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FACTORS INFLUENCING ONSET OF PUBERTY IN GILTS

by

Jennifer Lynne Patterson 

A thesis submitted to the Faculty of Graduate Studies and Research in partial fulfillment of the requirements for the degree of *Master of Science*

in

Animal Science

Department of Agricultural, Food and Nutritional Science

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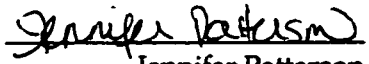

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Dr. George Foxcroft (supervisor)

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Date : 28/03/01

ABSTRACT

Sexual maturation in prepubertal gilts was examined in two studies. Study 1 tested the impact of method of boar exposure on puberty attainment: 1) gilts moved to a purpose built boar stimulation pen (GB), 2) boar moved to gilt pens (BG), 3) fenceline contact between boars and gilts in stalls (BS). GB gilts reached puberty earlier ($p < 0.05$) than BS gilts; BG gilts were intermediate. In study 2 the effects of feeding three different diets on lean growth rates and puberty attainment were studied. Although treatment affected lean growth rate ($p < 0.05$) there was no difference in age at puberty ($p > 0.05$). We conclude that direct exposure to the boar most effectively induces puberty in gilts, and that lean growth rates achieved did not affect age at puberty in gilts. However, late pubertal onset has important consequences for the weight of gilts at first estrus.

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Finally, I would like to thank my all friends and family, especially mom and dad for their support, encouragement and understanding throughout my studies.

This thesis is dedicated to my parents, Robert and Peggy Patterson,
for their love, support, and encouragement.

Table of Contents

LIST OF TABLES

LIST OF FIGURES

Figure A-4 Predicted time of ovulation as a percentage of duration of estrus. Ovulation was predicted to occur at 77.9 % of duration of estrus based on the average for CON and PGF sows. Grey bars represent the 24-h optimal period for ovulation to occur after the first AI. White bar represents the 24-h period for optimal fertilization following the second AI. Black bars represent the period which falls outside the optimal period for fertilization. If PGF were to exert an ovulation advancing effect, possibly more sows would fall within the optimal period for fertilization. 139

Glossary

GnRH	Gonadotrophin Releasing Hormone
LH	Luteinizing Hormone
FSH	Follicle Stimulating Hormone
VNO	Vomeronasal Organ
CNS	Central Nervous System
IGF-1	Insulin-like Growth Factor 1
GH	Growth Hormone
2DG	2-deoxyglucose
T4	Thyroxine
T3	Triiodothryonine
TSH	Thyroid stimulating hormone
TRH	Thyrotropin releasing hormone
HPA	Hypothalamic pituitary adrenal axis
CRH	Corticotropin-releasing Factor
SRIH	Somatostatin
EOP	Endogenous opioid peptides
POMC	Proopiomelanocortin
EAA	Excitatory-amino-acid
GHRH	Growth Hormone Releasing Hormone
NPY	Neuropeptide Y
GHRH	Growth hormone releasing factor
BPT	Back Pressure Test
RIA	Radioimmunoassay
CV	Coefficients of Variance
BMI	Body Mass Index
LEA	Loin Eye Area
FFL	Fat Free Lean
FFLG	Fat Free Lean Gain
FATGR	Fat Growth Rate
FIRE	Feed Intake Recording Equipment
WEI	Weaning to Estrus Interval
PGF2α	Prostaglandin F2α

CHAPTER 1

INTRODUCTION

The success of a commercial swine operation depends largely on the productivity of the breeding herd. In a typical commercial swine operation, the breeding herd is largely composed of multiparous sows. However, due to various reasons, large proportions of these sows are removed from the herd every year. Consequently, approximately 40 % of the reproductive herd is comprised of gilts. As reviewed by Levis (2000), as many as 54.4 % of the gilts selected for breeding purposes are culled without having a litter. As a result, a large pool of cycling gilts is necessary at all times in a herd to replace unproductive sows. Therefore, "gilt pools" have been established to ensure that a producer has a large number of gilts to select from as replacement animals. Without sufficient gilts readily available, service targets cannot be achieved and weaned pig volume declines (Dial et al., 2001). Thus, management of the gilt pool should then focus on reducing non-productive days (NPD) and entry to service intervals (ESI), increasing fertility of first parity sows, and developing programs so that at breeding, gilts have attained a target age, weight, and backfat (Dial et al., 2001; Foxcroft and Aherne, 2001). Therefore, sexual maturation and the attainment of puberty are critical factors in achieving these goals.

Largely based on data from other mammalian species, Foster and Nagatani (1999) and Foster et al. (1994) suggest that there are three developmental stages involved in sexual maturation, which would equally apply to gilts. The first stage is dependent on the development of the appropriate neural circuitry necessary for puberty to occur. In this state, innate differences in sexual development may limit the onset of puberty, irrespective of growth.

The second stage of development relates to the metabolic status of the animal. The metabolic state of the animal can be sensed through changes in circulating hormones such as leptin, insulin, and IGF-1 (Booth et al., 1994; Foster and Nagatani, 1999). Several studies have examined the onset of puberty as related to body condition

or metabolic status of the gilt. Sexual maturation has been associated with achievement of a certain fatness (Gaughan et al., 1997), growth rate (Beltranena, 1992), lean : fat ratio (Kirkwood and Aherne, 1985), metabolic state (Kennedy and Mitra, 1963) and protein mass (Cia et al., 1999). Because the current trend in pig production places emphasis on lean tissue growth rate, the relationships between high growth rates and reproductive performance should be considered. There is very little published information from which to elucidate these relationships. Gilts achieving commercially acceptable growth rates, through the availability of high planes of nutrition, and at a physiological age appropriate for induction of puberty, are not likely to be limited by some component of growth (Foxcroft, 1993). Gilts would likely possess a minimum threshold of growth necessary for pubertal onset. Any delay in the onset of puberty is thus likely to be due to the lack of development of the appropriate neural circuitry. From the data collected in our studies we were able to examine the effect of high lean growth rates on the attainment of puberty in gilts. However, the consequences on subsequent reproductive performance when the gilt enters the breeding herd remains to be examined.

The third stage of development involves the integration of several external cues regulating the onset of puberty, such as housing (Dyck, 1989), nutrition (Zimmerman et al., 1960; Le Cozler et al., 1999; Klindt et al., 1999), transportation and mixing (Pearce and Hughes, 1987) and the boar effect (Hughes et al., 1990). Although, numerous earlier studies have confirmed that direct exposure to boars is necessary for maximum stimulation (Zimmerman et al., 1998; Pearce and Hughes, 1985), fewer studies have examined whether the type of direct exposure is a determining factor in puberty stimulation (van Lunen and Aherne, 1987). It was considered important to re-examine this topic to determine which method of boar exposure is most effective in inducing early puberty in gilts in a modern, commercial production system.

The work undertaken in this thesis examines the effect of method of boar exposure and the effect of lean growth rate on puberty attainment in gilts. The second chapter is an in-depth review of the literature, examining the pertinent factors relating to sexual maturation in the gilt. The two studies undertaken are presented as sequential

chapters in an extended form of the papers that have been submitted for publication. In the final chapter of this thesis, the results from both studies are combined, and the implications of the two experimental designs are discussed as they relate to management and production implications. Future studies that could be used to further examine the effect of high growth rates on puberty attainment and the effect on subsequent reproductive performance are briefly discussed.

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CHAPTER 2

LITERATURE REVIEW

2.1 Biology of growth

Growth, in simplistic terms, is an increase in size, and total growth of an animal is the sum of the growth of all its parts (Price, 1996). Growth can occur in three ways: positive growth (weight gain), where tissue anabolism exceeds catabolism; negative growth (weight loss), where tissue catabolism exceeds anabolism; and static growth (weight maintenance), where tissue catabolism equals anabolism. Accretion of bone, fat and lean tissue are processes that involve positive growth.

A component of growth, known as development, relates to changes in shape, form and function of animals as growth progresses. Most development occurs prenatally and immediately postnatally (Price, 1996). Growth over time can be plotted as a sigmoid curve, starting from conception and continuing to maturity. Growth prior to birth occurs through hyperplasia (increase in cell number) and through hypertrophy (increase in cell size) after birth.

Growth in the pig can primarily be described in terms of its protein and lipid components. The relative proportion of protein and lipid in each kilogram of dissected lean varies among pigs. However, on average 1 kg of lean contains 20-23% protein, 70-75% water and 5-15% fat, and 1 kg of fat contains 80-95% lipid, 10-25% water and 2% protein (NRC, 1998). According to the NRC (1998) the energy cost of protein retention is 44.4 MJ of ME/kg, and that of fat retention is 52.3 MJ of ME/kg. Therefore, although the energy cost of production of protein or lipid deposited are approximately equal, lean is approximately four times more efficient to deposit than fat.

Whittemore (1998) further breaks down the cost of protein retention. One kilogram of protein retained in the body contains on average 23.6 MJ of energy. The energetic cost of protein retention associated with protein turnover (the process of

protein synthesis and degradation) requires, on average, 31 MJ. Therefore, the total energy cost for protein retention is the sum of the energy in one kilogram of retained protein plus the energetic cost of turnover, which will total 55 MJ of energy. Although this number is not equal to the value stated in the NRC (1998) it is well within the established range of values (28 – 59 MJ/kg).

The energetic cost of one kilogram of retained lipid is 39.3 MJ (Whittemore, 1998). Most of the energy associated with lipid retention is due to synthesis, as there is very little energy spent on turnover. In total, approximately 14 MJ/kg of energy is required for these processes. Therefore, the total energy cost of lipid retention is the sum of that for fatty tissue turnover and synthesis and the total cost in retained tissues, which is equivalent to 53 MJ/kg (Whittemore, 1998). This value is nearly equivalent to that in the NRC (1998) and again, fits well within the cited range.

2.1.1 Growth curves

Growth can be described by a sigmoid curve and there are three parts to the curve: accelerated growth, constant growth, and decelerated growth (Brody, 1945). In the accelerated growth portion of the curve, there is multiplication of individual cells, or organelles within the cells that can proceed without inhibition. The environment will then begin to offer resistance, whether it is by limiting nutrients or environmental limitations, causing a gradual slowing of growth. At this time, there will be an inflection in the curve and there is subsequently a period of relatively constant growth. As the environment continues to offer resistance, growth will enter a self-inhibiting phase, which is asymptotic (Price, 1996). Huxley (1932) first noted the fixed nature of the relationships between body components and the whole body during growth. It is important to note that the body-weight curve is the sum of all the separate growth curves of each part of the body (Price, 1996).

As a pig increases in age and liveweight, characteristic changes occur in its composition. Lipid and protein mass follow the typical sigmoid pattern of growth, and show a period

of acceleratory and deceleratory growth with an intermediary period of relatively constant growth (Whittemore, 1998). Whittemore (1998) suggests that there is a relative constancy of the lipid:protein ratio (1:1) between 25 and 100 kg liveweight. The asymptote of lipid mass is greater than that of protein and occurs later in time.

2.1.2 Lean tissue growth

In a stress-free environment and with adequate intakes of essential nutrients, protein deposition is determined by either energy intake or the genetically determined upper limit to body protein deposition (Möhn and de Lange, 1998). The upper limit for protein growth is determined by genotype and is approached when environment and nutrition are not limiting. However, in commercial conditions the maximum protein growth potential is seldom reached, and at best, only 75-80% of optimum protein growth is achieved (Schinckel and de Lange, 1996). The rate of protein (lean) deposition achieved by growing pigs is a function of a wide variety of environmental influences (temperature, humidity, herd health, social structure, space allowance, etc.) but is primarily based upon dietary amino acid and energy intake (Whittemore et al., 1988).

2.1.2.1 Genotype

Over the last several years, selection and performance testing have improved growth rate, feed efficiency, and carcass composition (Bikker and Bosch, 1998). Genotype, including sex, has significant effects on the pigs potential lean growth rate. These two factors are often related. The main difference between genotypes involves the overall mean protein accretion rate and the rate at which protein accretion declines after 90 kg live weight (Schinckel and de Lange, 1996). Typical differences occur between sexes: intact males have the highest lean growth potential, followed by gilts, and lastly castrated males (de Lange, 1998). Fuller et al. (1995) examined the response of growing pigs of different sex and genotype to different dietary energy and protein. Consistent with the literature, these researchers found that intact males grew faster than either females or castrated males and that genotype had significant effects on daily gains of

carcass protein and lipid. The authors suggest that a genotype's superiority may result from a difference in energy utilization, resulting in greater efficiency of protein utilization or a higher lean growth potential.

Schinckel and de Lange (1996) state that there are four growth parameters required to obtain the estimates of growth that characterize pig genotypes: 1) daily whole-body protein accretion potential; 2) partitioning of energy intake over maintenance between protein and lipid accretion; 3) energy requirements for maintenance; and 4) daily feed intake. The potential protein curve changes as body weight increases, while the shape of the curve reflects the changes that occur in the composition of the pig. In growing pigs under 50 kg, energy intake will determine the rate of protein deposition, whereas in finishing pigs over 50 kg, the upper limit to protein retention will determine the rate of protein deposition (de Lange, 1998; Schinckel and de Lange, 1996). The percentage of whole-body protein accretion increases from birth to approximately 45 to 65 kg (as the percentage of water decreases), whereas after 65 kg, the percentage of body protein decreases as the percentage of lipid increases (Schinckel and de Lange, 1996). Whole body protein includes protein in all body parts, including the head, carcass, organs, skin and bone (Schinckel, 1996).

2.1.1.2 Energy and protein intake

Maximal lean growth can be achieved only when nutrients and energy are provided in the appropriate ratio (Lawrence et al., 1994). In the growing pig it is well established that the optimum dietary lysine:digestible energy ratio is dependent upon daily live-weight gain and nitrogen deposition rate or the protein deposition rate potential of the animal (van Lunen and Cole, 1998). If energy is supplied in surplus of what is required for maintenance and protein deposition, the excess will be deposited as fat, while protein in excess of requirements will need to be deaminated and excreted (van Lunen and Cole, 1996). Reductions in dietary protein intake, but not energy intake, restricts lean tissue growth through inadequate availability of amino acids and allows redirection of dietary energy into fat deposition (Cia et al., 1998). When both energy and

protein are limited, fat and protein deposition rate will be reduced (van Lunen and Cole, 1996).

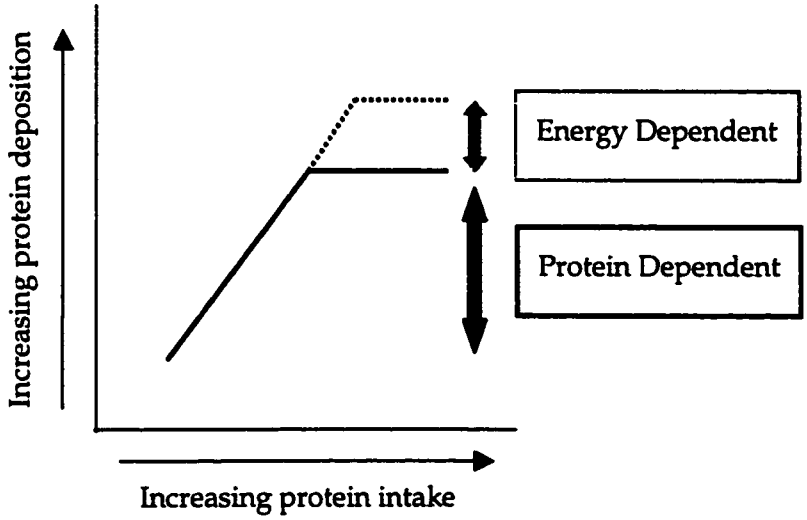

Figure 2.1 Relationship between energy and protein intake on protein deposition *(adapted from Bikker and Bosch, 1998).* Two different levels of energy intake are represented by "high" energy ----- and "low" energy ——— . In the protein dependent phase increasing energy intake (with adequate supplies of protein) will increase protein deposition. In the energy dependent phase, increasing protein (without adequate levels of energy to support protein deposition) will NOT further increase protein deposition.

The accretion of body protein requires both dietary energy and protein and there are typically two phases, an energy and a protein dependent phase as shown in Figure 2.1. When protein intake is below requirement, protein deposition is linearly related to protein intake and unaffected by energy intake (protein-dependent). When protein intake is in excess of requirement, protein retention is related to energy intake and unaffected by protein intake (energy-dependent) (Campbell, 1988). Energy intake plays an important role in the growth of pigs. At a constant energy intake, an increase in dietary protein causes a linear increase in daily protein deposition, indicating that protein gain is dependent on protein intake (Bikker and Bosch, 1998). A plateau in protein deposition occurs when the rate of protein deposition is maximized, and further increases in protein intake will not increase protein deposition. However, if energy

intake is increased, protein depostion can be improved (Bikker and Bosch, 1998). At this point, the lipid:protein ratio remains relatively constant at about 1:1. When energy is supplied in excess of requirement for maximum protein retention, the surplus energy will be directed towards lipid deposition (Schinckel and de Lange, 1996). The lipid: protein ratio will be greater than one. If energy intake is less than optimum, lipid retention will first be limited without affecting the daily protein deposition rate. However, further energy restrictions will reduce the rate of protein deposition.

Daily protein gain has been shown to increase with increasing intake of protein (Fuller, et al. 1995; Bikker and Bosch 1998). In the energy dependent phase, the utilization of protein and the upper limit to protein deposition is largely dependent on the digestibility, the availability and the amino acid composition of the available dietary protein (Bikker and Bosch, 1998). When the protein supply is less than optimal, any increase in energy will not increase protein deposition, but will lead to an increase in fat deposition in pigs.

When protein is supplied in excess of the amount needed for maximal protein accretion, heat production is increased and the efficiency of energy utilization is reduced (Campbell, 1988). It has been shown that the performance of growing-finishing pigs decreases as dietary crude protein concentrations are increased from adequate to excessive (Chen et al., 1995). Barrows and gilts showed a decrease in feed intake, average daily gain and fat accretion with an increase in liver, kidney and pancreas weights with increasing crude protein in the diet. Fluharty and McClure (1997) found that *ad libitum* fed ewes and wethers had significantly higher liver and kidney weights than their counterparts fed 85% of *ad libitum* intake. Chen et al. (1999) stated that finishing pigs fed high-protein diets utilize energy less efficiently, because more energy is required for ridding the body of excess protein; therefore, less energy will be available for growth.

The optimal amino acid:energy ratio decreases with increasing body weight of the pig, because increasing amounts of energy are used for maintenance and for lipid

deposition (Bikker and Bosch, 1998). van Lunen et al. (1998) suggested an optimal lysine:energy ratio for starter pigs from 5 to 25 kg was 1.2g/MJ. Bikker and Bosch (1998) reported that the range of lysine:energy ratios for pigs from 50 to 100 kg may range from 0.35 to 0.55 g/MJ. The recommended lysine:energy ratio in the literature varies, but in general, increasing lysine:energy ratio up to a point where it does not exceed 1.0 g/MJ, resulted in increases in daily liveweight gain and nitrogen deposition (Van Lunen and Cole, 1996) for fast growing genotypes from 25 to 90 kg. Similar results were obtained by Castell et al. (1994) and Cia et al. (1998) where increasing the lysine:energy ratio increased daily liveweight gain. Lysine:energy ratios beyond this point result in decreased gains and deposition rates.

2.1.1.3 Environmental stress

Crowding of pigs, excessive environmental temperatures and disease-causing organisms can prevent pigs from expressing their full, genetic performance potential (de Lange, 1998). Crowding and mixing of pigs depress growth rate by 15.7 and 7.1%, respectively (Hyun et al., 1998). As previously discussed, excessively high or low temperatures, will have detrimental effects on feed intake and growth performance. Some disease-causing organisms have been shown to decrease the lean potential of the pig by 30% (de Lange, 1998). The removal of pigs from a pen may reduce lean tissue growth rate due to environmental changes such as floor space, feeder space, social hierarchy, and effective temperature (Schinckel and de Lange, 1996).

2.1.3 Fat growth

Fatty tissue growth acts as a buffer or 'safeguard' to the environment. In comparison to lean, fat is later maturing and remains as a depot for excess energy long after the other tissues have ceased growing. There are several factors affecting fat growth: 1) unbalanced diets; 2) feed intake exceeds requirements for maintenance and lean tissue growth; 3) a change in the priority of accretion and 4) mature lean mass target is achieved. Total body lipid is comprised of three components: essential, target, and variable fat. Essential fat, accounting for approximately 5% of total fat, is the

minimum amount of fat required for normal metabolic function (Whittemore, 1998). Pigs have an increasing need to deposit essential body lipid as body weight increases (Möhn and de Lange, 1998). Target fat is the minimum amount of fat the body will lay down with gain. Once an animal achieves its target fat level, nutrients and energy can be used for other processes such as lean growth and pregnancy. At levels of fatness below target, the achievement of target levels will detract from the achievement of potential rates of protein retention (Whittemore, 1998). Until target levels of fat are reached, the animal will then direct nutrients and energy to reach that level, possibly at the expense of other processes (i.e. lean accretion, reproduction). Variable fat is any level of fat above target fat.

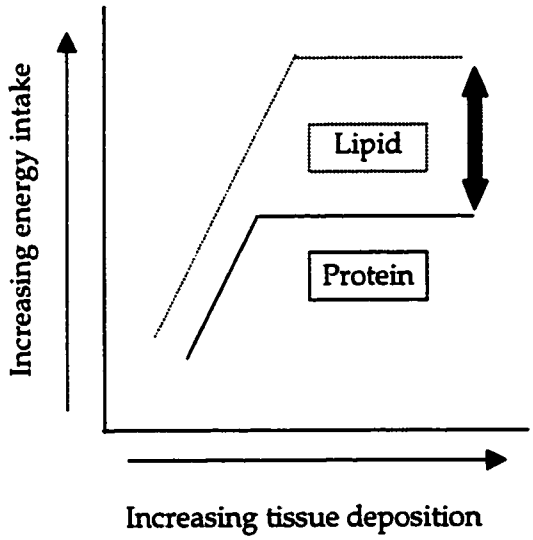

Figure 2.2 Relationship between increasing energy intake on protein and lipid deposition. *(adapted from Bikker and Bosch, 1998).* Increasing dietary energy intake above what is required for protein deposition will result in increases in lipid.

As shown before, the fat content of pigs increases with increasing body weights (Bikker and Bocsh, 1998). Unlike protein deposition, lipid deposition does not plateau or reach a maximum level. Energy supplied above requirements for protein deposition and maintenance will do nothing else but increase the deposition of fat (Figure 2.2). When maximal protein deposition is not yet achieved, a decrease in energy will result in

loss of variable lipid reserves, as the body's first goal is to maintain protein deposition. Another scenario where variable lipid levels would be reduced involves the situation where protein is supplied in excess, and thus energy will need to be diverted and used in deaminating the excess protein.

2.2 Puberty

Puberty can be defined as the phase that links immaturity and maturity and is the time of life in an animal when reproductive activity is initiated (Hughes and Varley, 1980). Gradual changes occur during development. These changes reflect a chain of events originating in the brain, including increasing production of sex steroids by the gonads occurring in response to the increasing secretion of gonadotrophins from the anterior pituitary gland; this, in turn, is driven by the increased secretion of GnRH from the hypothalamus (Foster and Nagatani, 1999).

2.2.1 Stages of development

How the body understands when is the appropriate time to begin high-frequency GnRH secretion is unknown, but it must relate to an organism's ability to sense changes in growth, possibly through changes in energy metabolism, and then to regulate their reproductive development accordingly (Foster and Nagatani, 1999). Prunier et al. (1993) reported that delayed puberty in underfed gilts is associated with an inhibition of pulsatile release of LH. Furthermore, Booth et al. (1994) demonstrated that short-term changes in metabolic state could produce significant differences in pituitary responsiveness to a GnRH challenge in the absence of differences in carcass composition. Foster and Nagatani (1999) hypothesized that the prepubertal rise in GnRH secretion may be the result of: 1) a changing energy metabolism with growth necessitated by a changing soma, 2) increasing energy reserves (fat), or 3) blood-borne signals reflecting changing energy metabolism and energy reserves.

Furthermore, Foster and Nagatani (1999) introduced the concept that multiple sequential signals may be involved during sexual maturation and that a developmental

block exists for the proximate signal to work at some stage. They hypothesize that there may be three developmental and responsiveness stages to metabolic cues and the timing of puberty (Figure 2.3). The period in which the development of neural pathways occurs and when the GnRH neurosecretory system is unresponsive to metabolic signals would be stage one. Foster et al. (1994) reported that perhaps the establishment of a functional GnRH neurosecretory system is solely governed by a genetic clock and is invariant in its time course of development.

Once the animal has developed the necessary neurosecretory system, it must have sufficient energy available to carry out reproduction (Foster et al., 1994). Adequate nutrition is a key component for the onset of normal reproductive function in animals. All animals possess the mechanisms to partition available nutrients according to priorities that ensure survival and long-term reproductive success (Wade and Schneider, 1992). Cellular maintenance, thermoregulation, and locomotion receive high priority because they are essential for the survival of the individual. When there is any deficit in nutrient availability, growth and reproduction will be compromised. As a result, underfeeding or inadequate diet formulation will have profound affects, resulting in decreased growth, and delayed puberty and sexual maturity.

During stage two, the GnRH neurosecretory pathways are in place and the animal achieves the appropriate energy balance to begin high frequency GnRH secretion (Foster et al., 1994). The systems regulating energy balance would send information to the appropriate systems signaling that sufficient resources are in place for reproduction to proceed. Foster et al. (1994) provided an alternative hypothesis at this stage; they predicted that it might be possible that development of the appropriate circuitry occurs after the attainment of an appropriate metabolic state. Similarly, Foxcroft (1980) stated that the attainment of a particular stage of growth might not necessarily be the effective stimulus for the attainment of puberty; the limiting factor may be the maturation of the positive feedback mechanism. Foxcroft (1993) stated that if high planes of nutrition are made available, then growth rate and body composition are unlikely to limit the attainment of puberty.

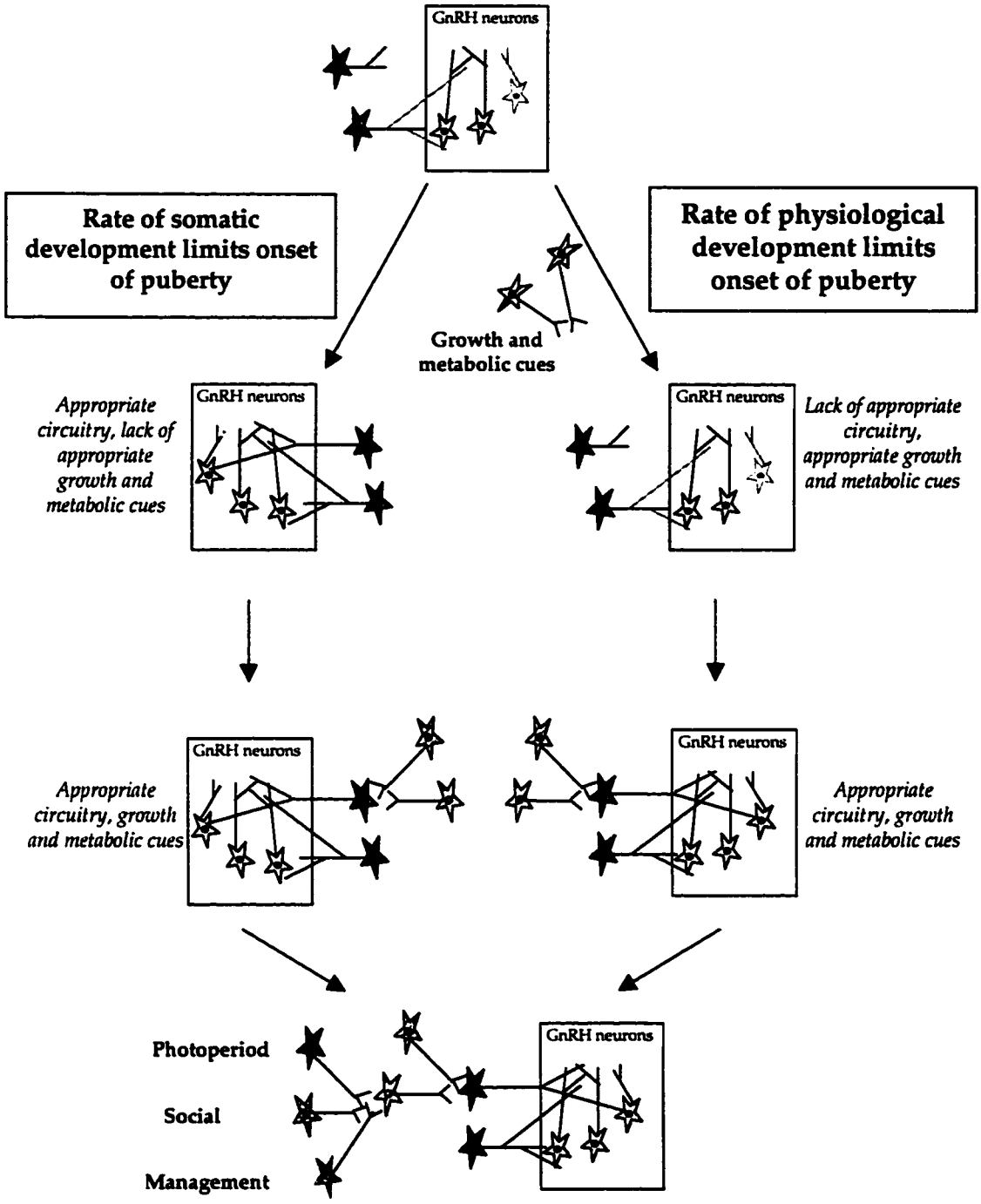

Figure 2.3 Factors regulating the onset of reproductive activity (redrawn from Foster et al. 1994, and Foster and Nagatani, 1999)

During stage three, environmental cues such as photoperiod, social stimuli, and management are integrated with the metabolic cues in timing the onset of puberty (Foster and Nagatani, 1999).

