Feeding Lactating Primiparous Sows to Establish Three Divergent Metabolic States: I. Associated Endocrine Changes and Postweaning Reproductive Performance[1,2]

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ABSTRACT: We investigated effects of different metabolic states on reproductive performance in lactating, primiparous sows. Sows were fed ad libitum (AL; n = 12), alimentated via a gastric cannula to 125% of AL feed intake (SA; n = 8), or restricted (R; n = 9) to 50% of AL from d 1 to 28 of lactation. At weaning, all sows were fed 2.5× maintenance energy requirements until standing heat and then fed twice maintenance energy requirement until slaughter. Sow weight, backfat, and litter weights were recorded weekly. After weaning, sows were tested twice daily for the onset of estrus and inseminated twice using pooled semen. At d 28 of gestation, sows were slaughtered, and the reproductive tracts were recovered to determine ovulation rate and embryo survival. Intensive blood sampling was performed before and after weaning for 12-h periods to characterize changes in plasma LH, insulin, and IGF-I. After weaning, additional samples were taken to monitor changes in LH and progesterone. Insulin and IGF-I were determined at standing heat. During lactation, AL and R sows lost, whereas SA sows gained, body weight and backfat ($P < .001$). Litter growth rates did not differ among treatments. Although plasma insulin was not different among treatments, plasma IGF-I concentration was lower ($P < .001$) in R sows. Mean LH and pulse frequency before ($P < .03$ and $P < .06$, respectively) and after ($P < .001$; for both) weaning were lower in R than in AL or SA sows. After weaning, SA sows lost more weight ($P < .01$) and backfat ($P < .01$) and ate less feed ($P < .001$) than AL or R sows. At standing heat, no differences in plasma IGF-I or insulin were observed, although energy balance for SA sows was lower ($P < .01$) than for AL or R sows. Weaning-to-estrus interval was extended ($P < .02$) in R sows. We observed no treatment difference in ovulation rate or embryo survival. Our results demonstrate that making sows anabolic during lactation did not ameliorate the negative impact of the suckling stimulus or improve fertility after weaning.

Key Words: Sows, Lactation, Feed Intake, Reproduction

Introduction

Metabolic correlates of nutritional state are a probable functional link with the reproductive axis (I'Anson et al., 1991; Baidoo et al., 1992; Wade and Schneider, 1992). During lactation, primiparous sows do not consume sufficient feed to maintain a positive energy or nitrogen balance (Aherne et al., 1995) and

mobilize body tissue to supply the substrates for milk production (King and Williams, 1984). Although the pig suckling stimulus provides the primary block to the resumption of estrus during lactation by reducing LH release (Britt et al., 1985), feed restriction can further exaggerate this problem (Foxcroft et al., 1995; Quesnel and Prunier, 1995). Confirmation that a lack of pulsatile LH secretion is the primary cause of lactational anestrus comes from studies in which treatment with exogenous GnRH during lactation resulted in follicular development, behavioral estrus, and ovulation (Cox and Britt, 1982; Rojanasthien et al., 1988; De Rensis et al., 1991). Therefore, although there is extensive evidence that lactational catabolism imposes profound inhibitory effects on reproductive function, it is not known whether anabolism during lactation could ameliorate the effects of suckling-induced inhibition of LH secretion during lactation and thereby postweaning fertility.

We undertook a comprehensive study to compare the effect of different nutritional regimens during lactation on nitrogen balance, milk production, and reproductive performance. In this part of the study, our objective was to test the hypothesis that making sows anabolic during lactation would have beneficial effects on postweaning fertility and also to identify the endocrine mechanisms mediating these effects.

Materials and Methods

Experimental Design

Primiparous sows (n = 36) were offered a high-quality diet during lactation using one of three feeding regimens, ad libitum (**AL**), restricted (**R**), or super-alimented (**SA**), designed so that sows were slightly catabolic, grossly catabolic, or anabolic, respectively. Group AL sows were offered feed on an ad libitum basis. From past experience at the University of Alberta Swine Research Center, gilts fed to ad libitum consume on average 5.0 kg/d and lose .3 kg/d of body weight. Gilts were made grossly catabolic (R) by restricting their feed intake during lactation to about 3 kg/d (fed at 0600, 1330, and 2100), which was calculated to mobilize in excess of 1 kg of body weight/ d. Sows were made anabolic by gastric alimentation of feed (superalimented), which commenced within 4 d of farrowing. They were offered feed on an ad libitum basis from their feed trough and given additional feed by means of a stomach cannula seven times a day (0600, 0830, 1100, 1330, 1600, 1800, and 2100). The amount of feed given via the cannula was adjusted so that the total intake of these sows was 125% of the estimated food intake of the AL group.

