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Original article

Influence of feed intake during pregnancy and lactation on fat body reserve mobilisation, plasma leptin and reproductive function of primiparous lactating sows

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Abstract — From day 23 of pregnancy, 24 gilts received either a medium (M, $n = 16$) or a high (H, $n = 8$) level of feeding calculated to meet 115% or 190% of energy for maintenance, respectively. During lactation, all H sows were fed ad libitum (H-AL) whereas M sows were fed either ad libitum (M-AL, $n = 8$) or were restricted (M-RE, $n = 8$) to the amount of feed ingested by H-AL sows. Increased feed intake during pregnancy increased live weight, backfat thickness, and estimated body lipid and protein on days 4 and 25 of lactation ($P < 0.05$). It also resulted in lower feed intake and higher lipid mobilisation during lactation ($P < 0.05$) without a detrimental influence on milk production. Activities of malic enzyme and glucose-6-phosphate dehydrogenase from neck fat samples were higher in H than M sows on day 4 ($P < 0.05$). They decreased during lactation in H sows ($P < 0.05$). Mean diameter of adipocytes decreased during lactation in the 3 groups ($P < 0.05$) but did not differ between groups on days 4 and 25. Plasma leptin on days 4, 11, 18 and 25 was higher in H than in M sows ($P < 0.05$) but was not influenced by lactational feed intake. Neither measured characteristics of gonadotrophin secretion on day 22, nor of ovarian activity on day 26, were significantly influenced by the level of feeding during pregnancy or lactation.

fat tissue / sow / pregnancy / lactation / gonadotrophin

Résumé — Influence de la consommation d'aliment pendant la gestation et la lactation sur la mobilisation des réserves adipeuses, le taux de leptine plasmatique et la fonction de reproduction de truies primipares allaitantes. À partir du 23e jour de gestation, 24 cochettes reçoivent un niveau alimentaire moyen (M, $n = 16$) ou élevé (H, $n = 8$) destiné à couvrir respectivement 115 et 190 % des besoins énergétiques d'entretien. Pendant la lactation, toutes les truies H sont nourries à volonté (H-AL) alors que les truies M sont nourries à volonté (M-AL, $n = 8$) ou rationnées (M-RE,

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n = 8) de façon à recevoir la même quantité d'aliment que celle consommée par les truies H. L'augmentation de la consommation d'aliment en gestation permet un accroissement du poids vif, de l'épaisseur de lard, du contenu en lipides et en protéines de la carcasse à 4 et 25 jours de lactation ($P < 0,05$). Cela induit une réduction de la consommation d'aliment en lactation et une augmentation de la mobilisation des réserves lipidiques ($P < 0,05$) sans diminution de la production laitière. Les activités de l'enzyme malique et de la glucose-6-phosphate deshydrogénase mesurées dans du tissu gras prélevé au niveau du cou sont plus élevées chez les truies H que chez les M, au jour 4 ($P < 0,05$). Elles diminuent pendant la lactation chez les truies H ($P < 0,05$). Le diamètre moyen des adipocytes diminue pendant la lactation dans les trois groupes ($P < 0,05$) mais ne diffère pas entre groupes aux jours 4 et 25 de lactation. La concentration de leptine plasmatique mesurée aux jours 4, 11, 18 et 25 est plus élevée chez les truies H que M et n'est pas influencée par le niveau alimentaire de lactation. Aucune des caractéristiques de la sécrétion des hormones gonadotropes au jour 22 et de l'activité ovarienne 4 jours plus tard n'est influencée significativement par le niveau alimentaire de gestation et de lactation.

tissu adipeux / truie / gestation / lactation / gonadotropine

1. INTRODUCTION

Reproductive performance of sows have been greatly improved during the past 20 years as shown by an increase of 4.5 piglets weaned per productive sow per year in French commercial herds [14, 15]. This improvement is primarily due to a rise in the litter size at birth without any increase in the piglet mortality. In parallel, improved genetic potential to produce milk, and increased teat stimulation by the piglets have increased milk production in the sow [10]. Although the increase in litter size and milk production is accompanied by an augmentation of feed intake in lactation this is not sufficient to compensate for nutrient requirements and highly productive sows must mobilise body reserves [28, 33]. Therefore, increasing feed intake during pregnancy in order to improve body reserves at farrowing may help highly prolific sows to meet the nutrient demand during lactation and to avoid subsequent reproductive disorders. Unfortunately, increased body fat reserves at farrowing have a detrimental effect on the appetite of lactating sows [7, 9, 35, 40–42], and may increase the nutritional deficit during lactation and have negative effects on reproductive function (for review, see [30, 33]). In order to discriminate between the positive and negative consequences of increased feed intake during pregnancy, three feeding strategies were compared. One group of young females received a high level of feeding during pregnancy and was fed ad libitum during lactation, one group received a medium level of feeding during pregnancy and was fed ad libitum during lactation, the third group also received a medium level of feeding during pregnancy and was pair-fed during lactation with the first group. Characteristics of the metabolic status (feed intake, energy balance, lipogenic activity in fat tissue, plasma leptin) and reproductive function (ovarian development, plasma gonadotrophins) were recorded.

