Parturition body size and body protein loss during lactation influence performance during lactation and ovarian function at weaning in first-parity sows

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Parturition body size and body protein loss during lactation influence performance during lactation and ovarian function at weaning in first-parity sows[1]

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ABSTRACT: We investigated the effect of body protein mass at parturition and different degrees of body protein loss in lactation on sow performance. In a 2 × 2 factorial arrangement, 77 Genex gilts were fed to achieve either a standard or high body mass at parturition and to lose either a moderate (MPL) or high (HPL) amount of protein in lactation. Pregnant gilts were fed either 24.4 MJ of ME, 266 g of CP, and 11 g of lysine/d or 34.0 MJ of ME, 436 g of CP, and 20 g of lysine/d resulting in divergent ($P < 0.01$) live weights (165 vs. 193 kg) and calculated protein masses (24.3 vs. 30.0 kg) and slightly different backfat depths (20.0 vs. 22.8mm; $P < 0.05$) at parturition. Diets fed during lactation were formulated to deliver 731 g of CP and 37 g of lysine/d or 416 g of CP and 22 g of lysine/d to induce differential body protein mobilization. Sows were slaughtered at weaning (d 26), and the weight of the organs and the lean, fat, and bone in five primal cuts was measured. The external diameter of the eight largest follicles on each ovary was recorded, and the follicular fluid from these follicles was collected, weighed, and analyzed for estradiol. Losses in lactational live weight (26 vs. 20 kg; $P < 0.01$) and calculated protein mass (17.8 vs. 10.7%; $P < 0.001$) were greater, and the carcass lean mass at weaning was 10% lighter ($P < 0.05$) in HPL sows. Backfat (5.1 ± 0.8 mm; $P = 0.29$) and calculated fat mass (25.8 ± 1.5%; $P = 0.84$) losses did not differ between treatments. Both sow body mass ($P < 0.05$) and lactation protein loss ($P < 0.01$) affected litter growth rate. Litter growth rate decreased ($P < 0.05$) at the end of lactation in HPL sows once these sows had lost 10 to 12% of their calculated protein mass. Ovarian follicular development was most advanced in high body mass sows that lost the least protein; these sows had the heaviest ($P < 0.05$) uterine weight and highest ($P < 0.05$) follicular fluid estradiol concentration. Follicular development was least advanced in standard body mass sows that lost the most protein. These sows had the lowest ($P < 0.05$) muscle:bone ratio at weaning and likely lost the largest proportion of their muscle mass compared wth the other treatments. In conclusion, ovarian function at weaning and litter performance was higher in high body mass sows and in sows that lost the least protein in lactation, suggesting that a larger lean mass may delay the onset of a decrease in performance in sows that lose protein in lactation.

Key Words: Lactation, Litter Performance, Pregnancy, Protein Intake, Reproductive Performance, Sows

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Introduction

Mobilization of body reserves allows lactation to occur with some independence from any limitation in dietary nutrient supply. However, depletion of maternal reserves may eventually compromise both the current lactation and subsequent reproduction (King and Mar-

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tin, 1989; Jones and Stahly, 1999a,b). We recently explored the role of protein mobilization on lactation and reproduction in first-parity sows (Clowes et al., 1998; 2003). Our results suggested that mobilization of less than 9 to 12% of a sow's protein mass present at parturition is without consequence for litter growth. A variety of ovarian variables, including number and size of follicles, follicular fluid volume and estradiol levels, and the ability of follicular fluid to advance in vitro maturation of oocytes, were also unaffected ($P > 0.25$). However, a decline in milk protein concentration, litter growth, and ovarian variables ensued at a loss of 12% or more of the sow's protein mass (Clowes et al., 2003).

Even at maximal voluntary feed intakes, contemporary commercial dam-line sows often mobilize body protein during lactation (Clowes et al., 1998). If dietary protein is limited, the sow becomes progressively more dependent on protein mobilization to support lactation. If the mobilizable protein reserve is a quantitatively important resource to sustain lactation, the absolute quantity of protein in the reserve (developed during the growth of gestating animals) may be important. We sought to test this by provoking two levels of protein mobilization during lactation in animals that had been fed during gestation to achieve two different body protein masses at the onset of lactation. We hypothesized that if dietary protein/lysine was limiting in lactation, a larger initial protein mass would sustain lactation for a longer duration before a decline in piglet growth or ovarian function could be detected.

Materials and Methods

Studies were conducted in accordance with the Canadian Council of Animal Care Guidelines, and were approved by the Institutional Animal Policy and Welfare Committee.

Experimental Treatments and Measurements

The experiment was conducted as a 2 × 2 factorial arrangement in five replicates. The treatments consisted of feeding animals in gestation to achieve two divergent body masses at parturition (high or standard) and in lactation to achieve either a moderate or high level of maternal protein loss. Seventy-seven Genex gilts (Manor Hybrid × Large White or Manor Hybrid × Landrace; Genex Swine Group Inc.) were selected at a live weight of 80 to 95 kg and placed into groups of five to seven animals in an environmentally controlled room. This study was not designed to measure differences among the sow genotypes, so to account for the inherent genetic differences due to the breed of the gilt's sire (Large White or Landrace), we randomized the two genotypes into the two gestational feeding treatments.

Gilts were checked for standing estrus by placing an intact boar into the pen for 15 min/d. Gilts were bred two or three times by AI, on at least their second estrus, with pooled semen (Alberta Swine Genetics Corp., Nisku, AB, Canada). Gilts were group-fed a conventional dry sow diet (12.1 MJ of ME/kg, 13.3% CP, and 0.55% lysine) until 3 to 7 d after breeding. They were then individually penned and randomly allocated to their respective gestation feeding regimens to achieve either a standard gain of about 30 kg or a high gain of about 65 kg of live weight during gestation. Gilt age (214 ± 1.2 d) and live weight (128 ± 1.0 kg) at breeding were similar among treatments.

Gilts were fed in gestation based on their ME and N requirements for maintenance and for fetal and maternal growth either 1.9 kg/d of the standard gain 10% CP mash diet to gain 4 to 5 g maternal N/d or 2.4 kg/d of the high gain 18% CP mash diet to gain 15 to 20 g maternal N/d (Table 1). The diets were designed so that gilts attained a midback backfat depth of 18 to 20 mm at parturition. It was assumed that the requirements for the products of conception and mammary gland were 2, 4, and 8 to 14 g of N/d in trimesters one, two and three of gestation (Noblet et al., 1985), respectively, and 1.3 MJ of ME/d throughout gestation (NRC, 1998), and that maternal gain was composed of 15% protein and 25% fat tissue. The sow's maintenance requirements are described in a later section. To maintain the desired gestational weight gains, individual sow feed intake was adjusted. Feed intake was increased in the standard- and high-gain groups to be 2.7 and 3.1 kg/d from d 107 of gestation, respectively. These intakes were in a range not considered to either enhance or inhibit fetal and mammary gland growth and development (Shields et al., 1985; Head and Williams, 1991; Kusina et al., 1999a). Approximately every 16 d during gestation, sow live weight was measured and backfat depth was measured ultrasonically with an Aloka SSD-210DXII Echo camera with a UST-5020 diagnostic real-time ultrasound (Aloka Co. Ltd., Tokyo, Japan) equipped with a 110-mm-wide, 3.5-MHz probe head (Overseas Monitor Corp. Ltd., Richmond, BC, Canada). Backfat was measured at three sites (loin, midback, and grade site) as described by Sather et al. (1991).

