Feed allowance and maternal backfat levels during gestation influence maternal cortisol levels, milk fat composition and offspring growth

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Abstract

The fetal and early postnatal environment can have a long-term influence on offspring growth. Using a pig model, we investigated the effects of maternal body condition (thin or fat) and maternal gestation feeding level (restricted, control or high) on maternal stress, milk composition, litter size, piglet birth weight and pre-weaning growth. A total of sixty-eight thin (backfat depth about 8 mm) and seventy-two fat (backfat depth about 12 mm) gilts were selected at about 22 weeks. This backfat difference was then accentuated nutritionally up to service at about 32 weeks. During gestation, individual gilts from within each group were randomly allocated to a gestation diet at the following feed allowances: 1·8 kg/d (restricted); 2·5 kg/d (control) and 3·5 kg/d (high) until day 90 of gestation. During gestation restricted gilts had higher levels of cortisol than high and control fed animals. Piglets born to fat gilts had higher average daily gain during the lactation period and higher weaning weights at day 28 than piglets born to thin gilts. Gilts on a high feed level had heavier piglets than those provided with restricted and control allocations. Fat gilts had less saturated fat in their milk at day 21 of lactation and higher unsaturated fat levels. No differences were found in the n-6:n-3 PUFA ratio in the milk between thin and fat gilts. In conclusion, maternal body condition influenced the daily weight gain of offspring up to weaning (day 28) and milk fat composition. Furthermore, maternal feed level during gestation alters maternal cortisol levels and milk fat composition.

Key words: Milk composition: Cortisol: Gestation feeding: Lactation: Fetal programming: Pigs

An adverse fetal environment can lead to permanent postnatal changes in the metabolism of the offspring with alterations to appetite regulation, fat deposition and muscle fibre composition. This predisposes the offspring to CVD, diabetes and obesity in later life. This phenomenon has been termed fetal programming. Previous studies in rodents have shown that offspring health and longevity are undermined when mothers are overfed or are nutrient or protein restricted during pregnancy. In addition, unique situations in human subjects such as the Dutch famine studied show that maternal undernutrition during gestation has important effects on the health of offspring in later life. Furthermore, there is evidence that not only feed level but also maternal body condition can affect offspring development. For example, in sheep, lambs born to obese mothers had increased adiposity compared with lambs born to normal-weight ewes. This has also been found in epidemiological studies in human subjects where babies born to obese women were more likely to be obese in childhood and adulthood. However, there is a dearth of literature on how the interaction between maternal feed levels during pregnancy and body condition could affect offspring development.

Abbreviations: DE, digestible energy; FAME, fatty acid methyl esters.

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Maternal endocrine status may make an impact on fetal development and programming. For example, levels of the stress hormone, cortisol, can cross the placental barrier. An exposure to cortisol in excess levels correlates with reduced birth weight and adverse outcomes in offspring such as hyperglycaemia in rats and hypertension in sheep. Low placental 11β-hydroxy-steroid dehydrogenase type 2 enzyme activity, which converts cortisol to the inert form cortisone, has been linked to lower birth weight, possibly due to an increased transplacental passage of active maternal glucocorticoids. Inhibition of placental 11β-hydroxy-steroid dehydrogenase type 2 increases glucocorticoid receptor mRNA expression of the amygala and increases anxiety in offspring. Excess cortisol levels during rapid brain growth in the guinea pig altered central corticosteroid receptor regulation. In addition, prenatal depression when urinary cortisol levels are high in women are associated with slower fetal growth rates and lower birth weight compared with offspring from non-depressed women with lower urinary cortisol levels. Therefore, stress can manifest itself through different kinds of influences, thus raising the question: is stress a compounding factor in undernutrition? Offspring growth may also be influenced by milk composition. For example, when sows were fed 8% added fat in the lactation diet compared with none, this changed the milk composition and increased litter weight gain from 57.9 kg in the control group to 68.7 kg in the experimental group. Furthermore, piglets reared by sows fed conjugated linoleic acid during pregnancy and lactation grew faster in the post-weaning period than piglets reared on sows fed linoleic acid during the same time period. It is known that fat and fatty acid concentrations in the milk of sows can be manipulated by dietary intervention during pregnancy and lactation. These levels are largely determined by the level and source of dietary fat. Furthermore, fatty acids have different functions: some regulate appetite, some mobilise fat tissue and influence energy metabolism. For example, feeding conjugated linoleic acid to sows during pregnancy and lactation altered the backfat and milk fatty acid composition, increasing fat metabolism in the tissue of sows. However, the sow’s body fat reserves can also influence the fat content of the sow’s milk. An imbalance in maternal micronutrients during pregnancy can lead to changes in milk composition and milk volume in rats. It is possible that the nutrient intake of sows during pregnancy and their body composition interact, causing differences in milk fat composition. The ratio of saturated:unsaturated fatty acids in the diet is also important in achieving an appropriate composition in developing tissue lipids. There is no conclusive evidence that high levels of saturated fats in the diet are linked directly to CVD, but there is strong evidence that vegetables and a Mediterranean diet, which is high in MUFA, can be protective against this. Furthermore, the ratio of ε-6,9-3 PUFA in human diets has changed dramatically over the last decade from a ratio of 1:1 to 15:1, and such changes are associated with CHD, hypertension and type 2 diabetes. Studies have also shown that components in the milk during early lactation might regulate growth and development and influence the programming of energy balance in later life.