2. MATERIALS AND METHODS

2.1. Animals and experimental design

Four replicates of six Large White × Landrace crossbred gilts were used. Gilts were inseminated at 246 ± 4 days of age and 141 ± 7 kg live weight (mean ± SD, *n* = 24). During the whole experiment, these females were reared under artificial light provided by incandescent lamps for 12 hours per day. During gestation, gilts were housed in groups of 5. They were moved from the gestation to the farrowing rooms on day 107 ± 1 of pregnancy. Thereafter, females were

tethered in individual farrowing crates measuring 2 × 2.5 m. When necessary, parturition was induced by a single intra-muscular injection of 2 mL cloprostenol (Planate®, Pitman-Moore, USA) on day 114 of pregnancy. Farrowing occurred on days 114 or 115 of pregnancy. Within 48 hours after birth, litters were standardised to 11–12 piglets. From 22 days of age, creep feed was offered to the piglets. Water was freely available for the sows and piglets throughout the experimental period. Sows were slaughtered at the local abattoir on days 26 or 27 of lactation.

During pregnancy, all gilts received a diet containing 2.88 Mcal of metabolic energy (ME) per kg, 13% crude proteins and 0.58% lysine (Tab. I). They were fed individually for one hour, twice daily from Monday to Friday and once daily during weekends. Between service and day 22, gilts received 2.3 kg of feed per day. From day 23 of pregnancy until farrowing, gilts received either a medium (M, n = 4/replicate) or a high (H, n = 2/replicate) level of feeding. Feed allowance was calculated to meet 115% (1.7 to 2.3 kg of feed/day) and 190% (2.9 to 3.8 kg of feed/day) of the energy requirements for maintenance in M and H groups, respectively. However, a ceiling of 3.8 kg of feed was introduced in H sows to avoid refusals. Maintenance requirements were

Table I. Composition of the diets (on a fed basis).

Ingredients (%, air dried basis)	Pregnancy	Lactation
Wheat	22.39	22.80
Yellow corn	10.00	12.00
Barley	33.00	25.00
Wheat bran	15.00	10.00
Soybean meal	9.00	21.00
Fat	2.00	2.00
Beat molasses	–	3.00
Sugar beet pulp	5.00	–
Calcium carbonate	1.92	1.30
Dicalcium phosphate	0.74	1.90
Salt	0.45	0.45
Vitamin and mineral premix[a]	0.50	0.50
L-Lysine HCl, 78%	–	0.05
Main characteristics[b]		
Dry matter (%)	87.4	86.9
Crude protein (%)	13.0	17.5
Metabolic energy (Mcal·kg^{-1})	2.88	3.01
Amino acids[b]		
Lysine (%)	0.58	0.90
Methionine + cystine (%)	0.46	0.60
Threonine (%)	0.47	0.63

[a] Supplied the following amounts per kg of diet: vitamin A, 10 000 IU; vitamin D3, 1 500 IU; vitamin E, 30 mg, vitamin K3, 2 mg; thiamin, 2 mg; riboflavin, 4 mg; nicotin acid, 20 mg; d-pantothenic acid, 10 mg; pyridoxine, 3 mg; d-biotin, 0.2 mg; folic acid, 3 mg; vitamin B12, 0.02 mg; choline, 500 mg; Fe, 80 mg; Cu, 10 mg; Mn, 40 mg; Zn, 100 mg; Co, 0.1 mg; I, 0.6 mg and Se, 0.15 mg.
[b] Calculated values (INRA, 1989).

calculated for individual gilts on the basis of their metabolic body weight (105 kcal ME·kg^{-1} of body weight$^{0.75}$ [27]) measured on day 23 and at 2-week interval thereafter.

Throughout lactation, all sows received a diet providing 3.01 Mcal of ME·kg^{-1}, 17.5% crude proteins and 0.9% lysine twice daily, around 8.30 and 16.00 (Tab. I). All H sows were fed ad libitum whereas M sows were either fed ad libitum (M-AL, n = 2/replicate) or were restricted (M-RE, n = 2/replicate) to the amount of feed ingested by H-AL sows. On the day of farrowing (day 0), all females were fed 1.0 kg of the lactation diet. The day after, all females received 2.5 kg. On day 2, H-AL and M-AL sows received 4.0 kg of the lactation diet whereas M-RE sows received 3.5 kg. Thereafter, M-AL and H-AL sows were given free access to feed. Their feed refusals were weighed daily before the morning meal and feed intake was then calculated. From day 3, M-RE sows received the average feed intake of the M-AL sows on the previous day. For the analyses, the average daily feed intake was calculated between days 3 to 7 (week 1), days 8 to 14 (week 2), and days 15 to 21 (week 3) as well as on the overall period between days 3 to 24 (some sows were missing on day 25).

2.2. Measurements and sampling

Piglets were weighed at birth and at 25 ± 1 days of age. Sows were weighed at insemination, at the end of pregnancy (days 111 or 112) and during lactation on day 4 ± 1 postpartum and 21 days later. On the same days, backfat thickness was measured ultrasonically on each side, 6.5 cm from the midline at the level of the last rib (P_2). The mean of both sides was calculated and used for statistical analyses. At the same stages of lactation, a biopsy of subcutaneous fat tissue was obtained without anaesthesia at the neck level (on the same side for both biopsies at about 10 cm from the midline). About 1.3 mg of fat tissue was collected, immediately frozen in liquid N_2 and stored at –80 °C until analysed.

On day 6 ± 1 of lactation, an indwelling Silastic® (Dow Corning, Midland, MI, USA) catheter was surgically inserted into the right jugular vein of all sows under general anaesthesia as previously described [3]. Single blood samples were collected at 14.00 by venipuncture on day 4 ± 1 of lactation, and via the catheter, on days 11 ± 1, 18 ± 1, and 25 ± 1 of lactation. Serial blood samples were collected via the catheter every 15 min from 8.00 to 16.00 on day 22 ± 1 of lactation. Blood samples were collected in heparinised tubes, placed immediately on ice and centrifuged within 15 min. Plasma was stored at –20 °C until assay. Due to catheter incompetency in one H-AL sow, data related to serial blood sampling were analysed in 23 sows.