2.2.2 Early pubertal development

Commencing early in gestation in the embryo, the reproductive system undergoes a series of developmental changes that result in the attainment of puberty (Dyck, 1988). Elevated levels of LH and FSH are characteristic of neonatal animals. During the first 30 to 50 days of life, FSH levels are maintained at levels equal to or greater than neonatal levels. During the period from 50 to approximately 150 days, circulating levels of FSH decrease. As puberty approaches, FSH levels remain relatively stable during the late pubertal period.

Circulating LH levels decrease after birth, reaching a nadir in the mid prepubertal period (Foxcroft et al., 1994). The rate of LH decrease after birth may be species-specific, as an immediate decrease in LH has been detected in gilts, while in female rats, the decrease in LH occurs at approximately 35 days of age. The pattern of LH change may be related to litter of origin and growth rate of young gilts (Deligeorgis et al., 1985). LH increases from mid to late prepuberty and reaches a plateau as puberty approaches (Foxcroft et al., 1989).

2.2.3 Prepubertal development

Lutz et al. (1984) characterized the serum profiles of LH and estrogen in the period immediately before the onset of puberty. LH was shown to increase in frequency and decrease in amplitude during the immediate prepubertal period. However, Diekman and Trout (1984) reported contradictory evidence that a decline in the frequency of LH was evident as gilts approach puberty. These differences most likely reflect variations in experimental design, not actual differences in prepubertal LH

profiles. Lutz et al. (1984) demonstrated that serum estrogen levels rose during the late prepubertal period in synchrony with LH.

The endocrine changes that occur in the immediate prepubertal period, resulting in first ovulation, have been well documented (Hughes et al., 1990). However, how these changes occur is poorly understood. The increase in gonadotrophins may be a result of one or more developmental changes: 1) a reduction in the sensitivity of the hypothalamic-pituitary unit to estrogen (gonadostat theory); 2) a reduction in the relative clearance of estrogen; 3) increased GnRH output from the hypothalamus, or 4) increased pituitary sensitivity to GnRH (Hughes et al., 1990).

The "gonadostat" theory can be used to explain the regulation of the release of gonadotrophins during development (Ramirez and McCann, 1963). Hughes and Varley (1982) outlined five phases of pubertal development, which involved a reduction in the sensitivity of the hypothalamic–pituitary unit to the inhibitory effects of estrogen and thus a change from negative to positive feedback of estrogen. Expression of the stimulatory estrogen feedback mechanism is essential for the onset of cyclic ovarian activity, because it triggers the surge release of LH, which in turn induces ovulation (Elsaesser, 1982). Elsaesser (1982) and Foxcroft et al. (1989) reported that there is no evidence for negative feedback regulation of endogenous ovarian factors on LH secretion in the gilt before day 60 and 80, respectively. Prior to this time, the lack of negative feedback may be related to a lack of ovarian secretory activity *per se* (Foxcroft et al., 1989). An estrogen challenge on day 6 and on day 60 resulted in suppression of LH. However, in the late prepubertal phase, administration of estrogen can induce premature ovulation, supporting the theory of a change in estrogen sensitivity of the hypothalamic-pituitary unit. Dyck (1988) demonstrated that an injection of estradiol benzoate at 160 days of age had a stimulatory effect on the occurrence of estrous, possibly involving a direct effect on the behavioral centers in the brain and stimulation of LH. Although the gilts were younger at puberty, an injection of estradiol benzoate resulted in more gilts being in estrus without ovulation, a reduction in ovulation rate, and an increase in the number of gilts with cystic ovaries (Dyck, 1988).

2.3 Factors affecting the onset of puberty

It has been well established that several factors play a role in the onset of puberty. For the purpose of this review, these factors are divided into three categories: environmental cues, nutrition and growth cues, and the effect of litter origin. Sexual development in gilts can be affected by numerous environmental cues: the boar effect, type, frequency and duration of boar contact, and housing. Nutrition and growth traits that influence pubertal onset include body weight, age, growth rate, fatness, lean growth, and metabolic rate. These effects are mediated through metabolic signals acting on the hypothalmo-hypophyseal-ovarian axis. Litter of origin has been shown to have significant effects of gilt performance.

2.3.1 Environmental cues

2.3.1.1 Boar effect

It is well established that exposure of prepubertal gilts to a mature boar will stimulate puberty (Dyck, 1988; Hughes et al., 1990). Introduction of mature, intact boars increased LH pulse frequency in gilts within six hours of initial contact (Kingsbury and Rawlings, 1993). In ewes, there is a rapid increase in the frequency of LH pulses, and a simultaneous increase in mean and basal levels that is presumably the result of pheromones (Martin et al., 1983). The ram stimulus is thought to act by inhibiting the negative feedback exerted by estradiol. It is suggested that the ovary responds to the higher concentration of LH by secreting estradiol, which in turn is responsible for the final maturation of the positive feedback system in the hypothalamus (Paterson, 1982). The boar has proven to be a potent stimulus for the onset of puberty in gilts. The stimulatory effect of the boar can be attributed to auditory, visual, olfactory and tactile cues (Hughes et al., 1990).

Although the data are somewhat limited, auditory and visual cues appear to play only a minor role in inducing puberty in gilts. Pearce and Hughes (1987a) have demonstrated that visual cues provided by a castrated male were less effective than an

intact male in stimulating puberty in gilts. Recordings of boar chants have been shown to be effective (Kinsey et al., 1976) and ineffective (Pearce and Hughes, 1987a) in inducing puberty.

Tactile cues essentially involve any physical interaction between boar and gilts, and may therefore refer to both casual contact and full contact behavior (Hughes et al., 1990). The stimulatory effect of the boar on inducing puberty in gilts has also been attributed to a significant elevation of cortisol (Pearce and Hughes, 1987b). Turner et al. (1998) demonstrated that introduction of gilts to boars resulted in a significant transient increase in plasma concentrations of cortisol, while there was no significant effect of using the back-pressure test during fenceline contact on plasma cortisol. In this study, introduction of gilts to boars may have stimulated some aspects of reproduction, because ovulation rate was increased in gilts that were introduced to boars compared with gilts that underwent the back-pressure test. The mechanism of this effect is thought to result from an increase in basal serum LH concentrations due to elevated cortisol, which may result in accelerated follicular development and estradiol production (Liptrap and Raeside, 1983). Pearce et al. (1988a) suggest that acute elevations in plasma cortisol concentration may be involved in mediating changes in the pituitary responsiveness and the secretion of LH in the prepubertal gilt. However, Kingsbury and Rawlings (1993) reported that the ability of a boar to induce puberty in gilts does not involve cortisol and, in fact, cortisol may have inhibitory effects. These authors found that gilts that released the most cortisol were most likely not to show signs of early estrus. However, in the study of Kingsbury and Rawlings (1993), only fenceline exposure to a mature boar was used, whereas Pearce and Hughes (1987b) found that full physical contact with the boar was necessary to induce the stress response in gilts.

The available literature relating cortisol to LH release seems to be conflicting. Sustained levels of cortisol impaired the LH surge and ovulation (Barb et al., 1982). Rivier and Rivest (1991) have shown that stress-related hormones impact reproduction by interfering with GnRH release and GnRH-induced LH release. However, they also concluded that the negative effects typically associated with reproduction are involved

in long-term stress and not acute stress stimuli. Furthermore, Turner et al. (1998) concluded that for cortisol to impair reproduction, its secretion must be elevated for a prolonged period of time. They found that in gilts with sustained levels of cortisol, LH amplitude, duration of surge, number displaying estrus, length of estrus, and ovulation rate were decreased compared with gilts with repeated acute elevations of cortisol.

As reviewed by Paterson (1982), olfactory stimuli can be attributed to pheromones, which can be divided into two categories: signaler pheromones, which cause a behavioral change, and primer pheromones, which cause a physiological change. Primer pheromones alter the endocrine status of the prepubertal gilt (Hughes et al., 1990). Kirkwood et al. (1981) concluded that the main stimulus involved in the boar effect was olfactory in nature. Prepubertal gilts made anosmic by surgically removing the olfactory bulbs from the brain were slightly older at puberty, suggesting that boar odors are involved in the overall male effect. It is well established that 16-androstene pheromones from the boar's submaxillary salivary glands are the primary pheromones involved in the boar effect (Pearce and Hughes, 1987b). Pheromaxein, a pheromone binding protein, must be present to transfer the pheromones from the boars submaxillary salivary glands to the vomeronasal organ (VNO) of the gilt (Booth, 1984; Pearce et al., 1988b). In order for this to occur, head-to-head contact between the boar and the gilt is essential. Pearce and Paterson (1992) found that the use of snout masks prevented contact of the gilt's snout with the sources of the boar's pheromones, resulted in reduced efficacy of puberty stimulation compared with full boar contact.

Ultimately, the boar effect operates via a synergistic action of several boar component stimuli including salivary and urinary pheromones, tactile and possibly visual and auditory stimuli (Hughes et al., 1990). There are several methods by which a boar can be used to stimulate puberty in gilts, and their effectiveness or ineffectiveness may be largely due to which boar component stimuli the gilt is exposed to.

2.3.1.2 Age of gilt at first exposure

Gilt age at first exposure to a boar will influence the age and interval from boar contact to puberty (Levis, 1997). The age range over which gilts reach first puberty is quite variable and dependent on a number of factors. However, Levis (2000) suggests starting boar exposure between 150 to 170 days for crossbred gilts and 170 to 190 days for purebred and late-maturing animals. The distribution of gilts reaching puberty is largely dependent on the age at first boar exposure (Levis et al., 2000). If a large number of gilts are required to reach a synchronous puberty, commencing boar exposure at an older age is desirable.

2.3.1.3 Age and sexual behavior of boar

As previously mentioned, the effectiveness of a boar in stimulating puberty is partially dependent on pheromones. A major source of pheromones is the submaxillary portion of the salivary gland, which does not commence development until about six months of age (Levis, 1997). It is suggested that boars be at least 10 months of age and exhibit a high libido. The proceptivity and sexual receptivity of gilts was increased when olfactory and auditory stimuli were added to 6-7 month old boars but not when added to 9-10 month old boars (Soede, 1993). Boars with a low libido are less effective in stimulating a younger puberty than boars with a high libido (Zimmerman et al., 1997; Hughes, 1994).

2.3.1.4 Type of boar contact

Siswadi and Hughes (1995) reported that daily boar exposure was effective in reducing the interval from initial boar exposure to puberty and increasing the proportion of gilts pubertal after 60 days on treatment. They concluded that full boar exposure is a potent stimulus for early puberty attainment in gilts and that full physical contact with the boar must occur to achieve the boar effect. Deligerorgis et al. (1984) and Karlbom (1981) reported similar results. It has been shown that physical contact between a boar and a group of gilts is a more effective stimulus than fenceline contact

for triggering an earlier first estrus in gilts (Pearce and Hughes, 1985; Zimmerman et al., 1998). Zimmerman et al. (1998) reported that when boar exposure was initiated at 160 days of age, boar contact effectively reduced the number of days to estrus by 9.5 days compared to fenceline stimulation. There are several methods by which a boar and gilt can have direct contact: moving the gilt to the boar pen, moving the boar to the gilt pen, moving both the boar and gilts to a common heat detection area, and continuously housing the boar and gilts together. Often housing, time, and labor constraints determine which method of boar exposure is utilized. van Lunen and Aherne (1987) reported that continuous boar exposure, or 30 minutes of daily exposure after moving gilts to a boar pen, were more effective than 30 minutes of daily exposure after moving a boar to the gilts' pen, fenceline exposure of gilts to a group of four boars, or no boar contact. However, in this experiment, gilts were only exposed to boars for a 10-day period after which all boar contact was removed, and a technician completed puberty detection in the absence of boar contact. Moreover, no significant differences were detected in age at puberty between treatments, and the only valid difference detected in the percent of gilts in heat by 270 d was between non-boar exposed gilts, and 24-h exposure and 30-min exposure of gilts moved to boar pen (29.4 vs 65.2 and 68.2 %). It is, therefore, not appropriate to conclude that the most appropriate method for detection of puberty is continuous boar exposure or 30-minute daily exposure moving gilts to a boar pen. Pearce and Hughes (1985) examined the effect of daily movement and the environment in which gilts were exposed to a boar on puberty attainment in gilts. The median interval to puberty in gilts exposed to a boar was significantly shorter than non-boar exposed gilts. However, no difference existed between gilts exposed in the boar pen or a "new" common pen. Table 2.1 summarizes the effect of type of boar exposure on puberty attainment.

One factor that must be considered is the ratio of boar to gilts used. It can be assumed that a larger ratio would result in a shorter time that the boar could stimulate each gilt. Therefore, the effectiveness of the reported results could be somewhat confounded if the ratios of boar to gilts were different. Generally, the ratios of boar to gilts in the studies reported were above similar.

Table 2.1 Summary of various techniques to stimulate puberty in gilts

	Exposure age	Boar : Gilt	Time (min)	Treatment		Interval or Age (d)
Zimmerman et al., (1998)	160	1 : 8	10	Fenceline	1x	36[I]
					2x	23.4
				Full Boar	1x	20.9
					2x	19.6
				Continuous	-	31.9
Van Lunen and Aherne (1987)[1]	130	1: 4	30	Gilts-Boar	-	209.9[A]
				Boar- Gilts	-	211.8
				Continuous	-	195.1
				FC[2] 4 – boars	-	211.7
				NC[3]		212.2
Pearce and Hughes (1985)	165	1:6	30	NC	-	80[aI]
				Gilts-boar	-	45.5[b]
				Gilts-empty pen	-	47.5[b]
				Gilts moved	-	76.5[a]
				Gilts – empty boar pen	-	80[a]

[1] Gilts received treatment for a period of 10 days only, after which all gilts were examined for signs of puberty by a technician
[2] FC – Fenceline contact with four boars
[3] NC – No contact of gilts with boars
[I, A] Interval from initial boar exposure to puberty, Age at puberty

In large modern production units, there is pressure to maintain a continuous flow of cyclic gilts to meet breeding targets. It is important that a producer adopt a method of puberty stimulation that most effectively induces early puberty in gilts. However, contemporary experimental evidence regarding the most effective method of puberty stimulation is lacking.

2.3.1.5 Frequency and duration of boar contact

Although the importance of direct boar contact was emphasized in the preceding discussion, the frequency and duration of boar contact must be considered. This is an important factor to consider because it will affect the efficacy of the gilt's response to the boar. Zimmerman et al. (1998) and Hughes and Thorogood (1999) demonstrated that puberty stimulation twice-a-day reduced age at puberty and the interval to estrus after initial boar exposure compared with once-a-day stimulation. These authors also suggest

that twice-a-day puberty stimulation may be more effective in gilts receiving fenceline contact than full boar contact. These extra stimuli may be necessary to make up for lack of boar stimuli in fenceline stimulation. Philip and Hughes (1992) found that increasing the frequency of boar exposure while holding duration of boar exposure constant, enhanced the pubertal response of the gilt. Continuous fenceline contact with boars was an ineffective method to stimulate early puberty in gilts (Zimmerman et al., 1998). This may be a result of habituation to the boar. Eastham et al. (1984) provided conflicting evidence as to whether rearing gilts with fenceline contact with a boar adversely affected age at puberty, ovulation rate, or embryo survival. Paterson et al. (1989a) reported that the proportion of gilts reaching puberty was not different between treatments consisting of 2, 5, or 7 times a week boar exposure. However, the mean interval to puberty varied over season and was affected by the number of times a week that gilts received boar contact. Gilts exposed to a boar for either ten or thirty minutes daily had a shorter interval to puberty than gilts exposed for two minutes a day. Paterson et al. (1989b) reported that when boar exposure was limited to 1 day, or 10 consecutive days, a degree of stimulation took place, but daily exposure continuing until puberty was necessary for the maximum stimulation of puberty.

2.3.1.6 Housing

Dyck (1989) reported that the interval from initial boar exposure to puberty for gilts housed in stalls was shorter than for gilts housed in group pens of eight (12.3 vs 18.7 d). However, these results may have been confounded by experimental design. All gilts were not treated equally, as the degree and length of pubertal stimulation, as well as boar age was different between treatments. Stall-housed gilts had a longer duration of estrus than pen grouped gilts, which may be partially attributed to the use of the mature boar at stimulation. As reviewed by Hughes (1982), crowding of gilts has been shown to be effective and ineffective in stimulating early puberty in gilts.

2.3.1.7 Transportation and mixing

Several researchers have demonstrated that crowding, mixing, and transportation of gilts will stimulate puberty. Pearce and Hughes (1987b) suggested that an important component in the stimulation of early puberty in gilts is an increase in cortisol, mediated through the stress of remixing and relocation. Hughes et al. (1997) demonstrated that there was no effect of transporting gilts 20 minutes daily for 10 days on puberty attainment in gilts. However, when transport and frequent boar contact (three times per day) were combined, the result was a shorter interval from initiation of boar contact until puberty compared with gilts receiving only frequent boar contact.

2.3.2 Nutrition and growth

Manipulation of nutritional regimes to achieve differences in growth, age, weight, and body composition have been used for many years to elucidate relationships with reproductive capability in gilts (Zimmerman et al., 1960; O'Bannon et al., 1966). Today, researchers still use nutritional manipulation to examine the relationships between reproduction, growth and body composition (Cia et al., 1998; Klindt et al., 1999; Le Cozler et al., 1999). Le Cozler et al. (1999) fed gilts at two different levels, one group *ad libitum* and the second group 80% of the *ad libitum* intake. This treatment led to the *ad libitum* gilts being younger, heavier and fatter at puberty than the gilts that were restricted. Similarly, Cia et al. (1998), through restricting the lysine:energy ratio, produced gilts of different body compositions at puberty. Gilts with the highest restriction of protein:energy (0.3) were lightest, had the greatest backfat, and the smallest muscle depth compared with gilts with a higher lysine:energy ratio (0.6 or 0.9).

Because the nutritional treatments imposed create differences in several body components, it is difficult to determine what, if any, of these components were specifically involved in altering pubertal onset. The next section will examine growth and its individual components and the effects they exert on the attainment of puberty in gilts.

It has been shown that the onset of puberty is genetically determined and that the course of events culminating in the attainment of puberty may be altered by environmental factors (Hughes and Varley, 1980). The onset of puberty has been associated with a critical age (Hughes and Varley, 1980), a minimum fat to lean ratio (Kirkwood and Aherne, 1985), growth rate (Rydhmer et al., 1994; Rozeboom et al., 1995) and minimum fatness (Gaughan et al., 1997). It seems from the literature available that these factors may be permissive, in that a minimum threshold of these factors must be reached before puberty can occur. Beltranena et al. (1993) concluded that genetic determinants of sexual maturity, and not growth performance, limit the attainment of puberty in modern gilts achieving commercially acceptable growth rates.

2.3.2.1 Age and weight

Kirkwood and Aherne (1985) predicted that neither age nor weights are reliable indices of reproductive development. This is supported by the fact there is a large range for both age and weight over which gilts reach puberty. Rozeboom et al. (1995) reported that little if any synchronization in puberty is due to age. However, there are minimum threshold values for these characteristics that must be achieved before puberty can occur, because puberty will not occur at earlier and earlier ages and weights. This suggests that while reaching the threshold levels for age and weight are necessary, their achievement is not in itself sufficient to trigger the onset of reproductive activity and does not preclude other factors, for example, nutrition, management, environment, and body composition, from having stimulatory or inhibitory effects (Kirkwood and Aherne, 1985). The age at which puberty stimulation is first introduced will have profound effects on the age at which gilts achieve puberty. Age at stimulation varies immensely over experiments, for example, 120 days (Rozeboom et al., 1995), 125 days (Holder et al., 1995), 175 days (Klindt et al., 1999), and 180 days (Le Cozler et al., 1999). As Holder et al. (1995) stress, it is important to recognize the difference between sexual age and chronological age when evaluating the effect of age at puberty on reproductive performance.

Growth rate has been negatively (Cunningham et al., 1974; Cameron et al., 1999) and positively (Rydhmer et al., 1992) correlated to age at puberty for gilts. It is well established that a severe restriction in nutrition that results in a slow growth rate, adversely affects age at puberty in gilts. Zimmerman et al. (1960) reported that nutritional deficiency during prepubertal development would be expected to influence subsequent reproductive performance by delaying puberty. Beltranena (1992) suggested that at growth rates above 600 g/d, the attainment of sexual maturity would not be affected.

Le Cozler et al. (1999) reported that large significant differences in live weight and backfat were established between gilts fed *ad libitum* and gilts restricted to 80 % of *ad libitum* from 25 kg until 180 days of age, whereas small significant differences were found in age at puberty; therefore, these authors speculate that age is the principle factor determining puberty. As discussed by Beltranena (1992), for gilts growing at less than .55 kg/d, somatic development likely limits the attainment of puberty. Although lifetime growth rates were not reported in Le Cozler's paper, at puberty, *ad libitum* and restricted gilts grew on average, .64 and .58 kg/d, respectively. Therefore, due to variation above and below the mean, it is likely that some of the restricted gilts were below the threshold of .55 kg/d that Beltranena (1992) described. It is not surprising then, that restrict fed gilts were slightly older at the onset of puberty, and it may not be valid to conclude that age was the factor regulating pubertal onset. Similarly, Cameron et al. (1999) found a significant negative correlation between growth rate from birth to puberty and age at puberty in gilts selected for various components of growth. However, at the initiation of estrus detection (150 days) lifetime growth rates were low between selection lines, .46, .53, and .57 kg/d.

Beltranena (1992) describes a second phase of development where lifetime growth rate does not limit the attainment of puberty. He predicted that when a growth rate of .6 kg/d was achieved, physiological age or maturity likely limits the onset of puberty, rather than some attainment of a minimum threshold of growth. There is little evidence regarding the effects of high growth rates on age at puberty in gilts.

2.3.2.2 Fatness

Frisch (1980) suggested that a female must store at least a threshold, or minimum, amount of body fat in order to begin and maintain menstrual cycles and hence the ability to reproduce. She also suggested (1977) that body weight (fatness) also directly influences estrogen metabolism and therefore may influence reproductive ability directly. Using dietary manipulation, Magowan (unpublished data), created a group of pubertal gilts whose average backfat was 5.3 mm, suggesting that the threshold for fat may be lower than suggested previously. King (1989) reported that reduced live weight and increased backfat depths in prepubertal gilts delayed the onset of pubertal estrus. In contrast to these findings, Gaughan et al. (1997) reported that backfat depth at selection had a major influence on puberty attainment. However, the delay in the onset of puberty may not be related to the rate of fat deposition or weight gain *per se*, but may be indicative of differences in physiological development. This is supported by the fact that although there was a significant difference in age at puberty for the first 67% of gilts to reach puberty, no difference was detected when all gilts on trial were included. This suggests that the distribution of gilts reaching puberty was different, and that later physiological development and not backfat depth at selection, was the primary factor limiting puberty onset. Further evidence supporting the lack of an effect of fat on puberty is provided by Beltranena (1992). In the presence of similar levels of lean deposition, no difference in basal and episodic LH secretion, follicular development, reproductive tract weight, and plasma or follicular fluid estradiol-17β concentrations were observed between gilts with reduced, compared with increased carcass fat. Rozeboom (1999) provided further evidence that a precocious puberty is not controlled by a specific amount of fat. However, even though fat may not be a major factor in the onset of puberty, as reviewed by Rozeboom (1999), there is conflicting evidence as to the effect of backfat depth at breeding and the ability of gilts to farrow four litters.

2.3.2.3 Lean growth rate

The data available concerning the effect of lean growth on reproductive parameters, in particular the attainment of puberty, is limited, and clear definitive

relationships are not yet available. Rydhmer et al. (1992) suggested that breeding for leanness might delay the onset of puberty. In contrast, Cunningham et al. (1974) reported that gilts with the highest lean growth rates were youngest at puberty, however, caution should be used in interpreting these data because growth rates in this trial were low compared with today's standards. More recent work by Cameron et al. (1999) suggests that selection for lean growth rate had no adverse effects on reproductive development. Having achieved differences in body composition in the gilts, it was determined that protein restriction in the rearing phase negatively impacted latency to estrus and ovulation rate (Cia et al., 1998), irrespective of increased body fatness. In another study, differences in the level or rate of fat deposition, in the presence of maximal protein accretion, did not influence reproductive development in the gilt (Beltranena et al., 1993). King (1989) suggests that lower or diminished body protein reserves have an adverse effect on the fertility of gilts. Cia et al. (1998) suggest that if body composition is affecting reproductive performance, the limiting factor may be protein mass, and if protein mass is the regulator, it may be related to the metabolic status of the animal.

2.3.2.4 Metabolic state

Kennedy and Mitra (1963) first proposed the idea that the transition between the fertile and infertile state (puberty) is related to a change in somatic metabolism. They concluded that information about the state of somatic development would be relayed to the hypothalamus to regulate puberty indirectly, through alterations in energy balance, and due in part to the changing relation between the growing animal and its environment. Thus, a decrease in the rate of metabolism occurs during growth to maintain a stable core temperature, and occurs when the rate of increase of the body mass (increase in heat production) exceeds the rate of the increase of surface area (heat dissipation) (Foster and Nagatani, 1999).

Secretion of GnRH is dependent on nutrient availability (Wade and Schneider, 1992; Miller et al., 1995). This suggests that the GnRH pulse generator must have some

way of sensing the metabolic status of the body (Miller et al., 1998). Circulating metabolic fuels and hormones identified as possible links between growth and reproduction include insulin/glucose availability, growth hormone/insulin-like growth factor, thyroid hormones, endogenous opioids, excitatory amino acids, neuropeptide Y, and leptin.

2.3.2.4.1 Insulin / glucose availability

Insulin is a hormone that stimulates tissue uptake of glucose and fatty acids, promotes lipogenesis and glycogen synthesis, and inhibits lipolysis (Wade and Schneider, 1992). Thus, insulin plays an important role in nutrient partitioning and the availability of metabolic fuels. There are numerous studies implicating insulin as an important factor in reproduction (Monget and Martin, 1997).

Until recently, insulin secreted from the endocrine pancreas (Schwartz et al., 1992) has been assumed to be the major link between nutrition and reproduction (as reviewed by Conway and Jacobs, 1997), acting as a peripheral adiposity signal to the central nervous system. This is supported by the fact that 1) plasma insulin levels vary directly with changes in energy balance and adiposity; 2) dose-dependent entry of circulating insulin into the CNS; 3) neurons express high concentrations of insulin receptors in brain regions known to participate in the regulation of feeding; and 4) insulin produces a marked reduction of food intake following either central or peripheral administration (as reviewed by Schwartz et al., 1992).

Many of the structures in the brain that control GnRH secretion have insulin receptors (Monget and Martin, 1997). Insulin in the brain is totally derived from the blood and insulin must cross the blood brain barrier by a saturable system (Banks et al., 1997). There is also evidence that insulin is a metabolic modulator of GnRH secretion and mediates the effects of nutrition on gonadotrophin secretion (Miller et al., 1995). These authors reported that rams given insulin and insulin+glucose showed increased LH pulses throughout the day (Miller et al., 1995).

Insulin has other important effects on the reproductive system. Cox et al., (1987) showed that gilts given an insulin injection and high energy diet had a larger number of corpora lutea compared with control gilts not given insulin with a lower energy diet. This increase in ovulation rate may have occurred because of recruitment of more follicles. Matamoros et al. (1990) confirmed these results, suggesting that a decrease in follicular atresia in response to exogenous insulin treatment in diabetic gilts contributed to an increase in ovulation rate in the absence of any detectable changes in LH. Insulin may act in concert with IGF-1 in producing these effects.

Foster and Nagatani (1999) discussed information suggesting that glucose availability itself is thought to be a metabolic signal: 1) glucose serves as a primary metabolic fuel for both the soma and brain; 2) glucose is a factor regulating appetite control; 3) glucose availability may be a regulator of LH secretion; 4) dietary restriction slows growth, reduces peripheral glucose and insulin concentrations; and 5) insulin modulates LH secretion.

