REPRODUCTIVE PERFORMANCE OF EARLY-WEANED GILTS AND FIRST PARITY SOWS FED DIFFERING PATTERNS OF FEED INTAKE DURING GESTATION AND LACTATION

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by

Darrelle Embury

In Partial Fulfillment of the

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Reproductive Performance of Early-Weaned Gilts and First Parity Sows Fed Differing Patterns of Feed Intake During Gestation and Lactation

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A Thesis/Practicum submitted to the Faculty of Graduate Studies of The University of Manitoba in partial fulfillment of the requirements of the degree of Master of Science

ABSTRACT

Two experiments were conducted to determine the effect of pattern of feed intake during gestation and lactation on the reproductive performance of two genotypes of sows.

In the first experiment, 60 National Pig Development (NPD) gilts and 53 first-parity sows were randomly assigned to one of two gestation treatments and subsequently one of two lactation treatments. Throughout gestation the control group (gC) (gilts, n=31; sows, n=26) was fed at 1.4 times maintenance d^{-1}, and the pattern group (gP) (gilts, n=29; sows, n=27) was fed in four stages based on body weight at d 1, d 30, d 60, and d 90. Each gestation group was further divided into two treatments for the 17-day lactation: the control group (lc) (gilts, n=30; sows, n=28) was 'full-fed', and the pattern group (lp) (gilts, n=30; sows, n=25) was fed in three stages based on body weight at d 1, d 6, and d 12.

In the second experiment, 18 Cotswold gilts were randomly assigned to one of two gestation treatments and one of two lactation treatments. Throughout gestation, control gilts (gC) (n=10) and the pattern group (gP) (n=8) were fed as described in Experiment I. Each gestation treatment was further divided into two treatments for the 18-day lactation: control gilts (lc) (n=9) were fed *ad libitum*, and the pattern group (lp) (n=8) was fed in three stages based on body weight at d 1, d 6, and d 12.

Average daily feed intake did not differ between treatments in Experiments I and II ($P>0.05$). Gestation treatment C consumed more feed in early gestation and gP consumed more in late gestation in both experiments ($P<0.05$). Total feed intake during

gestation was greater for gC gilts in Experiment II ($P<0.05$) and did not differ between treatments in Experiment I ($P>0.05$).

Although the patterns of P2 backfat change (Experiment II) and body weight change (Experiments I and II) differed due to gestation treatment, there were no differences between treatment groups by d 109 of pregnancy. Feed intake pattern had no effect on percent nutrient retention (Experiment II), serum urea nitrogen and progesterone (P_4) (Experiments I and II) during gestation ($P>0.05$).

Average daily feed intake and total lactation feed intake were lower for the lc treatment in Experiment I ($P<0.05$), but were not affected by feed intake pattern in Experiment II ($P>0.05$).

Backfat loss was greater for gP gilts and sows ($P<0.05$), while the gC group lost more body protein during lactation ($P<0.05$) in Experiment I. However, gestation treatment did not affect backfat depth and body protein levels at d 17 of lactation ($P>0.05$). Lactation treatment p had higher mean weight, predicted body protein and lipid contents during lactation in Experiment I ($P<0.05$). Gestation-lactation treatment combination Cc lost more backfat and body lipid during lactation, and had the lowest backfat and lipid reserves at weaning in Experiment I. Combination Cp maintained backfat depth and lost the smallest amount of body lipid during lactation. Maternal weight, backfat and predicted body composition did not differ at the end of lactation due to lactation treatment in Experiments I and II. However, the patterns of body protein and lipid utilization were different ($P<0.05$). Litter size born alive and at weaning (Experiment I), and piglet growth rate in late lactation

(Experiment II) were improved for gP gilts ($P<0.05$), but not for first parity sows in Experiment I. Lactation treatment c (Experiment I) resulted in larger litter size at weaning for gilts, but not for first parity sows ($P<0.05$).

Pattern of feed intake did not alter mean or baseline serum luteinizing hormone (LH) concentrations, LH pulse frequency, weaning-to-estrus interval, and ovulation rate in Experiment II ($P>0.05$). Gestation treatment P exhibited a greater rise in P_4 concentration postweaning and 45% more normal corpora lutea than gC gilts in Experiment II ($P<0.05$).

Lactation treatment p and the combination of Cp during gestation and lactation, extended the WEI of gilts relative to first parity sows in Experiment I.

These results indicate that pattern of feed intake during gestation produced beneficial effects in terms of reproductive performance for gilts, but did not produce these same effects for first parity sows.

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ABBREVIATIONS

AOAC	Association of Official Analytical Chemists
ADFI	average daily feed intake
CL	corpora lutea
d	day(s)
DE	digestible energy
d5pe	day 5 post-estrus
g	gram(s)
gC	control treatment during gestation
GnRH	gonadotropin releasing hormone
gP	pattern treatment during gestation
h	hour(s)
kcal	kilocalorie(s)
kg	kilogram(s)
$kg^{0.75}$	metabolic body weight
kJ	kilojoule(s)
lc	control treatment during lactation
LH	luteinizing hormone
lp	pattern treatment during lactation
Mcal	megacalorie(s)
ME	metabolizable energy

mg	milligram(s)
MJ	megajoule(s)
ml	millilitre(s)
mm	millimeter(s)
N	nitrogen
ng	nanogram(s)
NRC	National Research Council
P_4	progesterone
RIA	radioimmunoassay
SAS	Statistical Analysis System
S:P	secondary to primary muscle fiber ratio
USP	uterine secretory proteins
WEI	weaning-to-estrus interval
WSI	weaning-to-service interval

CHAPTER 1

INTRODUCTION

Recent decreases in the number of swine producers and the movement toward larger operations have resulted in the need for increased production efficiency. Sow reproductive performance is a significant component of the profitability of a swine operation and can be influenced by a variety of factors. Factors such as nutrition during gestation and lactation, and lactation length can be modified to optimize sow production efficiency without sacrificing the lifetime productivity of the sow.

Gilts and first parity sows frequently display poor reproductive performance associated with the conflicting requirements for growth to mature size and the requirements for fetal development and milk production during gestation and lactation (Pettigrew and Tokach, 1991; Aherne and Williams, 1992). As a result, young sows often exhibit prolonged weaning-to-estrus intervals (WEI) (Sterning *et al.*, 1990; Cosgrove *et al.*, 1997) and reduced litter size in the second parity (Kirkwood *et al.*, 1987a).

Nutrient requirements of the sow increase with the advancement of gestation and lactation, following the patterns of fetal growth and milk production (Noblet *et al.*, 1990; Whittemore and Morgan, 1990). Sows with insufficient total feed intake or receiving poorly-balanced rations mobilize body reserves to avoid negatively affecting litter growth (Willis and Maxwell, 1984; Neil and Ogle, 1996). Changes in maternal body composition due to loss of protein and lipid reserves affect sow body condition in gestation and lactation, and rebreeding later on.

Gestation feed intake has been associated with embryo mortality in early gestation (Jindal *et al.*, 1996, 1997), fetal growth (Aherne and Williams, 1992), as well as potential effects on milk production (Weldon *et al.*, 1991) and piglet growth in the postnatal period (Schoknecht *et al.*, 1993; Coffey *et al.*, 1994; Dwyer *et al.*, 1994). Additionally, feed intake during pregnancy has implications for sow body composition and performance during lactation (Mullan and Williams, 1989; Dourmad *et al.*, 1994). Gestation feed intake is negatively related to feed intake in lactation (Hughes, 1994), and voluntary feed intake during lactation is a factor limiting sow performance (Weldon et al., 1994a).

Young sows often cannot consume sufficient feed during the lactation period to meet the requirements for maintenance, milk production, and maternal growth (Cole, 1990). Feeding levels during lactation are connected to litter performance during lactation (King and Dunkin, 1986; Neil and Ogle, 1996) and subsequent reproductive performance of the sow (Whittemore and Yang, 1989; Dourmad, 1991). Body weight and backfat loss during lactation, as affected by gestation and lactation feeding levels, results in an extended WEI (Armstrong *et al.*, 1986). Feed intake during lactation is related to profiles of LH secretion at weaning (Tokach *et al.*, 1992), and P_4 concentration early in the subsequent pregnancy (Kirkwood *et al.*, 1987a). Alterations in secretion of these hormones have been associated with the length of the WEI and embryo survival, respectively.

The shift in management practices toward shorter lactation lengths (early weaning), in an attempt to increase the number of pigs per sow per year, may have negative effects on sow reproductive performance. In general, shorter lactation lengths are related to longer WEI

(Foxcroft *et al.*, 1995; Cosgrove *et al.*, 1997) and reduced subsequent litter size (Varley, 1982; Xue *et al.*, 1993), mediated by disruption of normal hormone profiles (Varley *et al.*, 1981; Archibong *et al.*, 1987; Kirkwood *et al.*, 1984). The reproductive problems associated with young sows may be compounded by the adoption of early weaning practices.

Current feeding practices during the gestation period provide gilts or sows with a fixed amount of feed during pregnancy, and may not consider individual requirements. This type of feeding system allows gilts and sows to become catabolic during late gestation if nutrient supply is insufficient. Conversely, nutrient oversupply during gestation results in reduced feed consumption during lactation.

Conventional lactation feeding systems restrict feed intake just prior to farrowing and increase the feed allowance gradually during the first few days of the lactation period to achieve *ad libitum* intake. Feed restriction in early lactation causes a reduction in total feed intake during lactation and may influence sow metabolic condition and reproductive performance in the subsequent cycle.

Little information is available on the effect of feed intake patterns during consecutive stages of the reproductive cycle of young sows. Previous studies have evaluated gestation and lactation feeding methods separately (Verstegen *et al.*, 1987; Cromwell *et al.*, 1989; Moser *et al.*, 1987; Koketsu *et al.*, 1996; Zak *et al.* 1997a). However, it is important to understand the relative contributions and influences of each stage of the production cycle on subsequent stages.

The objective of these studies was to modify the feed intake pattern of early-weaned gilts and first parity sows to reflect the changing maternal and piglet requirements during gestation and lactation. The influence of these altered feed intake patterns on reproductive performance was determined using two different sow genotypes.

CHAPTER 2

REVIEW OF THE LITERATURE

Introduction

Improving sow productivity is a major focus of modern swine systems. Sow productivity can be defined as the number of pigs weaned per sow per year, and is comprised of the number of piglets weaned per litter and the number of litters per sow per year. Litter size born alive and preweaning mortality influence the number of pigs weaned per litter. The number of piglets born alive is a function of fertilization rate, ovulation rate, and embryo survival (Varley, 1982). Gestation length, lactation length, and the number of non-productive days determine the number of litters per sow per year. Gestation length is a fixed biological effect that cannot be manipulated to improve production. However, lactation length and the number of non-productive days can be altered to affect sow productivity.

Productivity can be maximized by improving the efficiency of production and increasing sow longevity through optimum gestation and lactation performance, and a reduction in the number of non-productive days. Sow productivity is altered by factors including nutrition and management (ie. lactation length), and the influence of these factors on the metabolic and endocrine status of the sow.

Reproductive Performance: the gilt and first parity sow

The gilt and first parity sow represent a specific challenge in terms of improving productivity of the herd. Selection of gilts for leanness, rapid growth, increased milk production, and a reduced age at puberty have resulted in pigs with lower appetites (Aherne and Williams, 1992) and insufficient body reserves to support the increased demands of the modern production system (Rozeboom *et al.*, 1996). Consequently, gilts and first parity sows often exhibit decreased reproductive performance and low productivity.

Reproductive problems constitute approximately 30% of reasons for culling of first parity animals (Carroll *et al.*, 1996), resulting in a high herd replacement rate and reduced sow longevity in the herd. Reproductive failure may be related to genetic, nutritional, and environmental factors.

Early weaning and Productivity

Lactation length can be varied according to specific management objectives. The length of the lactation period has an effect on piglet performance and subsequent sow reproductive performance (Varley, 1982; Pettigrew *et al.*., 1995; Cosgrove *et al.*, 1997; Koketsu *et al.*, 1997). Traditional lactation periods were three to four weeks in length. The desire to increase efficiency of production has resulted in early weaning systems with lactation lengths of 10 to 18 days (Pettigrew *et al.*, 1995; Xue *et al.*, 1997a).

Early weaning has been adopted in an effort to improve the health status of the herd (Pettigrew *et al.*, 1995), to improve growth performance of weaned pigs, and to more efficiently utilize facilities (Dial *et al.*, 1992). However, early weaning may have a negative effect on certain aspects of sow reproductive performance. Shorter lactation lengths have been associated with decreased herd productivity, specifically with an increased weaning-to-estrus interval (WEI) (Foxcroft *et al.*, 1995; Cosgrove *et al.*, 1997; Xue *et al.*, 1997a) and reduced subsequent litter size (Varley, 1982; Xue *et al.*, 1993).

Weaning-to-Estrus Interval

The interval from weaning-to-estrus is increased by a reduction in lactation length (Varley, 1982; Xue *et al.*, 1997a). Xue *et al.* (1993) observed an increase in the weaning to service interval (WSI) with lactation lengths shorter than 17 days. However, WSI was unaffected by lactation lengths of 17-30 days (Foxcroft *et al.*, 1995). Early weaning of sows occurs at a time when the reproductive axis is suppressed, resulting in extension of the WEI (Cosgrove *et al.*, 1997).

Subsequent Litter Size

The influence of lactation length on subsequent litter size is mediated by the effects of early weaning on uterine environment suitability, ovulation and fertilization rates, and embryo survival.

Ovulation and fertilization rates and embryo survival

Varley and Cole (1976b) and Svajgr *et al.* (1974) found no effect of lactation length on ovulation rate when comparing sows weaned at 42-d versus 7-d. Subsequent work by Varley (1982) found similar results. Svajgr *et al.* (1974) found fertilization rates of early weaned sows to be comparable to sows weaned later in lactation.

Reduced embryo survival may contribute to the decrease in subsequent litter size observed with shorter lactation lengths. Embryo survival was negatively affected by shortened lactation length in a study by Varley and Cole (1976b). Evaluating embryo survival of sows weaned after 7-, 21- or 42-d lactation periods showed embryo survival rates of 59.2%, 63.9%, and 81.7%, respectively.

Numerous studies relate early weaning to a reduction in subsequent litter size (Moody and Speer, 1971; Cole *et al.*, 1975; Varley and Cole, 1976a). In many cases, these early findings utilize lactation lengths of greater than 21-d and multiparous sows. The effect of shortened lactation length on litter size is more readily observed in multiparous sows due to the inherently smaller litter size of first parity sows (Foxcroft *et al.*, 1995). Recent work supports this positive relationship between embryo survival, litter size and lactation length (Xue *et al.*, 1997a).

Uterine environment

Britt and Flowers (1997) and Cosgrove *et al.* (1997) cite incomplete uterine involution as a contributing factor to embryo mortality in sows; particularly those weaned before 14-d. The process of uterine involution begins in the first week of lactation and

proceeds until d 21 to d 28 post-farrowing (Varley, 1982), and may occur more quickly in sows with longer lactation lengths compared to early weaned sows (Cosgrove *et al.*, 1997). Endometrial repair is also occurring at this time and continues through d 14 to d 21 post-farrowing. Complete uterine involution is not necessary for successful establishment of pregnancy (Cosgrove *et al.*, 1997) and the uterus is capable of sustaining a pregnancy by d 18 post-farrowing (Levis, 1997). However, weaning earlier than 18 to 21 d post-partum may subject the embryos to unfavourable uterine conditions (Varley, 1982) and result in reduced embryo survival (Cosgrove *et al.*, 1997).

Abnormal levels of steroid hormones in early gestation could negatively influence embryo survival through disruption of the passage rate of the fertilized eggs along the oviducts or by exposing the fertilized eggs to unfavourable environmental conditions within the uterus (Varley, 1982). Varley *et al.* (1981) demonstrated an increased pattern of progesterone (P_4) secretion in the 26 d following mating for sows weaned at d 10 versus those weaned at d 42. The increased level of P_4 secretion was linked to a prolonged estrogen surge during and after mating in early weaned sows, however ovulation rate was not measured and this could contribute to the difference in P_4. Additional steroid-dependent factors, such as uterine secretory proteins (USP), are possible mediators of the synchronicity between the embryo and uterine lumen (Varley, 1982; Archibong *et al.*, 1987; Simmen and Simmen, 1990).

Gonadotropins

Alterations in gonadotropin profiles of the sow during late lactation, post-weaning, and early in the subsequent pregnancy may also be associated with the reduction in embryo survival (Varley, 1982). Conventional lactation lengths allow for a gradual decrease in the suppressory effects of suckling on hypothalamic-pituitary-ovarian activity (Varley, 1982; Kirkwood *et al.*, 1984).

In early weaned sows it is possible that the sensitivity of the hypothalamic-pituitary axis is reduced, or that weaning is occurring at a time of suppression of the reproductive axis (Cosgrove *et al.*, 1997). Edwards and Foxcroft (1983) demonstrated a diminished preovulatory luteinizing hormone (LH) surge in early weaned sows. Lower basal LH levels were observed by Kirkwood *et al.* (1984) in early weaned sows during the post-weaning period.

Follicular recruitment normally occurs during early lactation (Cosgrove *et al.*, 1997). In early weaned sows follicular development and recruitment may be occurring during late gestation, having implications for oocyte maturation, ovulation, and embryo survival (Cosgrove *et al.*, 1997). Zak *et al.* (1997b) recognize that the rate of development and maturation of follicles and oocytes may contribute to decreased embryo survival.

The reproductive problems associated with young sows may be compounded by the adoption of early weaning practices. Xue *et al.* (1997a) suggest longer lactation lengths for first parity sows to minimize the negative effects of early weaning on reproductive performance.

Nutrition and Productivity

The link between nutrition and reproduction has been recognized in previous research (Einarsson and Rojkittikhun, 1993; Carroll *et al.*, 1996; Xue *et al.*, 1997b; Pluske *et al.*, 1998). Nutritional effects on sow reproductive performance are mediated by changes in sow body composition, metabolic or endocrine factors. Sow nutrition during each stage of the reproductive cycle will influence nutrient requirements and performance in subsequent stages. Nutrition during the pre-breeding and gestation periods is associated with ovulation rate (Flowers *et al.*, 1989; Beltranena *et al.*, 1991), embryo survival (Jindal *et al.*, 1996; Jindal *et al.*, 1997), and fetal growth (Schoknecht *et al.*, 1993; Schoknecht, 1997). Potential improvements in litter size and litter birth weight, as well as sow body condition, and consequently, reproductive performance may be recognized during these time periods.

Pre-Breeding Nutrition

Nutrition and Ovulation Rate

Ovulation rate is a determinant of potential litter size and may be the initial limitation to maximal productivity in the gilt. The main factors influencing ovulation rate are individual animal effects such as age, or factors imposed on the animal such as nutrition (Hughes and Varley, 1980).

Short-term mediators of ovulation rate

Evidence suggests that increasing feed (or energy) intake above the maintenance requirement for a period of 8 to 14 d prior to breeding results in an increase in ovulation rate (Flowers *et al.*, 1989). This elevation in ovulation rate due to increased intake is known as 'flushing'(Beltranena *et al.*, 1991). The positive effect of increased feed intake on ovulation rate will be beneficial in animals where ovulation rate is below an acceptable level (Aherne and Williams, 1992; Hughes, 1994). This would be the case in young gilts bred at first estrus, and feed-restricted gilts. Mature sows, and gilts on full-feed will not benefit (Cox, 1997). Short-term nutritional modification (ie. flushing) near the time of estrus may modulate ovulation rate by stimulating the secretion of gonadotropins (Beltranena *et al.*, 1991), or through the involvement of metabolic hormones influenced by diet (Flowers *et al.*, 1989) in the absence of major changes in body weight or composition (Beltranena *et al.*, 1991).

Gonadotropins and ovulation rate

Nutrition can exert both hypothalamic-pituitary and direct ovarian effects on the ovary to alter ovulation rate. Gonadotropin stimulation is necessary to promote the maturation of preovulatory follicles (Foxcroft and Hunter, 1985). Short-term nutritional influences on reproduction may be insulin-mediated (Booth *et al.*, 1996) because observed effects on gonatropin secretion and ovarian development are occurring too rapidly to be explained by changes in body weight or composition.

Plasma levels of insulin may be involved in the stimulation of gonadotropin-releasing hormone (GnRH) release from the hypothalamus (Flowers *et al.*, 1989; Cosgrove *et al.*,

1997) and consequently in increasing LH and follicle stimulating hormone (FSH) levels (May and Schomberg, 1981; Cox *et al.*, 1987). Insulin-mediated changes in the frequency of GnRH secretion result in an increase in LH secretion, enhancing follicular development (Booth *et al.*, 1996). Conversely, short-term nutritional restriction associated with decreased insulin levels may suppress GnRH/LH release (Beltranena *et al.*, 1991). Insulin receptors have been identified in the region of the hypothalamus associated with GnRH activity in the pig and the rat (Cosgrove *et al.*, 1997). Realimentation of 7-d feed-restricted prepubertal gilts (Booth *et al.*, 1996) resulted in increased uterine weights and an increased number of total ovarian follicles, which could have implications for reproductive performance at puberty. These responses could be due to the noted increase in LH secretion. Flowers *et al.* (1989) observed an increase in concentrations of FSH and pulses of LH 5 d prior to estrus in gilts receiving 3.37 kg d^{-1} compared to gilts fed 1.70 kg d^{-1} for two weeks prior to estrus. The same study showed elevated plasma insulin for seven days before estrus in gilts with high feed intake. Booth *et al.* (1996) realimented feed-restricted prepubertal gilts, and found an increase in LH secretion five hours after realimentation.

Direct ovarian influences on ovulation rate

Short-term nutritional changes may have direct ovarian effects mediated by insulin (Ashworth, 1994). Insulin receptors have been identified on porcine granulosa cells (Otani *et al.*, 1985). *In vitro* work has shown insulin to be a critical component for certain aspects of porcine granulosa cell growth and development (May and Schomberg, 1981). As well, insulin potentiates FSH-stimulated LH receptor induction and steroidogenesis. Insulin may

increase ovulation rate through a reduction in follicular atresia (Booth, 1990; Ashworth, 1994) or an increase in follicular recruitment (Dailey *et al.*, 1975).

The size and heterogeneity of the preovulatory pool of follicles and follicular and oocyte quality (Foxcroft *et al.*, 1995; Zak *et al.*, 1997b) may be influenced by nutrition during the pre-breeding stage. Pope *et al.* (1990) have suggested that the preovulatory development of the follicle and oocyte has consequences for subsequent embryo survival.

Nutrition and Gestation

During gestation the sow requires sufficient nutrients for maintenance, maternal gain, and the development of reproductive tissue (mammary gland and uterus) and fetuses (Verstegen *et al.*, 1987; Genest and D'Allaire, 1995). In the case of the gilt and first parity sow, additional feed is required for growth to mature size (Verstegen *et al.*, 1987; Cosgrove *et al.*, 1997). There is increasing evidence to support a connection between nutrition during gestation and effects on embryo survival, fetal growth, and sow body composition.

Embryo Survival and Litter Size

Embryo survival/mortality contributes to the number of pigs born per litter, and therefore to sow productivity. Embryo mortality is defined as losses in the period from conception to approximately d 30 of gestation (Pere *et al.*, 1997), however the timing of embryonic death is inadequately defined. Proposed causes of embryo mortality during early

gestation include genetically defective embryos, asynchrony between the uterine environment and the embryos, asynchronous development of littermates, improper nutrition, insufficient P_4 and/or USP secretion, or combinations of these factors.

Nutrition during early pregnancy has been examined in connection with its effects on embryo mortality and litter size. During pregnancy the developing embryos receive high priority in terms of nutrient supply (Noblet *et al.*, 1990; Noblet *et al.*, 1997). Sows with insufficient total feed intake or receiving poorly-balanced rations mobilize body reserves to avoid negatively affecting fetal growth and development (Willis and Maxwell, 1984). Therefore, under-nutrition in early gestation must be severe to affect embryo survival (Aherne and Williams, 1992). Interestingly, some studies have found that high levels of feed intake during early gestation in the gilt have negative consequences for embryo survival (Jindal *et al.*, 1996; Jindal *et al.*, 1997). However, other research has not demonstrated similar effects of high levels of feed intake on embryo survival (Dyck *et al.*, 1980).

Progesterone and embryo survival

Progesterone is the primary steroid hormone involved in the maintenance of pregnancy and is important as a regulator of oviductal and endometrial development and embryo survival (Jindal *et al.*, 1996). Progesterone is secreted from the ovarian luteal cells (Hughes and Varley, 1980) and its concentration peaks at approximately d 10 of gestation, and subsequently declines to a relatively constant level by d 30.

Contradictory evidence exists relating to the effects of nutrition in early pregnancy on embryo survival. Some experiments have shown a relationship between embryo survival and

feed intake, but the results are often difficult to interpret because of differences in the duration of dietary treatment.

Dyck (1991) found no difference in embryo survival at d 30 of gestation when comparing gilts fed 2.5 kg d^{-1} versus gilts restricted to 1.25 kg d^{-1} from breeding until d 10 of gestation. Pharazyn *et al.* (1991) fed two levels of energy and protein to gilts from d 3 to d 15 of gestation and saw no effect on ovulation rate, plasma P_4 or embryo survival to d 28 of gestation. They proposed that in cases of already high embryo survival, lowering feed intake during early pregnancy will produce no observable benefit. Einarsson and Rojkittikhun (1993) suggest that the increase in embryo mortality related to high energy intake in gilts during the premating period and early gestation may be due to an increased ovulation rate, resulting in no net increase in litter size. The negative relationship between ovulation rate and embryo survival may explain the increased embryo mortality observed in multiparous sows.

Other researchers have demonstrated an inverse relationship between feed intake during early pregnancy, P_4 concentrations and embryo survival in gilts. The influence of feed intake on embryo survival may be mediated by changes in the metabolism or secretion of P_4 (Jindal *et al.*, 1996; Jindal *et al.*, 1997).

Dyck and Strain (1983) found that high feed intake from d 1 to d 10, but not from d 11 to d 20 post-mating, increased embryo mortality in gilts at d 30 of gestation. This led to the hypothesis that level of feeding may affect embryo survival during a critical period in early gestation. Jindal *et al.* (1996) compared the effects of three gestation feed intake levels on reproductive performance. Gilts were fed 2.5 kg d^{-1} prior to breeding and were either fed at

NRC (1988) recommended levels (approximately 1.9 kg d^{-1}) starting on d 1 or d 3 of gestation, or were fed 2.6 kg d^{-1} from d 1 until d 15 of pregnancy. Feed restriction implemented on d 1 allowed for effects of P_4 concentration at both the oviductal and uterine stages of embryo development to be examined. Delaying the feed reduction until d 3 allowed for development of the embryo within the uterus to be investigated (Foxcroft, 1997). Ovulation rate did not differ between treatments. Embryo survival was affected by feed intake level, with d 1-normal and d 1-high treatments differing (86% versus 67%, respectively). Plasma P_4 concentrations 3 d after estrus were highest in gilts that were fed at lower levels from d 1. Day 3-normal and d 1-high P_4 and embryo survival values did not differ. These authors suggest that the critical period during which feed intake has a positive effect on embryo survival is limited to the day after the onset of estrus, indicated by embryo survival and P_4 concentration in gilts fed at lower levels from d 1 of gestation. The lack of dietary intake effect on embryo survival reported in the previous section by Pharazyn *et al.* (1991) may be due to the delay in reduction of feed intake until d 3 of gestation. Further evidence for a role of P_4 in embryo survival was offered by Ashworth (1991). Progesterone concentrations in *ad libitum*-fed gilts were restored through administration of exogenous P_4 during the post-mating period, leading to improved embryo survival at d 30. A second experiment by Jindal *et al.* (1997) tested the hypothesis that nutritional effects on embryo survival are mediated by P_4. Gilts were allocated to a high level of feed intake (2.0 times maintenance) with or without P_4 injection commencing 24 h after the onset of estrus. Progesterone concentrations and embryonic survival were higher in gilts administered P_4.

Feed intake during early gestation may affect embryo survival via alterations in circulating P_4 concentration (Jindal *et al.*, 1996; Jindal *et al.*, 1997). Plasma P_4 concentration represents a balance between synthesis in the ovary and metabolic clearance by the kidney and liver (Jindal *et al.*, 1996). Increased feed intake may increase hepatic blood flow to metabolize the additional nutrients, leading to an elevated metabolic clearance rate of P_4 and therefore a lower hormone level in the blood (Hughes and Pearce, 1989; Jindal *et al.*, 1996).

Alternatively, differences in P_4 synthesis or an earlier increase of P_4 in relation to the timing of the preovulatory LH peak may explain the effects on embryo survivability (Jindal *et al.*, 1996). In a subsequent experiment, Jindal *et al.* (1997) fed gilts 2.5 kg d^{-1} for one estrous cycle, and at 1.5 or 2.0 times maintenance from d 1 of the next estrus. Progesterone concentration 72 h after onset of estrus and embryo survival were lower in gilts with the higher feed intake. The timing of the P_4 rise after the LH peak was delayed by 10 h in the gilts fed at the higher plane of nutrition (38 h versus 28 h). Foxcroft (1997) believes that this delay will be mirrored in a difference in oviduct concentration of P_4, exerting an effect on oviductal function in the periovulatory period. Dietary changes manifested in the oviductal environment, particularly with relation to steroid concentrations, may affect the transport of the embryo and subsequent synchrony with the uterus (Foxcroft, 1997), resulting in increased embryo mortality.

A delay in the rise of P_4 may also influence the timing of the required uterine changes (Jindal *et al.*, 1997), thereby compromising embryo survival in the early stages of development by disrupting uterine and embryo synchrony.