At slaughter, on day 26 ± 1 of lactation, both ovaries were collected within 15 min of death and weighed. Weights of the right and left ovaries were added for statistical analyses. The diameter of the largest ovarian follicle per sow was determined using a calliper.

2.3. Hormone assays

Plasma leptin concentrations were measured in single samples collected on days 4, 11, 18 and 25 of lactation. They were determined with a commercial kit (Multi-Species Leptin RIA kit, Linco Research Inc, USA) which was previously validated in the porcine species [32]. The percentage of cross reactions with porcine leptin was 67% as given by the manufacturer. The results are expressed in Human Equivalents (HE). Samples were analysed in duplicate within a single assay. Assay sensitivity, estimated as 90% of total binding, was 0.65 ng HE·mL^{-1} and the intraassay coefficient of variation (CV) was 6% at 2.1 ng HE·mL^{-1}.

Plasma LH concentrations were measured every 15 min and FSH concentrations every hour, in serial samples collected on day 22 of

lactation, using validated radioimmunoassays [3]. Samples were analysed in duplicate within single assays. Assay sensitivities, estimated as 90% of total binding, were 1.3 ng·mL^{-1} for LH and 1.4 ng·mL^{-1} for FSH. The intraassay CV was 8% at 2.2 ng·mL^{-1} for LH and 10% at 2.2 ng·mL^{-1} for FSH. The interassay CV was 16% at 1.5 ng·mL^{-1} for LH and 11% at 2.2 ng·mL^{-1} for FSH.

2.4. Enzyme assays and adipocyte diameters

Lipogenic enzyme activities were determined in the biopsies of subcutaneous adipose tissue. Weighed quantities of adipose tissue were homogenised in 0.25 M sucrose buffer and centrifuged at 30 000 *g* for 40 min. Supernatants were analysed for malic enzyme (MENZ, EC 1.1.1.40) and glucose-6-phosphate dehydrogenase (G6PDH, EC 1.1.1.49) activities according to methods previously described [11, 18]. Acetyl-CoA-carboxylase (ACX, EC 6.4.1.2) activity was assayed by the $H^{14}CO_3^-$ fixation method [4]. MENZ and G6PDH activities were expressed as micromoles of NADPH produced per gram adipose tissue per minute. ACX activity was expressed as nanomoles of bicarbonate incorporated per gram adipose tissue per minute. Due to technical difficulties, four out of 48 fat samples were not analysed for lipogenic enzymes.

For the determination of adipocyte size, tissue was mechanically minced and treated with 2% osmium tetroxide according to a method already described [16]. For each animal, 400 adipocytes were measured at random with an image analysis system using the NIH Image 1.56 software (J.C. Folmer, INRA Le Magneraud, France).

2.5. Calculations and statistical analyses

Body lipid, protein and energy were estimated on days 4 and 25 of lactation from the body weight and backfat thickness measurements using the equations proposed by Dourmad et al. [8]: lipid (kg) = $-26.4 + 0.221 \times EBW + 1.331 \times P_2$; energy (Mcal) = $-257 + 3.267 \times EBW + 10.99 \times P_2$; protein (kg) = $2.28 + 0.178 \times EBW - 0.333 \times P_2$, where EBW (kg) represents the sow empty live weight estimated from the live weight ($EBW = 0.905 \times BW^{1.013}$) and P_2 (mm) is the backfat thickness at the level of the last rib.

Profiles of LH were analysed as previously described [31]. Mean concentrations of FSH and characteristics of LH secretion (mean and basal concentrations, number of pulses) were calculated and used for statistical analyses. Data were analysed by analysis of variance using the GLM procedure of SAS [37]. All models included the effects of treatment and replicate and the treatment × replicate interaction. For plasma leptin concentration, the effect of the day of sampling and the day × treatment interaction was also analysed and tested against the within-animal error term. In all analyses, when the treatment × replicate interaction was not significant, it was removed from the model. When the treatment effect was significant, differences between means were assessed with the Scheffé test. Variations between days within experimental groups were assessed using a paired-t-test. Correlation coefficients were calculated between carcass characteristics, feed intake, hormonal data or ovarian weight. The Spearman coefficient was used for the correlation between the number of LH pulses (discontinuous variable) and ovarian weight. The Pearson coefficient was used for all other correlations.

Results are expressed as means ± SEM.

3. RESULTS

Higher feed allowance during the main part of gestation had a marked positive effect on the increase in live weight and backfat thickness during pregnancy (Tab. II). As a

Table II. Effects of feed intake during pregnancy (M or H) on sow live weight and backfat thickness during pregnancy and lactation and on feed intake during lactation (means ± SEM, n = 7 or 8 per group).