Food deprivation and thus glucose availability control reproductive activity through modulation of LH secretion in a number of species (Nagatani et al., 1996). In male and female rats given 2DG (a glucose inhibitor) there was a significant decrease in the frequency of LH pulses (Nagatani et al., 1996). Murashasi et al. (1996) suggested that glucose availability could influence LH and feeding, through a sensor in the lower brain stem that may act as a glucosensor involved in the modulation of LH secretion. Similarly, in male lambs, glucose administration increased LH secretion by acting through the central nervous system (Bucholtz et al., 1996). In feed restricted prepubertal gilts, glucose infusion increased insulin secretion and induced a rapid increase in episodic LH secretion (Booth, 1990).

2.3.2.4.2 Growth hormone --Insulin-like growth factor

The growth hormone (GH) neuroendocrine axis is exquisitely sensitive to changes in nutritional status (Shaffer Tannenbaum et al., 1998). GH plays a role in the onset of puberty. GH appears to augment the rate of sexual maturation once a pubertal pattern of gonadotrophin secretion is established. (Sharara and Giudice, 1997). Under

normal physiologic conditions, GH seems to be a major regulating factor influencing the synthesis and secretion of insulin-like growth factor (IGF) in swine or by acting directly on GH receptors localized recently in the granulosa cells and corpus luteum in the human ovary (Sharara and Giudice, 1997). IGF-1 is a peptide hormone involved in the metabolic regulation of growth and as an anabolic hormone it mediates many of the metabolic actions of GH (Mauras et al., 1996).

Historical data has shown that GH administration resulted in an increase in ovarian size and initiation of follicular development in dwarf mice (Bartke, 1964). More recently, GH has been shown to stimulate follicular growth, oocyte maturation and estradiol production in perfused rabbit ovaries (Yoshimura et al., 1993) and in human granulosa cells in the absence of LH and FSH (Mason et al., 1990; Tapanainen et al., 1992). Kirkwood et al. (1992) suggested that high levels of porcine growth hormone enhanced the in vitro steroidogenic capabilities of porcine granulosa cells and ovarian activity. The recent discovery that the human ovary has receptors for GH, and the fact that IGF-1 gene expression is only present in dominant and developing follicles, suggest that GH may act on the ovary to augment folliculogenesis and steroidogenesis (Sharara and Giudice, 1997). However, studies have shown that GH has adverse effects on reproductive activity, GH treatment has been shown to increase the occurrence of anestrus in gilts (Gilbertson et al., 1991) and a high incidence of cystic-like ovarian condition was observed following a gonadotrophin-stimulated ovulation in gilts pretreated with GH (Kirkwood et al., 1988).

It is important to point out a species difference in GH regulation. Unlike other species, GH secretion in rats is abolished during feed restriction. However, as reviewed by I'Anson et al. (1991), GH rises with low planes of nutrition or fasting in humans, pigs, sheep and cattle. These differences may be described by the catabolic and anabolic roles of GH.

Following acute feed restriction (48 hours) in barrows, plasma growth hormone increased while circulating levels of IGF-1, insulin, and thyroid hormones declined

(Buonomo and Baile, 1991). The reduction in plasma IGF-1 levels, despite the increase in GH, is controlled by the responsiveness of hepatic tissue to GH stimulation, where insulin acting as a key regulator of hepatic sensitivity (Foxcroft, 1990). Interestingly, after the feed restriction was removed in gilts, hepatic IGF-1 gene expression and circulating IGF-1 levels increased, however, there were no changes in follicular fluid IGF-1 concentrations or in ovarian IGF-1 gene expression (Charlton et al., 1993). This suggests that there is a different mechanism regulating IGF-1 expression in hepatic and ovarian tissues.

IGF-1 has been implicated in the onset of puberty. Hiney et al. (1991) suggest that IGF-1 may represent one of the "metabolic signals" thought to be involved in the initiation of puberty. As reviewed by Hiney et al. (1996), IGF-1 circulating levels increase strikingly during puberty across a wide range of species, and decreased levels of IGF-1 may contribute to a delayed onset of puberty. In addition, Wilson (1995) has demonstrated in the rhesus monkey, that injections of IGF-1 accelerate the process of puberty by decreasing the sensitivity of the hypothalamo-pituitary axis to estradiol negative feedback.

Hiney et al. (1996) provide evidence that IGF-1 may be one of the signals linking the development of the somatrophic axis to the activation of the LHRH/LH releasing system during female puberty. Intraventricular administration of small amounts of IGF-1 was capable of stimulating LH release. More recently, Adam et al. (1998) demonstrated that low doses of peripherally-administered IGF-1 stimulated LH output in sheep.

2.3.2.4.3 Thyroid

Thyroid hormones are of life sustaining importance in mammals, and are present in two forms, thyroxine (T4) and triiodothryonine (T3). Thyroid hormone release is stimulated by increasing thyroid stimulating hormone (TSH) levels as a result of activation by thyroid releasing hormone (TRH) in the paraventricular nucleus of the hypothalamus (Orban et al., 1998). Thyroid hormone is a major regulator of energy

homeostasis and thermeogeneis. Prolonged fasting decreases plasma T3 and T4, free T3 and T4, and produces low or normal levels of TSH (Orban et al., 1998).

TSH is a secondary hormone of reproduction, in that its actions are permissive and regulate physiological systems that indirectly, yet profoundly, influence reproduction (Estienne et al., 1997). Buonomo and Baile (1991) reported that thyroid hormones stimulate IGF-1 synthesis by the liver and potentiate the effects of GH on IGF-1 synthesis.

2.3.2.4.4 Hypothalamic-pituitary-adrenal axis

The hypothalamic-pituitary-adrenal (HPA) axis and the female reproductive system are intertwined and quite complex (Magiakou et al., 1997). Starvation, a type of stress, stimulates the HPA axis (Heiman et al., 1997) and is associated with suppression of the reproductive axis (Rabin et al., 1990). Most effects of HPA are inhibitory: CRH inhibits GnRH secretion (Rivier and Rivest, 1991), and glucocorticoids inhibit LH and ovarian estrogen and progesterone secretion and render estrogen-target tissues resistant to the gonadal steroid (Magiakou et al., 1997)

Activation of the HPA axis also has detrimental effects on the growth axis, as CRH inhibits SRIH and glucocorticoids reduce levels of IGF-1 and the sensitivity of tissues to IGF-1 (Magiakou et al., 1997). Likewise, CRH suppresses the thyroid axis (decreasing levels of TSH) (Magiakou et al., 1997).

2.3.2.4.5 Endogenous opioids

Endogeneous opioid peptides (EOP) are a group of neurotransmitters that control the secretion of hypothalamic hormones. In general, EOP (endorphins, enkephalins and dynorphins) inhibit GnRH, and hence LH, secretion through involvement with the catecholaminergic system (Estienne et al., 1997).

The precursor for β-endorphin is proopiomelanocortin (POMC) which has cell body fibres projecting to the medial basal hypothalamus, periventricular zone, preoptic area and median eminence (Estienne et al., 1997). POMC is implicated in the regulation of appetite and food intake, as well as influencing GnRH secretion. Neurons containing POMC make direct synaptic contact with GnRH-containing neurons (Cunningham et al., 1999) and POMC produces β-endorphin, which is a negative regulator of GnRH synthesis (Clarke and Henry, 1999).

2.3.2.4.6 Excitatory amino acids

Excitatory amino acid (EAA's) play an important role in regulation of pulsatile gonadotrophin release, induction of puberty and pre-ovulatory and steroid induced gonadotrophin surges (Brann and Mahesh, 1995). EAA neurotransmission is an essential component of the neuroendocrine transmission line that regulates anterior pituitary lutenizing hormone (LH) and follicle-stimulating hormone (FSH) (Brann and Mahesh, 1994). The effects of EAA's on stimulatory GnRH release requires an estrogen background, probably due to the increased sensitivity of GnRH neurons. Two EAA's of importance are glutamate and aspartate that are found in several regions of the hypothalamic nuclei, the arcuate nucleus, the suprachiasmatic nucleus, the supraoptic nucleus, the paraventricular nucleus, and the preoptic area (Brann and Mahesh, 1994). In contrast, Popwell et al. (1996) found that EAA's may have both an inhibitory and stimulatory effect of LH secretion, with the inhibitory effect occurring within the basal hypothalamus and the stimulatory effects lying within higher brain centers. Regulation of other releasing factors (TRH, CRF, GHRH) by EAA's is important and the stimulation of these factors and subsequent outcomes should be apparent by the preceding discussions.

2.3.2.4.7 Neuropeptide Y

Neuropeptide Y (NPY) is one of many peptides and neurotransmitters implicated in the regulation of appetite and food intake that are also known to influence the secretion of GnRH (Clarke and Henry, 1999). NPY is an important signal in the

hypothalamic circuitry engaged in the control of reproduction and can stimulate and inhibit release of luteinizing hormone (LH), one of the primary hormones regulating the pituitary-gonadal axis (Kalra and Kalra, 1996). NPY stimulates the release of GnRH (Kalra and Kalra, 1996). It is thought that the preovulatory gonadotrophin surge is dependent upon enhanced NPY output and that NPY may maintain LH secretion in circumstances such as nutritional challenges (Clarke and Henry, 1999; Kalra and Kalra, 1996). However, there seems to be a time period when NPY effects are inhibitory, because it has been shown that continuous infusion of NPY inhibits LH secretion when given to gonadectomized rats and sheep (as reviewed by Clarke and Henry, 1999). Malven et al. (1995) have shown that endogenous NPY in the brain of ovariectomized ewes seems to restrain or delay the onset of the surge-like secretion of LH and probably GnRH. The role NPY plays also seems to be dependent on levels of circulating steroids; NPY inhibits LH at low levels of steroids (Kalra and Kalra, 1996). The same authors state that NPY also stimulates the release of β-endorphins in the basal hypothalamus, which most likely suppresses the release of LH in these conditions. Central insulin (Schwartz et al., 1992) and leptin (Schwartz et al., 1996) administration inhibits the release of NPY.

2.3.2.4.8 Leptin

Leptin (from the Greek *leptos*, meaning thin) is a protein hormone with significant effects in regulating body weight, metabolism and reproductive function (Friedman and Halaas, 1998; Cunningham et al., 1999). Although leptin was not discovered until 1994 by Zhang and collegues, G.R. Hervey first hypothesized that the presence of a hormone in body fat regulated body weight through an interaction with the hypothalamus (see review of Considine and Caro, 1997). A review of the literature shows that the leptin receptor is not only expressed in the brain but in most other tissues in humans and rodents: heart, placenta, lung, liver, muscle, kidney, pancreas, spleen, thymus, prostate, testes, ovary, small intestine and colon (Keilar et al., 1998). This indicates that leptin may have a wide range of functions in the body. Evidence for a possible link between leptin and reproduction resulted from the determination that *ob/ob* mice were infertile due to reduced circulating gonadal steroids and insufficient

hypothalamic-pituitary drive (Cunningham et al., 1999). An injection of leptin into *ob/ob* mice restored reproduction function (Barash et al., 1996; Ahima et al., 1997).

It has been shown that leptin interfered with insulin secretion from pancreatic islets, reduced insulin-stimulated glucose uptake in adipocytes, and increased glucose transport, glycogen synthesis and fatty acid oxidation in skeletal muscle (Ceddia et al., 1998). Likewise, Fehmann et al. (1997) suggest that leptin is a mediator of insulin secretion.

Barash (1996) reported that ob/ob female mice (lack functional ob gene) given a single leptin treatment had greater ovarian and uterine weights than their untreated counterparts. Leptin treated ob/ob mice also showed a significantly larger total number of follicles consisting primarily of primary and graafian follicles. However, current literature suggests that the direct effects of leptin on the gonads are inhibitory rather than stimulatory. Zachow and Magoffin (1997) showed that leptin at high levels inhibits FSH- and IGF-1- simulated estrogen production. These authors suggest that, at high levels of leptin, a defect or block in the sensitizing action of IGF-1 on granulosa cell stimulation could contribute to the arrest of both granulosa cell differentiation and follicular growth. Spicer and Francisco (1997) have shown similar inhibitory effects of increasing leptin concentrations. Granulosa cells from small and large bovine follicles showed reduced insulin-stimulated progesterone and estradiol production in the presence of increasing leptin concentrations.

Leptin has been implicated in triggering the onset of puberty (Apter, 1997; Cunningham et al., 1999). This is supported by the discovery that all *ob/ob* female mice are infertile, probably because of reduced hypothalamic GnRH content and low plasma gonadotrophin concentrations (Clarke and Henry, 1999). Upon leptin treatment, infertility can be reversed and puberty can be advanced (Cheung et al., 1997). Cunningham et al. (1999) have suggested that leptin is more likely to be a permissive factor to the onset of puberty, and that, although some minimal threshold level of leptin is necessary for pubertal development, leptin by itself is not sufficient to initiate puberty.

The role of leptin in the onset of puberty remains a controversial topic. Cheung et al. (1997) have shown that during periods of feed and growth restriction, the onset of puberty is delayed and that leptin treatment with recombinant human leptin reverses this effect. The same authors have also shown that leptin does not advance puberty any earlier than would normally occur in normal rats with adequate access to feed.

In contrast, leptin has been shown to advance puberty in normal mice with no significant effects on body weight (Ahima et al., 1997). In this study, recombinant mouse leptin or saline treatment was given daily starting from 21 days of age until the mice reached puberty. In all factors that characterize pubertal onset in mice (vaginal opening, vaginal estrus, vaginal cycling), the leptin treated animals reached these markers significantly sooner than saline treated animal. One other study has confirmed these results. In an experiment much the same as Cheung et al. (1997), leptin treated mice showed advancement of vaginal opening and increased reproductive organ (uteri, ovaries, oviducts) weights (Chehab et al., 1997). These authors also determined that LH and 17-β estradiol concentrations were lower in leptin treated mice. Since all mice were killed on day 29, lower LH and 17-β estradiol levels would be expected in leptin treated animals since they progressed through puberty earlier than the other mice (Chehab et al., 1997). However, in the last study, the authors were unable to repeat these results. Likewise, Cunningham et al. (1999) were unable to replicate either result using rats.

2.3.3 Litter origin

The litter from which gilts originate has been shown to influence performance of gilts. This effect can be attributed to environmental (relating to maternal nutrition, litter size, birthweight) and non-environmental (differences in inherent physiology) factors. Deligeorgis et al. (1985) suggest that gilts originating from larger litters, litters with higher male to female ratios and litters with smaller variation in birth weight were more sexually mature at 160 days of age. Undernutrition *in utero* causes low birth weight and a reduction in postnatal growth rate (Dwyer et al., 1994). Mukasa-Mugerwa et al. (1991) reported that the onset of puberty was advanced by weaning weight, which was

correlated to birth weight and level of nutrition in ewe lambs. Deligeorgis et al. (1985) noted that the number of corpora lutea, number of follicles greater than 5 mm, ovarian and uterine weight, and LH and FSH responses to a GnRH challenge at 55 days of age were significantly affected by litter origin of gilts. More recently, Le Cozler et al. (1999) found that birth litter had a significant effect on several factors including age, weight and backfat depth at first physiological estrus and at first breeding. Perhaps age at pubertal onset is regulated by a genetic clock and is invariant in its time course of development (Foster et al. 1994). Almeida et al. (2001) provided evidence that litter had significant effects on growth characteristics such as body weight, growth rate, backfat depth and reproductive characteristics such as estrous cycle length, estrus duration, and various endocrine parameters.

2.4 Conclusions

The goal of this review of the literature was to explore and to understand a large area of study relating to the onset of puberty in gilts. An understanding of the fundamental components and processes of growth and puberty is first necessary to be able to determine the relationships that exist between the two. It was then essential to examine the factors that regulate and influence the onset of sexual maturity in gilts. The onset of puberty in largely controlled by internal and external cues, regarding the state of physiological development and the environmental conditions a gilt is exposed to (Figure 2.4). The work completed in this thesis examines both aspects of pubertal development.

The first experiment was designed to determine the effect of type of boar exposure on puberty attainment in gilts. Although previous studies undertaken in the last two decades examined this question, consensus in the literature as to which is the most efficient method is lacking. It was also important to revisit this question in terms of modern, contemporary genotypes and housing facilities.

The current trend in the production of market pigs places emphasis on lean tissue growth rate. The data available concerning the impact of lean growth rate on

sexual development of gilts is limited and somewhat contradictory. The second experiment addressed the question of the effect of lean growth rates on puberty attainment in gilts.

A third experiment was undertaken in the course of the thesis work, however, the experiment did not relate to puberty attainment in gilts. The study examined the effect of prostaglandin and weaning to estrus interval on subsequent reproductive performance in swine; the results are included in the appendix to this thesis.

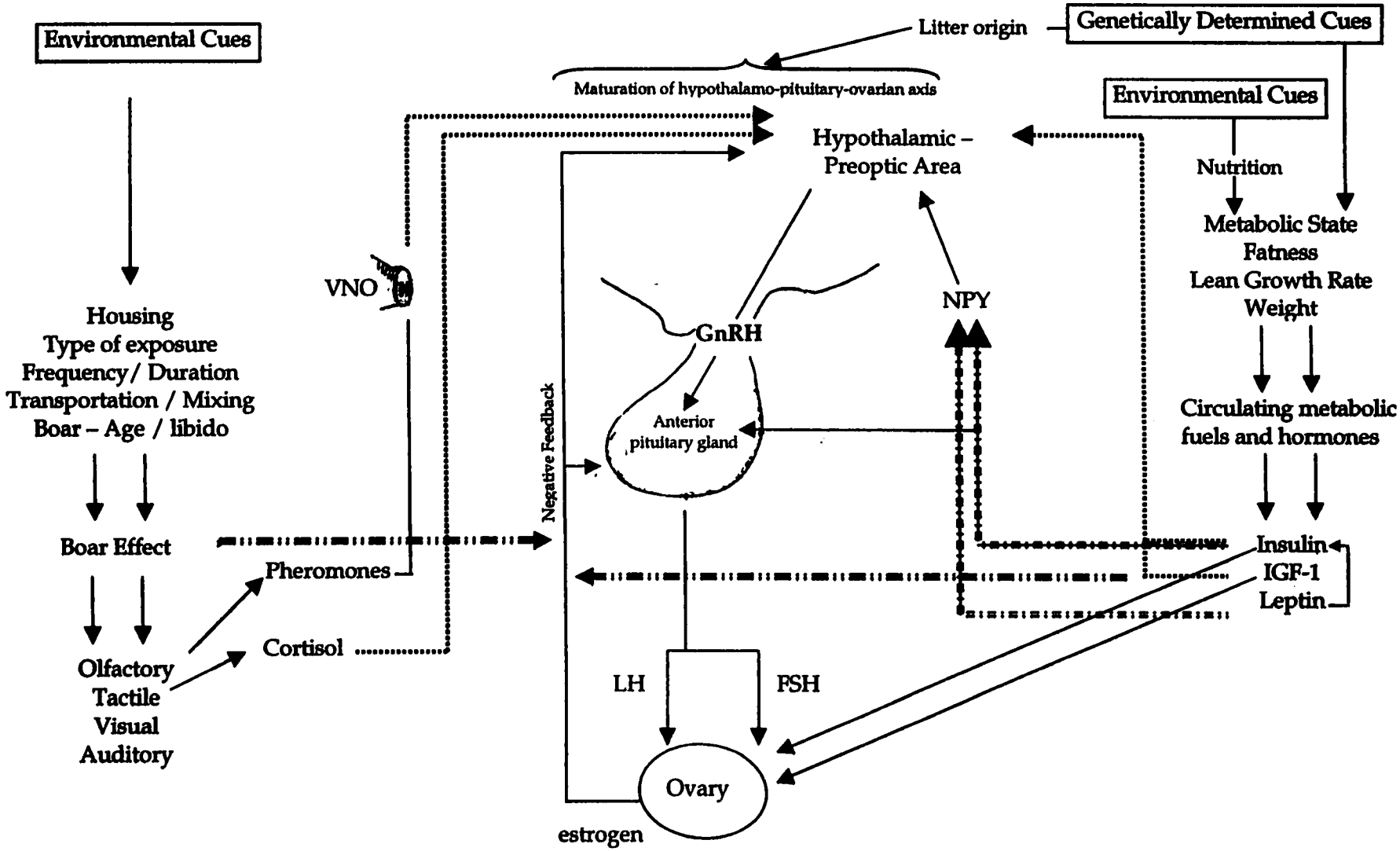

Figure 2.4 Summary of the factors affecting the onset of puberty. ⟶ stimulatory effects ▪▪▪▶ inhibitory effects.

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CHAPTER 3

IMPACT OF BOAR EXPOSURE ON PUBERTY ATTAINMENT AND BREEDING OUTCOMES IN GILTS

3.1 Introduction

It is well established that exposure of prepubertal gilts to a mature boar is an effective means of inducing puberty (Pearce and Hughes, 1985; van Lunen and Aherne, 1987). This effect is largely due to the synergistic action of the boar component stimuli: visual, tactile, olfactory and auditory cues (Hughes et al., 1990). There are several methods by which a mature boar can be used to stimulate puberty: Fenceline stimulation, gilts moved to the boar pen, gilts and boar moved to a common pen, boars moved to the gilt pen, and continuous housing of gilts and boars together (Pearce and Hughes, 1985; van Lunen and Aherne, 1987).

In large modern production units, there is pressure to maintain a continuous flow of cyclic gilts to meet breeding targets. Housing, time, and labor constraints often determine which method of boar exposure is utilized. However, it is important that a producer adopt a method of puberty stimulation that most effectively induces early puberty in gilts. It has been shown that physical contact between a boar and a group of gilts is a more effective stimulus than fenceline contact for triggering an earlier first estrus in gilts (Pearce and Hughes, 1985; Zimmerman et al., 1996). Moreover, van Lunen and Aherne (1987) reported that a larger proportion of gilts reach puberty by 270 d when gilts were taken to the boar, compared with when a boar was taken to the gilt pen for puberty stimulation.

Researchers have long sought a key metabolic signal for puberty onset in mammals (Kennedy and Mitra, 1963). The newly discovered ob gene, that codes for

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leptin, has been reported to be involved in the onset of puberty in mice (Cheung et al. 1997). Although leptin may be an important link signaling metabolic status to the reproductive system, it has already been determined that other metabolic cues, like insulin-like growth factor (IGF-1), may be involved in the timing of puberty (Hiney et al., 1996). However, the literature regarding leptin, IGF-1 and puberty in farm animals is limited.

The presence of a boar has been shown to exert physiologic effects on the female at breeding. Circulating oxytocin levels in the sow increase when sows are subjected to olfactory (Mattioli et al., 1986) and tactile stimulation (Claus, 1990), which stimulate uterine and oviductal contractions (Claus 1990). Sperm transport in the female tract is largely facilitated by smooth muscle contractions of the uterine myometrium (Hughes and Varley, 1980). Oxytocin also acts as a stimulus for prostaglandin secretion (Edgerton et al., 2000). Furthermore, both prostaglandin and oxytocin have been shown to be involved in fimbrial capture of the ovum and tubal transport of the ovum in the rabbit by stimulating contractility in the ampullary region of the oviduct (Osada et al., 1999).

The first goal of this study was to examine three different methods of boar exposure to determine the most effective method for stimulating gilts to reach puberty in a contemporary commercial genotype. A second goal of this study was to determine relationships between plasma IGF-1 and leptin concentrations and reproductive parameters. The third objective for this study was to determine if the lack of a boar at breeding has any negative consequences for the quality of the insemination and subsequent litter size.

3.2 Material and methods

3.2.1 Part 1

This study was completed at the University of Alberta Swine Research Unit with approval from the Faculty Animal Policy and Welfare Committee. Eighty-nine

prepubertal Large White grandparental gilts (Genex Swine Group) were used in this experiment. At approximately 160 days of age, groups of gilts were stratified by age and weight, and randomly allocated to one of three puberty stimulation treatments; 1) direct contact of group pens of gilts with a vasectomized boar in a purpose built boar stimulation area (GB: n=30); 2) direct contact with a vasectomized boar in gilt home pens (BG: n=31); 3) fenceline contact between a vasectomized boar and gilts housed in individual gilt stalls (BS: n=28). Gilts in pens were housed in groups of six. All gilts were housed on fully slatted floors, with natural daylight from overhead skylights. Pen floor space allowance was calculated as recommended for growing pigs on fully slatted floors (Agriculture and Agri-Food Canada, 1993); each gilt was permitted at least 1.2 m^2 space allowance throughout the entire trial.

All gilts were fed a grower diet, formulated to provide 16 % crude protein, 3.2 Mcal/kg of digestible energy and .75 % lysine. Pen-housed gilts were allowed *ad libitum* access to water and to feed from two-holed feeders throughout the trial. Stall-housed gilts were allowed *ad libitum* access to water and to feed from individual feeders in each gilt stall. In pens, feed trays were constructed to collect spilled feed, and thus to minimize feed wastage. Feed disappearance was measured weekly for group-housed gilts and daily for stall-housed gilts. In the BG treatment, feeders were removed from the pen during stimulation to prevent the vasectomized boar from eating.

Gilts were exposed to boars twice daily for the detection of the onset and stimulation of their pubertal estrus. Because not all treatments involved direct contact with a vasectomized boar, puberty attainment was defined as the first time a gilt exhibited a standing reflex in response to the back pressure test (BPT) by a technician, during twice daily (07:00 and 19:00 h) fenceline contact with a vasectomized boar (puberty detection). Any gilt exhibiting the standing response was removed from the pen. Gilts were then exposed to a vasectomized boar *as per* the method of boar stimulation they were assigned to, for a minimum of ten – fifteen minutes (puberty stimulation) (Figure 3.1). One of four mature vasectomized boars was used both for detection and stimulation and gilts were not exposed to the same boar twice in one day.

Age, weight and backfat depth at puberty and duration of pubertal estrus were recorded. At the onset of their pubertal estrus gilts were removed from the trial and group-housed gilts were relocated to individual stalls.

For statistical analysis, in part 1, all gilts with a progesterone concentration above 1 ng/ml before boar contact at 160 d of age were considered pubertal and removed from the analysis. Gilts were also removed from the analysis if they reached puberty within three days of the commencement of boar stimulation. Any lame gilt was removed from trial. If a gilt was not pubertal by d 215, her age on that day was used in the analysis. This age was chosen because it is likely a producer would not keep an anestrus gilt beyond this age.

On entry into the gilt facility, a 5 ml jugular blood sample was taken from all gilts. Every 10 days thereafter until d160 (commencement of boar stimulation) and every 10 days from d190 until d210, a 5 ml jugular blood sample was taken to ensure that gilts had not ovulated. All blood samples were taken during a brief period of nose-snare restraint. All samples were centrifuged at 1,500 x g for 15 minutes; plasma was decanted and stored at –20 ° C until analysis for plasma progesterone concentration. The d160 sample for all gilts, measured prior to any exposure to a vasectomized boar, was also analyzed for leptin and IGF-1.

For all radioimmunoassays (RIA), all samples were analyzed in duplicate. Assay sensitivity was calculated using the following equation: average of the zero binding tube [(Bmax) – 2SD(Bmax) / average (Bmax)] X 100. Plasma progesterone concentrations were determined in two assays using a kit RIA (Coat-a-Count Progesterone, Diagnostic Products Corporation, Los Angeles, CA, USA). The intra-assay coefficients of variance (CV) were 5.1 and 5.7% and the assay sensitivity was .01 and .01 ng/ml. The inter-assay CV was 7.7%. Plasma IGF-1 concentrations were determined using the homologous double antibody RIA as described by Cosgrove et al. (1992). 100 µl of plasma was initially extracted with 3 ml of acid ethanol. Radio inert recovery efficiency for the single assay run was 109%, the intra-assay CV was 8.9 %, and

sensitivity of the assay, estimated as 87% bound, was .012 ng/tube. Plasma leptin concentrations were determined using the multi-species double antibody kit assay previously validated by Mao et al. (1999) for use in our laboratory. The intra-assay CV was 5.1% and the sensitivity of the assay measured at 94.1% bound, was 0.2 ng/ml.