Cannulation

Between d 65 and 75 of gestation, 36 gilts (Camborough; Pig Improvement [Canada] Ltd.) un-

Table 1. Composition of experimental diets, percentage as based on air-dry analysis

Item	Gestation	Lactation
Ingredient		
Barley	56.3	24.0
Wheat	30.0	24.0
Soybean meal (44% CP)	7.0	22.0
Fish meal	—	5.5
Tallow	2.0	—
Sugar	—	16.0
Canola oil	—	5.0
Dicalcium phosphate	1.7	2.6
Limestone	1.4	—
Vitamin-mineral premix[a]	1.0	1.0
Iodized salt	.5	—
Chemical analysis		
Crude protein, %	13.7	18.6
Lysine, %	.56	1.05
Digestible energy, MJ DE/kg	13.4	15.4

[a]Supplied the following per kg of complete feed: 10,000 IU vitamin A, 1,000 IU vitamin D, 80 IU vitamin E, 2 mg vitamin K, 30 μg vitamin B_{12}, 12 mg riboflavin, 25 mg niacin, 25 mg calcium pantothenate, 600 mg choline, 200 μg biotin, 200 mg folic acid, 5 mg ethoxyquin, 150 mg iron, 12 mg manganese, 120 mg zinc, 12 mg copper, 200 μg iodine, and 100 μg selenium.

derwent surgery for the insertion of a stomach cannula (Pluske et al., 1995) in an experiment conducted in three replicates from March until November 1994. Surgical procedures were approved by the University of Alberta Animal Care Committee to ensure adherence to Canadian Council of Animal Care guidelines.

Diets and Management of Sows

Throughout gestation, gilts were individually housed and offered 2 to 2.3 kg of a conventional gestating sow diet according to their live weight (Table 1). At d 109 of gestation, gilts were moved into individual farrowing crates in a room accommodating 12 crates. The room temperature was maintained between 20 and 23°C, and an evaporative cooling system was used if the room temperature increased above 23°C. From d 109 of gestation and during lactation, all sows were fed a wheat-barley-soybean diet formulated to provide 15.4 MJ DE/kg and 18.6% crude protein (Table 1). All feed troughs were thoroughly cleaned daily, and any dry feed refusals were weighed. Water was freely available to the sows and pigs from nipple drinkers at all times.

At parturition, gilts were randomly allocated to one of three treatments, AL (n = 12), R (n = 9), and SA (n = 8). To facilitate gastric alimentation of SA sows, a suspension was created by adding water (2 parts water:1 part feed) and .5% xanthan gum (Pluske et al., 1995). Alimentation of SA sows commenced within 48 to 96 h after parturition. Litters were standardized to 8 to 10 pigs (8.6 ± .9) within 48 h after farrowing.

At 72 h of age, pigs were processed (teeth and tail clipping, and iron and antibiotic injection), and all males were castrated between d 15 and 19 of lactation. Creep feed was not available. Sow body weight and backfat depth (65 mm from the midline at the last rib; Scanoprobe II, Scano, Ithaca, NY) and litter weights were recorded within 24 h after farrowing and at weekly intervals thereafter.