At approximately d 109 of gestation, gilts were moved into individual crates in rooms containing five farrowing crates. At parturition, sows (n = 53) from the two gestational feeding treatments were randomly and equally allocated, based on the genotype of the gilt's sire, to lose a moderate or high level of protein, but a similar amount of fat tissue, in lactation. Sows allocated to these two treatments were fed diets that were formulated to provide 14.1 MJ of ME/kg, 17.5 or 11.5% CP, and 0.90 or 0.55% total lysine (Table 1). From d 1 of lactation, sows were offered 3.0 kg/d, and then feed offered was increased 1 kg/d every 5 d until d 15, after which, 5.5 kg/d was offered until weaning. To reduce the variance in feed intake between animals, feed levels were calculated to be approximately 85% of the ad libitum intake of first-parity sows in our herd. Feed not consumed was weighed daily.

Litter size was standardized to at least nine pigs within 2 d of parturition by cross-fostering within the

Table 1. Composition of the gestation and lactation sow diets (%, as-fed basis)

	Gestation diet[a]		Lactation diet[b]	
Ingredient	Standard gain	High gain	High loss[c]	Moderate loss[d]
Wheat	24.0	43.9	27.0	27.0
Hulless barley	—	—	34.4	34.4
Barley	62.9	23.8	—	—
Soybean meal (46% CP)	7.0	23.0	16.0	16.0
Fishmeal (herring)	—	—	—	8.0
Sugar	—	—	20.0	5.0
Oil (canola)	2.0	5.0	5.0	5.0
Iodized salt	0.6	0.6	0.6	0.6
Dicalcium phosphate	2.2	1.8	2.4	2.0
Limestone	1.4	1.4	1.4	1.3
Choline chloride (60%)	0.06	0.06	0.06	0.06
Vitamin premix[e]	0.20	0.20	0.20	0.20
Trace mineral premix[f]	0.15	0.15	0.15	0.15
Lysine HCl	0.10	0.05	0.10	—
Valine	—	—	0.08	0.05
Calculated analysis				
ME, MJ/kg	12.86	14.18	14.34	14.03
Crude fiber, %	4.69	3.66	2.76	3.24
Chemical analysis				
DM, %	89.6	90.2	92.3	91.7
CP, %	14.00	18.17	10.51	16.61
Lysine, %	0.59	0.82	0.51	0.84
Valine, %	0.67	0.87	0.58	0.87

[a]Formulated to 0.91% Ca, 0.75% P.
[b]Formulated to 0.93% Ca, 0.70% P.
[c]Diet fed to sows during lactation that was formulated to induce a high degree of body protein loss.
[d]Diet fed to sows during lactation that was formulated to induce a moderate degree of body protein loss.
[e]Supplied per kilogram of complete feed: 20,000 IU of vitamin A, 2,000 IU of vitamin D_3, 80 IU of vitamin E, 3.3 mg of vitamin K (menadione sodium bisulfite), 40 μg of vitamin B12, 10.7 mg of riboflavin, 47 mg of niacin, 33 mg of pantothenic acid (D-calcium pantothenate), 800 μg of biotin, 3.3 mg of folic acid, 3.3 mg of pyridoxine, and 3.3 mg of thiamine.
[f]Supplied per kilogram of complete feed: 113 mg of Fe (ferrous sulfate), 113 mg of Zn (zinc oxide), 56 mg of Mn (manganese oxide), 15 mg of Cu (copper sulfate), 750 μg of I (calcium iodate), and 225 μg of Se (sodium selenite).

gestation feeding treatments. Routine procedures (teeth clipping, tail docking, ear-notching, and iron injection) were conducted 2 d postpartum, and no creep feed was offered. Sows were weaned at about 0800 on approximately d 26 of lactation (range d 20 to 29). Sows and litters were weighed and sow backfat depth (midback) was measured ultrasonically on d 1 of lactation, every 5 d during lactation, and at weaning. Litters were also weighed on d 3. Sow backfat depth was measured ultrasonically at the three sites in early (d 1 to 2) and mid-lactation (approximately d 15) and at weaning. Milk samples (10 to 20 mL) were obtained twice from sows after an i.m. injection of 10 IU of oxytocin on d 10 and 20 of lactation, or 3 d prior to weaning, whichever came first. Milk samples were stored at –20°C and later analyzed for protein, fat, and lactose.

Feed was removed from the sows at least 16 h prior to slaughter. Sows were slaughtered 2 to 3 h after weaning. A blood sample was collected into a 10-mL heparinized tube at exsanguination and stored on ice until processing. The blood sample was centrifuged at 1,500 × *g* for 15 min, and the plasma was poured off and stored at –20°C for later insulin and IGF-I analysis. Both ovaries were collected and washed twice in sterile saline containing kanamycin (0.1 mg/mL; Sigma, St. Louis, MO).

Carcass Measures and Dissection (Cut-Out) of Primal Cuts. The mammary gland was removed from the carcass and weighed with skin attached. The remaining hide was stripped from the carcass, removing as little subcutaneous fat as possible, and weighed. The kidneys, heart, kidney fat, spleen, full gut, lungs, trachea, tongue, and skinned head were weighed. The liver was weighed after removal of the gall bladder, and the uterus was weighed after being trimmed of the mesenteric tissue, and sectioned immediately distal to the cervix. The carcasses were split longitudinally into equal halves and chilled for 24 h at 4°C after removal of the front feet. The chilled right-half side of the carcass was cut into the primal cuts: shoulder (picnic, hock, and butt), loin, ham, and belly. The belly and side ribs were reduced to a trimmed and squared product. The remaining primal cuts (shoulder, loin, and ham) were separated into muscle, fat, and bone, according to the procedure of Martin et al. (1981). The weights of these respective tissues were recorded, and the body cavity, subcutaneous, and intermuscular fat depots for each cut were weighed separately and added together for the

total fat in each depot. The muscle, fat, and bone in the primal cuts were calculated to be twice the muscle, fat, or bone in the half-carcass primal cuts. Total carcass fat was calculated to be the fat in the carcass primal cuts plus the kidney fat.

Determination of Sow Body Composition and Energy and Lysine Balance. Live weight and the midback backfat depth were used to indirectly estimate the sow's body protein and fat mass using preexisting equations (Whittemore and Yang, 1989). Because these equations do not distinguish between skeletal muscle protein and protein from other tissues and organs, the sow's muscle, bone, and fat wet-weight at weaning were measured by dissection of the primal cuts as previously described (Martin et al., 1981).

