Nutrition-Reproduction Interactions in Swine

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Pigs have developed the ability to store large reserves of protein, glycogen and fat in the body. The pig has also developed the ability to relate its nutrient intake, body composition and the level of tissue mobilization with the reproductive process. The possible physiological and endocrinological mechanisms by which genetically determined traits such as weight, fatness, growth rate or environmental cues such as nutrition affect the reproductive process have been extensively reviewed by Britt et al. (1988), Hughes and Pearce (1989) and Booth (1990). The concensus is that nutrient intake (total feed, energy or protein) will affect the levels of substrates, hormones and neurotransmitters in the plasma, and these can influence the hypothalamo-hypophyseal-ovarian axis. In the longer term, nutrient intake will influence growth rate, body composition (protein and fat) and the extent of tissue mobilization. The level of anabolism or catabolism of body tissues will influence a host of plasma substrates, hormones and neurotransmitters which will in turn affect the function of the hypothalamus, hypothysis and ovary (Figure 1).



Figure 1. A model for the control of onset of oestrous. The model proposes that nutrition induced change in metabolic hormones and substrates such as GH, insulin and IGF-1 modulate the activity of the hypothalmo-hypophyseal-ovarian axis.

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Puberty

The expression of puberty is dependent on a number of genetically determined traits such as body weight, fatness and growth rate and is also influenced by environmental factors such as nutrition, photoperiod and presence of a mature male (Figure 1.). The effects of these genetically-determined traits and environmetal cues on the onset of puberty are mediated through signals by a host of plasma substrates, hormones and neurotransmitters to the hypothalamo-hypophyseal-ovarian axis (Booth 1990). A review of the literature on puberty onset in gilts suggests that they are anatomically and physiologically ready for reproduction by five months of age, whereas puberty onset may not occur until six or seven months of age (Kirkwood and Aherne 1985). The application of exogenous hormones will trigger puberty in many gilts at 140 days of age (Paterson 1989). The conclusion from these studies may be that the trigger for puberty onset is through a required pulse frequency of gonadotrophin releasing hormone (GnRH) (Foster et al. 1988, Bronson and Manning 1988). A pulse of GnRH evokes a pulse of LH from the hypophysis, and above a certain frequency, this will stimulate follicular growth and ovulation (Martin 1984). Thus the effects of the genetically determined traits and the environmental cues on the onset of puberty is through the control of the GnRH pulse generator and changes in the sensitivity of the hypothalamo-hypophyseal-ovarian axis to metabolic and steriod hormones. The way nutrition influences the hypothalamo-hypophysealovarian axis is still speculative and a host of substrates, hormones, and neurotransmitters may be involved (Britt et al. 1988, Booth 1990).

The onset of puberty has been associated with attainment of a critical or threshold body weight, a minimum lean to fat ratio or minimum level of body fat (Paterson 1989, Burnett et al. 1988, Kirkwood et al. 1987). We believe that there is now sufficient evidence to say that the amount of fat in the body does not affect the attainment of puberty. If level of fatness is an important trigger for puberty onset, a clear difference in body fatness at any given age or weight would be anticipated between animals that do and do not achieve puberty. However, the study of Price et al. (1981) shows no difference in body composition at 109 kg between gilts that did or did not reach puberty before that weight. Also, if there is a critical pre-requisite fatness for puberty onset one might expect a skewing of the frequency distribution but, for a population of 349 gilts, Young et al. (1990) observed a normal distribution of fatness at puberty. Alternatively, metabolic mass and food intake or its correlated metabolic state may be the triggering mechanism to puberty onset (Booth 1990).

Further support to rule out fatness as **a major** determinant of onset of puberty comes from the experiments of Magowan (unpublished data) who manipulated the growth of gilts by controlling their food intake. His control was a group of gilts which he fed ad libitum from 50 kg liveweight. These gilts reached puberty as anticipated at 172 days of age and they weighed 108 kg and had 17.1 mm of **backfat**. He maintained another group of gilts at 50 kg liveweight. None of these had attained puberty by 270 days of age but, when changed to ad libitum feeding, they reached puberty 65 days later when they were 335 days old. These gilts had very little fat (5.3 mm) at puberty. Similarly when gilts were maintained at 80 kg liveweight until 270 days of age and then returned to full feeding they also reached puberty with very little body fat (6.7 mm). Even gilts that were restricted to 85 % of ad libitum from 50 kg liveweight were lean (9.6 mm) when they reached puberty at 238 days of age (Table 1). The relative unimportance of body fat in attainment of puberty is also demonstrated in the work of Britt et al. (1988) who induced gilts to stop regular oestrous cycles by restricting their energy intake. The return to **cyclicity** after realimentation was associated with an increase in weight gain but no increase in **backfat** depth.