At weaning, gastric alimentation of SA sows ceased and all experimental sows were fed a diet formulated to provide 13.4 MJ of DE/kg and 13.7% crude protein at 2.5 times their estimated energy maintenance requirements, based on their body weight at weaning. Sows were tested twice daily at 0700 and 1900 for the onset of standing estrus using fence line contact with a rotation of mature vasectomized boars for 15-min periods. The morning after first standing estrus, sows were weighed, and feed intake was reduced to twice maintenance energy requirements until slaughter. At 12 and 24 h after the onset of estrus, sows were artificially inseminated, by one of two inseminators, using pooled semen (3×10^9 spermatozoa/dose) from the same three boars (Alberta Swine Genetics Corp., Leduc, Alberta, Canada). The day of standing estrus was designated as d 0. Sows were slaughtered on d 28 (27.9 ± 1.2) of gestation at a local abattoir, and their reproductive tracts were recovered. Ovulation rate was determined by counting the number of corpora lutea on each ovary, and the number of embryos in utero was determined using the method described by Jindal et al. (1996).

Blood Sampling

At d 26 ± 1.3 of lactation, an indwelling jugular catheter was surgically implanted under general anesthesia via the cephalic vein (Cosgrove et al., 1993). On the day of blood sampling, feed was withheld during the first 2 h of sampling to allow for an estimate of preprandial insulin concentration. Blood samples (3 mL) were withdrawn at 10-min intervals for 12-h periods before and after weaning at 1800 on d 28 (27.9 ± 1.2) for the analysis of plasma LH concentration. Additional 15-mL samples were collected hourly for analysis of plasma IGF-I and insulin concentration. After weaning, 5-mL blood samples were collected at 8-h intervals (0700, 1500, and 2300) from 8 h after weaning until 4 to 5 d after standing estrus for the analysis of plasma LH concentration. Plasma progesterone concentration was analyzed daily from standing estrus until 4 d later. Plasma insulin and IGF-I concentrations were analyzed on the morning of standing estrus. Blood samples were collected into heparinized tubes and centrifuged at $1,500 \times g$ for 15 min. The plasma was decanted and stored at –30°C until analysis.

Estimation of Plasma Hormone Concentration

For RIA, all treatment groups were represented in every assay, and all samples from one sow were analyzed in the same assay. Plasma LH concentrations were determined using the homologous double antibody RIA previously described by Cosgrove et al. (1991). For LH, 200 μL of plasma was assayed; the intra- and interassay CV were 7.5 and 8.2%, respectively. Average sensitivity, estimated as 85% of total binding, was .01 ng/tube. Plasma insulin and IGF-I concentrations were determined in duplicate using the double antibody RIA previously described by Cosgrove et al. (1992). For insulin, 100 μL of plasma was assayed; the intra- and interassay CV were 6.0 and 6.1%, respectively. Sensitivity, defined as 88% of total binding, was .01 ng/tube. For plasma IGF-I, 100 μL of plasma was initially extracted; the intra- and interassay CV were 4.8 and 5.1%, respectively. Sensitivity of the assay, defined as 92% of total binding, was .08 ng/tube. Extraction efficiencies were routinely high, and plasma potencies were not corrected for recovery. Plasma progesterone concentration was determined with the extraction method previously described by Beltranena et al. (1991). Intra- and interassay CV were 4.7 and 11.2%, respectively, and sensitivity estimated as 85% of total binding was .05 ng/tube. Extraction efficiencies averaged 82%, and plasma potencies were corrected for recovery.

Statistical Analysis

All dependent variables were analyzed for normality using the Wilk-Shapiro test (SAS, 1990). Data for the dependent variables sow feed intake, body weight, backfat, litter weight at farrowing, d 7, d 14, d 21, and d 28, and progesterone (fitted to the time of peak LH surge) were analyzed by repeated measures analysis of variance, using the repeated measures GLM procedure of SAS (SAS, 1990). For all dependent variables, sources of variation were treatment, sow within treatment, and the repeated measure of day. In the event of a significant day × treatment interaction, differences among days were computed using split-plot analysis of variance within treatment (SAS, 1990).