Energy and lysine balance were calculated in lactation, based on the recorded measures of energy and total lysine intake minus the calculated requirements for maintenance and milk production. The maintenance requirements of the sow were assumed to be 444 kJ of ME/kg of BW $^{0.75}$ (106 kcal of ME/kg of BW $^{0.75}$; NRC 1998) and 0.039 g of lysine/kg of BW $^{0.75}$ (Fuller et al., 1989). The ME requirement for milk production (**Energy$_{milk}$**) was calculated from the equation of Noblet and Etienne (1989), modified by NRC (1998). The dietary efficiency of ME use for milk production was 72% (Noblet and Etienne, 1987). The total lysine requirement for milk production (Lysine$_{milk}$) was calculated from the equation of Pettigrew (1993):

$$\text{Energy}_{milk}\ (\text{kJ ME/d}) = [(4.92 \times \text{litter gain, g/d}) - (90 \times \text{No. pigs})]/0.72] \times 4.184$$

$$\text{Lysine}_{Milk}\ (\text{g/d}) = 26 \times \text{litter gain, kg/d}$$

Ovarian Measures. The external diameter of the eight largest follicles on each ovary, from each sow, was determined as the mean of two caliper measurements taken at 90° to one another. Treatment effects were established by comparing the proportion of the largest 16 follicles from each sow categorized as being either ≤3.5 mm or >3.5 mm external diameter. Follicular fluid from these follicles was then aspirated individually with a 250-μL Hamilton syringe and collected. The weight of the syringe before and after aspiration was recorded and the difference was taken as the follicular fluid weight. Follicular fluid volume was calculated assuming a density of 1 g/mL. Individual follicular fluid samples were diluted to 10% with tissue culture media (TCM 199 containing Earle's salts, L-glutamine, and no sodium bicarbonate; GibcoBRL/Life Technologies, Grand Island, NY) and stored at –30°C for later estradiol analysis. Collection of follicular fluid was not attempted on ovaries with follicles that had an external diameter of less than 2mm, but the follicular status of these ovaries was recorded. Uterine weight was used as a measure of the stimulatory effects of estradiol on the reproductive tract (Foxcroft et al., 1984).

Analyses

Feed and Milk Analyses. Feed samples were ground in a Wiley mill through a 0.8-mm screen, mixed well, and stored at 4°C until DM, N, and amino acid analysis. Feed N was analyzed with the FP-428 Determinator System: 601-700-900 (LECO Corp., St. Joseph, MI) and feed amino acid composition was determined by HPLC (Sedgwick et al., 1991). Methionine, cysteine, tryptophan, and proline were not determined. The concentrations of milk fat, protein, and lactose were determined by infrared analysis using a MilkoScan Analyzer (Foss Electrics, Denmark) at the Alberta Central Milk Testing Laboratory (Edmonton, AB, Canada).

Plasma Insulin and Insulin-Like Growth Factor-I Analyses. Plasma insulin concentrations were analyzed by the double-antibody RIA described by Cosgrove et al. (1992), with modifications described by Patterson et al. (2002). The mean sensitivity of the two insulin assays was 0.019 ng/tube and the mean intra- and interassay CV were 5.6 and 11.9%, respectively. Plasma IGF-I concentrations were determined by the double-antibody RIA of Glimm et al. (1990) after acid–ethanol extraction as described by Cosgrove et al. (1992). Extraction efficiency, based on an estimate of cold recovery of IGF-I added to the standard plasma pool, was 100%. The assay sensitivity, defined as 92% of the total binding, was 0.03 ng/mL, and the intraassay CV was 11.2%.

Follicular Fluid Estradiol Analysis. Diluted follicular fluid (10% in TCM 199) from each sow was pooled into three categories based on volume: the four largest, four smallest, and the four intermediate volumes. The pooled follicular fluid was further diluted 1:50 with PBS gelatin assay buffer to achieve a final dilution of 1:500. This assay buffer contained $NaH_2PO_4 \cdot H_2O$ (2.77 m*M*), NaH_2PO_4 (7.22 m*M*), NaCl (139.7 m*M*), NaN_3 (15.38 m*M*), and 0.1% (wt/vol) gelatin. Estradiol was measured on these pooled samples using a double-antibody estradiol RIA kit (Diagnostic Product Co., Los Angeles, California; catalog No. KE2D1), with a minor modification as described by Clowes et al. (2003). Assay sensitivity, defined as 95% of total binding, was 0.03 ng/mL, the intraassay CV for the two assays averaged 4.5%, and the interassay CV was 6.9%.

Statistical Analyses. Only animals that successfully completed the experiment were included in the analysis. Analyses involving continuous variables were computed using the GLM procedure of SAS (SAS Inst., Inc., Cary, NC). Effect of the gilt's live weight at parturition (standard [165 kg] or high [193 kg]), the gilt breed of sire (Large White or Landrace), and their interactions on sow variables during gestation were assessed by repeated measures ANOVA. Effect of the gilt's live weight at parturition, the gilt breed of sire, lactation protein loss (high [approximately 17%] or moderate [approximately 10%]), and their interactions on sow and litter measures during lactation were also assessed by repeated measures ANOVA. In the event of a significant ($P < 0.05$) interaction between time and sow body mass

Table 2. Carcass variables at weaning in first-parity sows that had a standard or high body mass at parturition and lost a moderate or high amount of protein in lactation[a]

Item	Standard body mass		High body mass		*P*-value	
	High	Moderate	High	Moderate	Parturition mass	Lactation loss
n	11	12	12	12		
Muscle mass, kg[b]	37.3 ± 1.4	42.1 ± 1.3	46.1 ± 1.5	49.9 ± 1.4	0.001	0.003
Bone mass, kg[c]	8.7 ± 0.2	8.5 ± 0.2	9.4 ± 0.2	10.0 ± 0.2	0.001	0.505
Muscle to bone ratio	4.25 ± 0.16[x]	5.00 ± 0.15[y]	4.89 ± 0.16[y]	5.01 ± 0.16[y]	0.052	0.007
Fat mass, kg[d]	14.5 ± 1.05	12.9 ± 0.99	17.1 ± 1.09	14.9 ± 1.05	0.036	0.064
Kidney fat	1.90 ± 0.19	1.47 ± 0.18	2.02 ± 0.20	1.64 ± 0.19	0.474	0.036
Total carcass fat, kg[e]	16.4 ± 1.2	14.3 ± 1.1	19.2 ± 1.2	16.5 ± 1.2	0.048	0.048
Other carcass variables						
Carcass length, cm	96.8 ± 1.2	95.2 ± 1.2	98.4 ± 1.3	103.0 ± 1.4	0.001	0.230
Hide, kg	11.6 ± 0.31	12.9 ± 0.29	14.3 ± 0.33	14.9 ± 0.31	0.001	0.003
Skinned head, kg	4.8 ± 0.1	4.8 ± 0.1	5.1 ± 0.1	5.3 ± 0.1	0.001	0.518
Ribs, kg	3.68 ± 0.18	3.86 ± 0.18	4.23 ± 0.19	4.60 ± 0.18	0.002	0.148
Belly, kg	7.9 ± 0.5	8.1 ± 0.5	9.7 ± 0.5	9.7 ± 0.5	0.003	0.822
Full gut, kg	10.2 ± 0.46	11.0 ± 0.43	10.5 ± 0.48	12.1 ± 0.46	0.132	0.011
Lungs, trachea & tongue, kg	1.99 ± 0.08[x]	2.07 ± 0.08[x]	2.05 ± 0.08[x]	2.45 ± 0.08[y]	0.010	0.004
Kidneys, kg	0.39 ± 0.02[x]	0.44 ± 0.01[y]	0.40 ± 0.02[xy]	0.52 ± 0.02[z]	0.001	0.001
Heart, kg	0.47 ± 0.02	0.50 ± 0.02	0.53 ± 0.02	0.62 ± 0.02	0.001	0.009
Liver, kg	2.11 ± 0.08	2.40 ± 0.07	2.22 ± 0.08	2.73 ± 0.08	0.010	0.001