	Treatment			
	M-AL	M-RE[1]	H-AL[2]	P-value
Live weight (kg)				
Day 23 p.c. (allotment)	149.2 ± 2.5	151.2 ± 1.8	148.8 ± 2.3	0.54
Day 112 p.c.	196.0 ± 3.4^{a}	196.1 ± 3.1^{a}	239.9 ± 2.7^{b}	0.001
Day 4 p.p.	181.7 ± 3.7^{a}	182.8 ± 3.4^{a}	214.0 ± 2.9^{b}	0.001
Day 25 p.p.	173.8 ± 3.8^{a}	168.2 ± 2.5^{a}	197.4 ± 4.0^{b}	0.001
Variation between days 23 and 112 p.p.	46.8 ± 2.8^{a}*	45.8 ± 1.7^{a}*	91.1 ± 1.4^{b}	0.001
Variation around farrowing	–14.3 ± 2.8^{a}*	–15.3 ± 3.8^{ab}*	–25.9 ± 2.4^{b}*	0.026
Variation between days 4 and 25 p.p.	–7.9 ± 2.2^{a}*	–17.0 ± 2.8^{b}*	–16.7 ± 3.1^{ab}*	0.029
Backfat thickness (mm)				
Day 23 p.c. (allotment)	15.9 ± 0.6	15.6 ± 0.6	17.3 ± 0.8	0.2
Day 112 p.c.	16.7 ± 0.7^{a}	16.6 ± 0.9^{a}	22.4 ± 1.0^{b}	0.001
Day 4 p.p.	15.3 ± 0.4^{a}	15.1 ± 0.6^{a}	25.1 ± 0.8^{b}	0.001
Day 25 p.p.	13.0 ± 0.7^{a}	11.4 ± 0.6^{a}	18.1 ± 0.5^{b}	0.001
Variation between days 23 and 112 p.p.	0.8 ± 0.8^{a}	1.1 ± 0.8^{a}	6.1 ± 1.0^{b}*	0.001
Variation around farrowing	–1.4 ± 0.6†	–1.9 ± 0.6*	–0.9 ± 0.6	0.64
Variation between days 4 and 25 p.p.	–2.3 ± 0.5^{u}*	–3.6 ± 0.3^{uv}*	–4.3 ± 0.8^{v}*	0.054
Feed intake (kg·day^{-1})				
Days 3 to 7 p.p.	4.8 ± 0.2^{a}	3.8 ± 0.1^{b}	3.4 ± 0.2^{b}	0.001
Days 8 to 14 p.p.	5.7 ± 0.3^{a}	4.8 ± 0.1^{b}	5.0 ± 0.4^{b}	0.010
Days 15 to 21 p.p.	6.8 ± 0.3	5.8 ± 0.3	6.2 ± 0.4	0.068
Days 3 to 24 p.p.	5.9 ± 0.2^{a}	5.0 ± 0.1^{b}	5.1 ± 0.3^{b}	0.008

[1] Number of observations = 7 for live weight at days 112 p.c., 25 p.p., its variation around farrowing and during lactation, and for backfat at day 112 p.c. and its variation during gestation and around farrowing.
[2] Number of observations = 7 for live weight at day 25 p.p. and its variation during lactation.
[a,b] Within a row, means lacking a common superscript letter differ at $P < 0.05$.
[u,v] Within a row, means lacking a common superscript letter differ at $P < 0.06$.
* $P < 0.05$ for the differences between days within groups.
† $P < 0.1$ for the differences between days within groups.

consequence, H sows were heavier and fatter at the end of gestation than M sows ($P < 0.001$). Live weight loss around farrowing was also significantly increased in H sows whereas backfat loss did not differ between experimental groups.

3.1. Sow and litter performance during lactation

The number of live piglets at birth (11.5 ± 0.7 piglets) and on day 2 of lactation (10.5 ± 0.3 piglets) did not differ between the treatment groups ($P > 0.1$). Similarly the average daily gain of the piglets (229 ± 8 g·day^{-1}) and of the litters during lactation (2480 ± 80 g·day^{-1}) were similar in the three experimental groups ($P > 0.1$).

Daily feed intake increased progressively between the first and third weeks of lactation in the three experimental groups (Tab. II, $P < 0.01$). As planned in the experimental design, feed intake was similar in M-RE and H-AL sows throughout lactation (Tab. II). Mean feed intake measured between days 3 and 24 of lactation was about 16% higher

in M-AL than H-AL sows ($P < 0.05$). This difference in feed intake between treatments decreased from 40% (week 1) to 14% (week 2) and 10% (week 3).

A higher feed intake during pregnancy (H vs. M sows) had a marked positive effect on sow live weight, backfat thickness and the lipid, protein and energy contents of the carcass estimated shortly after farrowing (Tabs. II and III). This effect was still present at the end of lactation, even though the difference was less marked between H-AL and M-AL sows than between H-AL and M-RE sows. Lipid and energy losses during lactation were similar in sows with the lower feed intake during lactation (M-RE and H-AL sows). They were significantly higher in H-AL than in M-AL sows whereas differences between M-AL and M-RE sows were close to significance ($P = 0.11$ for lipid losses and $P = 0.07$ for energy losses).

During the first week of lactation, feed intake of sows that were fed ad libitum (H-AL and M-AL sows) was negatively correlated with their backfat thickness ($r = –0.84$, $n = 16$, $P < 0.001$) and body lipid content ($r = –0.70$, $n = 16$, $P < 0.002$) determined on day 4 of lactation. Similarly, mean feed intake measured between days 3 and 24 of lactation was negatively correlated with backfat ($r = –0.62$, $n = 16$, $P < 0.01$) and body lipid content ($r = –0.49$, $n = 16$, $P < 0.05$) on day 4. In contrast, there was no clear relationship between feed intake and body protein content on day 4 ($P > 0.10$).

Table III. Effects of feed intake during pregnancy (M or H) and lactation (AL or RE) on sow estimated composition during lactation (means ± SEM, $n = 8$ per group).

