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THE EFFECT OF WEIGHT AND BODY FAT LOSS ON THE REPRODUCTIVE PERFORMANCE AND ENDOCRINOLOGICAL STATUS OF THE LACTATING AND POSTWEANING SOW

by

SAMUEL KOFI BAIDOO

A THESIS

SUBMITTED TO THE FACULTY OF GRADUATE STUDIES AND RESEARCH

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The undersigned certify that they have read, and recommend to the Faculty of Graduate Studies and Research, for acceptance, a thesis entitled THE EFFECT OF WEIGHT AND BODY FAT LOSS ON THE REPRODUCTIVE PERFORMANCE AND ENDOCRINOLOGICAL STATUS OF THE LACTATING AND POSTWEANING SOW submitted by SAMUEL KOFI BAIDOO in partial fulfilment of the requirements for the degree of DOCTOR OF PHILOSOPHY in ANIMAL NUTRITION.

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ABSTRACT

The effects of high (H, 6 kg d^{-1}) or low (L, 3 kg d^{-1}) levels of feed intake during lactation and the postweaning period on the length of the remating interval, ovulation rate, conception rate and percentage embryo survival were studied in eighty-nine second parity Lacombe sows. The endocrine status of sows on different lactation and postweaning nutritional regimes was also studied.

Sows on the L level of feeding catabolized more ($P<0.01$) body tissue to maintain milk yield and piglet growth. The sows fed H during lactation lost equal amounts of body fat tissue and lean body mass. The L fed sows lost more ($P<0.05$) body fat than lean body mass during lactation. The major benefit of increasing lactation feed allowance was in reducing the amount of maternal body weight and fatty tissue loss. Regression equations indicated that to maintain maternal body weight during lactation, the sow required 7.73 kg feed daily and 8 kg feed daily to maintain backfat. None of the piglet parameters studied were affected ($P>0.05$) by the level of feeding of the sows during lactation.

Twelve sows fed H and twelve sows fed L during lactation were fitted with indwelling vena cava cannulae and blood samples were collected at 1 h intervals for 24 h on d 2 and d 28 postpartum. Plasma samples were analysed by radioimmunoassays for concentration of growth hormone, insulin, glucagon, cortisol, prolactin, follicle stimulating hormone (FSH) and luteinizing hormone (LH). Concentration of glycerol in plasma was analysed by the enzymatic method. On d 2 postpartum, there was no difference ($P>0.05$) in plasma concentration of any of the blood hormones studied between treatment groups. On d 28 postpartum, the L fed sows had a higher ($P<0.05$) concentration of glycerol, growth hormone and cortisol and a lower ($P<0.05$) plasma concentration of insulin. There were no differences ($P>0.05$) in plasma concentration of glucagon, LH and FSH on d 28 postpartum, although they were significantly different ($P<0.05$) from the plasma levels observed on d 2 postpartum.

At weaning, sows were randomly assigned within lactation treatments to either a low (3 kg d^{-1}) (L) or a high (6 kg d^{-1}) (H) feeding regimen. Both L-H and L-L treatments

resulted in an increased ($P<0.05$) incidence of anestrus and extended ($P<0.05$) remating interval. Ovulation rate was not influenced ($P>0.05$) by feed intake during lactation and postweaning. Pregnancy rate and embryo survival were reduced ($P<0.05$) by low level feeding during lactation. Weaning was consistently associated with a rise ($P<0.05$) in basal LH concentrations whilst FSH secretion remained unaffected in all treatment groups. Plasma concentration of estradiol-17β was not influenced ($P>0.05$) by feed intake. Sows fed low levels during lactation had decreased ($P<0.05$) plasma levels of FSH and LH during the weaning to remating interval. However, there was no difference ($P>0.05$) between treatment groups in plasma concentrations of LH or FSH in sows that ovulated.

The results of this study suggest that low level feeding during lactation will result in body weight and backfat loss and will change the hormonal state of the sow favouring the catabolic hormones, namely cortisol and growth hormone. This catabolic state may hinder the synthesis and secretion of the gonadotropins. Thus extended remating interval, low conception rate and high embryo mortality observed in this study may primarily be due to lack of LH stimulation of the ovary.

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I. INTRODUCTION

An essential component of the overall reproductive efficiency of sows is the length of the interval from weaning to estrus. A short interval is necessary to maximize the number of pigs marketed per sow per year. About a third of the breeding sows are culled annually because of anestrus or infertility (Dagorn and Aumaitre, 1979; Fahmy, 1981; Friendship et al., 1986). Thus prolonged anestrus in weaned sows will decrease the number of litters per sow per year and as a consequence will reduce herd output. Inadequate nutrition of sows during lactation combined with excessive bodyweight and (or) fat losses have been implicated as major factors exerting an influence on the incidence of postweaning anestrus and embryo mortality (Aherne and Kirkwood, 1985).

A. LACTATION FEED INTAKE

There have been numerous contemporary reviews of sow nutrition as it affects reproductive performance (MacPherson et al., 1969; Elsley and MacPherson, 1972; ARC, 1981; Cole, 1982; Close et al., 1984; NRC, 1988). Many of the experiments reviewed dealt with a discrete area of the breeding cycle (i.e.pregnancy, lactation or the postweaning period) and as such only demonstrated direct effects. However, it is becoming increasingly apparent that nutrition may have indirect effects, the most notable being the influence of feed intake and changes in body weight and body composition during lactation on the postweaning and early gestation performance.

Factors affecting lactating sow feed intake

The sow's protein, vitamin and mineral requirements are generally met or exceeded in the lactation phase of production. However, the energy requirements frequently are not met during lactation due to the sow's inability to consume adequate amounts of feed (O'Grady et al., 1973; 1985). The energy requirements of sows vary due to differences in maintenance requirements which vary with sow body weight. In addition, the level of milk production varies significantly among sows (O'Grady et al., 1985) and thus influences energy

requirements. For example, young sows utilize some of their energy intake for growth which causes the additional drain on body energy reserves. Thus, the feed intake or energy intake of the sow during lactation is influenced by a number of environmental and nutritional factors in addition to the physiological state of the animal (NRC, 1987). Among the more important factors affecting sow feed intake are the following: temperature, genetics, system of feeding, gestation feed intake, energy density of the diet, protein content of the diet, dietary mineral and vitamin levels, and water.

1. Temperature

The temperature of the environment can have a significant effect on feed intake during lactation (Lynch, 1977; Armstrong et al., 1986). In the experiments of Lynch, sow feed intake was decreased by 12% from 5.23 kg d^{-1} at 21°C, to 4.58 kg d^{-1} at 27 °C. The growth rate of piglets was lower at the high temperature (168 g d^{-1} vs 195 g d^{-1}) which probably reflects a decrease in milk production, rather than a direct effect of temperature on piglets since the upper critical temperature for piglets is 31°C (Holmes and Close, 1985). However, the major effect of high environmental temperatures is an increase in heat production and a decrease in feed intake. This effect can be alleviated by the feeding of high energy diets to maintain energy intake (Cox et al., 1983).

In contrast to high temperature, cold temperatures will stimulate sow feed intake but will also increase the sows energy requirements. For each 1°C below the sow's lower critical temperature (18 - 20°C), there is an increase of approximately 4% in maintenance energy cost (Verstegen et al., 1987). The lower critical temperature is lower for heavier and/or fatter animals and it decreases as the intake of energy increases (Holmes and Close, 1985). The effective insulation value of subcutaneous fat was 30% lower in emaciated sows (95 kg after 3 parities) compared to standard sows (165 kg) (Hovell et al., 1977). Consequently heat loss by thin sows at 10°C below critical temperature was 25% greater than for fat sows (Holmes and Close, 1985).

2. Genetics

Part of the variability associated with feed intake can be attributed to differences in the genetic base or between breeds. The research in the area of genetic effects on feed intake is limited. However, there have been reports (Pond et al., 1981; O'Grady et al., 1985) that Large White and Landrace breeds consume more feed during lactation than do Yorkshire and Chester White breeds. Furthermore, O'Grady et al. (1985) showed that sows sired by Large White boars ate more in lactation than those sired by Landrace boars. Much of this difference may be in response to differences in milk production levels between these breeds.

3. System of feeding

A common feeding regimen used by producers is to restrict feed intake on the day of parturition and then gradually increase the feed allowed until the sow is fed ad libitum by 5 - 7 d postpartum (Krider et al., 1982). Recently Moser et al. (1987) demonstrated that ad libitum feed intake can be allowed immediately postpartum without detrimental effects on the general health of the sow and litter. Even with ad libitum feeding some lactating sows are unable to consume sufficient quantities of feed to satisfy their requirements for energy and protein (O'Grady et al., 1985). Average voluntary daily feed intake during lactation as low as 2.9 kg (Cox et al., 1983) and 3.1 kg (King et al., 1984) have been reported for primiparous sows in commercial piggeries. Others have also reported very low feed intakes for lactating sows fed ad libitum (King and Dunkin, 1986a; Britt, 1986; Armstrong et al., 1986; Kirkwood et al., 1988).

Feeding wet feed may have some merit increasing feed intake. O'Grady et al. (1985) indicated that sows consuming wet feed during lactation tended to consume more feed, lose less weight and wean heavier litters.

4. Parity effects

As a sow ages she gains weight and thus increases her maintenance requirements. In addition, she increases her milk production and litter size. This occurs in general until

the sixth parity and there is a parallel increase in lactational feed intake (O'Grady et al., 1985). Beyond the sixth parity a decrease in productivity occurs and a concomitant decrease in feed intake occurs (O'Grady et al., 1985).

5. Gestation feed intake

If pregnant sows are offered feed ad libitum, they will consume more energy during gestation than they require for maintenance and the development of the products of conception. The sows will therefore deposit body fat and protein. It is generally accepted that sows with higher rates of gain during pregnancy will lose more weight in lactation than those with lower rates of gain, even though identical feed intakes are maintained in lactation (Williams et al., 1985; Mullan, 1987; Roos et al., 1987; Yang et al., 1988). This may result from the higher maintenance requirement of the high-gain animals but that explains only part of the effect and it is possible that there is an enforced mobilization of body fat during lactation because large amounts of lipid have been deposited during pregnancy.

6. Energy density of the diet

Fats, oils, or carbohydrates are normally used to increase energy density of lactating sow diets. However, lactating sows tend to eat less of high energy diets and more of diets with low energy density. In some studies this compensation led to similar energy intakes on diets of very different energy density. Pollman et al. (1981) reported similar energy intakes by sows fed a control diet with or without 8% tallow added. A comparable effect was reported by Seerly et al. (1981) in that sows ate more of a control diet than of the same diet supplemented with corn oil but achieved similar digestible energy intakes. Several studies have shown an increase in energy intake by feeding 3 to 8% fat in the lactation diet (O'Grady et al., 1975; O,Grady and Lynch, 1978; Reese et al., 1982a; Cox et al., 1983).

7. Protein content of the diet

Mahan and Mangan (1975) reported that protein content of the diet fed to sows during pregnancy and lactation influenced feed intake during lactation. Gilts fed a 9% protein diet during pregnancy consumed almost 50% more feed when fed an 18% protein lactation diet than when fed a 12% protein diet. Sows fed a 17% protein gestation diet showed only a 5% increase in intake. Intake of the 18% protein lactation diet was independent of protein level in pregnancy, whereas intake of a 12% protein diet in lactation linearly increased with level of protein fed in pregnancy. Thus, it appears that feeding high protein diets in lactation stimulate feed intake only where the protein level fed in pregnancy is low.

8. Dietary mineral and vitamin levels

Sow feed intake was reported to increase significantly when dietary calcium was increased from 0.6% to 0.8% and phosphorus from 0.5% to 0.6%. However, feed intake declined when the calcium level was raised to 0.9% and the phosphorus level raised to 0.7% (Mahan and Fetter, 1982). Deficiencies or excesses of any of the mineral or vitamin components of the diet are likely to depress appetite (ARC, 1981; NRC, 1988).

9. Water

Water is involved in several physiological functions necessary for maximum animal production. Among these are temperature regulation, nutrient and waste transportation, metabolic processes, lubrication and milk production. Milk is about 80% water (Pond and Houpt, 1978) and is also a high-protein, high mineral food which can leave the body deficient in water. The water intake of lactating sows increases with dry matter intake (NRC, 1988) and it has been shown that lactating sows consume as much as 20 kg of water daily (Riley, 1978; Lightfood and Armsby, 1984).

Other husbandry and management factors such as disease and other environmental factors may influence the ad libitum intake of the sow, although the effects are difficult to quantify.

Effects of feed intake during lactation on the number of days to postweaning estrus

Several recent experiments have indicated that sows that lose excessive amounts of weight or body condition during lactation will have extended remating intervals and an increased incidence of anestrus (Reese et al., 1982a,b, 1984; King et al., 1982; King and Williams, 1984a,b; Hughes et al., 1984; Nelssen et al., 1985; King and Dunkin, 1986a,b; Johnston et al., 1986; Brendemuhl et al., 1987; Lythgoe, 1987; Kirkwood et al., 1987). Other studies (Eastham et al., 1988; Kirkwood et al., 1988) indicate that nutrition during lactation has no significant effect on the return to estrus by sows. The inconsistent response of sows to the level of nutrition during lactation between experiments could be attributted to differences in the body condition of sows at farrowing and the amount of weight lost during lactation.

In an experiment by King and Dunkin (1986a), gilts were bred at two different ages (341 vs 214 days at conception), the older sows were heavier (171 vs 143 kg) and had more backfat (31.0 vs 21.3 mm) at farrowing. The older sows lost more weight and back fat than the young gilts (31.4 vs 21.3 kg and 8.4 and 4.9 mm respectively). The interval between weaning and mating was shorter for the older than the younger animals. Nutrition during lactation does not affect the subsequent reproductive performance in older first-litter sows to the same extent as their younger counterparts (King and Dunkin, 1986a). This suggests that it is not the absolute amount of either liveweight or backfat which is lost that is important, but rather the proportion of that present at farrowing.

The effect of plane of nutrition during lactation on subsequent reproductive performance has also been studied in multiparous sows. Varley and Cole (1978) failed to show any relationship between the weight loss of sows during lactation and the interval between weaning and mating, despite liveweight losses of up to 30 kg. However, Hughes and Calder (1979) reported that the average interval between weaning and mating was increased from 5.2 to 18.2 d when the feed intake of sows was reduced from 8 to 4 kg d^{-1}. No details on the weight loss of these sows were given. Henry et al. (1984) also fed second to fifth parity sows either 7 or 3 kg d^{-1} during a 35 - d lactation. Sows fed 3 kg d^{-1} lost more liveweight (48 vs 16 kg) and backfat (5.7 vs 1.9 mm) during lactation and had an extended

interval between weaning and mating (9.8 vs 5.6 d).

The effect of feed restriction during lactation on the weaning to mating interval may be due to a decrease in the intake of either energy or protein. Recent studies (Reese et al., 1982a,b; Nelssen et al., 1985) have shown that with a protein intake of 630 g d^{-1}, energy intake could be reduced to 45 MJ DE d^{-1} with no increase in the weaning to estrus interval. If the weaning to mating interval is related to energy balance during lactation, then the sow must have a mechanism to monitor the degree of energy deficit. For example, an average energy balance for a sow during lactation may not be applicable for all sows, because it may be the energy balance just prior to weaning which is important in determining subsequent reproductive performance. Furthermore, it is unlikely that the same critical level of dietary energy intake would apply to all sows, since sows begin lactation at a range of body weights or levels of body condition.

Muscle wasting, as indicated by increased creatine concentrations, was reported by Reese et al. (1984) to be higher in restricted fed sows compared to ad libitum fed sows. However, this was considered not to be related to expression of estrus since the creatine concentrations were similar for restricted fed sows that did return to estrus and those that did not. On the contrary, recent studies (King and Williams, 1984b; King and Dunkin, 1986b; Brendemuhl et al., 1987) have indicated that though reduced energy intake (below 45 MJ DE d^{-1}) will increase sow weight and backfat loss, protein intake during lactation exerts a greater influence on the interval between weaning and estrus. Further indication of the importance of the level of protein intake of sows during lactation was demonstrated by King and Williams (1984a) and King and Dunkin (1986b) using nitrogen balance studies. In these studies, there was a positive association between estimated daily nitrogen loss during lactation and weaning to estrus interval. Mobilization during lactation of significant amounts of protein from muscle tissue is common in first litter sows allowed low protein intakes (Duee and Desmoulin, 1982). Interactions between protein and energy intakes may be more important during lactation than during other reproductive phases. The results of King and Williams (1984a) and Brendemuhl et al. (1987) indicate that if either protein or energy is limited during lactation, then the

results are similar, loss of weight and backfat and poor reproductive performance after weaning. However, these authors demonstrated that high energy intakes during lactation do not compensate for low protein intakes, but high protein intakes will partially offset effects of low energy intakes. Thus the substantial nitrogen loss sustained by lactating sows with low protein intakes was the primary nutritional factor responsible for the delayed weaning to estrus interval. There is the possibility that the female pig can monitor the degree to which body reserves are mobilized, and then relate this to subsequent reproductive function.

Effect of feed intake during lactation on ovulation rate

Brooks (1982) suggested that gilts that became catabolic during lactation may remain so after weaning and as a consequence have reduced ovulation rates. However, other studies have failed to detect an effect of weight loss during lactation on ovulation rate (den Hartog and van der Steen, 1981; King et al., 1984; King and Williams, 1984a,b; Hughes et al., 1984; King and Dunkin, 1986a,b).

Effect of feed intake during lactation on embryo survival

There is a general agreement that low level feeding in lactation adversely influences the weaning to remating interval, but has no effect on ovulation rate, however, the influence of lactation feed level on subsequent embryo survival is not clear. King and Williams (1984a) showed no influence of low lactation dietary energy or protein intakes on embryo survival, however, Hughes et al. (1984) and King and Williams (1984b) indicated an adverse effect of low level feeding. It is not known whether it is the backfat level or the amount of fat lost during lactation that affects embryo survival, that is will poor lactation nutrition only adversely affect those sows already relatively thin. If condition loss in lactation does affect embryo survival, the mechanism of action remains unclear.

B. FEED INTAKE DURING THE POSTWEANING PERIOD

The main objectives of feeding in the postweaning period is to stimulate a prompt return to estrus and support adequate ovulation and conception rates. MacLean (1969) recommended a "drying off" period of at least 24 h postweaning during which time the sow receives no food or water, as an effective means of shortening and synchronizing the return to estrus. Recent evidence indicates no benefit of this practice when conception rates are greater than 80% (Tribble and Orr, 1982) and it may be detrimental with some management systems (Allrich et al., 1979).

Effect of feed intake during the postweaning period on remating interval

Increasing the level of feeding during the postweaning period, has been reported to shorten the interval to service in primiparous sows (Brooks and Cole, 1972; King and Williams, 1984a), increase the number of sows exhibiting estrus within 10 days of weaning (Brooks and Cole, 1972; Fahmy and Dufour, 1976; King and Williams, 1984a) and increase the synchronization of estrus (Dyck, 1972). Other studies failed to confirm an effect of nutrition on the length of the weaning to estrus interval (den Hartog and van der Steen, 1981; Tribble and Orr, 1982). It is possible that the results of the study of Brooks and Cole (1972) can be explained by their use of gilts which may respond differently to postweaning feed intake than do multiparous sows. However, den Hartog and van der Steen (1981) also used primiparous sows and observed no response to variations in postweaning feed intake. It is therefore possible that a further indirect nutritional effect exists, that is lactation feed level (and thus weight change pattern) may affect the response of sows to the postweaning feed level.

Effect of feed intake during the postweaning period on ovulation rate

The available information on the influence of postweaning nutrition on ovulation rate at the first estrus and subsequent litter size is also nonconclusive. The normal remating interval for a sow can vary but may be as low as 4 to 5 days and as such, appears to

correspond to the follicular phase of a normal estrous cycle. Therefore it may be expected that sows will respond to nutritional changes in this period in a similar manner to that seen in gilts. However, there is little evidence (Fahmy and Dufour, 1976; Tribble and Orr, 1982) to support a claim for high level feeding during the remating interval affecting either the ovulation rate or subsequent litter size of sows. This may in part be due to the relatively short time span involved since Dyck (1974) reported that increasing the feed level will not affect the first postweaning ovulation rate, but does increase ovulation rate at the second postweaning estrus.

Effect of feed intake during the postweaning period on conception rate

Increasing postweaning feed levels for primiparous sows has been reported to improve conception rates (Brooks and Cole, 1972), although this was not confirmed by results with older sows (Dyck, 1972; Brooks et al., 1975; Tribble and Orr, 1982). However, the possibility of an interaction between lactation and postweaning feed levels remains to be adequately investigated. It is concluded that level of feeding in the remating interval is unlikely to improve reproductive performance unless it is to reverse the reduction in performance due to poor nutritional management during lactation (Pike and Boaz, 1972).

C. BODY COMPOSITION OF SOWS DURING LACTATION

Due to genetic selection, the backfat thickness of gilts at the time of breeding has been reduced considerably in recent years (Aherne and Kirkwood, 1985). Thus, modern sows begin their reproductive life with less body fat reserves over a reproductive cycle. If the nutrients for milk production are not met by diet then the sow must use her own body reserves to meet any shortage.

The relationship between the sow's digestible energy intake and milk production was demonstrated by King and Dunkin (1986a) by restricting the feed intake of first-litter sows during lactation. Milk production, as indicated by the growth rate of piglets, was similar for the first 21 d of lactation regardless of treatment. The restricted fed sows catabolized their

own tissue to meet the dietary shortfall and hence lost body weight. The results of King and Dunkin (1986a) indicated that the growth rate of piglets prior to weaning was affected by feed intake of sows during lactation, and that much of this effect on piglet growth rate was expressed during the last week of lactation. As lactation is extended so the energy intake of the sow increases and, given sufficient time the sow may begin to replenish body reserves prior to weaning. Replenishing of body reserves by the sow can also be achieved by split weaning (Edwards et al., 1985). Split weaning involves removal from the sow of half the litter, the bigger piglets, several days before smaller littermates.

A major component of the weight loss during lactation by first-litter sows can be accounted for by the loss of body lipid. Kotarbinska (1983) slaughtered first-litter sows at weaning after either ad libitum or restricted (2.4 kg d^{-1}) feeding during lactation. First-litter sows fed to appetite lost about 20 kg of carcass weight of which about one-half was lipid. The loss of lipid accounted for approximately 200 g kg^{-1} of the fat reserves present at farrowing. Most of the water lost could be accounted for by the loss of protein from lean because protein and water are usually associated together in the proportion 1:3.4 (Zoiopoulos et al., 1983) because of the chemical composition of muscular and other tissues.

Whittemore et al. (1980) studied the live weight and backfat changes of sows in several commercial herds and found that while there was a maternal weight gain from mating (parity 1) to weaning (parity 2) of 22 kg there was a concurrent loss of about 8 kg of fat tissue (8 kg of total fat is equivalent to 7.4 mm backfat). There was an indication that loss of fat did not only occur during lactation, but mobilization commenced in late pregnancy as indicated previously by Lodge (1972) and recently by Robinson (1986).

The extent to which energy reserves of the sow can be mobilized to support milk production can be demonstrated by calculations of energy balance. Sows fed 59.4 MJ ME d^{-1} partitioned a total of 31 MJ ME d^{-1} into milk production, of which 10% was derived from body fat reserves (Noblet and Etienne, 1986). Similarly animals receiving 43.5 MJ ME d^{-1} produced approximately the same energy per day per milk production (28 MJ ME d^{-1}) but 17 MJ or 60 per cent of this was produced from maternal fat. In a study by Kotarbinska (1983),

the contribution of body reserves to milk production can be estimated by assuming that the energy content of milk was similar for both treatments (5.2 MJ kg^{-1}) and that body reserves were used for milk production with an efficiency of 85% (Noblet and Etienne, 1987), and by calculating the change in the gross energy of the carcass from farrowing to weaning (lipid 39.7 MJ kg^{-1}, protein 23.8 MJ kg^{-1}) (ARC, 1981). Whereas the sows fed ad libitum produced about 25% of their milk from body reserves (equivalent to 10 MJ milk energy), the restricted sows mobilized sufficient energy reserves to account for almost all of the milk produced (30 MJ milk energy).

Shields et al. (1985) fed two gestation protein levels (5 or 14%) and three dietary lactation protein levels (5, 14 or 23%) to first parity sows. The growth of piglets, and hence milk production, was maximized by the feeding of a high protein diet during both pregnancy and lactation. These animals were in a zero nitrogen balance between day 7 and day 28 of lactation but did mobilized some fat reserves probably to supplement the low feed intake. However, details of the energy value for the diet were not given. Milk production by both groups receiving the low protein diet during lactation was depressed despite the mobilization of body protein.

The relationship between backfat thickness and body composition in gilts and sows

Backfat depth measured ultrasonically is a useful indicator of the fat content of live pigs and has been used successfully with animals of commercial weights. Recently there has been interest in using the same approach to monitor fat changes in sows through the reproductive cycle (Davies et al., 1985; Esbenshade et al., 1986; Eastham et al., 1988).

Prediction equations relating backfat depth to total body fat for sows have been determined (Whittemore et al., 1980; Shields et al., 1985; King et al., 1986). Whittemore et al. (1980) used sows slaughtered 10 d after weaning their second litter. These sows had lost 5.9 mm of backfat and an estimated 6.4 kg of fat tissue during their second lactation and since catabolism of body fat can continue after weaning (Brooks, 1982) these animals may not have commenced to replinsh their body fat reserves before slaughter.

Davies et al. (1985) using ultrasonic measurements indicated that for a population of sows fed a single level of feed, backfat depth will vary between individuals and that neither sow backfat at a given stage of production nor sow backfat changes during pregnancy will give an indication of sow productivity. Esbenshade et al. (1986) used visual appraisal for body condition scores and also failed to find a relationship between backfat measurements at various times during the reproductive cycle and subsequent productivity. This may be due to inaccuracy in prediction equations. Despite the shortcomings of prediction equations, the fact that carcass dissection or chemical composition methods require the slaughter of the sow and are costly, time consuming, and only one assesment of body composition can be made on each animal, prediction equations may be, the most practical alternative to carcass dissection in aiding the formulation of feeding stragies for the manipulation of body lipid reserves on a total reproductive cycle basis.

D. ENDOCRINE CONTROL OF ENERGY METABOLISM

Regulation of energy metabolism is primarily aimed at maintaining a constant level of glucose in the blood. Differences in energy intake essentially lead to either storage of excess nutrients or mobilization of body stores to provide for glucose synthesis. The regulation of storage, mobilization and interconversions of feedstuffs occur mostly in three tissues, liver, muscle and adipose tissue (Unger, 1981). The liver is the principal glucose producer by mobilization and interconversions by processes of glycogenesis, lipogenesis, and gluconeogenesis (Porte and Woods, 1981). During lactation the mammary gland is the principal consumer tissue and thus highly glycolytic. Adipose tissue stores free fatty acids (FFA) or fatty acids from lipoprotein as triglycerides and is the tissue in which lipogenesis and lipolysis mostly occur.

These processes of metabolism are regulated by a host of hormones with insulin and growth hormone as the regulators of energy anabolism (Unger, 1981). Other hormones involved in the regulation of energy catabolism include glucagon, growth hormone, glucocorticoids, the thyroid hormones and the catecholamines (Trenkle, 1978).

Insulin

Insulin, a polypeptide of 51 amino acid residues, is secreted by the beta cells of the islets of Langerhans of the pancreas. Insulin stimulates the uptake and utilization of glucose by peripheral tissues, inhibits gluconeogenesis and glucose release from the liver, stimulates the uptake and incorporation of amino acids into protein, inhibits proteolysis, stimulates lipogenesis and also reduces FFA levels by inhibiting lipolysis of triglycerides to form fatty acids (Straus, 1984). In general, insulin is hypoglycemic and promotes deposition of metabolites in peripheral tissues (Brockman and Laarveld, 1986). There are indications that insulin is very sensitive to changes in body glucose levels whether they occur with exercise or starvation (Warren, 1983). Indeed, in the absence of insulin, muscle wasting occurs due to excess mobilization of proteins (Bentley, 1982). Plasma concentrations of insulin have been shown to be depressed during dietary restriction in humans (Warren, 1983), sows (Lythgoe, 1987) and cows (Harrison and Randel, 1986). On the other hand, administration of insulin to lactating cows (Kronfeldt et al., 1963) and sows (Goldobin, 1976) resulted in decreased milk yield. Therefore, in contrast to the effects on nutrient uptake of peripheral tissues, insulin has no influence on glucose uptake by the mammary gland (Hove, 1978). However, Spencer (1985) noted that during energy deficit, plasma insulin levels are low, thus reducing nutrient deposition into tissues and allows the body to use the available energy for its immediate needs for survival.

Plasma insulin concentrations change rapidly (Woods and Porte, 1977). However, there is an indication that cerebrospinal fluid (CSF) insulin does not change nearly as rapidly as plasma insulin (Porte and Woods, 1981). Hence, CSF insulin concentration may have access to brain tissue and its dynamics may fulfill the criteria for a signal which would not change rapidly, and would represent body adiposity. Insulin in this state may play a role in the maintenance of body weight by action in the brain.

Acting at the brain, insulin influences the availability of subtrates that are essential for the synthesis of neurotransmitter systems in the brain, particularly that of norepinephrine and serotonin (Hashimoto and Kimura, 1987) both of which are thought to interact with

GnRH secretory mechanisms (Barraclough and Wise, 1982).

The effects of feed intake during lactation on plasma concentration of insulin, glucagon, growth hormone and cortisol have only partially been examined in the lactating sow. Armstrong et al. (1986) found no effect of restricted feeding on plasma insulin concentration in lactating sows. However, in this study plasma insulin concentrations were determined on the basis of a single sample taken on days 6, 12 and 20 of lactation, thus the true differences may have been missed. In another study, Lythgoe (1987) showed differences in plasma insulin concentration during lactation. Insulin concentration alone will not help in explaining the mechanism involved in energy metabolism during lactation in the sow because carbohydrate, protein and lipid metabolism are controlled by different aspects of the endocrine system.

During lactation lipogenesis tends to be suppressed in adipocytes by a reduction in the number and sensitivity of insulin receptors, by a depression in the activity of lipoprotein lipase and fatty acid synthetase, and by a reduction in the rate of fatty acid reesterification (Vernon and Flint, 1984).

Insulin has been shown to be necessary for reproductive cycling in some studies (Siegel and Wade, 1979; Kirchick et al., 1982; Cox et al., 1987). Ovulation rates in gilts have been demonstrated to be increased by exogenous insulin (Cox et al., 1987), although the increase in ovulation rate was not associated with changes in gonadotropins or estradiol-17β. The influence of insulin on ovulation rate was related to dietary energy, because the greatest increase in ovulation rate was in the gilts fed the high energy diet (Cox et al., 1987). Thus, it could be that the positive effects of insulin on ovulation rate are secondary to effects on body fat stores or body weight changes. Insulin has also been shown to enhance the stimulatory effects of FSH and LH on swine granulosa cells (Veldhuis et al., 1983). This may suggest that insulin acts directly at the ovary, rather than through the hypothalamic-pituitary-ovarian axis.