In this study, using a porcine model, we combined both maternal nutrition (restricted, control (normal) or high) and body composition (thin or fat) during gestation to investigate how maternal backfat and feeding levels interact to influence offspring growth. We also investigated the effect of maternal body condition and feed intake during gestation on subsequent sow reproduction. We hypothesised that the effect on offspring growth would be compounded by maternal stress levels and milk composition.

Materials and methods

All experiments complied with EU Council Directive 91/630/EEC, which lays down minimum standards for the protection of pigs, and EU Council Directive 98/58/EC, which concerns the protection of animals kept for farming purposes. This trial was conducted between November 2006 and August 2010.

Animals and design

Closely related F1 gilts (Large White × Landrace) were selected as replacement breeding stock at birth on a commercial breeding company’s (Hermitage AI) multiplier farm and exposed to the same group housing and feeding regimens up to final selection at 22 weeks of age. At this time, 158 gilts were selected on their backfat levels at the P2-site (7–9 mm or 10–14 mm, respectively) and categorised as thin (n 78) or fat (n 80). Backfat depth was measured at the last rib and 65 mm from the backbone of the gilt using an ultrasound scanner (Lean-meater, Renco Corporation) on both the left and right side and the mean was recorded. The difference in backfat between groups was nutritionally accentuated until service (32 weeks) when the backfat depths of thin and fat gilts were 12 (SEM 0.6) and 19 (SEM 0.6) mm, respectively. Between 22 and 30 weeks of age, thin gilts were fed 1.8 kg/d dry sow diet (619 g/kg lysine, 13.0 MJ digestible energy (DE)/kg) and the fat gilts were provided with ad libitum access to a gilt developer diet (5.85 g/kg lysine, 14.3 MJ DE/kg) (Table 1). For 4 weeks before planned mating, gilts were provided with constant boar contact by introducing a boar into an adjoining pen. At 2 weeks before service, all gilts were provided with ad libitum access to a lactation sow diet for the flushing effect (Table 1). A quantity of 9 ml of Regumate Equine (alretogest, 2.2 μg/ml; Intervet Productions S.A.) per gilt was added to the lactation sow diet daily for 6 d to synchronise gilts to oestrus. Gilts were provided with 10 min boar contact twice daily to aid oestrus detection once they came off the Regumate. Gilts were artificially inseminated at onset of standing oestrus and again 24 h later using semen pooled from eight closely related Hylean Large White boars (Hermitage AI). Immediately after service, gilts were moved to individual gestation pens (2.4 m × 0.6 m; O’Donovan Engineering) where they were fed, once per d, the dry sow diet (Table 1) until day 110 of gestation. Each gilt was fed 1.8 kg/d (23.4 MJ DE/d) for the first 25 d of gestation. On day 25 of gestation, gilts from each body condition group (fat or thin) were blocked according to weight and expected farrowing date and allocated at random to one of three feeding levels: (a) restricted (1.8 kg/d), (b) control...
At weaning, gilts were moved to individual pens in the service area and provided with *ad libitum* access to the lactation diet in pellet form until service. The weaning to oestrus interval and feed intake during this period were recorded. Immediately following service, gilts were returned to the dry sow accommodation and provided with the dry sow diet in liquid form (30 MJ DE/d (about 2.3 kg/d on a fresh weight basis)) through to the subsequent farrowing. Gilt reproductive performance at the subsequent farrowing was recorded.

Gilts were weighed and backfat measurements were taken at about 22 weeks, at service (about 32 weeks) on day 25, 50, 80 and 110 of gestation, at weaning and at the subsequent oestrus. Feed allocation is presented on a meal equivalent fresh weight (kg/d) basis for each period (Table 1).

### Table 1. Composition of experimental diets (on an air dry basis; g/kg)*

|                | Gilt developer diet | Dry sow diet | Lactation diet |
|----------------|---------------------|--------------|----------------|
| **Wheat**      | 832.9               | 892.9        | 423.9          |
| **Barley**     | 75                  | 75           | 160            |
| Soyabean meal  | 50 % crude protein  |              |                |
| Soyabean oil   | 70                  | 10           | 40             |
| Mineral and vitamins | 1.5    | 1.5          | 1.5            |
| Lysine HCl†    | 0.5                 | 0.5          | 2.0            |
| Ô-Methionine‡  | 0                   | 0            | 0.7            |
| Ô-Threonine‡   | 0                   | 0            | 0.8            |
| Di-calcium phosphate | 5       | 5            | 5              |
| Limestone flour | 11                  | 11           | 12             |
| Salt           | 4                   | 4            | 4              |
| Phytase‡       | 0.1                 | 0.1          | 0.1            |
| **Chemical composition** |             |              |                |
| DM             | 870                 | 871          | 873            |
| Crude protein  | 153                 | 116          | 158            |
| Crude fat      | 85                  | 26           | 39             |
| Crude fibre    | 32                  | 34           | 30             |
| Ash            | 39                  | 36           | 43             |
| Lysine§        | 5.85                | 6.2          | 9.1            |
| Digestible energy (MJ/kg)§ | 14.3 | 13.0 | 14.2 |