	Treatment			
	M-AL	M-RE[1]	H-AL[1]	*P*-value
Lipid (kg[2])				
Day 4	32.9 ± 1.3[a]	33.5 ± 1.1[a]	49.5 ± 1.3[b]	0.001
Day 25	28.1 ± 1.7[a]	25.3 ± 1.0[a]	39.9 ± 0.5[b]	0.001
Variation between days 4 and 25	–4.8 ± 0.9[a]*	–8.2 ± 0.7[ab]*	–9.6 ± 1.6[b]*	0.019
Protein (kg[2])				
Day 4	28.5 ± 0.6[a]	29.1 ± 0.5[a]	31.8 ± 0.9[b]	0.003
Day 25	27.9 ± 0.5[a]	27.3 ± 0.4[a]	30.4 ± 0.9[b]	0.003
Variation between days 4 and 25	–0.6 ± 0.4	–1.8 ± 0.5*	–1.4 ± 0.5*	0.095
Energy (Mcal[2])				
Day 4	486 ± 15[a]	496 ± 12[a]	669 ± 11[b]	0.001
Day 25	435 ± 19[a]	404 ± 11[a]	566 ± 9[b]	0.001
Variation between days 4 and 25	–51 ± 10[a]*	–92 ± 10[ab]*	–103 ± 17[b]*	0.017

[1] Number of observations = 7.
[2] The chemical composition of the body weight on days 4 and 25 of lactation as well as its variation were calculated from the body weight and backfat thickness measurements using the equations proposed by Dourmad et al. [8]: lipids (kg) = $–26.4 + 0.221 \times EBW + 1.331 \times P_2$, energy (Mcal) = $–257 + 3.267 \times EBW + 10.99 \times P_2$, protein (kg) = $2.28 + 0.178 \times EBW – 0.333 \times P_2$, where EBW (kg) represents the sow empty live weight ($EBW = 0.905 \times BW^{1.1013}$, BW: live weight in kg) and P_2 (mm) is the fat thickness measured at the last rib.
[a,b,c] Within a row, means lacking a common superscript letter differ.
* $P < 0.05$ for the differences between days within groups.

Table IV. Effects of feed intake during pregnancy (M or H) and lactation (AL or RE) on lipogenic enzyme activities and adipocyte diameter from subcutaneous fat tissue from primiparous lactating sows (means ± SEM).

	Treatment			
	M-AL[1]	M-RE[2]	H-AL[3]	*P*-value
Acetyl-Co-carboxylase (nmol HCO_3^-·g^{-1}·min^{-1})				
Day 4	0.123 ± 0.015	0.132 ± 0.019	0.173 ± 0.020	0.18
Day 25	0.098 ± 0.011[a]	0.084 ± 0.011[ab]	0.058 ± 0.006[b]	0.047
Variation between days 4 and 25	–0.025 ± 0.017	–0.051 ± 0.027	–0.106 ± 0.024*	0.077
Malic enzyme (µmol NADPH·g^{-1}·min^{-1})				
Day 4	1.37 ± 0.09[a]	1.17 ± 0.13[a]	2.91 ± 0.37[b]	0.001
Day 25	1.08 ± 0.11[ab]	0.86 ± 0.08[a]	1.46 ± 0.17[b]	0.035
Variation between days 4 and 25	–0.29 ± 0.16	–0.33 ± 0.24	1.25 ± 0.42*	0.050
Glucose-6-phosphate dehydrogenase (µmol NADPH·g^{-1}·min^{-1})				
Day 4	1.44 ± 0.11[a]	1.31 ± 0.15[a]	2.55 ± 0.30[b]	0.006
Day 25	0.95 ± 0.11	0.89 ± 0.15	0.87 ± 0.11	0.82
Variation between days 4 and 25	–0.46 ± 0.18[a]	–0.37 ± 0.33[a]	–1.54 ± 0.39[b]*	0.011
Mean diameter of the adipocytes (µm)				
Day 4	77 ± 2	82 ± 3	76 ± 2	0.20
Day 25	64 ± 3	59 ± 3	62 ± 4	0.50
Variation between days 4 and 25	–13 ± 4*	–24 ± 3*	–14 ± 4*	0.15

[1] Number of observations = 8 for the three enzymes and adipocytes on both days.
[2] Number of observations = 7 for the three enzymes and 6 for adipocytes on day 4; 6 for the three enzymes and 8 for adipocytes on day 25.
[3] Number of observations = 8 for the three enzymes and adipocytes on day 4; 7 for the three enzymes and 8 for adipocytes on day 25.
[a,b,c] Within a row, means lacking a common superscript letter differ at $P < 0.05$.
* $P < 0.05$ for the differences between days within groups.

3.2. Lipogenesis and adipocyte diameter

At the beginning of lactation (day 4), MENZ and G6PDH were higher ($P < 0.01$) in sows consuming the most feed during pregnancy whereas ACX was similar in the three experimental groups (Tab. IV). At the end of lactation, ACX and G6PDH were the lowest whereas MENZ was the highest in H-AL sows. Between days 4 and 25, all enzyme activities decreased in the three experimental groups but differences were more marked in H-AL sows and reached significance only in this latter group (Tab. IV). Mean diameter of the adipocytes did not vary among experimental groups at the beginning nor at the end of lactation ($P > 0.1$). It decreased ($P < 0.02$) between days 4 and 25 of lactation in the three groups of sows.