Pharazyn *et al.* (1991) confirmed that variability in P_4 secretion during early pregnancy is associated with differences in P_4 concentrations perfusing the oviductal vasculature. These differences may exert an effect on the oviductal environment and embryonic development and viability. Jindal *et al.* (1996; 1997) describe the importance of the oviductal environment in the cleavage, development and transport of the embryo from the oviduct to the uterus during very early pregnancy. The first cleavage of the embryo takes place within 14 to 16 h after ovulation (Hughes and Varley, 1980) while the embryo is in the oviduct (Jindal *et al.*, 1997). The embryo migrates to the uterus at 48 to 72 h after ovulation and remains near the uterotubal junction until d 5 to d 6 of gestation. It is possible that nutritional influences on P_4 secretion may alter the oviductal or uterine environment during the very early stages of gestation, by changing development or secretory activity, leading to asynchrony between the embryo and uterus, thereby impacting embryo survival (Pharazyn *et al.*, 1991; Jindal *et al.*, 1996; 1997).

Uterine and conceptus secretory proteins and embryo survival

Coordinated changes that occur between the maternal endometrium and conceptus during early pregnancy are critical to embryo survival. Considerable embryonic loss can occur during this period if the synchrony of these events are disrupted (Simmen and Simmen, 1990; Roberts *et al.*, 1993).

During gestation, pregnancy specific proteins are secreted by both the endometrium and conceptus which aid in the growth and development of the embryo (Simmen and Simmen, 1990; Jindal *et al.*, 1997). Progesterone is required for the production of several uterine

secretory proteins (USP) involved in the support of embryos throughout gestation (Varley, 1980; Roberts *et al.*, 1993). These proteins can be influenced by nutrient levels. If the level of feed intake during early gestation influences circulating P_4 concentrations, it is possible that the lower levels of P_4 influence the production of other factors necessary for normal fetal development and survival (Close, 1997).

Conceptus-derived proteins are also secreted which assist in the regulation of endometrial and fetal development (Simmen and Simmen, 1990). A drop in P_4 level in the blood may negatively affect the secretion of some of these proteins, and therefore increase embryo mortality (Close, 1997).

Mammary Gland Development

The period from mid- to late gestation is an important time in the development of the mammary gland of gilts (Weldon *et al.*, 1991; Aherne and Williams, 1992). Rapid growth of mammary tissue occurs between d 75 to d 105 of gestation, with the period between d 75 to d 90 being critical in the development of milk secretory tissue (Weldon *et al.*, 1991). Nutritional effects on mammary gland development in the gilt may influence milk production and piglet performance in lactation.

Weldon *et al.* (1991) fed gilts either adequate or high levels of protein (216 or 330 g d^{-1}) and energy (5.76 or 10.5 Mcal ME d^{-1}) from d 75 to d 105 of gestation. High dietary energy intake during this period was found to have negative effects on mammary development, including reduced mammary cell number and amount of milk-secreting tissue.

Fetal Growth

The rate of gain during the first trimester of pregnancy is relatively slow and occurs primarily in the placenta, fluids and uterus; as pregnancy progresses, gain occurs predominantly in the fetus (Noblet *et al.*, 1997).

Fetal growth is a high priority in terms of nutrient supply. Development of the fetuses will not be affected unless the sow is fed below the maintenance requirement (Einarsson and Rojkittikhun, 1993), in which case fetal growth may be reduced (de Lange *et al.*, 1980; Young *et al.*, 1990). Insufficient nutrient availability for fetal growth may result in mobilization of maternal reserves to meet fetal requirements (Close *et al.*, 1984). In early gestation fetal demands on the maternal system are relatively low (Close *et al.*, 1984) and variances in maternal feed intake will have little effect on fetal weight. However, during mid- to late gestation, maternal nutrition may affect piglet birth weight and subsequent performance (Pond *et al.*, 1992).

Mid-gestation

Maternal nutrition during mid-gestation may have influences on fetal growth that have consequences for body composition of the piglets at birth. Muscle fiber number is an important determinant of muscle mass in the pig (Miller *et al.*, 1975). The progression of muscle fiber development occurs with an initial rapid development of the primary muscle fibers beginning on d 50 of gestation, followed by a slower phase of secondary fiber development on the surface of the primary fiber (Dwyer *et al.*, 1994). Muscle fiber hyperplasia is complete in the piglet by d 90 of pregnancy. Primary muscle fiber numbers are

genetically determined and are relatively resistant to nutritional effects. However, secondary muscle fiber numbers are responsive to conditions *in utero*, such as maternal nutrition (Dwyer *et al.*, 1994), and are responsible for the variability in muscle fiber number seen within litters (Dwyer and Stickland, 1991). Prenatal conditions influencing fiber number have the potential for long-term effects on postnatal growth. Low birth weight pigs have a decreased fiber number caused by a reduced secondary fiber population (Wigmore and Stickland, 1983). Although maternal nutrition cannot increase muscle fiber number above a maximum level, muscle fiber number in low birth weight piglets may be increased by improved maternal nutrition. This would result in a more homogeneous distribution of muscle fiber number within the litter. The number of primary and secondary fibers that formed prenatally can be determined postnatally in the pig.

Dwyer *et al.* (1994) fed sows 2.5 kg d^{-1} (control) throughout gestation, or 5 kg d^{-1} from either d 25 to d 50, d 50 to d 80, or d 25 to d 80 of gestation. Muscle fiber number was estimated in the piglets at five weeks of age. The three high feed intake groups had larger mean ratios of secondary to primary muscle fiber number (S:P) than the control group. No difference existed between S:P for the high intake groups. Piglets from sows fed the high level throughout gestation had a faster growth rate from d 70 to slaughter than the control group. Experimental evidence has not consistently supported the influence of maternal feed intake during mid- to late gestation on secondary muscle fiber number and subsequent pig growth.

Late gestation

The nutrient requirements of the sow increase with the progression of gestation, reflecting the increase in maternal weight gain and pattern of fetal development (Verstegen *et al.*, 1987; Noblet *et al.*, 1997). During the last month of gestation, fetal growth is exponential (Noblet *et al.*, 1997). More specifically, fetal weight doubles during the last ten days of pregnancy (Aherne and Williams, 1992). Increasing sow feed intake during late gestation to improve piglet birth weight has resulted in variable success. Birth weight appears to be related to energy and protein intake of the sow during pregnancy (Cromwell *et al.*, 1989; Pond *et al.* 1992). Piglet birth weights increased as gestation energy intake increased, plateauing at an intake of 6 Mcal ME d^{-1} (Aherne and Williams, 1992). High energy intake until d 110 of gestation (2.27 kg d^{-1}; 7.4 Mcal ME d^{-1}) increased piglet birth weight and weight gain to weaning compared to normal intake levels (1.82 kg d^{-1}; 5.9 Mcal ME d^{-1}) (Coffey *et al.*, 1994). Piglet birth weight was heavier for primiparous sows fed to achieve 20 mm P2 backfat at farrowing versus sows farrowing with 12 mm P2 backfat (Yang *et al.*, 1989).

Other studies have examined the importance of protein nutrition of the sow during pregnancy and implications for fetal growth (Pond *et al.*, 1992; Schoknecht *et al.*, 1993). Birth weights of piglets born to sows fed a protein restricted (0.5%) diet during early (d 1 to d 44) or late gestation (d 81 to farrow), or throughout pregnancy, were lower than from sows fed a protein-adequate diet (13%) throughout pregnancy (Schoknecht *et al.*, 1993). Post-weaning performance of the piglets, as measured by average daily gain, was influenced by

maternal protein restriction throughout pregnancy.

The type of nutrient restriction during pregnancy, the time at which the restriction is imposed, and the growth requirements of the sow will influence fetal and subsequent piglet growth (Schoknecht, 1997).

Metabolic Indicators of Reproductive Status: Gestation

In addition to nutritional influences on fetal growth, changes in maternal body composition are affected by sow feeding during gestation (Cole, 1990). Differences in maternal body composition may contribute to the control of reproductive function. Additional factors regulating metabolic status, including alterations in nutrient balances and levels of metabolic hormones and substrates, may be responsible for the effects of nutrition on reproduction in the absence of changes in sow body composition.

Body Composition

Weight and backfat are general measures of changes occurring in the body of the sow (Whittemore and Yang, 1989) and can indicate alterations in metabolic status. The influence of nutrition on anabolic and catabolic processes occurring during the various phases of the reproductive cycle may be reflected in changes in body composition. However, nutritional modulation can induce acute or chronic changes in metabolic status and the reproductive axis in the absence of changes in body composition (Booth, 1990). Nutritional effects on sow

weight or backfat during pregnancy can have implications for subsequent reproductive performance, by influencing lactation feed intake, subsequent weight loss, and the WEI.

Feed intake during gestation should allow for gain in maternal tissues, taking into account the requirements for growth in the younger animal (Verstegen *et al.*, 1987; Noblet *et al.*, 1997), and the influence of previous lactation weight loss on body condition (Einarsson and Rojkittikhun, 1993). Nutrition during gestation should provide for a controlled amount of body fat and a large amount of body protein at farrowing to maximize subsequent lactation and reproductive performance (Pettigrew and Yang, 1997).

The sow can mobilize body reserves in late pregnancy to support the increasing fetal requirements if maternal nutrition is insufficient (Cole, 1990; Einarsson and Rojkittikhun, 1993). However, a catabolic state during late gestation may negatively affect lactation ability of the sow (Verstegen *et al.*, 1987).

Target values for maternal body weight gain of 45 kg (composed of 20 kg litter gain and 20 to 25 kg net maternal gain) for sows through gestation have been suggested (Verstegen *et al.*, 1987; Aherne and Williams, 1992). More generous recommendations for 25 to 40 kg net maternal gain during the first parity, and 25 to 30 kg for the second parity were proposed by Verstegen and Den Hartog (1989). Backfat thickness at the P2 site should reach 20 mm at farrowing for gilts (Yang *et al.*, 1989; Aherne and Williams, 1992). In the case of multiparous sows, weight loss during the previous lactation will influence production targets (Verstegen *et al.*, 1987).

Underfeeding during pregnancy is associated with lower body reserves at farrowing (Dourmad *et al.*, 1994). Gilts or sows consuming a high level of feed intake during gestation have higher body weight or condition at farrowing (Dourmad *et al.*, 1994). This increased feed intake and body weight gain during gestation negatively influences sow lactation feed intake, contributing to increased weight loss during lactation (Coffey *et al.*, 1994).

The main response to an increased level of feeding during gestation is an increase in maternal weight gain, with lean tissue gain being the primary area of weight gain in gilts (Aherne and Williams, 1992). The average composition of maternal gain during gestation is 70% lean and 30% lipid (Aherne and Williams, 1992).

Young *et al.* (1990) fed gilts low, medium or high energy levels until d 109 of gestation (22.2, 29.2, 36.2 MJ DE d^{-1}, respectively) for four parities. Gestation weight and backfat gain increased with feed intake level. Fewer sows fed the low gestation energy level completed parities three and four due to low backfat levels and poor conception rates.

Xue *et al.* (1997b) fed gilts normal (6.5 Mcal ME d^{-1}) or high (11 Mcal ME d^{-1}) levels from d 35 of gestation until farrowing. Gilts fed the high energy level gained more weight and backfat during gestation. Body fat at farrowing, due to gestation feeding, will influence primiparous sow performance during lactation, by influencing weight loss and lactation voluntary feed intake.

The influence of gestation feed intake on subsequent reproductive performance may be mediated through direct effects on body composition or indirectly through effects on feed intake during lactation (Xue *et al.*, 1997b). Difficulty in accurately predicting feed intake

requirements during gestation is due to individual sow needs for growth, maintenance, and production. At this time there is little concurring information on ideal gestation feed intake levels, gestation weight gain, and consequences for subsequent lactation performance.

Nutrient Utilization

Measurement of nitrogen and energy retention in the gilt or sow may provide information on the metabolic status of the animal due to imposed nutritional treatments. The balance trial is a suitable method of examining the differences in retention of energy and nitrogen between treatments over a limited time period (Everts and Dekker, 1994a).

Few studies have been conducted examining the effect of feed intake on energy or nitrogen retention in pregnant gilts or sows. Researchers have compared retention values of pregnant and non-pregnant gilts (De Wilde, 1980a,b; Close *et al.*, 1985) and studied metabolism of pregnant gilts (Noblet *et al.*, 1985; Noblet and Etienne, 1987; Dunn and Speer, 1991; Everts and Dekker, 1994b) with the purpose of defining the extent of nutrient partitioning, tissue deposition and utilization changes during gestation and using this information to estimate nutrient requirements.

Energy retention

Noblet *et al.* (1990) have shown that more than 75% of energy intake is used to meet maintenance requirements of the pregnant sow. Requirements for uterine growth represents approximately 5% of energy needs. However, the daily requirements for uterine and mammary tissue development increase during pregnancy and are high during the last week

of gestation (17% and 14% of total requirements, respectively) (Noblet *et al.*, 1990). Due to the increase in energy requirements during late gestation, Noblet *et al.* (1990) suggest feeding more energy during this stage to avoid mobilization of body reserves and to increase maternal protein deposition.

Energy retention in the pregnant sow occurs mainly in the maternal body, with some retention in the reproductive tissues and products of conception (Noblet *et al.*, 1997). Energy retention decreases with the progression of gestation at constant feeding levels, due to the increase in metabolic body weight of the sow (Close *et al.*, 1985; Noblet and Etienne, 1987; Noblet *et al.*, 1990). Energy retained in reproductive tissue and total protein deposition increases with the advancement of pregnancy, while energy retention in maternal tissue decreases (Noblet and Etienne, 1987).

Close *et al.* (1985) fed pregnant gilts low (1.8 kg d^{-1}) or high (2.5 kg d^{-1}) intakes during gestation and measured energy and nitrogen balances using a calorimeter. Pregnant gilts retained more energy at the high intake level during early-, mid- and late gestation. Energy retention decreased with progression of pregnancy; gilts on the low energy intake were in negative energy balance in late gestation.

Nitrogen retention

As previously mentioned, nutrient supply during pregnancy must meet the needs for maintenance and development as well as additional nutrients for growth in younger sows and to compensate for losses during the previous lactation in multiparous sows (Pettigrew and Tokach, 1991).

Nitrogen (or protein) intake has little influence on nitrogen (N) deposition in the products of conception (De Wilde, 1980a; Walach-Janiak *et al.*, 1986), however maternal N deposition is responsive to nutrient intake (Speer, 1990). Therefore, maternal gains can be targeted to levels for maximum reproductive performance.

In early pregnancy, N retention is primarily maternal (Dourmad *et al.*, 1996). The increase in N retention observed during early- to mid-gestation is associated with an increase in N retention in maternal tissues, whereas N retention in late gestation is primarily related to conceptus and mammary gland development (Dourmad *et al.*, 1996). With continuation of pregnancy, a larger proportion of energy is retained as protein (Noblet *et al.*, 1990).

Protein retention measured in pregnant versus non-pregnant gilts using the comparative slaughter method found late gestation to be the main period of protein deposition (De Wilde, 1980a). Protein deposition was dependent on protein intake for pregnant and non-pregnant gilts and stage of gestation for pregnant gilts (Close *et al.*, 1985).

Dunn and Speer (1991) conducted N balance trials on gilts and found that the pattern of N retention increased over the course of gestation, and with an increase in N intake. Similar results have been reported in other studies (Willis and Maxwell, 1984; King and Brown, 1993; Everts and Dekker, 1994b; Noblet *et al.*, 1997). This is consistent with the observation that N accretion in reproductive tissues increases during mid- to late gestation to provide for fetal growth and development (Dunn and Speer, 1991; King and Brown, 1993). Noblet *et al.* (1990) found that N retention in the conceptus and mammary gland increase from 2 g d^{-1} in mid pregnancy to 14 g d^{-1} in late pregnancy. King and Brown (1994) suggest

that the increase in N retention in the gravid uterus results in maternal N retention remaining almost constant during gestation. This is reflected in decreased urinary N over the course of pregnancy (Jones and Maxwell, 1982; Dunn and Speer, 1991), whereas fecal N excretion was not affected in the study by Jones and Maxwell (1982) but increased with pregnancy in a study by Dunn and Speer (1991). Willis and Maxwell (1984) found that gilts fed higher energy diets retained more N when compared to gilts fed moderate energy diets. Dietary protein intake and protein reserves during pregnancy influence subsequent fertility and lactation performance (Head and Williams, 1991). In this way, optimizing tissue protein deposition during gestation may positively influence reproductive performance. Willis and Maxwell (1984) suggest that the level of protein required to maintain maternal protein reserves is higher than that required to support fetal development. In general, N deposition responds to protein levels in the diet with a typical dose response pattern (King and Brown, 1993). Nitrogen retention increases with protein intake until the protein requirement is reached, beyond which there is no further increase in N retention. King and Brown (1993) fed gilts from 1.1 to 3.1 kg d^{-1} to provide different energy levels, but similar N intake levels to investigate the influence of dietary energy on N retention. Urinary and fecal N excretion increased with the increases in energy intake. This result was observed throughout pregnancy. Everts and Dekker (1994b) found that sows retained 14 g N d^{-1} during mid pregnancy and approximately 25 g d^{-1} during late gestation at standard feeding levels. In general, maternal body tissue accretion can be expected to decline with pregnancy, while nutrient deposition in reproductive tissues increases (Shields, Jr. *et al.*, 1985).

Serum Urea Nitrogen

Urea is the primary nitrogen end product produced from the catabolism of amino acids (Chen *et al.*,1995). Serum urea nitrogen concentrations are inversely related to the net protein utilization of the diet (Eggum, 1970) and are dependent on factors such as protein quality of the diet, and protein and energy intake (Mosenthin *et al.*, 1992; Cai *et al.*, 1995).

An increase in urea nitrogen concentration indicates a deficiency, excess, or imbalance of amino acids (Lewis and Speer, 1974). Consumption of excess protein increases urea synthesis and excretion which is reflected in elevated serum urea concentration (Eggum, 1970). In conditions of insufficient protein intake, mobilization of labile body protein stores to support the demands of gestation is also reflected in increasing serum urea nitrogen concentration (Einarsson and Rojkittikhun, 1993). Low daily energy intake causes amino acids to be deaminated and oxidized to meet maintenance energy requirements in growing pigs (Cai *et al.*, 1995) and primiparous sows (Nelssen *et al.*, 1985). Increasing serum urea nitrogen concentration indicates protein catabolism in animals whose dietary intake has not changed (Hulten *et al.*, 1993). Increasing energy intakes means fewer amino acids are oxidized, and more are incorporated into body proteins until the energy requirement for maximum protein accretion is reached. Nelssen *et al.* (1985) found serum urea concentration to be inversely related to energy intake. Urea nitrogen concentration in the blood is positively related to the rate of urea synthesis and therefore inversely related to the efficiency of nitrogen deposition (Cai *et al.* 1995; Coma *et al.*, 1995).

Nutrition and Lactation

Long-term nutritional strategies of the sow should concentrate on lactation, with the objectives of conserving maternal body condition (Cole, 1990), and maximizing piglet growth (King and Dunkin, 1986) and subsequent reproductive performance of the sow (King and Williams, 1984a).

Nutrition during lactation must provide for maintenance, milk production (Noblet *et al.*, 1990) and maternal growth in the case of young sows (Aherne and Williams, 1992). Nutritional effects on reproductive performance are evident in primiparous sows (Trottier and Easter, 1995) because of their nutrient requirements for growth, and because their voluntary feed intake is usually lower than multiparous sows (Genest and D'Allaire, 1995).

Voluntary Feed Intake

Voluntary feed intake of the modern gilt and sow during lactation is often insufficient to meet lactational demands (Cole, 1990), including maintenance of body weight (Noblet *et al.*, 1990), growth (King and Williams, 1984a) and milk production (Mullan *et al.*, 1989; Noblet *et al.*, 1990; Aherne and Williams, 1992; Clowes *et al.*, 1998). Factors influencing feed intake during lactation include parity, lactation length, and feed intake during gestation.

Low levels of feed consumption post-partum result in mobilization of maternal body reserves (Noblet *et al.*, 1994, Weldon *et al.*, 1994a). Excess weight and backfat loss during lactation is associated with decreased milk production (O'Grady *et al.*, 1973) and increased

occurrence of reproductive problems including delayed return to estrus (Dourmad *et al.*, 1994) and reduced subsequent litter size (Kirkwood *et al.*, 1987b).

Relationship to Gestation Feed Intake

Gestation feeding level influences voluntary feed intake during lactation (Einarsson and Rojkittikhun, 1993) and may be linked to changes in body composition during gestation and lactation. Increased feed intake during gestation results in lower feed intake during lactation (Mullan and Williams, 1989; Noblet *et al.*, 1990; Coffey *et al.*, 1994; Weldon *et al.*, 1994a). The relationship between gestation and lactation feeding affects sow body condition by influencing the extent of tissue mobilization and weight loss during lactation. This effect is pronounced in early lactation and in primiparous sows (Noblet *et al.*, 1990; Aherne and Williams, 1992). The negative association between gestation and lactation feed intake may be due in part to a reduced appetite in lactation (Einarsson and Rojkittikhun, 1993) related to body reserves of lipid and protein (Aherne and Williams, 1992; Revell and Williams, 1993). Increased body reserves at farrowing have been associated with depressed lactation feed intake (Mullan and Williams, 1989; Revell and Williams, 1993).

Cromwell *et al.* (1989) found higher gestation weight gain in sows was connected to greater lactation weight loss when comparing primiparous and multiparous sows fed normal levels throughout gestation, to those supplemented with 1.36 kg d^{-1} extra feed from d 90 of gestation.

Weldon *et al.* (1994a) fed gilts at NRC (1988) recommended levels or *ad libitum* from d 50 of gestation; all were fed *ad libitum* during lactation. *Ad libitum* gilts consumed more feed during the last 40 d of gestation, resulting in reduced feed consumption during each week of lactation. However, when total feed intake was calculated for the last 40 d of gestation plus the 28-d lactation, feed intake did not differ between treatment groups.

Dourmad (1991) fed gilts at low, medium or high levels during pregnancy (1.8, 2.25 or 2.7 kg d^{-1}) and *ad libitum* during lactation. Total feed intake over the four-week lactation did not differ between groups. However, feed intake during the first three weeks of lactation was lower in the sows fed *ad libitum* during gestation.

Mechanisms Controlling Voluntary Feed Intake

A variety of mechanisms have been proposed as regulators of the interaction between gestation and lactation feed intake. These include the actions of hormones such as insulin, and long-term fluctuations in nutrient balance (Revell and Williams, 1993).

Reduced insulin secretion (Weldon *et al.*, 1994a), and the development of insulin resistance (Revell and Williams, 1993; Weldon *et al.*, 1994b; Xue *et al.*, 1997b) have been implicated in the reduction in feed intake in early lactation associated with high levels of feeding during gestation. Research has not shown consistent results.

Insulin is involved in the regulation of plasma glucose and fatty acid levels and the control of carbohydrate, fat and protein balance in the body. Insulin promotes glucose

utilization by many body tissues and inhibits lipolysis. The mammary gland uses a large amount of available plasma glucose to produce milk (Spincer *et al.*, 1969).

In early lactation high insulin levels may lead to the suppression of voluntary feed intake. Plasma insulin concentrations have been seen to rise, due to the development of insulin resistance, when animals and humans are accumulating adipose tissue (McCann *et al.*, 1986; McNeill *et al.*, 1991). Insulin resistance (or insensitivity) may be involved in the reduced voluntary feed intake observed in obese animals (Revell and Williams, 1993). Insulin resistance in adipose tissue leads to an increase in blood glucose level that result in increased insulin concentration and reduced voluntary feed intake (Revell and Williams, 1993).

Weldon *et al.* (1994b) propose a similar role for insulin in the control of feed intake. Sows fed at NRC (1988) levels during gestation consumed more feed during lactation than sows fed *ad libitum* during gestation. The authors suggest that increased insulin concentrations in early lactation in the sows fed at NRC levels during gestation may increase glucose utilization and reduce mobilization of stored nutrients, causing feed intake to increase to maintain blood glucose levels (Weldon *et al.*, 1994a,b). Sows fed *ad libitum* during gestation developed insulin resistance and glucose intolerance, resulting in limited utilization of peripheral glucose and increased mobilization of stored nutrients, thereby increasing the availability of substrates for oxidation and promoting a reduction in feed intake during lactation (Weldon *et al.*, 1994b).

Piglet Performance

Milk production accounts for approximately 75% of the total energy requirements of the sow during lactation (Noblet *et al.*, 1990). Noblet and Etienne (1986) found that milk production of primiparous sows increased with the advancement of lactation.

Results indicating an influence of sow feed intake during lactation on piglet performance are contradictory. Neil and Ogle (1996) report that sow feed intake during lactation has implications for piglet performance during the lactation period due to the impact of feed intake on milk production. Sow lactation feed intake positively influences milk production particularly as lactation progresses, with milk production peaking later during the lactation period (Patience, 1993). Therefore lactation feeding level can be expected to influence piglet growth during the latter part of lactation (Mullan and Williams, 1989).

Sow body condition is also known to influence milk production (Patience, 1993). Sows with greater backfat thickness and body weight during late gestation may be better prepared for high milk production in lactation because they are able to catabolize these reserves and transfer more energy and nutrients into milk (Neil and Ogle, 1996).

Gilts were fed one of three gestation-lactation treatment combinations: a conventional gestation diet at 2.2 kg d^{-1} and restricted to a maximum of 7.0 kg d^{-1} during lactation, a simplified gestation diet and conventional diet *ad libitum* during lactation, or a conventional gestation diet and *ad libitum* during lactation (Neil and Ogle, 1996). Gestation treatment did not affect piglet birth weights. However, gestation-lactation treatment combination did influence piglet weaning weights. Piglets from sows fed the conventional-*ad libitum* diet

were heavier at weaning at five weeks of age than the other two treatments. Feed intake of primiparous sows fed one of six feed intake levels ranging from 1.5 to 4.8 kg d^{-1} during a 28-d lactation resulted in differences in piglet growth rate during the last week of lactation (King and Dunkin, 1986). In contrast, Kirkwood *et al.* (1987a) found that feeding level of second parity sows during lactation (3 kg d^{-1} versus 6 kg d^{-1}) produced no differences in piglet weaning weights. Pluske *et al.* (1998) fed primiparous sows *ad libitum*, restricted to 50% of *ad libitum* intake (3.0 kg d^{-1}), or superalimented to 125% of *ad libitum* intake during a 28-d lactation. Milk yield and litter weaning weights did not differ among treatments as measured during mid- and late lactation.

Verstegen *et al.* (1985) fed second parity sows at low (2.5 to 2.6 kg d^{-1}) or high (4.8 to 6.0 kg d^{-1}) feeding levels during lactation. The low feeding level was given to supply energy slightly above maintenance, with the sow mobilizing body reserves to supply the energy needed for milk production. The high level of feeding supplied sufficient energy for maintenance and milk production. Piglet weight gain after d 10 of lactation was affected by feeding level of the sow, with high level sows having heavier piglets.

Nutrition required during lactation to maximize lactational performance (ie. milk production, piglet growth) is less than that required to minimize body weight loss of the sow (King *et al.*, 1993). Lactational performance can be maximized even if sow feed intake during lactation is below the total requirements for milk production because of the contribution of mobilized body reserves (Clowes *et al.*, 1998). If dietary restriction during lactation is severe, and the sow cannot provide sufficient nutrients to supplement the dietary deficit by

catabolizing body tissues, milk production will decline (Whittemore *et al.*, 1988; Mullan and Williams, 1989). Restricted lactation feed intake reduced average piglet growth rate and weaning weights (Mullan and Williams, 1989). Interestingly, primiparous sows did not partition additional nutrients towards increased milk production but rather toward storage in maternal reserves in studies by Clowes *et al.* (1998) and Pluske *et al.* (1998). In this way, provision of excess nutrients during lactation did not improve piglet performance, but may have implications for sow metabolic condition and subsequent reproduction.

Metabolic Indicators of Reproductive Status: Lactation

A variety of factors have been proposed as predictors of subsequent sow reproductive performance, including weight and backfat loss, protein and lipid loss, and body composition of the sow at parturition and at weaning. However, as mentioned previously, nutrition can induce short- and long-term changes in the reproductive axis in the absence of changes in body composition (Booth, 1990; Beltranena *et al.*, 1991). Determination of the changing metabolic status of the sow throughout her reproductive lifetime is more likely to provide the link to reproductive performance (Foxcroft, 1992).

Sow Weight and Backfat

During late gestation or early lactation the sow often becomes catabolic, mobilizing both protein and fat reserves to support fetal growth and milk production (Aherne and Williams, 1992; Pluske *et al.*, 1998). Excess weight and backfat loss during lactation influences subsequent reproductive performance (King and Williams, 1984a).

Aherne and Williams (1992) suggest that the amount of body reserves at farrowing and weaning, rather than the amount of tissue catabolized during lactation, influence the reproductive performance of the sow. Weight and backfat gain during gestation is positively related to the level of weight and backfat depletion during lactation (Mullan and Williams, 1989).

