference in maternal plasma glucose between EG1 and EG2, which may in part explain why insulin concentrations are similar in these groups.

It is well documented that IGF-1 is an important regulator of postnatal growth and development and so, as expected, overall plasma IGF-1 concentrations were shown to increase from day 3 to day 21. Averette et al.\(^{(58)}\) has previously shown that IGF-1 is higher in the colostrums of sows fed fat and these authors proposed that this may be one explanation for the improved neonatal growth rate of their piglets. There is no evidence to support this hypothesis in the present study as differences in IGF-1 were not apparent, despite differences in growth between LBW and NBW piglets.

Thyroid hormones are known to influence both the physical and behavioural development of the newborn animal. Low plasma...
concentrations of thyroxine and triiodothyronine have been linked to LBW and poor growth performance in sheep but a similar situation was not observed in the present study.

Conclusions
Maternal dietary supplementation altered the distribution of piglet birth weights and improved the energy status of LBW piglets, irrespective of the fatty acid profile or timing of the supplement. Supplementation with MUFA during G1 appeared to have beneficial effects as it reduced the incidence of LBW, whereas offering a diet high in PUFA had the reverse effect.

Acknowledgements
The authors wish to acknowledge the financial support of the Commission of the European Communities (QLK1-2001-00138), PERILIP; this grant was awarded to P. F. D., L. C. and I. J. L. 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. The authors would also like to thank Emilio Herrera and Encarnacion Amusquivar (Universidad San Pablo-CEU, Madrid, Spain) for analysing the fatty acid profile of the experimental diets. The technical help of Kate Perkins and Kirsty Massey was also greatly appreciated. The investigation team are former members of the Animal Science Research Section of Imperial College London (Wye Campus). There are no conflicts of interest.

References