3.3. Plasma leptin

There was no treatment × day interaction for plasma leptin ($P > 0.1$). Plasma leptin decreased as lactation progressed (Fig. 1). It did not differ between M-RE and M-AL groups but was lower in M-RE sows ($P < 0.05$) and in M-AL sows ($P = 0.07$) than in H-AL sows. In sows which were fed

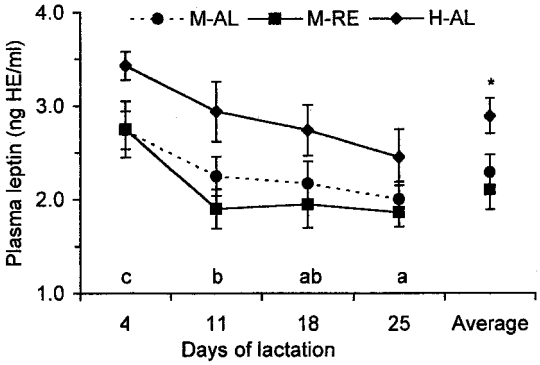

Figure 1. Day-related variations in plasma leptin in primiparous lactating sows submitted to various levels of feeding during pregnancy (M or H) and/or lactation (AL or RE); a,b,c, days with different superscripts differ at $P < 0.05$; * $P < 0.05$ for the treatment effect; $n = 8$ per group.

ad libitum during lactation (M-AL and H-AL sows), there was no clear relationship between plasma leptin and feed intake measured at various moments during lactation ($P > 0.1$). Across the three experimental groups, plasma leptin was positively correlated with backfat thickness and lipid body content on days 4 and 25 of lactation (Tab. V, $P < 0.05$). It was also positively correlated with adipocyte diameter on day 4 ($P < 0.05$) but not on day 25 ($P > 0.1$).

3.4. Gonadotrophin profiles and ovarian development

On day 22 of lactation, mean plasma FSH did not differ ($P > 0.1$) between experimental treatments (1.99 ± 0.07 ng·mL^{-1}, $n = 23$). Similarly, basal plasma LH (1.96 ± 0.05 ng·mL^{-1}, $n = 23$) and the number of LH pulses during 8 hours (0.74 ± 0.18 pulses, $n = 23$) were similar in the three groups of sows ($P > 0.1$). Mean plasma LH tended to be higher ($P < 0.1$) in M-AL (2.11 ± 0.05 ng·mL^{-1}, $n = 8$) than in H-AL sows (1.86 ± 0.06 ng·mL^{-1}, $n = 7$) and was intermediate in M-RE sows (1.98 ± 0.10 ng·mL^{-1}, $n = 8$). Across experimental groups, correlations between plasma leptin measured on day 25 and characteristics of gonadotrophin profiles were low (FSH: $r = –0.05$, $P > 0.1$; mean LH: $r = –0.19$, $P > 0.1$; basal LH: $r = –0.22$, $P > 0.1$; number of LH pulses: $r = 0.39$, $P < 0.1$).

On day 26 post-partum, the weight of both ovaries (9.0 ± 0.50 g, $n = 24$) and the diameter of the largest follicle (4.3 ± 0.1 mm, $n = 24$) were similar in the three experimental

Table V. Correlations between plasma leptin, and backfat thickness, adipocyte diameter or activity of lipogenic enzymes in primiparous lactating sows across the three experimental groups ($n = 21$ to 24).

Plasma leptin	Backfat thickness	Lipid body content	Adipocyte diameter	ACX activity	MENZ activity	G6PDH activity
At day 4 of lactation[1]	0.45*	0.50*	0.42*	0.39†	0.39†	0.40†
At day 25 of lactation[2]	0.43*	0.46*	0.10	0.04	–0.15	0.17

[1] Correlations between plasma leptin and metabolic criteria measured at day 4 of lactation.
[2] Correlations between plasma leptin and metabolic criteria measured at day 25 of lactation.
* $P < 0.05$.
† $P < 0.1$.

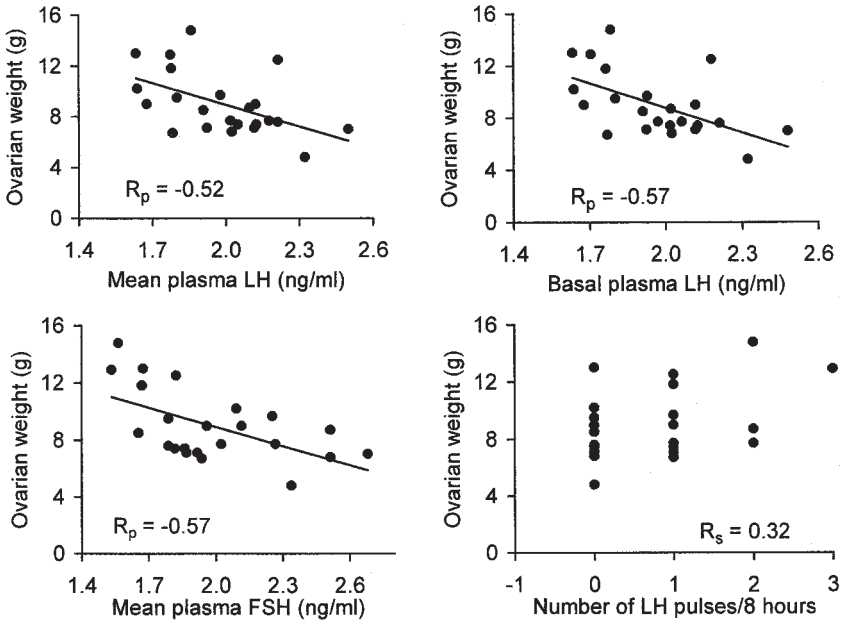

Figure 2. Relationships between characteristics of plasma gonadotrophins and the ovarian weight in lactating primiparous sows, n = 23; r_p Pearson coefficient of correlation; r_s Spearman coefficient of correlation.

groups. Ovarian weight was negatively correlated with mean plasma FSH, mean LH and basal LH ($P < 0.05$, Fig. 2).

