

Supplementing sow diets with palm oil during late gestation and lactation; effects on milk production, sow hormonal profiles, and growth and development of her offspring

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1 **Running title: Palm oil and lactating sow diets**

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3 **Supplementing sow diets with palm oil during late gestation and**
4 **lactation; Effects on milk production, sow hormonal profiles, and growth**
5 **and development of her offspring**

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Abstract: The supplementing of sow diets with lipids during pregnancy and lactation has been shown to reduce sow condition loss and improve piglet performance. The aim of this study was to determine the effects of supplemental palm oil (**PO**) on sow performance, plasma metabolites and hormones, milk profiles, and pre-weaning piglet development. A commercial sow ration (**C**) or an experimental diet supplemented with 10% extra energy in the form of PO, were provided from d 90 of gestation until weaning (24-28 d *post-partum*) in two groups of 8 multiparous sows. Gestation length of PO sows increased by 1 day ($P<0.05$). Maternal body weight changes were similar throughout the trial, but loss of backfat during lactation was reduced in PO animals (C: -3.6 ± 0.8 mm; PO: -0.1 ± 0.8 mm; $P<0.01$). Milk fat was increased by PO supplementation (C d3: $8.0\pm0.3\%$ fat; PO d3: $9.1\pm0.3\%$ fat; C d7: $7.8\pm0.5\%$ fat; PO d7: $9.9\pm0.5\%$ fat; $P<0.05$) and hence milk energy yield of PO sows was also elevated ($P<0.05$). The proportion of saturated fatty acids was greater in colostrum from PO sows (C: 29.19 ± 0.31 g/100g of fat; PO: 30.77 ± 0.36 g/100g of fat; $P<0.01$). Blood samples taken on 105 days of gestation, within 24 hours of farrowing, day 7 of lactation and at weaning (28 ± 3 days post-farrowing) showed there were no differences in plasma concentrations of triacylglycerol, non-esterified fatty acids, insulin or insulin-like growth factor-1 throughout the trial. However, circulating plasma concentrations of both glucose and leptin were elevated during lactation in PO sows ($P<0.05$ and $P<0.005$, respectively) and thyroxine was greater at weaning in PO sows ($P<0.05$). Piglet weight and body composition were similar at birth, as were piglet growth rates throughout the pre-weaning period. Seven days after birth, C piglets contained more body fat, as indicated by their

lower fat free mass per kg (C: 66.4 ± 0.8 arbitrary units/kg; PO: 69.7 ± 0.8 arbitrary unit/kg; $P < 0.01$), but by day 14 of life this situation was reversed (C: 65.8 ± 0.6 arbitrary units/kg; PO: 63.6 ± 0.6 arbitrary units/kg; $P < 0.05$). Following weaning, PO sows exhibited an increased ratio of male to female offspring at their subsequent farrowing (C: 1.0 ± 0.3 ; PO: 2.2 ± 0.2 ; $P < 0.05$). We conclude that supplementation of sow diets with PO during late gestation and lactation appears to increase sow milk fat content and hence energy supply to piglets. Furthermore, elevated glucose concentrations in the sow during lactation may be suggestive of impaired glucose homeostasis.

Key Words: Metabolites, Piglets, Body Composition, Milk, Fatty Acids.

Implications

Palm oil can be used as an effective energy source in the diets of pregnant and lactating sows, reducing sow body condition loss whilst improving the energy density of milk available to growing piglets. However, despite these benefits the feeding of palm oil during the latter stages of pregnancy also results in changes to glucose and thyroid metabolism. The causes, consequences and longer term implications of these changes are generally unknown and require further investigation.

Abbreviations: Control (C), fat-free mass (FFM), insulin-like growth factor-1 (IGF-1), non-esterified fatty acids (NEFA), palm oil (PO), polyunsaturated fatty acids (PUFA), radioimmunoassay (RIA), total-body electrical conductivity analyzing system (TOBEC), Triiodothyronine (T_3) and thyroxine (T_4)

70

71 **Introduction**

72 Nutrient requirements for lactation in sows are met both by dietary
73 sources, and by maternal tissue stores, resulting in mobilization of body
74 reserves and a reduction in maternal fat reserves by the time of weaning
75 (Mullan and Williams, 1989). Anestrous is highly inversely correlated with
76 body weight and backfat at weaning (Mullan and Williams, 1989; Johnston *et al.*, 1993); sows with lower body weight and backfat exhibit longer periods of
77 anestrous, thus reducing the efficiency of production. Supplementation of sow
78 diets with fats during late gestation and lactation can be used as a
79 concentrated source of energy, to increase the concentration of fat in
80 colostrum and milk (Quiniou *et al.*, 2008; Tummaruk *et al.*, 2014) and hence,
81 reduce dependence on maternal body stores and subsequent probability of
82 prolonged anestrous (Tantasuparuk *et al.*, 2001).