Comparisons of plasma insulin concentrations among treatments on d 28 before and after weaning were made over two time periods: preprandial (2 h before the morning feed) and postprandial (1st to 10th h after the morning feed). After weaning, all 11 hourly samples were used in the analysis. Pre- and postprandial and postweaning plasma insulin and pre- and postweaning IGF-I concentration profiles were analyzed with the method of Shaw and Foxcroft (1985) using a sliding window technique to determine the mean plasma concentration. At the time of standing estrus, plasma insulin and IGF-I concentration were compared to the mean postweaning concentration at d 28 of lactation. Mean LH concentrations before and after weaning at d 28 of lactation were calculated using the technique of Shaw and Foxcroft (1985). Episodic LH frequency was determined by visual appraisal using the method of McLeod and

Table 2. Least squares means (± SE) for feed intake, sow body weight, backfat,
and litter weights at weekly intervals during lactation, and from
weaning to standing estrus (SH)

	Treatment[a]		
Item	SA (n = 8)	AL (n = 12)	R (n = 9)
Average feed intake from d 1 to 28, kg/d	7.2 ± .4[x]	5.1 ± .9[y]	2.8 ± .4[z]
Feed intake from d 28 to SH, kg/d	1.36 ± .3[x]	2.30 ± .1[y]	3.2 ± .1[z]
Sow body weight, kg			
d 1	177 ± 12	179 ± 10	176 ± 10
Weight change from d 1 to 28, kg	+5.1 ± 1.2[x]	−16.3 ± .9[y]	−38.9 ± 1.1[z]
SH	165 ± 6[x]	155 ± 4[xy]	147 ± 3[y]
Weight change from d 28 to SH	−17.0 ± 1.8[x]	−7.4 ± 2.5[y]	+9.9 ± 3.0[z]
Backfat, mm			
d 1	17.0 ± 1.4	18.0 ± 1.7	18.3 ± 1.9
Backfat change from d 1 to 28	+1.8 ± .5[x]	−3.7 ± .8[y]	−8.9 ± .9[z]
SH	16.6 ± 1.0[x]	14.0 ± .9[y]	10.8 ± 1.3[z]
Backfat change from d 28 to SH	−2.2 ± 1.0[x]	−.3 ± .5[y]	+1.4 ± .8[z]
No. of pigs weaned	8.4 ± .9	8.5 ± .9	8.8 ± .8
Avg pig growth rate, g/d	235 ± 11.5	240 ± 9.5	214 ± 11.4
Energy balance[b] at d 28, MJ DE/d	+33.3 ± 5[c]	−13.2 ± 7[d]	−40.2 ± 5[e]
Energy balance[b] at SH, MJ DE/d	−1.9 ± .8[c]	+11.3 ± 3[d]	+24.4 ± 3[e]

[a]SA, AL, and R = sows with different lactational feed intakes. SA = sows receiving feed intake to 125% of ad libitum, AL = sows receiving ad libitum access to feed, and R = sows receiving 50% of estimated ad libitum feed intake.

[b]Energy balance = (energy for sow maintenance + energy for milk production) − energy in feed consumed. Energy for sow maintenance = Sow body weight$^{.75}$ (kg) × 110 kcal × 4.184. Energy for milk production = ([(2.54 × ADG) + (78.7 × BW) + 153]/1,000) × (4.184 × litter size)/k_1, where ADG = average daily gain (g) per pig during the period of lactation, BW = weight (kg) of the average pig at the beginning of the short period, and K_1 = efficiency of utilization (.72) of energy for milk production (Noblet et al., 1990).

[c,d,e]Means within a row lacking a common superscript letter differ ($P < .05$).

[x,y,z]Means within a row lacking a common superscript letter differ ($P < .001$).

Craigon (1985) and the pulse definition used by Cosgrove et al. (1991). For the dependent variables, mean plasma insulin, IGF-I, LH, and LH pulse frequency, sources of variation were treatment, sow within treatment, and day. Differences among day within treatment were computed using Duncan's multiple range test in a split-plot analysis of variance (SAS, 1990).

Energy balance at weaning and standing estrus, weaning to estrus interval, ovulation rate, and embryo survival were analyzed using analysis of variance, fitting treatment and the error term of sow within treatment. Differences among treatment means were determined using Duncan's multiple range test (SAS, 1990).