* Dry sow diet and lactation diet provided (mg/kg completed diet): Cu, 30 mg; Fe, 70 mg; Mn, 65 mg; Zn, 80 mg; I, 0.6 mg; Se, 0.2 mg; vitamin A as retinyl acetate, 3 mg; vitamin D₃ as cholecalciferol, 25 µg; vitamin E as α-tocopheryl acetate, 100 µg; vitamin K₂, 2 mg; vitamin B₁₂, 15 µg; riboflavin, 5 mg; nicotinic acid, 12 mg; pantothenic acid, 10 mg; thiamine chloride, 500 mg; biotin, 200 µg; folic acid, 5 mg; thiamine, 2 mg; pyridoxine, 3 mg.
† Synthetic amino acids.
‡ Sow diets contained 500 phytase units (FTU) per kg finished feed from Natuphos 5000 (BASF).
§ Calculated from standard book values for ingredients.

(2.5 kg/d) or (c) high feed level (3.5 kg/d), until day 90 of gestation. In total there were six treatment groups: thin restricted, thin control, fat restricted, fat control and 5 kg/d through to day 110. Water was available on an *ad libitum* basis throughout the experiment. At day 110 gilts were moved to individual farrowing pens and liquid fed (Big Dutchman) the lactation diet (Table 1) until farrowing at an allocation of 2.03 kg/d (28.8 MJ DE/d). Gilts were accommodated in farrowing rooms with ten gilts per room in National Pig Development Company type farrowing crates (O’Donovan Engineering) with hinged bottom bars. After farrowing, gilts were scale fed the lactation diet using a lactation feed curve increasing from 28.8 MJ DE/d at day 0 to 112.7 MJ DE/d at day 28 post-farrowing. Room temperature was maintained at 20°C except at farrowing when the temperature was increased to 24°C for 48 h. At farrowing litter weight, total born, born alive, stillbirths and mummies were recorded as well as individual piglet birth weights. Piglets were tagged at birth for identification purposes. Litter size was standardized at farrowing to approximately twelve pigs per litter by cross-fostering within treatment groups within 24 h of birth. The offspring were then kept with the gilt and suckled by her for the first 28 d of their life. In addition, a creep feed (16.5 MJ/DE, hyssine 1-6 %; Startrite 88; Nutec) was fed to all litters from day 12 postpartum to weaning at approximately day 28 postpartum. Pre-weaning mortality was recorded. Individual pig weight was recorded at weaning.

### Salivary cortisol measurement

To allow time for the gilts to adapt to their housing and their respective feeding levels, saliva was collected from gilts at day 80 of gestation. A cotton swab (Salivette Plain) was attached to a pair of surgical tongs and placed in the mouth of the gilt at three time points during the day: 09.30 (prior to feeding), 12.30 and 15.30 hours. These time points were chosen, as cortisol follows a circadian pattern(44). Gilts were allowed to chew on the cotton swab for approximately 30 s, or until saturated. Swabs were then returned to the container and centrifuged at 1000 g for 2 min at room temperature within 1 h of collection. The saliva was transferred to Eppendorf tubes and stored at −20°C. Saliva samples were assayed for cortisol levels in duplicate by a Salivary Cortisol EIA kit (Salimetrics). Cortisol concentration was quantified by interpolating absorbance readings from a standard curve generated in the same assay.

### Colostrum sampling

A colostrum sample (about 5 ml) was collected from each gilt, within 6 h of parturition. Colostrum samples were manually collected from teats at anterior, middle and posterior locations of the udder, pooled and immediately frozen at −20°C. Colostrum samples were analysed in duplicate for IgG levels using specific IgG pig-ELISA kits (Bethyl Laboratories Inc.). The IgG levels were quantified according to the manufacturer’s instructions by interpolating absorbance readings from standard curves generated in the same assay.