3.2.2 Part 2

A proportion of gilts from that completed experiment one were allocated to a breeding group and given 15.4 mg of Regumate daily as an oil-based top dressing from d15 of the first or second cycle to synchronize estrus. Regumate was removed 5 days prior to the desired breeding day. Gilts were assigned to one of two treatments based on weight at pubertal estrus; 1) fenceline contact with a boar during artificial insemination (BC, n=21), or 2) no contact with a boar for a minimum of 60 minutes prior to, or during, artificial insemination (NC, n=14). Gilts were checked twice daily following the removal of Regumate for onset of standing heat, at which time gilt weight, backfat depth, and heat number were recorded. Gilts were bred 12 and 24 hours after the onset of standing heat by artificial insemination using 3 X 10^9 sperm in 70 ml extended and pooled Genex semen. All NC gilts were checked for standing heat in the presence of fenceline contact with a vasectomized boar. Once all NC were heat checked, all BC gilts were heat checked and bred in the presence of a vasectomized boar. One hour after any contact with boars, NC gilts were bred without any contact with a vasectomized boar. All gilts were treated equally during insemination by the technician and minimal stimulation was used. Stimulation from the technician involved rubbing the flank and pressure on the back of the gilt. Breedings were scored using the following criteria: 1) Excellent breeding, gilt showed a standing reflex as soon as BPT was applied, gilt stood well for the entire breeding; 2) Very Good Breeding, gilt did not show a standing reflex as soon as BPT was applied, needed more stimulation than 1), but stood well for the entire breeding; 3) Good Breeding, gilt exhibited a standing reflex as soon as BPT was applied, but did not stand well to BPT through the entire breeding; 4) Satisfactory Breeding, gilt did not show a standing reflex as soon as BPT was applied, and did not stand through the entire breeding period; 5) Bad Breeding, gilt did not show a standing reflex as soon

as BPT was applied, needed more stimulation from technician than 4) to finally elicit a standing reflex, and did not exhibit the standing reflex through the entire breeding.

Differences between BG, GB and BS gilts for age at puberty, days to puberty from initial boar exposure, weight, backfat, duration of pubertal estrus, leptin and IGF-1 concentration and total and average feed intake were analyzed using the SAS GLM procedure (SAS, 1990). For all these variables, only treatment was included in the model because all other variables were not significant. Age, weight and backfat depth at 160 d were used as covariates for age, weight and backfat at puberty, respectively. If treatment differences were detected, multiple comparisons were performed between means, adjusted by Student-Newman-Keuls test (SAS, 1990). All data are represented by mean ± standard deviation. Differences in the number of gilts not pubertal at the completion of the trial were analyzed using chi-square analysis (SAS, 1990). SAS ANOVA was used to test for equal variances between treatments for age at puberty and days to puberty from initial boar exposure (SAS, 1990). Correlation analysis (SAS, 1990) was used to determine the associations between IGF-1, leptin, backfat depth and age at puberty.

Differences between NC and BC gilts for breed score 1 and 2, and subsequent litter size were analyzed using the SAS GLM procedure (SAS, 1990). Treatment and the cycle at which a gilt was bred were included in the model. If treatment differences were detected, multiple comparisons were performed between means, adjusted by Student-Newman-Keuls test (SAS, 1990).

3.3 Results

3.3.1 Part 1

Of the 89 gilts that were allocated to treatment, a total of 21 animals were removed from the analysis: six exhibited silent ovulations, nine reached their pubertal estrus within 3 d of the initiation of boar exposure and six gilts were lame. Of the remaining 68 gilts, there were no differences ($p > .05$) between GB, BG, and BS gilts in

age (159.2 ± 3.2, 159.8 ± 3.9 and 159.1 ± 3.5 d), weight (103.5 ± 8.5, 109.0 ± 6.8 and 108.2 ± 12.6) or backfat depth (12.3 ± 2.0, 13.1 ± 2.5 and 13.5 ± 4.0) respectively, at the start of treatment.

Table 3.1 shows the various parameters measured at the first pubertal estrus for gilts. GB gilts were younger ($p < .05$) than BS gilts, with BG gilts being intermediate for age at puberty. Direct boar contact reduced the interval from initial boar contact to puberty in GB and BG gilts compared with BS gilts ($p < .05$). There were no differences ($p > .05$) between treatment for pubertal weight, backfat thickness, lifetime growth rate, or duration of pubertal estrus. Treatment affected total and average feed disappearance ($p \leq .05$). However, when total feed disappearance was corrected number of days on trial, BS gilts consumed 13 and 5 % more feed than GB and BG gilts, respectively.

The effect of method of boar exposure on attainment of puberty is shown in Figure 3.2. GB and BG gilts showed a similar pattern of puberty onset; however, BS gilts showed a delay of 12 d before any gilts reached their pubertal estrus. The proportion of gilts to reach puberty by 215 d was not different between treatments, GB (96%; n=1), BG (82%, n=4) and BS (81%; n=4). The distribution of gilts reaching puberty by treatment is shown in Figure 3.3. A large range in age at pubertal estrus from the first gilt to the last gilt was found within each treatment group. All distributions were somewhat skewed to the right and did not represent a normal distribution. Analysis of variance results demonstrated that variances between treatments were not significantly different ($p = .22$).

There were no differences ($p > .05$) between GB, BG, and BS gilts in leptin concentration (3.7 ± 1.7, 3.4 ± 1.4 and 3.4 ± 2.2 ng/ml) or IGF-1 concentration (129.3 ± 20.2, 128.9 ± 29.5 and 132.6 ± 25.5 ng/ml), respectively, at the start of treatment. Backfat depth and leptin concentration at 160 d of age were positively correlated (Figure 3.4) ($p \leq .05$). No relationships were detected between leptin or IGF-1 concentration at 160 d of age and the interval from initial exposure to a vasectomized boar to puberty ($p > .05$) (Figure 3.5).

3.3.2 Part 2

At breeding there was no differences ($p > .05$) between BC and NC treatments in age (244.1 ± 120.1 and 241.3 ± 241.2), weight (156.9 ± 15.7 and 152.0 ± 13.5), backfat thickness (18.3 ± 2.3 and 18.3 ± 3.5) or estrus gilts were bred on (2.9 ± 0.8 and 2.6 ± 0.5). Table 3.2 shows the effect of the presence of a boar at breeding on the quality of breed, total piglets born alive and number of gilts not pregnant. Using the objective criteria outlined, NC had a higher score than BC for both breedings ($p \leq .002$). However, there was no difference ($p > .05$) between treatments for the total number of piglets born live at term or number of gilts not pregnant.

3.4 Discussion

Previous research has concluded that daily exposure to a mature boar is a potent stimulus for the attainment of puberty (Karlboom, 1981; Deligeorgis et al., 1984; Paterson et al., 1991). Zimmerman et al. (1996) found that physical boar contact reduced the age at puberty compared with fenceline contact with a boar (186.9 vs 199.4 d) with first contact with boars at 172 d. Our results confirm that puberty induction using fenceline stimulation is less effective than direct boar contact as measured by the number of days from initial boar exposure to puberty in gilts. Also, BS gilts were oldest at puberty, GB gilts were the youngest, with GB gilts being intermediate. Our results are similar to those reported by Pearce and Hughes (1985) who demonstrated that gilts reached puberty at a younger age with direct boar exposure. The method of direct boar contact did not affect age at puberty, supporting the results of van Lunen and Aherne (1987) who found no significant difference in age at puberty (209.9 vs 211.8 d), or the percentage of gilts reaching puberty by 270 d (68.2 vs 47.6 %), between gilts taken to the boar's pen or a boar moved to the gilts pen, starting at 140 d. However, Scheimann et al. (1976) reported that a higher proportion of gilts cycled when they were taken to the boar pen (68 %) rather than the boar was taken to the gilt's pen (48 %).

The different methods of boar exposure also affected the dynamics of the puberty response. BS experienced a delay in the first response to puberty stimulation as compared with GB and BG gilts. As reviewed by Hughes et al. (1990), the boar effect operates via a synergistic action of several boar stimuli including visual, tactile, auditory and olfactory stimuli. The increased days to estrus in stall-housed gilts may be due to the lack of any tactile stimulation from the boar.

Including only the gilts that reached puberty prior to the termination of the experiment, the age that gilts reached puberty was quite variable, ranging from 163 – 210 d. The distribution of age at which gilts reached puberty was somewhat skewed to the right probably because gilts were first exposed to a boar at 160 d of age in this experiment. If gilts were exposed to a boar at a younger age, we may have seen a more normal distribution. However, to a producer, the distribution of gilts reaching puberty is important. A more skewed distribution may be desirable since a higher percentage of gilts will cycle sooner after first boar contact and onset of first heat will be more synchronized (Levis, 2000).

Klindt et al. (1999) reported that reduced prepubertal feed intake from 13 to 25 wk of age in gilts did not adversely affect reproductive performance. These authors examined gilt efficiency as measured by quantity of feed consumed during development per live embryo in gestation and suggested that moderately energy-restricted gilts may be more efficient than ad-libitum fed gilts. Total and average feed intake to puberty appeared to differ among treatments in our experiment, as total feed disappearance for BS gilts was higher than GB gilts, with BG gilts being intermediate. However, BS gilts were on trial for approximately 10 more days than GB gilts. BS gilts ate 13 and 5 % more than GB and BG gilts, respectively. Gonyou et al. (1992) reported a 5 % increase in feed intake in individually penned grow-finish pigs compared with pigs in small groups over a ten week period. However, these pigs were younger and housed in smaller groups. One reason for the slight difference in feed intake we reported might be related to aggression between pigs. If a hierarchy were formed, it may result in a more dominant pig preventing a more timid pig from eating. Also, BS gilts were heavier at their

pubertal estrus than GB; thus, their energy requirements for maintenance would be higher and as a result they would have to consume more feed to meet these requirements.

Leptin and IGF-1 have been implicated as factors affecting the onset of puberty. However, Cheung et al. (1997) suggested that leptin was not the rate-limiting determinant for puberty onset, but is instead a permissive factor that allows puberty to proceed. Once the threshold of leptin is achieved, leptin may alert the reproductive system that the metabolic status is adequate to support and maintain a reproductive cycle. Hiney et al. (1991, 1996) suggest that IGF-1 may represent one of the "metabolic signals" thought to be involved in the initiation of puberty; IGF-1 circulating levels increase strikingly during puberty across a wide range of species, and a decreased level of IGF-1 may contribute to a delayed onset of puberty. Armstrong et al. (1994) demonstrated that, in gilts immunized against growth hormone to reduce serum IGF-1 concentrations, onset of puberty was not affected. However, ovulation rate was reduced. Because boar exposure began at d 160 in this trial, and we know that F1 gilts of the same genotype can reach puberty as young as 135 d (Patterson, Chapter 4), it is not surprising that our data did not detect a relationship between IGF-1 or leptin concentration and age at puberty. Further, Beltranena et al. (1991) demonstrated that in gilts achieving at least a growth rate of .60 kg/d, growth rate did not affect pubertal age. Therefore, it is doubtful that growth or metabolic state was limiting age at puberty in the present study, because the gilts in this study would have reached the minimum growth and metabolic state necessary to attain sexual maturity.

Backfat depth was significantly correlated to blood leptin concentration at 160 d of age. These results are consistent with the data of Hamann and Matthaei (1996), demonstrating that the amount of leptin secreted from adipocytes is significantly correlated to body mass index (BMI) and percent body fat in humans, and the results of Robert et al. (1998) indicating that leptin expression can be associated with subcutaneous fat accumulation in pigs. However, concentration of leptin at d160 was not correlated to age at pubertal estrus in our study.

Several authors have demonstrated that the presence of a boar at breeding provides important stimuli that may improve reproductive performance. As reviewed by Soede (1993), the duration of the standing response evoked by the back pressure test alone is shorter than the standing response evoked by the BPT where a boar is present to provide olfactory and auditory stimuli. Our data show that, using objective criteria, breedings without the presence of a boar scored lower than gilt breedings in the presence of a boar. Although litter size was not significantly different between treatments, the one pig difference in litter size would be an important economic advantage if substantiated in larger trials.

In conclusion, our data demonstrate that puberty induction using direct boar contact is unequivocally more effective than fenceline contact. No differences existed between the two direct boar contact treatment groups. Further, we have demonstrated that the presence of a boar at breeding improved the quality of artificial insemination.

Table 3.1 Mean (± sd) age, weight, backfat depth (P2), growth rate, estrus length and feed intake for gilts receiving direct contact with a vasectomized boar in a purpose built boar stimulation area (GB), direct contact with boars in gilt group pens (BG), and fenceline contact between boars and gilts housed in individual gilt stalls (BS).

	Pubertal Estrus						Feed Intake[3]	
	Age (d)	Days to estrus[1]	Weight (kg)	Backfat (mm)	Growth Rate[2] (kg/d)	Estrus Length[3] (h)	Total (kg)	Average (kg/d)
GB	180.9[a]	21.8[a]	124.3	15.8	.68	37.6	65.0[a]	3.0[a]
(n = 25)	(12.3)	(13.4)	(16.6)	(4.4)	(.06)	(15.4)	(35.1)	(.3)
BG	183.8[ab]	24.0[a]	129.8	15.7	.71	40.5	80.1[b]	3.1[ab]
(n = 22)	(16.4)	(18.6)	(20.0)	(3.0)	(.06)	(11.7)	(63.3)	(.3)
BS	191.1[b]	32.0[b]	139.2	17.3	.73	45.4	110.4[c]	3.3[b]
(n =21)	(13.0)	(14.6)	(20.3)	(4.3)	(.07)	(17.0)	(58.3)	(.5)
p	.039	.039	.12	.45	.35	.24	.017	.03

[1] Interval from initial exposure to a vasectomized boar until puberty

[2] Lifetime growth rate (birth – onset of puberty)

[3] Total and Average feed consumed over the entire period from initial boar exposure until puberty

[a,b,c] means in a column with different subscripts are different ($p < .05$).

Table 3.2 Mean (± sd) breeding scores and total number of piglets born live for gilts receiving fenceline contact with a boar during breeding (BC) and receiving no boar contact during breeding (NC).

Treatment	12-h Breed Score[1]	24-h Breed Score[1]	Total Liveborn Piglets	Number non-pregnant
BC (n=21)	1.4 (.76)	1.5 (.99)	11.1 (2.7)	1
NC (n=14)	2.7 (1.2)	2.9 (1.2)	10.1 (2.4)	0
p-value	0.001	0.002	.27	-

[1] Breed Score:

1 - Excellent breeding, gilt showed a standing reflex as soon as BPT was applied, gilt stood well for the entire breeding

2 - Very Good Breeding, gilt did not show a standing reflex as soon as BPT was applied, needed more stimulation than 1), but stood well for the entire breeding

3 - Good Breeding, gilt exhibited a standing reflex as soon as BPT was applied, but did not stand well to BPT through the entire breeding

4 - Satisfactory Breeding, gilt did not show a standing reflex as soon as BPT was applied, and did not stand through the entire breeding period

5 - Bad Breeding, gilt did not show a standing reflex as soon as BPT was applied, needed more stimulation from technician than 4) to finally elicit a standing reflex, and did not exhibit the standing reflex through the entire breeding.

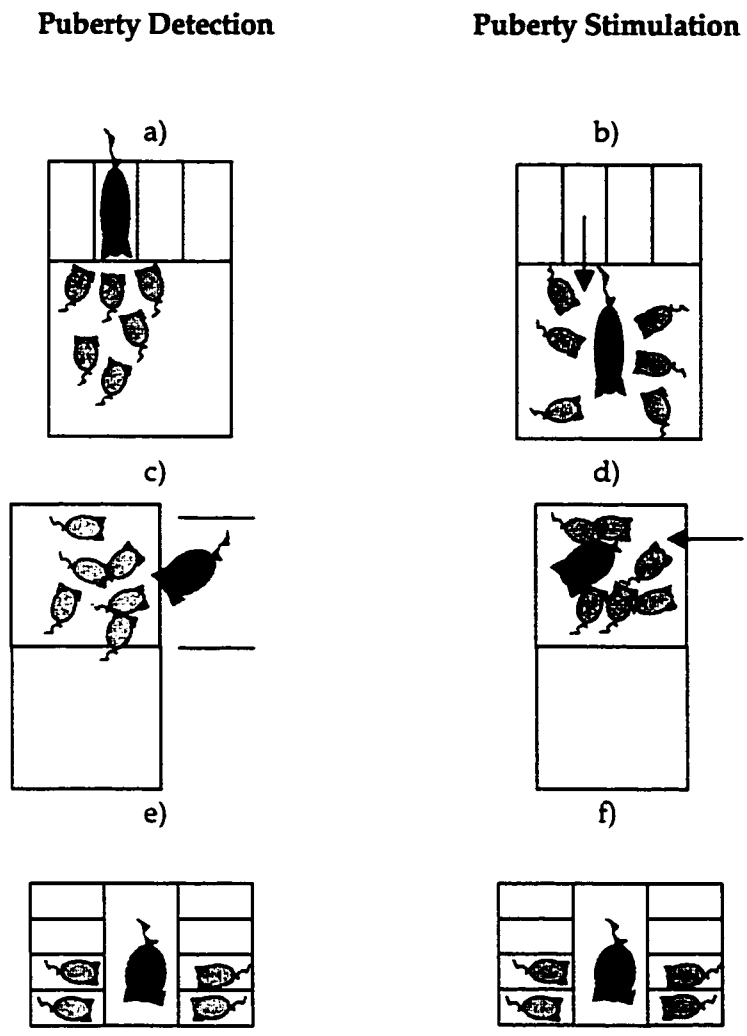


Figure 3.1 Method of puberty detection and puberty stimulation for Gilt-boar (GB), Boar-gilt (BG) and Boar-Stall (BS) treatment groups. All groups of gilts received fenceline contact with a vasectomized boar for puberty detection. a, b) puberty detection and stimulation for GB; c, d) puberty detection and stimulation for BG; and e, f) puberty detection and stimulation for BS.

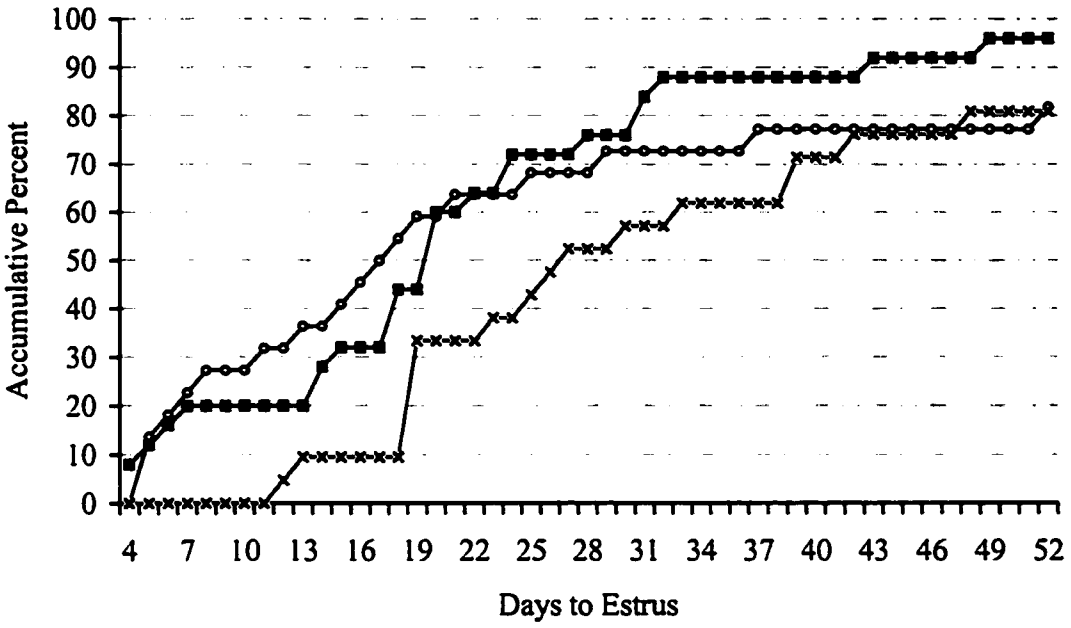
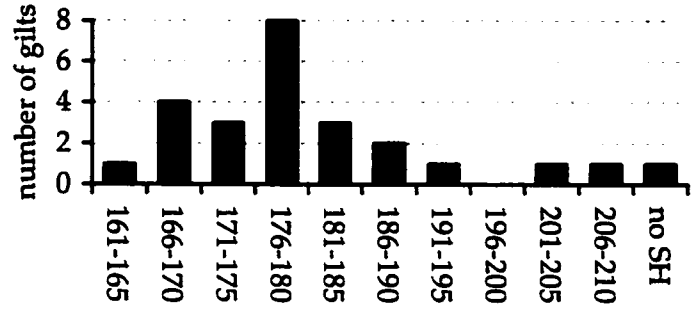
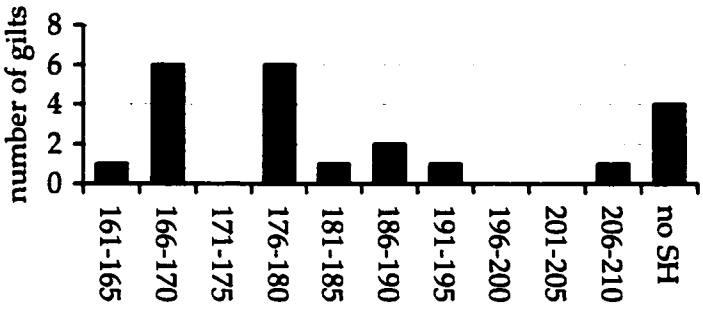


Figure 3.2 Accumulative percentage of gilts attaining puberty in response to direct contact with a vasectomized boar from 160 days in a purpose built boar stimulation area (GB), direct contact with boars in gilt home pens (BG), and fenceline contact between boars and gilts housed in individual gilt stalls (BS). Open circles represent BG, closed squares GB and x's BS.



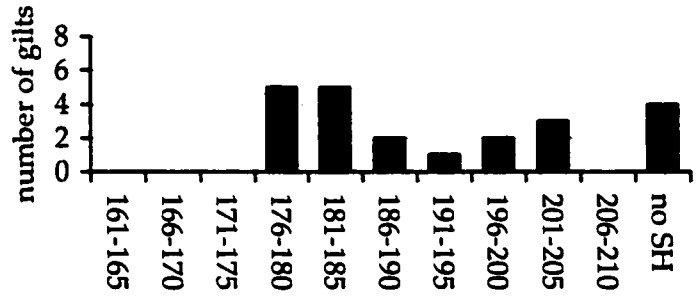
Average age – 180.9
Standard deviation – 12.3
Minimum age – 164
Maximum age - 210

a)



Average age – 183.8
Standard deviation – 16.4
Minimum age – 163
Maximum age - 209

b)



Average age – 191.1
Standard deviation – 13.0
Minimum age – 176
Maximum age - 203

c)

Figure 3.3 Number of gilts reaching puberty in successive 4-day periods after first exposure to a boar at 160 d; a) boar in a purpose built boar stimulation area (GB), b) boar in gilt group pens (BG) and, c) fenceline contact between boars and gilts housed in individual gilt stalls (BS).

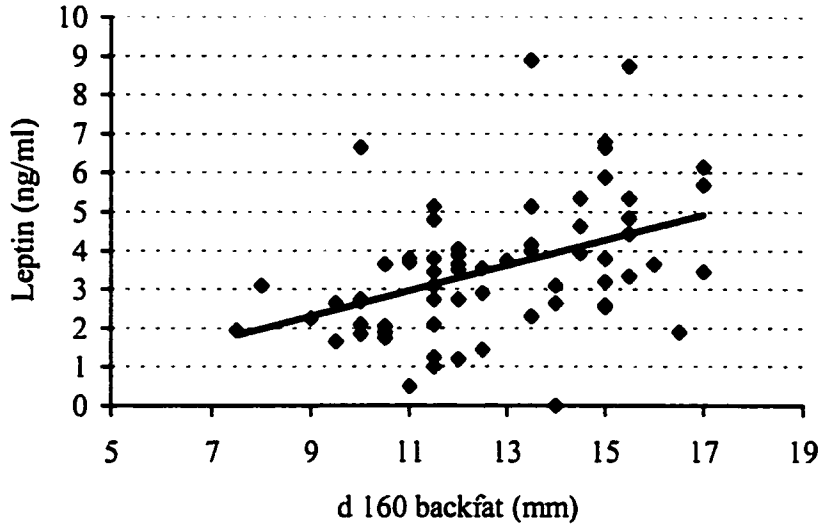
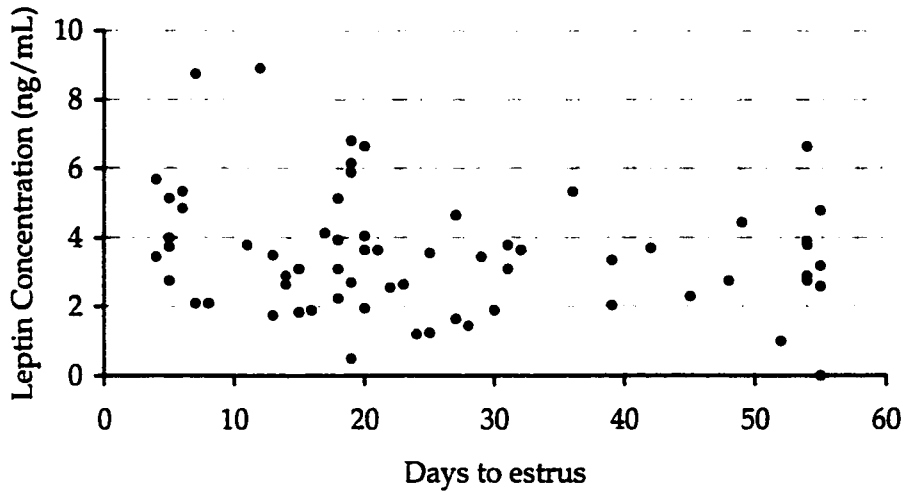
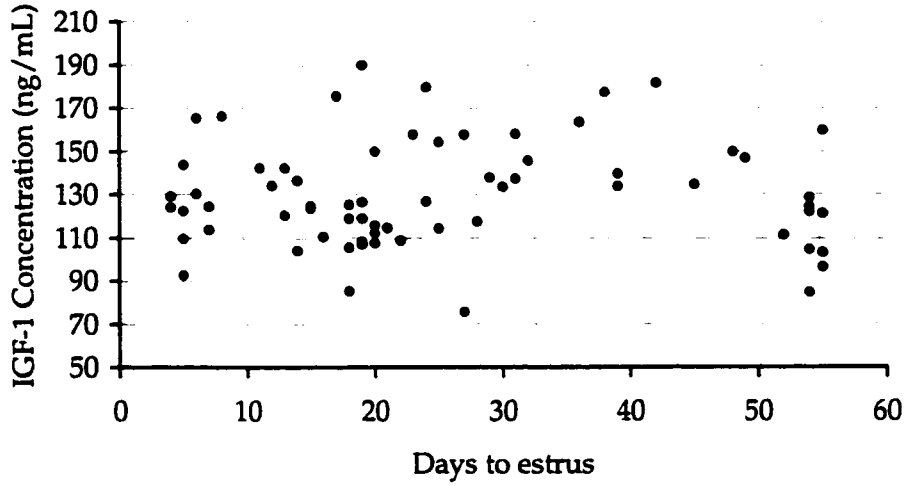


Figure 3.4 Relationship between plasma leptin concentration and backfat depth at 160 d; Leptin concentration d160 = -.64 + .32(d160 backfat depth), p =.0004, R^2=.18.



a)



b)

Figure 3.5 Lack of a relationship between interval from initial boar exposure until puberty attainment and a) leptin concentration at d160 (leptin concentration d160 = 4.07 - .02 (days to estrus), p=.12, R^2=.04) and b) IGF-1 concentration at 160 d (IGF-1 concentration d160 = 130.13 + .0005 (days to estrus), p = .998, R^2 = 0).

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CHAPTER 4

THE EFFECT OF LEAN GROWTH RATE ON PUBERTY ATTAINMENT IN GILTS

4.1. Introduction

The relationships that exist between age at sexual maturation and specific components of growth have been studied by a number of authors (Kirkwood and Aherne, 1985; Young et al., 1990; Rozeboom et al., 1995). Beltranena et al. (1993) suggest that within the range of acceptable commercial growth rates, the onset of puberty may be genetically determined rather than being dependent on growth performance. There is consensus in the literature concerning detrimental effects on puberty attainment in gilts by restricting growth. Recently, Le Cozler et al. (1999) have demonstrated that a restriction to 80 % *ad libitum* feed intake had negative effects on age at puberty. Gaughan et al. (1997) suggested that if gilts fail to reach their optimal level of protein retention, reproductive development might be affected. Similarly, Cia et al. (1998) reported that a reduction in protein mass or an impairment of the metabolic status of the animal has detrimental effects on subsequent reproductive performance.

The current trend in the production of market pigs places emphasis on lean tissue growth rate. The data available concerning the impact of high lean growth rate on sexual development of gilts is limited and somewhat contradictory. Rydhmer (1992) suggests that breeding for leanness may delay the onset of puberty. However, more recent work by Kerr and Cameron (1998) and Cameron et al. (1999) suggests that selection for lean growth rate had no adverse affects on sexual development. A comparison between gilts fed to achieve their maximum lean growth potential and those restricted in lean growth would provide additional information on this hypothesis.