Glucagon

Glucagon, a polypeptide of 29 amino acid residues, is secreted by the alpha cells of the islets of Langerhans of the pancreas. Glucagon stimulates hepatic glucose output by accelerating hepatic glycogenolysis and gluconeogenesis (Unger and Dobbs, 1978). The stimulation of glucose output by the liver is a prominent action of glucagon and is, perhaps, the only one of physiological importance in most mammalian species. Glucose output is increased by co-ordinated effects of four processes as outlined by Unger (1981). The processes which may promote glucose production, include the activation of glycogenolysis, inhibition of glycogen synthesis, stimulation of gluconeogenesis, and adaptive synthesis of some enzymes, such as liver phosphorylase. The stimulation of net glucose production by glucagon would be aided by co-ordinating the activation of glycogenolysis with a suppression of glycogen synthesis (Gerich et al., 1976). Glucagon also influences amino acids, inhibiting protein synthesis and enhancing glucose production from amino acids (Pilkis et al., 1978). Glucagon, according to Unger (1981), is also a potent stimulus of lipolysis in adipose tissue and thus there is release of FFA and glycerol for utilization by the liver and other tissues.

Plasma glucagon concentration in sows was shown to decrease at weaning while plasma insulin levels were increasing (Eriksson et al., 1987). This decrease in glucagon and increase in insulin could be due to the fact that nutrients meant to be incorporated into milk, pile up in the circulation until the maternal metabolism is adjusted to the non-lactating state.

Growth Hormone

Growth hormone (somatotropin), a polypeptide of 191 amino acid residues, is secreted by cells of the adenohypophysis. Growth hormone, essential for the growth of the animal, increases the rate of protein synthesis, increases fat mobilization for energy utilization and decreases carbohydrate utilization by increasing lipolysis (Goodman and Grichting, 1983), and decreasing lipogenesis by adipose tissue (Rudas and Scanes, 1983). The lipolytic effect of growth hormone is permissive by acting as a modulator or amplifier of physiological stimuli for lipid mobilization rather than as the initiator of lipolysis (Fain et al., 1965). Plasma

concentrations of growth hormone are markedly affected by various nutritional deficiencies. For example, plasma concentrations of growth hormone in chickens are elevated by starvation (Harvey et al., 1978) and chronic restriction of food intake (Engster et al., 1979). There is evidence that the increase in plasma concentration of growth hormone in feed restricted animals is due to a deficiency of protein. Scanes and Balthazart (1981) observed that chicks maintained on a 5% protein diet available ad libitum had elevated levels of growth hormone compared to chicks with free access to an isocaloric 20% protein diet. Thus it would appear that it is protein and not caloric intake that is responsible for changes in circulating growth hormone concentrations. It is unlikely that protein deprivation directly affects the synthesis of growth hormone owing to a restriction of the precursor pool of amino acids. Indeed, it may be presumed that the increase in plasma concentrations of growth hormone reflects increased synthesis and release. Furthermore, the effects of protein restriction on plasma concentrations of growth hormone may be due to deficiencies of dietary amino acids.

Growth hormone has also been shown to result in marked increases (25 - 40%) in milk production in high producing cows (Bauman et al., 1985). It plays a co-ordinating role by chronically altering metabolism so that more nutrients are partitioned to "high priority" processes like milk synthesis and growth (Peel et al., 1981). Basset (1972) showed that growth hormone concentrations decrease shortly after feeding and increase with time after feeding. Low concentration of energy metabolites have been shown to stimulate growth hormone secretion (Hart et al., 1979).

Glucocorticoids

The glucocorticoids are produced in the zona fasciculata and zona reticularis of the adrenal cortex. These portions of the gland depend on adrenocorticotropic hormone (ACTH) for viability and also for steroid production (Baird et al., 1969). Although the adrenal cortex secretes several classes of steroids, such as cortisol, corticosterone, cortisone, 11-deoxycortisol, progesterone, and aldosterone, cortisol is viewed as the most important adrenal steroid in stress response, in all vertebrates (Moderg, 1987). A major action of cortisol is to induce

protein catabolism in peripheral tissues. Cortisol stimulates catabolism of extrahepatic tissues and stimulate gluconeogenesis in the liver using the muscle and other peripheral tissues as sources of amino acids. The amino acids are transported to the liver where aminotransferases and other enzymes convert them to keto acids such as pyruvate, or α-oxoglutarate (Weber et al., 1966). These keto acids may be converted to oxaloacetate and thence to glucose via phosphoenolpyruvate and then ultimately to glucose. In adipose tissue, cortisol stimulates lipolysis and promotes the lipolytic effect of adrenalin and noradrenaline. Cortisol also increases the production of glycerol, a precursor of glucose carbon, and FFAs (Weber et al., 1966). Thus, gluconeogenesis is encouraged at the expense of glucose breakdown and utilization. In the absence of hepatic glycogen stores, the FFAs and glycerol provide metabolic substrates necessary for survival. Since plasma cortisol concentration is elevated during starvation, it is important in maintaining glucose levels during starvation.

High concentration of cortisol in plasma has been shown to depress reproductive performance. The administration of hydrocortisone acetate to castrated rhesus monkey by Dubey and Plant (1985), resulted in a correlation between elevated serum cortisol and the suppression of circulating levels of luteinising hormone (LH) and follicle stimulating hormone (FSH). Restraining cows suppressed the pulsatile secretion of LH in those animals which had a marked increase in plasma cortisol (Dobson, 1987). Furthermore, low plane feeding, at body maintenance level, has been reported to decrease basal plasma LH concentrations in humans (Warren, 1983), cattle (Rutter and Randel, 1984) and sheep (Rhind and McNeilly, 1986). These findings suggest that restricted feed intake, and the consequent stimulation of glucocorticoid secretion may play a role in suppressing gonadotropin releasing hormone (GnRH) secretion and/or block the pituitary's response to GnRH, lowering LH synthesis and secretion.

The Thyroid Hormones

Thyroid stimulating hormone regulates differentiated functions of thyroid cells. These include uptake of iodine, thyroglobulin biosynthesis, iodination of thyroglobulin to form the

thyroid hormones (thyroxine, T_4, and triiodothyronine, T_3) and the release of thyroid hormones into the blood stream (Forsling and Grossman, 1986). Most T_3 is formed by peripheral deiodination of T_4 in the circulation. The major effect of thyroid hormones is to increase the metabolic activities of the body, by increasing substrate availability and increasing glucose and free fatty acid uptake by cells. This effect is accomplished by increasing glycogenolysis, stimulating lipolysis, and increasing rate of absorption of glucose from the gastrointestinal tract (Brown et al., 1981). In mammals, the thyroid hormones cause an increase in nitrogen excretion, mostly in the form of urea and some creatine. Creatine and phosphocreatine are important in the energy exchange cycle in muscle function (Bentley, 1982). Thus, high levels of thyroxine in plasma of mammals causes wastage, weakness and fatigue of muscles (Hadley, 1987). In addition, hyperglycemia is produced by hyperthyroidism. In fact, thyroid hormones increase the supply of glucose for oxidation by their catabolic action on liver glycogen and by enhancing glucose synthesis in the liver (Edelman, 1974). Augmented glucose production may also be promoted by an increased availability of glycerol as a result of the accelerated lipolysis of the hyperthyroid state. In fact, a major effect of thyroid hormone on lipid metabolism is to enhance mobilization of FFAs from adipose tissue and to increase FFA oxidation (Klauser and Heinberg, 1967). Other studies have also shown that fat mobilization by glucagon, ACTH and growth hormone is also facilitated by thyroid hormones (Caldwell and Fain, 1971). In hyperthyroid patients and animals, Eisentein and Singh (1978) indicated that the respiratory quotient is lower than in normal subjects, indicating preferential oxidation of fat. Thus thyroxine's enhancement of fat catabolism outweighs its influence on fat synthesis.

The metabolic state of the sow at weaning may be important in determining the sows ability to return to estrus. Recent reports indicate that sows that were fed diets restricted in energy (Nelssen et al., 1985) and protein (Brendemuhl et al., 1987) during lactation and that did not return to estrus had lower concentration of serum T_4 and T_3 postweaning than sows that were fed diets restricted in energy and that did return to estrus.

Catecholamines

Catecholamines are derivatives of catechol, which, in turn, is a derivative of the amino acids phenylalanine and tyrosine (Weiner 1970). The catecholamines are synthesized by adrenal medullary and brain cells. The three main catecholamines are dopamine, norepinephrine and epinephrine. In most mammals epinephrine is the primary adrenal medullary hormone and is more able to elicit a generalised response than is norepinephrine; that is epinephrine is capable of activating all adrenergic receptors that are accessible from the blood stream (Tepperman, 1980). Epinephrine causes hyperglyceamia by several mechanisms including stimulation of hepatic gluconeogenesis from lactate produced in skeletal muscle, inhibition of insulin release, stimulation of glucagon release, and stimulation of ACTH release and consequently, of glucocorticoids by the adrenal cortex (Axelrod and Weinshilboum, 1972). Thus epinephrine and norepinephrine are potent lipolytic agents which induce a potent rise in plasma FFA. However, stimulation of lipogenesis is of lesser magnitude than that of lipid mobilization (Tepperman, 1986).

Glycerol

The rate of lipolysis in the lactating sow has not been studied extensively using glycerol as an indicator of body fat loss. Glycerol is a product of the metabolism of adipose tissue and since this tissue lacks the activating enzyme glycerokinase, adipose tissue cannot reuse glycerol for the synthesis of triglyceride. Thus the determination of the plasma concentrations of glycerol at the begining and end of lactation of sows fed different levels of feed, may enhance the understanding of the extent of lipid mobilization.

E. PHYSIOLOGY OF THE PIG ESTROUS CYCLE

The modern domestic pig does not have a specific breeding season. It is fertile throughout the year, and shows regular estrous cycles, with a duration of approximately 21 days (range 19 - 23 days) (Anderson, 1987). These cycles commence immediately following the attainment of puberty and continue throughout the female's life, interrupted only by

pregnancy and lactation.

Studies in the late prepubertal gilt, cyclic gilt and the lactating and weaned sow suggest that except in the follicular phase, continuous development and subsequent atresia maintains a proliferating pool of follicles of 1 to 6 mm in diameter (Foxcroft and Hunter, 1985). Estimates of the size of this pool vary, but Anderson (1987) suggested that the proliferating pool contained approximately 50 follicles between 2 and 5 mm in diameter. It has also been reported however, that the number of follicles within a specified size range was greater in breeds with high ovulation rates (Clark et al., 1973) and could therefore be a potential determinant of eventual ovulation rate and litter size, as suggested in some breeds of sheep (Lahlou-Kassi and Mariana, 1984).

Given an appropriate stimulus, recruitment occurs from the proliferating pool, which establishes a group of selected follicles destined to ovulate. The time at which this selection (or recruitment) occurs has been extensively studied in the cyclic gilt and data from experiments involving treatment with exogenous gonadotropins (Hunter et al., 1976), electrocautery of follicles (Clark et al., 1979) and unilateral ovariectomy (Coleman and Dailey, 1979) suggest that recruitment occurs days 14 and 16 of the estrous cycle.

Onset of estrus in the pig is characterized by gradual changes in behavioral patterns such as restlessness, mounting of other animals, vulva responses (swelling, pink red coloring) and occasionally a mucous discharge (Hughes and Varley, 1980). Sexual receptivity lasts an average of 40 to 60 h. Breed, seasonal variation (longest estrus in summer and shortest in winter) and endocrine abnormalities affect the duration of heat. Ova are released 38 to 42 h after the onset of estrus, and duration of this ovulatory process requires about 3.8 h, with ovulations occurring 4 h earlier in mated than in unmated animals (Hughes and Varley, 1980).

Pattern of pituitary gonadotropins and ovarian steroids

Time-related changes in plasma concentrations of LH, FSH, prolactin and estradiol-17β have been described in the cyclic domestic pig by several authors (Niswender et

al., 1970; Rayford et al., 1971, 1974; Aherne et al., 1976; Parvizi et al., 1976; Van de Wiel et al., 1981; Ziecik et al., 1981; Foxcroft and Van de Wiel, 1982; Tilton et al., 1982; Prunier et al., 1987).

During the pre-surge period, the follicular phase of the estrous cycle, estradiol-17β rises slowly at first, then rapidly, reaching maximum concentrations a day before the LH peak (Edwards and Foxcroft, 1983a). Luteinizing hormone concentrations increase in peripheral blood during this pre-surge phase of the estrous cycle, apparently due to decrease in progesterone concentration (Edwards, 1982). Increased LH concentrations during this period are characterized by increased frequency and decreased amplitude of pulsatile LH release in sows (van de Wiel et al., 1981). These pulses of LH may stimulate estradiol-17β secretion from the preovulatory follicles. The increase in estradiol-17β that occurs during the preovulatory period is clearly the stimulus that triggers the gonadotropin surge since exogenous estradiol-17β induces a preovulatory-like surge of LH in gilts (Elsaesser and Parvizi, 1979) and sows (Edwards and Foxcroft, 1983b).

The mechanism by which estradiol-17β induces the gonadotropin surge is not completely understood, but Hansel and Convey (1983) suggest that estradiol-17β acts to increase the capacity of pituitary gonadotrophs to release LH and FSH in response to GnRH. Estradiol-17β also increases GnRH self-priming, which is the process by which GnRH increases the capacity of the pituitary to respond to subsequent exposure to GnRH. Estradiol-17β also acts to set a timed mechanism in the hypothalamus which culminates in a surge release of GnRH that induces the gonadotropin surge. The capacity of the pituitary gland to release LH in response to GnRH is greatest during estrus and least during the luteal phase of the estrous cycle. Both increased GnRH secretion and increased pituitary responsiveness are necessary for the preovulatory LH and FSH surges (Kesner and Convey, 1982).

During the post-surge period, there is a marked decrease in estradiol-17β concentration in the peripheral blood (Foxcroft and van de Wiel, 1982). Approximately 24 h after the preovulatory gonadotropin surge, serum concentration of FSH increases slightly in

sows (van de Wiel et al., 1981). This increase in FSH may play a role in recruitment of pre-antral follicles, since Cahill et al. (1981) found a high correlation between the magnitude of this FSH peak and a number of antral follicles present 17 d later. Peripheral plasma levels of LH drop to low levels during the remainder of the estrous cycle (van de Wiel et al., 1981).

Steroidogenesis

The follicle is the ovarian compartment that enables the ovary to fulfill its dual function of gametogenesis and steroidogenesis. In the follicle the layer surrounding the germ cell is referred to as the granulosa layer, while the stroma cells immediately adjacent to this are the theca interna (Hafez, 1987). As proliferation takes place, fluid accumulates between the granulosa cells and forms a small cavity containing the follicular fluid (Foxcroft et al., 1987). During maturation of the follicle, the theca interna cells enlarge and are extensively vascularized, in contrast to the granulosa cells, which have no direct blood supply before ovulation (Hafez, 1987). Following ovulation the follicle collapses, and the granulosa and theca interna cells rapidly proliferate and at the same time become intensely vascularized. The ruptured follicle is transformed into the corpus luteum (Smith, 1986).

The two-cell systems of steroidogenesis is operative in swine (Evans et al., 1981). The granulosa cells are the predominant site of progesterone synthesis and are responsive to LH, FSH (Evans et al., 1981; Stoklosowa et al., 1982) and prolactin (Veldhuis et al., 1980). Although pig granulosa cells lack the 17α-hydroxylase enzymes necessary to synthesize androgens from progesterone or pregnenolone (Bjersing, 1967), substantial androgen synthesis by the thecal tissue of the pig follicles has been demonstrated (Tsang et al., 1979; Evans et al., 1981; Stoklosowa et al., 1982). The major androgen produced by the theca tissue is androstenedione (Evans et al., 1981) which, after transfer to the granulosa, is converted to testosterone and then aromatized to estradiol-17β.

F. HORMONES DURING AND AFTER LACTATION IN SOWS

Prolactin secretion during lactation

Prolactin plays a decisive role in the preparation and maintenance of the mammary gland for milk secretion during lactation. High levels of prolactin appear to be necessary for the initiation of lactation, but once mammary gland enzyme systems have become adapted to secrete milk, lactation can continue with lower prolactin concentrations (Hadley, 1986). Prolactin also increases the number of insulin receptors in the mammary gland and decreases them in maternal fat, which may lead to a preferential deposition of, for example, fat and glucose in the mammary gland (Flint, 1982). There are indications (Bevers et al., 1978; Stevenson et al., 1981) that nursing sows produce high levels of prolactin which is associated with anestrus in sows as a consequence of reduced secretion of the gonadotropins, especially LH (Parvizi et al., 1976; Stevenson and Britt, 1980; Stevenson et al., 1981; Mattioli et al., 1986). Among the different factors responsible for changes in LH secretory patterns, the suckling stimulus seems to be the most important (Edwards, 1982). As the response to GnRH is unimpaired (Bevers et al., 1981; Forsling and Grossman, 1986; Kirkwood et al., 1987; Rojanasthien et al., 1987) during lactation, the suckling stimulus appear to directly inhibit GnRH synthesis and secretion and by the same processes suppresses prolactin inhibiting hormone allowing the synthesis and secretion of prolactin by the anterior pituitary (Ben-Jonathan, 1985). Indeed an inhibition of hypothalamic GnRH, has been demonstrated in the sow (Cox and Britt, 1982b). High prolactin levels also have an inhibitory effect on the ovaries, which appear to be less responsive to FSH under these conditions (Forsling and Grossman, 1986).

Secretion of gonadotropins during lactation

The two gonadotropic hormones secreted by the anterior pituitary, FSH and LH, are themselves under hypothalamic control via the releasing hormone, GnRH. The data available on pituitary FSH and LH concentrations indicate that pituitary FSH is high, with little

evidence of change throughout lactation (Stevenson and Britt, 1980; Stevenson et al., 1981; Britt et al., 1985). On the other hand, there is a sharp drop in LH activity between the end of pregnancy and day 14 of lactation (Melampy et al., 1966). Stevenson et al. (1981) suggested that suckling was responsible for suppressing LH secretion, presumably by reducing the frequency of secretion of GnRH and/or gonadotropins which favor follicular growth as demonstrated by Cox and Britt (1982b). However, there is an indication that both serum LH and FSH concentration increased beyond the 3rd week of lactation in intact and ovariectomized sows and the gradual increase in gonatropins during lactation may be related to a natural reduction in suckling frequency of the litter (Stevenson et al., 1981). Whether this elevated secretion of FSH serves any physiological function is unclear at present but it appears to reflect the changes in ovarian follicular development seen as lactation progresses. Stevenson et al. (1981) suggested that FSH secretion during lactation may be controlled by inhibin which inhibits FSH release early in lactation. Inhibin, is a protein hormone produced by Sertoli cells in the male and granulosa cells in the female (Campbell and Schwarz, 1979; Franchimont et al., 1979). The biological activity of inhibin is neither species nor sex specific as preparations of different origins from both sexes reduce FSH secretion in a parallel fashion (Franchimont et al., 1979). As lactation progresses, there is an indication that the secretion of this FSH inhibitor (inhibin) may serve only to limit excess, but not apparently normal, FSH secretion (Stevenson et al., 1981).

Endocrinology of the sow at weaning

Weaning results in dramatic changes in hormone secretion and these changes seem to be influenced by duration of lactation. There are indications that FSH concentrations generally increase gradually after weaning (Cox and Britt, 1982a; Edwards and Foxcroft, 1983a,b; Shaw and Foxcroft, 1985; Foxcroft et al., 1987) and the increase in FSH is concomitant with follicular growth and maturation (Britt et al., 1985). In contrast, pituitary LH levels rise significantly at weaning (Cox and Britt, 1982a; Edwards and Foxcroft, 1983a; Shaw and Foxcroft, 1985; Foxcroft et al., 1987) and there is an increase in the frequency of

episodic pulses of LH over the next 2 - 3 days (Cox and Britt, 1982c; Shaw and Foxcroft, 1985). The increase in LH secretion is associated with similar increases in hypothalamic GnRH content (Cox and Britt, 1982b) within 60 h of weaning thereby increasing the potential for LH synthesis by the pituitary and thus for a sustained LH secretion which occurs at estrus.

Endocrine changes at the postweaning estrus

The underlying endocrine characteristics necessary for the resumption of estrus activity in weaned sows are still unclear. However, Edwards and Foxcroft (1983a,b) have indicated that two to six days after removal of the sow from her litter, plasma estradiol-17β begin to rise and remain elevated for 2 - 3 days prior to, and during part of the estrus period. This increase in estradiol-17β from developing follicles suppresses increased concentrations of gonadotropins within 60 h after weaning (Stevenson et al., 1981; Cox and Britt, 1982b; Edwards and Foxcroft, 1983a). After this period of suppression of gonadotropins by estradiol-17β, a further increase in estradiol-17β secretion triggers estrus and the preovulatory gonadotropin surges (Stevenson et al., 1981). The secretion of estradiol-17β is rapidly terminated by the preovulatory surge of LH (Edwards and Foxcroft, 1983a), an observation which is consistent with the reported ability of LH to terminate estradiol-17β secretion by cultured sheep Graafian follicles in vitro (Moor, 1974).

The preovulatory surge of LH is directly responsible for ovulation and any changes in its characteristics, particularly in relation to the length of lactation or the effect of nutrition could have a major influence on the postweaning reproductive performance of sows. A shorter lactation length ($<$ 21 days) significantly depressed LH in terms of mean LH and total LH secreted (Edwards and Foxcroft, 1983a; Kirkwood et al., 1984). This effect of length of lactation on LH concentration is similar to results obtained from starved humans (Warren, 1983) and sheep (Rhind et al., 1987). However, the effect of nutrition on the concentration of the gonadotropins during the postweaning period has not been reported for sows. Furthermore, the physiological basis for the decline in LH concentration due to length of

lactation or nutrition is not clear.

The results of Gauthier et al. (1983) with postpartum cows indicated that nutrition had a direct effect on hypothalamic function, and that nutritional deprivation during lactation reduced LH and FSH secretion and delayed postpartum estrus. Recently, Wright et al. (1987) suggested that the effect of body condition on the duration of the postpartum anestrus period is mediated through effects on LH pulse frequency. A similar relationship probably exists in the sow (Shaw and Foxcroft, 1985) in that mean LH levels during the 12 h period before weaning are inversely related to the interval from weaning to estrus.

G. CONCLUSION

The possible mode of action of sow body weight loss during lactation or during the postweaning period on hypothalamic function requires investigation. A definite characteristic of the metabolism of the lactating sow is the maintenance of milk production, even under conditions of low feed intake, by depletion of body tissues. Under conditions of dietary insufficiency, muscle protein synthesis is depressed and amino acids released from muscle degradation are channelled into milk protein production. Under conditions of severe protein deprivation, the supply of amino acids from skeletal muscle will not be sufficient for milk protein and milk production will be depressed. Although GnRH and the gonadotrophic hormones themselves are peptides containing amino acids, the amino acid requirement for hormone synthesis is very small and the effects of protein inadequacy on gonadotropin release will be mediated by pathways involving other metabolites. The effect of feed intake in lactating and postweaning sows on their endocrine profiles have received minor attention in most studies.

The objectives of the present study were, therefore:

1. To examine the effects of feed intake during lactation on changes in body condition and associated effects on the profiles of glycerol, prolactin, cortisol, growth hormone, insulin, glucagon, FSH and LH.
2. To determine the consequences of restricted feed intake during the weaning-to-estrus

interval on the length of the interval, ovulation rate, conception rate, and percentage embryo survival.

3. To study the estradiol-17β, FSH, and LH profiles of sows on different postweaning nutritional regimens.

A continuous experiment with the same sows from farrowing, lactation, through weaning to 25 days postcoitum was conducted in an attempt to achieve the above objectives.

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II. THE INFLUENCE OF LEVEL OF FEED INTAKE DURING LACTATION ON SOW AND LITTER PERFORMANCE AND SOW PLASMA GLYCEROL CONCENTRATION

A. INTRODUCTION

Sow productivity is frequently measured in terms of pigs weaned per sow per year and is influenced by litter size, time to postweaning estrus and conception rate. To maintain sow productivity therefore, the non-productive sows are culled. Reproductive failure represents nearly half of the reasons for culling sows (Dagorn and Aumaitre, 1979; Friendship et al., 1986). Of the several factors known to influence reproductive failure, the interval from weaning to estrus is the most common.

Feed intake, protein and (or) energy intake during lactation have been shown to influence the weaning to estrus interval (Reese et al., 1982a,b; King and Williams, 1984a,b; King and Duncan, 1986a,b; Lythgoe, 1987). Failure to meet the sows nutrient requirements during lactation results in sow weight loss and mobilization of body reserves (Whittemore et al., 1980; Aherne and Kirkwood, 1985; Mullan, 1987). It has been frequently demonstrated in recent years that excessive weight loss in lactation significantly influenced weaning to estrus interval (King and Williams, 1984a), number of sows returning to estrus (King and Dunkin, 1986a; Kirkwood et al., 1987), pregnancy rate and embryonic mortality (Hughes et al., 1984). However, other experiments have reported sow weight losses during lactation of in excess of 30 kg with no effect on the weaning to estrus interval (Eastham et al., 1988; Kirkwood et al., 1988). It appears that the sow can monitor her own protein and fat status and has a minimum threshold of both tissues below which reproductive function will be impaired. There is little agreement in the literature at present as to what that threshold is. Indeed there is still debate as to whether it is fat loss or protein loss that is most important in influencing the frequently observed reproductive problems. In the past, body weight loss during lactation was usually assumed to correspond mainly to loss of body fat. These assumptions were based on nitrogen balance data and changes in backfat thickness (Whittemore et al., 1980; Reese et al., 1982b). However, recent experiments based on comparative slaughter techniques have shown that both

muscle and adipose tissues are involved (Shields et al., 1985; King et al., 1986; Mullan, 1987). Although carcass dissection or chemical analyses are the most accurate physical means of determining carcass composition, they are costly and time consuming. Indirect methods, such as empirical equations may be used successfully in estimating body composition of sows by using backfat measurements or liveweights or both (Whittemore et al., 1980; King et al., 1986). In a recent study, King et al. (1986) demonstrated that the absolute weight of body fat could be accurately predicted from multiple regression equations combining live weight of the animal and the depth of backfat measured ultrasonically.

The concentration of glycerol in plasma reflects lipolysis in adipose tissue (Tepperman, 1980) and thus can provide an approximate index of fat mobilization. Therefore, determination of glycerol in plasma may be a very useful tool in determining any diet induced changes in fat mobilization.

In view of the significance of the events in lactation with regard to feed intake during lactation and weight loss, the present study was undertaken to examine the dynamics of weight and backfat loss in lactation, and the consequences of backfat and (or) weight loss during lactation on sow body condition and litter performance, the length of the remating interval, the percentage of sows in estrus by 5, 10, 15, 20 or 25 d postweaning, ovulation rate, pregnancy rate and embryo survival.

B. MATERIALS AND METHODS

Animal handling

The experiment was conducted at the Lacombe Research Station, Lacombe, Alberta. Eighty-nine second parity Lacombe sows were offered 2 kg d^{-1} of a barley - soybean meal diet (Table II.1) throughout gestation. The diet (Table II.1) was formulated to meet or exceed NRC (1979) recommended nutrient levels for gestating and lactating sows. Sows were moved into individual farrowing crates at approximately day 109 of gestation. The eighty-nine sows were randomly allotted on the basis of liveweight at day 109 of gestation to one of two

feeding levels. Sows were fed twice daily and offered a total of 3 kg (L) or 6 kg (H) of feed (Table II.1). The 6 kg feed was chosen as it represented the approximate daily feed intake for sows in the herd. The 3 kg daily feed intake represented a low feed intake that may arise under commercial conditions (Cox et al., 1983; King et al., 1984). At parturition, number of pigs farrowed alive and dead and individual pig birth weights were recorded. Within 48 h after farrowing, litters were adjusted to eight pigs to minimize litter size variation. Number and weight of individual pigs at farrowing, 14, 21 days postpartum and at weaning (28 days postpartum) were recorded. Water was available to both sow and the litter at all times during the experiment. The temperature of the farrowing unit was maintained at 21±2 °C throughout.

Body weight of sows was individually recorded within 12 h after farrowing, 14, 21, and 28 days postpartum, at breeding and at slaughter. Sow backfat depth (45 mm lateral to the mid line at the last rib) was measured ultrasonically (Kraut Kramer US 47) at 109 days of gestation, within 12 h after farrowing, and 14, 21 and 28 days postpartum. Daily feed consumption and feed refusals were recorded for each sow over the 28 d lactation.

At weaning, the sows were randomly allotted within lactation treatments to either a restricted intake (3 kg, L) or fed to appetite (H). On d 2 postweaning boars were introduced to sows on a daily basis to check for estrus. From d 3 sows were introduced individually to boar pens and the day of return to estrus was defined as the time at which sows first stood to be mated. Sows were bred a second time between 8 and 24 h after first service.

Blood sampling

On d 2 and d 28 of lactation, blood samples from sows fed high (n=8) or restricted (n=8) intakes were taken every 4 h (beginning at 0600) for 24 h from catheters inserted into the vena cava via an ear vein. Catheters were inserted at least 12 h before the first sample was taken. Catheters were flushed with saline (0.9% NaCl) containing 60 IU heparin ml^{-1}. All blood samples were collected into glass tubes containing heparin (10 IU ml^{-1}) and held at 4 °C until plasma was collected. Ten milliliters of heparinized blood was centrifuged at 3000 x g at

4 °C and the plasma stored at - 20 °C until analyzed for glycerol content.

Glycerol Analysis

Plasma samples were analysed for glycerol concentration by the enzymatic method of Boobis and Maughan (1983). To 1 ml of plasma or standard prepared from glycerol (99% pure, Sigma, London Chem. U.K.), was added 500 μl of ice-cold 2 N perchloric acid to deproteinize the sample. The mixture was centrifuged in an Eppendorf centrifuge for 60 s. Duplicate samples of the supernatant were used for the glycerol analysis. 25 μl of 5N potassium carbonate solution (pH 7-8) was added to 500 μl of the supernatant. This mixture was centrifuged at 3000 x g for 60 s. To 100 μl of this supernatant was added 500 μl of buffer (1 ml hydrazine hydrate, 0.95 ml 2-amino-2-methyl propan-1-ol, 37.2 mg EDTA (free acid) in 100 ml deionized water with pH adjusted to 9.9 with HCl), 20 μl β-nicotinamide adenine dinucleotide (NAD) and 20 μl of glycerol dehydrogenase (glycerol:NAD 2-oxidoreductase, EC 1.1.1.6) suspended in ammonium sulphate 20 mg ml^{-1} (BCL, The Boehringer Corporation (London) Ltd, U.K.). This mixture was incubated at room temperature for 60 min. At the end of the incubation period, 2 ml sodium carbonate buffer (168 mg sodium bicarbonate and 212 mg sodium carbonate in 200 ml water) pH 10.0 was added to each tube and the contents thoroughly mixed. Fluorescence was measured using a Fluorometer (Locarte Filter Fluorometer LFM8-9 with mercury arc lamp and quartz optics) (Locarte Co., U.K.). Flurometric readings were taken at 340 excitation and 463 emmission at range 11 and glycerol concentration of the samples were calculated from a standard curve and corrected for dilution. Repeated estimates of the glycerol content of a randomly chosen plasma (n=8) was 3.9±0.6 ng ml^{-1} with an intra-assay coefficient of variation of 9.6%.