84 A number of researchers have reported increased rates of weight gain
85 in piglets suckled by sows supplemented with animal fat during lactation
86 (Tilton *et al.*, 1999; Averette *et al.*, 1999). Piglets suckling from sows fed on
87 animal fat also lay down more fat during the pre-weaning period (Tilton *et al.*,
88 1999). The use of such animal by-products, certainly within the European
89 Union, is now restricted, primarily as a consequence of the BSE crisis
90 (Lauridsen *et al.*, 2007) and concerns about traceability, disease and chemical
91 residues. Consequently, alternative sources of fat are of increasing
92 importance and the use of palm oil is one such alternative to the use of animal
93 fats and carries none of the associated risks; although it should be noted that

lipid digestibility has been reported to decline with increasing free fatty acid concentration (Rosero *et al.*, 2016).

The use of palm oil in the diets of growing and fattening pigs has been reported by Teye *et al.* (2006) who demonstrated that there were no negative effects with regards to piglet performance by the use of palm oil. This group have previously shown that piglets born to mothers supplemented with palm oil during the first half of gestation were heavier and fatter (Laws *et al.*, 2007), whereas Almond *et al.* (2015) reported increased mortality in piglets born to sows fed palm oil throughout gestation. The aim of this study was to evaluate the effects of feeding 10% extra energy, in the form of palm oil, to sows during late gestation and through lactation on plasma metabolites and hormones, reproductive efficiency, neonatal outcome, and their subsequent growth and development.

Materials and Methods

Animals and Diets

All animals used in these studies were maintained at the Pig Research and Development Unit, Imperial College, London and protocols adopted were similar to previous studies conducted by this group (Laws *et al.*, 2009). Experimental procedures were undertaken in accordance with the Animals (Scientific Procedures) Act, 1986 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 (DEFRA, 2003), and fed commercially available diets to meet nutrient requirements.

118 Sixteen multiparous sows of a commercial genotype (25% Meishan;
119 12.5% Duroc; 62.5% Large White × Landrace), that had been artificially
120 inseminated with pooled semen from Large White boars (P17 2006, JSR
121 Genetics) were entered into the study on day 90 of gestation. Sows were
122 categorized by parity (C 5.7 ± 0.5 ; PO 5.3 ± 0.5) prior to being randomly
123 assigned to one of two dietary treatment groups to ensure that parity was
124 balanced across treatments. Maternal body weight (252 ± 5 kg Mean \pm SEM)
125 and backfat thickness (17 ± 1 mm Mean \pm SEM) at the start of the study was not
126 significantly different between treatments. Sows were assigned randomly to
127 either a control diet or one containing 10% extra energy in the form of a top
128 dressing of palm oil (**PO**) (33.54 MJ/kg; T Quality Ltd, Swindon, UK).
129 Experimental diets were supplied from day 90 of gestation (term \approx 115 days)
130 and consisted of either: i) control (**C**); 3 kg/d of the standard diet (ABN HE sow
131 pellets; ME 13.1 MJ/kg; Crude protein 12.7%; Oil 4.5% ; Fibre 4.8%; Ash
132 5.3%; Vitamin A 10000 (i.u./kg); Vitamin D3 1875 (i.u./kg); Vitamin E 60
133 (i.u./kg); Lysine 0.55%; Copper 21 mg/kg; ABN, Peterborough, UK); or ii) 3
134 kg/d of the standard diet plus 10% extra energy derived from PO (117g/d).
135 After parturition the following lactation diets were supplied: the C lactation diet
136 consisted 6-9 kg/d of the standard lactation pellets (ABN supreme lactation
137 pellets; ME 14.1 MJ/kg; Crude protein 18%; Oil 7.2%; Fibre 4.0%; Ash 5.0%;
138 Vitamin A 10000 (i.u./kg); Vitamin D3 1875 (i.u./kg); Vitamin E 75 (i.u./kg);
139 Lysine 0.95%; Copper 23 mg/kg; ABN, Peterborough, UK), and the PO
140 lactation diet consisted of 6-9 kg/d of the standard lactation pellets plus 10%
141 extra energy derived from palm oil (40 g palm oil per kg of feed). Sows were
142 offered a fixed amount of the appropriate feed daily (3-9 kg/day depending on

stage of gestation/lactation); there were no refusals and as a consequence there were no differences in feed intake observed between treatments. Fatty acid compositions of the experimental diets are shown in Table 1. Piglets had ad-libitum access to creep feed (Primary Select; 16.77 MJ/kg ME; 23.5% crude protein; Oil 9%; Fibre 2%; Ash 6.3%; Vitamin A 12500 (iu/kg); Vitamin D3 2000 iu/kg); Vitamin E 250 (iu/kg); Lysine 1.7%; Copper170 mg/kg; Primary Diets Ltd. UK) from day 14 of life.

Production Data

On d 90 and d 109 of gestation, and at weaning, sows were restrained in a weigh crate (UHL Products, UK) while their weight and ultrasonic measurements of backfat thickness (Aloka-echo camera 550-500, Aloka Ltd. Japan) were recorded. Backfat thickness was measured level with the head of the last rib, at the P1 (45 mm from the midline) and the P3 (80 mm from the midline) positions. The average of these two values was then calculated to give the P2 value. After farrowing the numbers of piglets born alive, stillborn and mummified and the number of male and female piglets born were recorded. The length of gestation was calculated from the day of insemination. After weaning, 24-28 days post-partum, sows were inseminated at their first oestrus, dates and results of the subsequent farrowing were recorded.