Results

Sow Responses to Treatment During Lactation

Although 36 gilts were placed on experiment, seven were withdrawn before completion of lactation: one gave birth to three pigs, and three litters developed severe diarrhea early in lactation and were considered aberrant. Three superalimented sows were withdrawn from the study, one because of metritis, another because of a rectal prolapse, and the third sow failed to recover from anesthesia associated with surgery at d 26 of lactation. Sows with ad libitum access to feed lost body weight ($P < .01$) and backfat ($P < .01$) during lactation, whereas SA sows, which received more feed ($P < .001$), gained weight ($P < .001$) and backfat ($P < .001$). Group R sows ate less ($P < .01$) and lost more weight ($P < .001$) and backfat ($P < .001$) than did AL sows during lactation. Litter weights increased curvilinearly during lactation ($P < .001$), but weaning weight and pig growth rate did not differ among treatments ($P > .05$); see Table 2 and Pluske et al. (1988) for further discussion.

There were no treatment differences in mean pre- or postprandial plasma insulin concentration during the 12-h period before or after weaning (Table 3). An effect of day ($P < .05$) showed that all treatment groups exhibited a decrease in plasma insulin concentration during the 12-h period after weaning that corresponded to an overnight feed deprivation. Analysis of treatment within day for mean plasma IGF-I concentration showed that it was lower ($P < .001$) in R than in AL or SA sows and remained so during the 12-h period after weaning ($P < .001$). No treatment differences in plasma IGF-I concentration were observed between SA and AL sows. During the 12-h period before weaning, mean plasma LH concen-
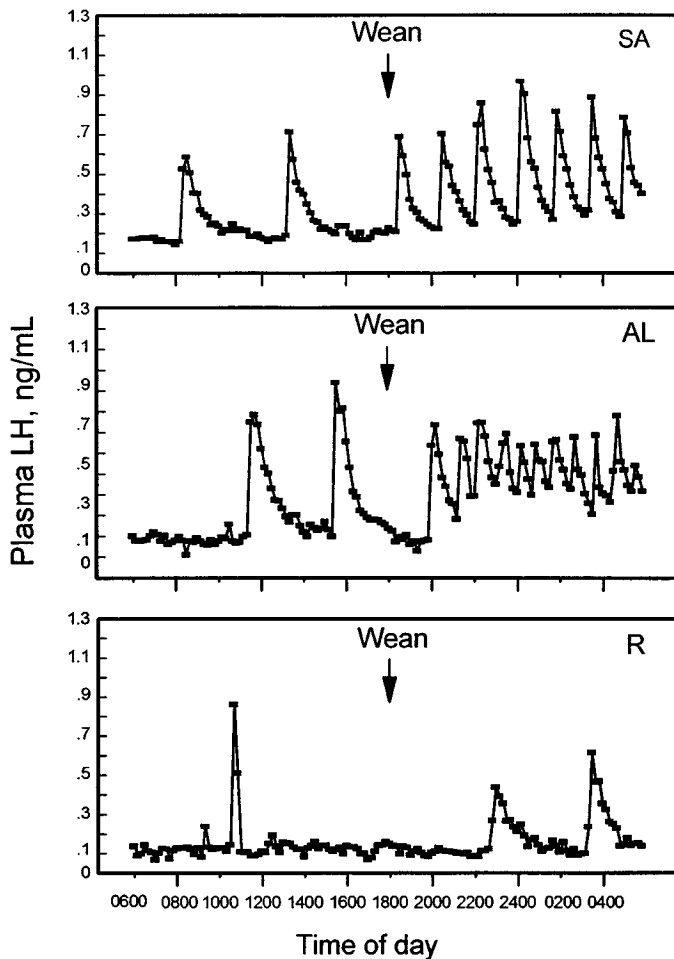
Figure 1. Representative profiles of plasma LH during a 24-h continuous bleed for 12 h before and 12 h after weaning at d 28 of lactation. Treatments are super-alimentation (SA), ad libitum (AL), and restricted (R).

tration and LH pulse frequency were lower ($P < .03$ and $P < .06$, respectively) for R sows than for AL or SA sows (Figure 1). In response to weaning, mean plasma LH concentration and pulse frequency increased ($P < .001$ for both) independent of treatment. After weaning, no treatment differences in mean plasma LH concentration or pulse frequency were observed between SA or AL sows; however, mean plasma LH concentration and pulse frequency continued to be lower in R sows ($P < .01$ for both).