[a]Least squares means ± standard error of the mean.
[b]Muscle mass (kg) = 2 × half carcass muscle in the primal cuts (shoulder, loin, and ham).
[c]Bone mass (kg) = 2 × half carcass bone in the primal cuts (shoulder, loin, and ham).
[d]Fat mass (kg) = 2 × half carcass fat in the primal cuts (shoulder, loin, and ham).
[e]Total carcass fat (kg) = kidney fat + fat mass.
[x,y,z]Within a row, interaction means that do not have a common superscript differ ($P < 0.05$).

or time and lactation protein loss, differences among time within each treatment were compared using *a priori* orthogonal contrasts. The sow, litter, carcass, and ovarian variables were analyzed using ANOVA and this model. If significant treatment differences were detected ($P < 0.05$), these means were compared using the LSD test. Follicular fluid estradiol concentrations were analyzed for the largest, smallest, and intermediate four follicular fluid volumes for each sow. Variation among experimental units (sow within body size × protein loss) was used as the estimate of experimental error and for significance testing of follicular fluid estradiol concentrations. The CATMOD procedure of SAS was used to determine differences in the proportion of follicles in the two follicle size categories (external diameter of ≤3.5 mm or >3.5 mm), between the two parturition body mass treatments, the two protein loss treatments in lactation, and the two-way interactions.

Results

Twenty-two gilts placed on test were not pregnant and two others were removed because of lameness. Six of the 53 gilts that farrowed were taken off test because of illness and/or low appetite (<3 kg/d in lactation). Thus, 11 gilts were available for allocation to the standard level of growth in gestation and to lose a high level of protein in lactation and 12 gilts were allocated to each of the other three treatments. Variation in the sow's genotype only affected a few measured variables. Gilts with a Landrace sire grew litters approximately 15% faster ($P < 0.01$) in lactation and had a 9% heavier ($P < 0.01$) liver weight at weaning. These gilts also had a 13% lower lean mass at weaning (40.4 vs. 46.9; $P < 0.001$) and approximately a 2-mm greater ($P < 0.06$) backfat depth throughout gestation.

Sow Body Reserves

There were interactions ($P < 0.05$) between sow body mass at parturition and protein loss in lactation for weight of the lungs, trachea and tongue, kidneys, and the muscle:bone ratio at weaning (Table 2). Standard body mass sows that lost the most protein in lactation had the lowest muscle:bone ratio at weaning and the lightest kidneys at weaning compared with the other three treatments. High body mass sows that lost the least protein in lactation had the heaviest kidneys and lungs, trachea and tongue at weaning.

Gilts on the higher plane of nutrition in gestation were larger ($P < 0.001$) at parturition (Tables 3). We observed a significant residual effect of the gestational treatments at weaning on the mass of various tissues (Table 2). These sows developed a larger ($P < 0.001$) skeletal structure, as indicated by a 10% larger bone mass and a 5% longer carcass at weaning. The belly, ribs, skinned head, hide, and most organs were also heavier ($P < 0.01$) at weaning in the high body mass sows. The magnitude of the difference between the gestational treatments ($P < 0.001$) in muscle mass at wean-

ing (+20%; 48 vs. 40 kg) was similar to the difference in the sow's calculated protein mass at parturition (+23%; 30.0 vs. 24.3 kg). Gilts on the higher plane of nutrition in gestation also achieved higher live weight gains during gestation (+61%; 66 vs. 41 kg; $P < 0.001$) as designed by the study, resulting in a larger live weight at parturition (+17%; 193 vs. 165 kg; $P < 0.001$).

The differences in muscle deposition during gestation were accompanied by parallel differences in fat deposition. Therefore, at parturition, the muscle:fat ratio was the same ($P > 0.40$) after both feeding regimens, regardless of whether the values were estimated (body protein to fat ratio; 0.57 ± 0.18) or measured by carcass dissection at weaning (muscle:fat ratio; 2.8 ± 0.2). The sow's backfat depth at parturition differed slightly ($P < 0.05$) between the gestational feeding treatments (Table 3). But, because of body size differences, high body mass sows had a greater calculated fat mass at parturition (+23%; $P < 0.001$), and more fat (+15%; $P < 0.05$) was dissected from the carcass cuts of these sows at weaning (Table 2).

Sow live weight losses in lactation and body weights at weaning reflected the level of protein fed during lactation and the size of the sow's body mass at parturition (Table 3). Sows fed less protein during lactation lost a larger fraction ($P < 0.01$) of their calculated protein mass at parturition (–17.8 vs. –10.7%; Table 3). This was concordant with the approximately 10% lower ($P < 0.01$) mass of muscle, organs (kidneys, heart, and liver) and hide at weaning in sows fed the lower protein level (Table 2). Standard body mass sows had a smaller muscle mass (39.7 vs. 48.0 kg; $P < 0.01$; Table 2), body weight (145 vs. 168 kg; $P < 0.01$), and calculated protein mass (20.8 vs. 24.9 kg; $P < 0.001$) at weaning than high body mass sows (Table 3). These effects were more prominent after restricted protein feeding in lactation because both the high and standard body mass sows that lost a large amount of protein in lactation lost a similar calculated amount of body protein mass. But standard body mass sows appeared to lose a larger proportion of their muscle tissue, as indicated by a lower muscle:bone ratio ($P < 0.05$; Table 2).

During lactation, all sows lost backfat depth (5 ± 0.9 mm) and body fat mass (25.8 ± 1.5%) (Table 3), but there was no effect of either parturition mass ($P > 0.32$) or lactational protein loss ($P > 0.28$) on the calculated fat mass or backfat depth loss in lactation (Table 3). However, other measured variables provide evidence that sows fed less protein in lactation lost slightly less fat tissue in lactation. These sows had more ($P < 0.05$) kidney fat and total carcass fat (Table 2) and a greater backfat depth ($P < 0.05$) at weaning (Table 3).