Piglet Growth and Composition

Growth performance of all piglets was observed throughout the neonatal period. Body weight and body composition were recorded at birth

and at 7, 14 and 21 days post-farrowing. Piglet growth rate was calculated by regression analysis of piglet weight against time. Body composition of all piglets was determined using a total-body electrical conductivity analyzing system (TOBEC, Model-SA3000 EMSCAN/TOBEC, SA-3203, Biotech Instruments Ltd. UK) on d 0, d 7, d 14 and d 21 of life. Body fat and lean mass tissues within an animal exhibit different conductivities. The increased conductivity of fat free mass is attributed to the presence of sodium (Na) and potassium (K), which in association with water exhibit electrical conductivity (EM-SCAN, 1992). When a subject is placed in the electromagnetic field, energy absorbed is a function of the area (A^2), magnetic field strength (B), conduction per unit volume at a specific frequency (c), and a number of constants (k), such that:

$$E = A^2 \times B \times c \times k \text{ (Mitchell and Scholz, 2001)}$$

The energy absorption signal produced by the TOBEC is primarily a function of the fat free mass (**FFM**) and is measured as the difference between coil impedance when empty and that with the subject within (EM-SCAN, 1992). As temperature affects electrical conductivity, it was maintained within the range of 18-22°C. Each piglet was positioned identically within a polycarbonate tube and held in place with a plunger to maintain constancy of position. Tube size was selected according to body weight (<3kg – 128 mm; 3-5kg – 150 mm; >5kg – 190mm). Piglet FFM per kg was calculated using the equation shown below, as suggested in the TOBEC manufacturer's instructions.

FFM (arbitrary units) = $\sqrt[3]{(\text{TOBEC reading} \times \text{Crown-rump-length/piglet weight (kg)})}$

Milk Composition

Colostrum samples were collected as near to birth as possible (within 12 hours of parturition) via milking by hand. Milk samples were collected on d 3, d 7, d 14 and d 21 of lactation following intra-muscular administration of 2 mL oxytocin (10 i.u./mL; NVS, UK). On each occasion 20 ml of milk were collected (milking by hand) and stored in azide coated sample pots at 4°C prior to analysis for gross milk composition by an automated infrared filtration system, which was conducted by National Milk Records (Harrogate, UK). A 1.5 ml milk sample was stored at -80°C prior to lipid extraction and purification by the method of Folch et al. (1957). Total lipid, neutral lipids or phospholipids 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.) An estimate of the total milk energy was calculated using the equation from Klaver et al. (1981):

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

Sow Blood Collection and Analyses

Samples of sow blood were collected (approximately 6 hours after the morning feed) into di-sodium EDTA blood tubes (Teklab, UK) from the jugular vein at 105 days of gestation, within 24 hours of farrowing, day 7 of lactation and at weaning (28 ± 3 days post-farrowing). Although all blood samples were taken within 6 hours of feed being offered, it is important to note that sow feed intake behaviour (irrespective of treatment) was not consistent around the time of parturition. Blood samples were centrifuged for 15 minutes at $1600g_{av}$ (Sci Quip 3K15, Sigma laboratory centrifuges, Osterode am Harz, Germany); plasma was collected and stored at -20°C until analysis. Plasma was analyzed for concentrations of glucose, non-esterified fatty acids (**NEFA**) and triacylglycerol (**TAG**), insulin-like growth factor 1 (**IGF-1**), insulin, thyroid hormones (Triiodothyronine (**T₃**) and thyroxine (**T₄**)) and Leptin using commercially available kits (glucose (GOD-PAP), and triacylglycerol (GPO-PAP) from Randox Laboratories Ltd. UK; NEFA C from Wako Chemical GmbH, Germany; IGF-1 IRMA from Diagnostic Systems Laboratories Inc., Webster, Texas, USA; Insulin, **T₃** and **T₄** radioimmunoassay (**RIA**) kits from ICN Pharmaceuticals, New York, USA; and leptin RIA assay kit from LINCO Research, St. Charles, Missouri, USA). Intra- and inter- assay Coefficients of Variation for insulin were 5.8 and 2.3%, respectively, 7.9 and 4.9%, respectively for IGF-1, 6.8 and 2.7%, respectively for leptin, 8.3 and 2.5%, respectively for **T₄**, 8.8 and 2.5%, respectively for **T₃**, 4.1 and 2.2%, respectively for glucose, 7.6 and 1.4%, respectively for NEFA, and 4.1 and 1.4%, respectively for TAG.

Statistical Analyses

Statistical differences between dietary treatments were determined by repeated measures using the mixed model procedure of SAS version 9.4 (SAS Institute Inc. Cary, NC, USA). Sources of variation within the model included diet (1 df), sample point (3 df for plasma hormones and 4 df for milk fatty acid analysis) with respect to each specific measure as previously described) and first order interactions between diet and sample point. Parity and litter size were used as covariates; parity was used as a covariate rather than class due to the spread and limited replication within and between treatments with respect to parity number. Individual animal was the repeated subject and sample point the repeated measure. Results are presented as least squares means with standard error and P value. Tukey's simultaneous tests were used to establish statistical difference between means (sample points and first order interactions). Probability values of less than 0.05 were considered to be statistically significant.