### Milk sampling

Milk samples (about 15 ml) were collected from gilts after their morning meal on day 21 of lactation which represents peak lactation(45). Piglets were removed from the gilt in the morning in order to facilitate refill of the mammary gland before sampling at noon. Milk samples were manually collected from teats at anterior, middle and posterior locations of the udder, after a 1 ml (10 IU) intramuscular injection of oxytocin (Eurovet Animal Health) to induce milk let down. All milk samples were frozen at −20°C until subsequent analysis. Before analysis, milk samples were thawed at 4°C and preservatives were added to prolong shelf life (Broad Spectrum Microtabs® II;
D&F Control Systems Inc.). Milk samples were diluted with distilled water prior to analysis to ensure that sample composition fell within the validated calibration range for the infrared analyser. Samples were heated to 40°C and analysed in duplicate for percentage fat, protein and lactose concentrations on an infrared analyser (Milkoscan™ FT 6000; Foss Electric). Protein content was validated using the Kjeldahl method (result $R^2$ 0.9396) according to ISO(48). Total fat was validated using the Rose–Gottlieb technique (result $R^2$ 0.9313) according to James(47). Following extraction, 30 mg of the respective milk fat were dissolved in hexane and transesterified at room temperature by the addition of 200 µl of 2 M-methanolic KOH. After 5 min the reaction was terminated using 0.5 g of sodium hydrogen sulphate monohydrate and the resulting fatty acid methyl esters (FAME) were analysed by GLC (3400; Varian) as previously described by Childs et al.(48).

Feed analyses
Feed samples for analysis were collected before feeding at intervals throughout the experiment and were pooled for analysis. DM, crude ash, crude protein, crude fat and crude fibre were analysed by the methods described previously(49).

Statistical analysis
Data were analysed using mixed models in SAS (SAS Institute, Inc.). For gilt production performance, the fixed effects were body condition (thin or fat), feed level (restricted, control and high), block and the body condition × feed level interaction. Litter birth weight was included as a covariate in the model. For the data describing body weight of the gilt, the weight of the gilts at day 25 of gestation was included as a covariate. For the milk and colostrum results, body condition, feed level and the interactions between body condition and feed level were included as fixed effects and litter size was included as a covariate. Cortisol data were analysed using repeated measurements in PROC MIXED. Time was included as a covariate. Cortisol data were analysed using PROC UNIVARIATE in SAS. Results were considered statistically significant when $P<0.05$ and were considered as trends when $P≤0.10$.

Results
Diet composition and analysis
The ingredient composition and the nutrient content of the gilt diets are presented in Table 1.

Production parameters – gilts
The final number of gilts included in the analysis for production parameters was sixty-eight thin and seventy-two fat gilts, due to sixteen repeating and two deaths. The effect of maternal body condition and feed level during gestation on gilt weight, backfat levels, lactation performance, length of pregnancy and subsequent reproductive performance is presented in Table 2.

Body weight and backfat
The body weight of pre-selected thin and fat gilts was different from day 25 of gestation onwards, with thin gilts being lighter throughout the trial ($P<0.05$). At day 25, body weights for restricted, control and high feed level gilts were similar ($P>0.05$). From day 50 onwards, however, feed level influenced body weight of the animals, with the restricted feeding being lighter than control fed gilts, and control fed gilts being lighter than high feed level gilts ($P<0.001$). Thin gilts had lower backfat levels than fat gilts at day 25 and throughout the trial ($P<0.001$). At day 25 of gestation, backfat depths were similar for restricted, control and high feed level gilts ($P>0.05$). From day 50 onwards, however, feed level influenced the backfat depth ($P<0.001$).

Lactation performance
Restricted gilts had a higher average daily feed intake during lactation than control and high feed level animals ($P<0.001$). There was no difference in daily feed intake for thin and fat gilts ($P>0.05$). During lactation, fat gilts lost more weight than thin gilts ($P<0.001$). Gilts restricted during gestation did not lose weight during lactation, while control and high feed level gilts did ($P<0.01$). Body condition or feed level did not influence the length of pregnancy ($P>0.05$) or lactation length ($P>0.05$).

Weaning to oestrus and subsequent performance
There was a tendency towards a body condition × feed level interaction for the weaning to oestrus interval. Fat control gilts tended to have a longer weaning to oestrus interval than thin control gilts (5.91 v. 5.05 d; SEM 0.212 d; $P=0.06$). Thin gilts had a shorter weaning to oestrus interval than fat gilts ($P<0.05$). There was no effect of the feed level or body condition during the first gestation on the number of pigs born alive ($P>0.05$) or born dead ($P>0.05$) at the second parity.

Piglet growth from birth to weaning
The main effects of maternal body condition and feed level during gestation on piglet growth up to weaning (day 28) are presented in Table 3. Restricted gilts had higher numbers of piglets born alive than gilts on the control feed level with both groups having similar values to that of high gilts ($P<0.05$). Restricted gilts gave birth to lighter piglets than gilts on the high feed level with both groups having similar values to that of control gilts ($P<0.05$). The mean weaning weight of the piglets was influenced by the body condition of the gilt, with piglets from the fat gilts being heavier than piglets from thin gilts ($P<0.05$). Piglets from fat gilts also had higher average daily gain from birth to weaning than piglets from thin gilts ($P<0.05$).