We conclude from the evidence above that prolonged undernutrition will not delay puberty indefinitely and that underfed gilts reach puberty at lighter weights and lower fatness levels than well-fed animals. These data also support the conclusion that level of fatness is not a critical prerequisite for the onset of puberty. Britt et al. (1988) and King (1989) have suggested that protein reserves or their metabolic correlates, rather than adiposity may influence **reproductive function.** Thus when fatness is not confounded with maturity, it has little influence on the onset of puberty.

Table 1. Age, weight and backfat at puberty for gilts fed ad libitum (AL) restricted fed to 85 % of ad libitum intake from 50 kg liveweight (ER50), or fed to maintain weight of 50 kg (M50) or 80 kg (MSO) liveweight and returned to full feding at 270 days of age. Each treatment had 12 gilts.

	M50	M80	ER50	AL.	±sem
Age (days) Weight (kg) Backfat (mm)	335a 88a 5 3a	269b 84a 6.7 ^b	238 ^c 104 ^b 9.6 ^c	172d 108b 17.1d	10.2 3.2 0.4
Backfat (mm)	5.3ª	6.70	9.60	17.14	0.4

In terms of priority of nutrients, the reproductive organs have a lower priority for nutrients than other tissues and organs. Therefore in times of nutrient deficiency growth and development of the reproductive organs will be retarded. This is consistent with the conclusion that restriction in feed intake (60 % to 85 % of ad libitum) of pigs in the growing (20 to 50 kg) period will delay the onset of puberty by about 10 to 14 days (King 1989 a,b). However, undernutrition may not prolong puberty indefinitely and, if normal feeding levels are resumed, underfed animals will reach puberty but at a lighter weight (Britt et al. 1988, Widdowson 198 1).

From the reviews of den Hartog and Van Kempen 1980 and Paterson 1989 evidence is presented for a positive, negative or zero relationship between growth rate and age at puberty depending on the data selected. Young et al. (1990) in a large scale experiment reported no significant correlation between growth rate and age at puberty. Beltranena et al. (1990) reported a negative quadratic relationship between age at puberty and lifetime growth rate to puberty. They interpret the data as shown in Figure 2.



Figure 2. Proposed relationships between age at puberty and lifetime growth rate (Beltranena et al. 1990)

For restrict-fed pigs and some ad libitum-fed pigs growing at less than 500 g/day from birth an increase in growth rate will reduce age at puberty. For gilts with growth rates of 500 to 650

g/day there is little influence on age at puberty but, for gilts with a growth rate greater than 650 g/day, a further increase in growth rate may retard animals reaching puberty. They suggest that this possibly reflects the need for further maturation of the hypothalamic-hypophysial-ovariarl axis or the need to attain a critical body weight consistent with the onset of puberty.

Potential size might be an important determinant of puberty. It has been suggested that gilts reach puberty when they achieve a certain proportion (approximately 30%) of their mature weight King (1989a). The concept that animals have different mature sizes helps to explain how attainment of puberty is influenced by growth rate. It would be expected that genotypes with the largest growth rate would also have the largest mature body size. Williams et al. (1985) estimated that the mature body weight of a modem sow, corresponding to zero nitrogen retention, was approximately 340 kg. In the study of Price et al. (1981) 90 gilts of mixed genotypes were monitored for onset of oestrus and slaughtered at 109 kg liveweight. Thirty-six percent of the gilts reached puberty before 109 kg. Growth rate was significantly greater for the population of pigs that did not reach puberty before 109 kg but, within the population of pigs that did reach puberty, the fastest growing gilts reached puberty earlier than those with slower growth rates. This apparent paradox may be explained as shown in Figure 3.



Figure 3 The relationship between mature body weight and onset of puberty (Price et al. 1981) indicates the weight (shaded area) at which gilts of different mature body weights might be expected to reach puberty.

The fastest growing gilts would have a larger mature body weight and 30 % of this weight would not occur until after 109 kg. Since the gilts in this experiment were slaughtered at 109 kg none of these animals reached puberty. In contrast the slower growing population that did reach puberty would have a mature body weight of 320 kg or less and therefore at 30 % of mature weight these gilts would be less than 109 kg liveweight.

How growth rate per se or attainment of a particular weight provides information to the reproductive system is a topic of much recent debate. As the animal increases in weight there is a change in the relationship between body weight and surface area. Thus, there is a change in the balance between heat producing mass and heat radiating surface area which requires an increase in heat loss or a reduction in metabolic rate if body temperature is to be maintained. It is possible that some aspect of this change in metabolism is monitored by the hypothalamus and is used to initiate the process of puberty onset (Kennedy and Mitra 1963)

OVULATION RATE

There is ample evidence to suggest that increasing the level of food or energy intake during the rearing period will significantly increase pubertal ovulation rate (den Hartog and Van Kempern 1980, Aheme and Kirkwood 1985). It is also well established that short-term, high-level feeding during the first oestrous cycle (flushing) increases ovulation rate (Hughes and Pearce 1989, Beltranena et al. 1990). Beltranena et al. 1990 also demonstrated that the flushing effect is not a superovulation but merely a normalizing of low ovulation rates seen in restrict-fed gilts.