Individual piglet growth rate was calculated by regression analysis of piglet weight against time. Analysis was conducted for the period between birth and weaning. The slope of the line gave a measure of their growth rate in kg /d.

Results

Sow weight change during gestation and lactation were similar, irrespective of treatment. No differences were observed in backfat at the P2 position during gestation, however mean backfat losses were lower in PO sows during lactation (C -3.6 ± 0.8 mm; PO -0.1 ± 0.8 mm; $P < 0.05$). Mean natural

gestation length was increased by 1 d for sows in the PO group ($C\ 117 \pm 0.3$ days; $PO\ 118 \pm 0.3$ days; $P < 0.05$). There were no significant differences in total litter size (mean \pm SEM: 11.5 ± 1.2), number of piglets born alive, stillborn or mummified or in the ratio of male to female piglets. In the subsequent reproductive cycle, weaning to service interval, percentage of successful inseminations, gestation length, litter size and number of piglets born alive, stillborn and mummified were similar for both treatment groups. However, in the subsequent litter the ratio of male to female piglets was two-fold higher in litters born to PO sows compared to C sows ($C\ 1.0 \pm 0.3$; $PO\ 2.2 \pm 0.2$; male:female; $P < 0.05$).

There were no differences in piglet body weight, either at birth or throughout the neonatal period (Table 2) and consequently piglet growth rates were also similar ($C\ 0.43 \pm 0.04$ kg/day, $PO\ 0.47 \pm 0.04$ kg/day; mean \pm SEM). All piglets became fatter with increasing age ($P < 0.05$), as indicated by their lower FFM/kg (Table 2). There were no differences in FFM/kg at birth, but by d 7 of life piglets born to C sows were fatter ($P < 0.01$). In contrast, by d 14, the piglets of PO sows were fatter ($P < 0.05$) but by d 21 no differences existed in piglet FFM between treatments, which may in part be due to the introduction of creep feed on d14.

There was no difference in the concentration of milk protein or lactose, but percentage of fat was increased in the milk of sows in the PO group, during the first week of lactation ($P < 0.05$; Table 3); this trend continued to day 21 of lactation ($P < 0.1$). As a consequence of the increased proportion of fat in the milk, energy yield was also found to be higher ($P < 0.05$; Table 3). As expected, the addition of palm oil to the maternal diet influenced the fatty acid

profile of both colostrum and milk, but only significant treatment differences are highlighted below. The percentage of saturated fatty acids was elevated in the colostrum of PO sows ($P<0.01$; Table 4). On day 3 of lactation the proportion of eicosadienoic (20:2 n-6) acid was lower in the milk of PO sows ($P<0.01$; Table 4). During mid-lactation (days 7 and 14) the percentage of myristoleic (14:1) acid was decreased in the milk of PO sows ($P<0.05$; Table 4). Similarly, by day 21 of lactation the proportion of α -linolenic acid (18:3 n-3) acid was reduced in the milk of PO sows ($P<0.05$; Table 4). There were effects of time on fatty acid profile such that the sum of saturates increased between subsequent sample points ($P<0.05$), the sum of monounsaturated fatty acids increased between 0 and 3 days post-partum and then remained similar. Conversely the sum of polyunsaturated fatty acids (**PUFA**), n-6, n-3 and the PUFA to saturated ratio declined over successive sample points. There were no interactions between diet and sample point.

There were no effects of PO on plasma concentrations of TAG, NEFA, insulin and IGF-1 at any time point (Table 5). Concentrations of glucose were greater in the PO group during lactation ($P<0.05$) as were concentrations of leptin ($P<0.005$), although these effects did not persist into weaning (Table 5). Concentrations of T_3 were greater in the PO group at weaning ($P<0.05$) although there were no effects of treatment or time point on concentrations of T_4 (Table 5). Circulating concentrations of IGF-1 were seen to increase with each successive time point ($P<0.001$) but these changes were not related to diet. There were no interactions between diet and sample point for any of the parameters determined in sow plasma (Table 5).

Discussion

Maternal Performance

Feeding animal fat during gestation has been shown to increase weight gain in sows (Avarette *et al.*, 2002), while the addition of fat to the lactation diet did not appear to influence maternal weight loss (Averette *et al.*, 1999). In the present study, neither weight gain during the last few weeks of pregnancy nor weight loss during lactation were affected by supplementation of sow diets with palm oil, which may be in part due to the timing and duration of fat supplementation. However, it should be noted that replication was limited and statistical differences may have been masked by variation due to the small number of animals in each group. Consequently care needs to be exercised when interpreting these results.

During gestation, sow backfat has been shown to increase linearly with increasing feed intake (Dourmad, 1991; Cools *et al.*, 2014) but this was not reflected in the current study. However, during lactation, backfat loss was lower in the PO group, which is in accordance with the findings of others (Tilton *et al.*, 1999; Avarette *et al.*, 2002). This apparent reduction in sow backfat loss at weaning could have been a result of increased dietary energy intake during lactation.