Clowes *et al.* (1998) fed primiparous sows *ad libitum*, restricted to 55% of *ad libitum* feed intake, or super-alimented to 125% of *ad libitum* feed intake during lactation. Restricted-fed sows were able to maintain milk production levels similar to the other treatments by mobilization of body protein and lipid reserves. The superalimented sows did not mobilize body protein reserves, and partitioned the additional nutrients toward maternal reserves rather than milk production. In general, feed restriction does not cause an initial decrease in milk production as sows are able to mobilize their protein reserves to maintain a satisfactory level of milk production during early lactation. In mid- to late lactation, restricted sows had reduced litter growth rates compared to *ad libitum* sows because they had mobilized a large percentage of maternal protein by mid- to late lactation. Lactating sows can mobilize up to 25% to 30% of their protein reserves (Mullan and Williams, 1990), however

lactational performance may be reduced when more than half of this protein reserve is catabolized (Clowes, 1998). King *et al.* (1993) state that the lactating sow requires more protein intake to maximize N balance than to maximize lactational performance. Therefore, sows fed sufficient nutrients to maintain a high level of milk production may still lose body condition during lactation (Foxcroft *et al.*, 1995).

The long-term reproductive performance of sows is met by minimizing lactation weight and backfat loss in order to limit the gain required to restore weight in the subsequent pregnancy (Einarsson and Rojkittikhun, 1993). Aherne and Williams (1992) suggest that sow weight loss is kept below 10 kg over the course of lactation. Young *et al.* (1991) propose that primiparous and second parity sows gain 16.8 and 13.4 kg, respectively, during lactation to prevent backfat loss.

King and Dunkin (1986) compared the reproductive performance of primiparous sows assigned to one of six feed intake levels (1.5, 2.2, 2.9, 3.6, 4.2 and 4.8 kg d^{-1}) during a 28-day lactation. Weight and backfat loss increased as lactation feed intake decreased, with weight and backfat loss varying from 9.0 to 44.5 kg and 4.0 to 8.9 mm, respectively, during the course of lactation over the range of high to low feed intakes.

King and Williams (1984a) fed primiparous sows *ad libitum* or restricted (2 kg d^{-1}) during lactation. Restricted-fed sows lost more weight and backfat during lactation, and these differences continued through the subsequent gestation.

Dourmad (1991) found gilt body weight and backfat levels during gestation increased in response to feed intake. Gilts were divided into three gestation treatments (1.8, 2.25, 2.7

kg d^{-1}) and were fed *ad libitum* during lactation. A positive relationship was demonstrated between weight gain during gestation, and lactation weight loss. Backfat loss during the lactation period was not affected by gestation treatment.

Mullan and Williams (1989) fed gilts one of three levels during gestation until parturition, 1.5, 2.0, or 3.0 kg d^{-1} (low, medium, and high, respectively) to achieve different levels of body weight and backfat at farrowing. During lactation gilts were either restricted (2.0 kg d^{-1}) or *ad libitum* fed. Weight and backfat changes during lactation were positively related to feed intake during the lactation period. Gilts given the high level of feed during gestation, and *ad libitum* access to feed during lactation, had the greatest lactational weight and backfat loss. This further demonstrates the inverse relationship between maternal weight gain during gestation and weight loss during lactation.

Body Composition

Changes in body composition during lactation can influence subsequent reproductive performance (Cole, 1990; Dourmad, 1991). Metabolic changes occurring during lactation have traditionally been assessed using sow weight and backfat (Cole, 1990), and the results were assumed to parallel changes in sow body composition. However, subsequent research confirms the limitations associated with the use of body weight measures as an indicator of nutritional alterations in body composition (Mullan and Williams, 1989). The relationship between body weight measures and fat reserves is unreliable as sows can gain weight and lose fat simultaneously (Whittemore *et al.*, 1980; Cole, 1990). Therefore, body weight at

parturition or body weight changes during pregnancy and lactation are not always good indicators of body fat reserves (Dourmad, 1991). Whittemore and Yang (1989) were able to predict body composition (lipid and protein content) of gilts using P2 backfat measurements and weight. Prediction of the absolute levels, and changes in the relative proportions, of protein and lipid stores in the maternal body over the course of lactation provide information on the influence of nutrition on metabolism.

Past nutritional recommendations for the lactating sow have been calculated based on the assumption that most of the weight loss during lactation represented the catabolism of fat reserves for milk production (Mullan and Williams, 1990). However, mobilization of both protein and lipid reserves account for a proportion of the total weight loss (Armstrong *et al.*, 1986; Whittemore and Yang, 1989; Mullan and Williams, 1990) and may be dependent on protein or energy intake during lactation and milk production of the sow (Mullan, 1991).

Sow body fat loss during lactation and resultant body weight at weaning, are influenced by subcutaneous fat depth at farrowing, lactation feeding level and litter size (Whittemore and Yang, 1989). Whittemore and Yang (1989) found the relationship between total body weight and body protein to be relatively resistant to change, whereas the relationship between body weight and body lipid is readily modified by the factors mentioned above.

Mobilization of maternal fat reserves during lactation is reflected in weight and backfat loss (Einarsson and Rojkittikhun, 1993), and is influenced by body weight and backfat thickness at farrowing, litter size, litter weight gain, and lactation feeding. However, the rate

of mobilization varies among individual sows fed at similar levels and supporting the same litter size (Einarsson and Rojkittikhun, 1993; Neil *et al.*, 1996).

Loss of body protein and lipid as influenced by nutrition during lactation may be responsible for decreased reproductive performance, including a positive relationship in primiparous sows between the level of protein loss during lactation (as a percent of total body protein at farrowing) and the weaning-to-estrus interval (WEI).

Mullan (1991) suggests that body fat at the start of lactation influences feed intake during lactation, and that an elevation above a critical level of body fat (approximately one third of body weight) is responsible for the decrease in voluntary feed intake observed in lactation.

Breeding sows may have a biological drive to attain a certain body protein level (Foxcroft *et al.*, 1995; Clowes *et al.*, 1998). Limited protein supply during gestation and protein losses due to tissue mobilization during lactation may impair sow reproductive performance (Foxcroft *et al.*, 1995). The drive for protein accretion would be expected to be higher for young sows, because they partition more nutrients towards maternal tissue accretion than multiparous sows (Clowes *et al.*, 1998).

Serum Urea Nitrogen

Similar responses in blood urea nitrogen concentration to dietary manipulation occur during lactation as discussed for gestation. Several studies have used serum urea nitrogen as a measure of the extent of protein mobilization (Coma *et al.*, 1996), assuming that the

concentration of urea in the serum is positively correlated to amino acid breakdown. During lactation, Nelssen *et al.* (1985) found that sows use amino acids as an energy source when their energy intake is restricted. This type of amino acid utilization results in deamination of the amino acids and subsequent urea synthesis in the liver (Nelssen *et al.*, 1985). Therefore, serum urea concentration is an indicator of amino acid degradation. Nelssen *et al.* (1985) found that serum urea nitrogen concentration increased from late gestation through mid- to late lactation (d 14) in primiparous sows, and decreased during late lactation (d 28). Brendemuhl *et al.* (1987) found serum urea nitrogen concentrations of lactating primiparous sows positively related to the level of protein intake and negatively related to the level of energy intake. These results agree with other research using primiparous sows (Nelssen *et al.*, 1985) and growing pigs (Cai *et al.*, 1995).

Nutrition and Subsequent Reproductive Performance

Nutrition during gestation and lactation influences the subsequent reproductive performance of the sow, including the WEI (Mullan and Williams, 1989; Whittemore and Yang, 1989; Dourmad, 1991), embryo survival (Aherne and Williams, 1992) and subsequent litter size (Kirkwood *et al.*, 1987b). Feed intake during lactation is positively related to lactation performance and subsequent reproductive performance (Koketsu *et al.*, 1996b). Mechanisms linking feed intake, body condition, metabolic status and reproductive performance may be controlled by changes associated with reduced feed intake and increased

body tissue mobilization, such as modified secretion of gonadotropins and metabolic hormones. The influence of lactation feed intake on post-weaning reproductive performance may be mediated indirectly by the hypothalamic-pituitary-ovarian axis, or through direct ovarian effects (Foxcroft *et al.*, 1995).

Lactational Anestrus

Lactation in the sow is recognized as a period of anestrus (Kirkwood *et al.*, 1987a; De Rensis *et al.*, 1993). The suckling stimulus provided by the piglets appears to be the primary factor involved in the suppression of reproductive activity during lactation (Varley and Foxcroft, 1990; De Rensis *et al.*, 1993; Foxcroft *et al.*, 1995). Other factors, such as nutritional influences on the metabolic state of the sow, and the length of the lactation period, may further suppress reproductive activity. As lactation progresses there is a gradual escape from inhibition (Varley, 1982; Kirkwood *et al.*, 1987a), perhaps due to a decreased suckling intensity (Varley and Foxcroft, 1990; Cosgrove *et al.*, 1997).

Suckling-mediated inhibition of gonadotropins

The neuroendocrine reflex stimulated by suckling suppresses GnRH/LH secretion, follicular development and estrus (Stevenson *et al.*, 1981; Foxcroft *et al.*, 1987; Varley and Foxcroft, 1990; De Rensis *et al.*, 1993; Sesti and Britt, 1993a; Cosgrove *et al.*, 1997). The reduction in gonadotropin secretion during lactation may be related to low secretion of GnRH, perhaps in combination with decreased pituitary sensitivity to GnRH (Cosgrove *et al.*, 1997). Suppression of LH secretion in primiparous sows was greater on d 7 of lactation than

in early lactation, however LH synthesis continued to occur during this time period (Tokach *et al.*, 1992b; Sesti and Britt, 1993b). Gonadotropin-releasing hormone levels, although suppressed by suckling, were sufficient to promote LH synthesis, but not LH release, resulting in accumulating pituitary stores of LH during early lactation. These pituitary stores provide readily releasable pools of LH (Sesti and Britt, 1993b). As lactation progresses there is an increase in synthesis and release of GnRH, inducing a gradual increase in basal gonadotropin secretion and follicular development (Sesti and Britt, 1993b) .

Weaning-to-Estrus Interval

The length of the interval required for return to breeding condition is an important factor determining sow productivity. Primiparous sows return to estrus as late as 7 to 10 d post-weaning (Carroll *et al.*, 1996). Studies reviewed by Sterning *et al.* (1990) show that a large proportion of primiparous sows return to estrus later than 10 d post-weaning. A number of factors influence return to estrus after weaning including, parity, lactation length, and nutrition during lactation (King and Williams, 1984b; Sterning *et al.*, 1990; Cole, 1990; Zak *et al.*, 1997a). The length of the WEI also shows a high degree of variability in the primiparous sow (Cosgrove *et al.*, 1997).

Luteinizing hormone secretion is a critical component in the resumption of estrus post-weaning (Koketsu *et al.*, 1996b). Studies have demonstrated that LH levels prior to weaning were related inversely to the WEI (Tokach *et al.*, 1992b; Xue *et al.*, 1997b). Armstrong *et al.* (1986) confirmed that primiparous sows with the highest LH pulse

frequency before weaning had the quickest return to estrus. Tokach *et al.* (1992b) found that mean LH concentrations and LH pulsatility from d 14 of lactation onwards were greater in sows that exhibited an early return to estrus (less than 9 d).

During early lactation, LH secretion continues in an active manner (Cosgrove *et al.*, 1997). By approximately d 2 to d 3 of lactation, the suckling stimulus inhibits LH secretion (Varley and Foxcroft, 1990; De Rensis *et al.*, 1993) by blocking the pulsatile release of GnRH from the hypothalamus (Foxcroft *et al.*, 1995; Cosgrove *et al.*, 1997). This results in suppression of ovarian follicular development in early lactation (Cosgrove *et al.*, 1997), and lactational anestrus (De Rensis *et al.*, 1993; Sesti and Britt, 1993a). It is the maturation of the follicles and their production of estrogen that defines the length of the weaning-to-estrus interval (Cosgrove *et al.*, 1997). Sows with shorter lactation lengths will be weaned at a time when the reproductive axis is suppressed, resulting in longer WEI (Cosgrove *et al*, 1997). However, as lactation progresses the secretion of gonadotropins increases, resulting in an increased number of medium to large follicles and serum concentration of estrogen (Sesti and Britt, 1993b).

Nutrition during lactation is related to gonadotropin secretion and the WEI (King and Williams, 1984b; Tokach *et al.*, 1992b; Einarsson and Rojkittikhun, 1993; Koketsu *et al.*, 1996b). Restriction of feed intake during lactation extended the WEI of primiparous sows in studies by King and Williams (1984a,b). Primiparous sows returning to estrus within 7 d consumed more energy during a 3-wk lactation period than those returning later than 7 d (Koketsu *et al.*, 1996b). These sows also lost less backfat during the lactation period than

sows which exhibited a delayed return to estrus. King and Dunkin (1986) observed decreased time from weaning to estrus with increased feed intake during lactation in primiparous sows. Kirkwood *et al.* (1987a) found that sows fed 3 kg d^{-1} took longer to return to estrus than sows fed 6 kg d^{-1} over the course of a 35-d lactation. The observation that feed restriction during lactation results in an extended WEI may be linked to changes in body composition over the course of lactation (Cole, 1990; Dourmad, 1991; Aherne and Williams, 1992; Einarsson and Rojkittikhun, 1993) related to the degree of tissue mobilization and the stage of lactation during which it occurs (Zak *et al.*, 1997a). In particular, loss of body lipid and protein reserves during lactation (Pettigrew and Tokach, 1991), as well as absolute levels at weaning are important (Dourmad *et al.*, 1994). Sow nutrition during lactation and post-weaning influences the rate of weight and/or backfat depletion (King and Williams, 1984a; Kirkwood *et al.*, 1987a; Carroll *et al.*, 1996; Xue *et al.*, 1997b). First parity sows exhibited extended WEI as influenced by subcutaneous fat depth at parturition, lactation feed intake and fat depth at weaning (Whittemore and Yang, 1989).

Nutrition during lactation has been correlated with altered patterns of LH secretion in the primiparous sow. Shaw and Foxcroft (1985) found a negative relationship between mean LH levels prior to weaning and the length of the WEI. They also showed that diet had no effect on plasma LH, when comparing sows which were fed *ad libitum* versus restricted. Zak *et al.* (1997a) fed primiparous sows *ad libitum* throughout a 28-d lactation, *ad libitum* to d 21 and restricted to 50% of *ad libitum* intake during the last week, or restricted until d 21 and *ad libitum* thereafter. Feed restriction during late lactation suppressed LH

concentration at d 28 more than feed restriction during early lactation. The WEI was extended in sows restricted in feed intake during lactation compared to those fed *ad libitum* throughout. Koketsu *et al.* (1996b) found a correlation between feed restriction during any week, as well as throughout a 21-d lactation, and reduced LH pulsatility, and this was associated with an increase in the duration of the WEI. Restricted nutrient or energy intake during lactation may influence the releasable pools of LH or the hypothalamic pulse generator (Armstrong and Britt, 1987).

The relationship between nutrition, gonadotropin secretion, and length of the WEI may be mediated by changes in metabolic status (Koketsu *et al.*, 1996b). Feed restriction during lactation suppresses plasma insulin concentration (Zak *et al.*, 1997a). Serum insulin concentrations were higher during early- and mid-lactation in primiparous sows that returned to estrus earlier (Tokach *et al.*, 1992b). Insulin concentration in mid-lactation was correlated with LH pulsatility (Tokach *et al.*, 1992b; Koketsu *et al.*, 1996b). These results suggest a possible role for insulin in nutritional effects on reproduction.

Follicular development and ovulation rate

Nutrition during late gestation (Cosgrove *et al.*, 1997) and lactation can influence the size of the follicles in the preovulatory pool, and the rate of oocyte maturation (Zak *et al.*, 1997b). The progression of lactation and increase in gonadotropin secretion stimulates growth of medium-sized (5 to 8mm) follicles in the ovary (Cosgrove *et al*, 1997). Changes in feed intake or metabolic status during late gestation or lactation may impact subsequent preovulatory follicles, particularly in early weaned sows (Foxcroft *et al.* 1995; Cosgrove *et*

al., 1997; Zak *et al.*, 1997b). This concept is known as follicular imprinting (Foxcroft *et al.*, 1995; Zak *et al*, 1997b). Nutritional changes imposed during the preovulatory period may promote variablity in follicular development (follicular heterogeneity) (Foxcroft *et al.*, 1995; Zak *et al.*, 1997b), having implications for oocyte maturation and embryo survival.

Zak *et al.* (1997a) observed a decrease in ovulation rate in primiparous sows feed-restricted for a 7-d period during early- or late lactation when compared to sows on full feed. The period of lactation when feed restriction was imposed did not influence ovulation rate. Previous studies reported no effect of nutrition during lactation on ovulation rate (King and Williams 1984b).

Current Feeding Systems

Gestation

Conventional gestation feeding systems provided gilts or sows with a fixed amount of feed throughout pregnancy to avoid a drop in feed intake in early lactation (Patience, 1993). This type of feeding system may not account for the individual feed requirements of sows. As a result, sows can become over- or under-conditioned with the advancement of pregnancy, resulting in declining body reserves with progressing parities (Patience, 1993).

Lactation

Traditionally, lactating sows have not been given *ad libitum* access to feed immediately following parturition (Moser *et al.*, 1987). Intake is often restricted just prior to farrowing and increased gradually during the first few days of the lactation period to achieve *ad libitum* intake (Neil, 1996). This early lactation feed restriction has been implemented to decrease the occurrence of lactation failure (Moser *et al.*, 1987). However, the restriction in early lactation reduces the total feed intake during lactation (Moser *et al.*, 1987) and may have negative influences on sow body condition and subsequent reproductive performance (Patience, 1993).

Pattern of Feed Intake

Few studies have been conducted to examine the effect of imposed pattern of feed intake on sow reproductive performance. Previous research examining the influence of feed intake pattern has evaluated only one stage of the reproductive cycle at a time (Coffey *et al.*, 1994). Interactions among the stages of the production cycle are an important consideration. Feed intake during gestation affects voluntary feed intake during lactation, and lactation intake influences feed required in the subsequent pregnancy to maximize reproductive performance (Dourmad, 1991).

Gestation

Verstegen *et al.* (1987) suggested a feeding strategy adjusted to the changing requirements of pregnancy. Feed requirements were calculated for various stages of pregnancy (d 0, d 30, d 60, d 90, and d 110) based on estimated sow weight, and gain in reproductive tissue in order to quantify the feed requirements during the various stages of gestation. Subsequently, a review of early findings by Cole (1990) stated that total feed intake in gestation is more important than pattern of feed intake.

Lactation

Neil (1996) varied the timing of introduction of *ad libitum* feeding during lactation to examine the influences on feed intake, and sow and piglet performance. *Ad libitum* feeding was introduced before farrowing (d 111 of gestation), on the day of farrowing, or 3 d after farrowing and continued to the end of the 35-d lactation. Sows provided *ad libitum* access to feed before or on the day of farrowing had higher total daily feed intake over the course of lactation. Treatment did not influence sow body composition or piglet performance. The author concluded that there is no benefit realized from delaying *ad libitum* feed intake until after farrowing. Moser *et al.* (1987) found no negative influence of *ad libitum* feeding imposed from the day of farrowing, on sow or litter performance when compared to restricted feeding during early lactation.

Altering the pattern of feed intake during lactation has been investigated in relation to sow and piglet performance during the lactation period, and subsequent sow reproductive

performance. Koketsu *et al.* (1996b) assigned primiparous sows to one of five feed intake patterns during lactation: high or low energy intake throughout a 3-wk lactation, or reduced intake during week 1, 2, or 3 of lactation, and examined the effects on reproductive performance. Restriction of energy intake in lactation influenced sow reproductive performance, backfat loss, and piglet performance depending upon the period of energy restriction. Zak *et al.* (1997a) fed primiparous sows to appetite from d 1 to d 28 of lactation, restricted to 50% of *ad libitum* intake from d 22 to d 28, or restricted from d 1 to d 21 of lactation. Feed restriction influenced body weight and backfat loss, and plasma metabolite and gonadotropin secretion, and WEI.

There is a shortage of data examining the connection between feed intake and reproduction in consecutive stages of the reproductive cycle of the young sow. The objective of these studies was to alter the feed intake pattern of gilts and first parity sows to reflect the changing maternal and piglet requirements during gestation and lactation, and to determine the influence of these altered feed intake patterns on reproductive performance.

CHAPTER 3

MANUSCRIPT I

REPRODUCTIVE PERFORMANCE OF EARLY-WEANED GILTS AND FIRST PARITY SOWS FED DIFFERING PATTERNS OF FEED INTAKE DURING GESTATION AND LACTATION

ABSTRACT

To study the effects of modified feed intake patterns during gestation and lactation on reproductive performance, 60 National Pig Development (NPD) gilts and 53 first-parity sows were randomly assigned to one of two gestation treatments and one of two lactation treatments. Throughout gestation, control (gC) gilts (n=31) and sows (n=26) were fed at 1.4 times maintenance d^{-1} and the pattern group (gP) (gilts, n=29; sows, n=27) was fed in four stages according to body weight on d 0, d 30, d 60, and d 90 of gestation. Each gestation group was further divided into two treatments for the 17-day lactation: control gilts and sows (lc) (gilts, n=30; sows, n=28) were full-fed, and the pattern group (lp; gilts, n=30; sows, n=25) was fed in three stages based on body weight at d 1, d 6, and d 12.

Average daily feed intake and total feed intake during gestation did not differ between groups ($P>0.05$). The gC group consumed more feed in early gestation and less feed in late gestation compared to treatment gP ($P<0.05$). Although pattern of body weight change was different during gestation there were no differences in body weight between treatment groups by d 109 of pregnancy. Weight gain of first parity sows followed the administered patterns of feed intake, while weight gain was lower throughout gestation for gP gilts ($P<0.05$). Gestation treatment had no effect on serum urea nitrogen and progesterone ($P>0.05$).

ADFI and total feed intake were lower for the lc treatment during lactation ($P<0.05$). Lactation treatment p had greater ADFI from d 7 of lactation onward ($P<0.05$). A larger difference between lactation treatments existed for first parity sows throughout lactation, while total feed intake of gilts was similar between treatments ($P<0.05$).

Backfat loss was greater for gP gilts and sows, while the gC group lost more body protein during lactation ($P<0.05$). However, there were no differences in backfat depth or body protein levels on d 17 of lactation due to gestation treatment. Lactation treatment p had higher weight, predicted body protein and lipid contents during lactation ($P<0.05$). Gestation-lactation treatment combination Cc lost more backfat and body lipid during lactation, and had the lowest reserves at weaning. The combination of Cp maintained a consistent level of backfat and lost the smallest amount of body lipid during lactation. Treatment combinations Pc and Pp resulted in backfat and lipid losses similar to treatment Cc during lactation.

Litter size born alive and litter size at weaning was larger for gP gilts, while these variables did not differ between treatments for first parity sows ($P<0.05$). Lactation treatment c gilts weaned larger litters, whereas lactation treatment did not influence litter size at weaning for first parity sows.

Lactation treatment p and the combination of Cp feeding patterns for gilts extended the WEI relative to first parity sows.

Overall, the administered patterns of feed intake during gestation and lactation did not result in consistent and similar improvements in reproductive performance of gilts and first parity sows.

INTRODUCTION

Appropriate nutrition during gestation and lactation is necessary for optimum reproductive performance. Young sows frequently display poor reproductive performance early in their lifetime because the additional requirement for growth to mature size makes them particularly sensitive to the effects of inadequate nutrition during the reproductive cycle (Dourmad *et al.*, 1994).

Interrelationships exist between successive stages of the reproductive cycle. For example, feed intake during gestation and its influence on maternal body composition affects voluntary feed intake, sow and litter performance during lactation and subsequent sow reproductive performance (Coffey *et al.*, 1994).

Current sow feeding programs generally do not account for differences in individual requirements. Gestation feeding level and voluntary feed intake of young sows during lactation are often insufficient to maintain maternal body weight, fetal growth, and milk production, as well as provide additional nutrients for maternal growth (Verstegen *et al.*, 1987; Aherne and Williams, 1992). Inadequate feed intake during lactation affects body lipid and protein reserves, and is associated with reduced litter growth during lactation (Brendemuhl *et al.*, 1989), prolonged WEI (Dourmad *et al.*, 1994), and reduced subsequent litter size (Kirkwood *et al.*, 1987b). Previous research has shown the need for refinement of feeding practices during gestation and lactation to more closely reflect the requirements of the growing sow.

The objective of this experiment was to assess the effects of feed intake patterns during gestation and lactation on the reproductive performance of gilts and first parity sows in a commercial farrow-to-wean facility.

MATERIALS AND METHODS

Experimental Design

Experiment 1 was performed at Kelly Farms, a commercial farrow-to-wean operation, near New Bothwell, MB. One hundred and thirteen National Pig Development gilts and sows (60 gilts, 200 days of age, and 53 first parity sows) were used during gestation and lactation. Animals were randomly assigned to one of two gestation treatments based on initial (d 1) body weight. At farrowing, each gestation treatment group was further subdivided, and animals within each group and parity were randomly assigned to one of two lactation treatments based on post-farrowing weight. The length of the lactation period was 17 days.

Animal Housing: Gestation

Animals were housed throughout the gestation period in individual gestation crates (0.61 m x 2.1 m). All animals were located in one large room (17.7 m x 39.3 m). Feed and water during gestation was provided in a trough along the front of the gestation crates. Water was present in the trough when feed was dropped, and was provided twice after all feed was consumed. Lighting during breeding and gestation was 16 hours of light and 8 hours of dark. Room temperature was set at 20°C.

Experimental Treatments: Gestation

Gilts were bred three times, twice by artificial insemination and once by natural mating at their second estrus. First parity sows were bred twice by natural mating. The day following the final insemination was designated as d 1 of gestation.

A 13.5% crude protein, barley-based, pelleted commercial dry sow ration (Landmark Feeds) was fed to gilts and sows from the first day of gestation (d 1) to the day of farrowing (Table 1). Feeding was done by automatic drop feeders once daily at 0700 h.

Gestation treatments differed in the assigned pattern of feed intake of the gilts and sows. Treatment 1 (Control) (gC) animals were fed at 1% of their body weight plus 0.7 kilograms (kg) of feed (Aherne, 1992) which is approximately 1.4 times their maintenance requirement, throughout gestation. Feed intakes were adjusted for changes in body weight at the end of each stage (d 30, d 60, d 90) to maintain feed intake at a constant proportion of body weight (Table 1). The control treatment was designed to meet NRC (1988) requirements for gestating sows.

Treatment 2 (Pattern) (gP) animals were fed in four increments during gestation with each stage being adjusted for body weight (Table 1). Treatment 2 was designed to provide the same average feed intake over gestation as Treatment 1 (1.4 times maintenance). The stages were designed as follows: Stage I: 1.1 times maintenance (d 1 to d 30 of gestation), Stage II: 1.3 times maintenance (d 31 to d 60 of gestation), Stage III: 1.5 times maintenance (d 61 to d 90 of gestation), and Stage IV: 1.7 times maintenance (d 91 to farrow).

The first two stages were designed to fall below NRC (1988) feed intake requirements for gestation. The final two stages exceeded NRC (1988) requirements.

Day one body weights were used to calculate gestation feed intakes for the first stage (d 1 to d 30). All gilts and sows were weighed at the end of each stage and these weights were used to determine feed intake for the subsequent stage. Maintenance requirements for gestation were calculated using Equation 1.

$$\text{Equation 1: Feed intake (maintenance)} = \frac{\text{Metabolic BW} * 461 \text{ kJ kg}^{-0.75}}{\text{DE content of the diet} * 4.18 \text{ kJ kcal}^{-1}}$$

Maintenance intakes were calculated using metabolic body weight (body weight $(kg)^{0.75}$), a maintenance energy allowance of 461 kJ of digestible energy (DE) $kg^{-0.75}$ (Jindal *et al.*, 1996), and the digestible energy content of the diet. This maintenance requirement was then multiplied by the corresponding factor for each stage to calculate feed intake. To convert metabolizable energy (ME) of the diets to DE, a factor of ME = 0.95DE was used. Gestation feed intakes were calculated using body weight rounded up to the nearest decimal place and grouped within a two kg weight range. The animals received their assigned feed intake for that entire gestational stage. Any feed not consumed by the following morning feeding was weighed back and recorded.

Table 1. Calculated gestation treatment feed intake levels based on maintenance feed intake requirements.

			GESTATION TREATMENT				
			Control (gC)	Pattern (gP)			
Body Weight (kg)	Metabolic BWt. (kg)	Maintenance feed rqt. (kg)	(d1to farrow) 1%BW+0.7kg (kg)	(d1 to d30) 1.1*M (kg)	(d31 to d60) 1.3*M (kg)	(d61 to d90) 1.5*M (kg)	(d91 to farrow) 1.7*M (kg)
100	31.62	1.20	1.69	1.33	1.57	1.81	2.05
102	32.10	1.22	1.71	1.35	1.59	1.83	2.08
104	32.57	1.24	1.74	1.36	1.61	1.86	2.11
106	33.04	1.26	1.76	1.38	1.64	1.89	2.14
108	33.50	1.28	1.79	1.40	1.66	1.91	2.17
110	33.97	1.29	1.81	1.42	1.68	1.94	2.20
112	34.43	1.31	1.84	1.44	1.71	1.97	2.23
114	34.89	1.33	1.86	1.46	1.73	1.99	2.26
116	35.35	1.35	1.89	1.48	1.75	2.02	2.29
118	35.80	1.36	1.91	1.50	1.77	2.05	2.32
120	36.26	1.38	1.93	1.52	1.80	2.07	2.35
122	36.71	1.40	1.96	1.54	1.82	2.10	2.38
124	37.16	1.42	1.98	1.56	1.84	2.12	2.41
126	37.61	1.43	2.01	1.58	1.86	2.15	2.44
128	38.10	1.45	2.03	1.60	1.89	2.18	2.47
130	38.50	1.47	2.05	1.61	1.91	2.20	2.49
132	38.94	1.48	2.08	1.63	1.93	2.23	2.52
134	39.38	1.50	2.10	1.65	1.95	2.25	2.55
136	39.82	1.52	2.12	1.67	1.97	2.28	2.58
138	40.26	1.53	2.15	1.69	1.99	2.30	2.61
140	40.70	1.55	2.17	1.71	2.02	2.33	2.64
142	41.14	1.57	2.19	1.72	2.04	2.35	2.66
144	41.57	1.58	2.22	1.74	2.06	2.38	2.69
146	42.00	1.60	2.24	1.76	2.08	2.40	2.72
148	42.43	1.62	2.26	1.78	2.10	2.42	2.75
150	42.86	1.63	2.29	1.80	2.12	2.45	2.78
152	43.29	1.65	2.31	1.81	2.14	2.47	2.80
154	43.72	1.67	2.33	1.83	2.17	2.50	2.83
156	44.14	1.68	2.35	1.85	2.19	2.52	2.86
158	44.56	1.70	2.38	1.87	2.21	2.55	2.89
160	44.99	1.71	2.40	1.89	2.23	2.57	2.91
162	45.41	1.73	2.42	1.90	2.25	2.59	2.94
164	45.83	1.75	2.44	1.92	2.27	2.62	2.97
166	46.25	1.76	2.47	1.94	2.29	2.64	3.00
168	46.66	1.78	2.49	1.96	2.31	2.67	3.02

Production Data: Gestation

All gilts and sows were weighed and had P2 backfat measurements taken (Scanmatic SM-1, Medimatic, Denmark) at the last rib, 6.5 cm from the midline, on d 1 of gestation and at the end of each stage (d 30, d 60, d 90 and d 109). Ultrasound (Preg-Tone®, Renco Corporation, Minneapolis, MN) was used to confirm pregnancy on d 35 and d 56 of gestation.