Nutrient Intake and Calculated Energy/Lysine Balance in Lactation

There were no interactions between body mass at parturition and protein loss in lactation for the nutrient

Table 3. Sow live weight, backfat, and calculated protein and fat mass during lactation in first-parity sows that had either a standard or high body mass at parturition and lost a moderate or high amount of protein in lactation[a]

	Standard body mass		High body mass		*P*-value	
Item	High	Moderate	High	Moderate	Parturition mass	Lactation loss
At parturition						
Live weight, kg	165 ± 1.7		193 ± 1.9		0.001	
Backfat depth, mm	20.0 ± 0.9		22.8 ± 1.0		0.045	
Protein mass, kg[b]	24.3 ± 0.35		30.0 ± 0.36		0.001	
Fat mass. kg[b]	44.4 ± 1.50		54.5 ± 1.68		0.001	
Sow live weight, kg						
At weaning, kg	141 ± 3.5	149 ± 3.3	163 ± 3.6	173 ± 3.5	0.003	0.013
Lactation loss, kg	23.1 ± 2.4	17.6 ± 2.2	29.7 ± 2.5	21.3 ± 2.4	0.032	0.006
Sow backfat depth[c]						
At weaning, mm	16.4 ± 1.2	15.1 ± 1.2	19.7 ± 1.2	15.7 ± 1.2	0.108	0.026
Lactation loss, mm	3.9 ± 0.9	4.6 ± 0.8	4.6 ± 0.9	5.7 ± 0.9	0.330	0.288
Calculated sow protein mass[b]						
At weaning, kg	19.9 ± 0.63	21.7 ± 0.60	23.6 ± 0.66	26.1 ± 0.63	0.001	0.001
Loss to d 15, %	9.8 ± 1.5	4.3 ± 1.4	9.6 ± 1.6	7.1 ± 1.5	0.402	0.012
Loss to d 20, %	12.5 ± 1.5	3.4 ± 1.4	12.4 ± 1.5	7.3 ± 1.5	0.195	0.001
Loss to weaning, %	17.3 ± 1.6	10.6 ± 1.5	16.7 ± 1.7	11.2 ± 1.6	0.991	0.001
Calculated sow fat mass[b]						
At weaning, kg	32.7 ± 2.1	32.5 ± 1.9	42.6 ± 2.1	38.4 ± 2.1	0.001	0.278
Loss to d 15, %	14.6 ± 2.2	12.6 ± 2.1	16.9 ± 2.3	16.4 ± 2.2	0.189	0.577
Loss to d 20, %	19.8 ± 2.6	21.7 ± 2.4	20.1 ± 2.7	20.2 ± 2.6	0.822	0.702
Loss to weaning, %	23.0 ± 3.7	25.0 ± 3.2	24.5 ± 3.5	23.9 ± 3.4	0.950	0.844

[a]Least squares means ± standard error of the mean.
[b]Body protein/fat mass calculated using the equations of Whittemore and Yang (1989), based on sow live weight and midback backfat depth.
[c]Average for all three sites.

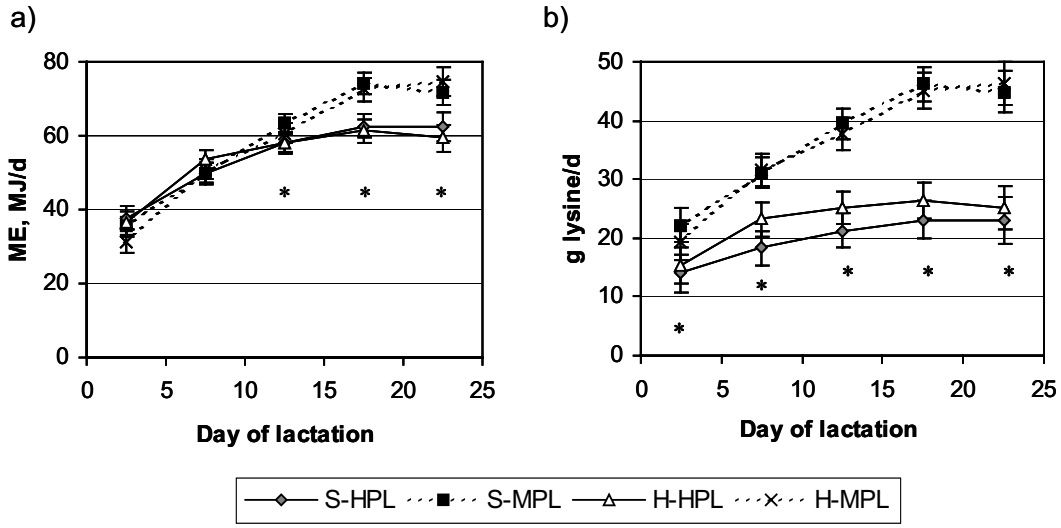

Figure 1. Intake of a) ME (MJ/d) and b) total lysine (g/d) in lactation by first-parity sows fed to achieve a standard (S) or high (H) body mass at parturition and lose either a moderate (MPL) or high (HPL) amount of protein in lactation. *The MPL sows had a higher ($P < 0.001$) intake than the HPL sows.

intake and calculated balance variables. Intakes of energy and lysine increased ($P < 0.001$) in all treatments until d 20 and increased no further thereafter (Figure 1a,b); CP intakes increased in a similar manner in lactation. Sows fed to lose a moderate amount of protein had higher ($P < 0.001$) protein (731 vs. 416 g/d) and total lysine (37.0 vs. 21.6 g/d) intakes during lactation than sows fed to lose a high amount of protein (Table 4). Energy intake did not differ among lactation treatments over the first 10 d of lactation, but thereafter it was about 10% higher ($P < 0.05$) in sows fed to lose a moderate rather than a high amount of protein (Figure 1a). The calculated energy balance for the whole of lactation was slightly more negative in the high than standard body mass sows (–23.0 vs. –20.0 MJ of ME/d; $P = 0.053$).

Litter Size and Growth

Litter size on d 0 and 3 of lactation and at weaning were similar among treatments and were 10.7 ± 0.22, 10.3 ± 0.22, and 9.9 ± 0.28 piglets, respectively. There were no interactions between sow body mass at parturition and lactation protein loss. Despite the differences in sow nutrition and growth in gestation, the feeding treatments imposed during gestation appeared to have little effect on mammary and fetal growth and development. Piglet birth weight (1.37 ± 0.05 kg) and the number of pigs born alive (10.8 ± 0.8), stillborn (0.53 ± 0.18), and mummified (0.21 ± 0.11) were similar ($P > 0.05$) among sows on the high and standard planes of nutrition in gestation. Also, mammary gland wet weight determined at weaning did not differ ($P = 0.12$) between gestation treatments (Table 5). However, over the first 5 d of lactation, the litters of standard body mass sows grew about 20% more slowly ($P < 0.01$) than those of high body mass sows (Table 5).