Salivary cortisol
The salivary cortisol levels in pregnant gilts are presented in Table 4. There was a time effect, with salivary cortisol
Table 2. Influence of the main effects, maternal body condition (thin or fat) and gestation feed level (restricted: 1·8 kg/d; control: 2·5 kg/d; or high feed level: 3·5 kg/d) on sow performance during gestation, during lactation and on subsequent reproductive performance (Adjusted mean values with their pooled standard errors)

| Treatment | Thin | Fat | SEM | P* | Restricted | Control | High feed | SEM | P* |
|-----------|------|-----|-----|----|------------|---------|-----------|-----|----|
| n         | 68   | 72  | 44  | 48 | 48         |          |           |      |    |
| Sow body weight (kg) |       |     |     |    |            |          |           |      |    |
| Day 25 of gestation | 141·7 | 158·5 | 1·61 | 0·001 | 149·7 | 149·9 | 150·7 | 2·04 | 0·92 |
| Day 50 of gestation | 161·0 | 176·6 | 1·63 | 0·001 | 160·5a | 167·6b | 173·3c | 2·07 | 0·001 |
| Day 80 of gestation | 182·6 | 198·0 | 1·64 | 0·001 | 175·9a | 189·0b | 206·1c | 2·08 | 0·001 |
| Day 110 of gestation | 198·6 | 213·5 | 1·76 | 0·001 | 191·9a | 204·3b | 222·0c | 2·20 | 0·001 |
| Farrowing weight† | 172·4 | 184·5 | 1·65 | 0·001 | 162·2a | 177·5b | 193·9c | 2·00 | 0·001 |
| Weaning weight | 167·6 | 173·8 | 2·01 | 0·05 | 161·6a | 170·4b | 180·4c | 2·51 | 0·001 |
| Sow backfat (mm) |       |     |     |    |            |          |           |      |    |
| Day 25 of gestation | 12·2 | 18·9 | 0·32 | 0·001 | 15·6 | 15·5 | 15·5 | 0·41 | 0·96 |
| Day 50 of gestation | 13·4 | 20·1 | 0·31 | 0·001 | 15·8a | 16·5a | 18·0b | 0·40 | 0·001 |
| Day 80 of gestation | 14·8 | 20·2 | 0·37 | 0·001 | 15·7a | 17·0a | 19·9b | 0·47 | 0·001 |
| Day 110 of gestation | 14·4 | 19·0 | 0·34 | 0·001 | 14·9a | 16·5a | 18·7b | 0·43 | 0·001 |
| Weaning backfat | 11·2 | 13·8 | 0·31 | 0·001 | 11·6a | 12·2a | 13·6b | 0·39 | 0·001 |
| Daily lactation feed intake (kg/d) | 4·98 | 4·84 | 0·124 | 0·43 | 5·43a | 4·69b | 4·61b | 0·154 | 0·001 |
| Lactation body weight change (%) | −1·03 | −5·94 | 1·040 | 0·001 | +0·02a | −3·94b | −5·58b | 1·284 | 0·01 |
| Length of pregnancy (d) | 115·5 | 115·5 | 0·19 | 0·98 | 115·5 | 115·4 | 115·6 | 0·24 | 0·06 |
| Lactation length (d) | 27·7 | 27·7 | 0·50 | 0·01 | 28·0 | 27·7 | 27·4 | 0·50 | 0·47 |
| Weaning to oestrus (d)* | 5·2 | 5·6 | 0·12 | 0·025 | 5·4 | 5·5 | 5·3 | 0·15 | 0·50 |
| Subsequent performance |       |     |     |    |            |          |           |      |    |
| Total pigs born/litter | 12·4 | 12·4 | 0·49 | 0·097 | 13·2 | 12·0 | 12·0 | 0·62 | 0·30 |
| Total pigs born dead/litter | 0·6 | 1·1 | 0·24 | 0·20 | 0·7 | 0·8 | 1·1 | 0·20 | 0·55 |

* Mean values within a row with unlike superscript letters were significantly different (P < 0·05) (Tukey-Kramer adjusted).
† Estimated value: empty farrowing weight = (sow weight at day 110 − (total born × 2·28)). The value of 2·28 kg is an estimate of the interaction of any of the variables tested.
‡ Salivary cortisol concentrations were 9·86, 5·04 and 5·33 nmol/l (SEM 0·419 nmol/l; P < 0·001) at 09·30, 12·30 and 15·30 hours, respectively. Thin gilts tended to have higher mean cortisol levels than fat gilts (7·34 vs. 6·15 nmol/l; SEM 0·493 nmol/l; P = 0·08). Cortisol levels for restricted gilts were higher (8·50 (SEM 0·611 nmol/l; P < 0·001)) than those for gilts fed the high feed level (5·00 nmol/l) and tended to be higher (SEM 0·611 nmol/l; P < 0·10) than those for gilts fed the control feed level (6·73 nmol/l). In turn, control fed gilts tended to have higher (P = 0·10) salivary cortisol concentrations than gilts fed the high feed level. Correlations between the average birth weight and maternal morning cortisol levels were calculated. There was a weak negative correlation (−0·2651; P < 0·02) between maternal morning cortisol levels and the average birth weight of the piglets. However, when