Reproductive Tract Dissection

Sows that did not show estrus within d 10 postweaning were monitored for another 25 d before slaughter. All other sows were slaughtered at d 25 post-insemination and the reproductive tracts removed from each sow. The cornua of the uterus were separated after

removing the mesometrium. Each cornus was cut along the mesometrial surface and the tissue separated with a forceps. Days to estrus, ovulation rate (determined by the number of corpora lutea) and the number and viability of embryos were recorded.

Statistical Analysis

Effects of level of feed intake effects on sow weight and backfat changes during lactation were analyzed using analysis of variance using Statistical Package for Social Sciences (SPSS INC. 1983). Estimates of feed intake to maintain weight and backfat during lactation were obtained from regression equations using Minitab Statistical Package (Minitab Inc. 1986). Chi-square analyses (Steel and Torrie, 1980) were used to test treatment effects for the following dependent variables: proportion in estrus, conception rate, ovulation rate and embryonic survival. Plasma glycerol concentrations were tested by split plot analysis of variance for repeated measures. All means reported are least-square means (Steel and Torrie, 1980).

C. RESULTS

Sows allocated to the two feeding levels during lactation did not differ significantly in live weight or backfat thickness at farrowing ($P>0.05$). Average daily feed intake of the sows were 5.5 kg and 3 kg, respectively. There was a steady increase in feed intake for the H sows during lactation (Table II.2). In week 4 of lactation, the H sows consumed ($P<0.01$) twice as much feed as the L sows.

Changes in body weight (Table II.3 and Figure II.1) and backfat (Table II.4 and Figure II.2) were directly related to dietary intake and length of lactation. As expected, a restriction in feed intake resulted in a higher ($P<0.01$) weight loss at d 21 of lactation (12.9% vs 5.3% of body weight at farrowing for L and H fed sows, respectively). Similar trends for weight loss were observed at d 28 postpartum (18% vs 7.4% for the L and H sows, respectively). In terms of backfat, the L fed sows lost more ($P<0.01$) backfat than the H fed sows (26% vs 11%, of the initial backfat at farrowing for L and H sows, respectively).

A 1-kg increase in daily feed allowance during lactation reduced sow live-weight and backfat losses by 0.27 kg and 0.03 mm respectively. These values were based on the following regression equations:

sow live-weight change in lactation (kg d^{-1})

= -2.08 (S.E. 0.06) + 0.269 (S.E. 0.014) feed intake; (RSD = 0.55, R^2 =0.82);

sow backfat change in lactation (mm d^{-1})

= -0.267 (S.E. 0.024) + 0.033 (S.E. 0.013) feed intake; (RSD = 0.344; R^2 =0.681)

Calculations based on these regression equations indicate that it would reguire 7.73 kg feed (1267 grams digestible crude protein and 95.85 MJ digestible energy) daily to prevent sow live-weight loss, but to avoid a loss in backfat the daily feed allowance would need to be 8.1 kg (1328.4 grams and 100.4 MJ digestible energy).

The regression equation reported by King et al. (1986) was used to estimate absolute weight of fat loss in the sow during lactation.

The absolute weight of fat was predicted from multiple regression equations combining LW with linear backfat measurements;

$$Y = -30.1 + 1.258(USP_1 + 0.322(LW) \text{ (RSD = 2.2; } R^2 = 0.965)$$

where Y = body fat (kg); USP_1 = Subcutaneous backfat measured ultrasonically at 45 mm from the last rib; LW = liveweight.

There was no difference ($P>0.05$) in absolute fat content between sows at farrrowing. However, as days in lactation increased, L sows lost significantly ($P<0.01$) more fat than did the H sows (Table II.5). It was estimated that restricted fed sows depleted 21 kg of fat (Table II.5) and 18 kg of lean and bone tissue during the 28 d lactation while the H sows depleted 8.1 kg of fat and 8.1 kg of lean body mass. These results suggest that the weight lost by restricted fed sows was slightly higher in fat than the loss by H fed sows (54% vs 50% for L and H fed sows, respectively).

Energy balances of the sows are presented in Table II.6. The maintenance energy requirement was significantly ($P<0.01$) lower in L fed sows. This was due to the greater weight loss of the L fed sows. Milk production computed from weight gains of pigs by the

methods of Verstegen et al. (1985), was similar (P>0.05) for both H and L sows (Table II.6). The L fed sows mobilized larger amounts of energy than did H fed sows as shown in Table II.6.

The effect of feed intake by sows during lactation on plasma glycerol concentration is presented in Figure II.3. Concentrations of plasma glycerol were not influenced by feed intake on d 2 postpartum. However, on d 28 of lactation, the L fed sows had higher (P<0.01) concentrations of plasma glycerol than the H fed sows. Thus, the rate of lipolysis was higher in the L fed sows than the H fed sows. Despite the loss in sow body condition during lactation, there were no significant (P>0.05) effects on litter size weaned and litter weight at weaning (Table II.7).

Severe sow weight loss in lactation resulted in an extension (P<0.05) of the weaning to estrus interval as presented in Table II.8 . A higher percentage of sows fed the H feed level returned to estrus by d 5 (P<0.01) postweaning compared with sows fed the L feed regimens during lactation (Figure II.4). Lactation feed intake had no effect (P>0.05) on ovulation rate at the postweaning estrus. However, lactation feed intake influenced (P<0.05) pregnancy rate (Table II.8) with the H fed sows having higher pregnancy rate. Embryo survival was influenced (P<0.05) by feed intake. The H fed sows during lactation had higher (P<0.05) embryo survival rate than the L fed sows.

D. DISCUSSION

In this study, the sow feeding levels utilized during a 28-d lactation produced differential sow live weight and backfat losses. Responses to feed level reflect responses to the nutrient components of the feed, especially energy and protein. The average daily energy and protein intake of the low fed sows were 492 gm and 37.2 MJ DE and for the high fed sows 902 gm and 74.4 MJ DE, respectively. These differences in energy and protein intake during lactation did not affect litter size and litter weight at weaning. Similar level of feed restriction for lactating sows (Adam and Shearer, 1975; Reese et al., 1982a) did not influence piglet weaning weights, during a single lactation. However, continued feed restriction for three

parities did reduce weaning weights and fewer pigs were weaned (Lythgoe, 1987).

At parturition, the mammary gland achieves metabolic priority over the other tissues to perform the synthesis and secretion of milk (Bauman and Currie, 1980; Collier et al., 1984). Thus, in terms of energy and protein metabolism, the burden of lactation is far greater than that of any other function in the breeding cycle. Apart from the energy required for maintenance, the sow has to produce milk to support her litter. With such heavy demands on the sow it is not surprising that sow body weight loss in lactation is negatively correlated with total feed intake. Similar conclusions were reported by Reese et al. (1982a,b; 1984), King and Williams (1984a), Noblet and Etienne (1986, 1987) and Lythgoe (1987). Regression relationships indicated that to maintain body weight during lactation, the sow should consume about 7.7 kg of feed and to maintain backfat during lactation, the required feed is 8.1 kg daily. Assuming a similar density of the diet, these results are similar to other estimations by Danielsen and Nielsen (1984) and Eastham et al. (1988). Danielsen and Nielsen (1984) estimated that the lactating sow required 7.07 kg and 7.3 kg of feed to maintain body weight and backfat respectively. Eastham et al. (1988) reported values of 5 kg and 8 kg of feed to maintain body weight and backfat. Thus depending on the body condition of the sow at the begining of lactation, the sow requires about 8 kg feed daily to maintain body weight and backfat. On the other hand if feed intake is below 8 kg then body catabolism occurs.

All sows catabolized body tissues during lactation. However, sows on the low level of feeding lost more backfat and lean body mass than the sows fed the high level of feeding. Using the regression equations of King et al. (1986), it was estimated that the mass of fatty tissue lost over lactation on feeding levels of 3 and 5.5 kg d^{-1} were 21 kg and 8.1 kg respectively, corresponding to sow body weight changes over lactation of -39.0 kg and -16.2 kg. Thus, by difference the estimate of lean tissue loss were 18.0 kg and 8.1 kg for the low fed sows and high fed sows, respectively. Similar weight losses in lactation has been reported in other studies (Kirkwood et al., 1987, 1988; Eastham et al., 1988).

Using the basis that 50.0 MJ DE and 15.0 MJ DE are required for the deposition of 1 kg fatty tissue and 1 kg lean tissue respectively (Whittemore and Esley, 1976) and that the

lactation diet contained 12.4 MJ DE kg^{-1}, tissue losses for the low fed sows would require the provision of 106.4 kg feed for their replacement which together with the 84 kg of feed given over the 28-d lactation, amounted to a total feed usage of 190.4 kg or feed intake of 6.8 kg d^{-1}. Using the same method of calculation, the high fed sows would require 42.4 kg feed for replacement of lost tissue which together with 154 kg of feed given over the 28-d lactation amounted to a total of 196.4 kg or 7.0 kg d^{-1}. Thus with the mobilization of body tissues the low fed sows compensated for lack of nutrients from feed by loosing both fat and lean tissue. The mean total liveweight gains of the litter from birth to weaning for the two lactation treatments were 54.1 kg and 55.4 kg, giving yield per input efficiencies of 0.284 and 0.281 respectively for the lowest and highest lactation feeding level treatments. These calculations indicate that efficiencies of conversion of total feed usage into piglet product were estimated to be similar for the two feeding levels during lactation. Eastham et al. (1988) using the same method of calculation reported yield:input efficiencies of 0.280 and 0.287 respectively for the lowest and highest feeding level treatments.

The higher fat mobilization in the low fed sows was reflected in the high levels of plasma glycerol concentration on d 28 postpartum. Noblet and Etienne (1987) reported that restricted fed sows had a lower respiratory quotient, indicating that fat was used as a source of energy instead of glucose. Furthermore, it has been demonstrated that cows on restricted feeding during lactation mobilize massive amounts of fatty acids from body stores to provide energy for milk synthesis and secretion (Bines and Hart, 1982).

The interval from weaning to estrus was influenced by lactation feed intake. The low fed sows during lactation lost more body weight and had extended remating interval. This is similar to previous studies (Reese et al., 1982a, 1984; King and Williams, 1984a; Kirkwood et al., 1987) and contrary to recent studies (Eastham et al., 1988; Kirkwood et al., 1988). The reasons for these discrepancies is not clear. However, the hormonal status of the sow during lactation and immediately postweaning is discussed in Chapter III of this report and may explain some of the discrepancies. Ninety eight percent of sows fed high during lactation displayed estrus by d 25 after weaning compared to 75% of those fed low levels. Aherne and

Kirkwood (1985) noted that sows having relatively high or low lactation feed intakes had anestrous rates of 1.2% and 20.1% respectively. It is apparent that the normal onset of estrus was delayed in sows having excess body weight and backfat loss during lactation. Studies with other species have also shown the adverse effect of condition loss on the duration of weaning to estrus interval. The rebreeding interval was extended in cattle (Rutter and Randel, 1984; Imakawa et al., 1984) and sheep (Rhind et al., 1986) which have lost condition during lactation.

The results of this study indicate that the weight loss of sows during lactation is due to loss of both adipose tissue and lean body mass. In addition, in the short term, sows given low levels of feed in lactation appear to be as efficient as those receiving high levels of feed in terms of total feed usage relative to pig product. There is an indication from these results that the appropriate feeding strategy in lactation should be one which approaches feeding ad libitum. However, the estimate of about 8 kg feed daily to prevent catabolism of body tissues suggests that some backfat loss during lactation will be unavoidable, since most modern sows will not eat that quantity of feed (Lynch, 1977). Difficulty in consuming 8 kg of feed daily, which is 22% more feed per day than suggested by NRC (1988) for lactating sow, may be due to low energy density or low protein level in the diet and depressed feed intake when environmental temperatures are high. Thus, low feed intake in lactation will result in excessive weight and backfat loss during lactation and will adversely affect sow reproductive performance by extending the remating interval and increasing the rate of embryo mortality.

Table II.1 Formulation and composition of sow diet.

Ingredients	% Diet
Barley	57.5
Wheat	23.4
Soybean meal (46% CP[1])	13.1
Calcium phosphate	1.5
Limestone	1.0
Vitamin-mineral premix[2]	1.0
Iodized salt	0.5
Tallow	2.0
Chemical analyses	
Dry matter (%)	89.3
Crude protein (%)	16.4
Lysine (%)	0.72
Digestible energy ($MJ.kg^{-1}$)[3]	12.4

[1]CP - Crude protein

[2]Supplied the following per kg of diet:120 mg zinc, 12 mg manganese, 150 mg iron, 12 mg copper, 0.1 mg selenium, 5000 IU vitamin A, 500 IU vitamin D_3, 22 IU vitamin E, 12 mg riboflavin, 45 mg niacin, 24 mg calcium pantothenate, 840 mg choline chloride, 30 μg vitamin B_{12}, 200 μg biotin.

[3]Calculated digestible energy.

Table II.2 Average daily feed intake (kg) of sows during lactation.

	High	Low	sed[1]	sig[2]
No. of sows	45	44		
Week				
1	4.80	3.00	0.11	**
2	5.18	3.00	0.10	**
3	5.92	3.00	0.08	**
4	6.00	3.00	0.16	**
MEAN	5.50	3.00	0.07	**

[1] sed = standard error of the difference

[2] High vs Low ** = significant (P<0.01).

Table II.3 Change in body weight during lactation of sows fed different levels of feed.

	Lactation feed level			
	High	Low	sed[1]	sig[2]
No. of sows	45	44		
Liveweight 109 d gestation (kg)	236.5	234.9	3.7	NS
Liveweight after farrowing (kg)	216.9	213.6	6.1	NS
Sow weight at weaning (kg)	200.7	174.6	3.6	**
Sow weight loss 21 d postpartum (kg)	11.5	27.5	1.7	**
Sow weight loss 28 d postpartum (kg)	16.2	39.0	1.8	**

[1] sed = standard error of the difference

[2] ** = significance($P<0.01$); ns = nonsignificant ($P>0.05$)

Table II.4 Influence of feed intake on sow backfat thickness (mm) during lactation.

	Lactation feed level			
	High	Low	sed[1]	sig[2]
No. of sows	45	44		
Time	Backfat thickness (mm)			
After farrowing	24.1	23.7	0.9	NS
21 d postpartum	22.6	19.5	0.7	**
28 d postpartum	21.8	17.3	0.7	**
Loss 21 d postpartum	1.5	4.2	0.3	**
Loss 28 d postpartum	2.3	6.4	0.3	**

[1] sed = standard error of the difference

[2] ** = significance (P<0.01); NS = nonsignificant (P>0.05)

TABLE II.5 Estimated body fat (kg) of sows fed different levels during lactation.

	Lactation feed level			
	High	Low	sed[1]	sig[2]
No. of sows	45	44		
Days postpartum	Estimated[3] body fat (kg)			
d 0[4]	66.8	64.2	2.4	NS
d 21	60.7	51.7	2.2	**
d 28	58.7	43.2	2.2	**
Fat loss (kg)	8.1	21.0	2.1	**

[1] sed = standard error of the difference

[2] ** =significant ($P<0.01$); NS = nonsignificant ($P>0.05$)

[3] Estimated using the equation $Y=-30.1+1.258(USP_1)+0.322(LW)$ where Y=body fat; USP_1=backfat measured ultrasonically at 45 mm from the midline at the level of the last rib; LW = liveweight

[4] d 0 refers to the day the sow farrowed.

Table II.6 Effect of lactation feed intake on performance and energy balance of sows over a 28-d lactation.

	Feed level[1]			
Lactation feed intake	High	Low	sed[2]	sig[3]
No. of sows	45	44		
Lactation performance				
Weight at farrowing, kg	216.9	213.6	6.1	NS
Lactation wt. loss, g/d	578.0	1392.9	65.0	**
Litter wt. gain, g/d	2035.5	1890.6	34.6	NS
Milk production, kg/d[4]	6.5	6.0	1.1	NS
Energy Balance, MJ/d				
ME intake	61.2	36.0	0.3	**
Maintenance energy[5]	25.0	23.5	1.3	**
Energy in milk[6]	54.6	50.8	1.7	NS
Maternal energy balance	-18.4	-38.3	3.4	**

[1] Avg. feed intake per day (kg) H=5.5 L=3.0

[2] sed = standard error of the difference

[3] NS = nonsignificant (P>0.05); ** = significant (P<0.01)

[4] Milk yield of sows estimated from weight gain of piglets (4 g milk / g gain) (ARC,1981)

[5] Maintenance energy requirement = (461 MJ DE)/$W^{0.75}$ per day (NRC,1988)

[6] Requirement for milk production = 8.4 MJ DE/kg based on energy content of 5.2 MJ/kg milk and efficiency of utilization of 65%. (ARC, 1981; Verstegen et al., 1985)

Table II.7 Influence of level of feed intake on sow and piglet performance through lactation.

Item	Lactation feed level			
	High	Low	sed[1]	sig[2]
No. of sows	45	44		
No. piglets born alive	10.10	10.20	0.64	NS
Avg. birthweight (kg)	1.64	1.63	0.04	NS
No. piglets weaned	8.13	8.22	0.32	NS
Avg. weaning weight(kg)	8.45	8.21	0.28	NS

[1] sed = standard error of the difference

[2] NS = Means are not significant (P>0.05)

Table II.8 Effect of lactation feed intake on reproductive performance of sows.

Lactation	Level		sed[1]	sig[2]
	High	Low		
No. of sows	45	44		
Mean interval weaning to estrus (days)	5.1	9.0	1.8	*
Pregnancy rate (%)[3]	84.5	65.5	4.2	*
Ovulation rate [4]	16.4	17.2	0.9	NS
Embryo survival (%)[5]	81.4	67.2	8.2	*

[1] sed = standard error of the difference

[2] NS = nonsignificant (P>0.05); * = significant (P<0.05)

[3] Percent of sows with embryos on d 25 postcoitum

[4] Number of corpora lutea determined on d 25 postcoitum

[5] Percent survival = embryos/corpora lutea, determined at 25 d postcoitum.

Figure II.1 Changes in body weight (kg) of sows fed high (■) and low (□) levels of feed during a 28 d lactation

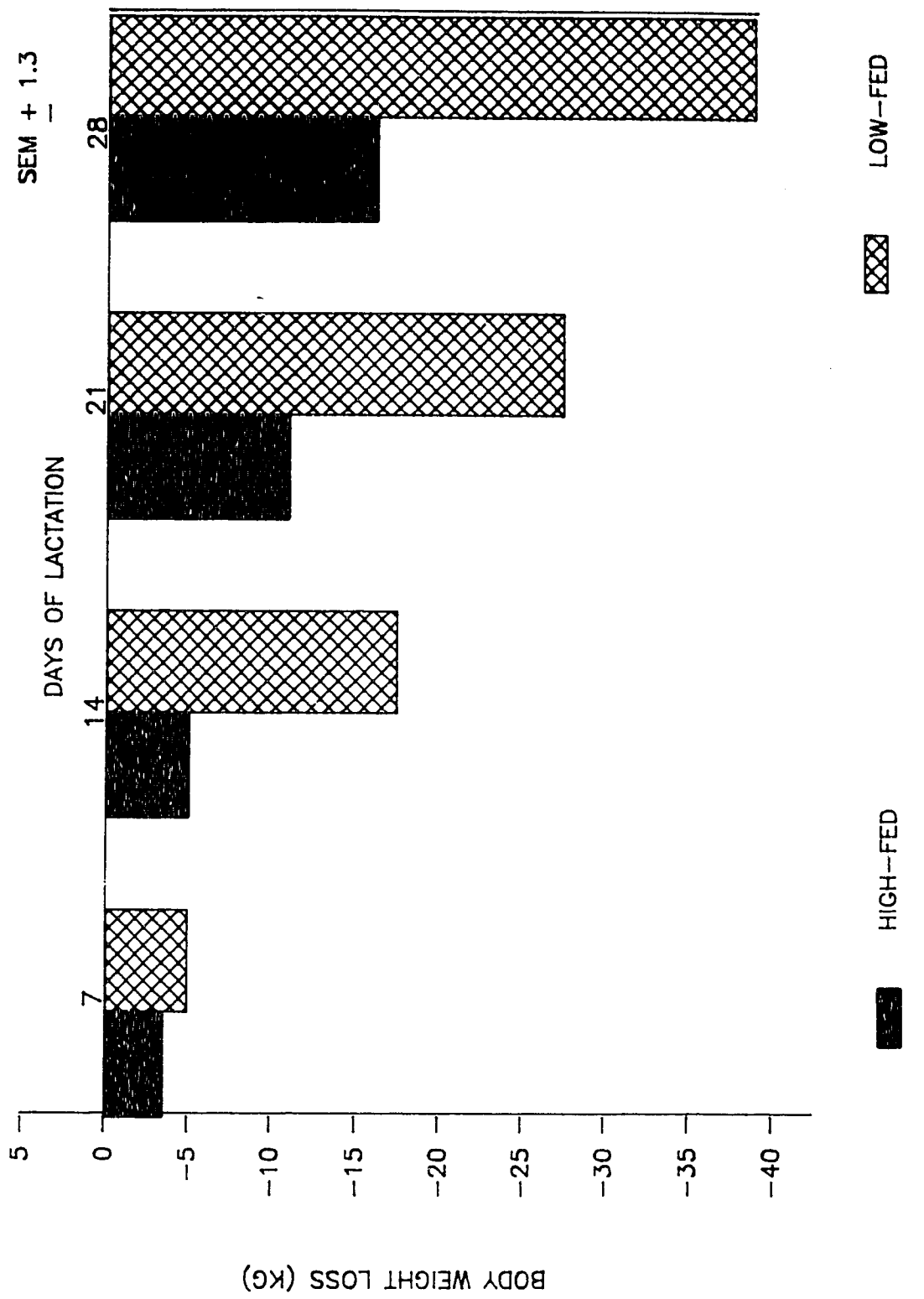

SEM + 1.3
28
21
14
7
DAYS OF LACTATION
5
0
-5
-10
-15
-20
-25
-30
-35
-40
BODY WEIGHT LOSS (KG)
HIGH-FED
LOW-FED

Figure II.2 Changes in backfat thickness (mm) in sows fed high (■) and low (□) levels of feed during a 28 d lactation

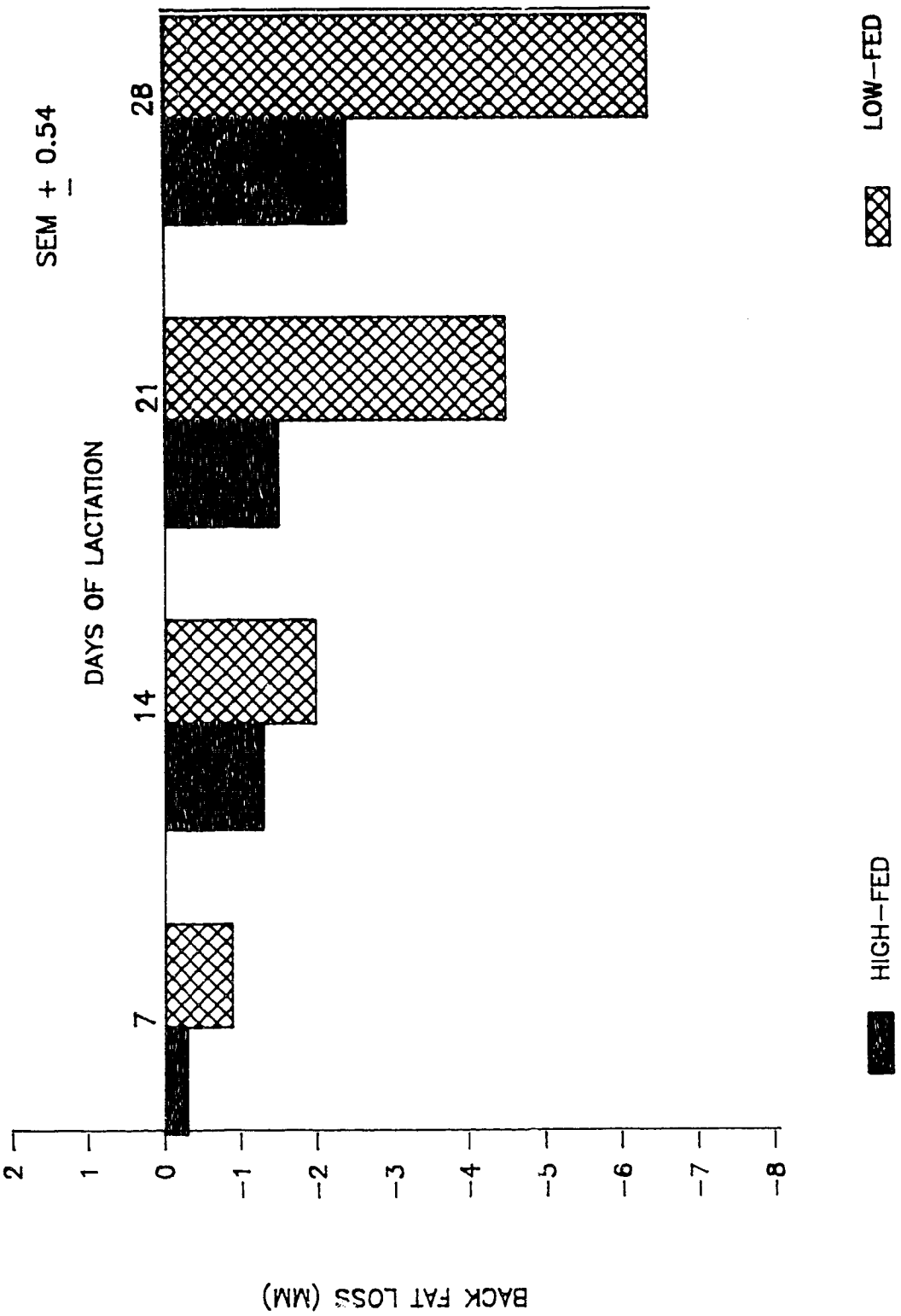

BACK FAT LOSS (MM)
2
1
0
-1
-2
-3
-4
-5
-6
-7
-8
7
14
21
28
DAYS OF LACTATION
SEM + 0.54
HIGH-FED
LOW-FED

Figure II.3 Plasma glycerol concentration on d 2 and d 28 of lactation in sows fed high (■, —) and low (+, ▵) levels of feed during lactation

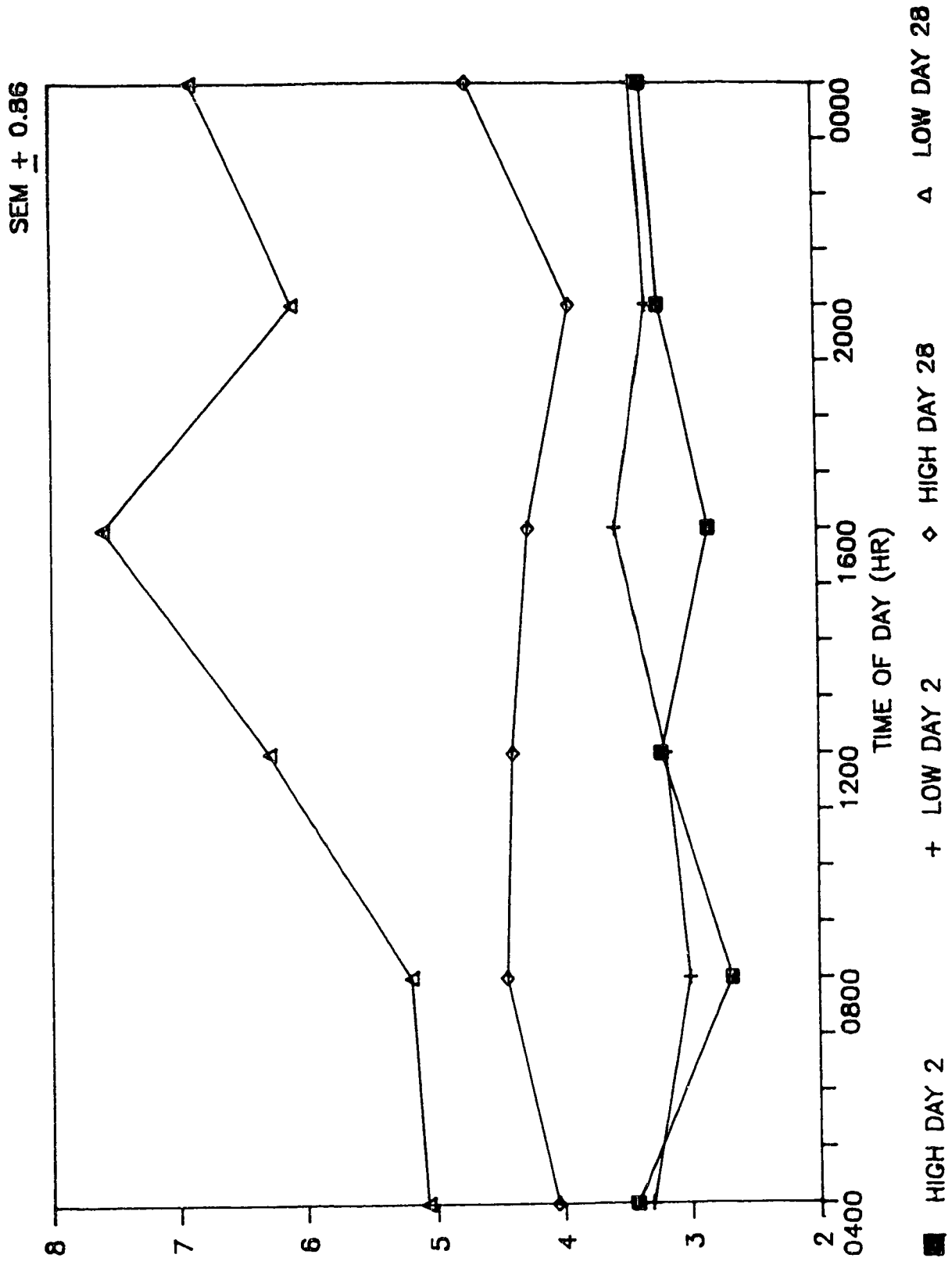
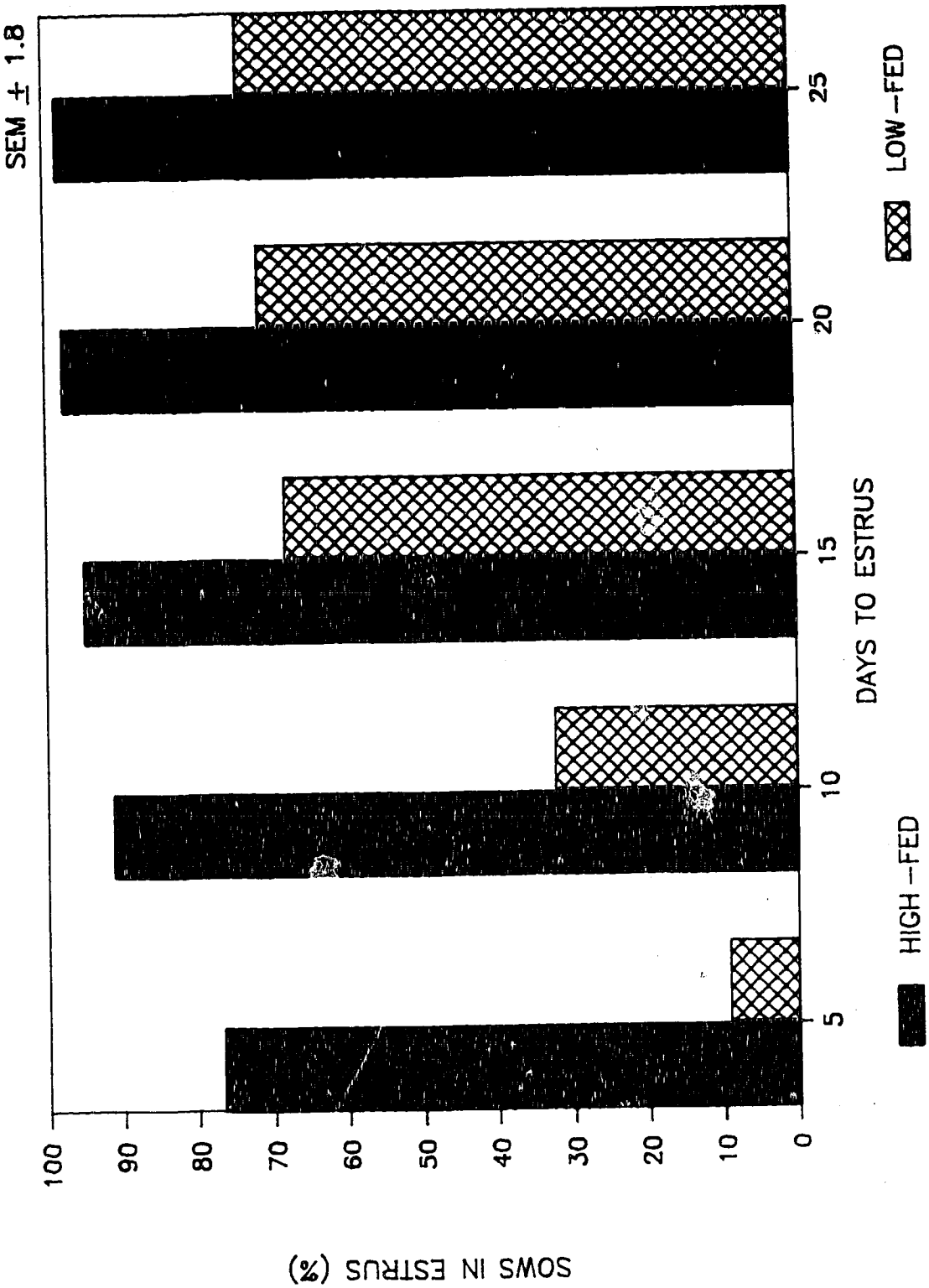