Avarette *et al.* (2002) found no difference in the gestational length of sows after supplementation with either medium or long chain (animal fat) triacylglycerols. Conversely, in the present study supplementation of the maternal diet with PO during the last few weeks of gestation was observed to

increase gestational length by one day. The review of Tanghe and De Smet (2013) into the effects of maternal fatty acid supplementation indicated that the effects that supplementary PUFA had on gestational length were not consistent. These authors suggested that the effects reported on gestational length may be a consequence of PUFA induced changes to eicosanoid production, or alterations to enzymes involved in steroid hormone production.

The sex-allocation hypothesis of Triver and Willard (1973) predicts that females in the best body condition will tend to produce offspring of the gender which favours the sex of greater variance (i.e. males). This hypothesis has been supported by observations in several species (Rosenfeld and Roberts, 2004), including pigs (Meikle *et al.*, 1996). In this experiment the subsequent litter of PO sows contained a higher proportion of male offspring, which may reflect the energy status of sows at the time of insemination/implantation. Rosenfeld *et al.*, (2003) observed similar results in mice fed either a high saturated fat or high carbohydrate diet; however, the mechanisms for diet-induced skewing of sex ratio is not known, but a number of possible mechanisms have been suggested and are discussed in a review by Rosenfeld and Roberts (2004).

Plasma Metabolites and hormones

It is well known that NEFA are a product of fat metabolism and a sign of catabolism of fat reserves. There were no effects of PO supplementation on circulating concentrations of NEFA at any time during the present study, which could reflect the minimal change in net backfat thickness. The net reduction in backfat depth of C sows during lactation might have been anticipated to result

in increased circulating concentrations of NEFA (Ren *et al.*, 2017) but surprisingly this was not the case in the current study.

Glucose concentrations were higher in the plasma of PO sows on day 7 of lactation. This reflects the findings of van der Peet-Schwering *et al.* (2004) who reported increased glucose concentrations in fat supplemented sows during lactation. These authors suggested that elevated glucose concentrations were a consequence of fat induced glucose intolerance, as sows fed an isocaloric diet containing starch did not exhibit the same alterations in glucose concentration. Within the current study the elevated concentrations of glucose in PO sows during lactation appear to coincide with higher circulating concentrations of insulin, although differences in circulating concentrations of insulin failed to achieve statistical significance. This might be indicative of insulin resistance/glucose intolerance as Almond *et al.* (2015) reported increased area under the curve following a glucose tolerance test in late gestation in sows supplemented with palm oil. Furthermore, they also reported a higher incidence of piglet mortality in palm oil supplemented sows, which was attributed to birth hypoglycemia. These authors proposed that maternal glucose intolerance resulted in impaired piglet cognition at birth leading to reduced suckling activity and hence increased incidence of hypoglycemia at birth. The elevated glucose and insulin concentrations recorded in the current study occurred during lactation rather than during pregnancy and so were unlikely to affect piglet glycemic status and subsequent mortality.

Circulating concentrations of leptin were also found to be higher in PO sows on day 7 of lactation. Leptin is mainly produced by adipocytes and

adipose tissue and circulating concentrations of leptin are linked to body stores (Summer *et al.*, 2009). A number of studies have shown a direct correlation with levels of sow adiposity and circulating concentrations of leptin (Estienne *et al.*, 2000; De Rensis *et al.*, 2005; Summer *et al.*, 2009). This is reflected in the current study whereby both PO and C sows gained similar levels of backfat during gestation but during lactation body stores were maintained in PO sows but reduced in C sows.

Although circulating concentrations of T₄ were not appreciably different between treatments or time points in the current study, those of T₃ were seen to be higher in PO supplemented sows, particularly at weaning (P<0.05). This mirrors the findings of Von Eder and Kirchgeßner (1997) who reported that circulating T₄ concentrations were not influenced by lipid supplementation but T₃ concentrations were increased when soya oil and olive oil were lipid sources, but the same effect was not apparent when beef fat was the source. These authors concluded that fat source rather than fat content influenced thyroid hormone metabolism.

Milk Composition

In the current study milk from PO sows contained more fat whereas protein and lactose concentrations were similar, irrespective of the maternal diet. These observations are in agreement with other published research, which found that protein and lactose concentrations varied little with dietary supplementation with animal fat (Shurson, 1986) or other lipid sources (Lauridsen and Danielsen, 2004). However, Lauridsen and Danielsen (2004) also went on to report that the addition of different fats and oils to sow diets

418 did not alter the total lipid content of milk. This disparity in findings between
419 these authors' and those of the current study may reflect the higher levels of
420 dietary fat used in the current study.

421 The higher fat percentage observed in the milk of PO animals resulted
422 in a greater energy yield per kg of milk produced. The increased percentage
423 of saturated fatty acids in the colostrum (day 0) of PO sows echoes the
424 elevated saturated fatty acid content of the maternal diet (Table 1). Previous
425 research has shown that milk fatty acid composition mirrors that of the
426 maternal diet (Lauridsen and Danielsen, 2004). However, there were no diet
427 induced differences in milk total saturates throughout the rest of lactation
428 despite the differences observed in the total saturates of the lactation diet.