Sow Responses After Weaning

There was a significant day × treatment interaction ($P < .05$) for feed intake after weaning. Although all sows were allowed to eat the equivalent of 2.5 times their estimated maintenance energy requirements during the period from weaning to standing estrus and twice maintenance energy requirements after standing estrus, SA sows ate less than AL or R sows ($P < .001$ for both). On average, SA sows ate only 38% of

their feed allowance after weaning compared to 70% by AL sows and 91% by R sows. From weaning to standing estrus, R sows gained weight and backfat ($P < .001$ for both), whereas AL and SA sows lost weight and backfat, and SA sows lost more ($P < .01$) weight and backfat than AL sows (Table 2). Nevertheless, at standing estrus, absolute values for body weight and backfat of SA sows were still greater than those of R sows ($P < .04$ and $P < .02$, respectively), whereas the body weight of AL sows was not different from that of either SA or R sows. Treatment differences in sow energy balance at d 28 ($P < .03$) and standing estrus ($P < .02$) showed that energy balance was greater in SA sows than in those on the other treatments, whereas at standing estrus the energy balance for SA sows was lower ($P < .01$) than that for AL and R sows.

From weaning to standing estrus, plasma IGF-I concentration in R sows increased ($P < .01$), whereas in AL and SA sows it did not (Table 3). From weaning to standing estrus, mean plasma insulin concentration in R and AL sows increased ($P < .035$); however, no change was observed in SA. At standing estrus there was no difference ($P > .05$) for mean plasma insulin or IGF-I concentration among treatments. After standing estrus, plasma progesterone concentration was not different ($P < .05$) among treatments within day; however, an effect of treatment × day ($P < .001$) showed that plasma progesterone increased linearly after the LH surge. Regression analysis revealed that embryo survival was correlated to plasma progesterone at 72 h after the LH surge (n = 20; $P < .01$; r = .7). Weaning-to-estrus interval was extended ($P < .02$) for R sows compared with SA or AL sows, and ovulation rate and embryo survival did not differ among treatments.

Discussion

Differential body weight and backfat changes were achieved by the feeding regimen imposed. The SA treatment increased body weight and backfat, whereas they were depleted in AL and R sows. Interestingly, SA did not increase milk production (Pluske et al., 1998). Hence, almost all of the additional nutrients available to the sows were directed toward tissue anabolism (see Clowes et al., 1998 for further discussion) and could have potentially ameliorated any inhibitory effects of lactational catabolism on reproductive performance. Consistent with the data reported by Nelssen et al. (1985), Mullan and Williams (1989), and Koketsu et al. (1996), restriction of feed intake during lactation seemed to decrease litter weight at weaning (Pluske et al., 1998) and extended the weaning-to-estrus interval (Reese et al., 1982; Mullan and Williams, 1989). Despite the increase in body weight and backfat during lactation, the fertility of SA sows was not different from that of

Table 3. Least squares means (± SE) for plasma insulin; IGF-I; LH concentration, and LH pulse frequency at 28 d of lactation, before and after weaning, and at standing estrus (SH); weaning-to-estrus interval (WEI), ovulation rate (OR), and embryo survival (ES)

| | Treatment[a] | | |
Item	SA (n = 8)	AL (n = 12)	R (n = 9)
Insulin, ng/mL			
Preprandial, d 28	1.42 ± .12	1.35 ± .16	1.22 ± .10
Postprandial, d 28	2.65 ± .21	2.57 ± .14	2.20 ± .16
After weaning	1.84 ± .2	2.10 ± .15	1.81 ± .16
SH	1.37 ± .3	2.76 ± .93	2.18 ± .43
Change from d 28 to SH, ng/mL	−.47 ± .41[d]	+.66 ± .21[e]	+.37 ± .31[f]
IGF-I, ng/mL			
d 28	51.2 ± 1.8[x]	53.2 ± 1.9[x]	25.5 ± 1.0[y]
After weaning	51.2 ± 1.8[x]	53.3 ± .1[x]	25.9 ± .1[y]
SH	51.6 ± .9	55.1 ± .2	52.1 ± .2
Change from d 28 to SH, ng/mL	+.4 ± .5[x]	+1.8 ± .2[x]	+26.2 ± .2[y]
Mean LH, ng/mL			
d 28	.33 ± .04[d]	.29 ± .03[d]	.22 ± .03[e]
After weaning	.51 ± .04[d]	.45 ± .03[d]	.36 ± .03[e]
LH frequency, pulses/12 h			
d 28	3.3 ± .8[d]	2.7 ± .6[d]	.2 ± .6[e]
After weaning	7.8 ± .8[d]	6.5 ± .6[d]	3.8 ± .6[e]
WEI, d	4.4 ± .8[d]	4.2 ± .1[d]	6.3 ± .1[e]
OR[b]	18.3 ± 3	14.4 ± 3	15.6 ± 3
ES, %[c]	73.2 ± 15.2	83.3 ± 10.1	72.3 ± 15.8