Blood Sample Collection: Gestation

Single blood samples were taken from a subsample of 20 gilts and 29 first parity sows four hours after feeding (1100 h) on d 30, d 60, d 90, and d 109. Animals were restrained using a wire nose snare. Blood samples were obtained from the jugular vein using 20-gauge, 1½ inch single-sample needles (Vacutainer, Becton Dickinson and Company, Franklin Lakes, NJ) and collected into 10 ml Vacutainer tubes for serum collection (Vacutainer, Franklin Lakes, NJ). Blood samples were stored overnight at 5°C. The following morning the samples were centrifuged at 1500 g for 30 min (CR3000, Jouan Inc., Winchester, VA) and the serum was separated, placed in glass vials, and frozen at -20°C until analysis.

Animal Housing: Lactation

Gilts and sows farrowed in individual farrowing crates (2.3 m x 0.25 m). Eight farrowing rooms (7.0 m x 11.6 m) were used during the lactation period. Each room contained 12 farrowing crates fitted with individual feeders and waterers. Piglets were provided with heat pads and lamps and did not have access to creep feed during lactation. Lights in the rooms were on continuously. Temperature of all rooms used during the lactation period was 20°C. A drip-cooling system was used for the sows and gilts during the summer months.

Experimental Treatments: Lactation

When farrowing was complete, each animal was weighed and assigned to one of two lactation treatments. Gilts and sows were assigned to lactation treatment by parity in order to equalize the distribution of gestation treatment across lactation treatment. Therefore, all combinations of gestation and lactation treatment were represented.

During lactation all animals were fed a 16% crude protein, barley-based, pelleted commercial nurser sow ration (Landmark Feeds). All animals were fed twice daily. On d 1 of lactation, control animals (lc) were given an amount of feed approximately equal to their final gestation stage daily feed intake (divided into two portions). If all feed was consumed

extra feed was added at each feeding in 0.2 kg increments until maximum feed intake was reached.

Treatment 2 (pattern) (lp) gilts and sows were fed in three stages during lactation (Table 2). Each daily ration was split into two portions and the animals were fed by hand twice daily, at 0700 h and 1430 h. The stages were designed as follows: stage I: 1.9 times the maintenance requirement (d 1 to d 6 of lactation), Stage II: 3.0 times maintenance (d 7 to d 12), and Stage III: 4.1 times maintenance (d 13 to d 17). Lactation feed intakes were calculated using Equation 1 based on a maintenance energy allowance of 461 kJ $kg^{-0.75}$ (Jindal *et al.*, 1996). Body weight taken at the end of each stage was used to calculate metabolic body weight. Lactation stage feed intakes were calculated using body weight rounded up to the nearest decimal place and grouped within a two kg weight range. Feed intakes were recorded for both treatments for the entire lactation period. Feed not consumed was weighed and recorded.

Table 2. Calculated lactation feed intake levels for treatment 2 (Pattern)(lp) based on maintenance feed intake requirements.

			Lactation Feed Intake		
Body Wt. (kg)	Metabolic Body Wt. (kg)	Maintenance Feed Rqt. (kg)	(d1 to d6) 1.9*M (kg)	(d7 to d12) 3.0*M (kg)	(d13 to d18) 4.1*M (kg)
138	40.26	1.37	2.61	4.14	5.66
140	40.70	1.39	2.64	4.18	5.74
142	41.14	1.40	2.66	4.23	5.81
144	41.57	1.42	2.69	4.28	5.86
146	42.00	1.43	2.72	4.32	5.92
148	42.43	1.45	2.75	4.36	5.98
150	42.86	1.46	2.78	4.40	6.04
152	43.29	1.47	2.80	4.45	6.10
154	43.72	1.49	2.83	4.50	6.16
156	44.14	1.50	2.86	4.54	6.22
158	44.56	1.52	2.89	4.58	6.28
160	44.99	1.53	2.91	4.62	6.34
162	45.41	1.55	2.94	4.67	6.40
164	45.83	1.56	2.97	4.71	6.46
166	46.25	1.58	3.00	4.75	6.52
168	46.66	1.59	3.02	4.81	6.60
170	47.08	1.60	3.05	4.83	6.62
172	47.49	1.62	3.08	4.88	6.69
174	47.91	1.63	3.10	4.94	6.75
176	48.32	1.65	3.13	4.97	6.81
178	48.73	1.66	3.16	5.01	6.87
180	49.14	1.67	3.18	5.05	6.93
182	49.55	1.69	3.21	5.10	6.99
184	49.96	1.70	3.24	5.13	7.03
186	50.37	1.72	3.26	5.18	7.10
188	50.77	1.73	3.28	5.21	7.14
190	51.18	1.74	3.31	5.27	7.21
192	51.58	1.76	3.33	5.29	7.25
194	51.98	1.77	3.37	5.35	7.33
196	52.38	1.78	3.40	5.40	7.40
198	52.78	1.80	3.42	5.43	7.44
200	53.18	1.81	3.45	5.48	7.51
202	53.58	1.83	3.47	5.51	7.55
204	53.98	1.84	3.50	5.56	7.62
206	54.38	1.85	3.52	5.59	7.66
208	54.77	1.87	3.55	5.64	7.73
210	55.17	1.88	3.57	5.67	7.77

Production Data: Lactation

On approximately d 109 of gestation, gilts and sows were moved into the farrowing crates. Sows were induced with 1.5 millilitres (ml) of Lutalyse® (intramuscular, im) (Upjohn Company, Animal Health Division, Orangeville, ON) on d 114 of gestation. Sows received 1.5 ml im of oxytocin (Vetoquinol Canada Inc., Joliette, P.Q.) if necessary. Gilts were not induced to farrow.

Gilt Production Data

Gilts and sows were weaned on Tuesdays and Thursdays closest to d 18 of lactation. Lactation length ranged from 14 - 20 d (mean, 17 d). All measurements (sow and piglet weights) were made on day of weaning. Gilts and sows were weighed after farrowing and at the end lactation. Lactation treatment p was weighed on d 6 and d 12 of lactation to enable determination of feed intake levels in the subsequent stages. P2 backfat measurements were recorded at the end of lactation.

Piglet Production Data

Records at birth included: total litter weight of liveborn piglets, total born, total born, born alive, stillborn, and number of mummies. All cross-fostering was done within 24 h of birth within lactation treatment. Litters were standardized to 12 piglets. All litters were weighed at weaning. Litter birth weights were assigned to their biological mother. Weaning

weights were assigned to the foster mother. Pre-weaning mortality and the number of piglets weaned were recorded.

Analytical Techniques

Feed Analysis

A subsample of each diet was analyzed for nitrogen, energy, and dry matter. Feed samples were ground in a Tecator cyclotec 1093 sample mill (Hoganas, Sweden). Dry matter content was determined after drying samples in a vacuum oven at 105°C for 24 hours. Dry matter and nitrogen content were determined according to the Association of Official Analytical Chemists (AOAC, 1990). Gross energy was determined using an adiabatic oxygen bomb calorimeter (Parr, model 1241).

Hormone and Metabolite Analyses

Blood Urea

Serum samples from d 30, d 60, d 90, and d 109 of gestation were analyzed for urea nitrogen concentrations using a standard kit (Procedure No.535) from Sigma Diagnostics (St. Louis, MO).

Urea concentration was measured without deproteinization of the samples. Twenty microlitres (µl) of serum was used to determine urea concentration. Standards ranged in value from 15 - 75 mg dl^{-1}. Samples, standards and controls were read at 540 nm within 20

minutes of removal from the water bath. Intraassay coefficients of variation were ≤9.6%. The interassay coefficient of variation was 3.3%. Blood urea nitrogen concentrations were expressed in mg dl^{-1}.

Progesterone

Serum samples from d 30, d 60, d 90, and d 109 of gestation were analyzed for progesterone (P_4) concentrations using solid-phase radioimmunoassay (RIA) (Coat-a-Count progesterone kit, Diagnostic Products Corporation, CA). ^{125}I-labelled progesterone was used as the tracer with counts of 70,000 cpm and maximum binding of ≤ 52.00 %. The standard curve range was 0.1 to 40 ng ml^{-1}. The method required 100 µl of standard or serum pipetted into anti-P_4 coated tubes, followed by the addition of 1 ml of tracer. Tubes were decanted after incubation for three hours at room temperature to isolate the antibody-bound P_4. Radioactivity was measured by a gamma counter (LKB Wallac 1282 CompuGamma Universal Gamma Counter). Nonspecific binding of the assay was ≤ 1.50 %. The sensitivity of the assay was 0.09 ng ml^{-1} at 90% binding. The intra-assay coefficients of variation were 3.16 %, 5.15 %, and 3.82 % for assays 1, 2, and 3, respectively. The interassay coefficient of variation was 5.56 %. Progesterone concentrations were expressed in ng ml^{-1}.

Statistical Analysis

Experiment 1 was analyzed as a two-way factorial during gestation (main effects: gestation treatment and parity), and as a three-way factorial design during lactation (main effects: gestation treatment, lactation treatment, and parity) using the General Linear Model of the Statistical Analysis System (1986). The level of significance was defined as $P<0.054$. A trend was defined as $P=0.055 - 0.08$.

Gestation

Gestation Model: $y_{ijk} = \mu + g_i + p_j + gp_{ij} + e_{ijk}$

Where:

μ = mean.

g_i = gestation treatment effect, i =1 to 2.

p_j = parity effect, j = 1 to 2.

gp_{ij} = interaction of gestation treatment and parity, ij = 1 to 4.

e_{ijk} = error.

To test for the effects of gestation treatment and parity during gestation, ADFI, sow weight and backfat, serum urea, and P_4 were analyzed as split plots. Repeated measures analysis was used with the gestation model and included the effects of stage of gestation, the interactions of gestation treatment*day, parity*day, and gestation treatment*parity*day. The effects of gestation treatment and parity were tested using sow within gestation

treatment*parity as the error term. When significant interactions occurred, contrasts were employed to determine differences between treatment groups or parity groups over time.

Total feed intake for each stage of gestation, as well as sow weight and backfat change during each stage of gestation, were analyzed as two-way factorials. Contrasts were utilized to determine differences between treatment or parity groups for significant interactions.

Lactation

Lactation model: $y_{ijkl} = \mu + g_i + l_j + p_k + gl_{ij} + gp_{ik} + lp_{jk} + glp_{ijk} + e_{ijkl}$

Where:

μ = mean.

g_i = gestation treatment effect, i = 1 to 2.

l_j = lactation treatment effect, j = 1 to 2.

p_k = parity effect, k = 1 to 2.

gl_{ij} = interaction of gestation treatment and lactation treatment, ij = 1 to 4.

gp_{ik} = interaction of gestation treatment and parity, ik = 1 to 4.

lp_{jk} = interaction of lactation treatment and parity, jk = 1 to 4.

glp_{ijk} = interaction of gestation treatment, lactation treatment and parity, ijk = 1 to 8.

e_{ijkl} = error.

To test for the effects of gestation treatment, lactation treatment and parity during lactation, ADFI during lactation, sow weight and backfat, predicted maternal body lipid and

protein composition during lactation, and average piglet weight, were analyzed as split plots. Repeated measures analysis was used for the above variables. The repeated measures model included stage of lactation, the interactions of gestation treatment*day, parity*day, lactation treatment*day, the three-way interactions of gestation treatment*lactation treatment*day, gestation treatment*parity*day, lactation treatment*parity*day, and the interaction of gestation treatment*lactation treatment*parity*day. Sow within gestation treatment*lactation treatment*parity was utilized as the error term to test the effects of gestation treatment, lactation treatment and parity. When significant interactions occurred, contrasts were employed to determine differences between treatment groups or parity groups over time. Differences between means existing at the start of lactation were defined using Bonferroni's test ($P<0.05$).

Total feed intake during each stage of lactation, sow weight and backfat changes and maternal body lipid and protein changes during each stage of lactation, litter size at birth and weaning, as well as WEI, were analyzed as three-way factorials. Contrasts were utilized to determine differences between treatment or parity groups for significant interactions.

Due to the weaning schedule of the barn, lactation lengths ranged from 14 to 20 d. In order to compare the lactation variables, lactation lengths were standardized to 17 d (Equations 2, 3, and 4) for the above measurements and the adjusted values were used in the statistical analyses. Average daily feed intake and total feed intake for lactation were computed by calculating the ADFI or total feed intake for each stage of lactation. The third stage (d 13 to weaning) was calculated by omitting feed intake values beyond d 17 of

lactation for sows that were weaned following lactation lengths of >17 d. Sows weaned earlier than d 17 of lactation had the third stage of feed intake calculated based on existing feed intake values. Sow weight and backfat values at weaning, and piglet weaning weights were adjusted to 17-d values using the formulae:

Equation 2: $$\text{Day 17 sow weight} = \frac{(\text{weaning weight} - \text{d 0 weight})}{\text{lactation length}} * 17 + \text{d 0 weight};$$

Equation 3: $$\text{Day 17 sow backfat} = \frac{(\text{weaning backfat} - \text{d 109 backfat})}{\text{lactation length}} * 17 + \text{d 109 backfat};$$

Equation 4: $$\text{Day 17 piglet weight} = \frac{(\text{weaning weight} - \text{birth weight})}{\text{lactation length}} * 17 + \text{birth weight};$$

Proportions of piglets born alive, stillborn, and necrotic were compared for gestation treatment, parity, and gestation treatment within parity using χ^2 analysis .

Prediction equations (Whittemore and Yang, 1989) were used to estimate total body protein (equation 5) and total body lipid (equation 6) of the gilts during lactation. The r^2 for protein and fat are $\geq$0.90 and $\geq$0.80, respectively. Body weight and backfat measurements were used from the beginning and end (adjusted to d 17) of lactation.

Equation 5: Protein (kg) = -2.3 + 0.19 live weight - 0.22 P2;

Equation 6: Lipid (kg) = -20.4 + 0.21 live weight + 1.5 P2;

RESULTS AND DISCUSSION

GESTATION

Gestation Feed Intake

Average Daily Feed Intake

The assigned pattern of feed intake resulted in no significant difference in average daily feed intake (ADFI) between Control (gC) and Pattern (gP) treatments over the course of gestation (Table 3). Gilts on average consumed 0.50 kg d^{-1} less feed than first parity sows during gestation ($P<0.05$). This can be expected due to the higher body weight of the first parity animals resulting in an increased maintenance feed intake requirement.

The interaction of gestation treatment*stage of gestation (Figure 1) confirmed the differential feed intake levels, with the difference in ADFI between treatments greater in early gestation, decreasing during mid-gestation, and becoming larger again in late gestation. Treatment gC consumed more feed per day during the first 60 d of gestation than treatment gP. From d 61 to farrowing, the gP animals had higher ADFI, although the difference in ADFI between treatments from d 61 to d 90 was smaller than during other stages.

The interactions of parity*stage and gestation treatment*parity*stage were also significant, reflecting the expected differences in feed intake during the course of gestation due to age (body size) of the animal, and imposed gestation treatment.

Table 3. Gestation ADFI (kg) of gilts and first parity sows

Factor			Average daily feed intake [b] (kg d^{-1})
Gestation Trt.			ns
		C	2.52 ± 0.02
		P	2.52 ± 0.02
Parity[c]			P = 0.0001
		0	2.27 ± 0.02
		1	2.77 ± 0.02
Gest.*Parity			ns
Stage[f]			P = 0.0001
		1	1.99[a] ± 0.01
		2	2.30[b] ± 0.01
		3	2.67[c] ± 0.01
		4	3.10[d] ± 0.01
Gest.*Stage**			P = 0.0001
	C	1	2.22 ± 0.01
		2	2.40 ± 0.01
		3	2.60 ± 0.01
		4	2.84 ± 0.01
	P	1	1.75 ± 0.01
		2	2.21 ± 0.01
		3	2.73 ± 0.01
		4	3.37 ± 0.01
Parity*Stage[g]			P = 0.0003
Gest.*Parity*Stage[g]			P = 0.0001

Values are LS means ● SEM.
ns = non-significant, P>0.05.
[a-d]within columns, means with unlike superscripts differ, P <0.05.
[c]parity 0 (gilt), parity 1 (first parity sow).
[f]Stage= gestation divided into 4 stages:
stage1 = d 1 to d 30, stage2 = d 31 to d 60, stage3 = d 61 to d 90, stage4 = d 91 to farrow.
[g]means not presented for these effects.
[h]LS means are average daily feed intake for each stage of gestation.
●*See Figure 1.

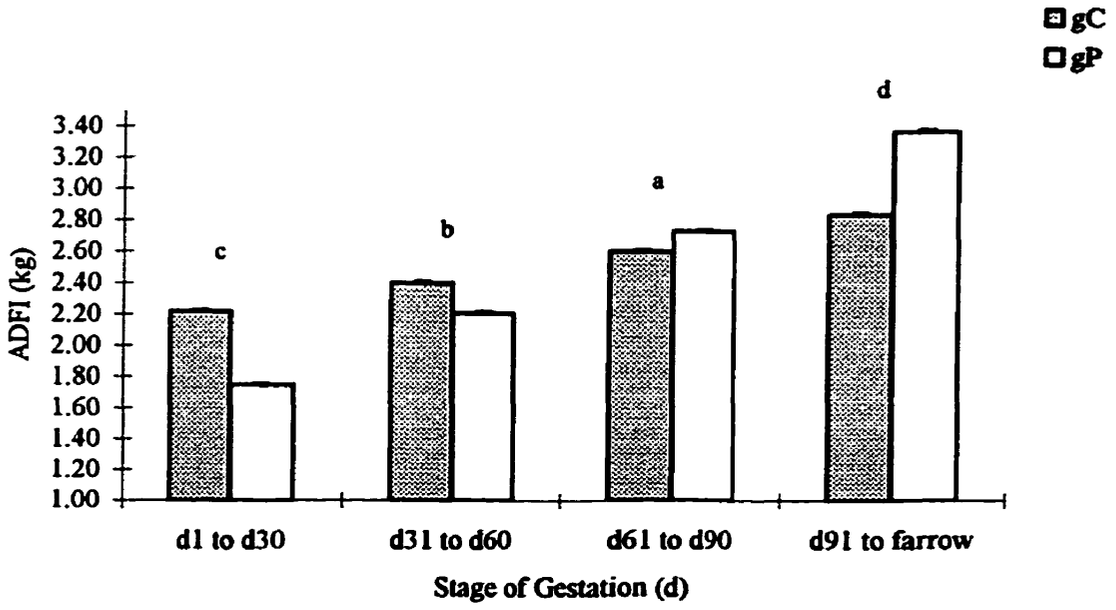

Figure 1. Gestation treatment*stage of gestation interaction for ADFI of gilts and first parity sows (LS means + SEM).

[a]different letters indicate that the difference between treatments in these time periods are not the same ($P<0.05$).

Total Gestation Feed Intake

Total feed intake during pregnancy was to remain equal to examine the effect of pattern of feed intake on the parameters of interest in the absence of differences in total gestational nutrient intake. This was achieved as shown in Table 4. Total feed intake during each stage of gestation differed between treatments ($P<0.05$), while total feed intake for the entire gestation period did not differ ($P>0.05$). As planned, feed intake of the gC treatment at 1.4 times maintenance during the first two stages of gestation was higher than the gP treatment (1.1 times maintenance and 1.3 times maintenance during stages 1 and 2, respectively). During the last two stages of gestation, the gP treatment consumed more feed (1.5 and 1.7 times maintenance in stages 3 and 4, respectively) than the gC treatment (1.4 times maintenance).

Body Composition

Backfat

Gestation treatment or parity did not affect mean P2 backfat levels during gestation ($P>0.05$) (Table 5). However, backfat measurements taken on d 109 of gestation tended ($P=0.06$) to be greater for gP gilts and sows. The interaction of gestation treatment*day was not significant. Variability associated with measurement of backfat depth (Mullan, 1991) may reduce the possibility of observing differences in P2 levels between treatments.

Table 4. Total feed intake (kg) of gilts and first parity sows during each stage of gestation

		Stage of Gestation				
Factor		**d 1 to d 30**	**d 31 to d 60**	**d 61 to d 90**	**d 91 to farrow**	**d 1 to farrow**
Gestation Trt.		P = 0.0001	P = 0.0001	P = 0.0001	P = 0.0001	ns
	C	66.67 ± 0.55	72.12 ± 0.60	78.10 ± 0.66	69.51 ± 0.75	286.40 ± 2.24
	P	52.66 ± 0.57	66.20 ± 0.62	81.99 ± 0.68	82.70 ± 0.77	283.55 ± 2.30
Parity[a]		P = 0.0001	P = 0.0001	P = 0.0001	P = 0.0001	P = 0.0001
	0	53.05 ± 0.54	61.54 ± 0.59	72.26 ± 0.65	70.03 ± 0.74	256.89 ± 2.20
	1	66.28 ± 0.58	76.78 ± 0.63	87.83 ± 0.69	82.17 ± 0.78	313.06 ± 2.33
Gest.*Parity		ns (0.0737)	ns	ns	ns	ns
C	0	59.34 ± 0.75	64.12 ± 0.81	70.87 ± 0.89	64.29 ± 1.02	258.64 ± 3.03
	1	74.00 ± 0.81	80.11 ± 0.89	85.33 ± 0.97	74.72 ± 1.11	314.16 ± 3.30
P	0	46.76 ± 0.79	58.96 ± 0.86	73.65 ± 0.94	75.77 ± 1.07	255.15 ± 3.19
	1	58.56 ± 0.81	73.44 ± 0.89	90.33 ± 0.97	89.62 ± 1.11	311.96 ± 3.30

Values are LS means ± SEM.
ns=non-significant, P>0.05.
[a]parity 0 (gilt), parity 1 (first parity sow).

Table 5. P2 backfat (mm) and weight (kg) of gilts and first parity sows during gestation

Factor		P2 Backfat (mm)	Weight (kg)
Gestation Trt.		ns	ns
	C	14.01 ± 0.30	179.76 ● 1.84
	P	14.51 ± 0.30	177.97 ± 1.86
Parity[f]		ns	P = 0.0001
	0	14.29 ± 0.29	156.30 ± 1.79
	1	14.22 ± 0.31	201.43 ± 1.91
Gest.*Parity[g]		ns	ns
Day		P = 0.0001	P = 0.0001
	1	13.16[a] ± 0.23	145.21[a] ± 0.53
	30	14.34[b] ± 0.23	159.43[b] ± 0.53
	60	14.59[b] ± 0.23	176.08[c] ± 0.53
	90	14.73[b] ± 0.23	197.70[d] ± 0.53
	109	14.47[b] ± 0.24	215.91[e] ± 0.54
Gest.*Day		ns[g]	P = 0.0064 **
	C 1		145.28 ± 0.74
	30		160.03 ± 0.74
	60		178.03 ± 0.74
	90		199.09 ± 0.74
	109		215.49 ± 0.76
	P 1		145.14 ± 0.76
	30		157.94 ± 0.76
	60		174.13 ± 0.76
	90		196.32 ± 0.76
	109		216.33 ± 0.76
Parity*Day[h]		P=0.0011	P=0.0110
Gest.*Parity*Day		ns[g]	P = 0.0110**

Values are LS means ± SEM.
ns=non-significant, P>0.05.
[a-c]within columns, means with unlike superscripts differ, P <0.05.
[f]parity 0 (gilt), parity 1 (first parity sow).
[g]means not presented for these non-significant effects.
[h]means not presented for these effects.
**See Figures 2,3.

In general, the level of backfat depth at parturition (13.82 ± 0.34 and 15.12 ± 0.34 mm, treatments gC and gP respectively), was lower than the 18 - 20 mm P2 depth for gilts and sows recommended by Aherne and Williams (1992) and Yang *et al.* (1989). The ADFI of 2.52 ± 0.02 kg d^{-1} during gestation for both treatments may have been inadequate to achieve the target levels of backfat. Yang *et al.* (1989) recommend 3 kg d^{-1} as a more suitable feed allowance during pregnancy to realize the target levels of P2 backfat at parturition. Target levels are suggested because P2 backfat depth at farrowing influences subsequent lactational and reproductive performance (Dourmad, 1991; Neil *et al.*, 1996).

Weight

Gestation treatment had no effect on sow weight during gestation (P>0.05) (Table 5). Due to the lack of difference in mean values for ADFI and total feed intake during pregnancy, differences in mean weight due to gestation treatment would not be expected.

The interaction of gestation treatment*day was significant (Figure 2). The treatment animals were of similar average weight at the start of the trial. The difference in body weight between treatments by d 60 of gestation was greater than the difference on d 1, with gilts and sows fed at 1.4 times maintenance (gC), gaining weight more rapidly during this early part of gestation than gilts fed below this level (gP). On d 109 of gestation there was no difference in body weight between treatments due to the increased rate of gain of the gP treatment group from mid-gestation to parturition. Therefore, the significance of the interaction coincides with the administered patterns of feed intake, and is similar to the

relationship between pregnancy weight gain and gestation feed intake in gilts reported by Dourmad (1991). Gestation weight gain (d 1 to d 109) reported in Appendix 2 was similar between treatments ($P>0.05$) at approximately 70 kg, although higher than the values of 45 - 60 kg gain suggested by Verstegen and Den Hartog (1989) and Aherne and Williams (1992).

Cromwell *et al.*, (1980) reported that pregnancy weight gain was influenced by total feed intake during gestation rather than pattern of feed intake. The absence of observed differences in maternal weight gain during gestation is an indication of the similarity in total gestation feed intake across treatments reported in this study.

The 3-way interaction of gestation treatment*parity*day was also significant (Figure 3). The parities differed in weight at the start of the trial ($P<0.05$), while weight within parity was similar for both treatments on d 1. Differences between treatments and parities at the start of gestation (d 1) were not the same as differences in weight at the end of gestation (d 109). The pattern of pregnancy weight gain of first parity sows responded differently to gestation treatment than gilts. The difference between gC and gP groups of first parity sows was small during early gestation, with weight of gC sows increasing above that of gP sows. A change in direction of the response resulted in gP sows increasing in weight during late gestation at a greater rate than the gC group. Gilts responded to gestation treatment in an opposite manner. The difference in initial body weight between gC and gP gilts was small. However, the difference between treatments increased during gestation, with gC gilts maintaining a higher body weight (rate of gain) throughout gestation. The difference in weight between treatments for first parity sows during gestation more closely reflected the

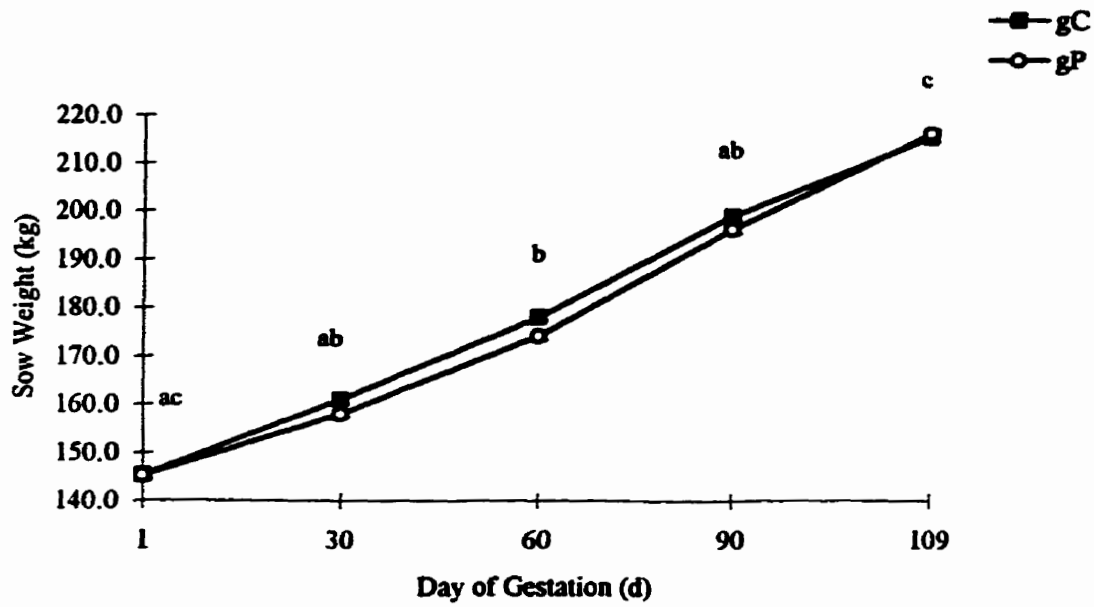

Figure 2. Gestation treatment*day of gestation interaction for gilt and first parity sow weight (LS means + SEM).