Prepubertal feed (kg/d)		2.0		Ad libitum
Postpubertal feed (kg/d)	2.0		2.0	Ad libitum
Ovulation at puberty		11.2 ± 0.4		13.3 ±0.8
Second ovulation	12.1 ±0.3		13.5 ±0.6	13.7 ±0.9

Table 2. Influence of feed intake on first and second ovulation rates. (Data from F.X. Aherne, University of Alberta 1990)

The most likely mechanism for the way flushing increases ovulation rate is by increasing plasma insulin, insulin-like growth factor 1 (IGF1) (Beltranena et al. 1990), increasing plasma follicle stimulating hormone (FSH), luteinizing hormone (LH) and increased pulse frequency of LH (Rhodes et al. 1987, Flowers et al. 1988). These results are consistant with the suggestions of Hughes and Pearce (1989) and Scaramuzzi and Campbell (1990) who presented a model for the regulation of ovulation rate. It is suggested that the increase in ovulation rate is determined by a reduced atresia of follicles rather than an increase in recruitment. The lack of a significant correlation between the increase in ovulation rate and change in any particular production trait from first to second oestrus for the flushed gilts suggests that realimentation may exert a short-term effect on ovulation rate through an improvement in metabolic status and not due to changes in body weight or fatness. This is in agreement with the suggestions of Booth (1990).

CONCEPTION RATE

A review of 26 experiments employing high or low feed intake up to puberty or **during** the oestrous cycle suggested that feed intake had no significant deleterious or beneficial effect on conception rate (den Hartog and Van Kempen, 1980).

EMBRYO SURVIVAL

High-level feeding during rearing or in the premating period is associated with an increased embryo mortality (Table 3). It is possible that the increased embryo mortality is due to an increased ovulation rate which would lead to a higher embryo mortality. The end result as can be seen in Table 3 is that nutrition in the prepubertal or premating periods did not affect number of embryos at 25 days of gestation.

Table 3. Influence of feeding level on embryo survival in gilts(adapted from den Hartog and Van Kempen 1980)

Period	No. of Trials	Energy Intake MJ ME/day	No. of Embryos at 25 days	Embryo survival (%)	
Prepubertal	19 46	35.7 22.8	9.8 10.0	69.7** 77.5	
Premating	36 31	38.5 21.6	10.1 9.7	73.2* 78.3	

**P < 0.01 *P < 0.04.

- GESTATION

Energy and nutrient requirements of the sow can be derived factorially from a summation of energy requirements for maintenance, maternal weight gain and products of conception. The developing foetus has the highest priority in allocation of nutrients except when gilts are still growing during their first gestation. Then the nutrient needs for body growth compete with those of the growing foetus. Several recent reviews and experiments have shown that the nutrient requirements of sows for maintenance, net body gain and products of conception can now be estimated with considerable accuracy (ARC 1990, NRC 1988, Noblet et al. 1990, Speer 1990, Mahan 1990). The energy cost of maintenance is about 462 kJDE/kg^{0.75} and does not vary throughout gestation (Noblet et al. 1990). During pregnancy, maintenance represents 75 to 85% of the total energy requirements and therefore an accurate estimate is very important. Maternal weight gain represents about 17% of energy requirements and the products of conception a further 5 %. The energy requirement for maternal weight gain is estimated as 4.6 MJDE/day for a maternal gain of 25 kg or approximately 21 MJDE/kg gain (NRC 1988). Noblet et al. (1990) suggests that the energy content of maternal gain can range from 8.4 to 18.8 MJ/kg with a mean value of 14.6 MJ/kg, depending on the composition of the gain. If the energy content of protein and fat is 23.8 and 39.7 MJ/kg, then the mean composition of maternal gain is approximately 73 % lean and 27 % lipid, with a range of 60 to 90 % lean. Noblet et al. (1990) suggest that the metabolisable energy (ME) is used for deposition in maternal tissues with a mean efficiency of 77%. Thus, the estimated cost of one kilogram of maternal gain would be approximately 20 MJDE/kg, which is in reasonable agreement with the NRC (1988) value of 21 MJDE/kg. The lean-to-fat ratio of maternal gain will vary with the amount and composition of the diet fed. During first pregnancy and in older animals with limited maternal weight gain, the net weight gain corresponds mainly to lean tissue or it may be that the chemical composition of the fat itself is changing, With a lower percent water in fat when the amount of fat deposited is low or when fat is being mobilised (Lee and Mitchell 1989,

Mullan and Williams 1990). Mullan and Williams (1990) highlight the danger in extrapolating prediction equations for body fatness to different situations or phases of the reproductive life.