A paper based on Chapter 4 will be submitted to Journal of Animal Science by J. L. Patterson, R. O. Ball, H. J. Willis, F. X. Aherne, and G. R. Foxcroft

Booth et al. (1994) demonstrated that changes in metabolic status could mediate short-term effects on reproductive function. Similarly, Prunier et al. (1993) indicated that feed restriction of 50% retarded growth and sexual development of gilts, and this effect was mediated by changes in plasma concentration and secretion of metabolic hormones. The metabolic state of an animal can be detected through changes in circulating hormones such as leptin, insulin and IGF-1 (Booth et al., 1994; Foster and Nagatani, 1999). Therefore, measurement of blood leptin, insulin and IGF-1 concentrations at critical time points may give an indication of the metabolic status of gilts receiving diets that would influence the rate of lean and fat deposition.

The objective of this study was to use dietary manipulation, within the limits achievable with typical farm ingredients, to determine the effect of different lean growth rates on puberty attainment in gilts. Weekly weight and ultrasound measurements to determine body components were used to characterize growth components in the gilts and to examine relationships with the onset of puberty.

4.2 Materials and Methods

4.2.1 Animals

Gilts were fed a standard grower diet until they reached approximately 50 kg, after which they were assigned to a treatment and fed one of two treatment diets until they reached puberty. All diets were commercially formulated and manufactured in collaboration with Vic Pouteaux, Landmark Feeds, Strathmore, Alberta, Canada. Each experiment was divided into two phases: *GROWTH* (50 kg to 130 d) and *STIMULATION* (130 d to puberty).

4.2.1.1 Experiment 1

One hundred and sixty-eight prepubertal Genex Manor hybrid F1 gilts, born to primiparous sows, were assigned to either; 1) a diet formulated to maximize lean growth potential (LP; n=84), or 2) a diet formulated to produce lower lean growth, but

similar fat growth to LP (LL; n=84) (Table 4.1). Gilts were housed in either barn 1 (n = 72) or barn 2 (n=96), in groups of six. Space allowance was calculated as recommended for growing pigs on fully slatted (barn 1) and partially slatted (barn 2) floors, with each gilt permitted at least 1.2 m^2 space allowance throughout the entire trial (Agriculture and Agri-Food Canada, 1993). In barn 1, gilts were housed on fully slatted floors, with natural daylight from overhead skylights. Gilts were stratified by weight, age, and litter origin to treatment. Littermate gilts were only selected from litters that had at least eight pigs born alive, of which 5 were gilts. Each treatment had two littermates, housed in separate pens. In barn 2, gilts were housed in pens with partially slatted floors, with artificial light from 0700 until 1700 h. Gilts were stratified by weight, age, and litter. Littermates were allocated across treatment as far as possible. All gilts, in both barns, were allowed *ad libitum* access to water and to treatment diets from two-holed feeders.

4.2.1.2 Experiment 2

Forty-eight prepubertal Genex Manor hybrid F1 gilts, born to second parity sows, were used in this trial. At approximately 50 kg, gilts were stratified by weight, age and litter origin and assigned to either; 1) the same diet formulated to maximize lean growth potential as used in Experiment 1 (LP; n=24); or 2), a diet formulated to restrict lean growth potential further than was achieved in LL in Experiment 1(RL; n=24) (Table 4.1). Gilts were stratified to treatment by weight, age, and litter. Littermates were allocated across treatment as far as possible. All gilts were fed treatment diets *ad libitum* and housed in groups of six with at least 1.2 m^2 of pen space per gilt. Gilts were housed in pens with partially slatted floors, with artificial light from 0700 until 1700 h. Each pen contained one electronic feeder (Feed Intake Recording Equipment, FIRE, Hunday Electronics, Newcastle, U.K.), which allowed access to the feeder by only one pig at a time. One week prior to beginning the trial, all gilts were fitted with an ear tag transponder, and became accustomed to the tag and feeder. When gilts visited the feeder, they were identified from an ear tag transponder. Entry and exit times, and total amount of feed consumed (kg), were transmitted to a dedicated on-line computer and all data were stored for later analysis.

4.2.2 Stimulation period

Starting at approximately 135 days of age, pens of gilts were taken to a boar stimulation pen, and received 20 min direct exposure to a boar daily as a pen group for pubertal stimulation. In barn 1, experiment 1, 4 mature vasectomized boars were used in rotation. In barn 2, experiment 1, 2 mature vasectomized boars and 4 boars of the same age as the gilts were used in rotation. In experiment 2, 4 mature vasectomized boars were used on a rotational basis. Physical signs, such as redness, degree of swelling, and discharge from the vulva were recorded daily during the stimulation period. Puberty attainment was determined as the day gilts first exhibited the standing reflex in response to contact with a boar. Boars were not permitted to breed the gilts. At pubertal estrus, body weight, backfat and loin depths were recorded.

4.2.3 Ultrasound and weight measurements

At weekly intervals, gilts were weighed and backfat and loin depth were determined ultrasonically (Ultra Scan 900, Alliance Medical Inc., St-Laurent, Quebec). A single longitudinal scan using a 3.5 MHz linear probe was obtained by placing the transducer probe parallel to the midline of the gilt approximately 5 cm from the midline. The scan was measured from the last rib until approximately the 3 or 4th last rib. A mark was then placed on the gilt's back with a permanent marker, and was still visible the following week, allowing successive scans to be taken from the same location. At the time of the ultrasound, the scan was visually appraised and loin and backfat depth measured.

4.2.4 Blood and feces collection

At the beginning of the *stimulation* period (135 d), a 5 ml jugular blood sample was taken from all gilts during a brief period of nose-snare restraint. All samples were centrifuged at 1,500 x g for 15 min and plasma decanted and stored at –20 ° C until analysis for leptin, IGF-1, and insulin concentration.

Gilts in experiment 1, barn 1, were relocated to a stall the day their first pubertal estrus was reached (day 1). Gilts from experiment 2 were housed in their original group pen until d 6 after estrus. All gilts were fed *ad libitum* until d 6, when feed was removed at 1600 h. At 0745 h on d 7, gilts were fed an amount of feed equaling 2 X maintenance requirements. The amount of feed consumed was measured and recorded. Gilts in experiment 1, barn 2, were not feed restricted, due to limitations in barn space and layout. At 0915 h, a 5 ml jugular blood sample was taken from all gilts.

Every 10 d from the start of puberty stimulation, a fecal sample was taken from each gilt using the technique of Sanders et al. (1994). All samples were stored at –30° C until subsequent analysis for progestagen concentration to determine reproductive activity in the gilts.

4.2.4 Radioimmunoassay

For all radioimmunoassay (RIA) analysis, all samples were analyzed in duplicate. The sensitivity of the assay was calculated using the following equation: average of the zero binding tube [(Bmax) – 2SD(Bmax) / average (Bmax)] X 100. Plasma IGF-1 concentrations were determined using the homologous double antibody RIA described by Cosgrove et al. (1992), with modifications relating to the antiserum as described by Novak et al. (2000). 100 µl of plasma was initially extracted with 3 ml of acid ethanol. Radio inert recovery efficiency was 109%. The intra-assay CV was 9.7%, and sensitivity of the assay, estimated as 95.2% bound, as 14.9 ng/ml. Plasma leptin concentrations were determined using the multi-species double antibody kit assay previously validated by Mao et al. (1999) for use in our laboratory. For the two assays run, intra-assay CV's were 9.2 and 8.9 %, the sensitivity of the assays determined at 91.5 and 109.7% bound were 1.7 and 1.4 ng/ml. The inter-assay CV was 10.6%. Plasma insulin concentrations were determined in duplicate using the homologous double antibody RIA described by Cosgrove et al. (1992), modified with the use of an anti porcine insulin first antibody purchased from ICN Biomedicals Inc. (1263 S. Chillicothe Road, Aurora, Ohio, USA, 44202, catalogue number 65-1041) and used at a final assay

dilution of 1/350 000. The intra assay CV's were 7.5 and 10.8 %, of the inter-assay CV was 2.2 %. 100µl of plasma was initially assayed. If necessary, high potency samples giving less than 26% binding were diluted and re-assayed. Sensitivity of the assays determined as 93.6 and 94.1% bound was 0.035 and 0.067 ng/ml.

Dr. Franz Schwarzenberger (Institut für Biochemie and Ludwig Boltzmann für veterinärmedizinische Endokrinologie, Vienna, Austria, personal communication) provided the procedure for fecal progesterone analysis. Fecal samples were thawed and thoroughly mixed by hand and 0.5 g of each sample, 0.5 ml water, and 4.0 ml 88% methanol were added to a screw-topped extraction vial. Tubes were vortexed for 30 min. After centrifugation (2000 g X 30 min), 0.06 ml of the methanol extract was diluted 1:5 with assay buffer and 0.05 ml of the diluted sample extract was taken to assay. Samples were assayed using a kit RIA (Coat-a-Count Progesterone, Diagnostic Products Corporation, Los Angeles, CA, USA). The intra- and inter- assay CV's for the three assays run were 13.4 and 7.0%, respectively. Assay sensitivity defined as 88% bound was 9.5 pg/tube. Percent hot recovery was 58.2 ± 5 % and data were not corrected for hot recovery. No difference was found between the slope of the standard curve and a control curve established with .050, .025 and .0125 mL of diluted extract per tube.

4.2.5 Statistical Analysis

Loin eye area (LEA), carcass fat-free lean (FFL), carcass fat-free lean gain (FFLG) and fat growth rate (FATGR) were estimated using the following equations:

LEA = exp (-0.54 + (.068895*loin) -(.000432*(loin)**2)).

FFL = 1.4357 + (.7632*2) - (14.7839*backfat depth) + (4.9574*LEA) + (.3312*live weight) (National Pork Producers Council, 1999)

$$\textbf{FFLG} = \frac{\text{(Final fat-free lean)} - \text{(Carcass fat-free lean at start of experiment)}}{\text{number of days from start to final measurement.}}$$

Total body lipid = (Backfat depth (mm) – 0.5) X body weight (kg) (Whittemore, 1988)

$$\textbf{FATGR} = \frac{\text{(Final lipid body wt)} - \text{(Initial lipid body wt at start of experiment)}}{\text{number of days from start to final measurement.}}$$

Because lean gain is influenced by starting and final weights, lean growth rate was determined using the adjustment factors in the NRC (1998).

For analysis, this experiment was divided into two phases: growth and stimulation. The *growth* phase covered the period from the start of the experiment until initiation of boar exposure (~ 50 kg – 135 d). The *stimulation* phase was from the initiation of boar exposure until detection of pubertal estrus (135 d – puberty). At the start of each phase, and at the time of onset of puberty, differences between treatment for weight, backfat and loin depth, and also age at puberty and days to puberty from initial boar exposure, were analyzed using the SAS GLM procedure (SAS, 1990). In experiment 1, treatment, barn, pen and litter were used as class variables, and pen(barn*trt) was used as the error term. In experiment 2, treatment and litter were used as class variables. For the analysis of weekly growth characteristics and feed intake over the growth and stimulation periods in experiment 1, treatment, barn, pen, time and litter were used as class variables in the analysis. Pen(barn*trt) was used as the error term. For experiment 2, treatment, time and litter were used as class variables in the analysis. Stepwise analysis from a full to a reduced model was completed. Any variable with $p > .20$ was removed from the model, and the model re-run. If treatment differences were detected, multiple comparisons were performed between lsmeans, adjusted by Tukey-Kramer test (SAS, 1990). Relationships between various components measured in this experiment were analyzed using SAS REG (SAS, 1990). PROC REG stepwise procedure was used to detect the relationships between age at puberty and components of growth (SAS, 1990). To detect differences in the proportion of gilts pubertal and prepubertal at each week after boar stimulation, a chi-square analysis was performed (SAS, 1990).

4.3 Results

4.3.1 General results

Of the 168 gilts that were allocated to treatment in experiment 1, a total of 16 animals were removed from the analysis: five gilts did not achieve pubertal estrus by 200 d, five gilts were lame, one gilt was a hermaphrodite, four gilts were removed on the basis of high fecal progesterone before recorded pubertal estrus (previously cyclic but anestrous) and one gilt was mistakenly removed from trial. Gilt removals were unrelated to treatment. Of the remaining 152 gilts, there were no differences ($p > .05$) between LP and LL gilts in weight (54.2 ± .3, and 53.8 ± .3 kg) or age (96.1 ± .3, and 96.6 ± .3 d), respectively, at the start of treatment.

Of the 48 gilts that were allocated to treatment in experiment 2, a total of 5 animals were removed from the analysis: one did not reach puberty by 200 d, three were removed because of ill health, and one gilt was lame. Gilt removals were unrelated to treatment. Of the remaining 43 gilts, there were no differences ($p > .05$) between treatments LP and RL gilts in weight (50.0 ± .7, and 51.2 ± .8 kg) or age (88.0 ± .8, and 87.3 ± .8 d), respectively, at the start of treatment.

4.3.2 Feed Intake and Growth Characteristics

Differences in average daily feed, lysine and metabolizable energy intake are shown in Table 4.2 and 4.3. In experiment 1, during the growth period, LP consumed more lysine and energy than RL ($p < .05$), however there was no difference in feed intake ($p > .05$). In experiment 2, during the growth period, LP consumed more lysine and energy than LL ($p < .05$) even though LL consumed more feed than LP ($p > .05$).

The use of ultrasound was an effective method to estimate body composition in gilts (Figure 4.1). There were no differences in any of the growth characteristics measured at the start of the trial (Tables 4.4 and 4.5). In experiment 1, differences in

weight, backfat and loin depth were detected ($p < .05$) at the start of puberty stimulation. However, treatment did not result in differences in weight, backfat and loin depth at the start of puberty stimulation in experiment 2 ($p > .05$).

Over the growth period, dietary treatment was effective in inducing differences in whole body growth rate and lean growth rate in experiment 1 ($p < .05$) (Table 4.2). In experiment 2, treatment resulted in differences in whole body, lean and fat growth rate ($p < .05$) (Table 4.3). Fat-free lean weight and fat weight increased linearly over the duration of the experiment ($p < .05$) (Figure 4.2). The effect of litter origin was highly significant ($p = .0001$) for all the parameters measured in both experiments.

4.3.3 Pubertal Data

No significant differences in age at puberty were detected in either experiment 1 or 2 ($p > .05$) (Table 4.4 and 4.5). However, in experiment 1, more LP gilts reached puberty within 7 days of initial boar contact than did LL gilts ($p = .046$) (Figure 4.3) with no difference in subsequent weeks. In experiment 2, there was no difference in the weekly proportion of gilts reaching puberty ($p > .05$). When comparing the common LP treatment between experiments, a larger proportion of LP gilts in experiment 1 attained puberty in weeks 1, 2, 3 and 4 than did LP gilts from experiment 2 ($p< .05$).

Average lean growth rate from the start of the growth phases is plotted against age at puberty in Figure 4.4. No relationship between age at puberty and lean growth rate during the growth period was detected using either a linear or second-degree polynomial model ($p > .05$). Lean and fat tissue growth rate over the stimulation period is illustrated in Figure 4.5. However, a plot of lean and fat tissue growth rate over the stimulation period for an individual gilt, illustrates a much higher R^2 for both lean and fat tissue growth rate (Figure 4.6). Average lean growth rate at puberty was negatively correlated to age at puberty ($p < .05$) in both experiments (Figure 4.7). At puberty, body composition of gilts varied considerably and the plot of fat-free lean weight at puberty represented a normal distribution (Figure 4.8).

Simple correlations between weight, backfat depth and fat-free lean weight at puberty are shown in Table 4.6. Stepwise linear regression was used to determine the most accurate equations to predict age at puberty in gilts. Age at puberty was estimated by weight and growth rate at puberty in experiment 1 and 2 (Table 4.7 and 4.8). Addition of various other body measurements and components only slightly improved the fit of the equation.

4.3.4 Leptin, IGF-1, and insulin concentrations

Total feed intake, total energy intake, IGF-1, leptin and insulin concentrations on day seven following the onset of standing heat are shown in Table 4.9 and 4.10. In experiment 1, total feed consumed and insulin concentration differed ($p < .05$). In experiment 2, total energy consumed differed between treatments ($p < .05$), no other significant relationships were detected.

No significant relationships were detected between plasma concentrations of leptin, IGF-1 and insulin at the time of initiation of boar exposure and age at the onset of puberty in experiment 1 ($p > .05$) (Figure 4.9). In experiment 2, no significant relationships were detected between leptin and insulin concentration and age at the onset of puberty ($p > .05$), however, a significant relationship was detected between IGF-1 concentration at the time of initiation of boar exposure and age at puberty ($p < .05$) (Figure 4.10).

4.4 Discussion

Puberty has been associated with a critical growth rate (Rydhmer, 1994; Beltranena, 1992), fatness (Frisch, 1980; Gaughan et al., 1997), and age (Hughes and Varley, 1980) however, the interactions amoung these factors is unclear. The current trend in pig production places emphasis on lean tissue growth. However, few data are available concerning the effect of high lean growth rates on sexual development in gilts.

Therefore, to examine the effects of high lean growth rate on puberty attainment in gilts, we used dietary manipulation to produce differences in body composition in gilts. The diets formulated in this trial were effective in inducing differences in weight, growth rate, lean and fat growth rate from the start of the experiment until gilts reached their first estrus.

The efficiency of production is related to how closely matched the rate of nutrient intake is to the growing pig's capacity for lean tissue growth (Castell et al. 1994). Daily intake of sufficient protein to meet the requirements will depend on the pig's capacity for growth and the concentration of nutrients (Castell et al. 1994). Consequently, maximal lean growth results when nutrients and energy are provided in the appropriate ratio (Lawrence et al. 1994).

The diets utilized in thus study were formulated and manufactured by a commercial feed mill (Vic Pouteaux, Landmark Feeds, Strathmore, Alberta, Canada). None of the diets were limiting in any nutrient. Because Tylan is a proven growth promotant, and the goal was to reduce growth in RL, it was specifically removed from the formulation for that diet. Energy intake and gut-fill capacity generally limit feed intake in growing pigs (Whittemore, 1998). Thus, the reason the diets contained various different ingredients is because it was hypothesized that maximum gut capacity would limit total feed intake. Therefore the diets formulated to reduce lean growth contained ingredients that "bulked up" the diet, and thus it was predicted that the intake of energy and lysine would be limited. In experiment 1, the diets were formulated to maintain a similar energy:protein ratio. This resulted in similar intake of feed, but because protein and energy content of the diets differed, total intake of lysine and energy per day differed. In experiment 2, a different approach was taken in formulation of the diets. Similar energy:protein intakes were not maintained; as a result, total feed consumed differed between treatments, and, although there was less of a difference in energy intake, energy and protein intake both differed between dietary treatments.

Differences in total feed intake between the common treatment within the two experiments may be related to the method of measuring feed intake. In experiment 1, average daily feed intake was measured on a group pen basis, whereas in experiment 2, the FIRE system was used to electronically measure individual feed intake. Feed wastage in experiment 1 may have been higher. A single feeder was placed in the pen, with spaces for two gilts to eat; however, competition to eat and fighting at the feeder may have resulted in feed wastage. In comparison, use of the FIRE system in experiment 2 most likely minimized feed wastage, as only one pig had access to the feeder at a time which would result in less feed wastage.

Total body, lean and fat growth rates achieved in this trial were similar to growth rates achieved in other trials. Utilizing finishing crossbred gilts (~60 –100 kg), Grandhi and Cliplef (1997) reported growth rates ranging from 912 –937 g/d. In gilts (30 – 110 kg) of the same genotype as used in our experiment, growth rates of 822 g /d were detected (Vic Pouteaux, personal communication). Similarly, the lean growth rates achieved in this experiment were within the range of previously published lean growth rates (Table 4.11). Mean fat-free lean gain can be used to estimate the daily protein accretion rate curve (Schinckel and de Lange, 1996). Whole-body protein accretion increases from birth to approximately 45 to 65 kg, whereas after 65 kg, the percentage of body protein decreases as the percentage of lipid increases (Schinckel and de Lange, 1996). Therefore, the period over which lean growth rate is measured will influence the lean growth rate measured. In this experiment the *growth* period corresponded with the period where the highest lean growth rates have been reported. Various factors such as stress, environmental conditions and space allowance, if not ideal, have been shown to have detrimental consequences on the lean tissue growth rates achieved (Schinckel and de Lange, 1996). Therefore, in this experiment, care was taken to ensure that ample space was provided for the gilts and that the environment the gilts were exposed was such that there would be no adverse affects on total body and lean tissue growth rate.

Although only one dietary treatment was common between the two experiments, differences were observed in growth between experiments for treatment LP. Although

lean growth rates achieved in experiment 1 and 2 were similar, overall growth rate and fat growth rate were higher in experiment 2. Several factors could have contributed to these differences, including; 1) birth and weaning weight, which were higher in experiment 2, 2) experiment one was completed during March – August, experiment two was completed during November – March; and 3) experiment 1 was initiated at approximately 54 kg and 96 days, in comparison to experiment 2 at 50 kg and 88 days of age (Table 4.12).

Differences in piglet birth weight between experiments are most likely due to the difference in parity of the dams between experiments. In the pig, undernutrition *in utero* causes low birth weight, a decrease in muscle fiber number, and a reduction in postnatal growth rate within a litter of pigs; as a consequence, lighter-weight pigs have probably suffered some deficit in muscle fiber number (Dwyer et al., 1994). Earlier research from this same group demonstrated that average daily gain from birth to 25 kg was correlated with birth weight but was not correlated with total muscle fiber number. However, from 25 kg to slaughter average daily gain was correlated with muscle fiber number and was not correlated with birth weight (Dwyer et al., 1993). Because of the differences in birth weights between experiments, it may be possible that muscle fiber number was also different, and thus the upper limit to protein deposition may have been higher for gilts from experiment two. The effects of the nursery environment, including low level of disease, ventilation, sanitation, air quality, animal comfort, social behavior, number of pigs per pen and space per pig, may limit the expression of the pig's genetic potential for lean growth from 22 to 50 kg (Schinckel and de Lange, 1996).

The higher growth rates in gilts in experiment two can be attributed to higher fat growth because lean growth rate was similar. Fat deposition occurs when energy intake exceeds needs for maintenance and lean tissue growth. In growing pigs under 50 kg energy intake will determine the rate of protein deposition, whereas in finishing pigs over 50 kg, the upper limit to protein retention will determine the rate of protein deposition (de Lange, 1998, Schinckel and de Lange, 1996). Although, it is likely that energy in the diets was not as formulated, and thus gilts in experiment 2 consumed

more energy than gilts in experiment 1, data on metabolizable content were not available.

Historically, to monitor reproductive activity in a gilt, serial blood samples were taken to determine progesterone status. Although this is an effective means to determine reproductive activity, animal welfare is a concern with repeated blood samples. In recent years, animal welfare issues have been an increasingly important concern to researchers and thus it is important to consider alternative approaches to determine reproductive status in animals. Monitoring corpus luteum function by measuring progestagens in feces is an effective, non-invasive technique used in several laboratories (Schwarzenberger et al., 1997). The technique to measure fecal progestagens has been validated in several species, including swine (Palme et al. 1996; Sanders et al. 1994). Steroid hormones are eliminated through the blood, metabolized in the liver and excreted in the urine and bile as conjugates (Palme et al., 1996). Subsequently, in the gut, steroids are deconjugated and partially reabsorbed; any steroids that are not reabsorbed are then excreted in the feces. We determined that using a simple extraction procedure and a commercially available progesterone kit was effective for detecting whether gilts underwent a silent heat.

It is well established that method of puberty stimulation (Zimmerman et al. 1998; Patterson, Chapter 3), frequency and duration of stimulation (Paterson et al., 1989) and age and sexual behavior of the boar (Zimmerman, 1997; Levis, 1997) are important factors regulating the effectiveness of the boar in puberty onset. For this reason, all gilts were taken to the boar pen for stimulation for a period of 20 minutes once a day. However, one variable that may have contributed to differences in age at puberty was the efficacy of the boar effect. Differences existed between experiments for the age and sexual behavior of the boar. Boars used in experiment 1, barn 1, and experiment 2, were mature boars; on the other hand, boars in experiment 1, barn 2, were young boars, similar in age to the gilts. The young boars had the highest libido, and thus gilts in this barn were possibly subjected to the highest degree of tactile stimulation. The older boars would likely produce more pheromones but had a somewhat lower libido, and

thus were observed closely to ensure they were working well. In all cases, pens of gilts within barn were rotated through the available boars on a daily basis, so the effect of individual differences in boars was removed.

Pubertal stimulation began at 135 days in this trial, which was younger than some other studies (Rydhmer et al., 1992; Gaughan et al., 1997; Cia et al., 1998; le Cozler et al., 1999). Age at puberty in the present experiment was similar to experiments where puberty stimulation was initiated at similar younger ages, 140 days (Young et al. 1990), 120 – 130 days (Rozeboom et al., 1995) and 130 days (Zimmerman et al., 2000) where puberty occurred on average at 167.2, 172.5 and 168.0 days, respectively. Rozeboom et al. (1995) also reported large ranges in weight, backfat, growth rate and lean and fat growth rate at the onset of puberty. Selection for lean growth has been shown to delay (Rydhmer et al., 1992) and to have no effect (Cameron et al., 1999) on puberty onset in gilts. Our results demonstrate that lean growth rate measured over the period from the start of the trial until the initiation of puberty stimulation had no effect on age at puberty in gilts. It is suggested that protein mass may be a regulator of reproductive performance (Cia et al., 1998). Young et al. (1990) speculated that if the onset of puberty were controlled by the accumulation of a certain amount of a body component, plotting a histogram would show skewing of the data at that specific threshold (e.g. weight). By plotting empty protein weight at puberty it was determined that empty protein weight is not a factor in regulating the onset of puberty in gilts (Rozeboom et al. 1995). Similarly, we determined that lean mass at puberty was normally distributed and therefore not a regulating factor in pubertal onset. Foxcroft et al. (1996) reported that the age at which gilts are first genetically capable of starting to cycle would be achieved well after they have reached the minimal growth rate or body weight needed to reach sexual maturity.

Age at puberty was negatively correlated to lean growth rate from the start of the trial until puberty. Protein accretion rates increase, reach a plateau from 35 to 65 kg, and then decline (Schinckel and de Lange, 1995). Therefore, protein accretion rates at higher weights will be lower than protein accretion rates achieved at younger weights and our

results demonstrated that lean growth rate declined as the gilts got older. It is then not surprising that gilts that reach puberty at older ages, due in our opinion to inherent differences in sexual maturation, would have a relatively lower lean growth rate than gilts that reach puberty at a younger age.

Stepwise linear regression was used to determine the most accurate equation to determine age at puberty in gilts. Weight at puberty and lifetime growth rate to puberty accounted for the largest proportion of total variance. Although weight explains a large proportion of the variance, it is not appropriate to assume that a specific weight achieved in this experiment was the "trigger" for the onset of puberty. Rather, weight at puberty is considered to be a consequence of the age at which a gilt reaches puberty on the basis that the threshold body weight, or some other body component necessary for puberty to occur, was likely exceeded in the gilts in both experiments. Thus, when gilts are growing at growth rates above the threshold for puberty attainment, the age at which they reach puberty will determine their weight at puberty. This conclusion is consistent with the suggestions of Beltranena et al. (1993) that it is not growth performance that determines the onset of sexual maturity, but innate variability in sexual development.

Litter origin was highly related to growth characteristics in both experiments, and related to age at puberty in experiment 1. Litter origin of gilts affected number of corpora lutea, follicles greater than 5 mm, ovarian and uterine weight, and LH and FSH responses to a GnRH challenge at 55 days of age in the study of Deligeorgis et al. (1985). Recently, Almeida et al. (2001) provided evidence that litter had significant effects on growth characteristics such as body weight, growth rate, backfat depth and reproductive characteristics such as estrous cycle length, estrus duration, and various endocrine parameters. Similarly, Le Cozler et al. (1999) found that birth litter had a significant effect on several factors including age, weight and backfat depth at first physiological estrus and at first breeding. Perhaps age at pubertal onset is regulated by a genetic clock and is invariant in its time course of development (Foster et al. 1994). Although, it is known that genetics play an important role in determining growth and reproductive

performance of pigs, it becomes important to understand just how much is genetic and how much we are able to control by management.

No relationship between leptin concentration and backfat depth was detected in this trial. This is in contrast to the results reported in the third chapter, and the data of Hamann and Matthaei (1996), who demonstrated that the amount of leptin secreted from adipocytes is significantly correlated to body mass index (BMI) and percent body fat in humans. Also, Robert et al. (1998) indicated that leptin expression could be associated with subcutaneous fat accumulation in pigs. No relationships were detected between age at puberty and insulin, IGF-1 or leptin concentration at 135 days of age in experiment 1. In experiment 2, no relationships were detected between age at puberty and insulin or leptin concentration; however a significant negative correlation was established between age at puberty and IGF-1 concentration at d 135. IGF-1 has been implicated as one of the signals that link the development of the somatrophic axis to the activation of the GnRH/LH releasing system during female puberty (Hiney et al., 1996). However, similar to phenotypic traits like weight and growth rate, it may be that the gilts used in this experiment were above the minimum threshold required and they acted as a permissive factor that allowed puberty to proceed.