Table 3. Influence of the main effects, maternal body condition (thin or fat) and gestation feed level (restricted: 1·8 kg/d; control: 2·5 kg/d; or high feed level: 3·5 kg/d) on litter size, piglet performance at birth and weaning (Adjusted mean values with their pooled standard errors)

| Treatment | Thin | Fat | SEM | P* | Restricted | Control | High feed | SEM | P* |
|-----------|------|-----|-----|----|------------|---------|-----------|-----|----|
| n         | 68   | 72  | 44  | 48 | 48         |          |           |      |    |
| Litter size |       |     |     |    |            |          |           |      |    |
| Litter birth weight (kg) | 16·5 | 17·3 | 0·49 | 0·25 | 17·1 | 16·5 | 17·0 | 0·62 | 0·72 |
| Total number born | 12·1 | 12·7 | 0·38 | 0·64 | 13·0 | 11·9 | 12·3 | 0·48 | 0·20 |
| Number born alive/litter | 11·4 | 11·9 | 0·38 | 0·89 | 12·4a | 11·1b | 11·4ab | 0·49 | 0·04 |
| Number born dead/litter | 0·76 | 0·80 | 0·130 | 0·59 | 0·64 | 0·80 | 0·90 | 0·165 | 0·51 |
| Number of pre-weaning deaths per litter | 1·79 | 1·98 | 0·229 | 0·52 | 1·87 | 2·14 | 1·64 | 0·287 | 0·43 |
| Total number weaned/litter† | 10·1 | 9·9 | 0·22 | 0·17 | 10·4 | 9·7 | 9·9 | 0·27 | 0·21 |
| Piglet body weight |       |     |     |    |            |          |           |      |    |
| Mean piglet birth weight (kg) | 1·47 | 1·49 | 0·028 | 0·67 | 1·41a | 1·50a, b | 1·53b | 0·036 | 0·05 |
| CV of birth weight (%)† | 18·36 | 19·44 | 0·705 | 0·23 | 19·05 | 18·87 | 18·78 | 0·884 | 0·99 |
| Mean weaning weight (kg) | 7·03 | 7·43 | 0·140 | 0·04 | 7·21 | 7·25 | 7·22 | 0·174 | 0·91 |
| CV of weaning weight (kg) | 17·09 | 18·15 | 0·821 | 0·33 | 16·23 | 18·15 | 18·49 | 1·029 | 0·23 |
| ADG (g/d) | 202·4 | 214·3 | 4·21 | 0·05 | 209·0 | 209·5 | 206·6 | 5·28 | 0·91 |
| CV of ADG‡ | 20·40 | 20·45 | 1·078 | 0·98 | 18·49 | 20·97 | 21·82 | 1·278 | 0·15 |

ADG, average daily gain.

* Mean values within a row with unlike superscript letters were significantly different (P < 0·05) (Tukey-Kramer adjusted).
† Litter size was standardised at farrowing to approximately twelve pigs within treatment groups.
‡ Within-litter CV values.
maternal body condition or feed level was analysed together with morning cortisol their correlations with average birth weight were: thin, $-0.0548 \ (P > 0.05)$; fat, $-0.4771 \ (P > 0.003)$; restricted, $-0.3755 \ (P > 0.05)$; control, $0.1177 \ (P > 0.05)$; and high feed level, $-0.3625 \ (P = 0.10)$.

**Colostrum samples**

The result of colostrum analysis at parturition for IgG levels is shown in Table 4. There were no differences between the colostrum IgG levels of thin (110-7 mg/ml) and fat (136-2 mg/ml) gilts ($\text{SEM} \ 15-23 \ mg/ml; P > 0.05$). In addition, feed level did not influence colostrum IgG levels for restricted (138-8 mg/ml), control (111-5 mg/ml) and high (119-8 mg/ml) feed level gilts ($\text{SEM} \ 13-51 \ mg/ml; P > 0.05$).

**Milk composition**

The effects of maternal body condition and gestation feed level on milk composition at day 21 postpartum are presented in Table 4. Fat gilts had a higher milk fat percentage compared with thin gilts (8.3 v. 6.5 % fat; $\text{SEM} \ 0.42 \%$; $P < 0.001$). Milk fat levels were not influenced by the level of feeding during gestation, being 7.6, 7.1, and 7.5 % ($\text{SEM} \ 0.55 \%$; $P > 0.05$) for restricted, control and high feed level, respectively. Gestation feeding level or body condition had no effect on protein and lactose levels in the milk.

**Fatty acid composition in milk**

Table 4. Effect of maternal body condition (thin or fat) and gestation feed level (restricted: 1.8 kg/d; control: 2.5 kg/d; or high feed level: 3.5 kg/d) on saliva cortisol levels at day 80 of pregnancy, and IgG levels in colostrum at parturition and day 21 milk composition

(Adjusted mean values with their pooled standard errors)

| Treatment | Thin | Fat | n | Cortisol (nmol/l) | SEM | P* Body condition | Feed level |
|-----------|------|-----|---|------------------|-----|------------------|------------|
| 1.8 kg    | 2.5 kg | 3.5 kg | 23 | 24 | SEM | 0.912 | 0.081 | 0.001 |
| 7.78 | 6.44 | 4.21 | 81 | 10 | 8 | 0.128 | 0.218 | 0.527 |
| 12.7 | 123.5 | 156.3 | 27.98 | 12 | 15 | 13 | 0.475 | 0.081 |

* There was no significant body condition x feed level interaction for cortisol, colostrum or milk composition.