Speer (1990) suggests that the nitrogen and protein requirements of a 120 kg sow gaining 25 kg of maternal body weight throughout gestation are met by about 23 gnitrogen intake/day (or about 144 g protein/day). This is dependent on the indispensible amino acids being fed at levels suggested by (NRC 1988) and supplemental nitrogen is supplied by Lglutamic acid. Speer (1990) also suggests that grain-soyabean meal diets supply a great excess of the non specific nitrogen component and all of the indispensible amino acids except lysine. From a review of the literature Aherne and Kirkwood (1985) suggested that sow weight gain will respond to increased protein intake up to levels of 300 g/day in gestation, but no apparent improvement in birth weight or reproductive performance is seen beyond a daily intake of approximately 140 g protein supplying 8-10 g lysine, 7-8 g threonine and 1.5 g tryptophan.

There is a high correlation (about 0.7) between feed intake and weight gain during gestation. Piglet birth weight is moderately correlated (0.4 - 0.5) with sow feed intake in pregnancy but poorly correlated with litter size (0.14) (Britt et al. 1988). The main effect of altering feed or energy intake during gestation is on the body weight of the sow rather than the performance of the litter. Because maternal deposition of protein and energy is responsive to dietary intake, specific maternal weight gain and level of fatness can be targeted with some degree of accuracy. However, the optimum level of maternal weight gain or level of fatness consistent with the long term and economical reproductive efficiency of the sow is not well known.

Whittemore et al (1988) have reported that sows may have a net gain of 15 kg in a preproductive cycle (25 kg gain in gestation followed by 10 kg loss in lactation) and yet lose substantial quantites of body fat. Thus weight gain in gestation will occur at feed intake levels that will not support fatty tissue gain. Modem hybrid sows usually begin their reproductive lives with less than 20 mm P2 backfat and in many situations do not consume sufficient food during lactation to meet their nutrient requirements for milk production. They do not maintain body weight or composition. After several parities these sows may have depleted their fat stores below 10 mm P2 backfat. It has been suggested by Whittemore et al. (1988) that sows are unwilling to deplete fat reserves below 10 mm and therefore such sows will reduce milk production and will have reduced post weaning reproductive efficiency. They suggest that a maternal body weight gain of about 28 kg in pregnancy is consistent with maintenance of backfat in first parity sows.

Target weight gains should reflect the expected feed intakes and condition loss during lactation. The amount of food fed to the sow in gestation and her targeted weight gain will also be influenced by the fact that there is a negative relationship between feed intake during gestation and the subsequent lactation (Cole 1990). Also recent studies at the University of Western Australia have shown that level of fatness at parturition, independent of body weight has a major influence on voluntary feed intake during lactation. Based on these considerations maternal weight gains of **30**, **25**, **25**, **20**, 15 and 10 kg in pregnancies one through six might be appropriate under good environmental conditions. If the sow has an innate drive to achieve her mature body weight, estimated by Williams et al (1985) to be 340 kg live weight for a modem sow, at the target maternal gains suggested previously and accepting a 10 kg loss in sow weight during lactation, a sow will only attain 50 - 55 % of her mature weight by the end of the sixth parity. The significance of this is not known.

The majority of experiments have demonstrated that although there may be a progressive increase in piglet birth weight with increasing sow feed or energy intake during pregnancy, the response above 25 MJDE/day was seldom significant, the major effect being seen with maternal weight gain. However, levels of energy below 20 MJDE/day have generally reduced birth weights significantly (Aheme and Kirkwood 1985, Lee and Mitchell 1989, Mullan and Williams 1989, Whittemore et al 1988, Young et al 1990).

Demand for energy and nutrients is low in gestation relative to lactation and the sow is usually fed only about one third of her voluntary-feed intake. It has been shown above that during gestation sows are generally fed about 2.0 kg food/day in one daily feed. Such a **feeding** system results in sows going from an absorptive phase to a lengthy post-absorptive phase and thus they experience a considerable period of fasting before their next meal. This period of fasting will significantly influence metabolic rate and a host of plasma metabolites and hormones of gestation and especially with first-parity gilts.

It is suggested that to ensure maximum stability in body composition of the sow throughout her reproductive life that, an appropriate feeding strategy should be to achieve a small weight or condition loss during lactation and a moderate weight gain over successive pregnancies. However, because the sow has the capacity to store and mobilize body tissue reserves and it is likely that nutrition during the course of a single pregnancy has at most only a limited direct influence on reproductive performance (Aherne and Kirkwood 1985).