In conclusion, results from our experiment demonstrate that a range of high lean growth rates do not affect the onset of sexual development in gilts. The next logical step would be to consider the consequences of high growth and lean growth on the subsequent performance of gilts in the breeding herd. Although, Rozeboom et al. (1996) reported that gilt age or body composition at first breeding had no effect on sow productivity and longevity over three parities, the consequences of increased mature body weight (increased maintenance costs, housing limitation, and welfare concerns) must be considered.

Table 4.1. Ingredient and nutrient composition of diets as formulated (form) and as fed (fed). Diets were formulated to maximize lean potential (LP), to produce lower lean growth (LL), and to reduce lean growth (RL)[Y].

	LP		LL		RL	
Treatment	Form	Fed	Form	Fed	Form	Fed
Ingredient Composition						
Wheat, %	44.5	-	19.2	-	-	-
Barley, %	4	-	43.8	-	13.8	-
Corn, %	8.5	-	-	-	-	-
Peas, field, clean, %	11.2	-	-	-	5.4	-
Canola Meal, %	15	-	15.0	-	-	-
Soy Meal, %	7.2	-	-	-	-	-
Wheat millrun (10%CF), %	-	-	17.5	-	29.0	-
Oats, ground, %	-	-	-	-	45.0	-
Beet pulp, %	-	-	-	-	1.95	-
Lime, 18- mesh %	1.3	-	1.6	-	1.8	-
Monodical Phos (21%), %	1.5	-	1.4	-	1.4	-
Grow-Finish Mix[1], %	.25	-	.25	-	.25	-
AP avfat (spray)[6], %	3.1	-	-	-	-	-
AP avfat (mix)[6], %	2.6	-	.5	-	.5	-
DL methionine, %	.02	-	-	-	-	-
L-lysine, %	.27	-	.016	-	.011	-
Salt, %	.34	-	.35	-	.36	-
Magox [4], %	.04	-	-	-	.14	-
Chol CL 70%[5], %	.065	-	.065	-	.064	-
L-Threonine, %	.085	-	.01	-	.045	-
Tylan 40[2], %	.025	-	.025	-	-	-
Dyna K (Bagged)[3], %	-	-	.13	-	.15	-
Nutrient Composition						
ME, Mcal/kg	3.282		2.869		2.550	
Crude Protein, %	18.98	18.7	16.10	15.2	13.0	13.4
Crude Fat, %	7.39	-	3.43	-	4.10	-
Crude Fibre, %	4.29	-	7.79	-	9.82	-
Calcium, %	.93	-	1.01	-	1.04	-
Phosphorus, total, %	.8	-	.87	-	.80	-
Phosphorus, available, %	.49	-	.45	-	.45	-
Lysine, %	1.113	1.18	.819	.84	.647	.64

[Y] All diets were commercially formulated by Vic Pouteaux, Landmark Feeds, Strathmore, Alberta, Canada.

[1] Landmark Feeds Grow-Finish Vitamin-trace Mineral Premix

[2] Tylosin Phosphate 88 g /kg

[3] Potassium Chloride (50 % potassium), IMC Agri

[4] Magnesium oxide 58 %, Graymont Western

[5] Choline chloride 70 %, Chinook

[6] Alberta Processors Animal / Vegetable Fat

Table 4.2 Summary statistics for performance measurements in experiment 1, for diets formulated to maximize lean potential (LP) and to produce lower lean growth (LL).

Experiment #1				P-value				
	LP	LL	Pool SEM	Trt	Barn	Time	Trt *time	Litter
Pen Feed Intake (kg/d)								
Growth	2.5	2.6	.02	.12	-	.0001	-	
Stimulation	2.7	3.1	.03	.0012	-	.006	-	
Overall	2.6	2.9	.02	.0006	-	.0001	.0351	
Pen Lysine Intake (g/d)								
Growth	28.0	21.5	.20	.0001	-	.0001	-	
Stimulation	30.7	25.4	.33	.0001	-	.0027	-	
Overall	29.4	23.6	.20	.0001	-	.0001	-	
Pen Metabolizable Energy Intake (Mcal/d)								
Growth	8.1	7.5	.06	.017	-	.0001	-	
Stimulation	8.9	8.9	.10	.88	-	.0038	-	
Overall	8.5	8.3	.06	.004	-	.0001	-	
Growth Rate (g/d)								
Growth	946	863	4.6	.0004	.0003	.0001	-	.0001
Stimulation	906	859	2.9	.018	-	-	.0001	.0001
Overall	914	869	2.7	.08	.0136	.0001	.0401	.0001
Lean Growth Rate (g/d)								
Growth	426	365	3.5	.0004	-	.0001	-	.0001
Stimulation	370	340	1.7	.0232	-	.0001	-	.0001
Overall	391	354	1.8	.0234	-	.0001	.0125	.0001
Fat Growth Rate (g/d)								
Growth	150	107	5.6	.09	-	.0001	-	.0001
Stimulation	275	227	2.5	.0063	-	.0001	-	.0001
Overall	229	182	2.5	.01	-	.0001	-	.0001
Gain : Feed								
Growth	.36	.33	.007	.059	-	.0001	-	
Stimulation	.33	.30	.0564	.012	-	.002	.0277	
Overall	.35	.31	.0011	.007	-	.0001	.0412	

Growth – Period from start of trial until 135 days of age
Stimulation – Period from 135 days of age until puberty
Overall – Period from start of trial until puberty

Table 4.3 Summary statistics for performance measurements in experiment 2, for diets formulated to maximize lean potential (LP) and to reduce lean growth (RL).

Experiment #2				P-value				
	LP	RL	Pool SEM	Trt	Barn	Time	Trt *time	Litter
Feed Intake (kg/d)								
Growth	2.1	2.5	.02	.0001		.0001	.0041	.0001
Stimulation	2.5	3.0	.03	.0001		.0001	.0007	.0001
Overall	2.3	2.8	02	.0001		.0001	.0001	.0001
Lysine Intake (g/d)								
Growth	24.0	16.6	.17	.0001		.0001	-	.0001
Stimulation	28.0	19.8	.30	.0001		.0001	.0147	.0001
Overall	26.3	18.5	.17	.0001		.0001	.0047	.0001
Metabolizable Energy Intake (Mcal/d)								
Growth	7.0	6.5	.05	.0001		.0001	-	.0001
Stimulation	8.1	7.7	.10	.0578		.0001	.0045	.0001
Overall	7.6	7.2	.05	.0003		.0001	.0004	.0001
Growth Rate (g/d)								
Growth	1038	969	10.6	.0001		-	-	.0001
Stimulation	969	968	7.9	.93		.0005	-	.0001
Overall	997	963	8.6	.0018		.0049	.0006	.0001
Lean Growth Rate (g/d)								
Growth	396	373	7.2	.024		.0354	-	.0001
Stimulation	380	374	4.6	.28		.0153	-	.0001
Overall	388	373	4.6	.0095		.0151	-	.0001
Fat Growth Rate (g/d)								
Growth	273	238	5.6	.0026		.0001	-	.0001
Stimulation	355	315	4.7	.0001		.0001	-	.0001
Overall	326	290	3.7	.0001		.0001	-	.0001
Gain : Feed								
Growth	.49	.39	.006	.0001		.0001	-	.0071
Stimulation	.45	.30	.030	.0474		.0333	-	-
Overall	.47	.34	.0001	.014		.0001	-	-

Growth – Period from start of trial until 135 days of age
Stimulation – Period from 135 days of age until puberty
Overall – Period from start of trial until puberty

Table 4.4 Summary statistics for weight, age, backfat and loin depth at the beginning of the trial (start), at the time of stimulation (stimulation) and at the onset of puberty (puberty) in experiment 1, for diets formulated to maximize lean potential (LP) and to produce lower lean growth (LL).

Experiment #1				P-value			
	LP	LL	Pool SEM	Trt	barn	Trt* barn	Litter
Start							
Weight (kg)	54.2	53.8	.35	.23	-	-	.0001
Age (d)	96.1	96.6	.28	.28	-	-	-
Backfat depth (mm)	11.8	12.1	.36	.70	-	-	-
Loin depth (mm)	34.7	34.2	.25	.40	-	-	-
Stimulation							
Weight (kg)	88.3	85.5	.60	.0096	.006	-	.0001
Age (d)	134.2	134.6	.28	.38	.0005	-	-
Backfat depth (mm)	13.4	12.4	.14	.0075	-	-	.0001
Loin depth (mm)	48.5	45.8	.32	.0001	-	-	.0001
Puberty							
Weight (kg)	109.3	106.2	1.21	.14	-	-	.0153
Age (d)	157.3	157.6	1.21	.75	-	-	.069
Backfat depth (mm)	16.9	14.3	.32	.0008	-	-	.0005
Loin depth (mm)	51.2	50.6	.35	.41	-	-	-

Table 4.5 Summary statistics for weight, age, backfat and loin depth at the beginning of the trial (start), at the time of stimulation (stimulation) and at the onset of puberty (puberty) in experiment 2, for diets formulated to maximize lean potential (LP) and to reduce lean growth (RL).

Experiment #2				P-value	
	LP	**RL**	Pool SEM	Trt	Litter
Start					
Weight (kg)	50.0	51.2	.69	.29	.0001
Age (d)	88.0	87.3	.55	.55	-
Backfat depth (mm)	9.7	10.0	.28	.64	.86
Loin depth (mm)	35.7	35.5	.78	.92	.69
Stimulation					
Weight (kg)	97.6	97.9	1.21	.87	.0001
Age (d)	135.0	134.3	.55	.56	-
Backfat depth (mm)	17.2	15.9	.52	.14	.083
Loin depth (mm)	49.5	50.6	.49	.20	.003
Puberty					
Weight (kg)	125.0	124.9	2.79	.98	.57
Age (d)	166.8	166.3	2.96	.94	.63
Backfat depth (mm)	20.6	21.1	.87	.89	.87
Loin depth (mm)	55.6	57.7	.98	.30	.56

Table 4.6 Simple correlations between age, body weight, and growth rate at the onset of puberty.

		Age (d)	Weight (kg)	Growth Rate (kg/d)
Experiment 1				
Age (d)	r	-	.80	.23
	p		.0001	.0054
Weight (kg)	r	.80	-	.77
	p	.0001		.0001
Growth Rate (kg/d)	r	.23	.77	-
	p	.0054	.0001	
Experiment 2				
Age (d)	r	-	.79	.05
	p		.0001	.68
Weight (kg)	r	.79	-	.55
	p	.0001		.0001
Growth Rate (kg/d)	r	.05	.66	-
	p	.68	.0001	

r = correlation coefficient
p = significance

Table 4.7 Percentage of total variance utilizing various body component measurements to predict age at puberty in gilts in experiment 1 from multiple-regression equations using a stepwise procedure.

Equation	Intercept	WT P	GR B-P	BF P	GR ST	LD ST	GR ST-P	R^2
1	75.7	.76						.64
2	156.9	1.44	-226.7					.994
3	156.5	1.46	-226.9	-.08				.995
4	155.3	1.46	-229.7	-.09	5.17			.995
5	155.6	1.46	-230.1	-.09	6.32	-.02		.995
6	155.9	1.48	-242.6	-.10	12.4	-.03	3.6	.995

WT P – body weight at first pubertal estrus (kg)
GR P – growth rate from birth to puberty (kg/d)
BF P- backfat depth at puberty (mm)
GR ST- growth rate at the start of trial (kg/d)
LD ST– loin depth at start of trial (mm)
GR ST-P – growth rate from start of trial to puberty (kg/d)

Table 4.8 Percentage of total variance utilizing various body component measurements to predict age at puberty in gilts in experiment 2 from multiple-regression equations using a stepwise procedure.

Equation	Intercept	WT SH	GR P	GR ST-S	LN P	LT	GR B-S	R^2
1	66.6	.8						.56
2	166.7	1.37	-230.2					.996
3	166.5	1.37	-212.5	-8.2				.996
4	168.0	1.33	-208.3	-7.7	-.009			.996
5	184.8	1.32	-206.0	-8.7	-.010	-.002		.996
6	184.8	1.31	-212.2	-10.8	-.009	-.002	7.8	.997

WT P – body weight at first pubertal estrus (kg)
GR P – growth rate from birth to puberty (kg/d)
GR ST-S– growth rate from start of trial until initiation of puberty stimulation (kg/d)
LN P – lean growth rate at puberty (kg/d)
LT – litter from which gilt originated
GR B-S – growth rate from birth to initiation of puberty stimulation (kg/d)

Table 4.9 Total feed offered, total feed and estimated energy consumed, and IGF-1, leptin and insulin concentration, for diets formulated to maximize lean potential (LP) and to produce lower lean growth (LL)[1].

	LP (n=33)	LL (n=30)	Pool SEM	p-value
Total feed offered (kg)	2.2	2.7	.04	.0001
Total feed consumed (kg)	1.4	1.7	.06	.0039
Total energy consumed (Mcal)	4.5	4.6	.2	-
Leptin (ng/mL)	2.1	1.9	.08	-
IGF-1 (ng/mL)	178.1	179.7	3.6	-
Insulin (ng/mL)	2.5	4.8	.2	.0001

[1] 75 min after total feed was offered, the remaining feed was removed and weighed to determine feed consumed and a single blood sample was taken to determine leptin, IGF-1 and insulin concentration.

Table 4.10 Total feed offered, total feed and estimated energy consumed, and IGF-1, leptin and insulin concentration, for diets formulated to maximize lean potential (LP) and to reduce lean growth (RL)[1].

	LP (n=20)	RL (n=21)	Pool SEM	p-value
Total Feed Offered (kg)	2.5	3.3	.04	.0001
Total Feed Consumed (kg)	1.2	1.1	.07	-
Total Energy Consumed (Mcal)	3.8	2.9	.2	.0368
Leptin (ng/mL)	2.1	1.8	.13	-
IGF-1 (ng/mL)	193.4	174.8	5.4	-
Insulin (ng/mL)	1.2	1.7	.23	-

[1] 75 min after total feed was offered, the remaining feed was removed and weighed to determine feed consumed and a single blood sample was taken to determine leptin, IGF-1 and insulin concentration.

Table 4.11 Summary of reported lean growth rates

Source	Period lean growth measured	Feed intake	Average lean growth rates achieved
Cameron and Curran (1995)	Birth – 82.6 kg	*Ad libitum*	491; 505; 520 g/d
Rozeboom et al., (1995)	22 – 104.9 kg	*Ad libitum*	429 g /d
Schinckel et al., (1996)	20 – 120 kg	?	260; 285; 310; 335; 360 g/d
NRC (1998)	20 – 120 kg	?	300; 325; 350 g/d
Vic Pouteaux (personal communication)	30 – 110 kg	?	458 g /d

Table 4.12 Summary of differences between experiment #1 and #2

	Experiment #1	**Experiment #2**
Birth weight	1.35	1.41
Weaning weight / age	5.65 kg / 21.1 d	7.3 kg / 22.8 d
Time of experiment	March - August	November – March
Start weight / age	54 kg / 96 d	50 kg / 88 d

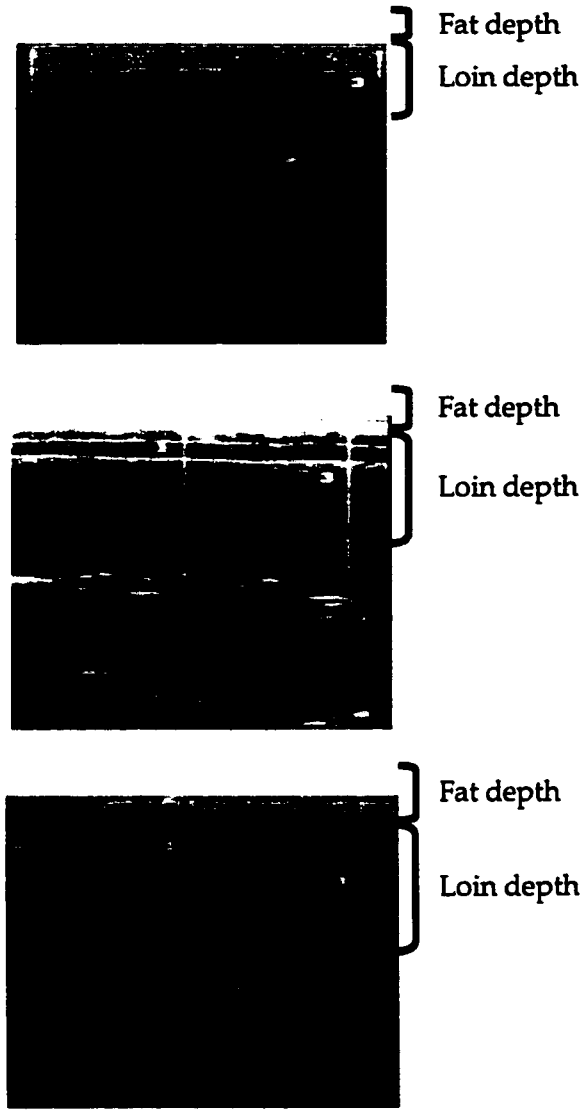


Figure 4.1 Series of sequential ultrasound pictures of one gilt over the experiment

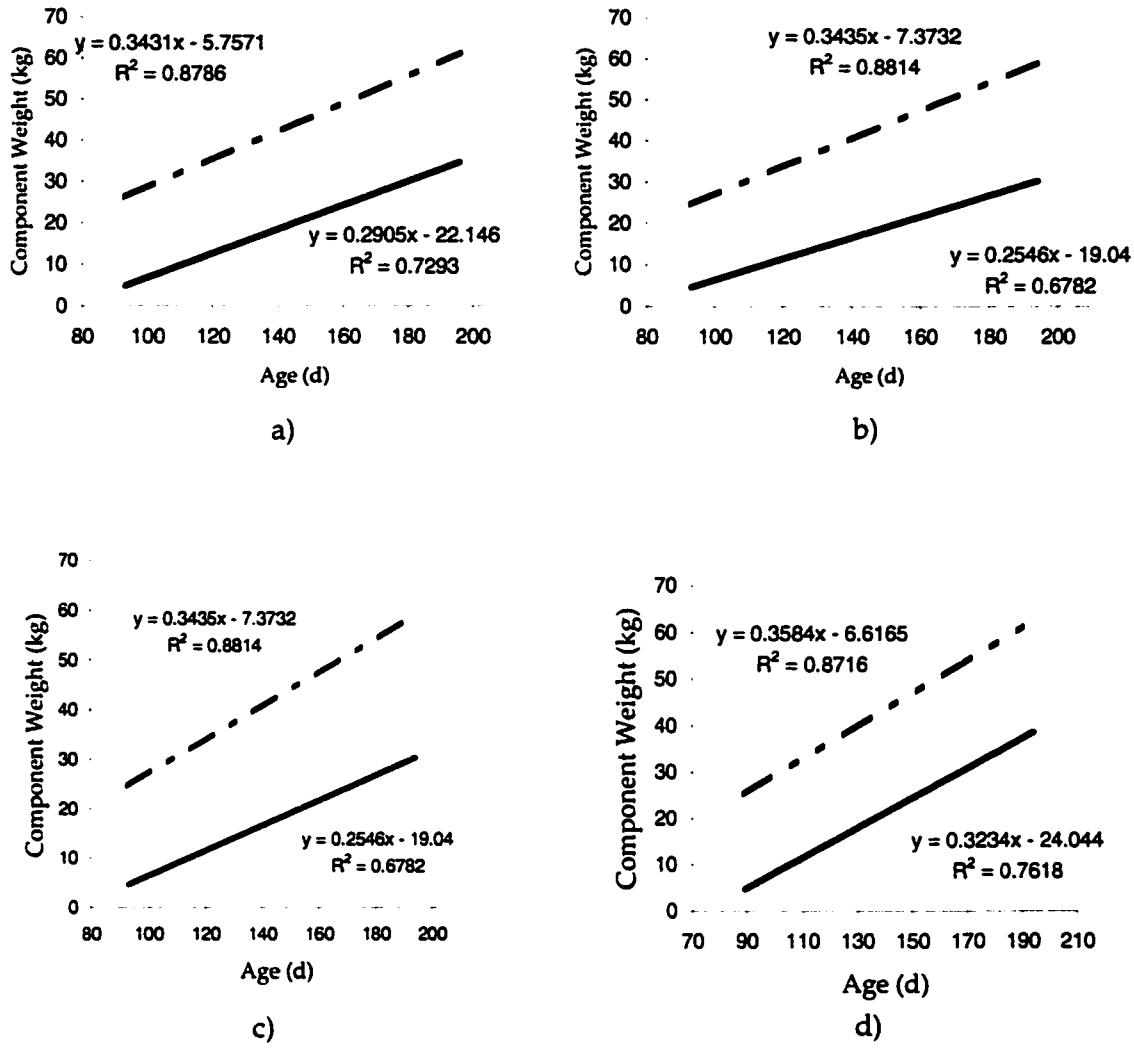


Figure 4.2 Relationship between age and component weight. Dashed line represents fat-free lean weight, solid line represents fat weight. a) Experiment 1, LP; b) Experiment 2, LL; c) Experiment 2, LP; d) Experiment 2, RL

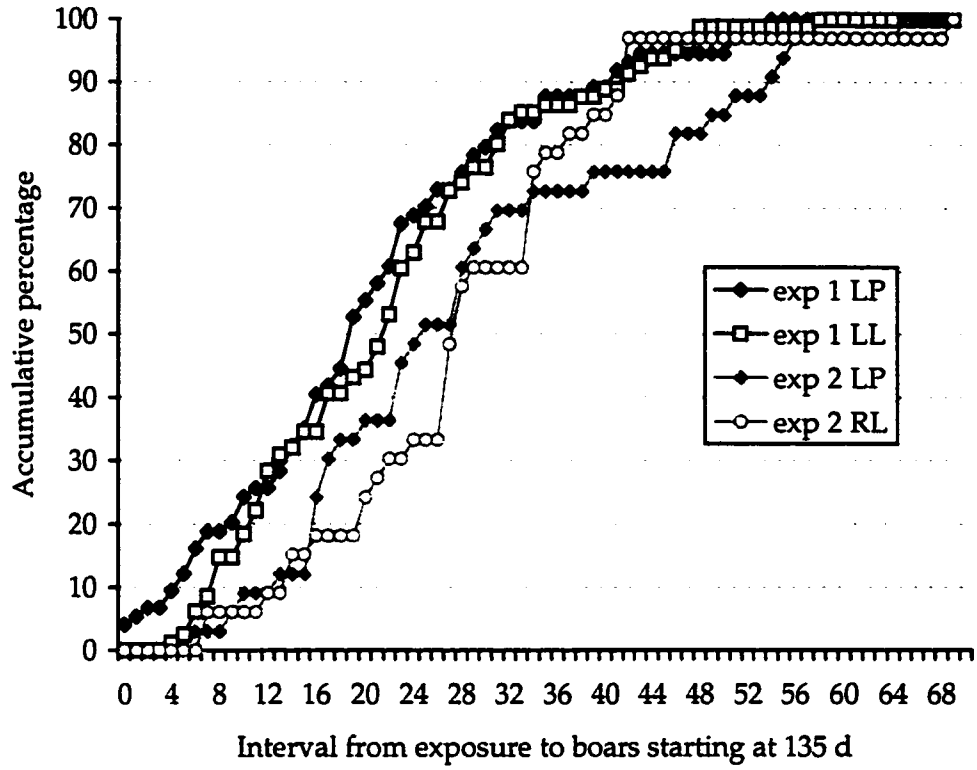


Figure 4.3 Accumulative percentage of gilts reaching puberty by interval from exposure to pubertal estrus in experiment 1 and 2.

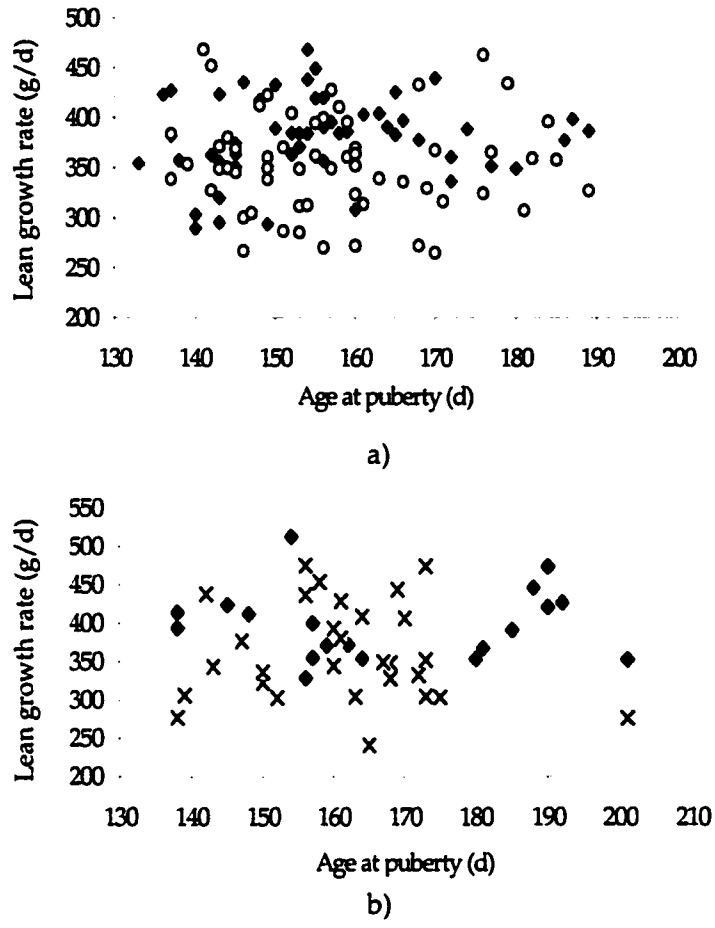


Figure 4.4 Relationship between average lean growth rate during the *growth* period (~ 50 kg to 135 d) and age at puberty. a) Experiment 1, closed diamonds represent LP, open circles represent LL; b) Experiment 2, closed diamonds represent LP, and crosses represent RL.

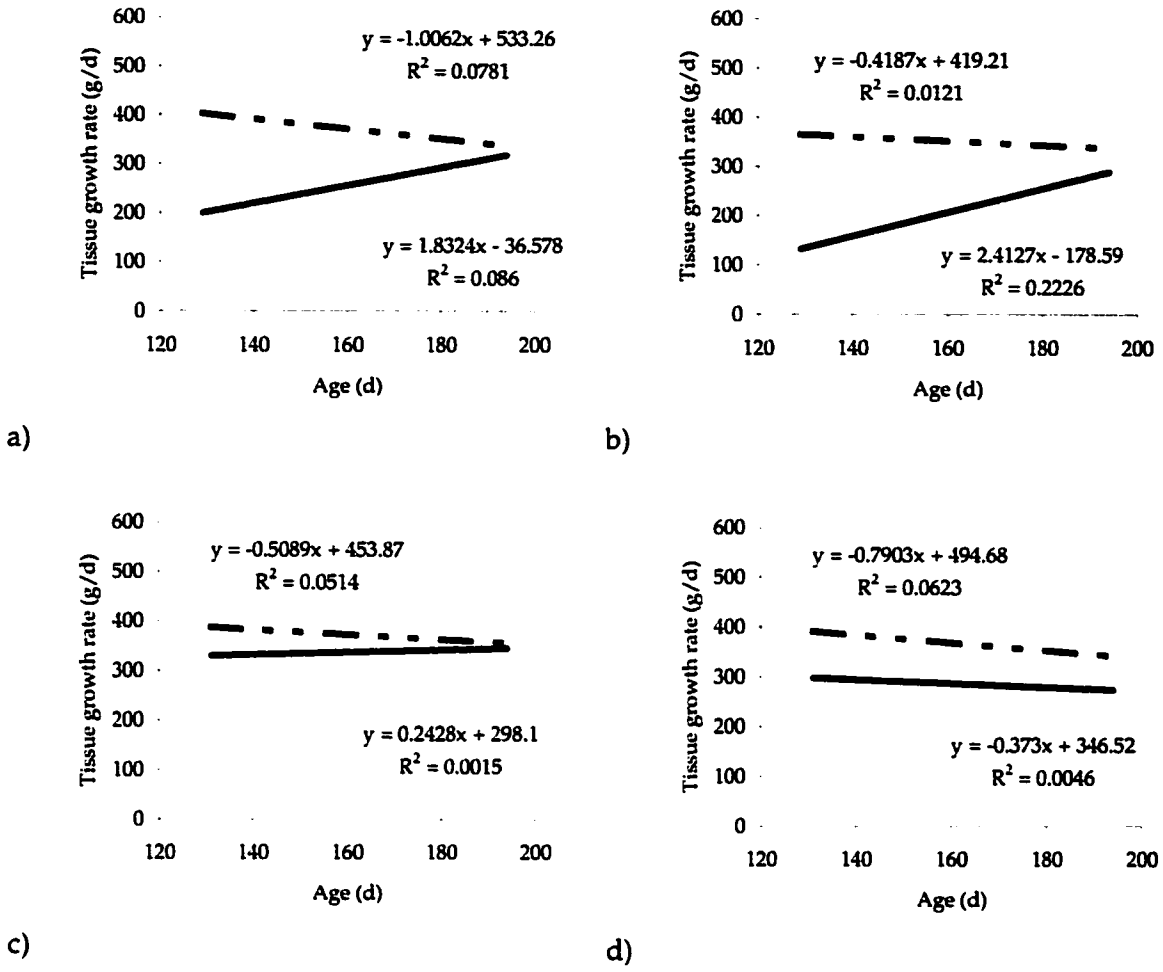


Figure 4.5 Relationship between tissue growth rate and age during the *stimulation* period. Lean tissue growth and fat tissue growth rates are represented by dashed and solid line, respectively. a) experiment 1, LP; b) experiment 1, LL; c) experiment 2, LP; d) experiment 2, RP.