SEM ± 0.86
GLYCEROL (ng/ml)
8
7
6
5
4
3
2
0400
0800
1200
1600
2000
0000
TIME OF DAY (HR)
HIGH DAY 2
LOW DAY 2
HIGH DAY 28
LOW DAY 28

Figure II.4 Effect of feed intake during lactation on weaning to estrus interval in sows fed high (■) and low (□) levels of feed during lactation


SEM ± 1.8
SOWS IN ESTRUS (%)
100
90
80
70
60
50
40
30
20
10
0
5
10
15
20
25
DAYS TO ESTRUS
HIGH-FED
LOW-FED

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III. A STUDY OF THE INFLUENCE OF FEED INTAKE DURING LACTATION ON PLASMA HORMONE LEVELS OF THE LACTATING SOW

A. INTRODUCTION

The effect of feed intake of sows during lactation has been extensively studied and it is accepted that plane of feeding will control the extent of weight and backfat loss during lactation, and that excessive weight and backfat loss during lactation will adversely affect subsequent reproductive performance (Reese et al., 1982a,b, 1984; King and Williams 1984a,b; King and Duncan, 1986a,b; Kirkwood et al., 1987; Lythgoe, 1987). A negative relationship between body condition loss during lactation and the duration of the remating interval is thus apparent.

The lactation period in the sow is accompanied by suppressed ovarian activity (Palmer et al., 1965) but follicular size increases with advancing stage of lactation (Kunavongkrit et al., 1982). Changes in luteinizing hormone (LH) and follicle stimulating hormone (FSH) secretion during the estrus cycle of the pig have been reported (Stevenson and Britt, 1980; Camous et al., 1985). Peripheral plasma levels of LH are low during lactation (Stevenson et al., 1981), however, a significant increase was observed as the lactation period progressed (Stevenson and Britt, 1980; Kunavongkrit, 1984). Levels of pituitary LH and hypothalamic gonadotropin-releasing hormone (GnRH) are also low during lactation (Cox and Britt, 1982a). Although, the hormonal status of the sow during lactation has also been described (Stevenson et al., 1981), there is, however, a paucity of data clarifying the mechanism whereby metabolic hormones influence the secretion of reproductive hormones in the pig. Attempts have been made to explain the influence of lactation feed intake on the endocrine status of the sow during lactation (Armstrong et al., 1986; Lythgoe, 1987). However, for various reasons the results of these studies have failed to explain the endocrinological reasons for the weight loss induced reproductive problems in sows.

Therefore, the experiment reported herein was designed to investigate the influence of feed intake during lactation on the relationship between plasma estradiol-17β, LH and FSH

concentrations and the plasma concentrations of the metabolic hormones, growth hormone, insulin, glucagon, cortisol and prolactin.

B. MATERIALS AND METHODS

Animal Handling

Eighty-nine second parity Lacombe sows were randomly assigned on the basis of d 109 of gestation weights to either 3 kg (L) or 6 kg (H) of feed daily during a 28-d lactation. The diet (Table III.1) was formulated to provide 16% crude protein and 12.4 MJ DE kg^{-1}. Litter size differences were minimized by cross-fostering within d 2 of farrowing. Water was available ad libitum to sows and piglets at all times during the experiment. Sows and litters were weighed immediately after parturition, and on 14, 21 and 28 days postpartum. Sow backfat depth (45 mm lateral to the midline at the last rib) was determined ultrasonically (Kraut Kramer US K7) immediately after farrowing, and 14, 21 and 28 days postpartum.

Blood Sampling

Blood samples were collected by means of an indwelling vena cava cannula from sub groups of the first twelve sows allocated to high and low feed intakes during lactation. Twelve hours before blood sampling, a cannula (PE 90, Clay Adams) was inserted into an ear vein through a 14 gauge needle (Monoject 200, Becton-Dickinson) until the distal end was located in the vena cava. This procedure was accomplished without the use of anesthesia using a rope snare as a restraint. Heparinized saline (60 IU heparin ml^{-1} saline) was used to keep the lumen of the cannula patent. At the time of sampling, a cannula extension was fitted and kept in place in a plastic bag attached to the back of the animal by cement glue. This allowed blood sampling without disturbing the animals. Approximately 3 ml (twice the void volume of the cannula) was drawn and discarded before each collection. After blood collection, cannulae were flushed with 4 ml heparinized saline. Blood samples were taken every hour for 24 h on d 2 postpartum. At the end of the 24-h sampling period the cannulas were removed. The

procedure was repeated for blood sampling on d 28 postpartum. Additional blood samples were collected from 0800 - 1200 at 15 min intervals for 4 h immediately after weaning to monitor basal levels of LH and FSH at weaning. All blood samples were collected into glass tubes with heparin (10 IU heparin ml^{-1} blood) and held at 4 °C until collection of plasma by centrifugation at 3000 x g for 15 min at 4 °C. Plasma was portioned into small samples into sterile plastic vials (1.25 x 2.5 cm) and stored at -20 °C until analyzed for growth hormone (GH), insulin, glucagon, cortisol, prolactin, FSH, LH and estradiol-17β. Plasma samples for glucagon contained 500 IU of trasylol (FBA Pharmaceuticals, Moberg Chem. Corp. NY) ml^{-1} to inhibit proteolysis of glucagon.

Radioimmunoassays

Growth Hormone

All plasma samples collected during lactation were assayed for GH by the method of Marple and Aberle (1972). Twenty five microlitres of 0.05 M phosphate buffered saline (PBS; pH 7.4) were added to a reaction vial containing 5 μl of purified pGH (NIH, Bethesda, Md). Five microlitres of $Na^{125}I$ (New England Nuclear, Boston, MA) were then added (0.7 to 0.8 mCi) to initiate the iodination, and 25 μl of chloramine T (1 mg ml^{-1}) were added to the reaction vial to enhance the oxidation of NaI. After 1 min of mixing by finger flicking of the reaction vial, 50 μl of sodium metabisulfite (2.5 mg ml^{-1}) were added to the reaction vial to stop the oxidation reaction. Twenty five microlitres of 2% bovine serum albumin - phosphate buffered saline (BSA-PBS) were added to the reaction vial and the contents of the reaction vial were transfered to a Sephadex G-75 column (a 10 ml disposable pipet) and eluted with 0.05 M sodium phosphate buffer, pH 7.5. Aliquots of 0.5 ml were collected into tubes that contained 100 μl of 2% BSA-PBS. Specific activity of ^{125}I-pGH was typically 100 to 105 $\mu Ci\ \mu g^{-1}$.

Porcine GH was assayed using a guinea pig anti-porcine GH serum (Dr. R.R. Kracling, U.S.D.A. Athens, GA) as the first antibody. To assay pGH, 300 μl of 1% BSA-PBS, pH 7.4, with 0.02% Na-azide, 200 μl of plasma or standard (1 to 100 ng ml^{-1})

and 100 μl of first antibody (final dilution 1:60 000 bound 30 - 35% of the radiolabeled pGH) were mixed and incubated in 12 x 75 mm borosilicate glass tubes at room temperature for 24 h. After 24 h, 100 μl of ^{125}I-pGH (12 000 cpm) was added, mixed and incubated for 48 h. At 72 h after initiation of the assay, 100μl of 1:100 normal guinea pig serum and 100 μl of sheep anti-guinea pig gamma globulin (Calbiochem, San Diego, CA) were added and incubated for another 48 h. On the fifth day of the assay, the tubes were mixed and centrifuged at 3000 x g for 30 min at 4 °C. The supernatant was decanted and the pellet was counted in a Beckman Gamma 8000 Counter (Scientific Inst. Div. CA. USA). Validation of the pGH assay was as described by Chard (1982). The apparent sensitivity of the pGH assay was 1 ng ml^{-1}. Intraassay and interassay coefficient of variability was 4.2% and 8.2%, respectively. Parallelism of the pGH assay was demonstrated by assaying 100, 200 and 300 μl of plasma, respectively. Plasma pGH concentration averaged 2.6, 4.8 and 6.5 ng ml^{-1} for these respective volumes. Quantitative recovery of pGH from plasma was evaluated by assaying a porcine pool after the addition of 0.5 ng to 3 ng of pGH. The average recovery was 105 ± 8.7% of the added pGH.

Insulin

Porcine insulin (Lot NO. 407696, Calbiochem, San Diego, CA) was iodinated as described above for pGH. Porcine insulin was analysed using the double anti-body assay procedure (Ray et al., 1983). The protocol was identical to that described above for pGH with the exception that the final dilution of the first antibody (IN-40 Lot 16 Batch 683, Endocrine Sciences, Tarzana, CA) was 1:6 000 with 1:400 normal guinea pig serum (bound 38% of the labeled insulin). The second antibody used was 1:50 goat anti-guinea pig immunoglobulin (Calbiochem, San Diego, CA). All samples were assayed in a single batch with an intraassay coefficient of variation of 5.1%. Parallelism of the insulin assay was verified by assaying 10, 50, 100, 200 and 300 μl of plasma. Plasma insulin concentration averaged 1, 5, 10, 18 and 31 ng ml^{-1} respectively. Recovery of insulin from plasma was conducted as described for the pGH assay. The average recovery was 95.3 ±

2.1%. The apparent sensitivity of the insulin assay was 2 ng per assay tube.

Glucagon

Plasma glucagon was measured by the method of Harris et al. (1979). Purified glucagon for standards, was generously provided by Dr. M.A. Root of Eli Lilly Co. Indianapolis, IN. Glucagon was assayed using 30 K beef-pork glucagon antiserum (O4A Pool 1 Lot 14, b3538 from Dr. R.H. Unger, University of Texas, Southwestern Medical School, Dallas, TX). To assay for glucagon concentration in plasma, 200 μl of plasma, 200 μl of glucagon antiserum with a final dilution of 1:115 000 (diluted with 0.25% normal human serum albumin (Hyland Laboratories, Los Angeles, CA) in sodium barbitol buffer, pH 8.6, and 200 μl of glucagon I^{125} (Lot No. N412119, New England Nuclear, Boston, MA; Specific activity 150-200 μl μg^{-1}) with 50 IU trasylol were incubated in 12 x 75 mm borosilicate glass tubes at 4 °C for 48 h. After 48 h, 100 μl of goat anti-gamma globulin (1:25) and 100 μl of normal rabbit serum were added and the vortexed and incubated for 24 h. At 72 h after the initiation of the analysis, the assay tubes were vortexed and centrifuged at 3500 x g for 15 min at 4 °C. The supernatant was decanted and the pellet was counted for 5 min in a Beckman Gamma 8000 counter. The apparent sensitivity of the glucagon assay was 40 pg ml^{-1}. Plasma samples were analyzed in a single assay. Intraassay coefficient of variation was 9%. Parallelism of the glucagon assay was determined by assaying 25, 50, 75 and 100 μl of pooled porcine plasma. Plasma glucagon concentration averaged 10, 18, 30 and 50 pg / tube for these respective volumes. Quantitative recovery of glucagon from plasma was evaluated by assaying a porcine plasma pool after the addition of 100 to 1000 pg of glucagon. The average recovery was 91 $\pm$ 5% of the added glucagon.

Cortisol

All plasma samples collected during lactation were assayed for total cortisol by the method of Evans (1987). Plasma samples (500 μl) were diluted with 500 μl Tris-gel buffer (0.05m Tris-HCL, 0.1M Nacl, 0.015 m sodium oxide, 0.1% gelatin) and heat

treated in 12 x 75mm glass tubes for 15 min at 95 °C to denature cortisol binding globulin (Forster and Dunn, 1974). Rabbit anticortisol-21 thyroglobin antisera (lot COR3, Miles-Yeda Ltd., Toronto, Ont) was added in 100 μl volume of Tris-gel buffer and incubated overnight at 4 °C. After the 24 h incubation, 100 μl of Tris buffer containing 14,000 DPM labelled cortisol (1,2,6,7 $^{-3}$H hydrocortisone, 93 Ci mmole $^{-1}$, New England Nuclear, Boston, MA) was added and the assay incubated overnight at 4 °C. Separation of free from bound cortisol was achieved by the addition of 200 μl of 0.5% charcoal (Norit A, Fisher Scientific, Fairlawn, NJ, U.S.A.) 0.05% Dextran (70,000 MWT, Sigma Chem. Co., St Louis, MO) in Tris-gel buffer. Following incubtion for 10 min at 4 °C, the contents were centrifuged at 3000 x g for 15 mins. To 500 μl aliquot of the supernatant was added 5 ml of liquid scintillation fluid, general purpose emulsifying scintillator (Beckman Instruments (Canada) Inc., Mississauga, Ontario) and counted in a liquid scintillation counter (Beckman LS 5801, Beckman Inst., Irvine, CA) for 2 min. Validation of the cortisol assay was as described for porcine growth hormone as above. Percent cross reactions of the anticortisol serum as reported by the supplier were : corticosterone, 11.7%; deoxycorticosterone, 6.0%; 17β-OH progesterone, 9.4%; progesterone, 6.9%; testosterone, 4.0%; prednisolone, <1.0%; aldosterone, androstenedione, <0.5%; dehydroepriandrosterone, estradiol-17β and dexamethasone, <0.01%. Accuracy of the cortisol assay was evaluated by adding known quantitites of standard (hydrocortisone, Sigma Chem. Co., St Louis, MO) covering the full range of the standard curve (from 1 to 200 ng ml^{-1} plasma for a 5 μl sample volume) to a control plasma. The average recovery was determined as 96.8 $\pm$ 3.2%. Cortisol potencies in unknown samples were not routinely corrected for recovery. Sensitivity of the assay was 1.0 ng ml^{-1} plasma. Intraassay and interassay coefficients of variation, determined with standard plasma, were 5.2 and 8.5%, respectively.

Prolactin

Plasma prolactin concentration was quantified by a homologous double-antibody radioimmunoassay by the method of Mulloy and Malven (1979). Purified porcine

prolactin, pPRL, (LER-2073 provided by Dr. L.E. Reichert, The Albany Medical College of Union University, Albany, NY) was used as the radioiodinated antigen and standard. Aliquots of 5 μg prolactin in 5 μl of distilled water were iodinated by the chloramine T method as described by Greenwood et al. (1963). For each iodination, 25 μl of 0.5 M phosphate buffer (pH 7.5) was mixed with 1 mCi $Na^{125}I$ in 20 μl of 0.05 M phosphate-buffered saline (PBS), pH 7.5, and 20 μl of chloramine T (1 mg ml^{-1} of .05 M phosphate buffer, pH 7.5) were mixed with the 5 μg aliqout of prolactin for 20 sec. The reaction was stopped with the addition of 50 ug sodium metabisulfate (1.0 $\mu g/\mu l$ of 0.5 M phosphate buffer). The reaction mixture was diluted with 100 μl of 1% potassium iodide and 100 μl assay diluent (0.05M PBS containing 2.7 mM EDTA and 1% bovine serum albumin (BSA). Free ^{125}I was separated from ^{125}I-labelled prolactin (^{125}I-PRL) on a Sephadex G 100 column. The ^{125}I-PRL was then purified on 9.6 x 17 cm ion exchange column. Maximal binding of label was observed in the fractions representing the final portion of the protein-bound peak and these were pooled and stored at 4 °C for subsequent use in the assay.

To assay for pPRL, 200 μl of 1% BSA-PBS, pH 7.5, 100 μl of plasma or standard (in 100 μl of 1% BSA-PBS, pH 7.5), 100 μl first antibody (anti porcine prolactin (ALM-5, Research Products International Co., IL) final dilution 1:50 000 in 1:200 normal goat serum) and 100 μl of 1% BSA-PBS containing ^{125}I-PRL (10 000 cpm) were mixed and incubated in 12 x 75 mm borosilicate glass tubes for 24 h at 4 °C. After 24 h, 200 μl of rabbit anti-goat gamma globulin (Calbiochem, San Diego, CA) diluted at 1:15 in 1% BSA-PBS was added to the assay tubes and incubated for a further 24 h at 4 °C. Three millilitres of cold PBS, pH 7.5, was added to each tube and centrifuged at 1500 x g for 30 min. The supernatant was aspirated and the radioactivity in the pellet was counted in a Beckman 8000 Gamma counter.

The dilution of first antibody (1:50 000) used bound 25 - 36% of ^{125}I-PRL in the absence of unlabelled hormone. Three volumes (100, 200 and 300 μl) of a pooled plasma sample included in each assay to test for parallelism to the standard curve gave

mean values of 1.3, 2.7 and 4.3 ng/tube. Accuracy, estimated as the recovery of prolactin at known concentrations from pig plasma, gave values ranging from 95.9 to 101.5%. The mean assay sensitivity was 0.2 ng/assay tube. Intraassay and interassay coefficients of variation were 8.5% and 10% respectively. The cross-reactivity of antiserum has previously been investigated by Kraeling et al. (1982) who found that cross-reaction with large quantities (2 - 200 ng) of FSH (NIH-FSH-P-2), LH (LER-778-4) and growth hormone (NIH-GH-P-5268) were all <1%.

Luteinizing Hormone

Plasma concentrations of porcine luteinizing hormone, pLH, were determined by double-antibody radioimmnuoassay as described by Kraeling et al. (1982). Purified pLH (LER 786-3, was provided by Dr. L.E. Reichert, Albany Med. College, Albany, NY) was used for iodination and standards (0.1 ng ml^{-1} to 10 ng ml^{-1}). Aliquots of 2.5 μg pLH in 2.5 μl of distilled water were iodinated by the chloramine-T method (Greenwood et al., 1963). At each iodination 20 μl of 0.5 M phosphate buffer, pH 7.5, were added to the reaction vial containing the pLH, and 30 μl of 0.05 M phosphate buffer was added to the vial containing 0.5 mCi $Na^{125}I$. The $Na^{125}I$ was added to the reaction vial and 25 μl chloramine-T (1 mg ml^{-1} in 0.5 M phosphate buffer, pH 7.5) were mixed with the pLH and $Na^{125}I$ for 2 min. The reaction was stopped with 50 μl sodium metabisulfite (2.5 mg ml^{-1} in 0.5 M phosphate buffer, pH 7.5) added to the reaction mixture for 1 min. The reaction mixture was diluted with 25 μl 2% BSA-PBS and 100 μl transfer solution (0.16 g ml^{-1} sucrose, 0.01 g ml^{-1} KI) and layered on Sephadex G 100 column (a 10 ml disposable pipette) for separation of free ^{125}I from ^{125}I-pLH. The reaction vial was rinsed with rinse solution (0.08 g ml^{-1}, 0.01 g ml^{-1} KI and 100 ng ml^{-1} bromophenol blue) and added to the layer on the column. The column was eluted with column buffer (0.05 M phosphate buffer, pH 7.5). Aliquots of 1 ml were collected into tubes containing 100 μl of 2% BSA-PBS.

The assay procedure was performed at 4 °C and all dilutions were made in 1% BSA-PBS, pH 7.5. Plasma or standard (300 μl) was added to 12 x 75 mm glass tube

containing 200 μl of buffer and 200 μl of diluted anti-pLH (rabbit-anti bovine, final dilution 1:160 000) containing 1:400 normal rabbit serum was added, mixed and incubated for 24 h. After 24 h incubation, 100 μl of ^{125}I-pLH (12 000 cpm) was added and incubated for 24 h. Then 200 μl of goat anti-rabbit gamma globulin (Lot No. 539845, Calbiochem, San Diego, CA) diluted at 1:25 in 1% normal rabbit serum in 0.05 M EDTA PBS, was added and incubated for 48 h. After 48 h the tubes were centrifuged at 3000 x g for 30 min to separate antibody bound ^{125}I-pLH from free ^{125}I-pLH. The supernatant was decanted and the remaining pellet was counted in a Bechman 8000 Gamma Counter (Scientific Instruments Division, Irvine, CA).

Dose response curves for pooled porcine plasma and increasing concentrations of the pLH standard (LER 786-3) added to the plasma pool were parallel ($P>0.5$). Assay sensitivity was 0.2 ng/tube. Recovery of purified standard added to control plasma was 95.4 ± 2.7%. The intraassay and interassay coefficients of variation were 4.9% and 5.2% respectively. Cross-reactions of the first antibody with large quantities of other preparations of pituitary hormones (0.1 to 1000 ng ml^{-1}) as reported by Kraeling et al. (1982) were: FSH (NIH-FSH-P-2), 2.4%; growth hormone (NIH-GH-P-526B), 2% and PRL (LER-2073), <0.001%.

Follicle Stimulating Hormone

All plasma samples were assayed for porcine follicle stimulating hormone (pFSH) by Dr. A.A.J. Vandalem and associates (Centre Hospitalier Universitaire, Sart Tilman, Belgium). The assay method was as described by Vandelem et al. (1979). Iodination of pFSH was by glucose oxidase method of Tower et al. (1977). Labeled pFSH was purified on a 15 x 1 cm column of Sephadex G 50, and the combined fractions of the first peak were further purified on a 90 x 1.5 cm column of Sephadex G 100. The fractions of the third peak were pooled and stored at 4 °C until use. This tracer had a specific activity of 100 - 200 μCi μg^{-1}. The antiserum specific for pFSH was obtained from Dr. G. Hennen (Academic Hospital, Liege, Belgium). The antiserum was used at a final dilution of 1:100 000, giving an initial binding of 30%. Twelve standard amounts of pFSH (G. Hennen)

ranging from 0.10 to 50 ng ml^{-1} were prepared in assay buffer (0.02 M $Na_2HPO_4.2H_2O$; 0.2% Na N_3; 1% BSA pH 7.6). Samples and standards were assayed in triplicate, and samples were assayed in two dilutions corresponding to 100 and 200 μl of undiluted plasma.

The percent cross reactivity of pLH, pTSH, pLHα, pLHβ, pTSHβ, pTSHα, pFSHα, pFSHβ and pPRL was <0.1, 7.8, <1.0, <1.0, <1.0, <1.0, <1.0, 40, and 0.1 respectively (subunits were tested by Dr. G. Hennen). Recovery of twelve quantities of pFSH (0.20-200 ng) added to 200 μl plasma containing 1.5 ng pFSH was 108.7 $\pm$ 10.2%. Serial dilutions of plasma sample ranging from 200 to 50 μl of undiluted plasma yielded pFSH concentrations of 13.8 $\pm$ 1.0 ng ml^{-1}. The intraassay and interassay coefficients of variation were 8.1% and 10% respectively. The sensitivity of the assay was 0.2 ng ml^{-1}.

Estradiol-17β

The radioimmunoassay for estradiol-17β was performed according to the procedures of Wettemann et al., (1972) and the validation procedure was as described for cortisol above. Aliquots of plasma (500 μl) were extracted twice with diethyl ether. Plasma samples and 5 ml of diethyl ether were added to extraction tubes mixed for 5 min and the aqueous phase frozen in a methanol-liquid nitrogen bath. The supernatant was poured into a 12 x 75 mm assay tubes and dried under nitrogen gas. Estradiol-17β standards (Sigma, Lot No. E8875, San Diego, CA) were prepared in ethanol and ranged in concentration from 5 to 5000 pg ml^{-1}. A 100 μl of standard was added to assay tubes and dried under nitrogen gas. A 200 μl antiserum (rabbit antiserum against estradiol-17β, Lot No. E-26-47, Endocrine Sciences, Tarzana, CA) with a final dilution of 1:120 000 was added to all tubes with standards and plasma samples. The mixture was vortexed and incubated for 1 h at 4 °C. A 100 μl of 12 000 dpm tracer ([2,4,6,7-^{3}v 2620H]-estradiol-17β, specific activity 250 μCi, New England Nuclear, Boston, MA) were added to reaction mixture and incubated for 24 h at 4 °C. After 24 h, 200 μl dextran charcoal suspension was added to the tubes and incubated for 10 min and then centrifuged for 10 min at 3000 x g at 4 °C. The supernatant was aspirated into

scintillation vials. Scintillation fluid, general purpose emulsifying scintillator (Beckman Instruments (Canada) Inc. Mississauga, Ontario) was added to the aspirated supernatant and incubated overnight at 4 °C and then counted in a Beckman LS 5801 Liquid Scintillation Counter.

Recovery of 5 to 100 pg estradiol-17β, added to 0.5 ml aliquots of a plasma sample containing 10 pg estradiol-17β per milliliter was 104 ± 10%. Estradiol-17β potencies in unknown samples were not routinely corrected for recovery. The intraassay and interassay coefficients of variation, determined with standard plasma in triplicates, were 5.9% and 12% respectively. Parallelism between the dose response curves for pooled porcine plasma and increasing concentrations of estradiol-17β standard was demonstrated ($P>0.5$). The sensitivity of the assay was 8.5 pg ml^{-1} plasma. Cross reactions of the rabbit antiserum against estradiol-17β as reported by the supplier (Endocrine Sciences, Tarzana, CA) were: estrone, 1.3%; estriol, 0.6%; 17α-estradiol, 0.1%; cortisol, corticosterone, progesterone, testosterone, all were <0.01%.

Statistical analysis

Data were analyzed for effects of dietary treatment during lactation by analysis of variance of the Statistical Package of Social Sciences (SPSS INC. 1984). Changes in hormone levels over time were analyzed by split plot analysis of variance using analysis of variance procedures of SPSSX. Comparisons between two means were made by Student's T-test (Steel and Torrie, 1980). The total hormone secreted over time during lactation was computed as area under the curve according to the method of Goodman and Karsch (1980). The area under the curve was calculated as the area above the baseline, and the corresponding means according to treatment were subjected to Student's T-test. Differences were considered to be insiginificant if $P>0.05$.

C. RESULTS

Sows and litter performance

The results of the performace of the sow and litter during lactation is as presented in Chapter II.

Effect of feed intake on hormone profiles during lactation

Growth hormone

Porcine GH concentrations were very similar ($P>0.05$) on d 2 postpartum for H and L fed sows (2.6 $\pm$.1 vs 2.5$\pm$.1). In both H and L fed sows GH increased ($P<0.05$) as lactation progressed (Figure III.1 and Table III.2). At weaning, GH was higher ($P<0.05$) in the L-fed sows than the H fed sows. The mean GH concentration and apparent amount of GH secreted were higher ($P<0.05$) in the L-fed sows than the H-fed sows on d 28 postpartum.

Insulin

There was no measurable postprandial increase in plasma insulin and no significant ($P>0.05$) treatment effect on the mean plasma insulin concentrations on d 2 postpartum (Table III.3 and Figure III.2). On d 28 postpartum, however, there was an increase ($P<0.01$) in plasma insulin concentration in sows in both treatment groups after the morning feed. Furthermore, the H-fed sows attained higher ($P<0.05$) peak amplitudes in response to feeding than the L-fed sows (26.2 $\pm$ 2.5 vs 23.9 $\pm$ 3.3 ng ml^{-1}). The response to feed intake by insulin after the afternoon feeding was not significant between the two treatments. The H-fed sows apparantly secreted ($P<0.05$) more insulin than the L-fed sows as indicated by the area under the insulin curve (Table III.3 and Figure III.2).

Glucagon

Plasma glucagon concentrations on d 2 postpartum were not influenced ($P>0.05$) by lactation feeding. Glucagon concentration on both d 2 and d 28, generally increased ($P>0.05$) from 1200 H until 1600 H and then decreased after the 1500 H feeding. However, on d 28 postpartum, glucagon levels increased slightly ($P>0.05$) in both treatment groups (Table III.4 and Figure III.3). The L-fed sows had higher levels of plasma glucagon on d 28 postpartum than the H-fed sows although the difference was not significant ($P>0.05$). The sows fed low levels of feed during lactation apparently secreted more glucagon ($P<0.05$) as indicated by the area under the glucagon curve on d 28 postpartum. Ratio of insulin to glucagon was low ($P>0.05$) on d 2 postpartum compared to the high ($P>0.05$) ratios on d 28 postpartum (Table III.4).