4. DISCUSSION

The present experiment indicates that a higher feed intake during pregnancy increases the amount of body reserves at farrowing but results in lower voluntary feed intake and higher mobilisation of body reserves during lactation in agreement with previous data [7, 9, 36, 40–42]. In these previous studies, backfat thickness in fat sows at farrowing was in a similar range to ours (26.0 mm vs. 29.2 mm in [7], 28.1 mm in [9], 24.3 mm in [35, 36], 29.4 mm in [41], about 30.0 mm in [42]). Since high energy intake during the first gestation inhibits appetite during lactation and may be detrimental to development of mammary secretory tissue at the end of gestation [39], it could decrease milk production and hence growth of the litters. Our data, in agreement with those of most previous studies [7, 9, 40–42], do not support this hypothesis. Increasing energy intake during gestation and fatness at farrowing had a non significant detrimental influence on milk production and litter growth in only one experiment [36]. Our data also confirmed that body energy reserves after 25 days of lactation may still be higher in sows receiving more food during pregnancy. Our data, which are in agreement with some studies [7, 40] but not with others [9, 42] show that the effect of the gestational feed intake on appetite decreases as lactation progresses. They also demonstrate that under our experimental conditions (nutrition and litter growth), both protein and adipose tissues were mobilised but that lipid tissue contributed to a larger proportion of the mobilised

tissues. When the rate of lipid mobilisation was calculated per kg of metabolic live weight and per day, it was in the same range (around 5 and 8 $g \cdot kg^{-0.75} \cdot day^{-1}$ in M and H sows, respectively) as in lactating dairy cows [5].

Two mechanisms may explain the influence of feed intake during pregnancy on appetite during lactation. Firstly, it may be proposed that body fat reserves and leptin production at farrowing are higher in sows with higher feed intakes during pregnancy and that the high leptin concentrations inhibit appetite during lactation. Supporting this hypothesis, our data show that plasma leptin concentrations were higher in sows receiving more food during pregnancy and were positively correlated with backfat thickness and lipid body content of the carcass either after 4 or 25 days of lactation. Moreover, feed intake of sows which were fed ad libitum during lactation was negatively correlated with their backfat thickness and body lipid content estimated at the beginning of lactation. This inverse relationship between fatness and appetite has already been described in lactating sows [7, 28] and cows [2, 13] and fits well with the lipostatic theory of long term regulation of feed intake. However, we did not observe any clear relationship between plasma leptin, measured either at day 4 or day 25 of lactation and feed intake. Therefore, leptin may be one of the mediators originating from the adipose tissue and acting on the brain to regulate appetite but other signals from the adipose tissue are likely to be involved (for review, see [1]).

The second hypothesis, developed by Weldon et al. [40, 41], is that postpartum hypophagia of sows which are fat at farrowing is due to an increased resistance to insulin and a reduced tolerance to glucose. It is clear that these phenomena allow plasma glucose to remain high after feed consumption which is supposed to delay the onset of the following meal. Moreover, insulin resistance favours mobilisation of stored nutrients into the plasma which is likely to reduce feed intake. A reduction in glucose tolerance and in spontaneous feed intake, which is in agreement with this theory, is observed in lactating sows that are fat at farrowing due to a liberal scale of feeding during rearing [19, 20] or gestation [40, 41]. Leptin may be implicated in this phenomenon since this hormone is known to attenuate the insulin action in various insulin-sensitive cell types (for review, see [17]).

Circulating concentrations of leptin were similar to those previously described in lactating sows [9, 23]. In contrast to Mao et al. [23] we did not show any effect of feed intake (M-AL vs. M-RE sows) on leptin concentrations measured after feeding. This discrepancy is probably due to a smaller difference in feed intake between experimental groups in our study. The positive correlation between plasma leptin and backfat thickness at the beginning of lactation agrees with previous data [9] and fits well with the theory that adipose tissue is the main site of leptin production (for review, see [17]). However, contrary to Estienne et al. [9], a high correlation was also maintained at day 25 of lactation in our sows. It should be noted that postprandial leptin may increase with feed intake [21, 23]. Therefore, body fat reserves may have a negative influence on concentrations of plasma leptin through inhibition of appetite. It is likely that the balance between the positive and negative effects of body fat reserves on leptin release varies as lactation progresses. In a previous study [9], the positive influence of body fat mass might have been counterbalanced during the 4th week of lactation by the marked reduction in appetite in fat sows. This phenomenon has probably played a minor role in our study since the difference in feed intake between fat and lean sows was very low at this stage of lactation.

In adipose tissue, the synthesis (lipogenesis) and incorporation of fatty acids into storage triglycerides and the release of fatty

acids (lipolysis) is a continuous cycle. The rates of these pathways adapt to changes in substrate intake and demand for energy during lactation to result in accretion or mobilisation of the adipose tissue reserves (for review, see [5, 22]). In the pig as in other mammalian species, fatty acids are generally stored during pregnancy and mobilised during lactation [5, 22]. It is generally accepted that the increase in backfat adipose tissue in the swine is mostly due to a hypertrophy of adipocytes [24]. However, in our study, the increase in backfat depth at farrowing in sows receiving the high level of feeding during pregnancy was not accompanied by an increase in adipocyte diameter. It can be hypothesised that small adipocytes (15 to 20 μm) were stimulated to store fatty acids in our females receiving a high level of energy. As a consequence, the average diameter was not modified whereas the number of active adipocytes was probably increased. In contrast, when gilts are feed restricted during pregnancy (100 vs. 130% of the maintenance energy requirements), mean diameter of adipocytes from subcutaneous adipose tissue is slightly reduced (113 vs. 120 μm at day 105 p.c.) [29]. Our data show a concomitant decrease in backfat depth (15 to 25%) and adipocyte diameter (15 to 30%) during lactation. Previously, Parmley et al. [29] also observed a reduction in adipocyte diameter (13 to 20%) in sows which were feed restricted during lactation but not in those which were well fed. The relatively low milk production as demonstrated by the litter gain (1750 g·day^{-1}) probably explains that well-fed sows did not mobilise fat tissue in this latter study on the contrary to ours (litter gain = 2480 g·day^{-1}).