Thin gilts had higher levels of total saturated fat (39.34 g/100 g of FAME) in their milk than fat gilts (36.91 g/100 g of FAME; $\text{SEM} \ 0.736 \ g/100 \ g \ of \ FAME, P < 0.01$) (Table 5). Fat gilts had higher levels of unsaturated fat in their milk (62.01 g/100 g of FAME) than thin gilts (59.72 g/100 g of FAME; $\text{SEM} \ 0.715 \ g/100 \ g \ of \ FAME, P < 0.05$). The unsaturated saturated fat ratio was 1.55:1 and 1.73:1 ($\text{SEM} \ 0.056$; $P = 0.05$) for thin and fat gilts, respectively. There was no effect of feed level during gestation on saturated or unsaturated fat concentration in milk. There was no effect of maternal body condition or feed level during gestation on the composition of $\omega-3$ and $\omega-6$ PUFA. Feed level during gestation significantly affected the concentration of C10 : 0, C12 : 0, C14 : 0, C14 : 1, C16 : 1, C16 : 1, C18 : 0, C18 : 1, C20 : 2 and tended to influence C6 : 0 and C15 : 0 concentrations. Body condition significantly affected the concentration of C10 : 0, C12 : 0, C14 : 0, C14 : 1, C15 : 0, C16 : 0, C16 : 1, C17 : 1, C18 : 0, C18 : 1 and C20 : 2 and tended to affect the concentrations of C10 : 1, C14 : 1, C19, $\Delta$11 conjugated linoleic acid and C20 : 0 (see Table 5).

**Discussion**

The present study demonstrates that gestation feeding level affects the numbers of offspring born alive per litter and offspring birth weight, while maternal body condition affects weaning weight and growth of offspring. This is, to our knowledge, the first study combining feed level and body condition interactions during pregnancy to determine their influences on postnatal piglet growth. However, in our study, very few interactions were observed. Restricted gilts had higher levels of...
Influence of maternal body condition during pregnancy

Piglets born to fat gilts had higher average daily gain between birth and weaning and were heavier at weaning than piglets born to thin gilts. This increased postnatal growth rate may be attributable to (a) the higher milk fat percentage of the fat gilts, (b) the difference in milk fat composition and/or (c) it could be due to other factors not measured such as appetite, which can be affected by programming(27) or possibly the uptake of nutrients in the gastrointestinal tract. A high fat content in sow milk is desirable as it promotes weight gain and fat deposition in piglets that functions as an insulator layer(50). Although milk fat and fatty acid composition levels are largely determined by the level and source of dietary fat used in the diet(29), Reveil et al(50) also observed that fat levels in the milk increased by 21% for fat sows (340 g of body fat/kg body weight) compared with thin (280 g of body fat/kg body weight) sows. Interestingly, in our study the fat gains on a high feed level during gestation had the highest ratio (1·89:1) of unsaturated:saturated fat in their milk. Milk TAG come from two sources: biosynthesis of fatty acids within the mammary gland (de novo synthesis) and uptake from the plasma by the mammary gland(51). Saturated fat contains mainly SCFA that arise predominately from de novo synthesis in the mammary gland, while longer-chain fatty acids arise directly from blood lipids from dietary fatty acids(52). As all gilts were given the same lactation diet and there was no difference in lactation feed intake, the milk fat difference between

| Treatment | Thin | Fat |
|-----------|-----|-----|
| n         | 18  | 18  |
| 1·8 kg    | 1·5 | 1·5 |
| 2·5 kg    | 1·5 | 1·5 |
| 3·5 kg    | 1·5 | 1·5 |
| 1·8 kg    | 1·5 | 1·5 |
| 2·5 kg    | 1·5 | 1·5 |
| 3·5 kg    | 1·5 | 1·5 |
| SEM       |     |     |
| Pt        |     |     |

Table 5. Effect of maternal body condition (thin or fat) and gestation feed level (restricted: 1·8 kg/d; control: 2·5 kg/d; or high feed level: 3·5 kg/d) on fatty acid composition of milk at day 21 of lactation (Adjusted mean values with their pooled standard errors)
thin and fat gilts observed may have arisen from differences in rates of de novo synthesis. In addition, the thin restricted gilts had higher levels of fat in their milk than the thin control and thin high feed level gilts, suggesting different pathways of energy distribution for maintaining high fat levels in the milk in these groups. It is probable that for the thin gilts the main fat source for milk production came from the feed, contrary to the fat gilts where the primary source could have been a mixture of endogenous and exogenous sources.