429 Lauridsen and Danielsen (2004) reported elevated levels of palmitic
430 acid in the milk of sows offered diets containing palm oil. The same effect
431 was not apparent in the present study, where there were no diet related
432 differences in the C16:0 content of milk at any time point despite the much
433 higher levels of C16:0 in the PO diet. There were some small transitory
434 differences in milk fatty acids 14:1, 18:3, 20:2 between diets, but these were
435 not consistent and followed no discernable pattern over the course of
436 lactation. The effects of time with respect to changes in milk total saturates,
437 monounsaturates and PUFA seen in the current study have been reported
438 previously (Skrzypczak et al., 2015) and are typical of changes seen in
439 porcine milk fat profiles with advancing stage of lactation.

440
441 *Growth and Development of Offspring*

In the present study birth weight of piglets were unaffected by manipulation of the maternal diet during late gestation. This is in agreement with findings of others following the provision of additional energy as fats during the latter stages of gestation (Seerley *et al.*, 1978; Quiniou *et al.*, 2008). However, Wang *et al.* (2016) reported a linear increase in piglet size with increasing energy provision during pregnancy, although it should be noted that total litter weight was not appreciably different between treatments.

In this trial piglet body composition (as indicated by FFM/kg) was also shown to be similar between treatments at birth. By d 7 of life, piglets suckling from C sows contained more fat, but by d 14 of life, piglets suckling from PO animals were the fattest (as indicated by their lower FFM/kg). The variation in body composition between birth and weaning between groups may be due to differences in milk yield and individual piglet suckling behavior. Previous studies have shown that piglets reared by sows receiving fat supplements during lactation grew faster; the composition of the increased weight gain was almost exclusively fat (Tilton *et al.*, 1999).

Conclusions

Results from this study suggests that the increase in energy intake by the sow, associated with palm oil supplementation, appears to alter milk composition, which may in turn influence early postnatal growth, development and body composition of suckling piglets. Elevated glucose concentrations in the sow seen during lactation may be suggestive of impaired glucose homeostasis. However, due to the limited replication within the current study

care needs to be exercised when interpreting data and as such further work is required in this area.

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Declaration of Interest

The authors declare that they have no conflict of interest

Ethics Statement

The study was subject to local ethical review and conducted in accordance with Imperial College's animal research policy and all procedures were conducted and conformed to the United Kingdom's Animal (Scientific Procedures) Act 1986.

Software and data repository sources

Data and models are not available in an official repository

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619 performance, and milk fat and protein output. Livestock Science. 194: 23-30.
620

621 **Table 1:** Fatty acid composition of sow diets

	Gestation diet		Lactation diet	
	Control	Palm Oil	Control	Palm Oil
12:O	ND	ND	0.26	0.15
14:O	0.53	0.80	0.85	0.90
15:O	0.09	0.15	ND	ND
16:O	18.24	28.07	20.45	25.58
16:1 (n-7)	0.32	0.40	0.24	0.21
17:1	0.08	0.08	0.07	0.09
18:O	3.14	3.73	5.22	5.32
18:1 (n-9)	19.98	29.35	32.81	35.80
18:2 (n-6)	50.33	33.46	34.83	28.88
18:3 (n-3)	4.76	2.93	3.97	1.71
20:O	ND	ND	0.53	0.47
20:1 (n-9)	0.66	0.46	0.36	0.54
20:2 (n-6)	0.25	0.08	ND	ND
20:3 (n-3)	0.04	ND	ND	ND
20:4 (n-6)	ND	ND	0.01	0.01
20:5 (n-3)	0.35	0.12	0.30	0.27
22:O	0.65	0.15	0.00	0.00
22:1 (n-9)	0.38	0.13	0.00	0.00
22:5 (n-3)	ND	0.10	0.00	0.00
22:6 (n-3)	0.21	ND	0.08	0.07
ΣS	22.65	32.90	27.31	32.43
ΣM	21.34	30.34	33.48	36.63
ΣP	55.94	36.68	39.21	30.94
Σn-6	50.58	33.54	34.84	28.90
Σn-3	5.36	3.15	4.36	2.04
P:S	2.47	1.11	1.44	0.95
n-6 :n-3	9.45	10.66	7.99	14.13

622 S = saturated fatty acids; M = monounsaturated fatty acids; P = Poly-unsaturated fatty acids;
623 ND = none detected. Values presented are mean percentages from 2 determinations of total
624 lipid fraction extracted from samples of diet
625
626
627

Table 2: Effect of maternal diet on piglet weight (kg) and fat-free mass/kg (FFM/kg)¹

Day ²	Weight (kg)		FFM/kg (arbitrary units)	
	C ³	PO ³	C	PO
0	1.64±0.04	1.61±0.04	79.1±0.9	77.9±0.9
7	2.91±0.08	2.91±0.08	66.4±0.8**	69.7±0.8**
14	4.79±0.12	5.05±0.12	65.8±0.6*	63.6±0.6*
21	6.91±0.17	6.72±0.17	60.2±0.7	61.1±0.7

¹ Data are presented as adjusted least squares means ± SEM (parity and litter size were analyzed as co-variates)

² Day of life (birth on day 0)

³ C= Control diet, PO = Palm oil diet.