[a]SA, AL, and R = sows with different lactational feed intakes. SA = sows receiving feed intake to 125% of ad libitum, AL = sows receiving ad libitum access to feed, and R = sows receiving 50% of estimated ad libitum feed intake.

[b]Number of observations: SA = 6, AL = 7, R = 7.

[c]Number of observations: SA = 5, AL = 7, R = 7.

[d,e,f]Means within a row lacking a common superscript letter differ ($P < .05$).

[x,y,z]Means within a row lacking a common superscript letter differ ($P < .01$).

sows fed on an ad libitum basis throughout lactation. In light of these observations, we must reject our hypothesis and conclude that feeding a sow beyond her ad libitum feed intake during lactation did not overcome the inherent inhibitory effects of pig suckling and improve fertility after weaning.

The metabolic state of these sows was characterized in part by plasma IGF-I and insulin concentrations. In lactating sows, the association between plasma insulin and feed intake is equivocal. Data reported in this experiment and that of Armstrong et al. (1986) and Mullan and Close (1991) did not find an association between feed intake and plasma insulin concentration, whereas Tokach et al. (1992), Koketsu et al. (1996), and Zak et al. (1997) all reported that periods of feed restriction during lactation were associated with reduced mean plasma insulin concentration and LH secretion. Plasma IGF-I concentration is associated with periods of restricted feed intake during lactation (Tokach et al., 1993; Zak et al., 1997) and also in this study with reduced plasma LH secretion. Plasma IGF-I may also be a likely candidate for the signaling of metabolic state to the hypothalamo-adenohypophyseal axis. Evidence for this comes from the data of Whitley et al. (1995), in which in vitro incubation of adenohypophyseal cells with IGF-I increased the

secretion of LH. However, as demonstrated by Booth et al. (1994) in prepubertal gilts, the increase in plasma IGF-I concentration in response to feeding to appetite after a period of feed restriction occurred after 30 h, whereas plasma LH secretion had already increased within a few hours after refeeding. The similar latent response of plasma IGF-I concentration to alterations in metabolic state at weaning in sows may modulate the LH response to the removal of the inhibitory effects of suckling, possibly explaining the lower episodic LH frequency in R sows after weaning.

Relatively severe catabolism experienced by R sows resulted in a suppression of LH secretion during lactation and in the immediate postweaning period, which is in agreement with the data reported by Baidoo et al. (1992) and Tokach et al. (1992). Even though SA sows gained body weight and backfat and were anabolic (Clowes et al., 1998), and AL sows were relatively catabolic during lactation, LH secretion before weaning was similar between these treatments. Contrary to this observation, plasma LH secretion was consistently less in sows suckled by 12 as opposed to six pigs (Mullan et al., 1991). These observations are consistent with the suggestion that the suckling stimulus provided by the pigs is the predominant inhibitor of LH secretion during lactation

in sows (Foxcroft, 1992), which cannot be overcome by additional feed intake and associated anabolism.

As expected from the data of Foxcroft et al. (1987), SA and AL sows responded to weaning with a robust increase in LH secretion. However, R sows exhibited a continued suppression of LH for at least 12 h after weaning and also exhibited a delay in the weaning-to-estrus interval. The weaning-to-estrus interval has previously been correlated with LH secretion before weaning (Armstrong et al., 1986; King and Martin, 1989; Tokach et al., 1992); thus, the delay in the time to ovulation in R sows may be dependent on the tonic inhibition of LH release during lactation, as a result of increased catabolism. However, LH secretion in these sows was still suppressed after weaning compared with that in AL or SA sows. The prolonged weaning-to-estrus interval in R sows may be due to retarded ovarian development in lactation as a result of severe catabolism (Miller, 1996), and hence a lack of responsiveness to gonadotropin stimulation. In addition, the lower frequency of episodic LH secretion in R sows that persisted into the 12-h period after weaning may have been a less effective signal for follicle recruitment and selection (Foxcroft, 1992).