Cortisol

The plasma cortisol profiles for d 2 and d 28 postpartum are shown in Figure III.4 and Table III.5. The characteristics of the cortisol profile is presented in Table III.5. There were no treatment differences ($P>0.05$) in plasma cortisol concentration on d 2 postpartum (21.1 vs 21.9 ng ml^{-1} for H and L fed sows, respectively). On d 28 postpartum, both treatment groups showed high levels ($P<0.05$) of cortisol at 1600 H and 0200 H (Figure III.5). In both time periods, the L-fed sows had higher ($P<0.05$) plasma cortisol concentration than the H-fed sows. On d 28 postpartum, mean plasma cortisol concentrations, and apparent total secretion of cortisol within 24 h were significantly higher ($P<0.01$) in the L-fed sows than the H-fed sows.

Prolactin

Mean plasma prolactin profiles in lactating sows are illustrated in figure III.5 and the characteristics of the profile are presented in Table III.6. Plasma prolactin concentration were not significantly affected by lactation feed intake on d 2 and d 28 postpartum. In both treatment groups, plasma prolactin concentration decreased from 108 ng ml^{-1} on d 2 postpartum to 37 ng ml^{-1} on d 28 postpartum. The low fed sows tended

($P>0.05$) to secrete more prolactin as depicted by the area under the prolactin curves on d 28 of lactation (Table III.6).

Follicle stimulating hormone

Plasma FSH concentrations were uniformly low on d 2 postpartum for both treatment groups. But plasma concentrations of FSH increased ($P<0.01$) in both L-fed and H-fed sows as period of lactation increased from d 2 to d 28 postpartum (Figure III.6 and Table III.7). The H-fed sows tended to have a higher ($P>0.05$) mean plasma FSH concentration on d 28 postpartum than the L-fed sows as shown in figure III.6. Total secretion of FSH on d 28 postpartum, as indicated by the area under the curve (Figure III.6) the H-fed sows had higher levels of FSH secreted ($P<0.05$) than the L-fed sows.

Luteinizing hormone

Plasma LH concentration was below the level of sensitivity of the assay (0.2 ng ml^{-1}) on d 2 postpartum. On d 28 postpartum, LH concentrations were higher in H-fed sows than the L-fed sows but the differences were not significant ($P>0.05$) (Figure III.7 and Table III.8). The apparent total amount of LH secreted on d 28 postpartum was higher ($P<0.05$) in the H fed sows than in L fed sows (45.8 ± 3.9 vs 32.2 ± 2.2) with an arbitary units of h. ng ml^{-1}).

Estradiol-17β

The peripheral plasma concentration of estradiol-17β were low throughout lactation in both treatment groups as shown in Table III.9. The mean estradiol-17β concentration on d 28 postpartum for the H-fed sows was 28 pg ml^{-1} and 23 pg ml^{-1} in the L-fed sows.

Hormonal Profiles During Postweaning Period

Frequent Sampling

The characteristics of episodic LH secretion as determined by the periods of frequent sampling (every 15 min for 4 h) immediately after weaning is presented in Figure III.8 and Table III.10. There was a mean increase in basal LH concentration in both H fed and L fed sows in lactation after 120 min from weaning (figure III.8). Overall, the H fed sows, had higher basal levels of LH than the L fed sows, although the differences were not significant ($P>0.05$) and there were no consistent effects of lactation feed intake on the pattern of episodic LH secretion (mean episodic frequency and amplitude for H fed sows during lactation were 0.75 ± 0.3 pulses per 4 h, 1.36 ± 0.2 ng ml^{-1} and for L fed sows were 0.63 ± 0.3 pulses per 4 h and 1.22 ± 0.3 ng ml^{-1}).

The mean plasma FSH concentration measured during the 4 h frequent sampling period (Table III.10 and Figure III.8) immediately after weaning was not consistently ($P>0.05$) influenced by feed intake during lactation.

D. DISCUSSION

During lactation, the L fed sows lost significantly more weight than the H fed sows. This weight loss was due to depletion of both fat and lean tissue as demonstrated in chapter II. The greater weight loss in L fed sows was reflected in the characteristics of the hormonal profiles of these animals during lactation. As substrate supply has a major effect on the metabolic processes and energy metabolism is subject to hormonal regulation the changes in growth hormone, insulin, glucagon and cortisol in both groups of sows indicated the extent to which lactation feed intake affected general metabolism.

The higher plasma concentration of growth hormone in late lactation may be associated with body metabolic changes in response to lactation and the corresponding increased demand for nutrients. High concentrations of growth hormone during lactation are believed (Hart, 1983) to facilitate the partitioning of nutrients, at least glucose and lipid, to the mammary gland. Increased levels of growth hormone during lactation were observed in

both H fed and L fed sows; however, the L fed sows had significantly higher levels of mean plasma growth hormone and apparent total secretion of growth hormone than the L fed sows. The L fed sows as shown in Chapter II lost significantly greater amounts of fat than the H fed sows. It has been shown that in undernutrition, plasma growth hormone levels are higher in humans (Warren, 1983) and swine (Lythgoe, 1987). Furthermore, there are indications that, average daily secretion of growth hormone is negatively correlated with the carcass fat in pigs (Machlin, 1972), lambs (Wagner and Veenhuizen, 1978) and cattle (Trenkle and Topel, 1978). In malnutrition high growth hormone levels are useful for their lipolytic effect, thus allowing an increased use of fat as an energy source. In this study, the increased plasma growth hormone concentration as sows advanced into lactation are consistent with mobilization of lipid from adipose tissue to provide energy substrates for body functions and milk synthesis.

Low plasma insulin concentrations during d 2 postpartum are presumably related to energy balance rather than stage of lactation per se. Bauman and Currie (1980) indictated that available glucose is preferentially shunted towards mammary metabolism at the begining of lactation. In contrast to insulin, plasma prolactin concentrations were very high at the begining of lactation. The high prolactin concentrations may have contributed to the low plasma level of insulin in both H fed and L fed sows on d 2 postpartum as prolactin is known to increase the number of insulin receptors in the mammary gland during lactation (Flint, 1982). Removal of litters from lactating rats caused a decrease in number of insulin receptors in the mammary gland and an increased plasma level of insulin (Flint, 1982). Therefore, changes in the number of insulin receptors in the mammary gland, mediated by changes in prolactin may have contributed to the low levels of plasma insulin concentration on d 2 and high plasma insulin levels on d 28 postpartum.

Concentrations of insulin in plasma have also been shown to be positively correlated with feed intake (Basset, 1974) however, pattern of insulin secretion was very similar for both H fed and L fed sows on d 2. Postprandial insulin responses were present after feeding on d 28 with differences between groups of sows. Insulin enhances the utilization of glucose and

amino acids by peripheral body tissues (Spencer, 1985). Loss in body weight as sow progress from early to late lactation, points to energy deficit situations and expexted dominance of plasma glucagon over plasma insulin levels. This is consistent with the present the study in that the L-fed sows had higher plasma glucagon concentrations on d 28 postpartum than the H fed sows. High plasma glucagon levels represent a dominance of the glycogenolytic and gluconeogenic actions of glucagon and peripheral utilization of free fatty acids (FFAs) and amino acids for convertion to glucose (Brockman and Laarveld, 1986) and there are indications that high plasma concentrations of glucagon are asscioclated with elevations of plasma FFAs (Basset, 1974) and glycerol (Brockman et al., 1975) concentrations. The high levels of FFAs in plasma reported by Lythgoe (1987) after feeding at low levels (six percent of metabolic body weight) during lactation over three parities and the high glycerol concentrations reported in late lactation in Chapter II of the present study both indicate that lipolysis occurred during lactation.

Plasma cortisol concentrations were significantly higher in the L-fed sows on d 28 postpartum than in the H-fed sows. Cortisol also potentiates the hepatic actions of glucagon (Unger, 1981) by causing an impairment of glucose metabolism in adipose tissue by decreasing the available glycerol phosphate and thus inhibiting fat synthesis (Brockman and Laarveld, 1986): Fat is thus made available for milk synthesis. Increased cortisol, therefore, seems to increase the availability of glucose and energy for the mammary gland.

Cortisol together with epinephrine and glucagon act as the conter regulatory hormones to insulin in the regulation of blood glucose levels (De Fronzo et al., 1980). Epinephrine and glucagon are fast acting, whereas cortisol enhances and prolongs for several hours the increase in blood glucose due to epinephrine or glucagon (Munck, 1970). In addition cortisol inhibits the secretion of insulin and stimulates secretion of glucagon (Yasuda et al., 1982). Thus cortisol can also be viewed as making use of a variety of mechanisms to protect the organism against overactivity of the prime regulator of blood glucose, insulin.

Plasma concentrations of FSH and LH were very low on d 2 postpartum. Lactation in the sow is normally characterized by low plasma concentrations of gonadotropins and

hyperprolactinaemia (Britt et al., 1985). The low level of LH secretion during lactation may be due to suppression by suckling (Stevenson et al., 1981) and possibly acts as a means of preventing ovulation too soon after parturition. The low plasma concentration of FSH during lactation is suggested to be due to inhibin produced by the ovaries (follicles) (Stevenson et al., 1981). As lactation progressed from d 2 to d 28 postpartum, the level of FSH and LH increased, while plasma concentration of estradiol-17β decreased slightly. The high plasma concentration of estradiol-17β on d 2 postpartum could have originated from sources other than the ovaries which are putatively quiescent during lactation. Body fat deposits may be a possible source but pigs do not have the capacity to accumulate steroids in body fat to the same extent as dairy cows (McCraken, 1964). MacDonald (1975) (cited by Varley et al., 1984) concluded that the most likely source of origin is the adrenal glands which, under certain conditions, have the competence for significant estradiol-17β production. Parturition and the initiation of lactation is a time of such physiological change and upheaval that ostensibly the animal is under acute stress. Under these conditions the adrenals may therefore secrete estradiol-17β. On the other hand, Holeness and Hunter (1975) reported that the high levels of estradiol-17β at parturition is apparently due to the peak of feto-placental estrogens that occurs at parturition.

Undernutrition resulted in changes in the metabolic state of the animal such that there was an inbalance of catabolic to anabolic hormones in favor of the catabolic hormones. The catabolic hormone, cortisol is also known to reduce the sensitivity of the pituitary gland to GnRH (Barb et al., 1982) resulting in reduced secretion of LH and FSH from the gonadotrophs. For example, Dubey and Plant (1985) reported that the major site of this inhibitory action of cortisol on gonadotropin secretion resides at a supra pituitary level and is mediated by interuption of hypothalamic GnRH release.

The observed elevations of plasma growth hormone, glucagon and cortisol with a concomitant reduction in plasma insulin concentration in the low-fed sows was associated with reduction in plasma levels of FSH and LH. Pearce et al. (1988) observed that elevated plasma cortisol concentrations can reduce pituitary responsiveness to exogenous GnRH and

elevations in plasma cortisol has been shown to depress the preovulatory surge of LH in mature gilts (Barb et al., 1982) and LH secretion in boars (Pitzel et al., 1984).

In conclusion, this current data have provided further evidence to indicate that weight and fat loss during lactation in sows is associated with changes in the profiles of metabolic and reproductive hormones. The catabolic hormones, especially cortisol and a reduction in plasma insulin is suggested to have caused the reduction in plasma concentrations of LH and FSH. The depressed secretion of the gonadotrophins, LH and FSH, in the low fed sows could reduce subsequent ovarian development in the sow since a functional relationship between LH secretion in lactation and follicular development has been suggested (Foxcroft et al., 1987).

Early observations of the changes in LH concentration indicated that the secretion of LH was unaffected at weaning (Aherne et al., 1976; Parvizi et al., 1976; Stevenson and Britt, 1980; Kirkwood et al., 1984). As reported by other authors (Edwards and Foxcroft, 1983; Shaw and Foxcroft, 1985; Foxcroft et al., 1987) and as shown in the present study, the use of frequent sampling procedures indicate that there is a rise in LH secretion immediately after weaning. This increase in LH secretion, especially in the H-fed sows, may be important for initiating sustained follicular growth as shown by experiments in which exogenous LHRH treatment promoted follicular development during lactation and postweaning anestrus (Cox and Britt, 182b; Britt et al., 1985; Ramirez et al., 1985; Rojanasthien et al., 1987). Similar observations have been noted in sheep resuming estrus activity after a seasonal anestrus period (Rhind et al., 1986) and in cattle (Wright et al., 1987). This initial increase was similar for both groups of sows fed high and low during lactation. Since this basal increase was not significantly influenced by nutrition during lactation, it indicates that the rise in LH basal levels may possibly be due to removal of the suckling stimulus as demonstrated by Stevenson et al. (1981) and Cox and Britt (1986). The increase in basal LH may be caused by the decline in neural inhibition of GnRH synthesis as shown in the ewe (Foster and Olster 1985).

Follicle stimulating hormone (FSH) characteristics immediately after weaning were also not influenced by lactation feed intake although plasma FSH concentration in the high fed sows tended to be higher than in the low fed sows. Plasma FSH concentration did not

appear to be significantly depressed in the present study during lactation (Chapter III) when compared to FSH levels after weaning and during the estrous cycle of the sow (Foxcroft and Van de Wiel, 1982). This may suggest that removal of the suckling stimulus at weaning does not influence the pattern of FSH secretion. There is a suggestion that FSH may respond more to non-steroidal follicular signals such as folliculostatin (Stevenson et al., 1981) than to the removal of the suckling stimulus. However, in this experiment it was observed that FSH levels increased after weaning. Therefore, there could be a mechanism overriding the inhibitory effect of inhibin and allowing the increase in FSH concentration at weaning.

The present results would therefore suggest that the effects of weight loss and the consequent increase in the catabolic hormone, cortisol and a decrease in the anabolic hormone, insulin, may influence the rate of neurotransmitter synthesis required for GnRH secretion or enhance pituitary responsiveness to GnRH. This area requires further study before a complete understanding may be achieved.

Table III.1 Formulation and composition of sow diet.

Ingredients	% Diet
Barley	57.5
Wheat	23.4
Soybean meal (46% CP[1])	13.1
Calcium phosphate	1.5
Limestone	1.0
Vitamin-mineral premix[2]	1.0
Iodized salt	0.5
Tallow	2.0
<u>Chemical analyses</u>	
Dry matter (%)	89.3
Crude protein (%)	16.4
Lysine (%)	0.72
Digestible energy ($MJ.kg^{-1}$)[3]	12.4

[1]CP = crude protein

[2]Supplied the following per kg of diet:120 mg zinc, 12 mg manganese, 150 mg iron, 12 mg copper, 0.1 mg selenium, 5000 IU vitamin A, 500 IU vitamin D_3, 22 IU vitamin E, 12 mg riboflavin, 45 mg niacin, 24 mg calcium pantothenate, 840 mg choline chloride, 30 μg vitamin B_{12}, 200 μg biotin.

[3] Calculated digestible energy

Table III.2 Characteristics of plasma concentrations of growth hormone in sows fed different levels during lactation

	Lactation feed intake[1]			
	High	Low	sed[2]	sig[3]
No. of sows	12	12		
D 2 postpartum				
Mean (ng/ml)	2.62	2.52	0.1	NS
Area under the curve(h. ng/ml)	29.90	31.50	5.9	NS
D 28 postpartum				
Mean (ng/ml)	4.61	5.44	0.9	**
Area under the curve(h. ng/ml)	45.80	52.80	11.5	**

[1]High = 5.5 kg; Low = 3.0 kg.

[2]sed = standard error of the difference

[3]NS = nonsignificant (P>0.05); ** = significant (P<0.01).

Table III.3 Chararcteristics of plasma concentrations of insulin in sows fed different levels during lactation

	Lactation feed intake[1]			
	High	Low	sed[2]	sig[3]
No. of sows	12	12		
D 2 postpartum				
Mean (ng/ml)	5.12	4.83	0.4	NS
Amplitude of peaks (ng/ml)	3.21	4.02	0.6	NS
Area under the curve(h. ng/ml)	28.80	29.70	4.4	NS
D 28 postpartum				
Mean (ng/ml)	7.80	6.90	0.4	*
Amplitude of peaks (ng/ml)	26.20	23.90	3.9	*
Area under the curve(h. ng/ml)	51.60	47.40	7.8	*

[1]High = 5.5 kg; Low = 3.0 kg.

[2]sed = standard error of the difference

[3]NS = nonsignificant (P>0.05); * = significant (P<0.05).

Table III.4 Characteristics of plasma concentration of glucagon in sows fed different levels during lactation.

	Lactation feed intake[1]			
	High	Low	sed[2]	sig[3]
No. of sows	12	12		
D 2 postpartum				
Mean (pg/ml)	191.9	187.2	47.1	NS
Area under the curve (h. pg/ml)	2546.3	2423.1	141.7	NS
D 28 postpartum				
Mean (pg/ml)	227.6	264.8	45.3	NS
Area under the curve (h. pg/ml)	3657.2	4560.4	173.1	*

[1] High = 5.5 kg; Low = 3.0 kg.

[2] sed = standard error of the difference

[3] NS = nonsignificant (P>0.05); * = significant (P<0.05).

Table III.5 Characteristics of plasma concentration of cortisol in sows fed different levels during lactation.

	Lactation feed intake[1]			
	High	Low	sed[2]	sig[3]
No. of sows	12	12		
D 2 postpartum				
Mean (ng/ml)	21.10	21.90	3.3	NS
Area under the curve (h. ng/ml)	316.60	369.90	15.6	NS
D 28 postpartum				
Mean (ng/ml)	21.90	30.80	2.3	**
Area under the curve (h. ng/ml)	452.20	572.30	18.5	**

[1] High = 5.5 kg; Low = 3.0 kg.

[2] sed = standard error of the difference

[3] NS = nonsignificant (P>0.05); ** = significant (P<0.01).

Table III.6 Characteristics of plasma concentration of prolactin in sows fed different levels during lactation.

	Lactation feed intake[1]			
	High	Low	sed[2]	sig[3]
No. of sows	12	12		
D 2 postpartum				
Mean (ng/ml)	106.10	109.90	3.4	NS
Area under the curve (h. ng/ml)	967.80	998.90	13.2	NS
D 28 postpartum				
Mean (ng/ml)	36.20	37.80	1.7	NS
Area under the curve (h. ng/ml)	233.80	287.10	11.5	NS

[1] High = 5.5 kg; Low = 3.0 kg.

[2] sed = standard error of the difference

[3] NS = nonsignificant (P>0.05)

Table III.7. Characteristics of plasma concentrations of follicle stimulating hormone (FSH) in sows fed different levels during lactation.

	Lactation feed intake[1]			
	High	Low	sed[2]	sig[3]
No. of sows	12	12		
D 2 postpartum				
Mean (ng/ml)	0.43	0.42	0.1	NS
Area under the curve(h. ng/ml)	35.30	34.50	5.9	NS
D 28 postpartum				
Mean (ng/ml)	1.61	1.25	1.3	NS
Area under the curve(h. ng/ml)	115.80	92.93	12.4	*

[1] High = 5.5 kg; Low = 3.0 kg;

[2] sed = standard error of the difference.

[3] NS = nonsignificant (P>0.05); * = significant (P<0.05)

Table III.8. Chararcteristics of plasma concentrations of luteinizing hormone (LH) fed different levels during lactation.

	Lactation feed intake[1]			
	High	Low	sed[2]	sig[3]
No. of sows	12	12		
D 28 postpartum				
Mean (ng/ml)	0.65	0.48	0.1	NS
Area under the curve (h. ng/ml)	45.83	32.17	4.8	*

[1] High = 5.5 kg; Low = 3.0 kg;

[2] sem = standard error of the difference.

[3] NS = nonsignificant ($P>0.05$); * = significant ($P<0.05$).

Table III.9. Effect of feed intake during lactation on mean plasma concentration (pg ml^{-1}) of estradiol-17β

	Lactation feed intake[1]			
	High	Low	sed[2]	sig[3]
No. of sows	12	12		
D 2 postpartum	35.8	38.3	2.4	NS
D 28 postpartum	28.6	23.4	4.1	NS

[1] High = 5.5 kg; Low = 3.0 kg

[2] sed = standard error of the difference

[3] NS = nonsignificant (P>0.05)

Table III.10 Effect of lactation feed intake on mean (± sem)[1] plasma LH and FSH characteristics at weaning [2]

	Lactation feed intake		
	High	Low	sig[3]
No. of sows	12	12	
LH			
Frequency of LH pulses (/4 h)	0.75 ± 0.3	0.63 ± 0.3	NS
Time between pulses (min)	45.9 ± 3.4	50.9 ± 4.5	NS
Amplitude attained (ng/ml)	1.35 ± 0.8	1.22 ± 0.9	NS
Mean LH (ng/ml)	1.06 ± 0.1	0.83 ± 0.1	NS
FSH			
Amplitude attained (ng/ml)	1.40 ± 0.4	1.24 ± 0.5	NS
Mean FSH (ng/ml)	1.30 ± 0.06	1.14 ± 0.06	NS

[1] standard error of the mean

[2] First 4 h after weaning

[3] NS = nonsiginificant (P>0.05)

Figure III.1 Mean plasma concentrations of growth hormone on day 2 and day 28 postpartum in sows fed high (■, —) and low (+, Δ) levels of feed during lactation. Arrows denote time of feeding.

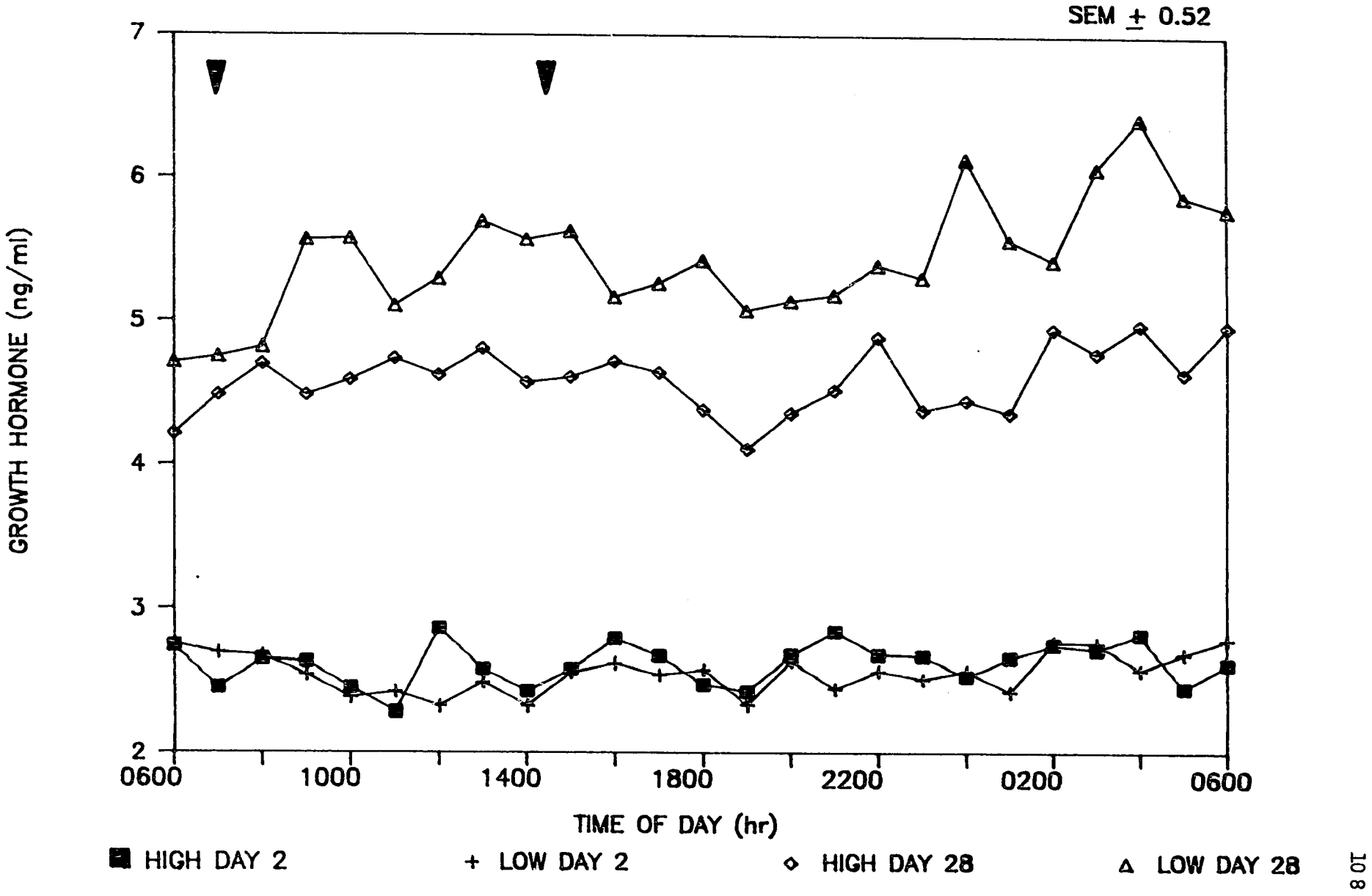

SEM ± 0.52
GROWTH HORMONE (ng/ml)
7
6
5
4
3
2
0600
1000
1400
1800
2200
0200
0600
TIME OF DAY (hr)
HIGH DAY 2
LOW DAY 2
HIGH DAY 28
LOW DAY 28

Figure III.2 Mean plasma concentrations of insulin on day 2 and day 28 postpartum in sows fed high (■, —) and low (+, ▵) levels of feed during lactation. Arrows denote time of feeding.

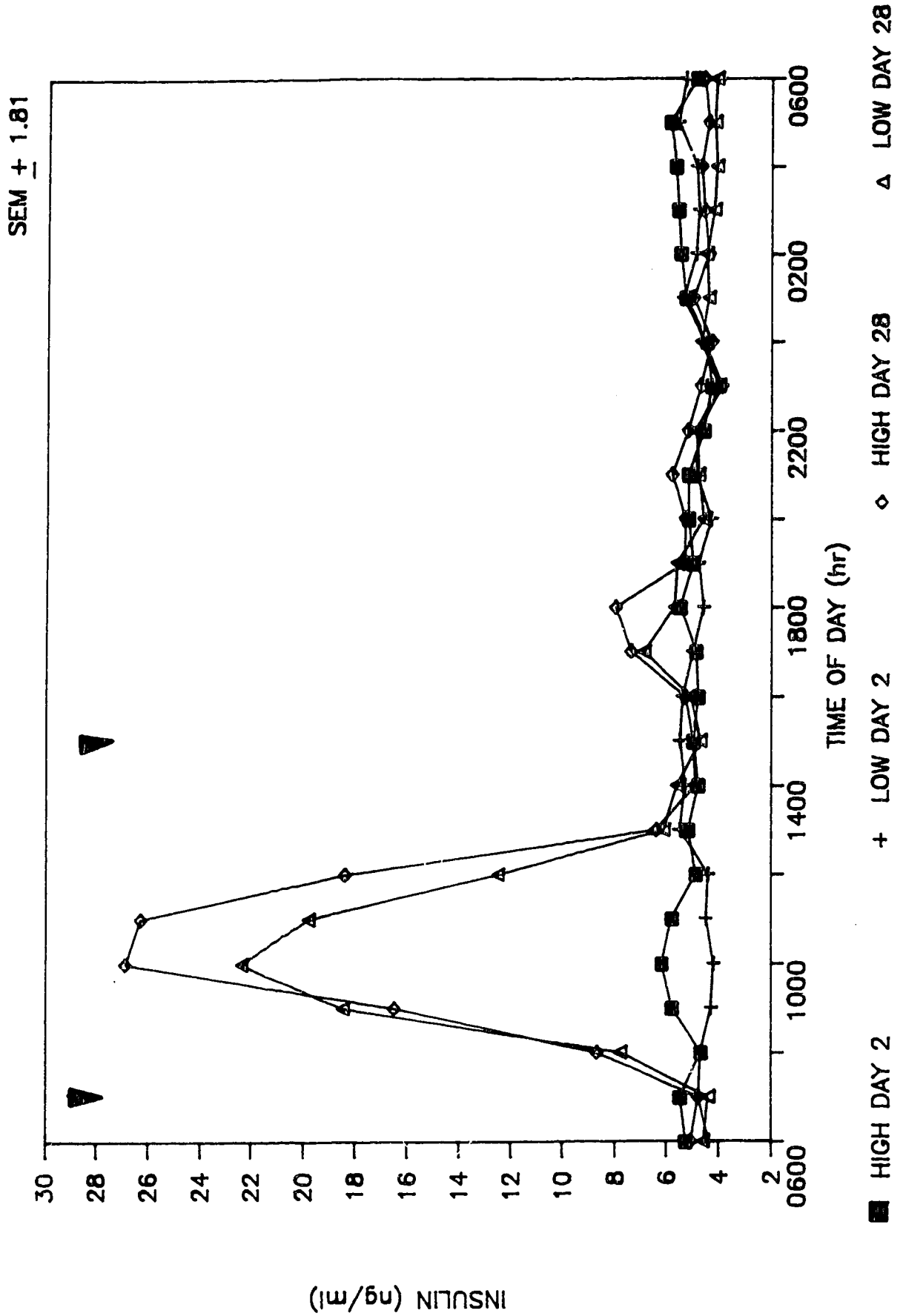

SEM ± 1.81
INSULIN (ng/ml)
30
28
26
24
22
20
18
16
14
12
10
8
6
4
2
0600
1000
1400
1800
2200
0200
0600
TIME OF DAY (hr)
HIGH DAY 2
+ LOW DAY 2
◇ HIGH DAY 28
△ LOW DAY 28

Figure III.3 Mean plasma concentrations of glucagon on day 2 and day 28 postpartum in sows fed high (■, —) and low (+, Δ) levels of feed during lactation. Arrows denote time of feeding.