* Denotes significant differences (*: $P<0.05$; **: $P<0.01$) between treatments (C vs. PO) in FFM/kg

638 **Table 3:** Effect of maternal diet on sow milk composition and milk energy¹

Day ³	Fat %		Protein %		Lactose %		Energy (MJ/kg) ²	
	C ⁴	PO	C	PO	C	PO	C	PO
3	8.0±0.3*	9.1±0.3*	4.8±0.2	4.9±0.2	5.4±0.1	5.1±0.1	5.70±0.25	5.64±0.25
7	7.8±0.5*	9.9±0.5*	5.0±0.4	4.6±0.4	5.2±0.4	5.2±0.4	4.99±0.15**	5.84±0.15**
14	8.1±0.5	8.5±0.5	4.4±0.1	4.7±0.1	5.7±0.1	5.5±0.1	5.06±0.15	5.34±0.15
21	7.0±0.4	8.4±0.4	4.6±0.2	4.6±0.2	5.5±0.1	5.4±0.1	4.69±0.16*	5.24±0.18*

639

640 ¹ Data are presented as adjusted least squares means ± SEM

641 ² MJ/kg of milk, MJ calculated as 0.0042 x (92.2 x fat + 61.3 x protein + 35.6 x lactose)

642 ³ Days since parturition

643 ⁴ C= Control diet, PO = Palm oil diet.

644 * Denotes significant differences (*: $P<0.05$; **: $P<0.01$) between treatments (C vs. PO) within
645 each milk fraction.

646 **Table 4:** Mean effects of sow diet during late gestation and lactation on the fatty acid profile (g/100g fatty acid) of their colostrum
647 and milk over a 21 day lactation.

Fatty Acid	Day 0		Day 3		Day 7		Day 14		Day 21		P-value	
	C	PO	C	PO	C	PO	C	PO	C	PO	Diet	Day
14:0	1.47±0.16	0.35±0.18	2.15±0.24	2.37±0.24	2.99±0.21	2.84±0.21	3.32±0.27	2.77±0.27	3.25±0.20	2.83±0.20	0.097	<0.001
14:1	0.03±0.04	0.02±0.01	0.11±0.03	0.13±0.03	0.20±0.03*	0.09±0.03*	0.24±0.04*	0.08±0.04*	0.20±0.04	0.09±0.04	0.001	0.003
16:0	22.1±0.40	23.4±0.47	26.1±1.21	29.2±1.21	31.3±1.32	29.9±1.32	33.6±1.74	32.4±1.74	34.7±1.45	33.6±1.45	0.618	<0.001
16:1(n-7)	3.24±0.55	3.47±0.63	6.29±0.67	7.92±0.67	8.94±0.90	7.47±0.90	9.77±1.04	7.64±1.04	10.22±0.81	8.26±0.81	0.066	<0.001
18:0	5.46±0.27	5.68±0.31	5.76±0.33	4.86±0.33	5.11±0.32	5.14±0.32	4.72±0.35	4.74±0.35	4.32±0.27	4.59±0.27	0.745	<0.001
18:1(n-9)	35.8±1.25	35.7±1.45	38.2±1.19	35.5±1.19	31.8±1.68*	34.8±1.68*	30.5±2.04	33.7±2.04	29.6±1.55	33.3±1.55	0.048	0.002
18:2(n-6)	26.0±1.28	24.3±1.47	16.9±0.76	16.0±0.76	15.6±0.77	15.8±0.77	14.4±0.86	15.2±0.86	14.3±0.57	14.0±0.57	0.761	<0.001
18:3(n-6)	0.37±0.04	0.24±0.05	0.05±0.04	0.08±0.04	0.04±0.02	0.05±0.02	0.03±0.01	0.02±0.01	0.04±0.02	0.03±0.02	0.370	<0.001
18:3(n-3)	1.46±0.25	1.69±0.29	1.13±0.05	1.03±0.05	1.20±0.08	1.13±0.08	1.14±0.08	1.07±0.08	1.11±0.05*	0.96±0.05*	0.050	0.003
20:1(n-9)	0.32±0.06	.21±0.06	0.39±0.04	0.35±0.07	0.33±0.07	0.34±0.07	0.35±0.10	0.32±0.06	0.32±0.07	0.29±0.07	0.416	0.262
20:2(n-6)	0.52±0.08	.47±0.09	0.57±0.05**	0.28±0.05**	0.42±0.06	0.33±0.06	0.31±0.07	0.29±0.07	0.33±0.05	0.26±0.05	0.049	0.007
20:4(n-6)	1.07±0.09	1.01±0.11	0.83±0.07	0.79±0.07	0.65±0.04	0.64±0.04	0.48±0.04	0.51±0.04	0.45±0.03	0.44±0.03	0.702	<0.001
20:5(n-3)	0.24±0.11	0.25±0.13	0.43±0.11	0.30±0.11	0.38±0.03	0.46±0.31	0.26±0.07	0.32±0.07	0.45±0.14	0.46±0.14	0.177	<0.001
22:1(n-9)	0.10±0.04	0.12±0.04	0.09±0.02	0.12±0.02	0.11±0.01	0.09±0.01	0.07±0.02	0.10±0.02	0.08±0.02	0.10±0.02	0.437	0.761
22:2(n-6)	0.05±0.22	0.44±0.25	ND	0.01±0.00	0.06±0.03	0.01±0.03	0.01±0.00	0.01±0.00	0.01±0.00	0.01±0.00	0.167	0.087
22:5(n-3)	0.43±0.05	0.34±0.06	0.32±1.03	0.29±0.03	0.24±0.03	0.26±0.03	0.21±0.02	0.19±0.02	0.20±0.02	0.17±0.02	0.177	<0.001
22:6(n-3)	0.33±0.05	0.32±0.06	0.22±0.02	0.24±0.02	0.02±0.03	0.18±0.03	0.17±0.03	0.14±0.03	0.16±0.03	0.12±0.03	0.662	<0.001
ΣS	29.2±0.31**	30.8±0.36**	34.2±1.24	36.7±1.24	39.7±1.36	39.7±1.36	41.9±1.82	40.2±1.82	42.5±1.44	41.3±1.44	0.049	<0.001
ΣM	39.6±1.58	39.6±1.83	45.1±0.82	44.1±0.82	41.4±1.20	42.9±1.20	40.9±1.46	41.9±1.46	40.4±0.97	42.1±0.97	0.276	0.001
ΣP	31.1±1.52	29.6±1.75	20.6±0.93	19.2±0.93	19.2±0.93	18.9±0.90	18.9±0.90	17.1±0.98	17.8±0.74	16.6±0.74	0.735	<0.001
Σn-6	28.8±1.37	27.2±1.58	18.7±0.85	17.6±0.85	17.1±0.82	17.1±0.82	15.5±0.88	16.24±0.88	15.3±0.66	15.0±0.66	0.577	<0.001
Σn-3	2.61±0.24	2.73±0.28	2.12±0.12	1.93±0.12	2.04±0.32	2.03±0.32	1.79±0.12	1.73±0.12	1.93±0.20	1.74±0.20	0.733	<0.001
P:S	1.06±0.05	0.96±0.06	0.61±0.04	0.54±0.04	0.48±0.04	0.50±0.04	0.41±0.04	0.46±0.04	0.40±0.03	0.41±0.03	0.735	<0.001
n-6:n-3	12.4±1.48	10.0±1.71	8.99±0.41	9.19±0.41	8.60±0.74	9.28±0.74	8.72±0.39	9.55±0.39	8.31±0.81	9.18±0.81	0.798	0.032