Litter growth rate increased ($P < 0.001$) over the first 10 d of lactation for sows on all treatments and thereafter remained unchanged until d 20, when a decline in litter growth was observed (Table 5). There was a

Table 4. Feed intake and calculated energy and lysine balance over lactation in first-parity sows that had a standard or high body mass at parturition and lost a moderate or high amount of protein in lactation[a]

	Standard body mass		High body mass		*P*-value	
Item	High	Moderate	High	Moderate	Parturition mass	Lactation loss
Lactation intake						
ME, MJ/d	56.6 ± 2.3	62.9 ± 2.2	55.9 ± 2.4	61.0 ± 2.3	0.590	0.016
CP, g/d	415 ± 23	743 ± 22	416 ± 24	720 ± 23	0.625	0.001
Total lysine, g/d	20.1 ± 1.3	37.7 ± 1.3	23.1 ± 1.4	36.2 ± 1.3	0.582	0.001
Calculated balances in overall lactation						
ME, MJ/d[b]	–16.9 ± 3.0	–20.4 ± 2.7	–23.2 ± 2.8	–25.7 ± 2.9	0.053	0.298
Lysine, g/d[b]	–23.4 ± 1.9	–16.8 ± 1.7	–23.6 ± 1.7	–19.1 ± 1.8	0.500	0.003

[a]Least squares means ± standard error of the mean.
[b]Energy and lysine balance calculated based on the sow's recorded energy and lysine intake minus the sow's calculated requirements for maintenance and milk production.

Table 5. Litter variables in first-parity sows that had a standard or high body mass at parturition and lost a moderate or high amount of protein in lactation[a]

Item	Standard body mass		High body mass		*P*-value	
	High	Moderate	High	Moderate	Parturition mass	Lactation loss
Average litter growth rate, kg/d	1.93 ± 0.09	2.16 ± 0.08	2.11 ± 0.09	2.29 ± 0.09	0.082	0.024
Mammary gland[b]	6.96 ± 0.29	7.68 ± 0.27	7.41 ± 0.30	8.16 ± 0.29	0.124	0.014
Litter growth, kg/d[cd]						
d 0 to 5[e]	1.42 ± 0.13	1.37 ± 0.12	1.72 ± 0.14	1.82 ± 0.13	0.008	0.860
d 5 to 10	2.18 ± 0.13	2.67 ± 0.12	2.41 ± 0.13	2.47 ± 0.13	0.885	0.028
d 10 to15	2.27 ± 0.10	2.41 ± 0.10	2.37 ± 0.11	2.56 ± 0.10	0.221	0.103
d 15 to 20	2.11 ± 0.12	2.41 ± 0.11	2.35 ± 0.12	2.66 ± 0.12	0.044	0.011
d 20 to 26	1.76 ± 0.16	2.21 ± 0.16	1.83 ± 0.17	2.37 ± 0.16	0.485	0.004
Litter growth change, d 10 to 15 vs. d 20 to 26, %[e]	–19.5 ± 6.9	–6.8 ± 6.5	–21.6 ± 7.2	–6.4 ± 6.9	0.908	0.045

[a]Least squares mean ± standard error of the mean.
[b]Mammary weight at weaning includes attached skin, connective tissue, and muscle remnants.
[c]Parturition mass ($P = 0.046$) and lactation protein loss ($P = 0.004$) differed in the repeated measures analysis of litter growth rate.
[d]The effect ($P < 0.001$) of 5-d time period in lactation indicated that litter growth rate increased over the first 10 d of lactation in all treatments, and thereafter remained unchanged until d 20, when it decreased.
[e]There was an interaction ($P < 0.05$) between 5-d time period in lactation and lactation protein loss such that the decrease in litter growth rate between d 10 to 15 and the end (d 20 to 26) of lactation was greater in sows that lost the most protein.

significant ($P < 0.05$) lactation protein loss × time interaction for litter growth rate. Sows that lost the most body protein, regardless of their body mass at parturition, had difficulties in maintaining their litter growth rate toward the end of lactation (Table 5). Average litter growth rate over the whole of lactation was approximately 9% lower ($P < 0.05$); the decline in litter growth rate between d 10 and 15 and the end (d 20 to 26) of lactation was greater (–21 vs. –7%; $P < 0.05$) and mammary gland wet weight at weaning was 9% lighter ($P < 0.02$) in sows that lost the most protein in lactation (Table 5). Milk protein concentration was also lower in these sows ($P < 0.05$), but milk fat (Figure 2) and lactose concentrations did not differ among treatments. Milk protein concentration was unaffected by stage of lactation, but milk fat concentrations declined (Figure 2b; $P < 0.001$), and milk lactose concentrations increased slightly (5.4 vs. 5.5; $P < 0.001$) between d 10 and 20 of lactation.

A smaller body mass at parturition exacerbated the effects of restricted protein intake in lactation. Although the degree of decline in litter growth rate in late lactation did not differ between high and standard body mass sows, litters from standard body mass sows grew more slowly ($P < 0.05$) than those from high body mass sows (Table 5). The average litter growth rate of standard body mass sows tended ($P = 0.08$) to be lower than for high body mass sows (2.05 vs. 2.21 kg/d; Table

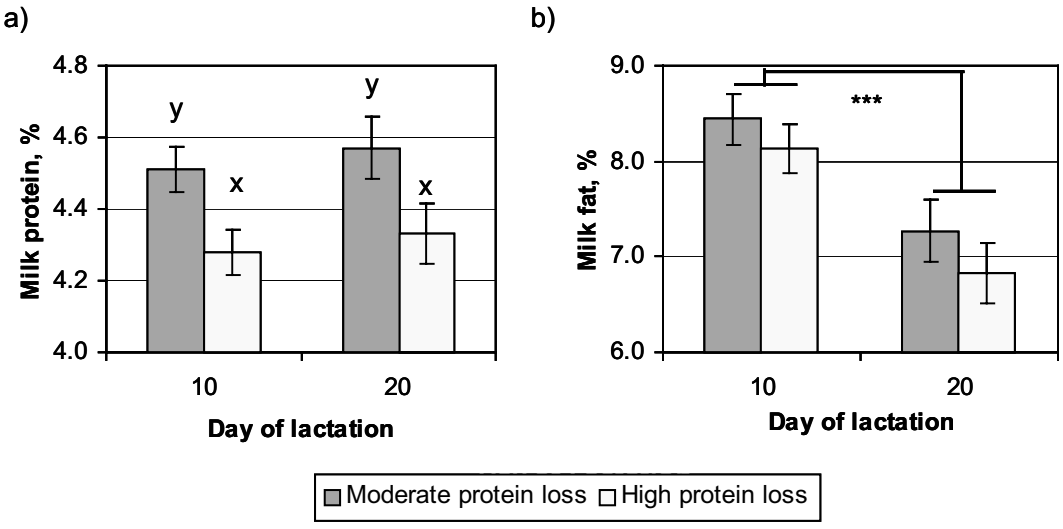

Figure 2. Milk a) protein and b) fat composition on d 10 and 20 of lactation in first-parity sows that lost a moderate or high amount of protein in lactation. Bars within a day of lactation that do not have a common letter designation differ ($P < 0.05$). ***Milk fat concentration on d 10 of lactation was higher ($P < 0.001$) than on d 20.