[a]different letters indicate that the difference between treatments in these time periods are not the same (P<0.05).

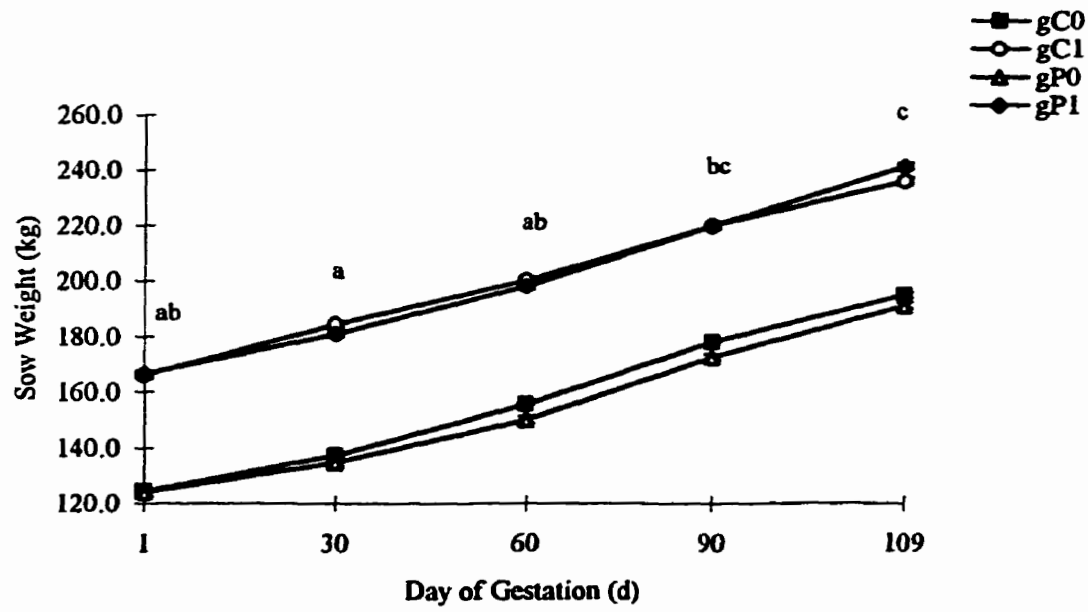

Figure 3. Gestation treatment*parity*day of gestation interaction for gilt and first parity sow weight (LS means + SEM).

[a]different letters indicate that the difference between treatments in these time periods are not the same (P<0.05).

pattern of ADFI (Figure 1). Conversely, the difference between gC and gP gilts demonstrated an inability of gP gilts to increase in weight during the period of increased feed intake in late gestation to the same extent as was noted for first parity sows. In general, feeding gilts at a constant proportion of body weight (gC), or pattern-feeding (gP) during gestation resulted in similar pregnancy weight gain (Appendix 2). However, pattern feeding (gP) resulted in a larger weight gain in first parity sows compared to the gC treatment.

Serum Urea Nitrogen

Mean serum urea nitrogen concentrations were not influenced by gestation treatment ($P>0.05$) (Table 6). If serum urea N concentrations are an indicator of catabolism of amino acids from exogenous or endogenous sources (ie. dietary versus body protein catabolism) (Chen *et al.*, 1995), the lack of treatment-induced differences in urea N would indicate that the imposed dietary treatments did not appear to cause mobilization of body protein. This conclusion is supported by the positive overall increase in body condition during gestation.

Table 6. Mean serum urea nitrogen (mg dl^{-1}) of gilts and first parity sows during gestation

Factor			Serum Urea Nitrogen (mg dl^{-1})
Gestation Trt.			ns
		C	15.33 ● 0.47
		P	15.96 ± 0.49
Parity[a]			P = 0.0001
		0	14.13 ± 0.53
		1	17.15 ± 0.43
Gest.*Parity			ns
	C	0	13.81 ± 0.75
		1	16.84 ± 0.57
	P	0	14.46 ± 0.74
		1	17.46 ± 0.65
Day[b]			ns
Gest.*Day[b]			ns
Parity*Day[b]			ns
Gest.*Parity*Day[b]			ns

Values are LS means ± SEM.
ns = non-significant, P>0.05.
[a]parity 0 (gilt), parity 1(first-parity sow).
[b]means not presented for these non-significant effects.

Progesterone

There were no effects of gestation treatment, parity, or their interaction on mean serum progesterone (P_4) concentrations during pregnancy ($P>0.05$) (Table 7). Progesterone concentrations decreased from d 60 to d 109 for all gilts and sows, in agreement with Dyck *et al.* (1980) who state that from approximately d 30 to d 100 of pregnancy, P_4 levels are relatively constant and decline to base levels by parturition.

Table 7. Mean serum progesterone (P_4) (ng ml^{-1}) of gilts and first parity sows during gestation

Factor			P_4 (ng ml^{-1})
Gestation Trt.			ns
		C	15.99 ● 0.59
		P	15.23 ± 0.59
Parity[d]			ns
		0	15.16 ± 0.58
		1	16.07 ± 0.60
Gest.*Parity			ns
	C	0	15.49 ± 0.86
		1	16.51 ± 0.82
	P	0	14.82 ± 0.77
		1	15.63 ± 0.88
Day			P = 0.0001
		30	17.92[a] ± 0.41
		60	17.85[a] ± 0.41
		90	14.39[b] ± 0.38
		109	12.30[c] ± 0.40
Gest.*Day[e]			ns
Parity*Day[f]			P=0.0246
Gest.*Parity*Day[e]			ns

Values are LS means ● SEM.
ns = non-significant, P>0.05.
[a-c]within columns, means with unlike superscripts differ, P<0.05.
[d]parity 0 (gilt), parity 1 (first parity sow).
[e]means not presented for these non-significant effects.
[f]means not presented for this effect.

LACTATION

Lactation Feed Intake

Average daily feed intake

Previous research has shown that an inverse relationship exists between lactation feed intake and average feed intake in the previous gestation (Mullan and Williams, 1989; Revell and Williams, 1993). As well, a negative relationship between sow body weight or fatness at parturition, as influenced by gestation feeding level, and lactation feed intake has been reported (Dourmad, 1991; Koketsu *et al.*, 1996a; Neil *et al.*, 1996).

However, in the current study there was no effect of gestation treatment on ADFI in the subsequent lactation ($P>0.05$) (Table 8). Cromwell *et al.* (1989) found that additional feed from d 90 of gestation to farrowing, resulted in increased total gestation feed intake and did not influence ADFI during lactation. Little data exists on the influence of pattern of feed intake during gestation on feed intake in the subsequent lactation. Treatment similarities in ADFI and total feed intake during pregnancy, and the absence of treatment differences in sow weight or backfat depth at d 109, may explain why the negative relationship between gestation and lactation feed intake was not observed in this study.

Lactation treatment significantly affected ADFI during the lactation period ($P<0.05$). Pattern (lp) gilts and sows consumed, on average, 0.35 kg d^{-1} more feed than control (lc) animals. The lc treatment was administered at a level which may be defined as full-feeding

Table 8.Gilt and first parity sow ADFI (kg) during lactation

Factor			Average Daily Feed Intake[a] (kg d^{-1})
Gestation Trt.			ns
C			4.74 ± 0.07
P			4.76 ± 0.07
Lactation Trt.			P = 0.0004
c			4.57 ± 0.06
p			4.92 ± 0.07
Parity[d]			P = 0.0001
0			4.46 ± 0.06
1			5.04 ± 0.07
Gest.*Lact.			ns
	C	c	4.56 ± 0.08
		p	4.92 ± 0.10
	P	c	4.59 ± 0.10
		p	4.92 ± 0.09
Gest.*Parity[f]			ns
Lact.*Parity			P = 0.0266
	c	0	4.39 ± 0.10
		1	4.76 ± 0.10
	p	0	4.52 ± 0.10
		1	5.32 ± 0.10
Gest.*Lact.*Parity[f]			ns

Table 8.Gilt and first parity sow ADFI (kg) during lactation (continued)

Factor			Average Daily Feed Intake[g] (kg d^{-1})
Stage[e]			P = 0.0001
		1	3.12[a] ± 0.05
		2	4.95[b] ± 0.05
		3	6.19[c] ± 0.05
Gest.*Stage[f]			ns
Lact.*Stage**			P = 0.0001
	c	1	3.11 ± 0.07
		2	4.79 ± 0.07
		3	5.83 ± 0.07
	p	1	3.13 ± 0.07
		2	5.10 ± 0.07
		3	6.54 ± 0.07
Parity*Stage[h]			P=0.0020
Gest.*Lact.*Stage[f]			ns
Gest.*Parity*Stage[f]			ns
Lact.*Parity*Stage[f]			ns
Gest.*Lact.*Parity*Stage[f]			ns

Values are LS means ± SEM.
ns=non-significant, P>0.05.
[a-c]within columns, means with unlike superscripts differ, P<0.05.
[d]parity 0 (gilt), parity 1 (first parity sow).
[e]Stage= lactation divided into 3 stages:
stage1 = d 1 to d 6, stage2 = d 7 to d 12, stage3 = d 13 to d 17.
[f]means not presented for these non-significant effects.
[g]LS means are average daily feed intake (adfi) for each stage of lactation adjusted for 17-d lactation using adfi for each stage.
[h]means not presented for this effect.
**See Figures 4, 5.

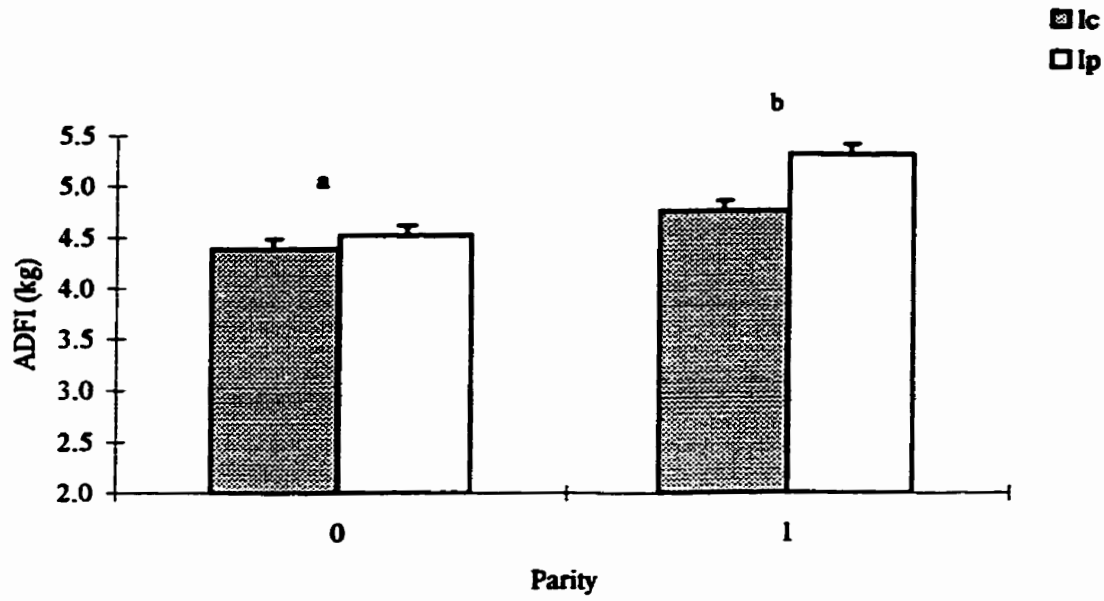

Figure 4. Lactation treatment*parity interaction for ADFI of gilts and first parity sows (LS means + SEM).
[a]different letters indicate that the difference between treatments in these time periods are not the same (P<0.05).

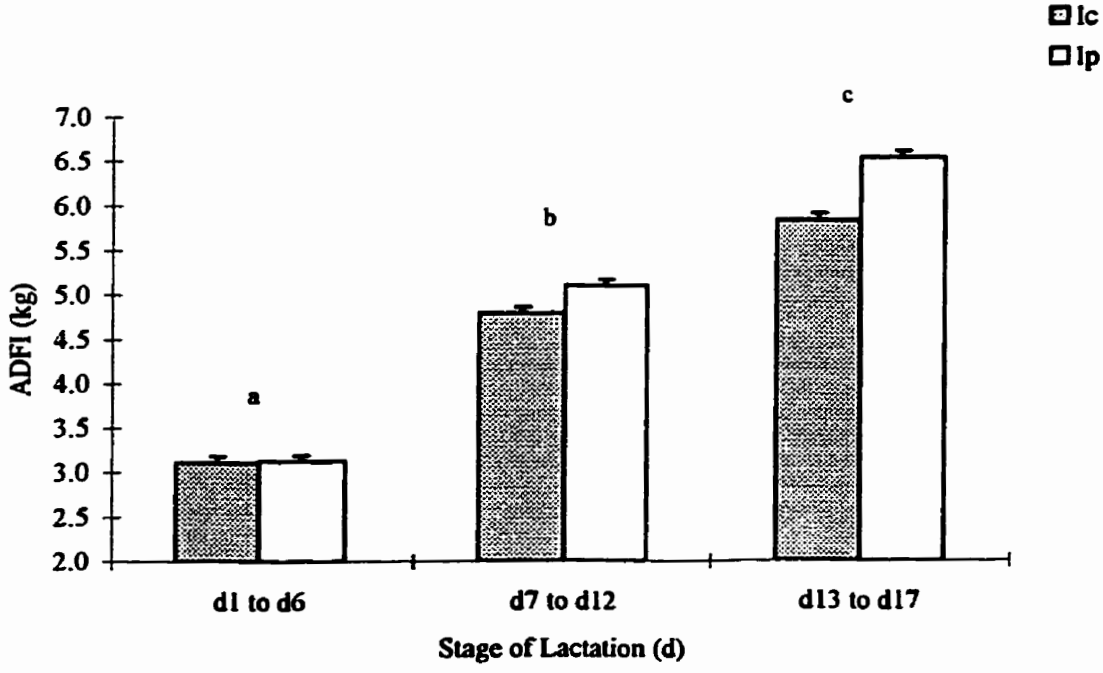

Figure 5. Lactation treatment*stage of lactation interaction for ADFI of gilts and first parity sows (LS means + SEM).
[a]different letters indicate that the difference between treatments in these time periods are not the same (P<0.05).

rather than *ad libitum*-feeding. This experiment was conducted in a commercial operation and the feed intake level of lc gilts and sows was restricted during early lactation and increased in increments of 0.2 kg per feeding (twice daily) according to existing management practices of the unit. As a result, the pre-calculated feed intake levels of the lp treatment during the second and third stages of lactation were greater than the standard levels (lc) of lactation feed intake administered.

The interaction between lactation treatment*parity ($P<0.05$) (Figure 4) indicates that the difference in ADFI between treatments increases with parity. Average daily feed intake of gilts was similar for lactation treatments lc and lp. First parity sows assigned to the lc treatment had ADFI similar to both treatment groups of gilts, but lower ADFI than first parity sows receiving treatment lp. Koketsu *et al.* (1996), in a characterization of feed intake patterns of commercial swine herds during lactation, found a significant difference in ADFI of gilts and first parity sows. Other authors also report a lower voluntary feed intake in gilts compared to that of multiparous sows during lactation (Mullan and Close, 1989; Patience, 1993; Genest and D'Allaire, 1995). Assignment of feed intake during the lactation period based on metabolic body weight (lp) versus the level considered suitable to satisfy voluntary feed intake (lc) in gilts were similar, probably due to the lower voluntary feed intake of gilts (Genest and D'Allaire, 1995). The interaction of lactation treatment*parity may be explained by the possible limitation of voluntary feed intake of first parity sows in the lc group.

Average daily feed intake increased during lactation regardless of treatment pattern ($P<0.05$). The interaction of lactation treatment*stage of lactation illustrated in Figure 5

shows that the difference in ADFI between treatments increased with stage of lactation ($P<0.05$). During early lactation (d 1 to d 6), ADFI of both lactation treatments was similar. Mid- to late lactation saw an increase in ADFI of the lp gilts and sows compared to the lc group. Since the feed allowance for both lc and lp gilts and sows was restricted in early lactation, the difference between treatments from d 1 to d 6 was small. By raising the feed allowance by 0.2 kg per feeding for the lc group, and by increasing the feed allowance of the lp gilts and sows at a greater rate, the difference in ADFI between treatments increased as lactation progressed. The difference between treatments was greatest during the third stage of lactation. These results suggest that feed intake of the control gilts and sows was underestimated by the feeding method utilized, resulting in a lower ADFI of the lc group.

Total feed intake

Factors which influence feed intake during lactation include gestation feeding level and body condition at parturition (Coffey *et al.*, 1994; Dourmad *et al.*, 1994). Gestation treatment did not induce differences in total gestation feed intake or body composition (weight and backfat) at parturition. Maternal body lipid content at the start of lactation was not different between treatments, and will be discussed in a subsequent section.

Total feed intake for each stage of lactation is presented in Table 9. Pattern of feed intake during gestation did not influence total feed intake during lactation ($P>0.05$). Research demonstrating a significant negative relationship between gestation and lactation feed intake employed high ADFI throughout gestation (Dourmad, 1991) or *ad libitum* feed intake during

late gestation (Weldon *et al.*, 1994a), resulting in differences in total gestation feed intake between treatments. Average daily feed intake and total gestation feed intake did not differ between gestation treatments in this trial, explaining the absence of a gestation treatment effect on lactation feed intake. Body composition, including weight, backfat and protein and lipid content, at the start of lactation was not affected ($P>0.05$) by treatment during the previous gestation period.

Differences in total feed intake due to lactation treatment were only significant during the periods from d 7 to d 12, and d 13 to d 17, with lactation treatment lp consuming more feed during these periods, as discussed in the previous section. Overall, lp animals consumed 7.2 % more feed over the total lactation period compared to lc animals.

Table 9. Total feed intake (kg) of gilts and first parity sows during each stage of lactation

			Stage of Lactation			
Factor			**d 1 to d 6**	**d 7 to d 12**	**d 13 to d 17[c]**	**d 1 to d 17[c]**
Gestation Trt.			ns	ns	ns	ns
		C	18.54 ● 0.42	29.76 ● 0.48	30.85 ● 0.56	79.15 ● 1.12
		P	18.86 ● 0.42	29.61 ● 0.48	31.01 ● 0.56	79.48 ● 1.12
Lactation Trt.			ns	P = 0.0086	P = 0.0001	P = 0.0007
		c	18.64 ● 0.41	28.77 ● 0.47	29.14 ● 0.55	76.56 ● 1.10
		p	18.76 ± 0.43	30.60 ± 0.49	32.73 ● 0.57	82.07 ● 1.14
Parity[a]			P = 0.0046	P = 0.0001	P = 0.0001	P = 0.0001
		0	17.83 ± 0.41	27.55 ± 0.46	29.03 ± 0.54	74.41 ± 1.08
		1	19.56 ± 0.44	31.82 ± 0.50	32.83 ± 0.58	84.22 ± 1.16
Gest.*Lact.			ns	ns	ns	ns
	C	c	18.29 ± 0.54	28.95 ● 0.62	29.02 ● 0.72	76.26 ± 1.44
		p	18.79 ● 0.65	30.56 ● 0.74	32.69 ± 0.86	82.04 ● 1.71
	P	c	18.99 ± 0.63	28.60 ± 0.71	29.27 ± 0.83	76.85 ± 1.66
		p	18.72 ± 0.56	30.63 ± 0.64	32.76 ± 0.76	82.10 ± 1.51
Gest.*Parity[b]			ns	ns	ns	ns
Lact.*Parity			P = 0.0249	ns (0.0799)	ns	P = 0.0244
	c	0	18.46 ± 0.58	27.24 ± 0.66	27.77 ± 0.76	73.46 ± 1.52
		1	18.83 ± 0.60	30.31 ± 0.68	30.52 ± 0.79	79.65 ± 1.58
	p	0	17.21 ± 0.57	27.86 ± 0.65	30.30 ± 0.77	75.35 ± 1.54
		1	20.30 ± 0.64	33.33 ± 0.73	35.15 ± 0.85	88.78 ± 1.69
Gest.*Lact.*Parity[b]			ns	ns	ns	ns

Values are LS means ± SEM.
ns=non-significant, P>0.05.
[a]parity 0 (gilt), parity 1 (first parity sow).
[b]means not presented for these non-significant effects.
[c]feed intake adjusted to 17-d lactation:
for lactation length < 17 d: total intake for d 13 to d 17 = (average feed intake from d 12 to weaning) x 5.
for lactation length > 17 d: omitted feed intake above d 17.

Body Composition

Backfat

There were no significant differences in mean P2 backfat depth for the main effects or their interactions during lactation (Table 10). Moser *et al.* (1987) reported no effect of restricted versus *ad libitum* feeding method on sow backfat loss during lactation.

As lactation progressed, all sows and gilts lost backfat ($P<0.05$) consistent with other data sources indicating catabolism of body fat reserves during lactation (Moser *et al.*, 1987; Yang *et al.*, 1989; Young *et al.*, 1991; Einarsson and Rojkittikhun, 1993).

The interaction of gestation treatment*day of lactation was significant (Figure 6). The difference between gC and gP treatments at d 0 of lactation was greater than the difference between these treatments at d 17. Gilts and sows which received the gP treatment mobilized a greater amount of backfat during the lactation period than the gC group. Gilts and sows which had received the gP treatment during pregnancy tended ($P=0.06$) to have more backfat at the end of pregnancy / start of lactation, than the gC group. Changes in backfat depth are presented in Appendix 3. Mullan and Williams (1989) found that gilts and sows with a higher backfat level in late gestation mobilized more backfat during lactation, and that this relationship was related to a higher level of feed intake during gestation. Other studies also observed greater backfat losses occuring during lactation in sows that had more backfat at farrowing (Sterning *et al.*, 1990; Einarsson and Rojkittikhun, 1993). However, no association was made between backfat levels and gestation feed intake.

Table 10. P2 backfat (mm) and weight (kg) of gilts and first parity sows during lactation

Factor		P2 Backfat[c] (mm)	Weight[c](kg)
Gestation Trt.		ns	ns
C		13.40 ± 0.40	189.89 ± 2.09
P		14.08 ± 0.39	190.58 ● 1.99
Lactation Trt.		ns	P = 0.0219
c		13.48 ± 0.39	186.83 ± 2.05
p		13.99 ± 0.40	193.55 ± 2.02
Parity[a]		ns (0.0720)	P = 0.0001
0		13.22 ± 0.38	168.76 ± 1.96
1		14.25 ± 0.41	211.62 ± 2.11
Gest.*Lact.[b]		ns	ns
Gest.*Parity[b]		ns	ns
Lact.*Parity[b]		ns	ns
Gest.*Lact.*Parity[b]		ns	ns
Day		P = 0.0001	P = 0.0001
	0	14.51 ± 0.15[d]	194.25 ± 0.51
	17	12.96 ± 0.15	186.13 ± 0.51

Table 10. P2 backfat (mm) and weight (kg) of gilts and first parity sows during lactation (continued)

Factor			P2 Backfat[c] (mm)	Weight[c](kg)
Gest.*Day			P = 0.0320**	ns[b]
	C	0	13.94 ± 0.22	
		17	12.85 ± 0.22	
	P	0	15.09 ± 0.21	
		17	13.06 ● 0.21	
Lact.*Day[b]			ns	ns
Parity*Day			ns[b]	P = 0.0002[c]
Gest.*Lact.*Day			P = 0.0058**	ns[b]
	Cc	0	14.02 ● 0.28	
		17	12.00 ± 0.29	
	Cp	0	13.86 ± 0.33	
		17	13.71 ● 0.33	
	Pc	0	14.81 ± 0.32	
		17	13.07 ± 0.32	
	Pp	0	15.37 ± 0.29	
		17	13.05 ± 0.29	
Gest.*Parity*Day[b]			ns	ns
Lact.*Parity*Day[b]			ns	ns
Gest.*Lact.*Parity *Day[b]			ns	ns

Values are LS means ● SEM.
ns=non-significant at P>0.05.
[a]parity 0 (gilt), parity 1 (first parity sow).
[b]means not presented for these non-significant effects.
[c]adjusted to 17-d lactation length:
adjusted d 17 backfat =(((backfat at weaning - d 109 backfat)/lactation length)*17) + d 109 backfat.
adjusted d 17 weight = (((weight at weaning - d 0 wt.)/lactation length)*17) + d 0 wt.
[d]P2 values on d 0 of lactation = actual values taken on d 109 of gestation.
[e]means not presented for this effect.
**See Figure 6, 7.

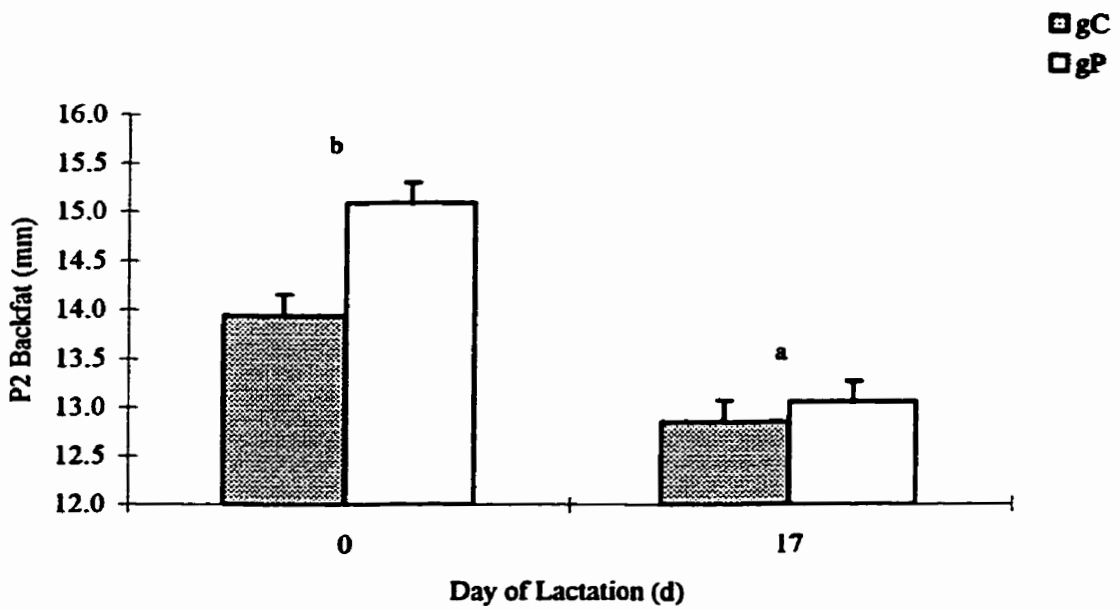

Figure 6. Gestation treatment*day of lactation interaction for gilt and first parity sow P2 backfat (LS means + SEM).

[a]different letters indicate that the difference between treatments in these time periods are not the same ($P<0.05$).

Mullan and Williams (1989) and Mullan (1991) describe an inverse relationship between backfat depth at farrowing and lactation feed intake (Mullan and Williams, 1989; Mullan, 1991). However, in the present study there was no apparent connection between backfat depth at parturition and lactation feed intake. Similarly, Yang *et al.* (1989) found that backfat level at farrowing did not appear to be inversely related to lactation feed intake.

The 3-way interaction of gestation treatment*lactation treatment*day of lactation was also significant (Figure 7). The difference between gilts and sows that received the Control treatment during gestation and either the control (c) or pattern (p) treatment during lactation was smaller on d 0 than on d 17 of lactation. Of the gilts and sows that had received the gC treatment, those assigned to the p group in lactation maintained a consistent level of backfat during lactation, while the combination of Cc lost a larger amount of backfat and had the lowest backfat depth at the end of lactation. Differences in ADFI (and total feed intake) influence backfat loss (King and Williams, 1984; King and Dunkin, 1986; Patience, 1993) and may explain the decrease in backfat during lactation in the Cc treatment group. King and Dunkin (1986) found a linear decrease in backfat and weight loss as feed intake during lactation increased. Gilts and sows assigned to the gC treatment during gestation tended to have lower backfat depth at farrowing, and this gestation treatment combined with the control treatment in lactation, may have resulted in greater mobilization of backfat reserves to support lactation demands. The gC sows and gilts assigned to the pattern treatment during lactation (treatment combination Cp), were provided a higher level of feed intake during lactation which may have allowed for maintenance of P2 backfat depth. Yang *et al.* (1989) found that

backfat loss during lactation was less in gilts and sows that were thinner at farrowing and fed *ad libitum* during lactation. Sows that were fed to attain 12 mm backfat at parturition, and fed *ad libitum* during lactation did not lose backfat during lactation.

The difference in P2 backfat depth between Pc and Pp treatments on d 0 was greater than the difference on d 17. Backfat loss during lactation for sows that received the pattern treatment during gestation (gP) did not seem to be influenced by lactation treatment. Feeding at increasing levels times maintenance during gestation (gP) tended to result in greater backfat depth at farrowing, resulting in a higher rate of mobilization of these reserves in lactation (Hulten *et al.*, 1993). Gestation treatment P, paired with the control treatment during lactation consumed the lowest feed intake level during lactation which may have further accelerated backfat mobilization. Lactation treatment p, which provided greater lactational feed intake, combined with gestation treatment P, somewhat balanced the degree of backfat loss in this group.