648 C= Control diet, PO = Palm oil diet; S = saturated fatty acids; M = monounsaturated fatty acids; P = Poly-unsaturated fatty acids; ND= none detected;

649 Data presented are adjusted least squares means ± SEM, Tukey's test was used to determine differences between treatment means.

650 * Denotes significant differences (*: $P<0.05$; **: $P<0.01$) between treatments (C vs. PO) within each sample time point.

651

652 **Table 5:** Effect of sow diet on circulating concentrations of leptin, insulin, insulin-like growth factor-1 (IGF-1), T₃, T₄, glucose and
653 lipids

Sample time	105d Gestation		Farrowing ¹		Lactation ¹		Weaning ¹		P-values	
	C	PO	C	PO	C	PO	C	PO	Diet	Time
Glucose (mM)	4.47±0.28	4.26±0.29	4.36±0.42	5.14±0.24	4.69±0.28*	5.41±0.26*	4.96±0.29	5.02±0.32	0.049	0.125
TAG (mM)	0.31±0.09	0.37±0.09	0.33±0.12	0.23±0.09	0.31±0.09	0.26±0.09	0.33±0.09	0.55±0.10	0.805	0.097
NEFA (mM)	0.22±0.05	0.21±0.05	0.34±0.07	0.29±0.05	0.24±0.05	0.36±0.05	0.18±0.05	0.27±0.06	0.588	0.075
Leptin (ng/ml)	3.29±0.41	4.14±0.42	3.10±0.84	3.79±0.42	2.94±0.45***	5.02±0.42***	3.24±0.41	4.34±0.46	0.035	0.774
Insulin (μIU/ml)	44.2±10.14	30.6±9.92	39.0±15.12	29.6±9.32	40.7±9.73	57.7±9.64	44.3±9.73	37.3±11.04	0.701	0.540
IGF-1 (ng/ml)	180±96.5	145±112.4	275±121.3	252±113.1	358±109.4	387±112.7	373±109.4	396±114.3	0.995	<0.001
T ₃ (ng/ml)	0.45±0.51	0.63±0.52	0.55±0.76	1.85±0.53	0.55±0.51	1.09±0.52	0.66±0.52*	2.40±0.53*	0.049	0.065
T ₄ (ng/ml)	30.9±1.83	32.2±1.88	24.1±2.73	30.5±1.90	29.4±1.83	32.7±1.88	31.7±1.83	33.7±1.98	0.514	0.485

654 ¹ Sample time: Farrowing = within 24 hours of parturition; Lactation = d7 of lactation; Weaning = d 28±3 of lactation)
655 C= Control diet, PO = Palm oil diet, TAG = Triacylglycerol, NEFA = Non esterified fatty acid, IGF-1 = Insulin-like growth factor 1, T₃ = Triiodothyronine, T₄ =
656 thyroxine.
657 * Denotes significant differences (*: $P<0.05$; **: $P<0.01$; ***: $P<0.005$) between treatments (C vs. PO) within each sample time point.