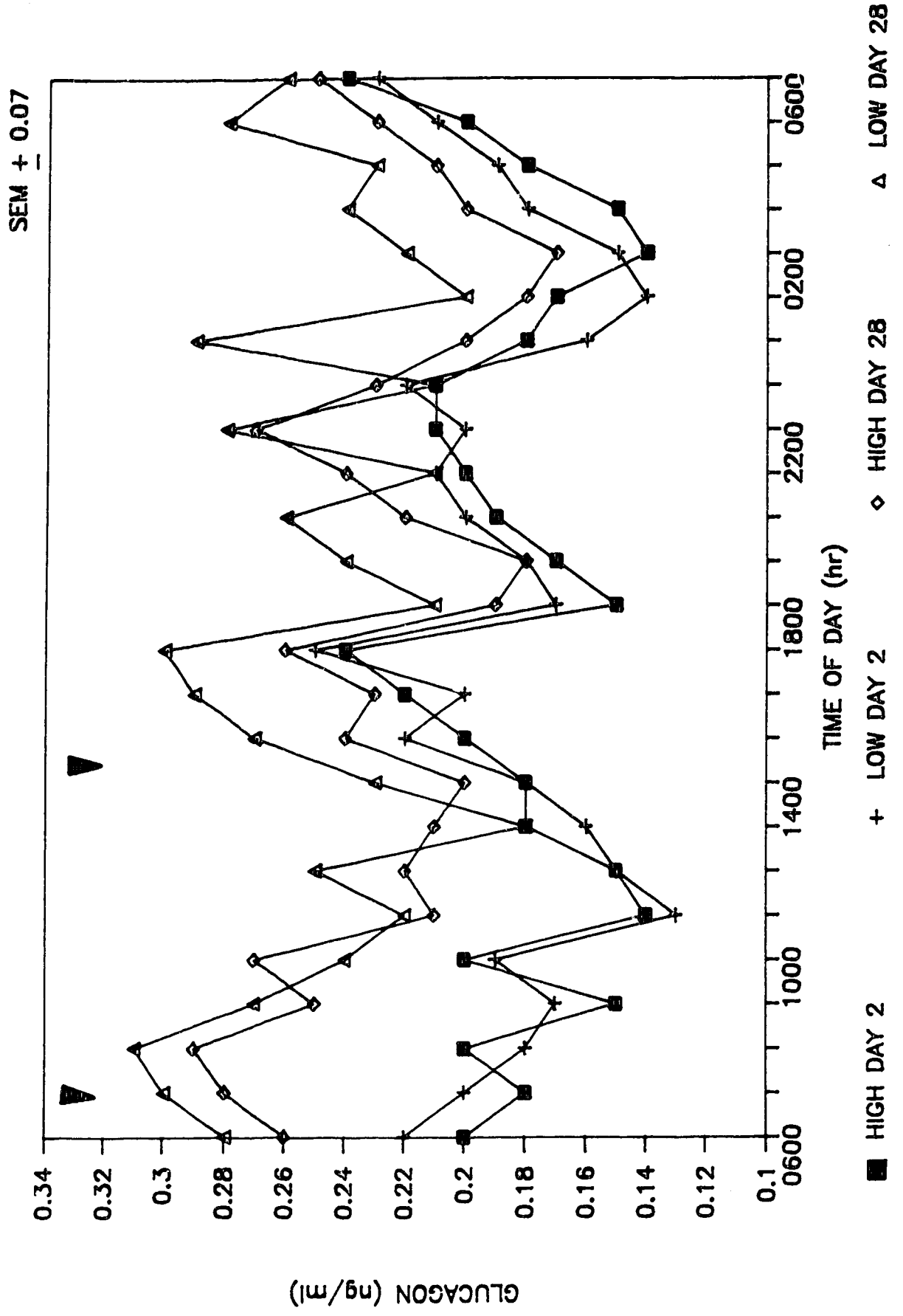

SEM ± 0.07
GLUCAGON (ng/ml)
0.34
0.32
0.3
0.28
0.26
0.24
0.22
0.2
0.18
0.16
0.14
0.12
0.1
0600
1000
1400
1800
2200
0200
0600
TIME OF DAY (hr)
HIGH DAY 2
LOW DAY 2
HIGH DAY 28
LOW DAY 28

Figure III.4 Mean plasma concentrations of cortisol on day 2 and day 28 postpartum in sows fed high (■, —) and low (+, Δ) levels of feed during lactation. Arrows denote time of feeding.

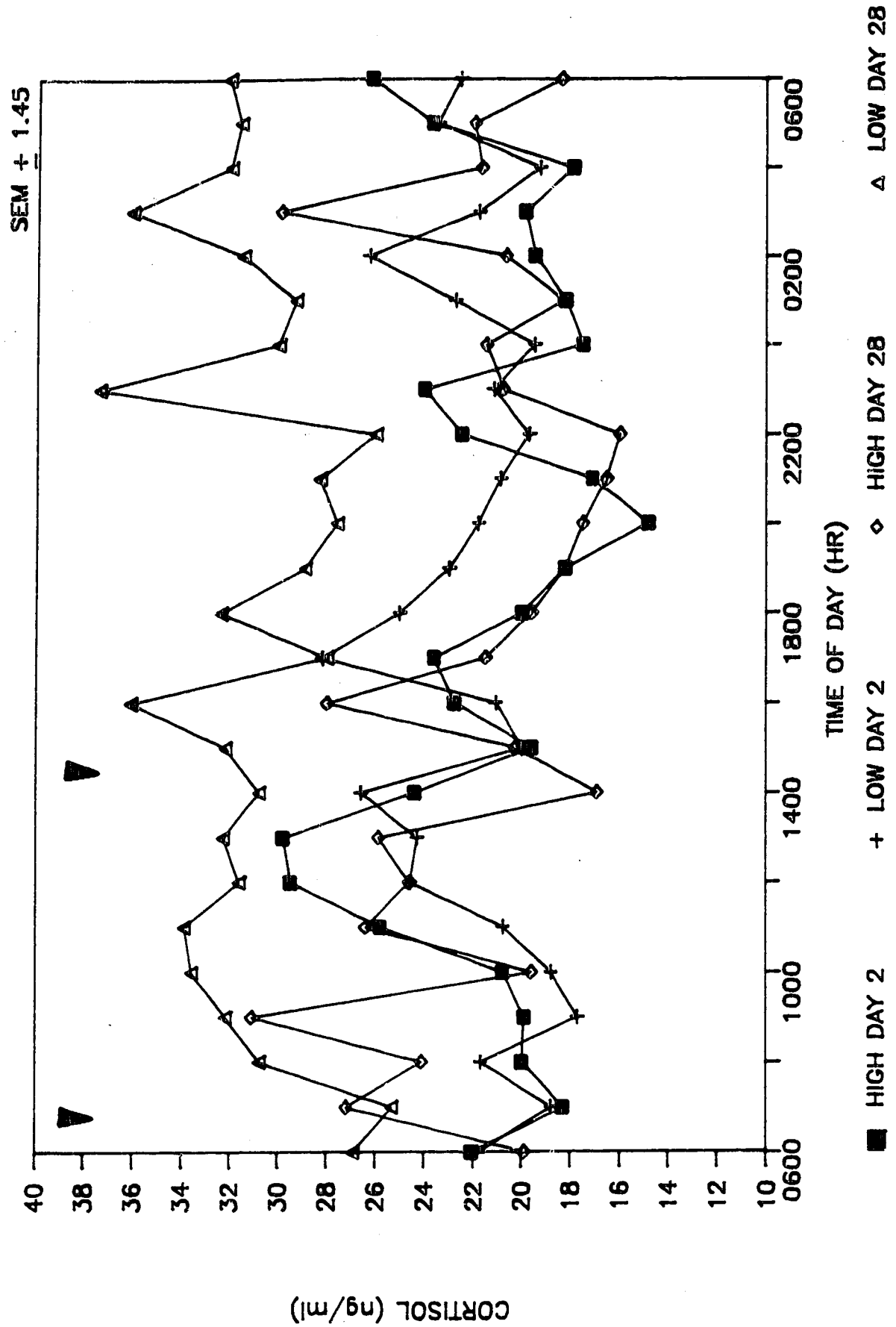

SEM ± 1.45
CORTISOL (ng/ml)
40
38
36
34
32
30
28
26
24
22
20
18
16
14
12
10
0600
1000
1400
1800
2200
0200
0600
TIME OF DAY (HR)
HIGH DAY 2
+ LOW DAY 2
◇ HIGH DAY 28
△ LOW DAY 28

Figure III.5 Mean plasma concentrations of prolactin on day 2 and day 28 postpartum in sows fed high (■, —) and low (+, ∆) levels of feed during lactation. Arrows denote time of feeding.

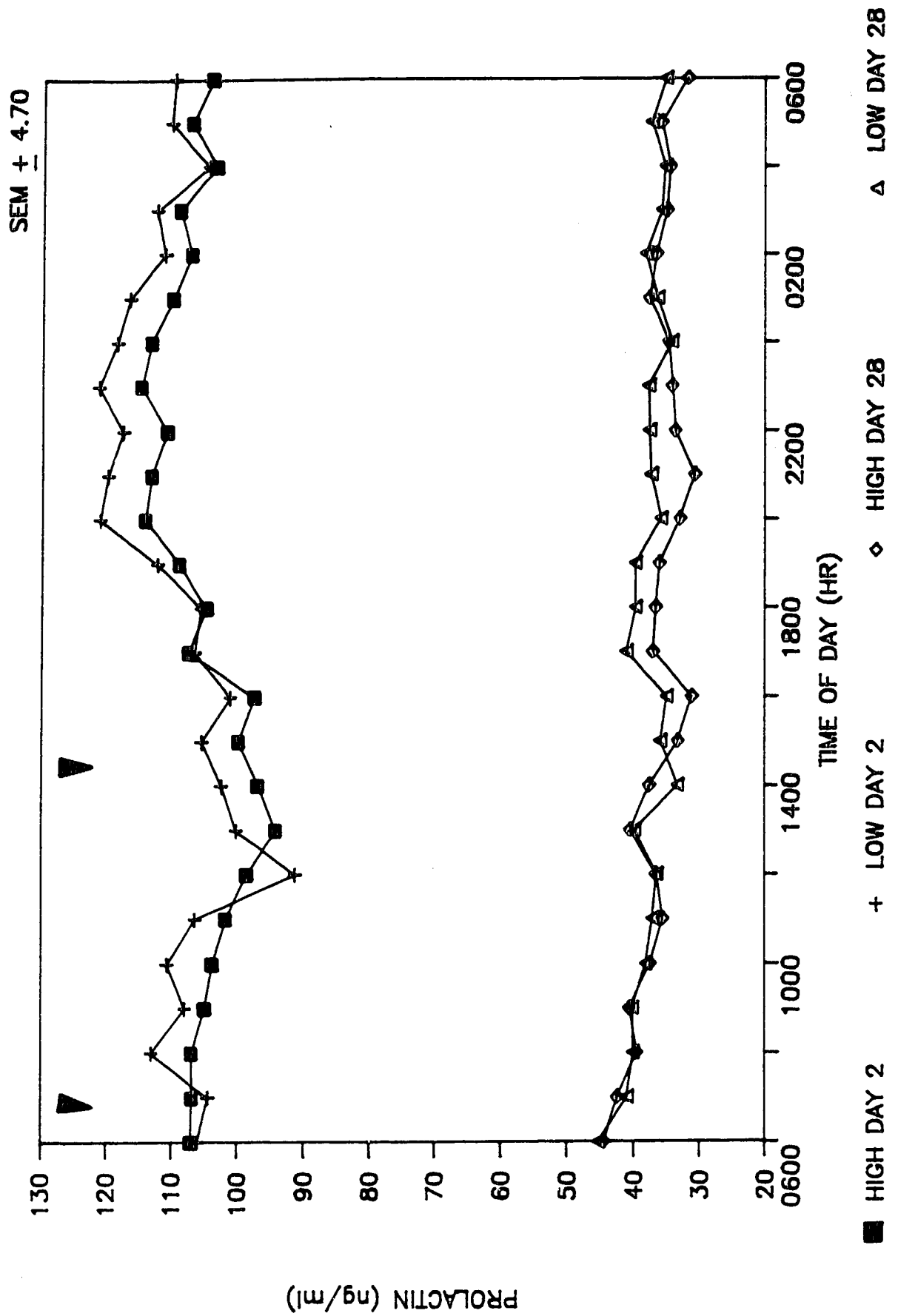

SEM ± 4.70
PROLACTIN (ng/ml)
130
120
110
100
90
80
70
60
50
40
30
20
0600
1000
1400
1800
2200
0200
0600
TIME OF DAY (HR)
HIGH DAY 2
LOW DAY 2
HIGH DAY 28
LOW DAY 28

Figure III.6 Mean plasma concentrations of FSH on day 2 and day 28 post partum in sows fed high (■, —) and low (+, ∆) levels of feed during lactation.

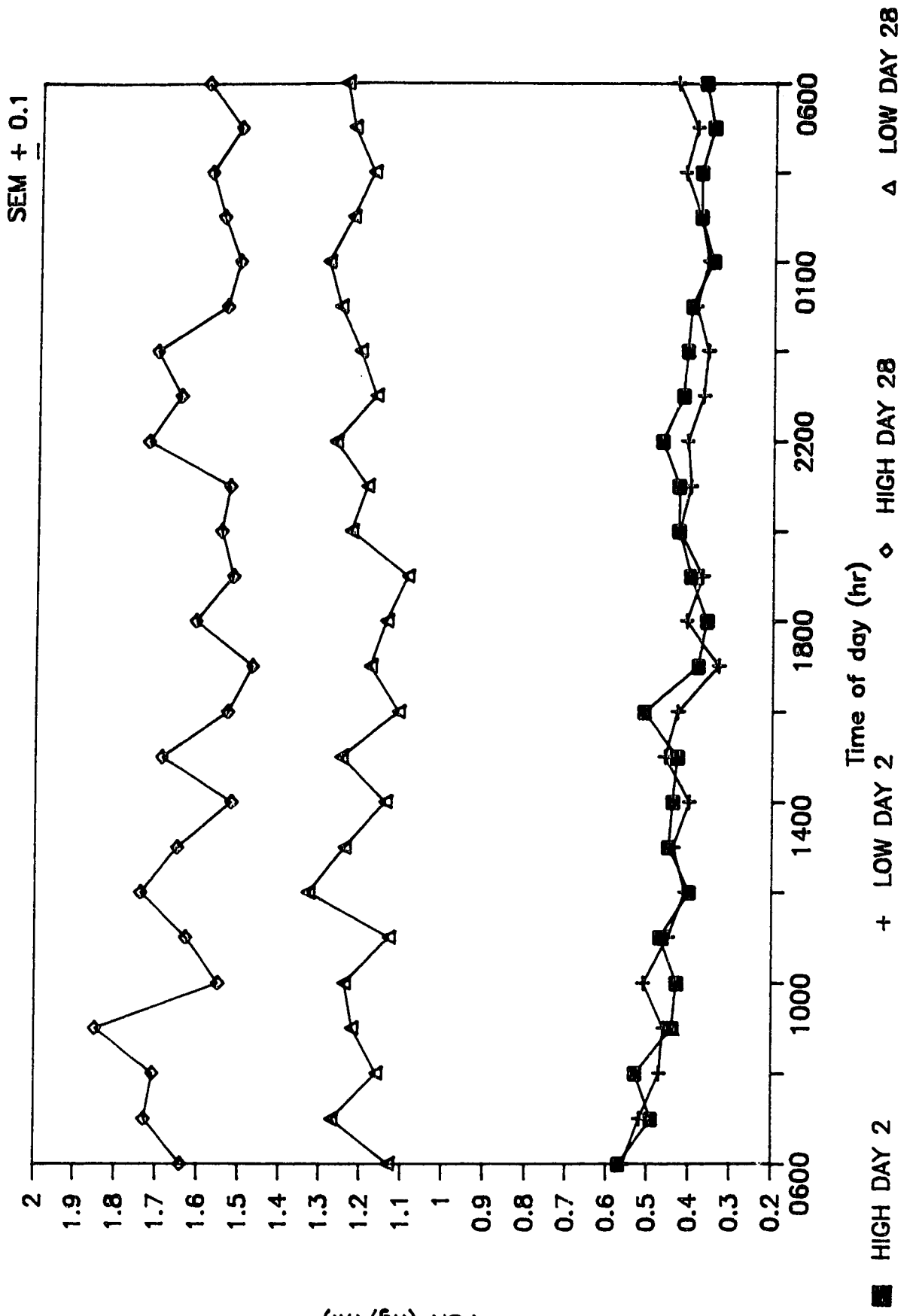

SEM + 0.1
FSH (ng/ml)
2
1.9
1.8
1.7
1.6
1.5
1.4
1.3
1.2
1.1
1
0.9
0.8
0.7
0.6
0.5
0.4
0.3
0.2
0600
1000
1400
1800
2200
0100
0600
Time of day (hr)
HIGH DAY 2
LOW DAY 2
HIGH DAY 28
LOW DAY 28

Figure III.7 Mean plasma concentrations of LH on day 2 and day 28 post partum in sows fed high (■, —) and low (+, ∆) levels of feed during lactation.

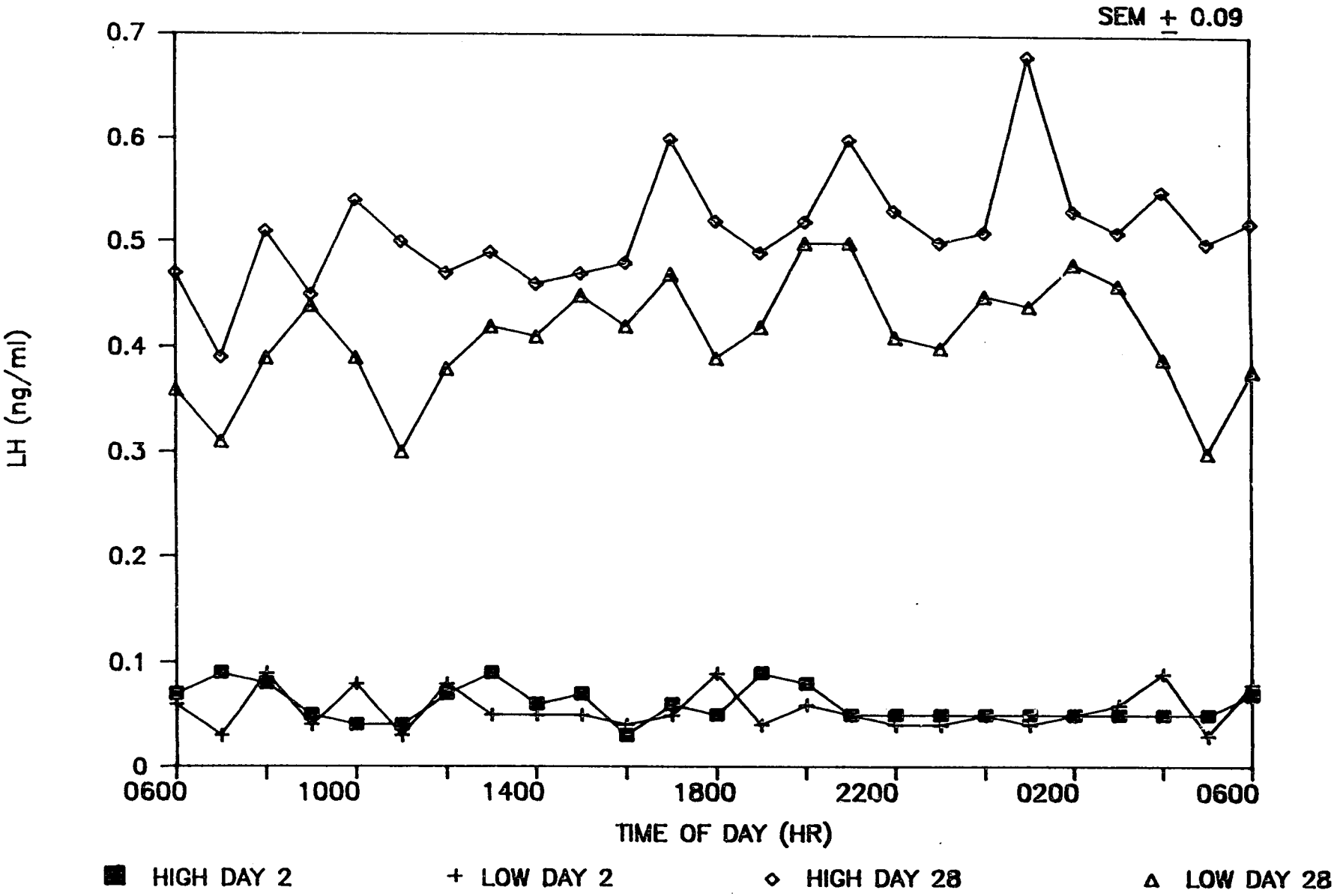

SEM ± 0.09
0.7
0.6
0.5
0.4
0.3
0.2
0.1
0
LH (ng/ml)
0600
1000
1400
1800
2200
0200
0600
TIME OF DAY (HR)
HIGH DAY 2
LOW DAY 2
HIGH DAY 28
LOW DAY 28

Figure III.8 Mean plasma LH concentrations obtained from frequent samples taken every 15 min for 4 h immediately after weaning from 0800 to 1200. Mean plasma profile of sows with corpora lutea (C.L.), (■, —) and sows with no C.L. (+, ▵) at slaughter 25 d postcoitum.

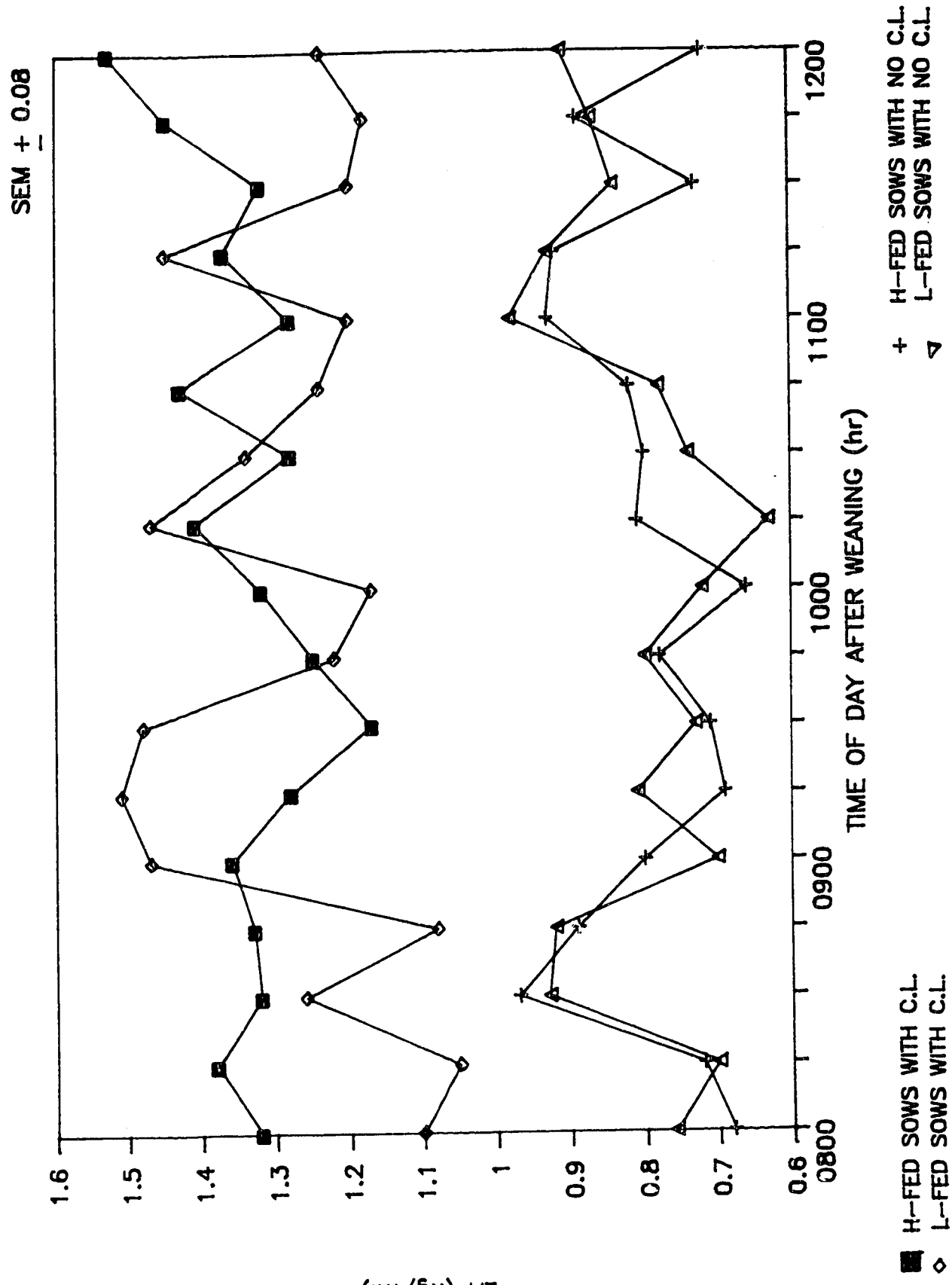
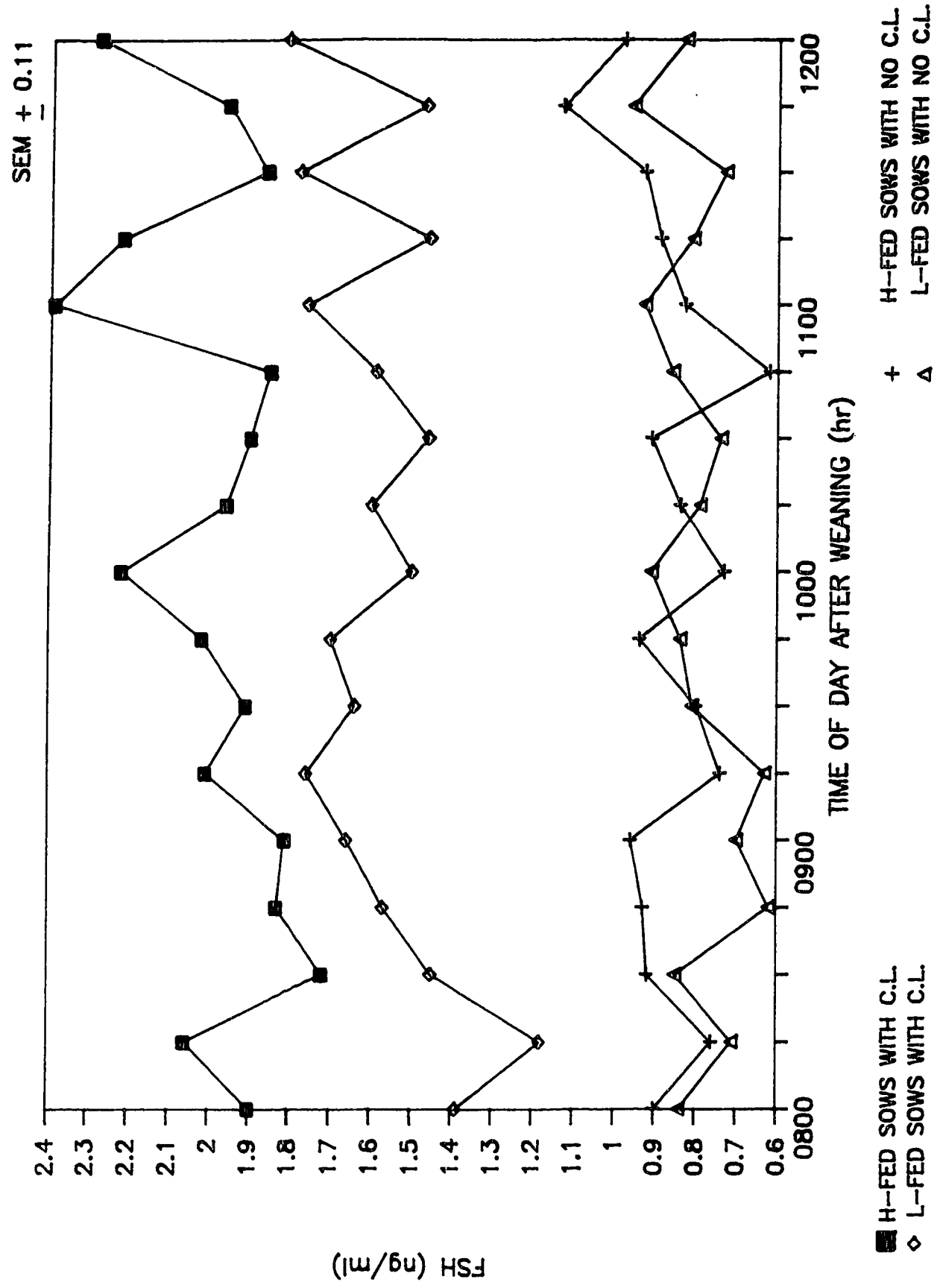

SEM ± 0.08
LH (ng/ml)
1.6
1.5
1.4
1.3
1.2
1.1
1
0.9
0.8
0.7
0.6
0800
0900
1000
1100
1200
TIME OF DAY AFTER WEANING (hr)
H-FED SOWS WITH C.L.
L-FED SOWS WITH C.L.
H-FED SOWS WITH NO C.L.
L-FED SOWS WITH NO C.L.

Figure III.9 Mean plasma FSH concentrations obtained from frequent samples taken every 15 min for 4 h immediately after weaning from 0800 to 1200. Mean plasma profile of sows with corpora lutea (C.L.), (■, —) and sows with no C.L. (+, ▵) at slaughter 25 d postcoitum.


SEM ± 0.11
FSH (ng/ml)
2.4
2.3
2.2
2.1
2
1.9
1.8
1.7
1.6
1.5
1.4
1.3
1.2
1.1
1
0.9
0.8
0.7
0.6
0800
0900
1000
1100
1200
TIME OF DAY AFTER WEANING (hr)
H-FED SOWS WITH C.L.
L-FED SOWS WITH C.L.
H-FED SOWS WITH NO C.L.
L-FED SOWS WITH NO C.L.

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IV. THE INFLUENCE OF FEEDING LEVEL DURING LACTATION AND BETWEEN WEANING AND MATING ON THE OCCURRENCE AND ENDOCRINOLOGY OF THE POSTWEANING ESTRUS IN SOWS

A. INTRODUCTION

The most frequent reason for culling sows is infertility, such as failure to rebreed promptly after weaning. A variety of factors known to affect the weaning to mating interval have been described (Reese et al., 1984). Energy and (or) protein intake resulting in excessive weight or condition loss during lactation affect rebreeding performance (Reese et al., 1984; King and Williams, 1984a,b; Lythgoe, 1987). The adverse effect of condition loss on the duration of the calving to estrus interval has also been reported in cattle (Rutter and Randel, 1984; Imakawa et al., 1984) and sheep (Rhind and McNeilly, 1986).

The physiological mechanisms whereby change in body condition during lactation affects the remating interval in the pig are unknown. Studies with cattle have demonstrated a negative correlation between basal plasma LH during the remating interval and weight loss (Rutter and Randel, 1984; Wright et al., 1987). Similar conditions occur in sheep (Foster and Olster, 1985; Rhind and McNeilly, 1986). The available information on the influence of postweaning nutrition on the weaning to estrus interval in the pig is inconclusive. Increasing the level of feeding during the postweaning period has been reported to shorten the weaning to estrus interval in primiparous sows (Brooks and Cole, 1972), but not in multiparous sows, and to increase the number exhibiting estrus within 10 days of weaning (Fahmy and Dufour, 1976). Recent reports failed to show benefits of increasing feed intake for reducing the weaning to estrus interval (Tribble and Orr, 1982; Johnston et al., 1986). Because of such contradictory reports there is a need for basic information on the mechanisms mediating the influence of postweaning feed level on the reproductive performance of sows which have had different nutritional regimens during lactation.