In general, three of the four gestation-lactation treatment combinations (Cc, Pc and Pp) showed greater backfat loss over the course of lactation. The lower feed intake of lc sows during lactation may have resulted in greater mobilization of body fat reserves to support the demands of lactation (King and Dunkin, 1986; Noblet *et al.*, 1990; Patience, 1993). Zak *et al.* (1997) found that primiparous sows fed either *ad libitum* for the first 22 days of a 28-day lactation, and restricted to 50 % of *ad libitum* intake thereafter, or restricted to 50 % of *ad libitum* intake to d 21 and fed *ad libitum* to d 28 lost more backfat than sows fed *ad libitum* throughout lactation.

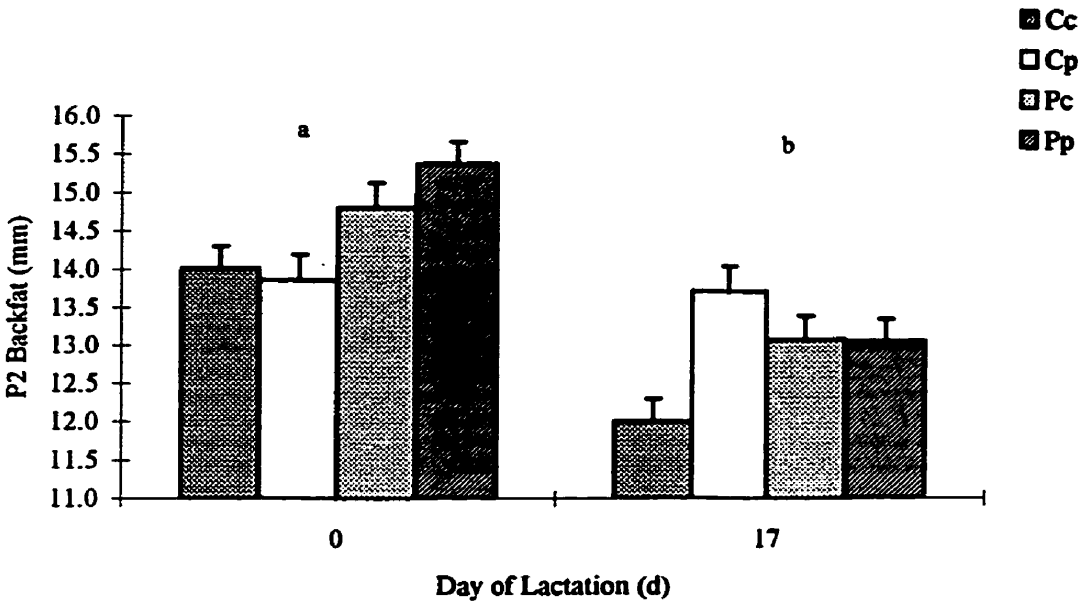

Figure 7. Gestation treatment*lactation treatment*day of lactation interaction for gilt and first parity sow P2 backfat (LS means + SEM).

*different letters indicate that the difference between treatments in these time periods are not the same ($P<0.05$).

Weight

Sow weight during lactation was not affected by feed intake pattern during the gestation period (P>0.05) (Table 10). This is contrary to the reports of others (Cromwell *et al.*, 1989; Dourmad 1991) who observed a positive relationship between pregnancy weight gain (associated with level of gestation feed intake) and lactation weight loss. Because gilts and sows consumed similar total levels of feed during pregnancy in this experiment, and body weight at the end of gestation was not different between gestation treatments, differences in lactation weight loss due to gestation treatment would not be expected.

The pattern of lactation feed intake resulted in lp animals having a higher average weight during lactation than the lc group (P<0.05). Koketsu *et al.* (1996) found that the pattern of lactational energy intake of gilts influenced weight loss during a three-week lactation period. Low energy intake during any week of lactation, or throughout lactation, resulted in loss of sow body weight. Gilt and sow weight change as illustrated in Appendix 3 was not affected by gestation or lactation treatment (P>0.05). However, considerable backfat loss during lactation (as observed for treatment gP), can be associated with a positive change in weight (Dourmad, 1991), or as observed in this experiment, no effect on weight loss. In contrast to the results of this trial, Dourmad (1991) found a positive relationship between gestation feed intake (gestation weight gain) and lactation weight loss, but no effect of gestation treatment on backfat loss during lactation. The study by Dourmad (1991) utilized different levels of total feed intake during gestation, contributing to a reduction in lactation feed intake for gilts on the high plane of gestation intake, resulting in the significant

response in lactational weight loss. Most sows lose some weight and backfat during lactation when fed at NRC (1988) recommended levels during gestation (Whittemore and Yang, 1989).

Body Protein

Maternal body composition can be predicted using backfat and weight measurements, and the levels of lipid and protein reserves are indicators of sow metabolic condition. The predicted maternal body protein content of gilts and first parity sows is shown in Table 11. Feed intake pattern during gestation had no effect on average maternal body protein content during lactation ($P>0.05$), and this corresponds with the absence of gestation treatment effects on mean weight or backfat during lactation.

Gilts and sows that were fed the control treatment during lactation (lc) had lower mean body protein content than lp sows ($P<0.05$). The higher level of feed intake, and resultant body weight, of the lp treatment during lactation explains the greater body protein content. Whittemore and Yang (1989) cited a strict relationship between body protein levels and changes in weight during lactation.

The interaction of gestation treatment*day of lactation was significant (Figure 8) ($P<0.05$). The difference in predicted maternal body protein content between gestation treatments on d 0 of lactation was not the same as the difference on d 17. This interaction suggests that gilts and sows which received the gC treatment during gestation lost a greater amount of body protein during lactation than treatment gP. It is possible that animals in the the gC treatment were slightly catabolic in late pregnancy due to the lower level of feed intake

at this time (2.84 ● 0.01 kg d^{-1}), predisposing them to greater protein loss during lactation. Catabolic condition of the sow during late gestation can affect lactation performance (Verstegen *et al.*, 1987).

All gilts and sows lost a significant ($P<0.05$), although moderate, amount of body protein during lactation. The average loss of 1.18 kg of body protein for gilts and first parity sows during the 17-day lactation period in this trial is acceptable when considering the 3 kg average protein loss during a 28-day lactation for parities one through four reported by Whittemore and Yang (1989).

Body Lipid

Predicted maternal body lipid content is presented in Table 11. As with maternal body protein, the mean predicted lipid content of the maternal body was not affected by gestation treatment ($P>0.05$). Lactation treatment significantly affected the mean body lipid content during lactation. Gilts and sows that received the lp treatment during lactation had higher mean body lipid during lactation than the lc treatment. This treatment effect was also observed for body weight and protein as discussed previously.

The interaction of gestation treatment*lactation treatment*day of lactation was significant (Figure 9). Comparison of the differences between treatment combinations on d 0 of lactation to d 17 showed that the difference between the treatment combinations was larger on d 17 of lactation. Gilts and sows that received the control treatment in gestation followed by the control treatment in lactation (treatment combination Cc), lost a greater

amount of body lipid during the 17-day lactation, and had the lowest lipid reserves on d 17. Treatment combination Cp appeared to lose the smallest amount of maternal lipid during lactation. The second gestation treatment (gP) in combination with lactation treatment c or p resulted in lipid loss during lactation similar to treatment Cc. In general, all gestation-lactation treatment combinations mobilized body lipid stores to support lactation demands. However, the extent of body lipid utilization during lactation was not affected by lactation treatment when gilts and sows received the P treatment during gestation. The loss of body fat due to mobilization of fat reserves is evident from the decrease in P2 backfat depth and the decrease in body lipid content during lactation. The patterns of lipid loss are similar to backfat losses during lactation due to the interaction of gestation treatment*lactation treatment*day of lactation.

Shields and Mahan (1983) reported that only maternal body fat reserves fluctuated during lactation, while body protein remained fairly constant. In contrast, other researchers observed substantial losses of body lipid and modest losses of body protein during lactation in sows (Whittemore and Yang, 1989; Dourmad, 1991). The composition of the loss varies according to parity of the sow. Multiparous sows mobilize fat reserves during lactation (due to larger labile lipid reserves), while primiparous sows catabolize both fat and protein reserves (Cole, 1990). In this experiment, first parity sows lost more body protein and lipid during lactation than gilts ($P<0.05$) (Appendix 4). Within parity, lipid losses were greater than protein losses for gilts and first parity sows.

Table 11. Predicted maternal body protein and lipid content (kg) of gilts and first parity sows during lactation

Factor		Body Protein (kg)[a]	Body Lipid (kg)[a]
Gestation Trt.		ns	ns
	C	30.84 ± 0.38	39.04 ± 0.92
	P	30.81 ± 0.35	40.74 ± 0.87
Lactation Trt		P = 0.0306	P = 0.0361
	c	30.26 ± 0.37	38.55 ± 0.91
	p	31.40 ± 0.36	41.24 ± 0.88
Parity[a]		P = 0.0001	P = 0.0001
	0	26.86 ± 0.35	34.39 ± 0.86
	1	34.79 ± 0.38	45.40 ± 0.93
Gest.*Lact.[b]		ns	ns
Gest.*Parity[b]		ns	ns
Lact.*Parity[b]		ns	ns
Gest.*Lact.*Parity[b]		ns	ns
Day		P = 0.0001	P = 0.0001
	0	31.42 ± 0.09	41.92 ± 0.28
	17	30.24 ± 0.09	37.86 ± 0.29

Table 11. **Predicted maternal body protein and lipid content (kg) of gilts and first parity sows during lactation (continued)**

Factor			Body Protein (kg)[c]	Body Lipid (kg)[c]
Gest.*Day			P = 0.0265**	ns[b]
	C	0	31.58 ± 0.14	
		17	30.10 ± 0.14	
	P	0	31.25 ± 0.13	
		17	30.37 ± 0.13	
Lact.*Day[b]			ns	ns
Gest.*Lact.*Day			ns[b]	P = 0.0181**
	Cc	0		39.78 ± 0.55
		17		34.59 ± 0.59
	Cp	0		41.92 ± 0.60
		17		39.89 ± 0.60
	Pc	0		41.97 ± 0.58
		17		37.83 ± 0.58
	Pp	0		44.01 ± 0.52
		17		39.13 ± 0.52
Parity*Day			P=0.0001[d]	ns[b]
Gest.*Parity*Day[b]			ns	ns
Lact.*Parity*Day[b]			ns	ns
Gest.*Lact.*Parity*Day[b]			ns	ns

Values are LS means ± SEM.
ns=non-significant at P>0.05.
[a]parity 0 (gilt), parity 1 (first parity sow).
[b]means not presented for these non-significant effects.
[c]Calculated using the equations of Whittemore and Yang (1989), using d 17 adjusted weight and backfat.
[d]means not presented for this effect.
**See Figures 8, 9.

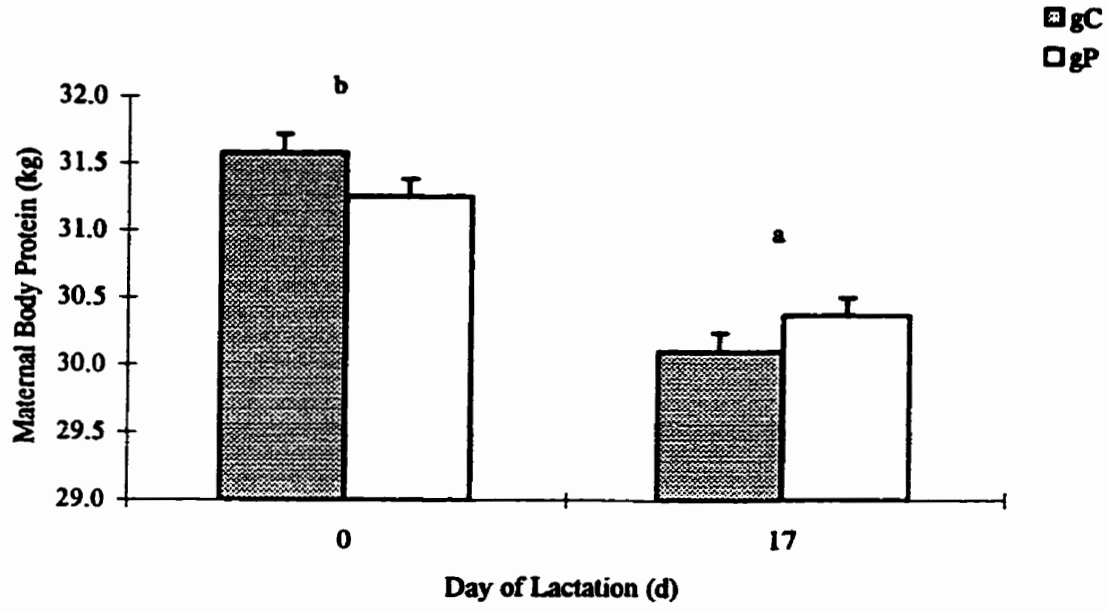

Figure 8. Gestation treatment*day of lactation interaction for predicted maternal body protein content of gilts and first parity sows (LS means + SEM).

[a]different letters indicate that the difference between treatments in these time periods are not the same (P<0.05).

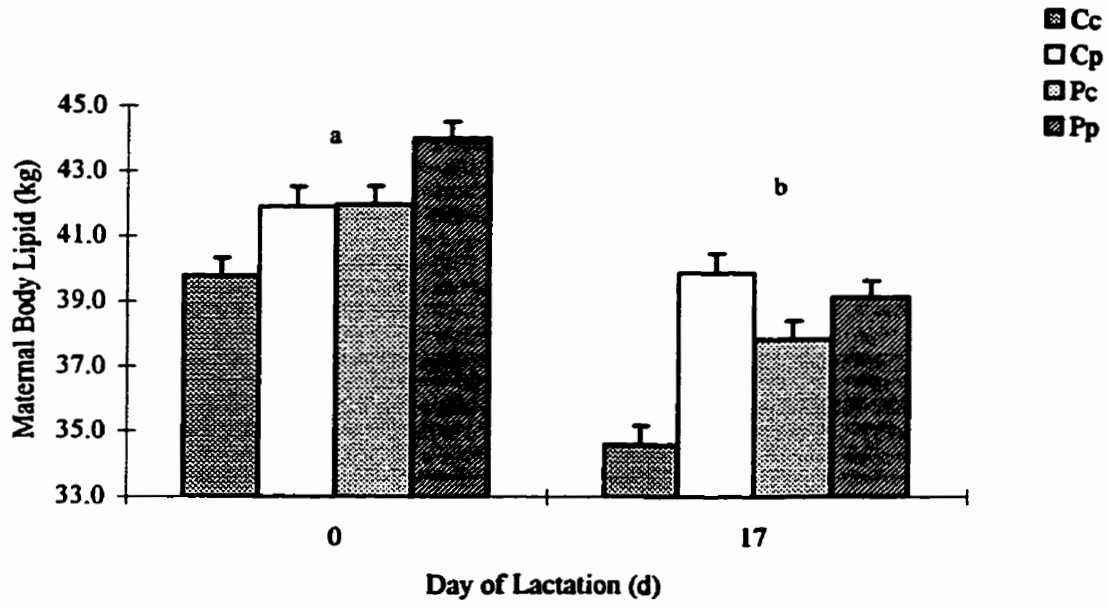

Figure 9. Gestation treatment*lactation treatment*day of lactation interaction for predicted maternal body lipid content of gilts and first parity sows (LS means + SEM).

[a]different letters indicate that the difference between treatments in these time periods are not the same (P<0.05).

Litter characteristics

Piglet Weight

Piglet performance may be affected by maternal nutrition during pregnancy and lactation (Neil and Ogle, 1996). Research has been conducted examining the effect of additional nutrients in late gestation with the assumption that the sow will partition these extra nutrients toward fetal growth (Britt, 1986). Cromwell *et al.* (1989) observed a positive effect of an additional 1.36 kg d^{-1} of feed from d 90 of gestation on piglet birth weight. Dietary protein restriction of the gilt decreased piglet birth weight (Pond *et al.*, 1992) and postnatal growth (Pond *et al.*, 1992; Schoknecht *et al.*, 1993). Protein restriction in late pregnancy also influenced milk production in lactation as evident by smaller piglets at weaning in the study by Schoknecht *et al.* (1993). Milk production of the sow is influenced by maternal body reserves at the beginning of lactation (Pomar *et al.*, 1991), and may be affected by nutrition during the period of mammary gland development in gestation (Weldon *et al.*, 1991). Feed intake, particularly in late lactation, affects milk yield (Mullan and Williams, 1989; Neil and Ogle, 1996). Sows with low feed intake in lactation have more difficulty maintaining milk production using their body reserves in late lactation. Therefore, piglet growth may be influenced by the effect of maternal nutrition on milk production in late lactation (King and Dunkin, 1986; Mullan and Williams, 1989).

Mean piglet weight during lactation was not affected by gestation or lactation treatment in this experiment ($P>0.05$) (Table 12). The interaction of gestation treatment*parity tended ($P=0.08$) to result in a greater difference in mean piglet weight between gestation treatments.

Table 12. Piglet weight (kg) during lactation

Factor			Mean Piglet Weight (kg)
Gestation Trt.			ns
		C	3.33 ± 0.06
		P	3.24 ± 0.06
Lactation Trt.			ns
		c	3.25 ± 0.06
		p	3.31 ± 0.06
Parity[a]			P = 0.0001
		0	3.01 ± 0.06
		1	3.56 ± 0.06
Gest.*Lact.[b]			ns
Gest.*Parity			ns (0.0792)
	C	0	3.13 ± 0.08
		1	3.53 ± 0.09
	P	0	2.88 ± 0.09
		1	3.59 ± 0.09
Lact.*Parity[b]			ns
Gest.*Lact.*Parity[b]			ns
Day[c]			P = 0.0001
		0	1.43 ± 0.05
		17	5.14 ± 0.05
Parity*Day[d]			P=0.0001
Gest.*Day[b]			ns
Lact.*Day[b]			ns
Gest.*Lact.*Day[b]			ns
Gest.*Parity*Day[b]			ns
Lact.*Parity*Day[b]			ns
Gest.*Lact.*Parity*Day[b]			ns

Values are LS means ● SEM.
ns=non-significant at P>0.05.
[a]parity 0 (gilt), parity 1 (first parity sow).
[b]means not presented for these non-significant effects.
[c]day17=piglet weight adjusted to 17-d lactation.
d 17wt. = ((weaning wt. - birth wt.)/lactation length) *17 + birth wt.
[d]means not presented for this effect.

for gilts compared to first parity sows. Piglets from gilts that had received the gC treatment tended to be heavier than gP piglets. Conversely, piglet weights were similar across gestation treatments for first parity sows.

Piglet performance during lactation was not affected by lactation treatment, possibly due to increased catabolism of maternal body reserves to maintain milk production. Yang *et al.* (1989) found that sows which were thin at parturition (12 mm backfat) and receiving 3 kg d^{-1} during lactation continued to mobilize their low fat reserves to maintain milk production when nutrition during lactation was inadequate.

Born Alive, Stillborn, Necrotics

Chi-square analyses of the litter characteristics in Table 13 show significant effects of gestation treatment, parity and treatment within parity. The chi-square test comparing the proportions of piglets born alive, stillborn, and necrotic was significant for gestation treatment. The proportion of piglets in each birth category was not the same for the gestation treatments. The number of piglets born alive as a proportion of the total number of piglets born was smaller for the gC treatment, and this is reflected by an increase in the proportions of stillborn and necrotic piglets. The proportion of piglets in the three categories also differed between gilts and first parity sows. First parity sows had a larger number of piglets born alive as a proportion of the total number of piglets born. This coincided with a reduction in the proportions of stillborn and necrotic piglets for the older sows.

Table 13. Litter characteristics of gilts and first parity sows

		Variable				
Factor		**Total Born**	**Born Alive**	**Stillborn**	**Necrotic**	χ^2
Gestation Trt.						5.99**
	C	665	0.90	0.06	0.04	
	P	636	0.95	0.03	0.02	
Parity						13.55**
	0	725	0.90	0.06	0.04	
	1	576	0.95	0.03	0.02	
Gilt						
	C	366	0.87	0.08	0.05	8.78**
	P	359	0.93	0.04	0.03	
First Parity						1.04ns
	C	299	0.95	0.03	0.02	
	P	277	0.96	0.02	0.02	

*χ^2 analysis: testing the hypothesis that the proportion of piglets in the three birth categories (born alive, still born, necrotics) are the same for the two groups for each factor (Gestation Trt., Parity, etc).
ns = non-significant: where $\chi^2_{0.05} > \chi^2$.
Rejection region: where $\chi^2 > \chi^2_{.05}$.
Number of litters (n) per treatment:
Gilts:
C n = 26
P n = 27
First Parity:
C n = 31
P n = 29

Analyzing each parity separately, gilts that were assigned the gC treatment had a lower proportion of piglets born alive, and larger proportions of stillborn and necrotic piglets compared to gilts fed at increasing levels throughout gestation (gP). Proportions of piglets in the three birth categories were not different for the two treatment groups for first parity sows. Conversely, gestation treatment did not have an effect on the proportions of piglets in each birth category for first parity sows. Differences in the number of piglets born alive to gilts may be related to uterine capacity in later gestation rather than feeding level per se.

Analysis of litter size at birth (Table 14) showed no effect of gestation treatment on the mean number of piglets born alive and total born. Parity did not significantly affect the number of piglets born alive. However, the total number of piglets born was greater for gilts compared to first parity sows ($P<0.05$). The number of piglets born alive in each gestation treatment was dependent on parity as illustrated by the significant interaction of gestation treatment*parity. The number of piglets born alive to gilts that had received the gP treatment was higher than for first parity sows fed in the same manner. Gilts receiving the gC treatment had fewer piglets born alive than gP gilts. The number of piglets born alive to first parity sows was similar for both gestation treatments. Pattern-feeding of gilts during pregnancy proved beneficial in terms of the number of piglets born alive. The interaction of gestation treatment*parity tended ($P=0.07$) to result in fewer total piglets born for first parity sows that were pattern-fed during gestation, while pattern-feeding tended to result in a greater total number of piglets born to gilts. These result indicate that gilts may be more sensitive than first parity sows to the effects of nutritionally-induced changes in concentrations of P_4 and

Table 14. Litter size at birth and weaning of gilts and first parity sows

Factor			Total Born	Born Alive	Weaned
Gestation Trt.			ns	ns	ns
		C	11.65 ● 0.31	10.57 ± 0.32	10.11 ● 0.15
		P	11.23 ± 0.32	10.72 ± 0.32	10.14 ● 0.15
Lactation Trt.			**	**	ns
		c			10.21 ± 0.15
		p			10.04 ● 0.15
Gest.*Lact.			**	**	ns[b]
Parity[a]			P=0.0133	ns	ns
		0	12.01 ± 0.33	10.90 ± 0.31	10.05 ± 0.15
		1	10.88 ± 0.33	10.39 ± 0.33	10.20 ± 0.16
Gest.*Parity			ns (0.0697)	0.0135	0.0149
	C	0	11.80 ± 0.43	10.26 ± 0.44	9.77 ± 0.20
		1	11.50 ± 0.47	10.88 ± 0.47	10.46 ± 0.23
	P	0	12.21 ± 0.44	11.55 ± 0.45	10.33 ± 0.21
		1	10.26 ± 0.46	9.89 ± 0.46	9.95 ± 0.22
Lact.*Parity			**	**	0.0453
	c	0			10.36 ± 0.21
		1			10.07 ± 0.22
	p	0			9.74 ± 0.21
		1			10.33 ± 0.23
Gest.*Lact.*Parity			**	**	ns[b]

Values are LS means ± SEM.
ns=non-significant at P>0.05.
[a]parity 0 (gilt), parity 1 (first parity sow).
[b]means not presented for these non-significant effects.
**lactation treatment not included in the model for variable born alive.

other pregnancy-specific proteins during early- to mid-pregnancy which are necessary for normal fetal development and survival. Close (1997) suggests that multiparous sows have higher blood P4 levels and this may explain why the negative relationship between feed intake in early gestation and embryo survival has not been observed in multiparous sows.

There was no effect of gestation treatment, lactation treatment or their interaction on the mean number of piglets weaned ($P>0.05$). Cromwell *et al.* (1989) found that additional feed in late gestation did not result in increased survival at weaning. A previous study by Pettigrew (1981) improved piglet survival at weaning by feeding supplemental fat to the sows in late gestation. However, survival was only improved if average survival from birth to weaning was less than 80%. The lack of response to additional feed in the study by Cromwell *et al.* (1989) may also be due to the relatively high survival (mean, 84%). Piglet survival to weaning in this trial (93%) may also explain the absence of treatment effects on litter size at weaning.

The interaction of gestation treatment*parity indicates that gilts responded differently to gestation treatment compared to first parity sows in the number of piglets weaned ($P<0.05$). Gilts that had received the gC treatment weaned fewer piglets compared to gP gilts. First parity sows weaned similar numbers of piglets regardless of gestation treatment. These results contradict information reported by Cromwell *et al.* (1989) where piglet survival to weaning was positively affected by birth weight (gestation feeding). However, the gC treatment tended ($P=0.08$) to have heavier piglets at birth than the gP treatment in the present

study. The larger litter size at weaning of gP gilts corresponds to the larger litter size at birth for this treatment.

The lactation treatment*parity interaction was also significant. The response to lactation treatment in terms of number of piglets weaned differed depending on parity of the dam. Gilts fed the lc treatment during lactation weaned more piglets than gilts restricted (lp) in feed intake, while the difference between lactation treatments was small for first parity sows. Therefore, lactation treatment did not seem to influence the number of pigs weaned by first parity sows.

Weaning-to-Estrus Interval

The length of the WEI is an important factor influencing sow productivity. Weaning of the sow is related to an increase in LH concentration and LH pulsatility (Einarsson and Rojkittikhun, 1993). The length of the WEI is influenced by sow body condition at farrowing and weaning, as well as the amount of tissue mobilized during lactation (Mullan and Williams, 1989; Yang *et al.*, 1989; Sterning *et al.*, 1990; Koketsu *et al.*, 1996). Sows that have lost larger amounts of body weight have extended WEI (Einarsson and Rojkittikhun, 1993).

Weaning-to-estrus intervals for gilts and first parity sows are presented in Table 15. Gestation treatment and lactation treatment had no effect on WEI of gilts and first parity sows ($P>0.05$). The absence of lactation treatment effect on WEI may be due to the fact that both lactation treatments resulted in a restriction in feed intake during early lactation. Koketsu *et*

al. (1996) found that energy restriction during lactation decreased LH pulsatility and extended the WEI.

The interaction of lactation*parity on WEI was significant (Figure 10). The difference in length of the WEI between lactation treatments was smaller and responded in a different manner for first parity sows than for gilts. The WEI of gilts and first parity sows that had received the control treatment (lc) during lactation were similar. However, gilts responded differently than sows to the pattern (lp) treatment during lactation. Gilts that had received the pattern treatment in lactation had longer WEI than first parity sows of the same treatment. Interpretation of these results leads to the conclusion that lp feeding of gilts more negatively affected the WEI than did this feeding strategy for first parity sows. Koketsu *et al.* (1996) found that restriction of energy intake during the first week of a 21-day lactation, adversely affected the WEI. Average daily feed intake of gilts during the first stage of lactation was 3.08 ± 0.10 and 2.87 ± 0.10 kg for lc and lp lactation treatments, respectively, while ADFI was 3.14 ± 0.10 and 3.38 ± 0.11 kg for first parity sows, lc and lp, respectively.

The 3-way interaction of gestation treatment*lactation treatment*parity was also significant (Figure 11). Gilts responded differently to the combinations of gestation-lactation treatments than first parity sows. In particular, the combination of Cp resulted in a lengthened WEI for gilts relative to first parity sows. The other treatment combinations did not produce these divergent effects between the two parities. Treatment combinations Cc, Pc, and Pp resulted in similar WEI for both gilts and first parity sows. The extended length of the WEI due to gestation-lactation treatment combination Cp does not agree with data concerning

backfat, weight, and body protein and lipid loss during lactation, and the relationship between body condition and WEI discussed earlier. These results indicate that loss of backfat and body lipid stores may not be as important in the regulation of resumption of estrus post-weaning.

Table 15. Weaning-to-estrus interval (WEI) (d) of gilts and first parity sows

Factor			WEI (days)
Gestation Trt.			ns
		C	8.06 ± 0.66
		P	7.35 ± 0.64
Lactation Trt.			ns
		c	7.55 ± 0.65
		p	7.86 ± 0.65
Parity[a]			P = 0.0001
		0	9.52 ± 0.63
		1	5.89 ± 0.67
Gest.*Lact.[b]			ns
Gest.*Parity[b]			ns
Lact.*Parity			P = 0.0332**
	c	0	8.37 ± 0.90
		1	6.73 ± 0.94
	p	0	10.67 ± 0.87
		1	5.05 ± 0.97
Gest.*Lact.*Parity			P = 0.0355**
	Cc	0	7.67 ± 1.23
		1	7.78 ± 1.27
	Cp	0	12.28 ± 1.27
		1	4.50 ± 1.50
	Pc	0	9.08 ± 1.32
		1	5.67 ± 1.37
	Pp	0	9.06 ± 1.19
		1	5.60 ± 1.23

Values are LS means ± SEM.
ns = non-significant, P>0.05.
[a]parity 0 (gilt), parity 1 (first parity sow).
[b]means not presented for these non-significant effects.
**See Figures 10, 11.