PATTERN OF FEEDING IN EARLY GESTATION

The results of many studies have shown that under-nutrition in early gestation has to be very severe to reduce embryo survival (Speer 1982, Pond et al. 1968). Once an ovum is fertilized the embryo is given a very high priority in nutrient supply. Hughes (1989) presented data on the relationship between early gestation feed level and embryo survival for studies in which pre-mating level of feed was standardised. These data show an adverse effect of high plane of feeding in early gestation on embryo survival (Table 4)

Table 4. The effects of feed level in early gestation on embryo survival in sows.(Summary of 12 experiments, feed intake standardized pre-mating).Adapted from Hughes (1989)

	GESTATION FEE High	EDING LEVEL Low	
Ovulation rate	15.4	15.5	
Number or viable embryos	11.8	12.7	
Embryo survival (%)	77	82	

However, the difference between 77 and 82 % embryo survival is unlikely to be statistically significant. Critical to the nutrition of the embryo in early gestation is the supply of uterine specific proteins (USP). The secretion of USP is stimulated by the ovarian steroid hormones, especially progesterone. Dyck et al. (1980) suggested that increased gestation feed intake was associated with a decrease in plasma progesterone and a reduction in embryo survival. Rhind et al. (1989) concluded that embryo mortality is unlikely to be related to lower circulating progesterone levels per se but may be attributable to a reduction in LH pulse frequency. Grandhi (1988) reported no consistent effect of high energy intake in early gestation on embryo survival but plasma progesterone levels were reduced. It has been suggested that high levels of feeding might affect embryo survival by increasing hepatic blood flow and consequently the rate of clearance and plasma concentration of steroids (Parr et al. 1987, Hughes 1989 and Symonds and Prime 1989).

In a recent experiment at the University of Alberta, 48 gilts were fed high or low energy levels and high or low protein levels from three days post-mating to day 28 of gestation. The results showed that ovulation rate did not differ significantly among treatment groups. Level of energy or protein fed had no affect on embryo survival to day 28 of gestation. (Table 5).

Energy intake Protein intake	Low Low	Low High	High Low	High High	±SE	SIG
Food/day (kg) MJ DE/d g protein/d No. corpora lutea No. embryos	1.8 24.7 207 13.7 12.2	1.8 24.7 364 15.1 12.8	2.8 40.2 207 14.2 12.4	2.8 40.2 364 14.8 11.6	0.60 0.55	NS NS
Embryo Survival (%)	85.7	88.9	85.6	80.2	3.80	NS

Table 5. Effect	of energy a	and protein	intak	e during	early		
gestation	on embry	o survival,	(from	Pharazy	n and	Aherne,	1989)

Plasma progesterone rose from day 3 to day 15 but there was no significant difference in levels between treatment groups at days 3 to 15 but, on day 9, gilts receiving high protein levels had higher plasma progesterone levels than gilts with lower protein intakes. Separating gilts on the basis of plasma progesterone suggested that embryo survival was greater (P = 0.09) and variability in embryo survival was smaller (P = 0.07) at the higher concentration of progesterone levels reported in this study were higher than those reported by Dyck et al. (1980) or Grandhi (1988) and it could be speculated that,where higher plasma progesterone levels exist, hormonal status of gilts may be less sensitive to changes in feed intake and an effect on embryo mortality may not occur.

In a second experiment at the University of Alberta sows were fed either 6 kg or 3 kg feed per day during lactation. From 48 hours after mating, sows from each lactation treatment group were allocated equally to receive the same diet at 3.6 kg or 1.8 kg per day until slaughtered at day 25 of gestation. Gestation feeding level did not influence number of embryos or embryo survival for the sows fed 6 kg/day during lactation (Table 6).

6		3	,	
3.6	1.8	3.6	1.8	±SE
28	28	25	24	
13.8a	14.5a	13.1a	11.5b	0.43
80.6a	84.6a	75.8b	67.4c	2.41
	6 3.6 28 13.8a 80.6a	6 3.6 1.8 28 28 13.8a 14.5a 80.6a 84.6a	6 3 3.6 1.8 3.6 28 28 25 13.8a 14.5a 13.1a 80.6a 84.6a 75.8b	$ \begin{array}{c ccccccccccccccccccccccccccccccccccc$

Table 6. Effects of feed intake during lactation and gestation on
the number of embryos, and embryo survival. Baidoo (1989)

However, for sows fed low levels during lactation number of embryos and percent embryo survival were significantly improved by the higher feed intake in gestation. These data suggest an interaction between lactation and gestation feed **intake.The** fewest embryos and greatest embryo mortality were associated with sows on a low plane of feeding in both lactation and gestation. Plasma LH concentrations were also lower in this group. A link between plasma LH and corpora **lutea** function in sows during early pregnancy has been documented (Parvizi et al. 1976). High feed intake in early gestation did not significantly influence plasma progesterone. These data indicate that factors in addition to plasma progesterone may be involved in the determination of embryo survival.

MID-GESTATION

The period from 75 to 105 days of gestation is a critical period in the development of the mammary gland of gilts. (Hacker and Hill 1972, Kensinger et al. 1982). Recently Weldon et al. (1990), have reported that high dietary energy intakes by sows during the period of maximal mammary development (70 to 105 days of gestation) has deleterious effects on total DNA content of parenchymal cells. This reduction in total DNA reflects a reduced cell number which may impair milk production. Recent research at the University of Western Australia (Head, unpublished data) suggests that sows whose diet was manipulated to produce fat or lean sows of the same weight at parturition differed significantly in number of milk secretory cells as the lean sows, and produced less milk.