There is a general agreement that weight loss during lactation has no effect on ovulation rate (Pike and Boaz, 1972; King et al., 1982; King and Williams, 1984a,b; King and

Dunkin, 1986a,b). However, the evidence for an effect of lactation and postweaning feed intake on embryo survival is not clear. King and Williams (1984a) reported no effect of lactation feed intake on subsequent embryo survival and Reese et al. (1982) reported no effect on subsequent litter size. In contrast Hughes et al. (1984) reported that low feed intake during lactation will reduce subsequent embryo survival. It is apparent that the influence of lactation and postweaning feed intake on subsequent embryo survival requires further investigation. Changes in plasma concentration of gonadotropic hormones and estradiol-17β during the weaning to estrus interval have been reported (Edwards and Foxcroft, 1983a,b; Cox and Britt, 1986). However, there are no data concerning the effect of lactation and postweaning feed intake on the hormonal status of the sow during the weaning to mating interval.

The purpose of this study was therefore to determine the effects of high or low levels of feed intake during lactation and the postweaning period on the length of the remating interval, ovulation rate, conception rate and embryo survival. The endocrine status of sows on different lactation and postweaning nutritional regimens was also studied.

B. MATERIALS AND METHODS

Animal Management

Eighty-nine second parity Lacombe sows were fed either 3 kg (L) or 6 kg (H) of a diet containing 16% crude protein and 12.4 MJ ME kg^{-1} (Table IV.1) daily during a 28 d lactation. Water was available ad libitum at all times during the experiment. Litter size differences were minimized by cross-fostering within d 2 of farrowing. At weaning, sows were randomly allotted within lactation treatments to either a restricted (3kg, L) feeding regimen or fed to appetite (H) (Figure IV .1).

Sows were moved to pens containing a boar to check for estrus on daily basis. On d 1 and d 2 postweaning, boars were introduced to sows on a daily basis to check for estrus; from d 3 sows were introduced individually to boar pens and the day of return to estrus was defined as the time at which sows first stood to be mated; sows were bred a second time between 8

and 24 h after first service.

Blood Sampling

On the 27 d postpartum, 24 H and 24 L (12 H-H, 12 H-L, 12 L-H, and 12 L-L) fed sows were fitted, without use of anesthetic, with indwelling polyethylene vena cava cannulae (Polyvinyl chloride tubing, SV. 61, Dural Plastics, Dural, N.S.W. Australia) inserted via a prominent ear vein while the sow was restrained with a rope snare. The cannula was attached to the animal's ear and back with tag cement and adhesive tape. The lumen of the cannula was kept patent with heparin saline solution (60 IU heparin ml^{-1} sterile, isotonic saline). The cannula was secured and protected on the outside of the neck by a 7.62 cm wide elastic bandage wrapped around the neck and shoulder area. In all these sows, blood samples were collected every 6 h, commencing at 0600 h on the second day (d 1) after weaning (d 0) until 24 h post insemination or d 10 postweaning, which ever came first. All blood samples were collected into glass tubes with heparin (10 IU ml^{-1} blood) and kept at 4 °C for no more than 6 h until centrifugation at 3000 x g for 15 min at 4 °C. Plasma samples were then frozen and stored at -20 °C until assayed for estradiol-17β, LH and FSH by radioimmunoassays.

Radioimmunoassays

Plasma estradiol-17β concentration was measured by radioimmunoassay using rabbit antiserum against estradiol-17β (E-26-47, Endocrine Sciences, Tarzana, CA) as described previously (Chapter III). Intraassay and interassay coefficients of variation were 5.9% and 12.9% respectively. Porcine LH and FSH concentrations were determined as described in Chapter III using rabbit-antibovine-LH and antiserum specific for pFSH, respectively. The intraassay and interassay coefficients of variation for LH and FSH were 3.2% and 5.1% and 7.2% and 8.1% respectively.

Reproductive Tract Dissection

Sows that did not show estrus by d 10 postweaning were monitored for another 25 d before slaughter. All other sows were slaughtered at d 25 post-insemination and the reproductive tracts removed from each sow. The cornua of the uterus were separated after removing the mesometrium. Each cornua was opened by means of a cut along the mesometrial surface and the tissue separated with a forceps. The number and viability of each embryo was determined. Days to estrus, ovulation rate (determined by the number of copora lutea) and number of viable embryos were recorded.

Statistical Analysis

The experimental design is presented in Figure IV.1. Sow performance data were subjected to a two way analysis of variance. Lactation and rebreeding diets were the two effects for analysis of rebreeding data used in the split plot design (Steel and Torrie, 1980). During the postweaning period, sows were divided into four treatment groups (H-H, H-L, L-H, L-L) on the basis of lactation and postweaning feed intake. Orthogonal contrasts were used to compare lactation diets (H-H + H-L vs L-H + L-L). Likewise, postweaning feed intake were compared using the orthogonal comparison (L-H + H-H vs L-L + H-L). Chi-square analysis (Steel and Torrie, 1980) was used to test treatment effects for the following dependent variables: proportion in estrus, conception rate, ovulation rate and embryonic survival. The differences of the means were evaluated by two way analysis of variance and differences evaluated by Student-Newman-Keuls' test (Steel and Torrie, 1980).

Data from hormone analysis were subjected to split plot analysis of variance (Steel and Torrie, 1980). Mean hormonal level was determined for LH, FSH and estradiol-17β by obtaining the average of all the concentrations during the entire period of collection of samples.

C. RESULTS

Sow Performance

Low level feeding in lactation resulted in significant body weight loss and backfat loss ($P<0.01$) as indcated in Chapter II. The performance of sows during the postweaning period is shown in Table IV.2. Sows fed H during the postweaning period consumed ($P<0.05$)more than the sows fed low. The average weight change in sows during the postweaning period was not significantly different ($P>0.05$) between treatments. The results indicate that lactation feed intake markedly influenced sow weight change, however, the feed intake during the postweaning period had little influence on weight change during the weaning to remating interval.

Severe sow weight loss during lactation resulted in an extension ($P<0.05$) of the weaning to estrus interval as presented in Figure IV.2 and Table IV.2. A higher number ($P<0.05$) of sows fed the H-H and H-L (21 and 19 respectively) returned to estrus within 10 days after weaning compared with sows fed the L-H and L-L fed regimes (8 and 6 respectively). Of those sows that returned to estrus within 10 days postweaning there was no significant difference ($P>0.05$) in mean interval weaning to estrus, however, there were differences ($P<0.05$) by d 26 postweaning (Table IV.2). High level of feeding during the postweaning period did not significantly ($P>0.05$) shorten the weaning to estrus interval.

Postweaning feed intake had no effect ($P>0.05$) on ovulation rate at the postweaning estrus. However, lactation feed intake influenced ($P<0.05$) pregnancy rate (Table IV.2) with sows fed H-H and H-L having higher pregnancy rates. Embryo survival was influenced ($P<0.05$) by feed intake. The sows fed high during lactation and low during the postweaning period had higher ($P<0.05$) embryo survival rate than the other treatments. Low feed intake during lactation reduced ($P<0.05$) embryo survival.

Hormonal Profiles During Postweaning Period

Estradiol-17β

The level of feed intake during lactation and (or) during the postweaning period did not ($P>0.05$) influence the overall mean plasma levels of estradiol-17β (Table IV.3). However, lactation feed intake influenced ($P<0.05$) the number of sows with peak levels of estradiol-17β by d 10 postweaning (Table IV.4). Overall mean peak estradiol-17β levels attained between treatments were not significantly different ($P>0.05$) (Table IV.4 and Figure IV.3). The number of sows (3, 2, 5, 6 for H-H, H-L, L-H and L-L respectively) that did not show any sign of ovulation at slaughter, had significant ($P<0.01$) low levels of estradiol-17β compared with sows that ovulated (Figure IV.4).

Gonadotropins

The level of feed intake during lactation influenced the overall mean plasma levels of FSH ($P<0.05$) and LH ($P<0.01$) during the postweaning period (Table IV.3). Feed intake during the postweaning period did not influence ($P>0.05$) the overall mean plasma levels of the gonadotropins, LH and FSH (Table IV.3).

The mean interval from weaning to peak LH surge levels was significantly different ($P<0.05$) between the lactation treatment groups (Table IV.4): with a shorter time to the postweaning LH surge in H-L compared to the L-L fed sows (106.2 h vs 160 h). The postweaning feed levels did not significantly ($P>0.05$) influence the time to preovulatory surge of LH. Neither lactation feed level nor postweaning feed level significantly ($P>0.05$) influenced the time to preovulatory FSH surge. The mean peak LH concentrations for H-H, H-L, L-H and L-L fed sows were 9.2 $\pm$ 0.9, 9.5 $\pm$ 0.8, 8.1 $\pm$ 0.9 and 7.6 $\pm$ 0.6 ng ml^{-1}, respectively. Lactation feed intake had a significant ($P<0.05$) effect on peak LH concentrations and total amount of LH secreted during the preovulatory surge (Table IV.4). The LH secretion in sows which demonstrated a LH surge, returned to basal levels after the surge. Lactation feed and (or) postweaning feed level did not influence ($P>0.05$) the peak FSH attained nor the amount of FSH secreted

(Table IV.4). The LH surge and FSH surge durations were not significantly ($P>0.05$) influenced by lactation and (or) the postweaning feed levels (Table IV.4).

Comparison between sows that ovulated and sows that did not ovulate showed that the plasma concentrations of LH and FSH were higher ($P<0.01$) in sows that ovulated (Figures IV.5 and IV.6). Plasma concentrations of the gonadotropins in the sows that did not ovulate were at basal levels.

D. DISCUSSION

The postweaning estrus, which is the most important event in rebreeding the sow postpartum, was influenced by lactation feed intake as indicated in Chapter II. Postweaning feed intake did not affect the weaning do estrus interval as reported in other studies (Fahmy and Dufour, 1976; Den Hartog and Van Der Steen, 1981; Tribble and Orr, 1982). The L-H fed sows did not respond to high levels of feed during the postweaning period. Sows fed high during lactation took a shorter time to return to estrus than the low fed sows.

Relationships between changes in plasma estradiol-17β, LH and FSH after weaning were similar to those in previous studies (Stevenson et al., 1978; Edwards and Foxcroft, 1983a,b; Shaw and Foxcroft, 1985; Cox and Britt, 1986) and concentration around estrus were not different from those reported for cycling sows (Edwards and Foxcroft, 1983a; Shaw and Foxcroft, 1985). However, the absolute amount of LH released during the preovulatory surge after weaning was significantly reduced in low fed sows compared to the high fed sows during lactation. This result is similar to the observation by Edwards and Foxcroft (1983a,b) which showed the absolute amount of LH released during the preovulatory surge after 21 d of lactation was significantly reduced in comparison to surge in LH release in 35 d weaned or cyclic sows at oestrus. Thus early weaned sows may have the same endocrinological status in relation to reproductive behavior such as return to estrus similar to sows that have lost body condition during lactation as was indicated by Kirkwood et al. (1987). The physiological basis for this observation is not clear, but could involve reduced pituitary sensitivity to endogenous GnRH and (or) an actual decrease in endogenous GnRH release by the hypothalamus.

The ovulation rate was similar in both high and low-fed sows. All sows showing signs of estrus in both the high and low-fed sows ovulated. At slaughter, it was found that six sows which did not show any signs of estrus, had ovulated. Later plasma analysis of LH, FSH and estradiol-17β indicated there was a surge of LH and normal estradiol-17β concentration. There was a lack of FSH surge in these sows. It could therefore be suggested that the preovulatory FSH surge may not be of any physiological significance in considering the process of ovulation. Thus, it seems that the most important endocrine event during the postweaning period is secretion of LH. However, Shaw and Foxcroft (1985) reported a significant positive correlation between FSH levels after weaning and ovulation rate and this raises the possibilty that FSH may be involved in determining the final munber of follicles within the ovulatory population. Plasma estradiol-17β concentrations were not influenced by feed intake during the postweaning period in sows that returned to estrus. However, the attainment of estradiol-17β preovulatory rise took a longer time to occur in the low fed sows compared to the high fed sows.

Pregnancy rate was influenced by feed intake. Sows fed high during lactation had a higher pregnancy rate. Embryo mortality was highest for the L-L fed sows. The H-L fed sows had the highest rate of embryo survival. The reasons for this are speculative and were discussed by Aherne and Kirkwood (1985): one suggestion was that low levels of LH during the preovulatory LH surge may influence luteinization of the copora lutea thus resulting in decrease in plasma progesterone levels in early pregnancy and consequent adverse effect on uterine secretions and embryo survival. A primary role of the corpus luteum is to synthesize and secrete progesterone, which is essential for the maintenance of pregnancy. Progesterone synthesis and secretion are requlated by luteotrophic and luteolytic mechanisms (Smith, 1986). Luteinizing hormone stimulates progesterone synthesis (Thanki and Channing, 1978) and thus can prolong luteal lifespan (Donaldson and Hansel, 1965). Follicle stimulating hormone stimulated progesterone secretion in pig granulosa cells (Smith, 1986). These results suggest that both FSH and LH may have a luteotropic role in luteal function. Since FSH and LH concentrations were high in sows fed high during lactation, this could account for the high

incidence of pregnancy in sows fed high levels of feed during lactation. Plasma progesterone concentrations were not determined in this study, therefore this suggestion requires further investigation.

Comparison between the hormonal profiles of sows that ovulated and did not ovulate indicated a quiescent reproductive system in the sows that did not ovulate in terms of hormonal profile. This occurrence were observed in H-H, H-L, L-H and L-L fed sows during the weaning to estrus interval. There were low levels of FSH, estradiol-17β and LH in plasma of sows that did not ovulate. The reason for this observation is not clear, however, Almond et al. (1986) suggested that the ovaries of the postweaning anoestrous sows exert sufficient inhibitory feed back through an unknown mechanism to inhibit normal hypothalamo-hypophyseal axis control of follicular development of the ovaries. However, it could be due to lack of synthesis of GnRH since hourly pulses of GnRH stimulated estrus and ovulation in anestrus sows (Armstrong and Britt, 1985).

In conclusion, excessive weight and (or) backfat loss during lactation will adversely affect sow reproductive performance by extending the remating interval and increase the rate of embryo mortality. The extension of the remating interval appeared to be related to LH secretion after weaning. Lactation feed intake had a profound effect on the occurrence of estrus during the postweaning period, since the high fed sows during lactation had elevated circulatory levels of plasma LH than low fed sows during lactation. The postweaning feed intake had no significant effect on incidence of estrus after weaning. It appears that body condition at weaning is more important than the postweaning feed intake in determining the length of the weaning to estrus interval.

Table IV.1 Formulation and composition of the diet.

Ingredients	% Diet
Barley	57.5
Wheat	23.4
Soybean meal (46 %CP[1])	13.1
Calcium phosphate	1.5
Limestone	1.0
Vitamin-mineral premix[2]	1.0
Iodized salt	0.5
Tallow	2.0
Chemical analyses	
Dry matter (%)	89.3
Crude protein (%)	16.4
Lysine (%)	0.72
Digestible energy ($MJ.kg^{-1}$)[3]	12.4

[1] CP = Crude protein

[2] Supplied the following per kg of diet: 120 mg zinc, 12 mg manganese, 150 mg iron, 12 mg copper, 0.1 mg selenium, 5000 IU vitamin A, 500 IU vitamin D_3, 22 IU vitamin E, 12 mg riboflavin, 45 mg niacin, 24 mg calcium pantothenate, 840 mg choline chloride, 30 μg vitamin B_{12}, 200 μg biotin.

[3] Calculated digestible energy

Table IV.2 Effect of lactation and postweaning feed intake on reproductive performance of sows.

	Level				
Lactation	High		Low		
Postweaning	High	Low	High	Low	sed[1]
No. of sows	23	22	22	22	
Feed intake (kg d^{-1})[2]	4.7[a]	3.0[b]	5.2[a]	3.0[b]	0.6
Lactation weight loss (kg)	16.3[a]	15.9[b]	38.9[a]	39.2[b]	2.3
Lactation backfat loss (mm)	2.1[a]	2.4[b]	6.2[a]	6.6[b]	0.6
Weight change remating interval (kg)	2.2	0.9	3.4	1.4	0.7
Mean interval weaning to estrus (days)	4.6[c]	5.5[c]	8.5[d]	10.5[d]	1.8
Pregnancy rate (%)[3]	86.9[c]	82.0[c]	69.0[d]	62.0[d]	3.1
Ovulation rate [4]	16.6	16.2	17.7	16.7	0.9
Embryo survival (%)[5]	77.7[cd]	85.0[c]	70.4[d]	64.0[e]	8.2

[1] sed = standard error of the difference

[2] [a,b] Means with different superscripts differ ($P<0.05$);
[c,d,e] Means with different superscripts differ ($P<0.01$)

[3] Percent of sows with embryos on d 25 postcoitum.

[4] Number of corpora lutea determined on d 25 postcoitum

[5] Percent survival = embryos/corpora lutea, determined at 25 d postcoitum.

Table IV.3. Mean (± sem)[1] LH, FSH and estradiol-17β concentrations of sows during the postweaning period.

Treatment	No. of sows	FSH (ng/ml)	LH (ng/ml)	Estradiol (pg/ml)
HH	12	1.58 ± 0.06	2.31 ± 0.15	43.72 ± 6.1
HL	12	1.47 ± 0.11	2.33 ± 0.15	41.50 ± 4.7
LH	12	1.24 ± 0.12	1.51 ± 0.16	37.50 ± 4.8
LL	12	1.04 ± 0.15	1.47 ± 0.23	38.44 ± 5.0
Sig[2]		*	**	NS

[1] sem = standard error of the mean

[2] ** = significant ($P<0.01$); * = significant ($P<0.05$); NS = nonsignificant ($P>0.05$)

Table IV.4. Effect of lactation and postweaning feed intake on the mean estradiol-17β induced LH and FSH surge characteristics, in sows during the postweaning estrus.

	Feed Intake[1]				
Lactation	High		Low		
Post Weaning	High	Low	High	Low	sed[2]
No. in estrus/ No. of sows	9 / 12	10 / 12	7 / 12	6 / 12	
Peak estradiol pre-ovulatory level (pg/ml)	85.2	71.6	73.7	72.9	6.9
Time to LH surge (h)	108.0[a]	106.2[a]	147.4[b]	160.0[b]	10.7
Time to FSH surge (h)	104.6	102.0	132.0	151.0	10.2
Peak LH level attained (ng/ml)	9.2	9.5	8.1	7.6	0.6
Area under the LH curve (h. ng/ml)	3080[a]	3175[a]	2711[b]	2544[b]	169.1
Peak FSH level attained (ng/ml)	2.9	3.2	2.7	2.8	0.1
Area under the FSH curve (h. ng/ml)	924.3	1020.2	860.5	892.5	42.3
LH surge duration (h)	44.7	46.2	44.2	44.0	2.1
FSH surge duration (h)	39.3	48.0	40.3	41.0	2.8

[1] High = 6 kg; Low = 3 kg

[2] sed = standard error of the difference

[ab] Means with different superscripts differ ($P<0.05$)

Figure IV.1 Feeding regimens of sows during the lactation and postweaning periods

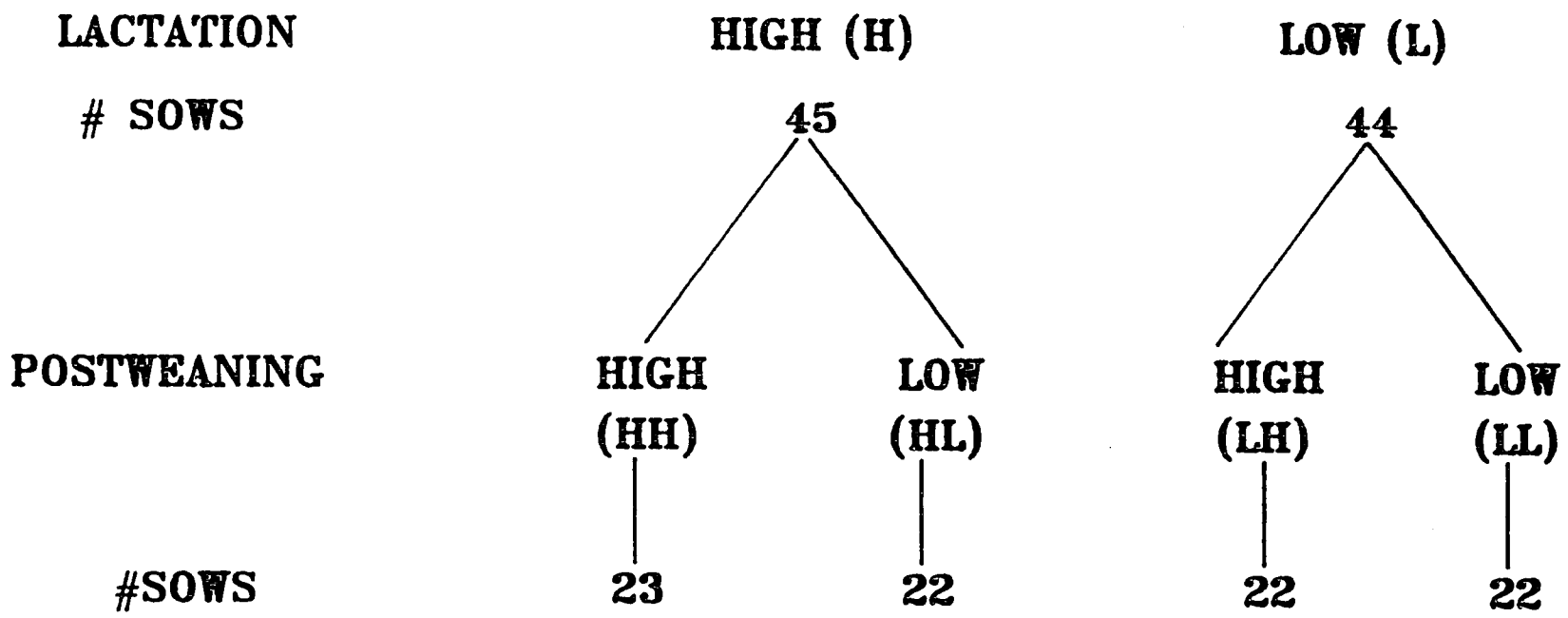

LACTATION
HIGH (H)
LOW (L)
SOWS
45
44
POSTWEANING
HIGH (HH)
LOW (HL)
HIGH (LH)
LOW (LL)
#SOWS
23
22
22
22

Figure IV.2 Effect of lactation and postweaning feed intake on the weaning to estrus interval in sows

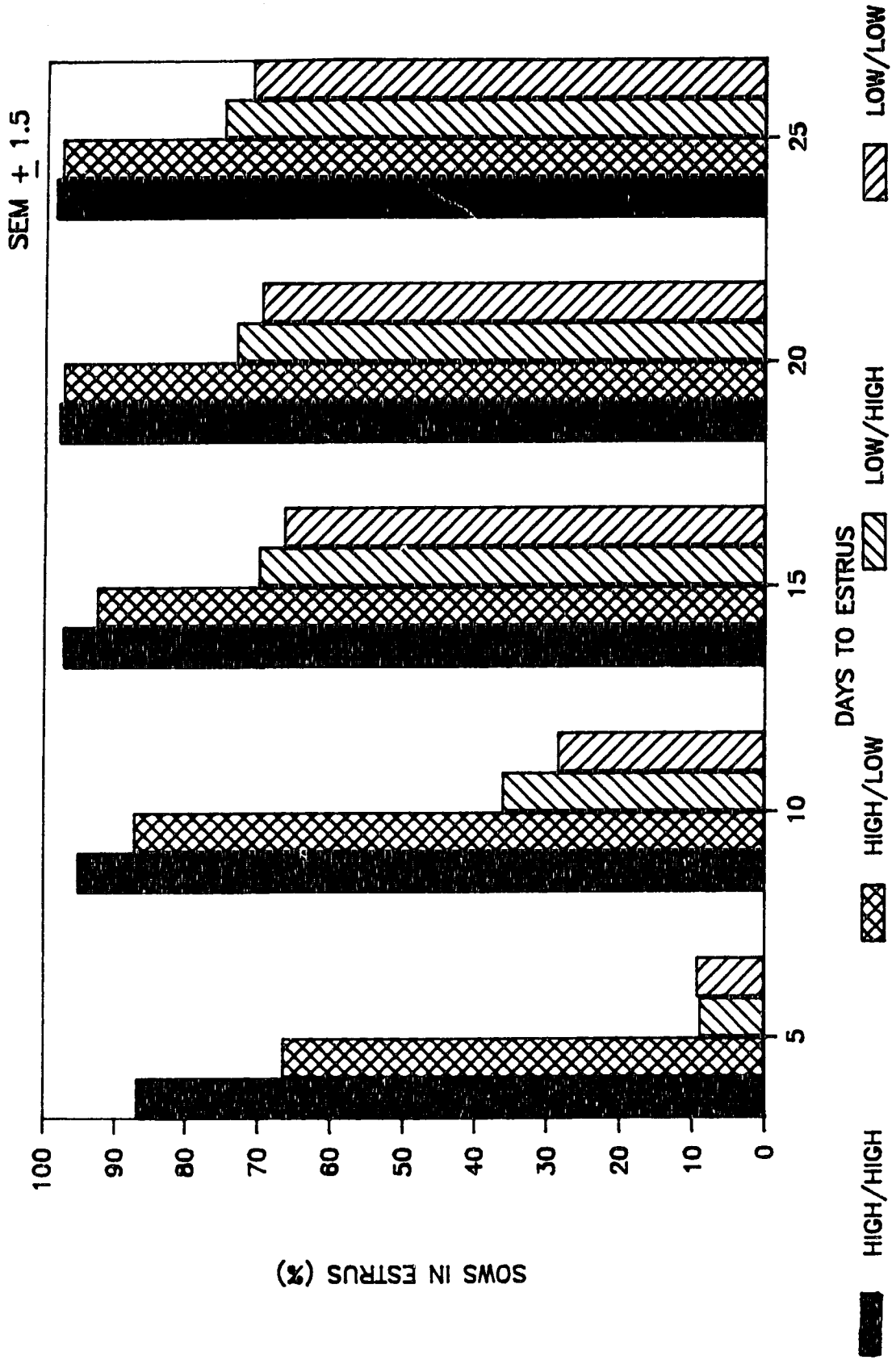

SEM + 1.5
SOWS IN ESTRUS (%)
100
90
80
70
60
50
40
30
20
10
0
5
10
15
20
25
DAYS TO ESTRUS
HIGH/HIGH
HIGH/LOW
LOW/HIGH
LOW/LOW

Figure IV.3 Plasma levels of estradiol-17β concentrations during the postweaning period in sows fed high and low levels of feed during lactation and the postweaning period

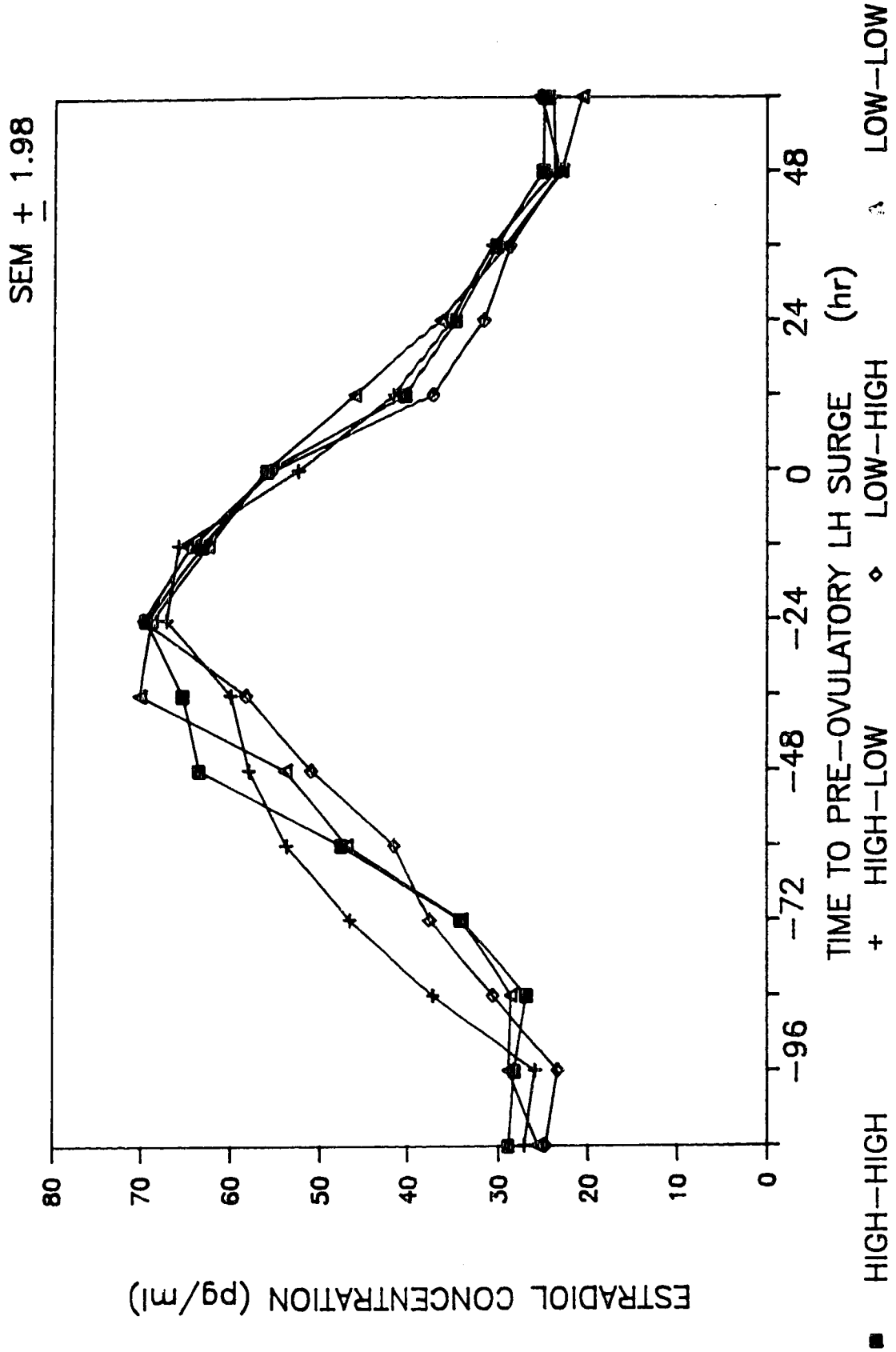

SEM + 1.98
ESTRADIOL CONCENTRATION (pg/ml)
0
10
20
30
40
50
60
70
80
-96
-72
-48
-24
0
24
48
TIME TO PRE-OVULATORY LH SURGE (hr)
■ HIGH-HIGH
+ HIGH-LOW
◇ LOW-HIGH
LOW-LOW

Figure IV.4 Mean plasma estradiol-17β concentrations in sows with corpora lutea (C.L.) (■) and sows with no C.L. (+) at slaughter d 25 postcoitum

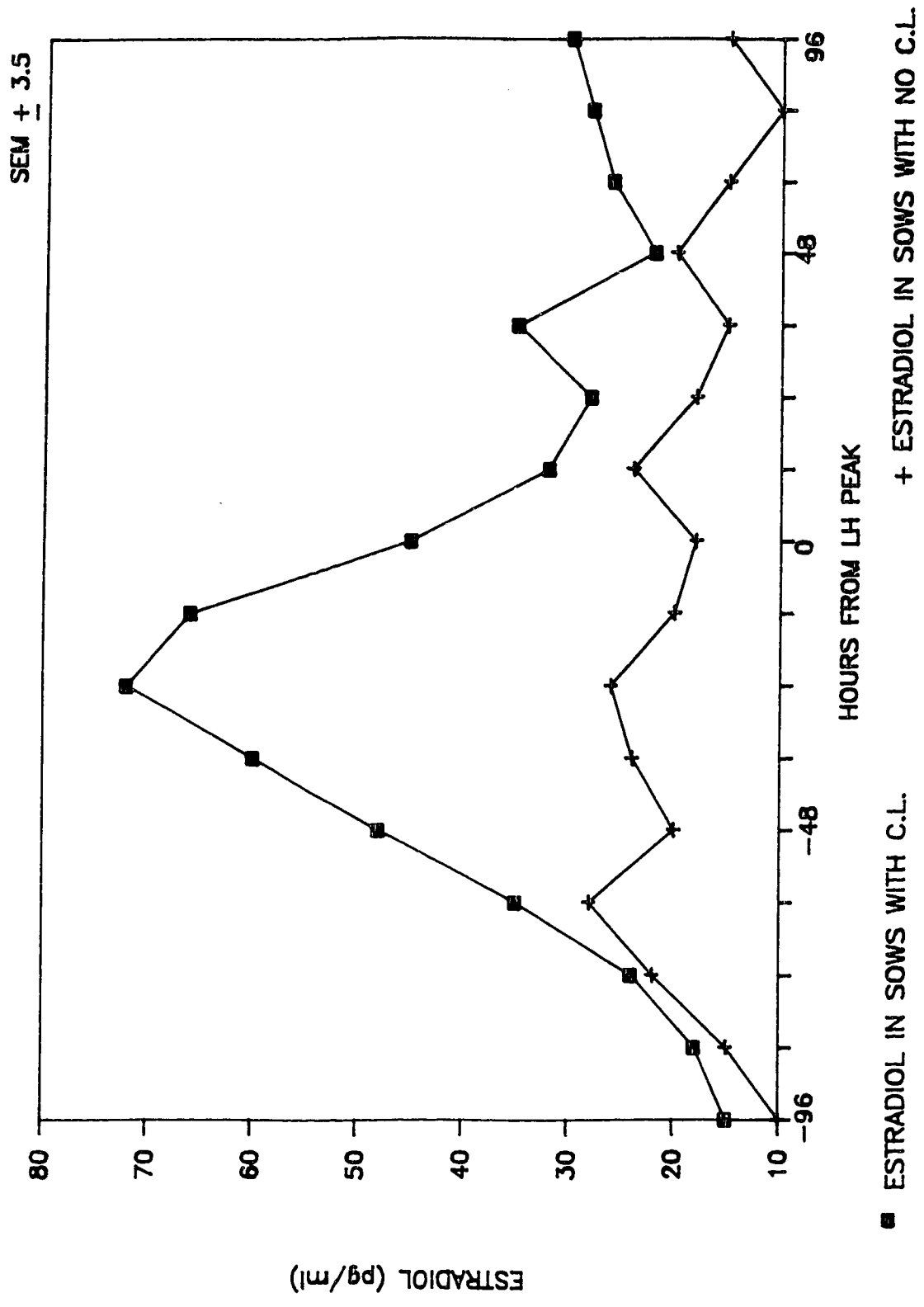

SEM ± 3.5
ESTRADIOL (pg/ml)
80
70
60
50
40
30
20
10
-96
-48
0
48
96
HOURS FROM LH PEAK
■ ESTRADIOL IN SOWS WITH C.L.
+ ESTRADIOL IN SOWS WITH NO C.L.