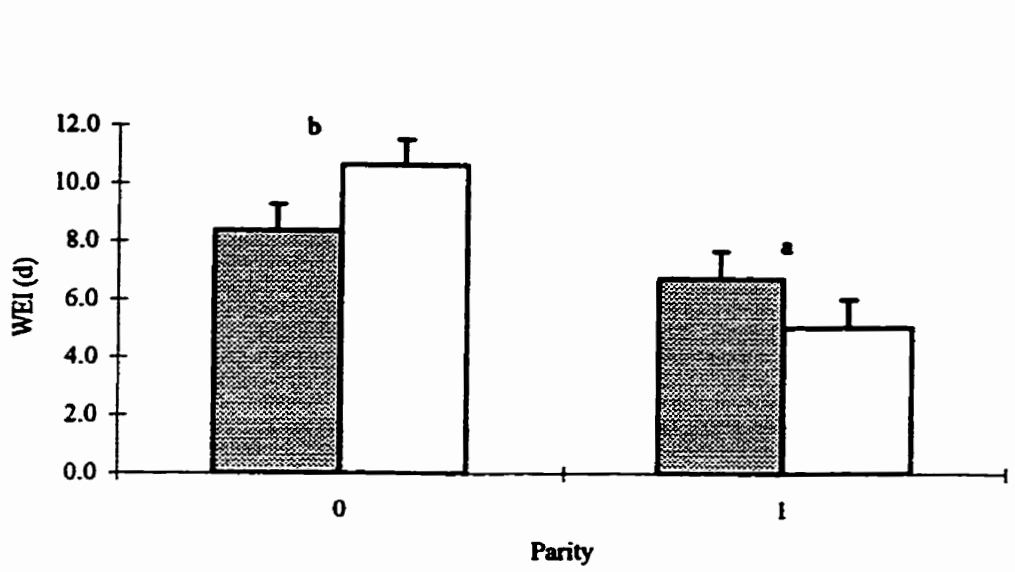

Figure 10. Lactation treatment*parity interaction for WEI of gilts and first parity sows (LS means + SEM).

[a]different letters indicate that the difference between treatments in these time periods are not the same (P<0.05).

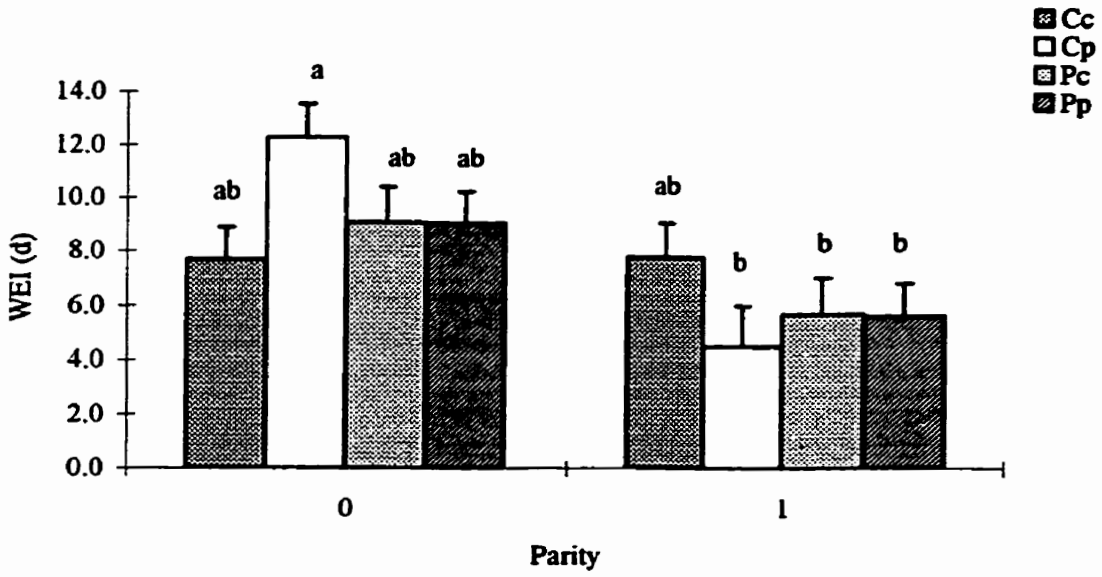

Figure 11. Gestation treatment*lactation treatment*parity interaction for WEI of gilts and first parity sows (LS means + SEM).

[a]different letters indicate differences in the length of the WEI (P<0.05).

CHAPTER 4

MANUSCRIPT II

REPRODUCTIVE PERFORMANCE OF EARLY-WEANED GILTS FED DIFFERING PATTERNS OF FEED INTAKE DURING GESTATION AND LACTATION

ABSTRACT

To study the effects of feed intake patterns during gestation and lactation on reproductive performance, 18 Cotswold gilts were randomly assigned to one of two gestation treatments and one of two lactation treatments. Throughout gestation, control gilts (gC; n=10) were fed at 1.4 times maintenance d^{-1}, and the pattern group (gP; n=8) was fed in four stages based on body weight at d 1, d 30, d 60, and d 90. During an 18-day lactation each gestation group was further divided, and control gilts (lc; n=9) were fed *ad libitum*, and the pattern group (lp; n=8) was fed in three stages based on body weight at d 1, d 6, and d 12.

Average daily feed intake (ADFI) during gestation did not differ ($P>0.05$) between treatments. Gestation treatment C consumed more feed in early gestation and overall ($P<0.05$). Although the patterns of backfat and body weight change were different ($P<0.05$), there were no differences between treatments by d 109. Gestation treatment influenced daily nutrient retention ($P<0.05$) but did not affect percent nutrient retention, serum urea nitrogen and progesterone (P_4) ($P>0.05$).

ADFI and total feed intake during lactation were not affected by gestation or lactation treatments ($P>0.05$). Lactation treatment did not result in differences in maternal weight, backfat or body composition at weaning. Patterns of body protein and lipid utilization differed ($P<0.05$). Treatments did not alter mean or baseline LH concentrations, pulse frequency, weaning-to-estrus interval, and ovulation rate ($P>0.05$). Gestation feeding method resulted in gP gilts supporting improved piglet growth rate in late lactation, a greater increase in P_4 concentration post-weaning, and 45% more normal corpora lutea than gC gilts ($P<0.05$).

INTRODUCTION

Nutrition during gestation and lactation, and the interactions between feed intake levels during these periods, play an important role in the reproductive performance of the gilt. Previous research demonstrates the importance of nutrition during pregnancy and lactation in order to meet the requirements for litter growth, milk production, and subsequent reproductive performance (Cole, 1990; Ashworth, 1991; Tokach *et al.*, 1992; Jindal *et al.*, 1996; Noblet *et al.*, 1997). The nutrient requirements for true growth occurring in the gilt and the additional requirements necessary to maintain production at a satisfactory level during the reproductive cycle, result in a unique situation with respect to the young sows susceptibility to the effects of inadequate nutrition on reproductive performance.

Nutritional influences on the metabolic state of the gilt during one stage of the production cycle will influence successive stages (Coffey *et al.*, 1994). Feed intake during gestation and its influence on maternal body composition affects voluntary feed intake and performance during lactation (Noblet *et al.*, 1990; Einarsson and Rojkittikhun, 1993), and post-weaning reproduction (Koketsu *et al.*, 1996; Zak *et al.*, 1997a). A sparing effect on nutrient requirements due to catabolism of body reserves of lipid and protein complicates the assessment of feed intake requirements from one stage of the reproductive cycle to the next.

Current sow feeding programs generally do not account for individual sow requirements. As a result, feed intake of young sows is often insufficient to maintain maternal body condition, milk production, and piglet growth, while providing the additional nutrients needed for maternal growth.

Inadequate feed intake during lactation affects body lipid and protein reserves and is associated with reduced litter growth during the lactation period (Brendemuhl *et al.*, 1989), prolonged WEI (Dourmad *et al.*, 1994), and reduced subsequent litter size (Kirkwood *et al.*, 1987b).

The objective of this experiment was to modify the feed intake patterns of gilts to reflect the changing maternal and piglet requirements throughout the reproductive cycle, and to assess the effects of these altered feed intake patterns on reproductive performance using metabolic, endocrine and production data.

MATERIALS AND METHODS

Experimental Design

Experiment II was performed at the Animal Science Research Unit (ASRU) located at the University of Manitoba, Fort Garry Campus. Twenty-four Cotswold gilts (175 days of age) were used in one experiment conducted during gestation and lactation. Each gilt was randomly assigned to one of two gestation treatments based on initial (pre-breeding) body weight. At farrowing, each gestation treatment was further subdivided based on gilt weight immediately post-farrowing, and gilts were randomly assigned to one of two lactation treatments. The length of the lactation period was 18 days.

Animal Housing: Gestation

Gilts were housed throughout the pre-breeding and gestation periods in individual pens in two rooms with eight pens (1.2 m wide x 2.4 m long) and 16 pens (1.2 m wide x 2.4 m long), respectively. Each pen had an individual feeder and waterer. All rooms used during the gestation phase of the study had light:dark cycles of 10:14 hours during the breeding period, and 12:12 hours during gestation. Room temperatures were set at 20°C.

Pre-Breeding: Estrous Synchronization

Gilts were weighed upon arrival in the ASRU and this initial weight was used to randomly assign them to a gestation treatment, and to allocate them to Regu-mate (Hoechst Canada Inc., Agriculture Division, SK) treatment groups as described below. The gilts were then randomly assigned to pens in one of the two rooms.

During the pre-breeding period gilts were fed 2.5 kilograms (kg) of a 16% crude protein diet once daily. Animals were randomly assigned to one of four groups for Regu-mate® administration to synchronize their estrous cycles. All gilts received 7.5 millilitres (ml) of Regu-mate®, administered using a drench gun, as a top dress with 25% of their daily ration for 14, 16, and 18 days (groups A, B, and C&D, respectively). Gilts were given Regu-mate® at 0800 h and the balance of their daily feed allotment one hour later. Beginning on d 12 of Regu-mate® treatment, the gilts were observed twice daily for signs of estrus using a boar as well as visual observation. Gilts were bred twice by artificial insemination with Cotswold line 30 mixed semen at first estrus following Regu-mate® withdrawal. The first insemination occurred when back pressure elicited a strong standing response. The second insemination occurred approximately twelve hours later. The day following the second insemination was designated as day one of gestation.

Experimental Treatments: Gestation

A 13.5% crude protein, barley-based, pelleted commercial dry sow ration (as in Experiment I) (supplied by Landmark Feeds) was fed to gilts once daily (0800 h) from the first day of gestation (d 1) to the day of farrowing (Table 1). Chromium oxide was included in the diet at 1 g kg^{-1} as an indigestible marker during periods of feces collection.

Gestation treatments differed in the assigned pattern of feed intake of the gilts. Treatment 1 (Control) (gC) gilts were fed at 1% of their body weight plus 0.7 kg of feed (Aherne, 1992), which was approximately 1.4 times their maintenance requirement, throughout gestation. Feed intakes were adjusted for changes in body weight at the end of each stage to maintain feed intake at a constant proportion of body weight (Table 1). The control treatment was designed to meet NRC (1988) requirements for pregnant gilts.

Treatment 2 (Pattern) (gP) gilts were fed in four increments during gestation with each stage being adjusted for gilt body weight (Table 1). Treatment 2 was designed to provide the same average feed intake over gestation as Treatment 1 (1.4 times maintenance). The stages were designed as follows: Stage I: 1.1 times maintenance (d 1 to d 30 of gestation), Stage II: 1.3 times maintenance (d 31 to d 60 of gestation), Stage III: 1.5 times maintenance (d 61 to d 90 of gestation), and Stage IV: 1.7 times maintenance (d 91 to farrow). The first two stages were designed to fall below NRC (1988) feed intake requirements for gilts during gestation. The final two stages exceeded NRC (1988) requirements.

Day one body weights were used to calculate gestation feed intakes for the first stage (d 1 to d 30). All gilts were weighed at the end of each stage and these weights were used to determine feed intake for the subsequent stage. Maintenance requirements for gestation were calculated using Equation 1.

$$\text{Equation 1: Feed intake (maintenance)} = \frac{\text{Metabolic BW} * 461 \text{ kJ kg}^{-0.75}}{\text{DE content of diet (kcal kg}^{-1}) * 4.18 \text{ kJ kcal}^{-1}}$$

Maintenance intakes were calculated using metabolic body weight, a maintenance energy allowance of 461 kJ of digestible energy (DE) $kg^{-0.75}$ (Jindal *et al.*, 1996), and the digestible energy content of the diet. This maintenance requirement was then multiplied by the corresponding factor for each stage to calculate feed intake. To convert metabolizable energy (ME) of the diets to DE, a factor of ME = 0.95DE was used. Gestation feed intakes were calculated using body weight rounded up to the nearest decimal place and grouped within a two kg weight range. The gilts received their assigned feed intake for that entire gestational stage. Any feed not consumed by the following morning feeding was weighed back and recorded.

Production Data: Gestation

Gilts were weighed using a portable scale (Gascoigne Pig Weigher, Gascoigne Readings, England) and had P2 backfat measurements taken at the last rib, 6.5 cm from the midline (Renco Lean-meater®, Renco Corporation, Minneapolis, MN), on d 1 of gestation and at the end of each stage (d 30, d 60, d 90 and d 109).

Commencing on d 30 of the experiment, ultrasound (Preg-Tone®)(Renco Corporation, Minneapolis, MN) was used to confirm pregnancy. Nine gilts that were not confirmed pregnant by ultrasound and returned to estrus after 21 days were rebred. Subsequently, six gilts were determined not pregnant and were removed from the experiment. On d 104 of gestation one gilt aborted her litter of 12 piglets. Therefore, data from this gilt was included until d 90 of gestation.

Metabolism Data: Gestation

Data on nitrogen and energy metabolism were collected at the end of each stage of gestation during a 48-hour collection period. The metabolism trials were staggered because there were only eight crates for 24 animals. Therefore, the collection periods commenced between d 5 to 8, d 25 to d 28, d 55 to d 58, and d 85 to d 88 of gestation. All gilts received the dry sow diet with chromium oxide added for five days prior to movement to the metabolism crates. The gilts were then placed in the metabolism crates one day in advance of the collection period.

Metabolism Crate Design

Eight metabolism crates (1.7 m length x 0.8 m height x 0.37 to 1.1 m adjustable width) were located in a third room with temperature and lighting conditions as previously described. Each crate had a feeder and waterer. The crate flooring was plastic coated

expanded metal (Tendernova™, Minneapolis, MN). Removable stainless steel trays were located beneath the flooring of the crate for urine collection. The trays sloped toward the front of the crate and a plastic stopcock was located at the deepest part of the tray to facilitate urine flow from the tray. Screens were mounted on frames to fit the inner dimensions of the tray to reduce fecal and feed contamination of the urine.

Excreta Collection

At 0800 h on the first day of collection the crates were thoroughly sprayed with water to remove any fecal and feed material. The crates were then allowed to drip dry.

Eighty ml of 1 M sulfuric acid was added to the tray prior to the start of each collection period. Urine was collected into pails by straining through cheesecloth (4-ply, grade 50, Veratec Inc. Graphic Arts Products, Wapole, MA). Total urine volume and weight was recorded. Urine was collected in this manner twice daily at 0800 h and 1700 h. Urine from the two-day collection period was pooled for each gilt and stored in individual pails in a fridge at 7°C. Four representative samples were collected in 20 ml vials from each gilt urine pool at the end of the 48-h collection. The vials were stored in a -20°C freezer until analysis.

Fresh fecal samples were collected twice daily throughout the 48-hour period and immediately placed in plastic freezer bags and frozen at -20°C until analysis. All fecal material collected over the two-day period was freeze-dried, mixed, and a representative sample was taken for each gilt. Feces and urine samples were later analyzed for dry matter, nitrogen and energy content.

Blood Sample Collection: Gestation

Single blood samples were taken from each gilt five hours after feeding (1300 h) on d 1, d 30, d 60, d 90, and d 109. The gilts were restrained using a wire nose snare. Blood samples were obtained from the jugular vein using 20-gauge, 1½ inch single-sample needles (Vacutainer, Becton Dickinson and Company, Franklin Lakes, NJ) and collected into 10 ml Vacutainer tubes for serum collection (Vacutainer, Franklin Lakes, NJ). Blood samples were stored overnight at 5°C. The following morning the samples were centrifuged at 1500 g for 30 min (CR3000, Jouan Inc., Winchester, VA) and the serum was separated, placed in glass vials, and frozen at -20°C until analysis. Gestation samples were analyzed for serum urea nitrogen and progesterone.

Animal Housing: Lactation

Gilts farrowed in individual farrowing crates (1.5 m width x 2.4 m length). The farrowing room held ten crates. Each crate had Tendernova™ flooring and individual feeders and waterers. Piglets were provided with heating pads (Stanfield, Osborne Industries Inc., KS) located on one side of the crate and did not have access to creep feed during lactation. Lights in the rooms were on continuously and room temperature was maintained at 23°C during lactation.

Two to three days after farrowing, gilts and litters were moved to pens located in an adjacent room. Each pen was 1.8 m width x 2.4 m length with an individual feeder and waterer and Tendernova™ flooring. A section of pen railing was attached to perpendicular walls in the back corner of each pen to form a creep area. A heat lamp was hung behind the partition for the piglets.

Experimental Treatments: Lactation

When farrowing was complete, each gilt was weighed and assigned to one of two lactation treatments. Gilts were assigned to lactation treatment in order to equalize the distribution of gestation treatment across lactation treatment. Therefore, all combinations of gestation-lactation treatment were represented.

During lactation all gilts were fed a 16% crude protein, barley-based, pelleted commercial nursing sow ration (as in Experiment I) (supplied by Landmark Feeds).

Treatment 1 (control) (lc) gilts were fed *ad libitum*. On d 1 of lactation *ad libitum* gilts were given an amount of feed equal to their final gestation stage daily feed intake. If all feed was consumed, extra feed was added each day in 0.5 kg increments to ensure *ad libitum* intake. In this way a small quantity of feed was always present in the feeder.

Treatment 2 (pattern) (lp) gilts were fed in three stages during lactation (Table 2). Each daily ration was split into two portions and the gilts were fed twice daily at 0800 h and 1600 h. The stages were designed as follows: Stage I: 1.9 times the maintenance

requirements (d 1 to d 6 of lactation), Stage II: 3.0 times maintenance (d 7 to d 12), and Stage III: 4.1 times maintenance (d 13 to d 18). Lactation feed intakes were calculated using Equation 1 based on a maintenance energy allowance of 461 kJ $kg^{-0.75}$ (Jindal *et al.*, 1996) and using the digestible energy content of the diet. Gilt weight taken at the end of each stage was used to calculate metabolic body weight. Lactation stage feed intakes were calculated using body weight rounded up to the nearest decimal place and grouped within a two kg weight range. Feed intakes were recorded for both treatments for the entire lactation period. Feed not consumed by the following morning was weighed and recorded. During the post-weaning period all gilts were fed 2.50 kg of feed daily at 0800 h. Gilts were observed twice daily for signs of estrus.

Production Data: Lactation

On d 109 of gestation, gilts were moved into the farrowing crates. Gilts were induced with two ml of Lutalyse® (intramuscular, im) (Upjohn Company, Animal Health Division, Orangeville, ON) at approximately 1400 h on d 114 of gestation. Eighteen h following Lutalyse® administration (at 0800 h the following morning) gilts received two ml of oxytocin im (Vetoquinol Canada Inc., Joliette, P.Q.).

Gilt Production Data

Gilts were weighed (Scale: Hiqual Manufacturing Ltd., MB) after farrowing and at the end of each lactation stage (d 6, d 12, and d 18). P2 backfat measurements were recorded on d 6, d 12, and d 18 of lactation. Lactation weight and backfat measurements were used to determine gilt body composition from prediction equations (equations 2 and 3) (Whittemore and Yang, 1989).

Piglet Production Data

At birth, piglets were weighed and given an identification number using a felt marker. Records at birth included: time of birth of each piglet, total born, total born alive, stillborn, and number of mummies. All cross-fostering was done within 24 h of birth. All piglets were weighed using a bucket placed on a scale, 24 h after birth, and on d 6, d 12 and d 18 of lactation. Piglet birth weights were assigned to their biological mother. Weaning weights were assigned to the foster mother. Pre-weaning mortality and the number of piglets weaned were recorded. Piglets were weaned from the sow before 1000 h on d 19.

Blood Sample Collection: Lactation

Post-farrowing Blood Samples

Single blood samples were taken from sows after farrowing by jugular venipuncture using the method described for blood sample collection during gestation in Experiment II.

Catheterization Procedure

On d 5 of lactation, gilts were catheterized for serial sampling to take place over the course of lactation. Catheters were placed in the central or lateral ear vein. This catheterization technique allowed for the repetitive blood sampling necessary for reproductive hormone analysis. Two gilts were not catheterized due to poor ear veins.

Gilts were restrained using a wire nose snare for the duration of the catheterization procedure. The ear and back of the gilts neck were shaved. An elastic band was placed around the base of the gilts ear to raise the ear vein and the ear was then swabbed with alcohol. A 14 gauge, 3 inch thin-walled needle was inserted into the central or lateral ear vein. Once the needle was in the ear vein, the catheter tubing (vinyl 70: ID 1.00 mm, OD 1.50 mm; Dural Plastics & Engineering, Australia) was slotted through the needle until the tip reached the vena cava. One metre of catheter tubing was used. The length of tubing required to reach the vena cava (approximately 0.5 m) was marked prior to insertion. The 14 gauge needle was removed by sliding it from the tubing. A blunt-end 19 gauge needle was placed in the end of the catheter and a PRN adapter injection cap (Becton Dickinson, Utah)

was used to close the end of the catheter by screwing it onto the blunt needle. Catheter patency was tested using heparinized saline (100 units ml^{-1}). A gauze square was taped over the exit point of the catheter. The exterior portion of the catheter was secured to the ear and neck using 2.5 cm white tape. Livestock glue (Ag-Tek Cement®, Kane Enterprises, SD) was spread along the path of the catheter, and Elastoplast® tape (three inch width) was used to cover the catheter and white tape. The catheter was secured to allow for movement of the ear, head, and neck of the animal. The catheter was then threaded into a Whirl-Pak® bag through a small hole cut at the bottom and the bag was glued to the back of the neck using livestock cement. White and Elastoplast® tape were used to cover the Whirl-pak® with an opening at the end to allow access to the catheter. White tape was used to cover this opening when the catheter was not in use. Catheter patency was maintained by flushing catheters twice daily with saline and filling the length of tubing with heparinized saline (100 units ml^{-1}) when not in use.

Serial Sampling Technique

Lactation Samples

Serial blood sampling periods during lactation occurred on d 6, d 12 and d 18. Blood samples were taken at 15-minute intervals for four hours starting at approximately 1400 h. Five ml of blood were drawn off and discarded before every sample to ensure that the samples weren't contaminated with heparinized saline. Ten-ml blood samples were taken using syringes and the blood collected was immediately placed in glass test tubes, covered with

parafilm, and stored in a basin of cool water. Between blood sampling intervals, 3 ml of saline was injected into the catheter and the injection cap was replaced. At the end of the collection period, 2-3 ml of heparinized saline was injected into the catheter and all blood samples were placed in a 4°C cool room overnight. The following morning the samples were centrifuged and separated as described for gestation.

Post-weaning Samples

Blood samples were taken every fifteen minutes during a 3 hour sampling period beginning at 1400 h on the day of weaning. At estrus, blood samples were collected from 10 remaining catheterized gilts at 15-minute intervals for four hours.

Estimation of Ovulation Rate

Approximately 5 - 10 d after returning to estrus following lactation, the gilts were slaughtered to obtain their reproductive tracts. To estimate ovulation rate, the number of corpora lutea (CL) present on each ovary were counted. The total number of CL (left plus right ovary) gave an estimation of the number of ova shed. The number of normal CL (pink to purple in colour, solid, vascular appearance) were also counted, excluding cysts and abnormal CL. Appearance of the CL, as well as numbers of ovulation stigma, follicles, corpora albicans and cysts were noted. Five gilts that showed no visible signs of estrus were slaughtered at d 15, d 20, d 30, and d 38 after weaning, respectively.

Analytical Techniques

Feed Analysis

A subsample of each diet was analyzed for nitrogen, energy, and dry matter. Feed samples were ground in a Tecator cyclotec 1093 sample mill (Hoganas, Sweden). Dry matter content was determined after drying samples in a vacuum oven at 105°C for 24 h. Dry matter and nitrogen content (Kjeltec Auto 1030 Analyzer, Hoganas, Sweden) were determined according to the Association of Official Analytical Chemists (AOAC, 1990). Gross energy was determined using an adiabatic oxygen bomb calorimeter (Parr, model 1241, Moline, IL).

Fecal Analysis

All fecal samples collected were freeze-dried (Virtis Consol 25LL, The Virtis Company, Gardiner, NY). Freeze-dried samples were ground from each collection period for each gilt. A subsample of fecal material was taken from each gilt during each period and analyzed for dry matter, nitrogen and energy content according to the AOAC (1990).

Urine Analysis

Urine samples were removed from the freezer and thawed at room temperature. Samples were filtered (#541 Whatman filter paper, Whatman International Ltd., Maidstone, England) and mixed.

To determine the nitrogen content of the urine sample, fresh urine was used. The AOAC (1990) method for nitrogen determination was used except that two ml of sample was pipetted into a protein tube.

To determine the energy content, 50 ml of previously frozen urine was added to two grams of Alphacel (ICN Biomedicals, Inc., Aurora, OH) in a petri dish and stirred to completely dissolve the Alphacel in the urine. A blank was also prepared and run at the same time using 50 ml of distilled water and two grams of Alphacel. Samples were placed in the freeze dryer for 48 hours at -45°C. Dried samples were ground using a mortar and pestle, transferred to plastic vials and stored in a dessicator until analysis. Pellets weighing one gram were made of the urine-alphacel mixture. Gross energy was determined by the AOAC (1990) method and corrected for the alphacel blank.

Hormone and Metabolite Analyses

Blood Urea

Serum samples from d 1, d 30, d 60, d 90, and d 109 of gestation, farrowing, d 6, d 12, d 18 of lactation, weaning (d 19), and estrus were analyzed for urea nitrogen concentrations using a standard kit (Procedure No.535) from Sigma Diagnostics (St. Louis, MO).

Urea concentration was measured without deproteinization of the samples. Twenty microlitres (µl) of serum was used to determine urea concentration. Standards ranged in value from 15 - 75 mg dl^{-1}. Samples, standards and controls were read at 540 nm within 20

minutes of removal from the water bath. Intraassay coefficients of variation were ≤9.6%. The interassay coefficient of variation was 3.3%. Blood urea nitrogen concentrations were expressed in mg dl^{-1}.

Progesterone

Serum samples from d 1, d 30, d 60, d 90, and d 109 of gestation, farrowing, d 6, d 12, d 18, weaning, estrus, and five days following estrus were analyzed for progesterone (P_4) concentrations using solid-phase radioimmunoassay (RIA) (Coat-A-Count progesterone kit, Diagnostic Products Corporation, CA). ^{125}I-labelled progesterone was used as the tracer with counts of 70000 cpm and maximum binding of ≤ 52.00 %. The standard curve range was 0.1 to 40 ng ml^{-1}. The method required 100 µl of standard or serum sample pipetted into anti-P_4 coated tubes. One ml of tracer was added to each tube. Tubes were decanted after incubation for three hours at room temperature to isolate the antibody-bound P_4. Radioactivity was measured by a gamma counter (LKB Wallac 1282 Compu Gamma Universal Gamma Counter). Nonspecific binding of the assay was ≤ 1.50 %. The sensitivity of the assay was 0.09 ng ml^{-1} at 90% binding. The intraassay coefficients of variation were 3.16 %, 5.15 %, and 3.82 %, for assays 1, 2, and 3, respectively. The interassay coefficient of variation was 5.56 %. Progesterone concentrations were expressed in ng ml^{-1}.

Luteinizing Hormone

Serum samples from farrowing (single samples) and serial samples from d 6, d 12, d 18 of lactation, weaning, and estrus were analyzed for luteinizing hormone (LH) concentration. Samples were analyzed at the University of Saskatchewan using RIA following the method described by Kingsbury and Rawlings (1993). Double-antibody RIA was used to determine LH concentration in 200 µl aliquots of serum. The initial antibody was raised in rabbits against bovine LH at a dilution of 1:40000. The second antibody was sheep-anti-rabbit gamma-globulins. Iodinated bovine LH was used as the tracer with 13000 - 18000 cpm in 200 µl.

Sensitivity of the assay was defined as the concentration of the lowest standard different from zero and was equal to 0.06 ng ml^{-1}. The intraassay and interassay coefficients of variation were ≤18.11% and 25.23%, respectively. Luteinizing hormone concentrations were expressed in ng ml^{-1}.

Equations Used in Determining Nutrient Intake and Retention

All calculations are expressed on dry matter basis and per day.

Energy Intake (kcal) = ((Energy in feed (kcal g^{-1})*dry matter intake (g))

Energy excreted in feces (kcal) = (fecal energy (kcal g^{-1})*weight of feces (g))/1000

Urinary energy (kcal) =
((total volume (ml)*urine energy (kcal g^{-1})*urine dry wt (g))/sample volume (ml))/2

$$\text{Energy Retention(\%)} = \frac{(\text{energy intake (kcal)} - \text{fecal energy (kcal)} - \text{urinary energy(kcal)})*100}{\text{energy intake (kcal)}}$$

Nitrogen Intake (g) = (Nitrogen in feed (%)*dry matter intake (g))

Nitrogen excreted in feces (g) = (fecal nitrogen (g)*weight of feces (g))/1000

Nitrogen excreted in urine (g) = (total volume (ml)*urinary nitrogen (g ml^{-1}))/2

$$\text{Nitrogen Retention (\%)} = \frac{(\text{nitrogen intake (g)} - \text{fecal nitrogen (g)} - \text{urinary nitrogen(g)})*100}{\text{nitrogen intake (g)}}$$

Statistical Analysis

Experiment II was analyzed as a completely randomized design during gestation (main effect: gestation treatment), and as a two-way factorial design during lactation (main effects: gestation treatment and lactation treatment) using the General Linear Model of the Statistical Analysis System (1986). The level of significance was defined as $P<0.054$. A trend was defined as $P=0.055 - 0.08$.