LATE GESTATION

The nutrient requirements of sows increase with advance in pregnancy following the pattern of foetal development. Foetal weight is doubled over the last month of pregnancy with growth acceleration occurring especially in the last 10 days. However, efforts to increase birth weight of piglets by increasing sow feed intake in late gestation have been variable and unspectacular (Aheme and Kirkwood, 1985). Dietary treatments that increase birth weight and/or glycogen and fat reserves of the new-born piglet may give it a survival advantage if its birth weight is less than 1.0 kg (Aheme and Kirkwood 1985). The major advantage to increasing sow feed intake in late gestation accrues to the sow herself. Cole (1990) reported that a daily energy intake of 39.6 MJ DE/day was required from day 90 of pregnancy in order to maintain P2 backfat (Table 7).

Gestation	Days 0 - 90	Days	s 90 - 110	
Energy intake MJ DE /day	26.4	26.4	39.6	52.8
Weight	36.2	10.0	18.9	26.1
Backfat	4.1	-0.8	0.5	1.7

Table 7: Influence of digestible energy (MJDE/day)intake in late gestation on change in weight (kg) and backfat (P2mm). (from Cole 1990).

These results are in agreement with those of Close et al. (1985) who showed that sows fed 21 MJDE/day mobilized fat at day 87 of pregnancy and that losses during late pregnancy could account for up to 4.8 kg fat or 20 % of fat reserves. Noblet et al. (1990) observed that sows fed 24.7 MJDE/day will mobilize energy reserves during late pregnancy whereas maternal energy balance was zero and sows increased protein deposition and did not mobilize body reserves when fed 30.4 MJDE/day. Forbes (1987) suggested that high plasma estrogen in late gestation may reduce feed intake. It is interesting to speculate that an increased feed intake in late gestation. However, Sterling and Cline (1986) suggest that increasing energy intake in late gestation may result in a decrease in voluntary feed intake in lactation.

LACTATION

The energy and protein requirements of the lactating sow will depend on the weight of the sow, her milk yield and its composition, and the change during lactation in body weight and body composition. In general milk production accounts for about 75 % of the total energy requirements of the lactating sow. The maintenance (MEm) requirement of the lactating sow is about 485 kJDE/kg^{0.75}. Estimates of the energy cost of milk production range from 7.6 to 8.4 MJDE/kg (NRC 1988, Noblet et al. 1990). These values assume an energy content in milk of 5.4 MJ/kg and an efficiency of utilization of DE for energy in milk of 65 - 68 %. The efficiency of utilization of energy from body reserves for milk production is estimated to be 88 % (Noblet et al. 1990). The energy utilization of DE for deposition in maternal tissues in pregnant sows is about 80%. The combined efficiency of tissue gain during pregnancy and its mobilization during lactation therefore is about 68 %. This suggests that the storage of body fat during pregnancy and subsequent utilization during lactation results in an overall efficiency that is very similar to the efficiency of direct utilization of DE for milk production during lactation (Noblet et al. 1990). Based on these and other data ARC (1990), NRC (1988), Cole (1990) and Williams and Mullan (1989), have presented estimates of energy and protein requirements for lactating sows of different body weights and with different levels of milk production. Most of these estimates take for granted that most lactating sows will not consume sufficient feed to meet their nutrient requirements and therefore will mobilize some body reserves in order to meet their requirements. The amount of tissue mobilized will depend upon the extent to which nutrient requirements for maintenance and milk production exceed nutrient intake. Noblet et al. (1990) suggest that the amount of tissue mobilized is equivalent to: (ME requirements - ME intake) x 0.82 where 0.82 represents the ratio between the efficiency of utilization of dietary ME and ME mobilized from body reserves (72:88). Therefore milk production in sows is reasonably independent of energy intake unless the level of body reserves at farrowing is low.

There is considerable evidence that the voluntary food intake of modem day sows during lactation is low and often does not provide sufficient energy or nutrients for maintenance and milk production (Williams and Mullan 1989 and Cole 1990). The NRC (1986) reported that even with ad libitum feeding average daily feed intakes over a 28-day lactation was 4.27 kg (range 3.76 - 4.80 kg) for gilts and 4.90 kg (range 4.30 to 6.60 kg) for sows. Lynch (1989) reported that approximately 40 % of first-litter gilts, 50 % of second-parity sows and 70 % or more of third- and greater-parity sows do not consume the levels of feed recommended by NRC (1988). These sows will mobilize their body tissues, both protein and fat, to meet the demands of milk production. The two most important negative influences on feed intake during lactation are feed intake during gestation and ambient temperature (Mullan 1987, Hughes 1989, Cole 1989, 1990). Cole (1989, 1990) and Noblet et al. (1990), have discussed these and many other factors that effect appetite in sows. Daily lactation food intake is reduced by approximately 2.1 MJDE for each additional 4.2 MJ consumed daily during pregnancy (Noblet et al. 1990). This well-documented relationship between feed intake in gestation and feed intake in lactation is weak in early lactation and for parity-one sows (Whittemore et al. 1988, Young et al. 1990, Noblet et al. 1990).