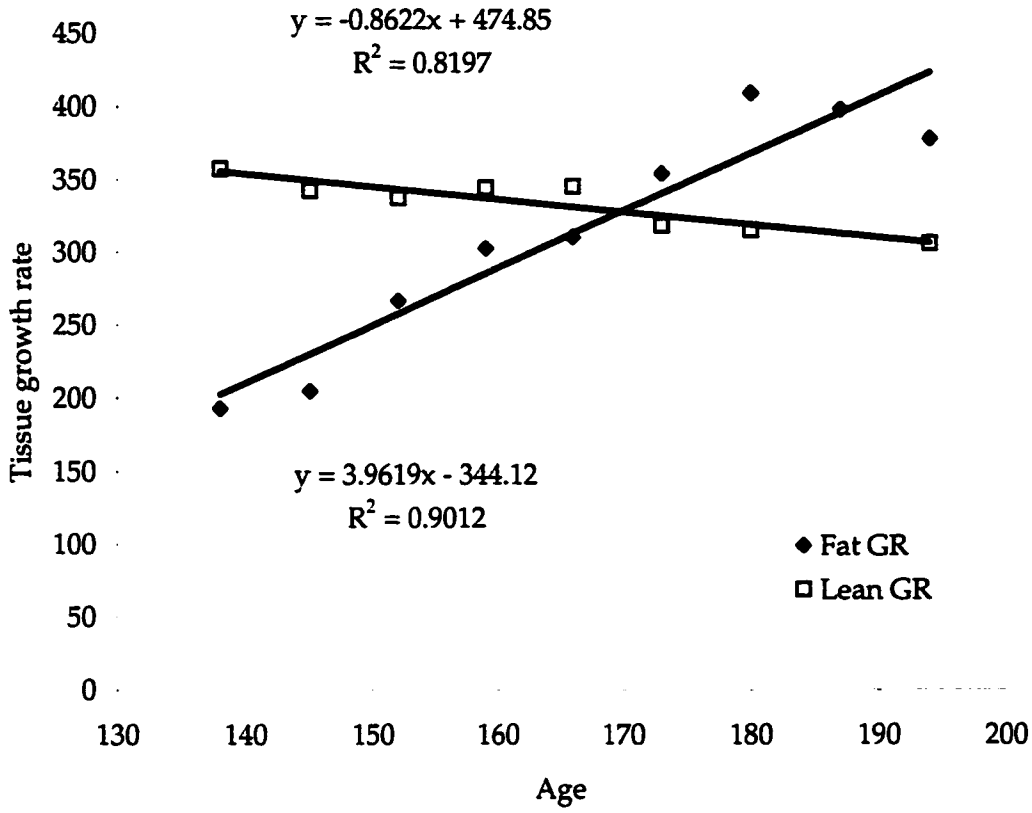
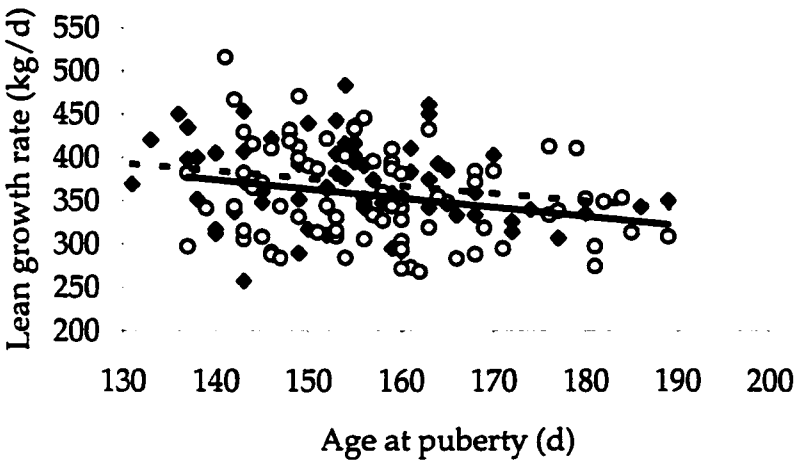
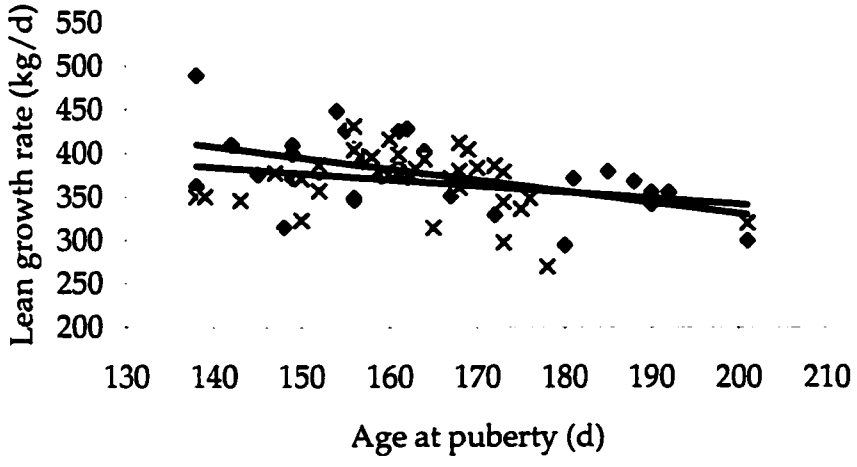


Figure 4.6 Relationship between tissue growth rate and age during the *stimulation* period in gilt #381 (age at puberty = 194 d). Lean tissue growth rate and fat tissue growth rates are represented by dashed and solid line, respectively.

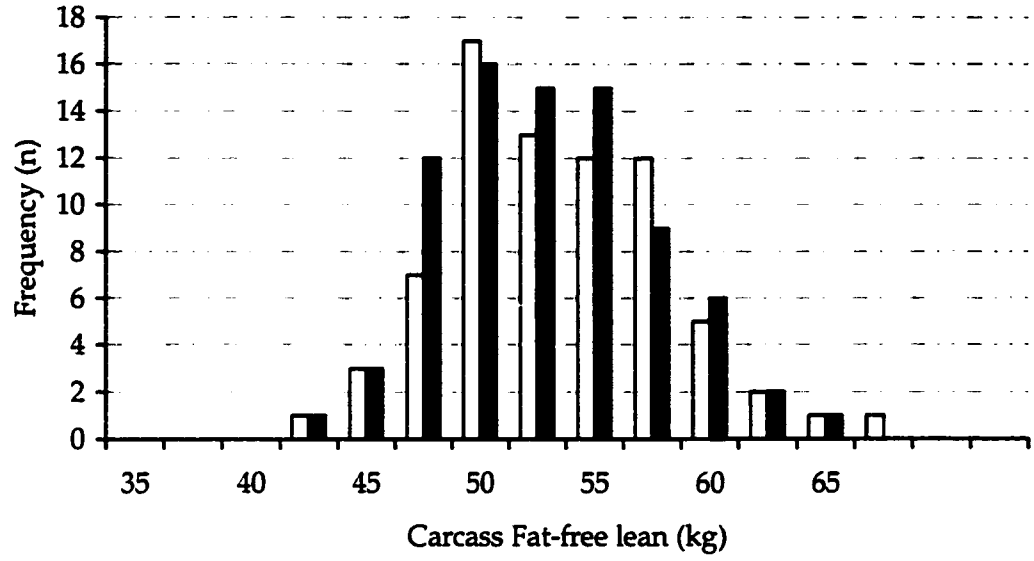


a)

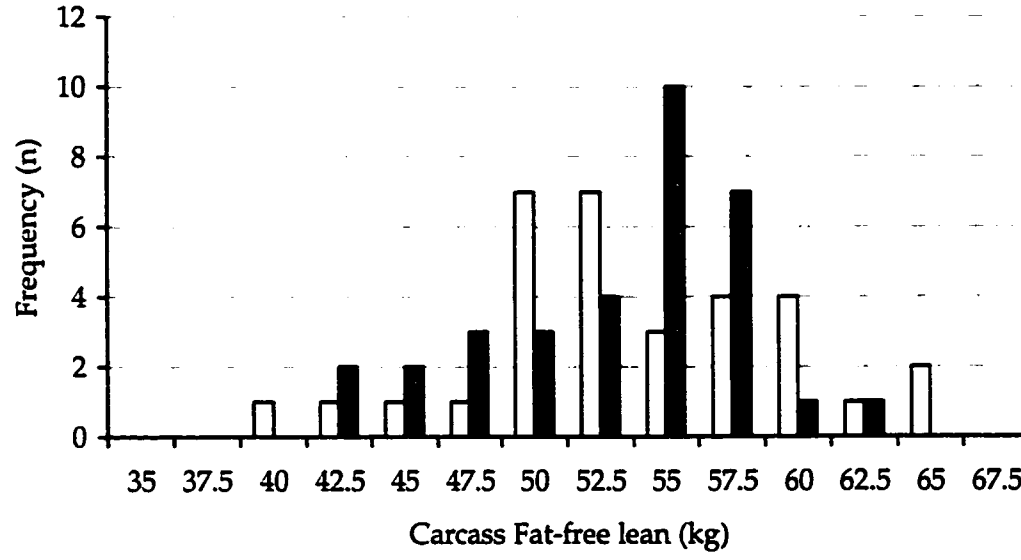


b)

Figure 4.7 Relationship between lean growth rate from start (~ 50 kg) until age at puberty a) experiment 1: closed diamonds represent LP, [Age at puberty = 176.3 - .059 (lean growth rate), p = .059, R^2=-.23]; open circles represent LL, [Age at puberty = 177.3 - .055 (lean growth rate), p = .003, R^2 = -.25]. b) Experiment 2: closed diamonds represent LP, [Age at puberty = 239.3 - .19 (lean growth rate), p = .003, R^2 = -.48]; crosses represent RL, [Age at puberty = 239.0 - .19 (lean growth rate), p = .01, R^2 = -.53].



a)



b)

Figure 4.8 Distribution of fat-free carcass lean weight at puberty a) experiment 1; white bars LP, black bars LL; b) experiment 2, white bars LP, gray bars RL.

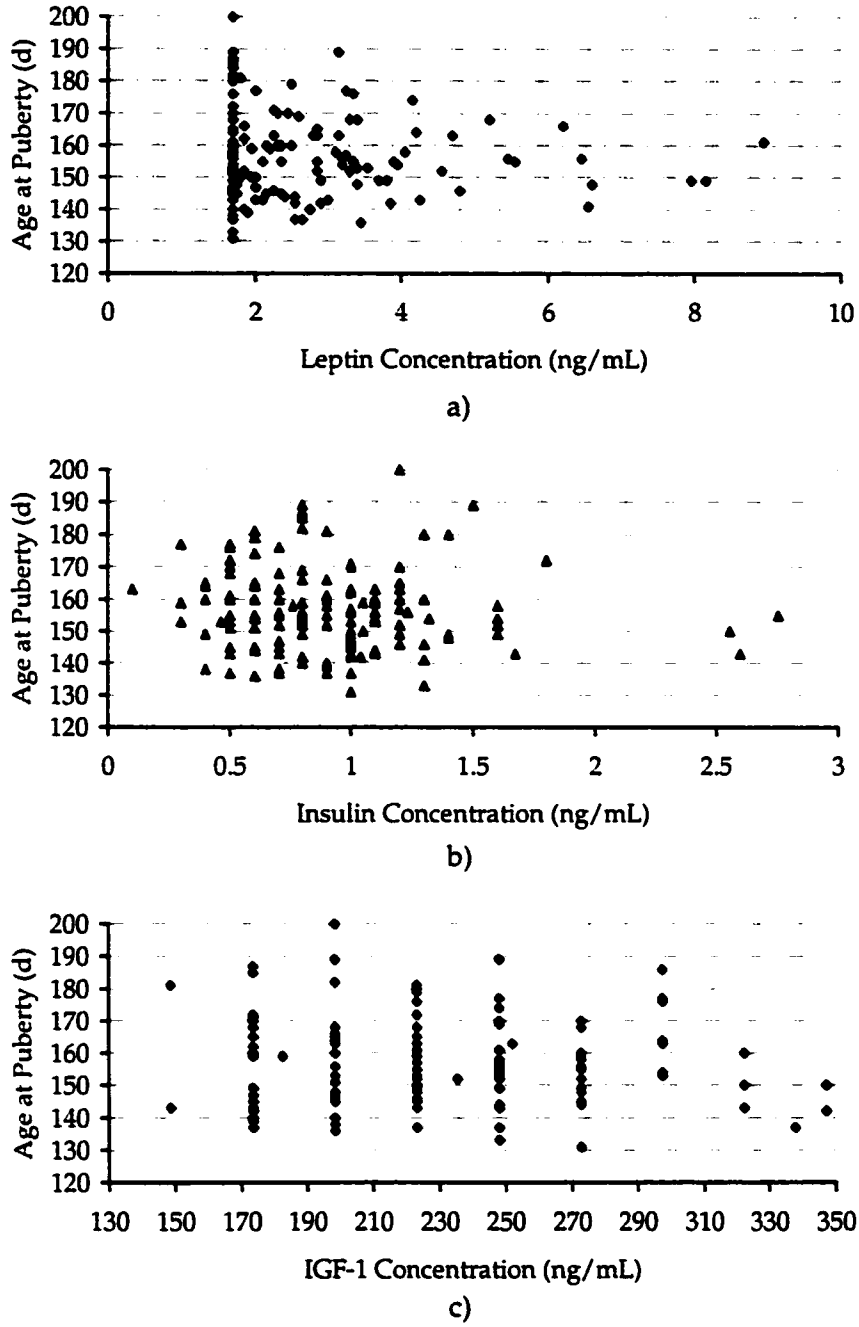
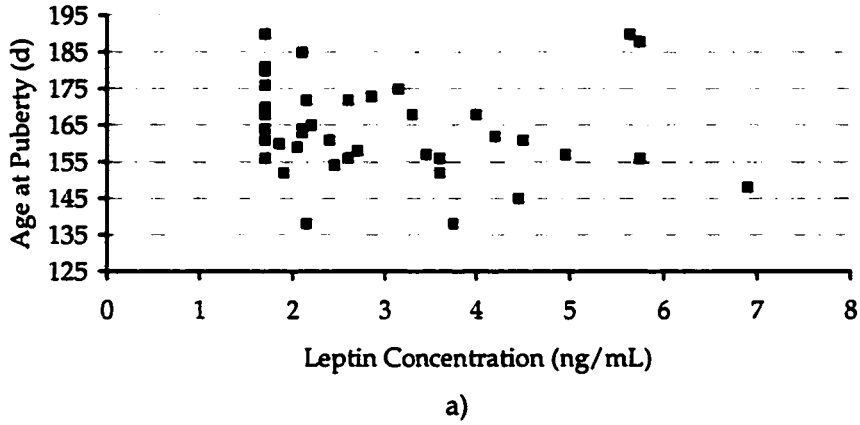
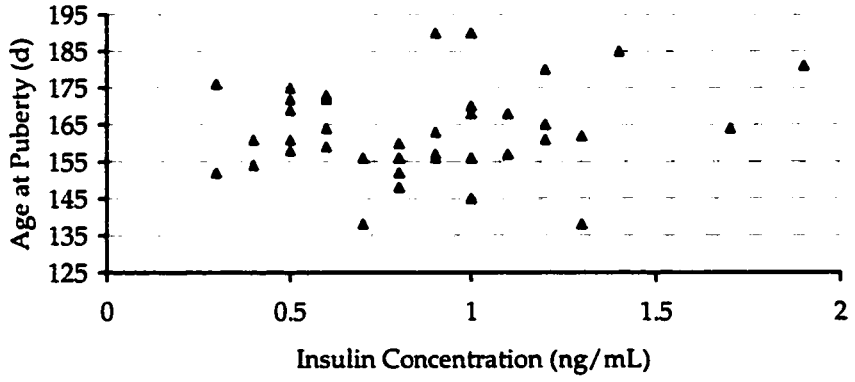


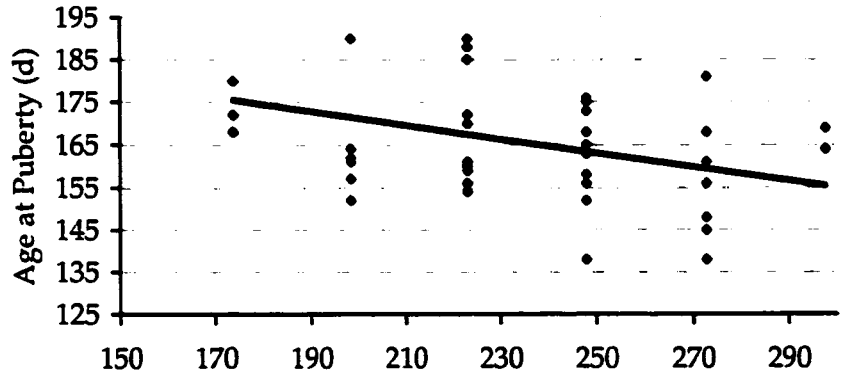
Figure 4.9 Lack of a relationship between age at puberty and a) leptin concentration at d 135 (age at puberty = 158.4 - .66(leptin concentration d135), $R^2 = 0$, p = .46); b) insulin concentration at d135 (age at puberty = 159.8 – 3.28(insulin concentration d135), $R^2 = .001$, p =.23); and c) IGF-1 concentration at d135 (age at puberty = 165.5 - .04(IGF-1 concentration d135), $R^2 = .02$, p = .10).



a)



b)



c)

Figure 4.10 Relationship between age at puberty and a) leptin concentration at d 135 (age at puberty = 171.1 – 1.7(leptin concentration d135), R^2 = .03, p = .31); b) insulin concentration at d135 (age at puberty = 165.6 – .26(insulin concentration d135), R^2 = 0, p =.97); and c) IGF-1 concentration at d135 (age at puberty = 203.6 - .16(IGF-1 concentration d135), R^2 = .14, p = .01).

4.5 References

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Chapter 5

5.1 General Discussion

In a typical swine herd, 40% of the females that make up the breeding herd are gilts, and as many as 54.5 % of these gilts will be culled without ever farrowing a litter (see review, Levis, 2000). It is therefore extremely important to a producer that a constant supply of cyclic gilts is readily available to use in the herd. As a result, the onset of sexual maturation in gilts is crucial to the sustainability of a swine operation. It is therefore necessary to gain an understanding of the onset of puberty in gilts. Attainment of sexual maturity is the result of a cumulative series of physiological events that are regulated and influenced by a number of factors. Extensive research has been undertaken to detect and establish relationships regarding onset of puberty.

The numerous factors that influence the onset of puberty can be broadly grouped into two categories: genetically determined factors and environmental factors. In this thesis, factors falling into both categories were studied. It has been shown that the environment in which the gilt is housed (Dyck, 1989), method of boar exposure (van Lunen and Aherne, 1987; Zimmerman et al., 1998), and the frequency and duration of exposure (Paterson et al., 1989) play critical roles in the onset of puberty in gilts. The first experiment (chapter 3) examined the effect of method of pubertal stimulation on puberty attainment in gilts. Confirming previous studies, direct contact with a boar was a superior method for stimulating puberty in gilts compared with fenceline contact. Our results demonstrate that taking a group of gilt's to a boar pen and taking a boar to a group of gilts home pen, were equally effective in inducing puberty. Because the primary factor to achieve maximal pubertal stimulation in gilts is direct physical contact, the decision of what method of direct boar contact to use will be the manager's choice. Which method to use will depend largely on ease of handling, barn layout, space, and labor constraints. It may be easier to move a single animal (boar) rather than a group of animals (gilts), thereby making the boar to gilt method of puberty stimulation easier. Secondly, by moving only the boar, one would not have to deal with the difficulty of moving a gilt in estrus. However, there are some downsides to this method. It is critical

that the boar is not permitted to consume food from the gilt's feeders, as he will be more interested in eating that working with the gilts. Safety also becomes an issue, because it may be more of a risk to move a boar than gilts. It is necessary to have adequate available pens empty in which to complete puberty stimulation. As the study in chapter 3 demonstrated, the most effective method to stimulate puberty is through direct physical contact of the boar and gilt.

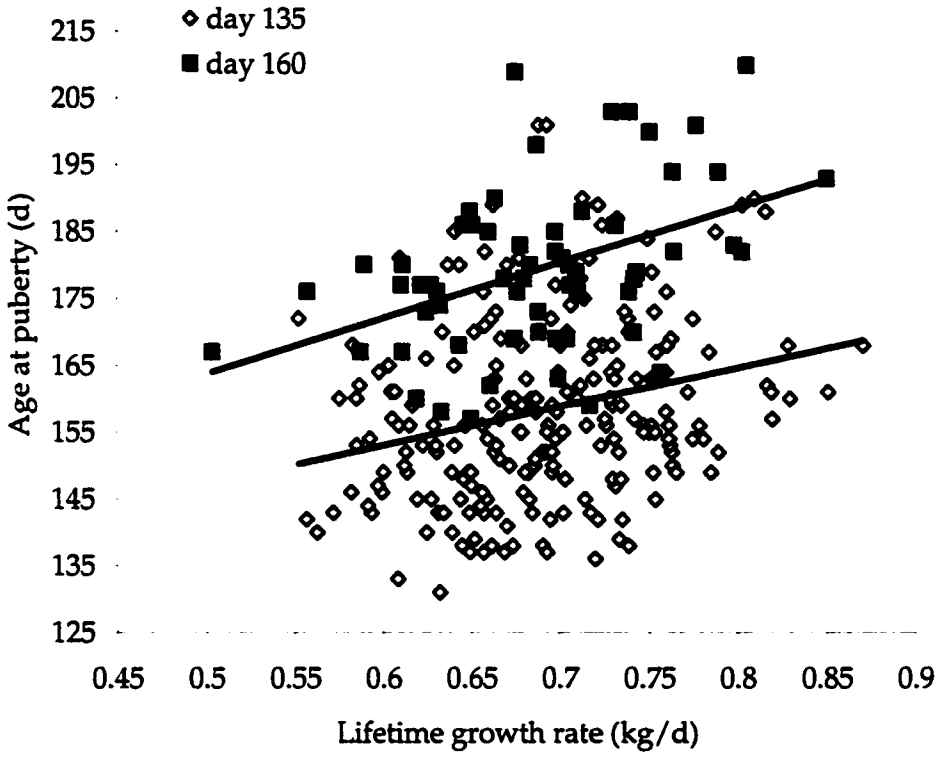

Figure 5.1 Effect of puberty stimulation in the gilt commencing either at 160 or 135 d of age. Closed squares represent pubertal stimulation at 160; Age at puberty (d) = 83.2 (lifetime growth rate) + 122.13, R^2 = .19, p =0.0003). Open diamonds represent pubertal stimulation at 135 d; Age at puberty (d) = 58.3 (lifetime growth rate) + 118.0, R^2 = .07, p =0.0001).

The age at which the gilts were first exposed to the boar differed between experiments. Gilts in the first (chapter 3) and second (chapter 4) experiment were first exposed to a mature boar at 160 and 135 days of age, respectively. If achievement of a minimum threshold level of a specific component of growth is necessary before puberty

can occur, it is evident from both experiments that all gilts had achieved this minimum threshold. As described by Kirkwood and Aherne (1985) achievement of this threshold value is not sufficient itself to trigger the onset of puberty and does not preclude other factors such as nutrition and management from having stimulatory or inhibitory effects. It is evident from Figure 5.1 that age at first exposure to the boar had significant effects on the age at puberty in the gilts. It is apparent that boar stimulation is a sufficient trigger to stimulate puberty onset in gilts.

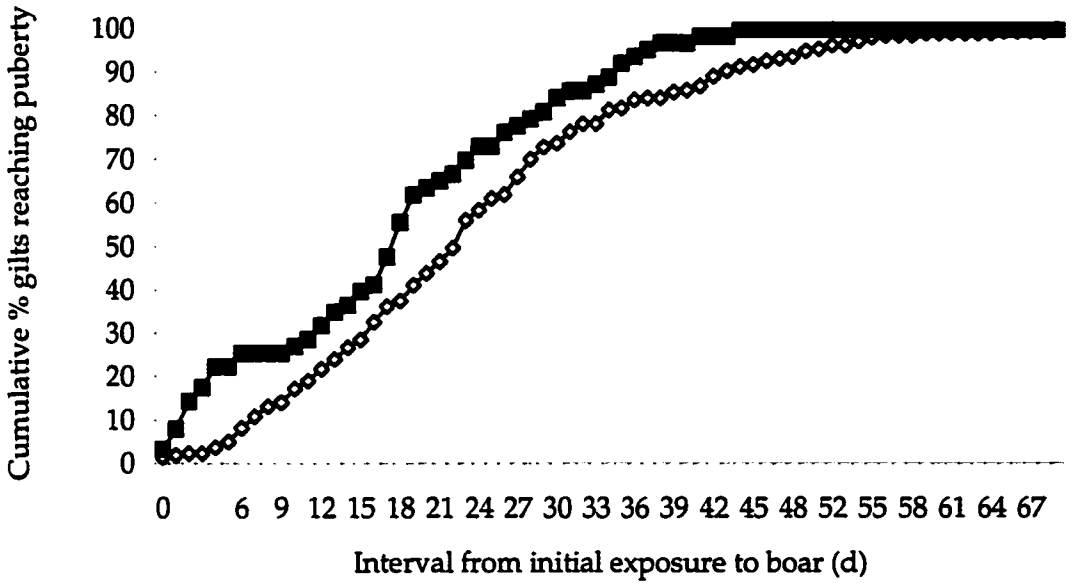

Figure 5.2 Accumulative percentages of gilts reaching puberty by interval from initial boar exposure until puberty. Closed squares and open diamonds represent initiation of puberty stimulation at 160 and 135 d, respectively.

There is an abundance of information in the literature regarding the effects of chronological age at puberty and subsequent reproductive performance of gilts. Withholding initial boar exposure until 160 days resulted in differences in the distribution of gilts coming into heat. Initial stimulation at 160 days resulted in a skewed distribution whereas stimulation at 135 resulted in a normal distribution. When the stimuli are applied at 160 days, there is an increase in the number of gilts that reach estrus immediately following boar introduction (Foxcroft and Aherne, 2001). At each time point along the distribution curve, a higher percentage of gilts were pubertal when

stimulated at 160 days compared with 135 days (Figure 5.2). Depending on the manager's requirements for gilts, the distribution of gilts reaching puberty will differ. If a large proportion of gilts are required to reach a synchronous puberty, commencing boar exposure at an older age is desirable (Levis, 2000). However, stimulating gilts at an earlier age has several benefits. Stimulating gilts at a young age enables the producer to identify gilts that are most sexually mature (Foxcroft and Aherne, 2001). Stimulating gilts early would permit a producer to cull gilts that are not cycling, and as a result reduce the number of non-productive days a gilt adds to the herd. A producer is able to manage gilts so that at breeding, gilts have achieved a target weight and body condition. A third benefit of early stimulation would allow a producer more to synchronize estrus in gilts and thus organize breeding groups. Finally, early stimulation of gilts would permit producers to take advantage of the increased productivity of being bred at a high estrus. This is however, a balance that must be achieved to maximize productivity. Although, breeding on a higher estrus is beneficial, the gilt accumulates non-productive days.

There is no evidence that early puberty has any negative impacts on gilt performance, and in fact, may have positive effects. Rozeboom et al. (1996) reported that neither age nor weight at puberty affected gilt performance over four parities. Sterning et al. (1998) reported that gilts expressing puberty at younger age are more likely to show a standing reflex and to ovulate within 10 days of weaning than gilts expressing puberty at older ages.