Although the main limiting factor for intake in piglets is milk volume\(^{53}\), the differences found in piglet growth between thin and fat gilts could also be influenced by the differences in milk fatty acid composition. For example, the amount of oleic acid (C18:1\(\text{cis}9\)) was higher in the milk from fat gilts and increased with gestation feeding level. In vitro cellular models altering the conformation of a C18:1 double bond from \(\text{cis}\) to \(\text{trans}\) (oleic acid to elaidic acid) decreases cholecystokinin secretion, a satiety hormone involved in appetite regulation\(^{31}\). Oleic acid also provides a signal of nutrient abundance which switches fuel sources from carbohydrates to lipids\(^{54}\). This would indicate that offspring from fat gilts were receiving a healthier milk composition, with regard to appetite regulation. Of the \(n\)-3 and \(n\)-6 PUFA, only C20:3\(\text{cis}6\), a precursor for the synthesis of prostaglandins and other eicosanoids, was increased in the milk of fat gilts. An alteration in the balance of eicosanoid synthesis can cause chronic inflammation, arterial hypertension, CHD, atherosclerosis and diabetes mellitus\(^{55,56}\) later in life. Saturated fats C10:0 (capric acid), C12:0 (lauric acid) and C14:0 (myristic acid) increased in milk both from restricted fed gilts and from thin gilts. The importance of the lactation period has been demonstrated in rats where postnatal factors overcame both genetic predisposition and prenatal factors in determining the development of adiposity, insulin sensitivity and brain pathways that mediate these functions\(^{57}\). Feeding regimens and body condition did not influence the \(n\)-6 to \(n\)-3 PUFA levels, indicating that supplementation with different dietary fats would be required to change \(n\)-6: \(n\)-3 ratios\(^{25,37}\).

**Influence of maternal feed level during pregnancy**

Restricted gilts had the highest level of salivary cortisol, most likely due to undernutrition and suboptimal gut feeding stress to the pregnant animal. High levels of cortisol during pregnancy may lead to in utero growth restriction as thin restricted gilts gave birth to piglets with the lowest birth weights. Undernutrition per se can reduce birth weight in offspring due to the reduced nutrient supply to the fetus as a consequence of suboptimal feeding of the mother. For example, a reduction to 30 % of ad libitum intake in pregnant rats resulted in newborns with a 25 % reduced birth weight compared with the control\(^{58}\). However, in addition there is also substantial evidence that maternal glucocorticoid levels, such as cortisol, affect offspring birth weight and glucose metabolism\(^{25}\). Monkeys subjected to a mild stressor in the form of noise and removal from their cage during pregnancy gave birth to lower birth weight offspring\(^{59}\). Furthermore, glucocorticoid administration during pregnancy reduces birth weight by 9 % in human subjects\(^{60}\), and by 15 % in response to one dose of 0.5 mg/kg betamethasone in sheep\(^{61}\). In addition, pregnant sows treated orally with hydrocortisone-acetate gave birth to piglets with lower birth weight (1.5 kg) compared with piglets born to control sows (1.65 kg)\(^{26}\). Surprisingly, restricted gilts gave birth to the highest number of piglets born alive. This could be because of differences in placental attachment and blood flow due to treatment. However, it could also be due to a higher exposure to cortisol. Kranendonk et al.\(^{22}\) showed that oral administration of hydrocortisone-acetate to sows during pregnancy (day 21 to 110) increased the number of piglets born alive. They hypothesized that this could be due to enhanced maturation of the organs of the piglets born to cortisol-treated sows\(^{25}\). Mullan & Williams\(^{62}\) also found that total birth weights were lower for gilts restricted to 1.5 kg feed per d during pregnancy. One suggested pathway is that low placental 11β-hydroxy-steroid dehydrogenase type 2 activity correlates with lower birth weight because of increased transplacental passage of active maternal glucocorticoids\(^{25}\). As cortisol is an inhibitor of the growth hormone–insulin-like growth factor 1 axis\(^{63}\), this elevated level might suppress insulin-like growth factor 1 actions, causing growth retardation\(^{64}\).

Our results suggest that even though restrictive feeding increased the number of piglets born alive per litter, a fixed uterine capacity resulted in lighter individual piglets in these larger litters. Restricting feed intake during pregnancy did not increase the total number of piglets born per litter, indicating that despite the lack of a significant effect of feed level on the number of piglets born dead, a numerical reduction in the number of piglets born dead was in part responsible for the increase in the number of piglets born alive from restricted gilts.

In conclusion, few maternal body condition × gestation feed level interaction effects were observed for offspring growth. During gestation, feed-restricted gilts had higher cortisol levels and gave birth to lighter piglets. This response to restricted feeding was greatest for the fat gilts. Weaning weights were heavier and average daily gain was greater in piglets born to fat gilts. Furthermore, body condition of gilts and feed level during gestation altered the milk fat percentage and profile, with thin gilts having higher levels of saturated fat than fat gilts.

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C. S. were responsible for running the GC and analysis. P. G. L., N. C. S. and L. G. secured funding for this study. The initial draft of the manuscript was written by C. A. and critically revised by P. G. L., L. G. and N. C. S. The final version was compiled by C. A. and all the authors read and approved the final manuscript.

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