The mechanism by which body composition modulates food intake is as yet speculative. It has been suggested that the effect of feed intake in gestation on feed intake in lactation is possibly mediated through the level of fatness of the sow at parturition (Whittemore et al. 1988). Matzat et al. (1990) have also reported a significant decrease in feed intake for sows having increased **backfat** at **farrowing**. Recently Williams and **Mullan** (1989) demonstrated that when body fat exceeds about one third of body weight then voluntary energy intake declines (Figure 4). Total body fat of the sow can be calculated using the regression equation of **Mullan** (1987):

TOTAL BODY FAT = -33 + 0.32 LW + 1.105 P2

Using this regression equation it can be calculated that sows of 120 to 180 kg liveweight at parturition can have a **backfat** thickness of 25 mm P2 before level of fatness will have a negative effect on voluntary food intake.



Figure 4. Voluntary food intake (VFI) in lactation vs body fat after farrowing. The regression equation was; $Y = 5.6126 - 0.2521X + 2.9558 \times 10^{-3} X^2 (r^2 = 0.99)$

Whittemore et al. (1988) suggest that the negative effect of increased feed intake during pregnancy on weight loss during lactation is partially but not completely caused by a reduction in voluntary feed intake during lactation. They reported that when sows were fed the same amount of feed in lactation the fatter sows at farrowing mobilized more body reserves. Gainsworthy and Topps (1982 a, b) also reported that high levels of fat at calving had an inhibitory effect on food intake during lactation and fat cows produced more of their milk from body fat then directly from food.

Tybirk (1989) has proposed that the two main regulatory factors involved in the control of food intake are :

(i) metabolic regulation (plasma concentrations of substrates, hormones and neurotransmitters), and

(ii) physical regulation (capacity of the gut).

Among the metabolic regulators suggested to influence feed intake Tybirk (1989) suggests that insulin plays a central role. From the evidence provided by Booth (1990) it is possible that feed intake regulation by insulin is mediated not by plasma insulin but by insulin concentration in the cerebral spinal fluid(CSF). It is known that the concentration of insulin in CSF responds very slowly to changes in plasma insulin. It is possible that sows that are fat at parturition or that have been well fed in pregnancy will have higher concentrations of insulin in their CSF than leaner sows. High levels of insulin in CSF will signal the hypothalamus to reduce food intake. Reduced feed intake will decrease plasma insulin and increase plasma cortisol and catecholamines thus stimulating an increase in lipolysis (mobilization of fat). This hypothesis would explain the all too common scenario in which lactating sows that are fed palatable, wellbalanced diets ad libitum choose to limit their daily feed intake and instead mobilize body tissues to meet the demands of milk production.

A second hypothesis is that it is the circulating levels of certain substrates in the plasma which act as signals of metabolic status to the liver and brain and thus regulate feed intake. Our studies and those of others have shown that feed intake in lactation is negatively correlated with the level of fatness of the sow at parturition. The greater the amount of fat in the body the higher the turnover and the greater the release of fatty acids and glycerol into the blood stream. Similarly upon lipolysis plasma levels of glycerol and fatty acids increase. Because the only possible source of free glycerol in the plasma is from mobilised body fat (free glycerol is not absorbed from the gastrointestinal tract) plasma glycerol itself should provide a very useful index of fat mobilization in the body. It is also known that metabolic receptors in the liver are sensitive to the oxidation of free fatty acids and glycerol. The oxidation of these substrates is believed to alter the firing rate of vagal afferent nerves in the liver that relay information to the central nervous system, in particular to the hypothalamus (Booth, 1990). If this is so then the experimental manipulation of plasma levels of glycerol and free fatty acids should influence food intake through their effect on the hypothalamus.

Many recent investigations have firmly established that sows, especially primiparous sows, losing excessive amounts of live weight or body condition (both protein and fat) will have extended remating intervals, a lower percentage of sows in oestrus within 10 days of weaning, reduced pregnancy rate and reduced embryo survival (Aherne and Kirkwood, 1985, Hughes and Pearce 1989, Cole 1989,1990). Typical of the data reported are those of Baidoo (1987) (Table 8).

	Ad libitum	Restricted	±SEM	
Number of sows	93	92		
Feed/day (kg) Weight loss (kg) Backfat loss (mm)	5.8 14.5 1.8	3.3 42.4 7.2	1.2 0.2	
Days to oestrus Pregnancy rate (%) Ovulation rate Embryo survival (%)	5.1 84.5 16.4 81.4	9.0 65.5 17.2 67.2	1.8 4.2 0.9 2.9	

Table 8: Effect of feed intake in lactation on sow reproductive performance.(from Baidoo 1989)

Feed intake in lactation does not appear to reduce ovulation rate. Hughes and Pearce (1989) suggest that the lack of a difference in ovulation rate from under-feeding in lactation may be due to the longer weaning-to-remating interval allowing an extended flushing period in the sows fed low levels in lactation.