Data reported for postpartum dairy cows demonstrated a correlation between the timing of the first postpartum estrus and the time at which cows approached a relatively less negative energy balance after the energy balance nadir (Cranfield and Butler, 1990; Zurek et al., 1995; Senatore et al., 1996). In the present study, AL and R sows were also approaching a relatively more positive energy balance between weaning and estrus. In contrast, although the weaning-to-estrus interval was not different between AL and SA sows, when estrus occurred SA sows were in a negative energy balance. If the situation between weaning and estrus was analogous to that of lactation, a sow approaching a more catabolic state would be expected to exhibit an extended weaning-to-estrus interval (Reese et al., 1982; Armstrong et al., 1986; Koketsu et al., 1996). However, data from King and Williams (1984) have shown that the weaning-to-estrus interval cannot be reduced by additional feed consumption after weaning, and conversely, restriction of feed intake after weaning in sows that were fed 6 kg during lactation did not extend the time to return to estrus (Baidoo et al., 1992). The inability of postweaning feed intake to affect return-to-estrus interval, combined with the observation that SA sows approached a less positive energy balance after weaning, would suggest that the rate-limiting events culminating in estrus are dependent on events during lactation. If we accept that metabolic state in lactation exerts the predominant effect on fertility after weaning, then central and ovarian aspects are potentially involved (Foxcroft et al., 1995). Indeed, LH has the ability to affect ovarian development directly (Cox and Britt, 1982; Rojanasthien et al., 1988; De Rensis

et al., 1991). Local effects of insulin (Cox et al., 1987; Meurer et al., 1991) and nutrition (Flowers et al., 1988) on the number of follicles selected to ovulate have been established in gilts. However, the results of this experiment clearly indicate that the availability of additional nutrients during lactation did not increase ovulation rate or decrease the time to return to estrus. Thus, follicle sensitivity and ability to respond to the final recruitment signal at weaning (Foxcroft et al., 1995) may be dependent on factors other than the availability of nutrients, and we speculate that the pig suckling stimulus per se, independent of its effects on LH secretion, may have a direct role in the regulation of ovarian development during lactation.

In contrast to the data reported by Kirkwood et al. (1987), Baidoo et al. (1992), and Zak et al. (1997), restriction of feed intake during lactation did not affect embryo survival compared to those animals maintained on a high feed intake during lactation, although the absolute values reported for high and restricted-fed lactational sows were similar in all experimental paradigms. Plasma progesterone is generally considered to have a critical role in mediating embryo survival by affecting the secretion of uterine factors (Roberts and Bazer, 1988). Independent of treatment, a correlation between embryo survival and plasma progesterone was observed at 72 h after the LH surge in the present study. This observation is consistent with the data reported by Pharazyn et al. (1991) and Jindal et al. (1996) for cyclic gilts and with data from a recent study with weaned primiparous sows (Zak, Jindal, Aherne, and Foxcroft, unpublished observation).

In conclusion, the data are not consistent with the hypothesis that making sows anabolic during lactation will improve fertility after weaning. Collectively, these data suggest that the infusion of additional nutrients during lactation did not improve the weaning-to-estrus interval compared to supplying feed for consumption on an ad libitum basis. Weaning-to-estrus interval is more dependent on metabolic status during lactation rather than on changes in metabolic state after weaning, and in part this observation may be dependent on mean plasma IGF-I concentration and LH secretion at the time of weaning.

Implications

These data demonstrate unequivocally that the effects of suckling by pigs through the tonic inhibition of luteinizing hormone secretion (or via other aspects of ovarian function) is the predominant controller of fertility during lactation and that making sows anabolic during lactation will not abrogate this effect. Producers should manage lactating sows so that catabolism of body tissue is minimized during lacta-

tion; however, no further advantage to the sows' fertility will be gained by making them anabolic.

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