Figure IV.5 Mean plasma LH concentrations during the postweaning period in sows fed high or low levels of feed during lactation and the postweaning period

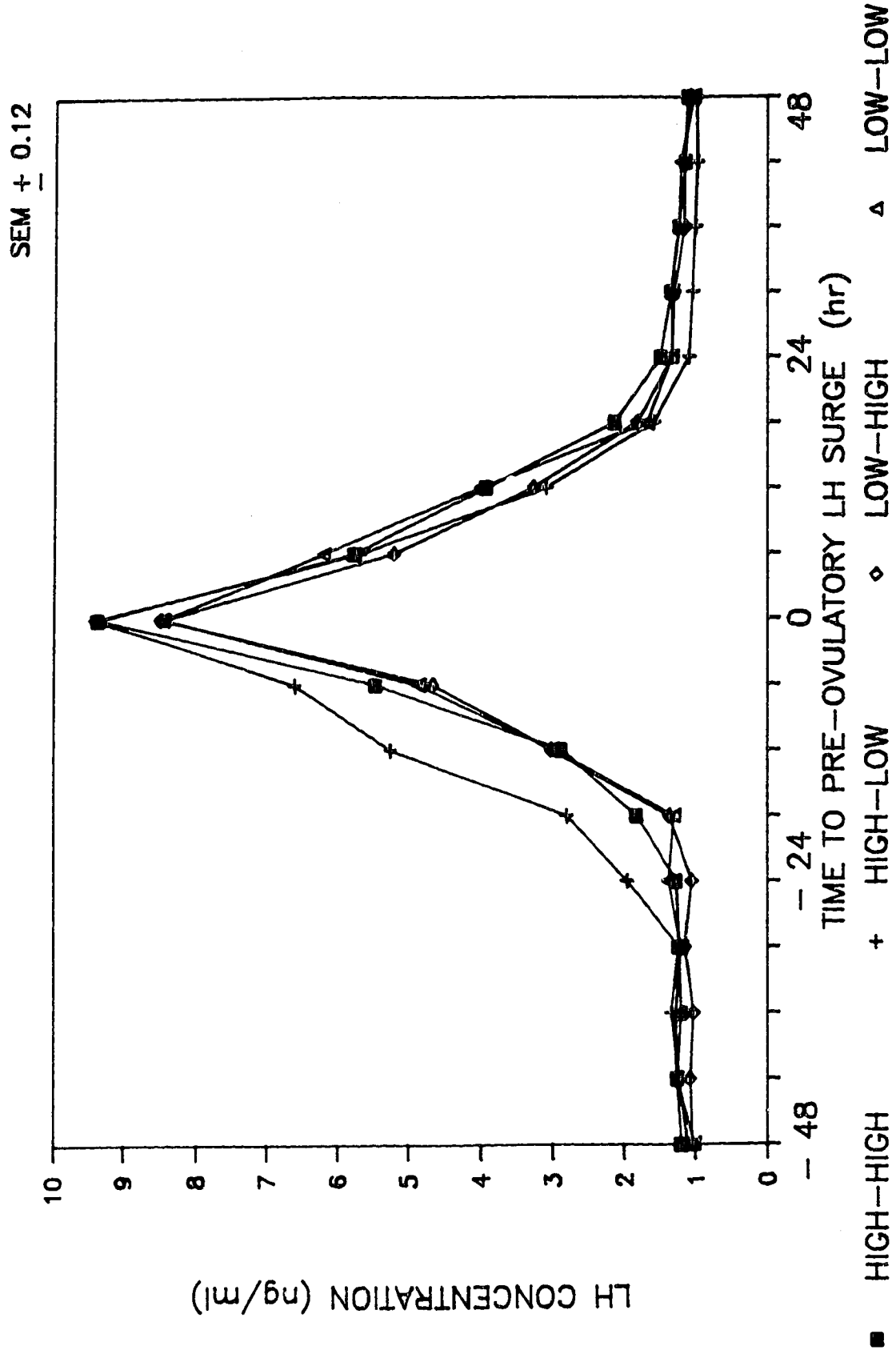

SEM ± 0.12
LH CONCENTRATION (ng/ml)
10
9
8
7
6
5
4
3
2
1
0
– 48
– 24
0
24
48
TIME TO PRE–OVULATORY LH SURGE (hr)
■ HIGH–HIGH
+ HIGH–LOW
◇ LOW–HIGH
△ LOW–LOW

Figure IV.6 Mean plasma FSH concentrations during the postweaning period in sows fed high or low levels of feed during lactation and the postweaning period

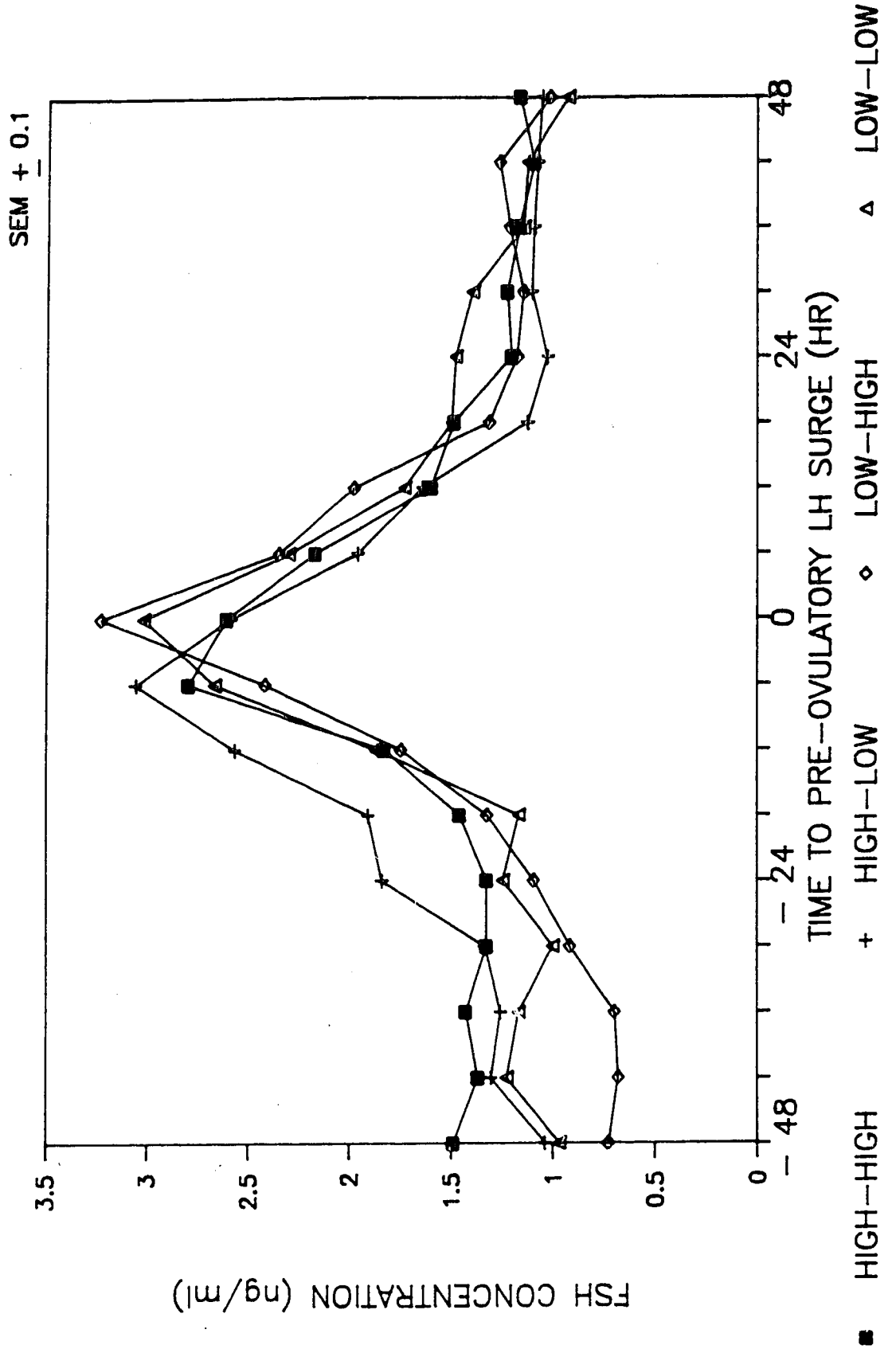

SEM + 0.1
FSH CONCENTRATION (ng/ml)
3.5
3
2.5
2
1.5
1
0.5
0
– 48
– 24
0
24
48
TIME TO PRE–OVULATORY LH SURGE (HR)
HIGH–HIGH
HIGH–LOW
LOW–HIGH
LOW–LOW

Figure IV.7 Mean plasma LH concentrations in sows with corpora lutea (C.L.) (■) and sows with no C.L. (+) by d 25 postcoitum

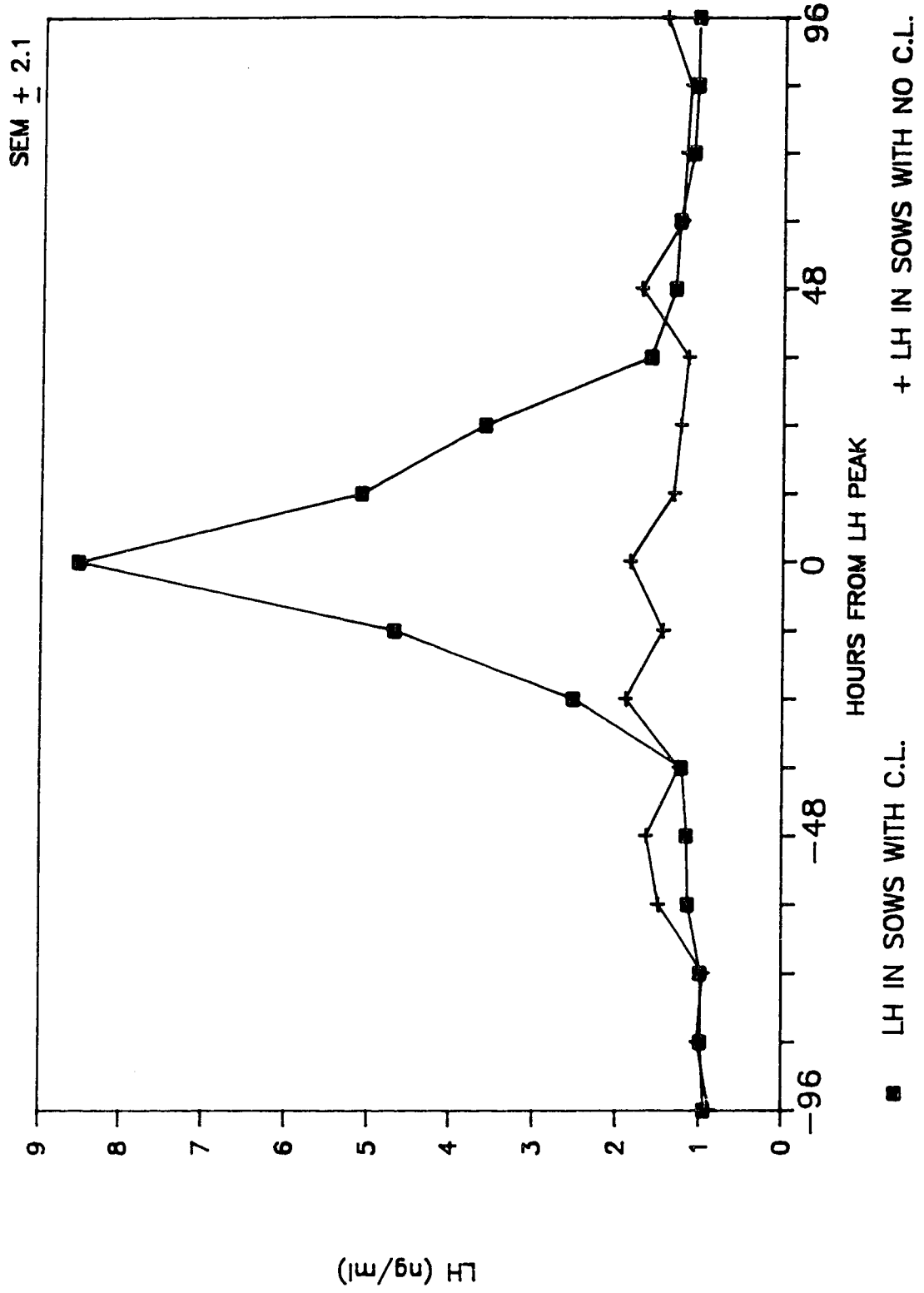
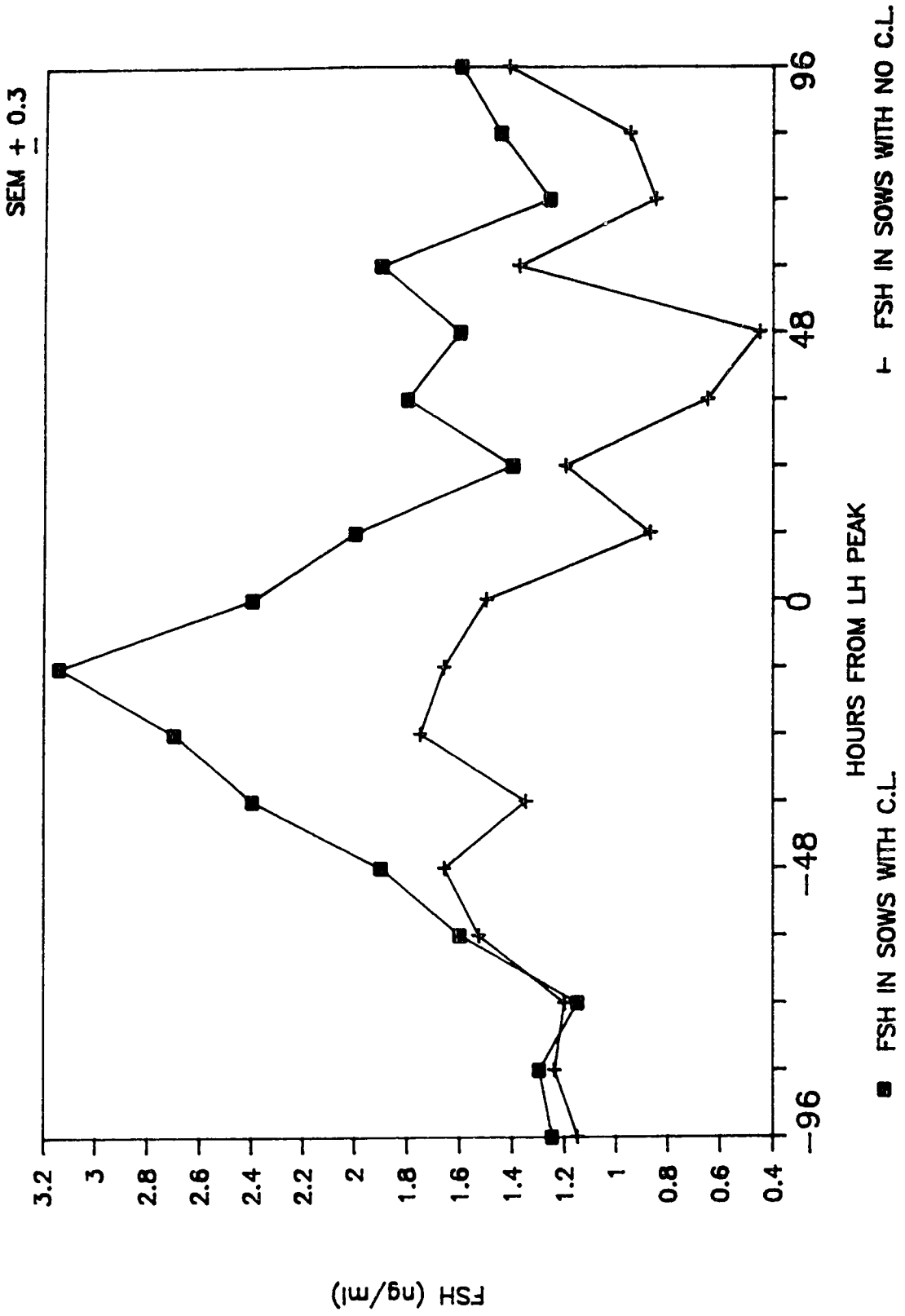

SEM ± 2.1
LH (ng/ml)
0
1
2
3
4
5
6
7
8
9
−96
−48
0
48
96
HOURS FROM LH PEAK
■ LH IN SOWS WITH C.L.
+ LH IN SOWS WITH NO C.L.

Figure IV.8 Mean plasma FSH concentrations in sows with corpora lutea (C.L.) (■) and sows with no C.L. (+) by d 25 postcoitum


SEM + 0.3
FSH (ng/ml)
3.2
3
2.8
2.6
2.4
2.2
2
1.8
1.6
1.4
1.2
1
0.8
0.6
0.4
-96
-48
0
48
96
HOURS FROM LH PEAK
FSH IN SOWS WITH C.L.
FSH IN SOWS WITH NO C.L.

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V. GENERAL DISCUSSION

For many reasons, sow feed intake during lactation can be highly variable. The data from experiment 1 (Chapter II) support the conclusion that low feed intake during lactation results in extensive body weight loss. The components of the body weight loss include both lean body mass and body fat tissue which is in agreement with the results of several other studies (Etienne et al., 1985; King et al., 1986; Mullan, 1987). The H sows lost equal amounts (kg) of body fat tissue and lean body mass, but the low fed sows lost more body fat than body lean tissue. In situations where lactation feed intake is inadequate, the demands of lactation are met through mobilization of maternal body reserves. To prevent body catabolism it was estimated (Chapter II) that a sow with postpartum weight of about 200 kg would require 8 kg of feed daily to maintain body weight and backfat.

It was shown (Chapter II) that there was no significant difference in weight gain in piglets in spite of the level of feed intake of the sows during lactation. This observation is consistent with a previous report that there was no significant differences in piglet weight gain in piglets on sows restricted and ad lib feeding regimes during a single lactation (Reese et al., 1984). Thus body reserves during a single lactation may be sufficient to enable the sow to withstand nutritional restrictions during lactation. However, it has been shown that continued feed restriction for more than two parities did reduce weaning weights of the piglets (Lythgoe, 1987).

The excessive loss of body weight and backfat during lactation was reflected in the profiles of plasma glycerol, growth hormone, cortisol, insulin and glucagon. Thus the response to feed restriction during lactation and during the postweaning period were related to changes in plasma concentrations of the hormones studied. Most studies of the physiological effects of hormones have been based on changes in serum or plasma concentrations. The plasma concentration, however, represents a balance between secretion into and clearance from the circulation. A change in plasma concentration may therefore reflect a change in either secretion or clearance or both, but it seems that most changes in plasma hormone concentrations are caused by changes in secretion (Trenkle, 1978). Also, the biological effects

of a given plasma concentration depends on blood flow to the target tissue, the receptor concentration in the target tissue, and the presence of an active amplification system. However, receptor concentrations have been found to vary from one physiological state to another (Trenkle, 1978).

Major shifts in energy metabolism occur during under feeding. Glycerol, growth hormone, cortisol, insulin and glucagon concentrations on d 2 postpartum were not different between low and high fed sows, presumably because the sows were consuming similar amounts of feed and the sows had similar amounts of body reserves at the begining of lactation. As lactation progressed the L fed sows lost more weight and body fat than did the H fed sows. At weaning the mean concentrations of glycerol, growth hormone and cortisol in the low fed sows were significantly higher than in the high fed sows. Growth hormone, in addition to its importance for growth is also suggested to play a vital role in mobilizing body fat and diverting energy from body tissue synthesis towards milk production (Bines and Hart, 1978).

Plasma cortisol concentrations were significantly elevated in the low fed sows. Cortisol is known to be catabolic and inhibits uptake of glucose by body tissues and stimulates release of glucose into the circulation. This effect of cortisol has been demonstrated in both fed (Reilly and Black, 1973) and fasted (Basset, 1968) sheep. The secretion of both growth hormone and cortisol during undernutrition should facilitate energy mobilization from body tissues to satisfy demands for maintenance amd productive processes such as lactation.

Insulin was not responsive to feed intake on d 2 postpartum which may have been related to a lack of any differences in consumption at this time. However, there is a suggestion that glucose released from feed at the beginning of lactation is shunted to the mammary gland for milk synthesis (Bauman and Currie, 1980). Furthermore, high plasma concentration of prolactin on d 2 postpartum is known to increase the number of insulin receptors in the mammary gland during lactation and to decrease plasma level of insulin (Flint, 1982). This may help explain the lack of response to feed on d 2 postpartum. On d 28 postpartum, a postprandial rise in insulin secretion occurred in both low and high fed sows. The greater response after the morning compared to the afternoon feed is presumed to relate

to the previous overnight fast. At the end of lactation the total amount of insulin secreted by the high fed sows was greater than the amount secreted by the low fed sows. Insulin has been suggested to play a key role in reproduction. Steiner (1987) suggested that insulin binds specifically to receptors in the arcuate nucleus and median eminence of the brain, regions regarded to serve important regulatory functions for the control of GnRH secretion. Acting at the brain, insulin influences the availability of substrates for the synthesis of neurotransmitter systems in the brain, particularly that of norepinephrine and serotonin, both of which are thought to interact with GnRH secretory mechanisms. It is therefore possible that by influencing the synthesis of certain neurotransmitters critical for maintaining GnRH secretion, insulin could provide a link between nutritional status and reproductive function. Recently, Cox et al. (1987) demonstrated that ovulation rates could be increased by dietary energy and exogenous insulin. Effects of insulin and high energy on ovulation rate may involve the entire hypothalamo-hypophyseal-ovarian axis and interactions among its components. In the hypothalamus, insulin and insulin receptors were identified in the vicinity of gonadotropin releasing hormone (GnRH) producing neurons (Havrankova et al., 1978), and estrogen binding was promoted by insulin (Siegel and Wade, 1979). Direct effects of insulin on ovaries have also been identified, including promotion of production of progesterone by granulosa (May and Schomberg, 1981; Veldhuis et al., 1985), and luteal cells (Ladenheim et al., 1984) in culture, and androstenedione by thecal cells (Barbieri et al., 1983). Thus tropnic actions of insulin and / or insulin-like peptides and the synergism of insulin with classical ovarian effector hormones are likely to be of physiological importance to the differentiation of granulosa cells in the developing ovarian follicle.

Plasma prolactin levels were very high at the beginning of lactation. It has been shown that at the beginning of parturition, plasma prolactin levels increase to prepare the mammary gland for milk synthesis (Leong et al., 1983) and that this increase is related to the suckling intensity of the piglets (Stevenson et al., 1981). At the end of lactation the suckling intensity of piglets declines and plasma prolactin concentrations susequently decrease.

During lactation the gonadotropins were at very low levels. Indeed LH was not detectable by the LH assay on d 2 postpartum. There was a gradual increase in LH concentration as lactation period increased. This phenomenon of LH increase with duration in lactation has been demonstrated in sows (Stevenson and Britt, 1980; Bevers et al., 1978). The use of dopamine (Ben-Jonathan, 1985), naloxone (Mattioli et al., 1986; Armstrong et al., 1988) and bromocryptine (Bevers et al., 1981) have all shown that the release of prolactin per se is independent of the release of LH. The explanation may be that the suckling stimulus stimulates neurons which inhibits the release of GnRH resulting in inhibition of LH synthesis and secretion.

Plasma FSH increased with duration of lactation and plasma FSH concentrations during lactation may be controlled by a different physiological mechanism to that regulating LH secretion. The control appears to be due to a secretory product of the ovary (Stevenson et al., 1981). As lactation progresses, this inhibition seem to diminish or is countered by a positive effect on FSH release and such changes were particularly apparent in sows fed high levels during lactation.

The sows which lost excessive body weight and backfat had extended remating intervals. Brooks (1982) suggested that sows losing excessive weight during lactation remain catabolic for a longer period after weaning than sows losing less weight. It was further suggested that a persistent net catabolism may be related to the delay in estrus onset. The low fed sows had significantly high levels of the catabolic hormones, namely cortisol and growth hormone. There is evidence indicating that high levels of cortisol will inhibit the hypothalamic pituitary axis secretory activities (Dobson, 1987), by the adrenal modulation of the gonadal axis (Moberg, 1987). Immobilization of female rats has also been reported to increase circulating corticosteroids and to cause a corresponding drop in plasma LH (Tache et al., 1976); the FSH response to immobilization was much more variable (Tache et al., 1976), sometimes being suppressed by stress and other times unaffected. Recently, another component of the adrenal axis, corticotrophin releasing hormone (CRH), has been found to inhibit the hypothalamic release of LH, but not FSH (Ono et al., 1984; Rivier and Vale,

1984). It remains to be determined whether the neurosecretory cells that inhibit GnRH are also the cells that release CRH into the hypothalamic-hypophyseal portal system to control the pituitary corticotrophs. In addition, ACTH, which induces synthesis and release of cortisol, suppresses the secretion of gonadotropins. This effect is attributted to the adrenal progesterone secreted in response to ACTH (Moberg, 1987). However, the importance of adrenal progesterone in regulating gonadotropin secretion has not been established.

The observation in this report that low plane feeding during lactation decreased embryo survival is consistent with results from other studies (Johnson et al., 1986; Hughes et al., 1984). Various possible etiologies for increased embryo mortality associated with low plane feeding have been suggested (Aherne and Kirkwood, 1985). The speculation was that there is a reduced amount of LH released at estrus and a lowering of plasma progesterone during early pregnancy. The low fed sows during lactation had a reduction in total LH and FSH concentration in plasma during the postweaning period. Thus the gonadotropins, especially LH could possibly play a key role in the high embryo survival in the lactation high fed sows. The H-L sows had a higher peak amplitude of LH and also had the highest rate of embryo survival. Thus the relative levels of LH and FSH may influence subsequent luteal function as suggested by Smith (1986) and studies with ewes in poor condition indicated that poor body condition was associated with reduced FSH / LH levels (Rhind and McNeilly, 1986; McLeod and Haresign, 1987). Armstrong and Britt (1985) also suggested that, administration of hourly pulses of GnRH to weaned anestrus primiparous sows imposed a proper stimulation to the anterior pituitary that in turn lead to an increased gonadotropin secretion that induced estrus and ovulation.

The present studies would therefore suggest that, excessive weight and (or) backfat loss during lactation will adversely affect sow reproductive performance by increasing the weaning to remating interval. Postweaning feed intake had no effect on the reproductive performance of the sow during the weaning to effective mating interval. A majority of sows showed elevations of both LH and FSH secretion after weaning with the sows fed high during lactation showing marked elevations. Estradiol-17β was not influenced by treatment. Thus, it

could be concluded that undernutrition with the consequent weight and backfat loss will reduce the secretion of LH and FSH. The conclusions of this study are based on plasma hormonal levels. The respective concentrations of the metabolic hormones, growth hormone, insulin and cortisol have been described under a state of restricted feeding. There is paucity of information on the effect of undernutrition on receptor characteristics. Changes in receptor concentrations may be an important factor in explaining the mechanism(s) involving undernutrition and reproductive performance.

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