King (1987) suggested a similar influence of both tissue weight at weaning and tissue loss during lactation on weaning-to-oestrus interval. It has been suggested by **Mullan** (1987) that the amount of body reserves at far-rowing and weaning are the critical factors affecting sow reproductive performance, rather than the amount of tissue mobilized during lactation. Thus, a small weight loss in a thin sow may be more serious than a large weight loss in a **well**-conditioned sow. Both fat loss and protein loss have been associated with poor reproductive **performance** (Cole 1990, **Noblet** et al. 1990).

However, several experiments have shown that when first-litter sows consume sufficient **food** during lactation to maintain body weight then the level of reserves at **farrowing** has no effect on subsequent reproductive performance (**Spicer** and Aheme, 1989). These studies and those of Anderson et **al.**(1990) and **Mullan** and Close (1989) suggest that although selection programmes directed principally against **backfat** may have resulted in sows of large mature size and lower voluntary food intake, mobilization of significant amounts of maternal tissue may not be a necessary and unavoidable part of the reproductive cycle as suggested by Lindsay et al. (1991).

A reported range in the energy content of body weight loss in lactation is 12.6 to 27.7 MJ/kg (Noblet et al. 1990). Assuming an energy content of lipid and lean tissue loss of 39.8

MJ/kg and 23.8 MJ/kg it could be calculated that the body weight loss could range in composition from 35 to 80 % lean and 20 to 65% fat (Kotarbinska, 1983) These estimates are consistant with values reported in the literature (Williams and Mullan 1989, Baidoo 1989, Noblet et al. 1990, Cole 1990). It could be concluded that for sow weight loss during lactation of 20 to 40 kg the composition of the weight loss will vary from 50 to 60% lean, with the suggestion that greater or lesser weight loss will contain higher percent lean.

Williams and Mullan (1989) suggested that weaning-to-mating interval is related to live weight of the sow and that she will exhibit the minimum interval of about 5 days if weaned after a lactation of three weeks or more with a weight of 150 kg or greater (Figure 5). Black et al. (1986) suggested that weaning-to-mating interval could be estimated using the regression equation:

Weaning-to-mating interval (days) = 78 - 0.5 weight of sow at weaning (kg).





It is difficult to explain how weight per se could influence the hypothalamo-hypophysealovarian axis. Also in the studies of Baidoo (1989) sows weighing greater then 150 kg at weaning and having greater than 17.3 mm backfat had delayed returns to oestrus. Both Steiner (1987), and Booth (1990) suggested that we should be considering some measure of the animal's metabolic status which would reflect both body composition and mobilization of body reserves.

Little attention has been paid to when the sow becomes anabolic again in late lactation. Brooks and Smith (1980) suggested that the catabolic phase may persist at least through the weaning-to-oestrus period. Booth (1990) considered the physiological mechanisms mediating the effects of weight and/or fat or protein loss in lactation with subsequent reproductive function and suggests an important role for **GnRH/LH** regulation. Several other reports have provided evidence that mean LH concentration and LH pulse frequency are decreased in late lactation and postweaning in sows underfed during lactation (Hughes and Pearce, 1989, King and Martin, 1989, Cole, 1990, Baidoo 1987). Several studies have shown an inverse relationship between LH concentration before weaning and the interval between weaning and oestrus (Shaw and Foxcroft, 1985). A central role of reduced insulin, insulin like growth factor 1 (IGFI) increased plasma levels of cortisol and growth hormone in inhibiting the activity of the hypothalamic-hypophyseal-ovarian axis is suggested (Booth 1990, Baidoo 1989). Baidoo (1989) suggests that a low plasma concentration of LH may allow corpora lutea to luteinize causing a decrease in plasma progesterone and, as a consequence, adverse effects on uterine secretions and embryo survival. Indeed, Kirkwood et al. (1987a), and Kirkwood et al. (1990) have reported reduced plasma progesterone concentration during early pregnancy in sows following low-plane feeding during lactation.

It may be concluded that the decision when to rebreed is made some time before weaning and is mediated by a host of substrates, hormones, and neurotransmitters as shown in Figure 6. The nutritional modulation of reproductive function can occur over a period of hours, days or weeks (Booth 1990), but it is likely that the sow must be anabolic for 10 days before the onset of oestrus will occur. Thus a sow can monitor changes in its own body reserves and through changes in the plasma concentration or fluxes of metabolic hormones and substrates send signals which affect the activity of the hypothalamo-hypophyseal-ovarian axis.



Figure 6. Metabolic and endocrine responses of the lactating sow to changes in nutrient and energy availability (adapted from Foxcroft 1990).

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