Table 6. Ovarian measures and plasma hormone concentrations at weaning in first-parity sows that had a standard or high body mass at parturition and lost a moderate or high amount of protein in lactation[a]

Item	Standard body mass		High body mass		*P*-value	
	High	Moderate	High	Moderate	Parturition loss	Lactation loss
No. of sows	11	12	12	12		
Plasma IGF-I, ng/mL[b]	54.9 ± 4.6	57.4 ± 4.5	59.7 ± 4.8	66.2 ± 4.6	0.157	0.322
Plasma insulin, pg/mL[b]	350 ± 62	427 ± 58	361 ± 64	569 ± 62	0.231	0.024
Uterine weight, kg[c]	0.24 ± 0.02[x]	0.22 ± 0.02[x]	0.23 ± 0.02[x]	0.30 ± 0.02[y]	0.060	0.126
Percentage of largest 16 follicles						
≤3.5 mm diameter	73.9	61.4	47.4	41.7	0.001[d]	0.001[d]
>3.5 mm diameter	26.1	38.5	52.6	58.3		
Largest 16 follicles						
No. of sows	6	10	8	8		
Diameter, mm	3.4 ± 0.31	3.3 ± 0.26	3.8 ± 0.28	3.9 ± 0.27	0.065	0.866
Follicular fluid volume, μL	24.9 ± 4.6	21.9 ± 3.8	29.0 ± 4.1	33.1 ± 4.0	0.072	0.91
Follicular fluid estradiol, ng/mL	0.22 ± 0.12	0.29 ± 0.10	0.28 ± 0.10	0.65 ± 0.10	0.051	0.040

[a]Least squares mean ± standard error of the mean.
[b]Plasma samples were collected preprandially 2 or 3 min after slaughter.
[c]Uterine weight at weaning from all sows, trimmed of excess connective tissue.
[d]χ^2 square value for the parturition mass effect was 79.9, and for the lean loss effect was 13.0. Both treatments had one degree of freedom.
[xy]Within a row, interaction means that do not have a common superscript ($P < 0.05$).

5), and there was an indication that the decline in litter growth rate occurred earlier in these sows.

Ovarian Function

The proportion of different size class follicles was measured on all sows, but follicular fluid variables were only measured in the last four replicates of the experiment (32 of the 47 sows that completed lactation). There was only an interaction between sow body mass at parturition and protein loss in lactation for uterine weight ($P < 0.05$). High body mass sows that lost a moderate amount of protein in lactation had a higher uterine weight at weaning. Fasting plasma IGF-I concentrations did not differ ($P > 0.15$) among treatments, but sows that lost the most protein in lactation had the lowest ($P < 0.05$) fasting plasma insulin concentrations at weaning (356 vs. 498 pg/mL; Table 6).

The sow's body mass at parturition had the greatest effect on the ovarian variables measured; protein loss in lactation had a lesser effect (Table 6). At weaning, high body mass sows had more ($P < 0.001$) large follicles (greater than 3.5 mm in diameter). They also had a higher ($P = 0.051$) follicular fluid estradiol concentration and this was reflected in a higher ($P = 0.06$) uterine weight at weaning. Also, sows that lost a moderate amount of protein in lactation had more ($P < 0.001$) large follicles on their ovaries and a higher ($P < 0.05$) follicular fluid estradiol concentration at weaning than sows that lost a high amount of protein. Of the sows that lost a high amount of protein in lactation, more high than standard body mass sows (83 vs. 67%) had follicles with an external diameter greater than 3.5 mm at weaning. Similarly, of the sows that lost a moderate amount of protein in lactation, more high than standard body mass sows (83 vs. 45%) had follicles with an external diameter greater than 3.5 mm at weaning. Thus, at weaning, limited ovarian follicular development was seen in standard body mass sows that lost the most protein in lactation compared with high body mass sows that lost a moderate amount of protein in lactation. The latter sows showed the greatest follicular development.

Discussion

The gestation feeding treatment to produce sows of a standard body mass was similar to industry standards for energy, protein, and lysine intake. The higher level of feeding during gestation permitted more growth and produced animals of greater body size and mass, but similar body composition at parturition. Sows had parturition body protein reserves of either 24.3 or 30.0 kg to draw upon in lactation. By feeding sows an isoenergetic diet in lactation that contained two levels of protein, sows were induced to lose differential proportions of their protein mass. The gestation feeding regimens generated no differences in mammary development at d 26 of lactation, or on fetal growth and development evident at birth or during lactation. Lactation protein loss and gestation treatment effects on the growth of the gilt's progeny to market weight (106 kg) and their carcass composition at slaughter are described in Fortin et al. (2003).

The results of this study are concordant with our prior work showing a decline in litter growth and ovarian variables after the loss of at least 9 to 12% of the sow's calculated body protein mass present at parturition (Clowes et al., 2003). In the present experiment, we hypothesized that a larger initial protein mass would sustain lactation for a longer duration, before a decline in litter growth or ovarian variables could be detected. The results show that the poorest litter growth rate in

lactation and lowest ovarian development were observed in animals that were initially smaller and had mobilized the most body protein during lactation. A larger body mass at parturition ensured a higher litter growth rate and was associated with improved ovarian follicle development.

Body Composition Change

In our prior work, we assessed body composition indirectly from live weight and backfat thickness with equations developed by Whittemore and Yang (1989). The carcass dissection in this study allows further discrimination of body composition. In particular, skeletal muscle is regarded as the body's physiological protein reserve available for mobilization (Allison and Wannemacher, 1965; Swick and Benevenga, 1977). A substantial fraction of the differences in body weight and protein at the end of lactation were attributable to skeletal muscle. At slaughter, muscle mass ranged from 37.3 ± 1.4 kg in the standard body mass sows with the greatest mobilization to 49.9 ± 1.4 kg in the high body mass sows with a moderate amount of mobilization. Smaller fractions of the differences in live weight were attributable to the hide, internal organs, and body fat.

Ideally, in an experiment of this type, the only differences among the animals would be the initial body protein mass and the rate at which it is mobilized during lactation to ensure that the results are not confounded by differential fat loss. From a practical standpoint, this may be impossible to achieve. Some mobilization of fat may be inevitable and there may be secondary effects related to the level of protein feeding. For example, the energy expenditure for milk production was about 10% lower in sows fed less protein in lactation, especially at the end of lactation, because of the lower milk production. This likely reduced the need of these animals to mobilize their adipose tissue reserves, and as a result they had a slightly higher body fat mass (+15%; 17.8 vs. 15.4 kg), kidney fat mass (+27%; 1.96 vs. 1.54 kg) and backfat depth (+18%; 18.0 vs. 15.4 mm) at weaning. This is consistent with observations by others (King et al., 1993; Everts and Dekker, 1994; Sauber et al., 1998). Care must also be taken in commercial conditions to ensure that the sow does not become too fat in gestation and as a consequence experience reduced feed intake and performance in lactation (Revell et al., 1998).