It is well established that estrus number at first breeding has an effect on number of piglets born live (Levis, 2000). MacPherson et al. (1977) reported an increase of 2.6 piglets per litter when gilts were bred on their 1st estrus compared to the 3rd. Although not significant, Young and King (1981) demonstrated that gilts mated on their third estrus had more pigs born alive than gilts bred on their first estrus. Stimulating gilts at a younger age, thus resulting in a younger age at puberty, gives the producer the ability to take advantage of the beneficiary effects of breeding at a later estrous. If gilts reach puberty at a younger age, they can be managed through a number of estrus cycles to

achieve a target breeding weight and backfat. Aherne et al. (1999) suggested that, at second estrus, gilts should possess around 125 kg body weight, and 15 mm backfat.

The current trend in the pork industry places emphasis on lean tissue growth rate. However, it is unclear whether an unfavorable relationship exists between production and reproduction traits. In chapter 4, the main hypothesis tested was that high lean growth rates achieved through nutritional treatments of diets fed under commercial conditions would affect age at puberty in gilts. Although the different diets utilized resulted in differences in growth characteristics, no impact on pubertal onset was established. We, therefore, again conclude that the growth rates and composition of growth established, were not limiting factors for sexual maturation and onset of puberty in response to boar stimulation. The positive linear relationship between age at puberty and lifetime growth rate must be addressed. Although the model accounts for little of the variation in age at puberty, it is still a significant fit. However, if a minimum threshold of growth components exist, it is more than likely that these gilts possess the minimum threshold. The factor controlling the onset of puberty would not be age or weight, but it is lack of the development of the appropriate neural circuitry necessary for the onset of puberty. Therefore, to limit these gilts from achieving high rates of growth, and hence large body weight at puberty, it may be necessary to prohibit *ad libitum* access to feed to gilts, and/or develop a gilt 'conditioning' diet designed to limit high growth rates without adversely affecting puberty onset.

Although the growth rates achieved were not a limiting factor for age at puberty, the consequences of variable and uncontrolled growth rate for weight at breeding, longevity and sow welfare need to be addressed. Aherne et al. (1999) has suggested that, at second estrus, gilts should possess around 125 kg body weight, and 15 mm backfat. Whether puberty stimulation was initiated at 135 or 160 d, a similar range in lifetime growth rate at puberty was detected. However, as shown in Figure 6.1, a lifetime growth rate of .7 kg/d would result in gilts weighing 110 and 127 kg at puberty onset as a consequence of initiation of stimulation at 135 and 160d, respectively. Gilts stimulated at 160 d have already surpassed the target weight, but through nutritional

management gilts stimulated at 135 d will reach the target weight by their second estrus. To further describe the effect of high growth rate on body composition at puberty, the following example is outlined, using two gilts, gilt #1 and #2, with similar ages at puberty (165 and 167, respectively). Gilt #1, at puberty had a lifetime growth rate of .61 kg/d, body weight of 100.6 kg, and backfat depth of 14.4 mm. In contrast, gilt #2 at puberty, had a lifetime growth rate of .79 kg/d, body weight of 132 kg, and a backfat depth of 27.8 mm. This creates problems for the producer, as he will have to implement different strategies to manage these two gilts. To meet the target breeding conditions, gilt #1 will have to gain body condition; however, gilt #2 has surpassed her target weight and backfat.

It has been well established that the use of a mature boar is necessary for the maximal stimulation of gilts. The goal of the experiment undertaken in chapter 3 was to confirm these results in a modern, commercial genotype and production system. The results in chapter 4 show that although high lean growth rate did not adversely affect age at puberty, further studies are necessary. The effect of lean growth rate on the reproductive performance of gilts must be examined; for example, the relationships between lean growth rate, ovulation rate, litter size, lactation, ability to rebreed, remain unknown. Work should also be undertaken in development of gilt conditioning diets that result in gilts achieving puberty as early as possible, and preset target breeding weights and backfat.

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APPENDIX

LACK OF AN EFFECT OF PROSTAGLANDIN INJECTION AT ESTRUS ONSET ON THE TIME OF OVULATION AND ON REPRODUCTIVE PERFORMANCE IN WEANED SOWS

Introduction

Soede et al. (1995) have shown that ovulation occurs at approximately 72 ± 15 % of estrus duration in sows and optimal fertilization occurs when sows are bred between 0 and 24 hours before ovulation. Several reports have shown that insemination using boar seminal plasma is effective in advancing ovulation in sows (Waberski et al., 1997; Weitze et al., 1990). Estrogens present in boar seminal plasma cause the release of prostaglandins from the endometrium (Claus, 1989; Kemp and Soede, 1996). There is evidence that PGF2α may play a role in improving litter size and farrowing rate in herds with lower productivity, particularly herds with low conception rates and in which summer infertility and poor management are a problem (Peña et al. 1998; Peña et al., 2000). Peña et al. (1998) showed that an intravulva injection of PGF2α at insemination increased sow fertility. The mechanisms regulating these effects remain to be determined, but possibly include an improvement in sperm transport along the female reproductive tract, advancement of ovulation, or improvement in the fertilization of ova (Peña et al., 1998).

The experiment reported in this paper was designed to further elucidate these mechanisms, and the primary goals were 1) to determine if PGF2α is effective in improving ovulation rate and conception rate in a high producing herd, and 2), to determine whether the positive effects of PGF2α treatment on sow fertility were mediated by an advance in the time of ovulation.

A version of this paper has been submitted to Theriogenology, by J.L. Patterson, H.J. Willis, R.N. Kirkwood, and G.R. Foxcroft

A third objective was to use the data accumulated in this trial to explore relationships between post-weaning characteristics and subsequent sow reproductive performance. Steverink et al. (1999) have shown that litter size and farrowing rate are greatest when the WEI is between 2 and 4 days (d), then decreases when WEI increases from 4 to 7 d. Furthermore, several authors have reported a negative relationship between WEI and duration of estrus (Soede et al., 1995; Steverink et al., 1999). Because these relationships exist, it was relevant to examine them as potentially confounding factors in this experiment.

Materials and methods

This study was completed at the University of Alberta Swine Research Unit and with approval by the Faculty Animal Policy and Welfare Committee. Forty-seven multiparous PIC (Pig Improvement Canada) Camborough sows were used in the study and weaned between 20 and 29 d of lactation. After weaning, sows were housed individually in the breeding room. From d 3 after weaning, sows were allowed 10 min of fenceline contact twice a day (8:00 a.m. and 8:00 p.m.) with one of two mature boars. The onset of standing heat was recorded as the first time the sow exhibited the standing reflex to the back pressure test by a technician, in the presence of a boar. Sows continued to be heat checked until the end of standing heat to determine duration of estrus. If sows did not show standing heat by day 10 after weaning, they were removed from the trial. Sows were weighed and P2 backfat depth (Scanoprobe II, Scano, Ithaca, New York) was measured at farrowing and weaning. Sow weight was also recorded at standing heat. At the onset of standing heat, sows were assigned to treatment based on parity and weight at weaning, and PGF sows received 0.5 mL of a PGF2α analogue (7.5 mg/ml luprostiol, Prosolvin, Intervet, Boxmeer, Holland) (PGF) or acted as a non-injected control (CON). The vulva was first cleaned and PGF2α injected into the external vulva at the junction of the vulva with the normal cornified skin. To minimize trauma, a half-inch (12 mm) 20 gauge needle was used and directed medially and 20 to 30° cranially. From start of weaning until slaughter, sows were fed a gestation diet *ad libitum* that contained 13.4 percent crude protein, 3.1 Mcal/kg

digestible energy, and 0.6 percent lysine. At breeding, sow feed was reduced to 2.0 kg. All sows had *ad libitum* access to water.

Twelve and twenty-four h after the onset of standing heat, sows were inseminated using pooled Dalland semen at a dose of 3.0 X 10^9 morphologically normal sperm (Alberta Swine Genetics Corporation, Leduc, Alberta, Canada) per insemination. Semen was no older than 3 d from the collection date. Sows were allowed fenceline contact with one of two boars during insemination.

Transcutaneous ultrasound (Pie Medical Scanner 200, model 41480, Can Medical, Kingston, Ontario, Canada), using a 5.0 to 7.5 MHz multiple scan transducer of the right ovary, was carried out every 6 h beginning 24 h after the onset of estrus, until ovulation was detected. The time of ovulation was recorded as half the time between the last detection of follicles and their subsequent disappearance. At 36, 54, and 72 h after the onset of standing heat a 2.5 mL sample of blood was taken from an ear vein during a period of nose-snare restraint when necessary. These samples were subsequently analyzed for progesterone. After breeding, sows were moved to individual stalls in the gestation room. Twenty-eight d after insemination, sows were transported to a local abattoir for slaughter and recovery of reproductive tracts. The number of corpora lutea on each ovary were counted to determine ovulation rate, and the uterus was dissected to determine total number of live embryos, chorioallontoic fluid volume, and embryo length.

Statistical Analysis

Differences between control and prostaglandin treated sows for duration of estrus, ovulation rate, number of viable embryos and embryonic survival at d 30 of gestation were analyzed using the SAS GLM procedure (SAS, 1990). For all these variables, treatment, and WEI were included in the model. Lactation length was used as a covariate since it varied between sows. Because not all sows were slaughtered at d 30, due to restrictions at the abbatoir, d was used as a covariate for chorioallantoic fluid

volume and embryo length. To determine whether different durations of estrus affected the response to treatment, sows were grouped on the basis of a mean duration of estrus less (SHORT) or greater than (LONG) the overall mean. As progesterone concentrations were not represented by a normal distribution, the data was log-transformed prior to analysis. Data were examined using repeated measures analysis of variance (SAS, 1990) to determine overall treatment, group, and the interaction effects and the treatment and group over time by the use of orthogonal contrasts. Differences between groups for progesterone concentration, ovulation rate, number of live embryos and embryo survival were analyzed using the SAS GLM procedure (SAS, 1990).

Results

Of the 47 sows that were allocated to treatment at weaning, a total of seven animals were removed from the trial: five sows did not return to estrus within 10 d of weaning, one sow was lame, and one sow was detected as non-pregnant at day 30 of gestation. Data from these animals were not included in the analysis. The remaining 40 sows were divided evenly among treatment and the stratified allocation to treatment resulted in no difference in parity (mean ± sd) (4.3 ± 1.9 vs 3.8 ± 1.8) or weaning weight (250.1 ± 36.0 vs 240.1 ± 36.3) between CON and PGF sows.

The effect of an intravulval injection of PGF2α at the detection of estrus on the duration of estrus, ovulation rate, number of live embryos, embryonic survival, chorioallantoic fluid volume and embryo length are shown in Table A-1. Treatment had no effect ($p > .05$) on any of these parameters. Use of ultrasound was effective in determining time of ovulation in sows (Figure A-1). However, due to problems with the ultrasound machine, ultrasound data were only obtained for 13 sows on trial.

Table A-2 shows ovulation rate, number of live embryos and embryonic survival at d 30 of gestation for sows with a duration of estrus equal to or less than 60 h (short; average = 49.1 ± 9.7 h) and greater than 60 h (long; average = 75.3 ± 6.9 h).

Duration of estrus had no effect on any of these parameters ($p > .05$). Also, the treatment*estrus duration interaction was not significant, indicating that treatment was not effective in increasing ovulation rate, number of live embryos or embryo survival even in sows a short or long duration of estrus.

WEI was negatively associated with ovulation rate (Figure A-2a), number of live embryos (Figure A-2b) and duration of estrus (Figure A-2c). One sow was considered as an outlier and removed from the analysis because her WEI was greater than two standard deviations from the mean. However, even when including data from this sow the same relationships existed, Ovulation Rate = 30.5 – 0.1 WEI (h), $r = -.38$, $p = .02$; Duration of estrus = 88.7 - .29 WEI (h), $r = -.32$, $p = .05$; and Embryo survival = .84 (WEI) - .0017; $r = -.16$; $p = .34$.

The overall progesterone concentration in CON and PGF gilts 36, 54 and 72 h after the detection of the onset of estrus (Figure A-3a) was not different between PGF and CON sows ($p = .20$) and concentrations at 36 h after onset of estrus differed between treatments ($p < .05$). For sows grouped on the basis of duration of estrus, the relationship between progesterone concentration and time is shown in Figure A-3b, and overall, progesterone concentrations differed among groups (mean ± SD), short: .60 ± .42; long: -.017 ± .50; $p = .0006$.

Discussion

Our study shows that PGF2α given at the onset of standing heat had no effect on ovulation rate, number of live embryos, embryo survival, chorioallantoic fluid volume or embryo length at d 30, or conception rate. Peña et al (1998) reported that a prostaglandin injection at insemination improved farrowing rate (78.5 vs 54.4) and litter size (10.8 vs 9.1) in sows during the low fertility summer season, and addition of PGF2α to semen doses improved conception rate and litter size (Peña et al., 2000; Takacs et al., 1985). However, in these studies farrowing rates (54.4%) and conception rates (75%) in control sows were low and the 98% conception rate in control sows in

our study virtually removed any possibility of demonstrating the efficacy of PGF2α treatment.

Even though we did not have ultrasound data to identify when ovulation occurred in all sows, we believe that the difference in progesterone concentration at 36 h after the detection of onset of estrus was different between treatments and was probably related to variation in time of ovulation and estrus duration. Neither our ultrasound data, nor the progesterone data, are consistent with the hypothesis that PGF2α treatment advanced ovulation.

Waberski et al. (1997) has reported that ovulation inducing factors may be more beneficial for sows with a longer duration of estrus. It would be predicted that sows with a longer duration of estrus would ovulate relatively later than sows with a short duration of estrus, thus explaining the difference in progesterone concentration between short and long sows. Soede et al. (1995) have shown that optimal fertilization occurs 0 to 24 h prior to ovulation and that the percentage of sows with 90% normal embryos decreases with every 12 - h interval before ovulation. Therefore, insemination at fixed times after the detection of the onset of estrus may result in sows with a long duration of estrus being inappropriately inseminated. If PGF2αwas effective in advancing ovulation in a long duration of estrus, we would have predicted that PGF sows would have an improved number of live embryos and embryo survival compared to CON sows, owing to the fact more sows would fall into the optimal period (Figure A-4). However, when the effectiveness of a PGF2α injection was determined for sows grouped as having a longer duration of estrus, our results still indicate that treatment was ineffective in improving ovulation rate, number of live embryos or embryo survival.

In the context of differences to previous studies, it is possible that the positive effects reported by Peña et al. (1998) were not due to advanced ovulation but to enhanced sperm transport in the female reproductive tract. The experiment of Peña et al. (1998) differed from our own in three important elements; 1) there was no boar

stimulation at insemination, 2) sows were given a PGF2α injection at insemination rather than the onset of estrus and 3), summer infertility was a problem. Boar stimulation results in a release of oxytocin that subsequently results in a rise of prostaglandin, both enhancing muscle contractions. Sows that do not receive boar stimulation will not profit from the beneficial effects of enhanced sperm transport and thus treatment with PGF2α may improve sperm transport and improve fertility. Also, the farm in which our trial was undertaken was intensively managed, had few reproductive problems, and did not suffer from summer infertility. Under conditions of lower productivity, PGF2α may have exerted an effect.

We cannot rule out the possibility that an injection of PGF2α had a negative effect on early corpus luteum activity. It is well known that PGF2α is involved in the regulation of corpus luteum function, acting to inhibit progesterone production and induce luteolysis (Peña et al., 1998). However, pooled progesterone concentration, ovulation rate, number of live embryos and embryo survival were not different between treatment, which leads us to believe if early luteal function did differ between PGF and CON sows, it did not have long-term effects.

Litter size and farrowing rate have shown to be influenced by the day of return to estrus after weaning (Kemp and Soede, 1996, Steverink et al., 1999). Several authors have reported a negative relationship between WEI and duration of estrus (Kemp and Soede, 1996; Steverink et al., 1999); and our data confirms these results. In previously unpublished data, Steverink et al. (1999) reported that ovulation rate decreased from 21.6 to 19.7 oocytes, when WEI increased from 3 to 6 d. Because no relationship between WEI and embryo survival ($r = -.12$, $p = .44$) was detected, our data provide the first published evidence demonstrating that lower ovulation rate ($r = -.54$, $p = .0005$) associated with increasing WEI, probably contributes to lower litter size.

In conclusion, we found no benefit of a PGF2α injection in sows with already acceptable reproductive performance, suggesting that the PGF2α injection may only be a useful breeding tool on farms where summer infertility or poor management

strategies are a problem. PGF2α did not affect the overall progesterone concentration and thus did not appear to affect the time of ovulation of sows. A negative relationship existed between increasing WEI with ovulation rate and number of live embryos, providing a mechanism to explain why a decrease in WEI is associated with lower litter size.

Table A-1 Conception rate, ovulation rate, live embryos, embryonic survival, estrus to ovulation interval and the percentage of ovulation of estrus duration (mean±sd) for control (CON) and prostaglandin (PGF) sows.

	CON (n = 20)		PGF (n=20)		
	MEAN	SD	MEAN	SD	P
Duration of estrus (h)	58.8	16.0	63.0	15.5	.53
Ovulation rate (#)	21.1	4.3	21.6	4.0	.72
Total live embryos (#)	14.5	4.6	14.3	5.5	.88
Embryonic survival (%)	68.2	16.1	66.2	21.8	.86
Embryo length (mm)	23.0	3.9	23.4	3.6	.71
Chorioallantoic fluid volume (mL)	166.5	72.0	172.6	60.8	.65
Estrus to ovulation interval (h)[1]	43.0[♦]	4.9	46.3[δ]	6.9	.52
Ovulation / estrus duration (%)	78.3[♦]	13.5	81.9[δ]	13.0	.88
Conception rate (%)	100	-	95	-	.32

[1] Interval from the initiation of estrus until ovulation
[♦] n=6
[δ] n=7

Table A-2 Number of live embryos and embryonic survival of gilts with a short (SHORT) and long (LONG) duration of estrus.

	GROUP 1		GROUP 2		p	
	MEAN	SD	MEAN	SD	GROUP	TRT* GROUP
Ovulation rate	20.4	4.2	22.3	4.5	.20	.76
Live embryos (#)	14.1	14.8	14.8	5.4	.70	.80
Embryo survival (%)	68.5	19.0	65.9	19.5	.69	.68
Embryo length	22.0	4.2	24.8	2.3	.29	.54
Chorioallantoic fluid volume (mL)	153.2	68.5	189.5	56.7	.65	.69
Estrus to ovulation interval (h)	21.7	5.0	24.1	3.5	.81	.18

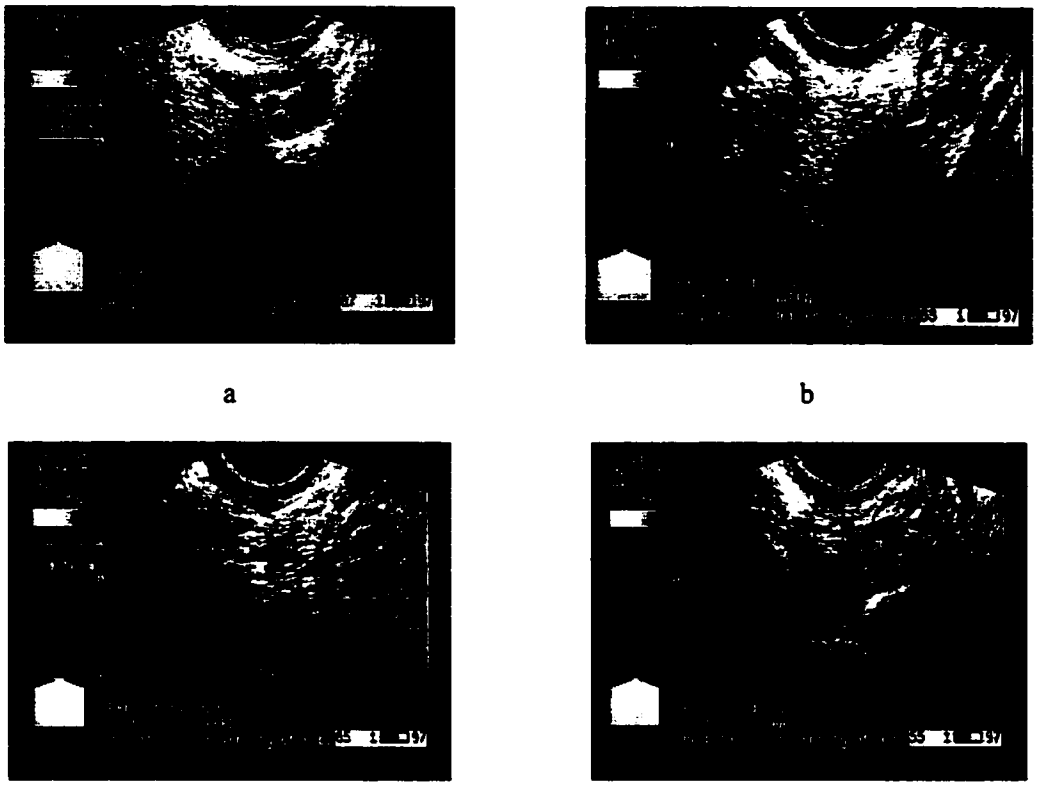

Figure A-1 Series of ultrasound images of follicular development; a) 22 h prior to ovulation, follicle diameter = 0.56 cm; b) 4 hours prior to ovulation (right and left ovary); c) 4 hours prior to ovulation, follicle diameter = 0.79 cm; and d) ovulated ovary.

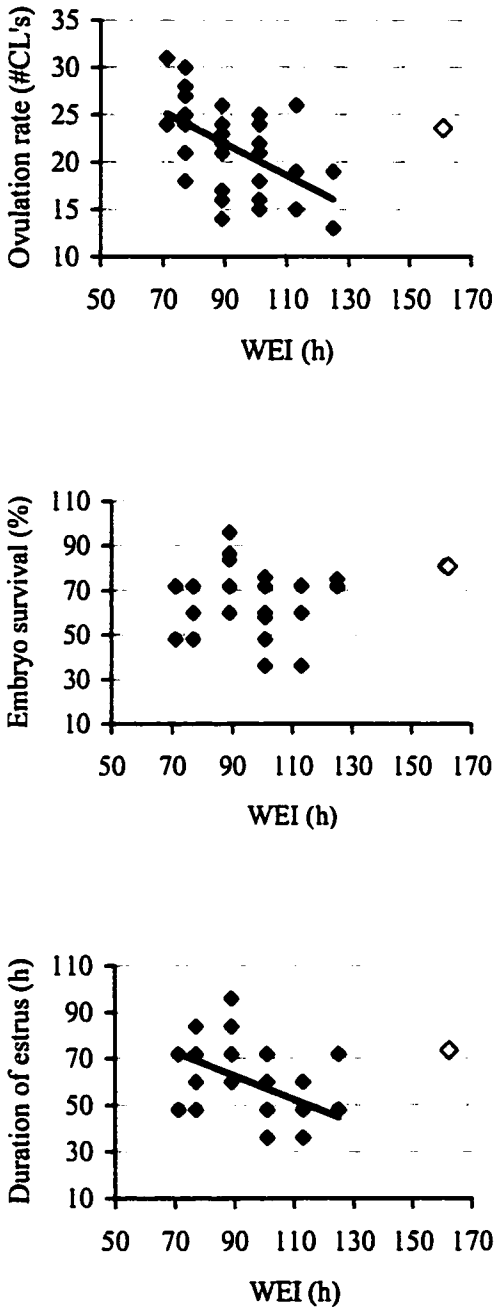

Figure A-2 .Relationships of WEI and different reproductive characteristics. a) Ovulation Rate = 37.12 - .17 (WEI); r = -.54, p = .0005; b) Estrus duration = 113.3 - .56 (WEI); r = -.49, p = .002; c) Embryo survival = .84 - .0017 (WEI); r = -.12, p = .44. Closed diamonds represent data used in the analysis, open diamond was flagged as an outlier and removed from analysis.

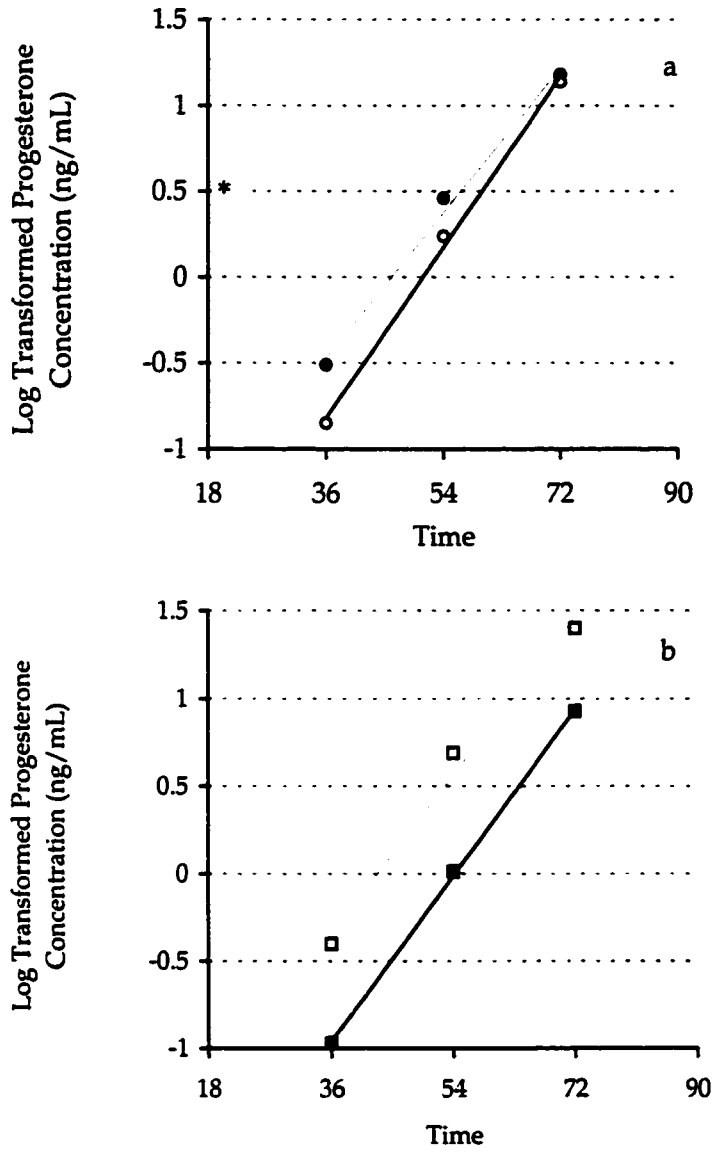

Figure A-3 a) Log transformed progesterone concentrations at 36, 54 and 72 hours after the onset of estrus (time) in CON (closed circles) and PGF (open circles) sows. *p < .05 for differences between treatment within time of sampling. b) Log transformed progesterone concentrations in GROUP1 and GROUP2 sows over time; GROUP1 (open squares) GROUP2 (closed squares).

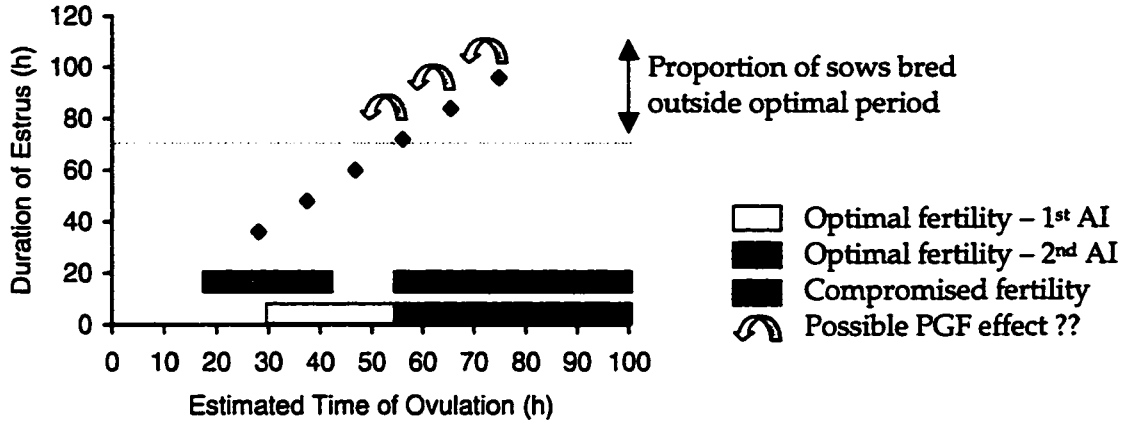

Figure A-4 Predicted time of ovulation as a percentage of duration of estrus. Ovulation was predicted to occur at 77.9 % of duration of estrus based on the average for CON and PGF sows. Grey bars represent the 24-h optimal period for ovulation to occur after the first AI. White bar represents the 24-h period for optimal fertilization following the second AI. Black bars represent the period which falls outside the optimal period for fertilization. If PGF were to exert an ovulation advancing effect, possibly more sows would fall within the optimal period for fertilization.

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