To assess changes in lipogenesis, the activity of various key enzymes involved in fatty acid synthesis was measured. The co-enzyme NADPH is necessary for the biosynthesis of fatty acids and, in the pig, MENZ and G6PDH are recognised to be the main enzymes that supply NADPH [43]. Activities of both enzymes were lower in the fat tissues of our lactating sows than in growing pigs [26]. In dairy cows, G6PDH and MENZ activities of the adipose tissue are also very low at the beginning of lactation (week 3) to save glucose and to favour the supply of fatty acids and energy to the mammary glands [6]. In our experiment, higher feed intake during pregnancy stimulated activities of both enzymes measured 4 days after farrowing. This difference in lipogenic activity is probably due to a difference in enzymatic activity that already exists during pregnancy. Indeed, Parmley et al. [29] observed that incorporation of glucose into fatty acids and palmitate incorporation into triglycerides measured in vitro in subcutaneous fat tissue from pregnant gilts (day 105 p.c.) increased with the level of feeding. Both G6PDH and MENZ activities decreased as lactation progressed and the variation was more marked in sows which were fatter at farrowing (H-AL group). As a consequence, G6PDH activity no longer differed between groups of sows after 25 days of lactation. Our data do not show any clear relationship between plasma leptin and enzyme activities either at day 4 or at day 25 of lactation. This suggests that the lipogenic potential of adipocytes and leptin secretion by these cells are independent.

The enzyme ACX catalyzes the first step of the fatty acid biosynthesis and is considered as a rate-limiting enzyme for lipogenesis in pigs [24]. Compared with growing pigs [25], ACX activity in adipose tissue was relatively low throughout lactation. Feed intake during pregnancy had no clear effect on ACX activity at the beginning of lactation. It decreased progressively during lactation in all groups of sows but the decrease was more marked in sows with the higher feed intake during pregnancy.

To our knowledge, this is the first report studying the influence of body reserves at farrowing on the reproductive axis of sows having the same feed intake during lactation (M-RE vs. H-AL sows). Our data do not show any effect of these body reserves on any characteristics of gonadotrophin

release nor of ovarian activity during the 4th week of lactation. Similarly, feed intake during lactation (M-AL vs. M-RE sows) had no effect on any characteristics of the reproductive axis. The reduction in the feed intake of M-RE sows (–0.9 kg·day^{-1}) was moderate and probably too low to influence the gonadotrophin release and ovarian activity during lactation [30]. Similarly, simultaneously varying fat reserves at farrowing and feed intake during lactation (M-AL vs. H-AL sows) had no clear influence on the gonadotrophin and ovarian characteristics of sows during the 4th week of lactation. There was only a tendency for lower mean LH in sows which were fatter at farrowing. More profound negative effects of body fat at farrowing on LH secretion during lactation and one day after weaning were observed by Xue et al. [42]. This difference between studies may be related to the sow average feed intake. In a previous experiment, the feed intake of fatter sows was much lower than that of the fatter sows in our experiment (3.2 vs. 5.1 kg·day^{-1}) and was in a range that was likely to inhibit gonadotrophin release [30, 34, 44].

To determine whether body reserves may influence gonadotrophin secretion through variations in leptin as suggested in numerous studies (for reviews, see [1, 17]), correlations between plasma leptin on day 25 and gonadotrophin characteristics on day 22 were calculated. These correlations were low and non significant. In contrast, Mao et al. [23] observed a positive association between plasma leptin and mean LH concentrations on days 21 and 28 of lactation. However, in their study, low plasma leptin concentrations were associated with a marked reduction in feed intake (2.7 vs. 5.8 kg·day^{-1}) which itself is known to inhibit LH secretion [30, 34, 44].

Our results show negative correlations between the ovarian weight on the one hand, mean FSH, mean LH or basal LH on the other hand. These correlations were expected and clearly illustrate the negative feedback exerted by the ovaries on the pituitary release of LH and FSH. Similarly, lower FSH concentrations were observed in lactating sows with higher plasma oestradiol [26] as well as negative correlations between plasma FSH measured during lactation or within 48 hours after weaning and follicular characteristics (diameter of 10 biggest follicles, follicular fluid oestradiol, aromatase activity) measured 48 hours after weaning [12]. In addition, negative relationships between plasma oestradiol and inhibin on the one hand and plasma FSH on the other hand were observed on the days after weaning [38]. Therefore, low mean concentrations of LH and FSH in lactating sows may indicate that the overall hypothalamo-pituitary-ovarian axis is strongly inhibited or that folliculogenesis is very active and inhibits the basal release of gonadotrophins. Even though non significant, the correlation between the ovarian weight and the number of LH pulses was positive in accordance with the stimulatory influence of LH pulses on folliculogenesis [12, 30]. This result also suggests that ovarian steroids may exert positive effects on LH pulse frequency [12].

In conclusion, our data show that a high feed intake during pregnancy resulting in high body fat reserves at farrowing has no clear detrimental influence on the reproductive axis observed during the 4th week of lactation as well as on the overall milk production despite the reduction in feed intake especially at the beginning of lactation. The role of leptin in mediating the effect of body reserves on the reproductive function and appetite is not clear and requires further investigation.

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