Impact of Protein Loss on Performance During Lactation

A number of mechanisms are conceivably involved in the decline in lactation performance observed in sows that lose large amounts of their protein mass. The first is simply based on the concept that as animals mobilize body protein in lactation to maintain milk production, their muscle mass progressively declines. The fractional rate of muscle protein mobilization must therefore increase to provide a consistent total amino acid supply for milk production. For example, the muscle mass at weaning in standard body mass sows was 25% less than in the high body mass sows. To release the same total amount of amino acids, sows with the least muscle would require a rate of mobilization about 30% higher than sows that have a larger muscle mass. With continuous loss of body protein throughout lactation, at some point, the maximal fractional rate of muscle protein mobilization may no longer supply all the amino acids required to maintain milk production, and as a result, milk production will fall. This would make the absolute size of the protein reserve at parturition a key factor in supporting lactation for a lengthy duration and would be consistent with an earlier decline in litter growth in standard body mass animals.

The second is based on the suggestion that the quality of the amino acid mixture released from mobilized body protein does not match that required for milk protein synthesis. The composition of the amino acids mobilized from internal reserves, such as skeletal muscle, is dictated by the amino acid sequence of the constituent proteins and by any metabolism that may take place. It is not clear how well this supply resembles the optimal amino acid mixture for supporting lactation. If the qualities of dietary and mobilized protein were similar, lactation would be equally well supported by either. If the amino acid mixture released from endogenous protein does not match that required by the lactating mammary gland, then milk production would be less efficient at using the mobilized protein as a substrate. This is consistent with our observations here that even sows with a large initial lean body mass had a lower milk protein concentration on d 10 and 20 of lactation. A similar reduction in milk protein concentration in first-parity sows fed low lysine intakes in lactation (10 vs. 30 g/d) was observed by Kusina et al. (1999b) as early as d 8 of lactation and was still apparent on d 18.

It is also important to consider that mobilization of nonmuscle tissues may be implicated in the decline of milk production. The effects of progressive mobilization on the amount of functional secretory tissue in the mammary gland would be of interest since at weaning, the total organ weight differed among treatments by as much as 1.13 kg. Further work is required to clarify this area. The 9% lower wet mammary gland weight at weaning in sows that lost the most protein in lactation most likely reflects a reduction in mammary gland secretory tissue and production at the end of lactation because at the end of lactation, litter growth (and therefore milk production) was about 20% lower in these animals.

Impact of Protein Loss on Ovarian Function

Parturition body size had the largest impact on ovarian variables studied here. High body mass sows on the higher protein intake had the most follicular development at weaning. Standard body mass sows fed the

lowest protein intake in lactation had the least follicular development at weaning. These sows had lost the most muscle protein in lactation and had the lowest muscle mass at weaning, as indicated by the smallest muscle:bone ratio. This ratio can be used as an index of relative muscle mass because bone mass is maintained, but not mobilized to any great extent, even under conditions of extreme weight loss in adult animals (Seebeck, 1973; Kempster, 1978).

A low muscle mass may initiate warning signals that delay or even prevent the subsequent reproductive cycle to allow the animal to recover from the large metabolic insult incurred in lactation. Peripheral insulin and IGF-I concentrations were unlikely to mediate this effect. In this and our prior work (Clowes et al., 2003), peripheral IGF-I levels after an overnight fast did not differ among treatments, and only in the present study were peripheral insulin concentrations higher in sows that lost the least protein in lactation. However, insulin and IGF-I tissue sensitivity (e.g., receptor number, affinity of ligand binding, and signal transduction) was not measured here and could potentially account for any differences in the signal transmitted by circulating hormones and could affect the hypothalamo-pituitary-ovarian axis either directly or indirectly.

Restriction of feed (Zak et al., 1997a; Quesnel et al., 1998; van den Brand et al., 2000) and protein intake in lactation (King and Martin, 1989; Jones and Stahly, 1999b; Yang et al., 2000b) reduced sow LH pulsatility in late lactation and after weaning. This indicates inhibition of the hypothalamic–pituitary axis due to a slowing of the hypothalamic GnRH pulse generator (I'Anson et al., 1991; Wade et al., 1996). Changes in substrate and hormone levels induced by diet directly inhibit ovarian function, follicle growth and development, and the quality of the follicle and oocyte within that follicle (Foxcroft, 1990; 1992). First-parity sows restricted in feed (Zak et al., 1997b; Quesnel et al., 1998) and protein intake (Yang et al., 2000a; Clowes et al., 2003) during lactation had fewer potential preovulatory follicles at weaning and poorer quality follicles after weaning. First-parity sows restricted in feed intake in lactation also had lower ovulation rates (Zak et al., 1997a; van den Brand et al., 2000). The ovary is likely inhibited by the sow's nutritional status at any stage of lactation because preovulatory follicles (>3 mm diameter) probably undergo antral formation in early lactation. The lactation length of sows in this experiment was 26 d, and antral follicles may be recruited into the preovulatory pool over a 19- to 21-d period (Morbeck et al., 1992).

It is possible that peripheral amino acid concentrations differed among sows on the different treatments. We previously observed that first-parity sows fed to lose divergent amounts of body protein in lactation had differing muscle-free amino acid profiles (Clowes et al., 2000). Also, like lactating sows, postsurgery patients mobilized their body protein and changed their muscle and peripheral free amino acid profile (Askanazi et al., 1980; Petersson et al., 1992). Therefore, we conjecture that the availability of certain amino acids in the peripheral circulation could influence ovarian function or the hypothalamic–pituitary axis. The changes in peripheral amino acid patterns could increase the competition for central amino acid transporter uptake of key neurotransmitter precursors and thus alter central neurotransmitter concentrations; some amino acids act directly as neurotransmitters or indirectly as precursors for neurotransmitters. Amino acid supply could also impact on oocyte development and maturation. The extent and nature of such effects remain to be determined.

Implications

Extensive protein mobilization decreases litter performance during lactation and ovarian follicular development at weaning. Our results suggest that careful feeding of replacement gilts in their first gestation and lactation may limit or abolish these problems. Even when gilts are small at breeding, higher intakes during gestation (to increase protein reserves) and optimal feeding during lactation (to limit protein mobilization) may be used to maintain animal performance. Sows with large protein reserves at parturition cope better with poor lactation nutrition. Thus, producing heavier gilts at farrowing by breeding at a heavier weight and/or feeding a higher energy/protein intake during gestation could be a useful management tool when poor appetite and subsequent reproductive performance are a problem in a commercial herd. The economic impact of these findings and the effects on overall animal productivity need to be further studied.

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