Gestation

Gestation Model: $y_{ij} = \mu + g_i + e_{ij}$

Where:

μ = mean.

g_i = gestation treatment effect, i =1 to 2.

e_{ij} = error.

To test for the effect of gestation treatment during gestation, average daily feed intake, sow weight and backfat, serum urea nitrogen, nitrogen and energy retention, and P_4 were analyzed as split plots. Repeated measures analysis was used with the gestation model and included the effects of stage of gestation (day), and the interaction of gestation treatment*day. The effect of gestation treatment was tested using sow within gestation treatment as the error term. When significant interactions occurred, contrasts were employed to determine differences between treatment groups over time.

Total feed intake for each stage of gestation, as well as sow weight and backfat change during each stage of gestation, were analyzed as a completely randomized design.

Lactation

Lactation model: $y_{ijk} = \mu + g_i + l_j + gl_{ij} + e_{ijk}$

Where:

μ = mean.

g_i = gestation treatment effect, i = 1 to 2.

l_j = lactation treatment effect, j = 1 to 2.

gl_{ij} = interaction of gestation treatment and lactation treatment, ij = 1 to 4.

e_{ijk} = error.

To test for the effects of gestation treatment and lactation treatment during lactation, average daily feed intake during lactation, sow weight and backfat, predicted maternal body lipid and protein composition, average piglet weight, serum urea nitrogen, LH, and post-weaning P_4 were analyzed as split plots. Repeated measures analysis was used for the above variables. The repeated measures model included stage of lactation (day), the interactions of gestation treatment*day, lactation treatment*day, and the 3-way interaction of gestation treatment*lactation treatment*day. The effects of gestation and lactation treatments were tested using sow within gestation treatment*lactation treatment as the error term. When a significant interaction occurred, contrasts were employed to determine differences between treatment groups over time. Differences between means existing at the start of lactation were defined using Bonferroni's test ($P<0.05$).

Total feed intake during each stage of lactation, sow weight and backfat changes and maternal body lipid and protein changes during each stage of lactation, as well as WEI, litter size at birth and weaning, and ovulation rate were analyzed as two-way factorials.

Prediction equations (Whittemore and Yang, 1989) were used to estimate total body protein and total body lipid of the gilts during lactation. The r^2 for protein and fat are $\geq$0.90 and $\geq$0.80, respectively. Body weight and backfat measurements were used from the beginning and end of lactation.

Equation 2: Protein (kg) = -2.3 + 0.19 live weight - 0.22 P2;

Equation 3: Lipid (kg) = -20.4 + 0.21 live weight + 1.5 P2;

Energy (kcal d^{-1}) and nitrogen intake (g d^{-1}), and energy (kcal d^{-1}) and nitrogen (g d^{-1}) excreted were calculated using equations reported previously. These values were then used to calculate energy retention and nitrogen retention for the end of each gestation stage.

Luteinizing hormone pulsatility and baseline concentrations were determined by the method of Evans *et al.* (1994). Serial samples for each gilt from each sampling period (ie. day of lactation) were analyzed separately. LH peaks were characterized as any point(s) greater than 3 standard deviations above the mean for that gilt. Baseline concentrations were defined as the average LH concentration when all points greater than 3 standard deviations had been removed.

RESULTS AND DISCUSSION

Gestation

Gestation Feed Intake

Average Daily Feed Intake

Gilt average daily feed intake (ADFI) is presented in Table 16. There was no effect of gestation treatment on ADFI during pregnancy (P>0.05). This result confirms the desired feeding strategy as the treatments were designed to provide equal gestational ADFI.

The interaction of gestation treatment*stage of gestation was significant (Figure 12) reflecting the imposed patterns of feed intake due to treatment. The difference in ADFI between gestation treatments was greater in early and late gestation, and smaller during mid-gestation. Control (gC) gilts fed at a constant proportion of their metabolic body weight throughout gestation consumed more feed than Pattern (gP) gilts during the first two stages of gestation. ADFI was similar for gC and gP treatment groups from d 61 to d 90 of gestation. During the final stage of gestation, gP gilts had higher ADFI than gC gilts.

Total gestation feed intake

Control gilts consumed more feed than gP gilts during the first two stages of gestation (P<0.05) (Table 17). Differences in total feed intake due to gestation treatment were not significant during the periods from d 61 to d 90 and from d 91 to farrowing (P>0.05). Feed

Table 16. Gestation ADFI (kg) of gilts

Factor			Average Daily Feed Intake[f] (kg d^{-1})
Gestation Trt.			ns
		C	2.24 ● 0.03
		P	2.18 ± 0.03
Stage			P = 0.0001
		1	1.83[a] ± 0.01
		2	2.02[b] ± 0.01
		3	2.31[c] ± 0.01
		4	2.67[d] ± 0.01
Gest.*Stage**			P = 0.0001
	C	1	2.06 ± 0.02
		2	2.14 ± 0.02
		3	2.29 ± 0.02
		4	2.45 ± 0.02
	P	1	1.61 ± 0.02
		2	1.89 ± 0.02
		3	2.32 ± 0.02
		4	2.88 ± 0.02

Values are LS means ● SEM.
ns = non-significant, P>0.05.
[a-d] within columns, means with unlike superscripts differ, P<0.05.
[e]Stage= gestation divided into 4 stages:
Stage1 = d 1 to d 30, Stage2 = d 31 to d 60, Stage3 = d 61 to d 90, Stage4 = d 91 to farrow.
[f]LS means are average daily feed intake for each stage of gestation:
Stage1 = average daily feed intake from d 1 to d 30, stage2 = d 31 to d 60, etc.
**See Figure 12.

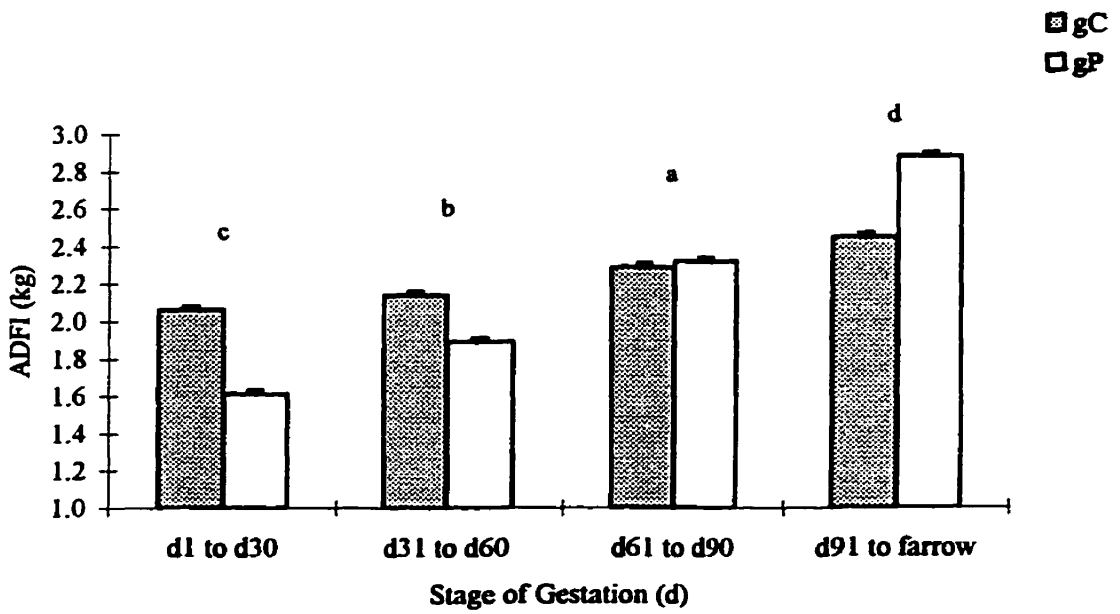

Figure 12. Gestation treatment*stage of gestation interaction for ADFI of gilts (LS means ± SEM).

[a]different letters indicate that the difference between treatments in these time periods are not the same ($P<0.05$).

Table 17. Total feed intake (kg) of gilts during each stage of gestation

		Stage of Gestation				
Factor		**d 1 to d 30**	**d 31 to d 60**	**d 61 to d 90**	**d 91 to farrow**	**d 1 to farrow**
Gestation Trt.		P = 0.0001	P = 0.0001	ns	ns	P = 0.0325
	C	61.77 ± 0.76	64.29 ± 0.74	68.70 ± 0.98	61.06 ± 2.65	255.82 ± 3.98
	P	48.34 ± 0.85	56.81 ± 0.83	69.71 ± 1.10	66.99 ± 2.97	241.86 ± 4.45

Values are LS means ± SEM.
ns = non-significant, P>0.05.

intake during the period from d 91 to parturition was not affected by gestation treatment ($P>0.05$), although the intention was for the gP treatment to consume more feed during this stage. Total feed intake during pregnancy was significantly affected by gestation treatment ($P<0.05$). Control gilts consumed 13.96 kg more feed than gP gilts, although total feed intake was designed to be equal for the two gestation treatments. The total number of days from d 1 of gestation to farrowing were 114.90 ● 0.92 and 113.25 ± 1.02 days for gC and gP gilts, respectively. Although the length of the gestation period did not differ ($P>0.05$) between treatments, comparison of ADFI and total feed intake for the different stages of gestation indicate that length of the gestation period may have influenced total feed intake. Treatment differences were consistent when comparing ADFI to total feed intake results for the first three stages of gestation. However, examination of the final stage of gestation shows that ADFI was greater for gP gilts. Total feed intake values for this period were not significantly different indicating that the shorter gestation length of the gP gilts resulted in total feed intake during the final stage of gestation below expected levels, and a reduction in total feed intake during gestation. Lower body weight of the gP gilts during mid- to late gestation may also have contributed to the reduction in total feed intake.

Body Composition

Backfat

Gilt P2 backfat measurements during gestation are presented in Table 18. Imposed pattern of feed intake did not affect P2 backfat levels (P>0.05). The level of backfat depth at parturition (mean, 17.21 ● 0.32 mm) was lower than the recommended level of 20 mm for gilts (Yang *et al.*, 1989; Aherne and Williams, 1992). The ADFI of 2.24 ± 0.03 and 2.18 ● 0.03 kg d^{-1} for gC and gP treatments may have been inadequate to achieve the target backfat levels. As well, variability associated with the measurement of backfat may have precluded the observation of treatment effects during individual stages of gestation (Mullan, 1991). Gestation feed intake of 3 kg d^{-1} was recommended by Yang *et al.* (1989) to achieve target backfat depth at farrowing. Changes in backfat depth are shown in Appendix 5.

Weight

Gilt body weight is shown in Table 18. There was no effect of gestation treatment on mean gestation weight during pregnancy (P>0.05). Due to the lack of difference in ADFI and total feed intake during gestation, differences in mean weight due to gestation treatment would not be expected.

The interaction of gestation treatment*day of gestation was significant (Figure 13), reflecting the effect of treatment feed intake patterns on maternal weight gain. There was no difference in maternal body weight between treatments gC and gP on d 1 of gestation. The

Table 18. Gilt P2 backfat (mm) and weight (kg) during gestation

Factor			P2 Backfat (mm)	Weight (kg)
Gestation Trt.			ns	ns
		C	15.85 ± 0.59	152.19 ± 2.68
		P	16.20 ± 0.67	146.64 ± 3.00
Day			P = 0.0001	P = 0.0001
		1	$13.93^{a} \pm 0.27$	$129.55^{a} \pm 1.05$
		30	$15.51^{b} \pm 0.27$	$132.56^{a} \pm 1.05$
		60	$16.46^{bc} \pm 0.27$	$144.51^{b} \pm 1.05$
		90	$17.01^{c} \pm 0.27$	$161.32^{c} \pm 1.05$
		109	$17.21^{c} \pm 0.32$	$179.12^{d} \pm 1.13$
Gest.*Day			ns	P = 0.0047**
	C	1	13.55 ± 0.36	129.90 ± 1.40
		30	15.65 ± 0.36	136.99 ± 1.40
		60	16.55 ± 0.36	149.45 ± 1.40
		90	16.65 ± 0.36	164.95 ± 1.40
		109	16.86 ± 0.42	179.64 ± 1.49
	P	1	14.32 ± 0.41	129.20 ± 1.57
		30	15.37 ± 0.41	128.12 ± 1.57
		60	16.37 ± 0.41	139.56 ± 1.57
		90	17.37 ± 0.41	157.69 ± 1.57
		109	17.55 ± 0.49	178.60 ± 1.70

Values are LS means ± SEM.
ns = non-significant, P>0.05.
$^{a-d}$ within columns, means with unlike superscripts differ, P<0.05.
**See Figure 13.

differences between treatments on d 30, d 60 and d 90 of gestation were greater than the initial difference. Body weight did not differ between treatments on d 109 of gestation. The pattern of weight gain of the gilts during mid- to late gestation was dependent upon treatment. The difference in weight between treatments increased as a result of a greater rate of weight gain by the gC group during mid- to late gestation. From d 90 to d 109 of gestation, the gP treatment increased in weight to attain a similar weight at d 109. The significance of the interaction coincides with the administered patterns of feed intake, and is similar to the relationship between gilt weight gain and gestation feed intake reported in Manuscript 1. Values for gestation weight change are presented in Appendix 6, and illustrate the pattern of weight gain by the gC group and weight loss by gP gilts in early gestation as shown in Figure 13. Since the requirements of reproductive tissue and fetuses are low during this period of pregnancy (Close *et al.*, 1984; Noblet *et al.*, 1997) it is reasonable to assume that the feed intake of the gP group was insufficient to support maintenance and maternal (or true) growth. During the second stage of gestation both treatments gained similar amounts of weight ($P>0.05$). The final stage of gestation (d 90 to d 109) resulted in gP gilts gaining more weight than gC gilts ($P<0.05$). The shift from weight loss in early gestation to weight gain by the gP treatment may help to explain the relatively slow increase in weight of this treatment before d 90 of gestation. Over the entire gestation period, there was no difference in weight change (gain) between treatments ($P>0.05$). The increase in weight during gestation of approximately 50 kg agreed with the maternal plus reproductive gain of 45 - 60 kg suggested by Verstegen and Den Hartog (1989) and Aherne and Williams (1992).

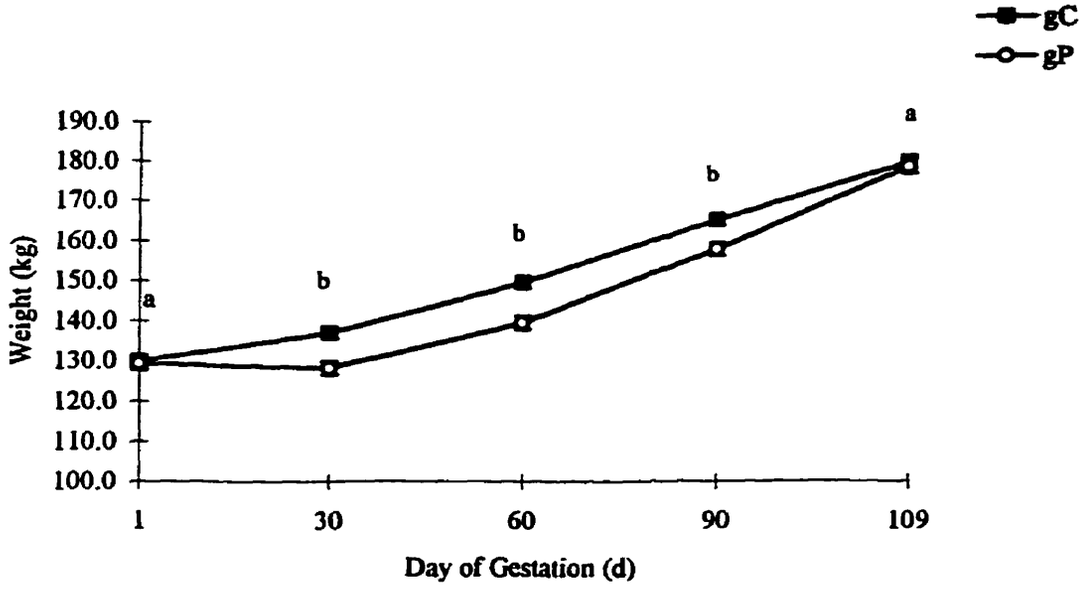

Figure 13. Gestation treatment*day of gestation interaction for gilt weight (LS means ± SEM).
*different letters indicate that the difference between treatments in these time periods are not the same ($P<0.05$).

Nutrient Retention

Values of mean daily nitrogen and energy excretion in feces and urine are shown in Table 19. Energy and nitrogen retained per day are also presented.

Energy

Fecal energy excretion was influenced by gestation treatment, with gC gilts excreting more energy in their feces than gP gilts ($P<0.05$). Urinary energy excretion tended ($P=0.06$) to be greater in gC gilts than gP gilts. The amount of energy retained per day was greater for gC gilts than for gP gilts ($P<0.05$).

The interaction of gestation treatment*day of gestation had a significant effect on energy retention (Figure 14). A difference in energy retention between treatments existed on d 5 of gestation ($P<0.05$), with gC gilts retaining more energy per day than gP gilts. The difference between treatments on d 90 of gestation was smaller and not the same as the initial difference (d 5). While energy retention of the gC gilts remained at a relatively constant level, gP gilts continued to retain increasing amounts of energy as gestation progressed. The pattern of energy retention of the gC treatment was consistent with the administration of the feeding level for this group. Maintaining feed intake at a level of 1.4 times the maintenance requirement resulted in a uniform level of energy retention per day. Several studies (Close *et al.*, 1985; Noblet and Etienne, 1987; Noblet *et al.*, 1997) report a decrease in energy retention with pregnancy when gilts are fed at constant levels (due to an increase in metabolic

body weight and therefore an increase in the maintenance energy requirement). The adjustment of feed intake at the start of each stage (based on metabolic body weight) for gC gilts resulted in a constant level of energy retention. The pattern of energy retained per day for the gP treatment group reflects the increasing level of feed intake times maintenance during gestation.

Nitrogen

Mean fecal nitrogen (N) excretion per day was also affected by gestation treatment ($P<0.05$). Control gilts had higher fecal N excretion than gP gilts during gestation ($P<0.05$). Dunn and Speer (1991) found that fecal N excretion increased as daily N intake increased, and with progression of pregnancy.

Mean daily urinary nitrogen excretion was not significantly affected by gestation treatment, day, or their interaction. Increased urinary N excretion with an increase in N intake had been reported in some studies (Dunn and Speer, 1991). However, urinary nitrogen excretion in this study was highly variable. Mean daily N retention was not significantly altered by gestation treatment.

The interaction of gestation treatment*day was significant (Figure 15) and illustrates an initial difference (d 5) between treatments in nitrogen retention ($P<0.05$) as observed for energy retention in Figure 14, with gC gilts retaining more N per day than gP gilts. The interaction between treatment and day can be explained by a steady level of N retention during pregnancy by gC gilts, while gP gilts exhibited a sharper increase in N retention

beyond d 30. As a result, the difference in N retention between treatments on d 90 of gestation was not the same as the initial difference (d 5), with the daily N retention values of gP gilts increasing above the gC group. The pattern of increase observed in N retention of gP gilts is supported by Dunn and Speer (1991) who found that the pattern of N retention increased with an increase in feed intake. Nitrogen retention values during pregnancy in this experiment were in the range of 10 - 16 g d^{-1} reported by Everts and Dekker (1994).

Table 19. Daily fecal and urinary nutrient excretion during gestation

Factor		Variable[d]					
		Fecal Energy Excreted (kcal d^{-1})	Urine Energy Excreted (kcal d^{-1})	Energy Retained (kcal d^{-1})	Fecal Nitrogen Excreted (g d^{-1})	Urine Nitrogen Excreted (g d^{-1})	Nitrogen Retained (g d^{-1})
Gestation Trt.		P = 0.0061	ns (0.0564)	P = 0.0031	P = 0.0316	ns	ns
	C	2519.36 ± 72.26	191.25 ± 8.34	5674.25 ± 136.55	12.65 ■ 0.48	18.19 ± 0.89	17.48 ± 1.10
	P	2181.73 ± 78.93	166.07 ± 8.96	4971.52 ± 149.16	10.97 ± 0.53	16.01 ± 0.99	15.37 ± 1.20
Day		P = 0.0013	ns	P = 0.0001	P = 0.0001	ns	P = 0.0001
	5	2162.06[a] ± 94.02	180.07 ± 9.57	4870.10[a] ± 98.29	10.31[a] ± 0.50	17.21 ± 0.88	14.20[a] ± 1.06
	30	2114.48[a] ± 98.87	165.75 ± 9.57	4945.70[a] ± 103.37	10.64[a] ± 0.53	16.86 ± 0.88	13.97[a] ± 1.12
	60	2498.16[ac] ± 98.87	176.72 ± 9.57	5262.50[a] ± 103.37	12.85[b] ± 0.53	17.23 ± 0.88	16.18[a] ± 1.12
	90	2627.48[bc] ± 105.28	192.10 ± 10.26	6213.23[b] ± 110.06	13.44[b] ± 0.56	17.11 ± 0.88	21.33[b] ± 1.19
Gest.*Day		ns	ns	P = 0.0001*	ns	ns	P = 0.0321
C	5	2407.95 ± 125.35	197.37 ± 12.76	5486.77 ± 131.05	11.62 ± 0.67	18.35 ± 1.17	16.84 ± 1.42
	30	2285.24 ± 125.35	174.14 ± 12.76	5632.71 ± 131.05	11.55 ± 0.67	18.51 ± 1.17	16.76 ± 1.42
	60	2765.38 ± 125.35	191.29 ± 12.76	5465.55 ± 131.05	14.05 ± 0.67	18.18 ± 1.17	16.48 ± 1.42
	90	2618.86 ± 157.14	202.22 ± 14.74	6111.96 ± 164.28	13.38 ± 0.84	17.72 ± 1.17	19.83 ± 1.78
P	5	1916.17 ± 140.15	162.78 ± 14.27	4253.43 ± 146.52	8.99 ± 0.75	16.08 ± 1.31	11.57 ± 1.58
	30	1943.72 ± 152.94	157.35 ± 14.27	4258.69 ± 159.90	9.74 ± 0.82	15.20 ± 1.31	11.19 ± 1.73
	60	2230.94 ± 152.94	162.15 ± 14.27	5059.44 ± 159.9	11.65 ± 0.82	16.29 ± 1.31	15.88 ± 1.73
	90	2636.10 ■ 140.15	181.98 ± 14.27	6314.50 ± 146.52	13.50 ± 0.75	16.49 ± 1.31	22.84 ± 1.58

Values are LS means ± SEM.
ns = non-significant, P>0.05.
[a-c] within columns, means with unlike superscripts differ, P<0.05.
[d]Values are means calculated from total collection over a 48-hr period.
*See Figures 14, 15.

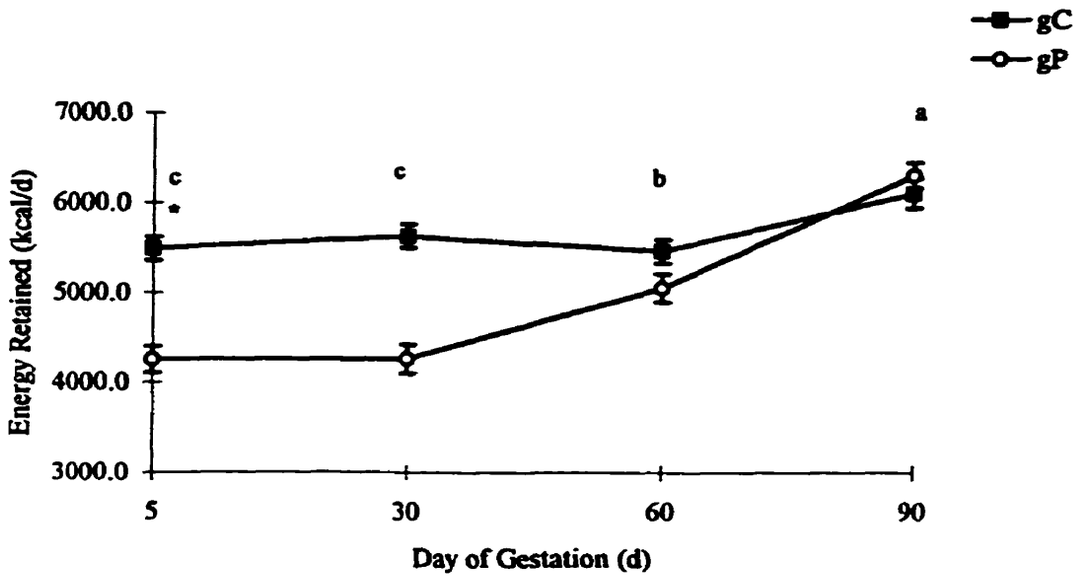

Figure 14. Gestation treatment*day of gestation interaction for daily energy retention of gilts (LS means ± SEM).

[a]different letters indicate that the difference between treatments in these time periods are not the same (P<0.05).

*denotes a significant difference between treatments within day (P<0.05).

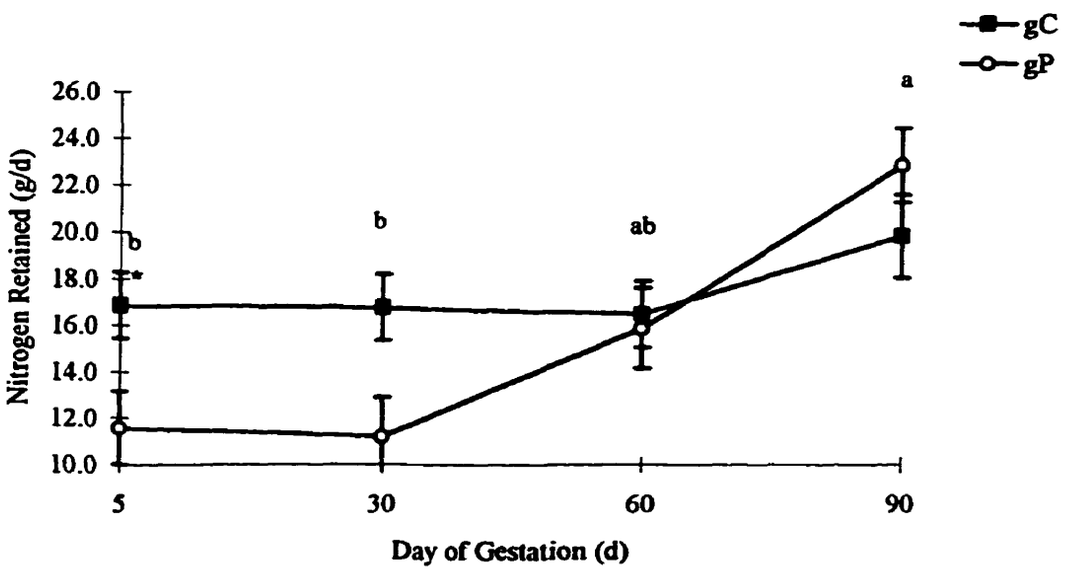

Figure 15. Gestation treatment*day of gestation interaction for daily nitrogen retention of gilts (LS means ± SEM).

[a]different letters indicate that the difference between treatments in these time periods are not the same (P<0.05).

*denotes a significant difference between treatments within day (P<0.05).

Table 20. Nutrient utilization by gilts during gestation

Factor			Energy Retained (%)[a]	Nitrogen Retained (%)[a]
Gestation Trt.			ns	ns
		C	67.55 ● 1.08	35.85 ± 2.07
		P	67.79 ± 1.18	35.19 ± 2.27
Day			ns	ns
		5	67.47 ± 1.21	33.71 ± 2.15
		30	68.30 ± 1.27	32.71 ± 2.26
		60	66.23 ± 1.27	35.10 ± 2.26
		90	68.67 ± 1.35	40.56 ± 2.41
Gest.*Day			ns	ns
	C	5	67.79 ± 1.61	35.94 ± 2.87
		30	69.56 ± 1.61	35.62 ± 2.87
		60	64.58 ± 1.61	33.49 ± 2.87
		90	68.25 ± 2.02	38.34 ± 3.59
	P	5	67.15 ± 1.80	31.47 ± 3.21
		30	67.04 ± 1.96	29.79 ± 3.50
		60	67.88 ± 1.96	36.72 ± 3.50
		90	69.10 ± 1.80	42.78 ± 3.21

Values are LS means ± SEM.
ns = non-significant, P>0.05.
[a]Values expressed as percent of nutrient intake.

Percent Energy and Nitrogen Retention

Nutrient retention expressed as a percent of nutrient intake is presented in Table 20. Gestation treatment, day and their interaction had no effect on energy or nitrogen retention as a percent of nutrient intake ($P>0.05$). Therefore, the significant differences in E and N retention (daily values) (Table 19) can be explained by treatment differences in daily feed intake.

Serum Urea Nitrogen

Catabolism of amino acids from exogenous and endogenous sources results in elevated serum urea N concentrations (Eggum, 1970; Cai *et al.*, 1995; Chen *et al.*, 1995). In this experiment, serum urea N levels during pregnancy were not influenced by gestation feed intake pattern ($P>0.05$) (Table 21).

The observed increases in nutrient retention, gain in weight and backfat and the lack of treatment-induced differences in mean body condition, nutrient retention, and serum urea N levels indicate that the gilts in both treatments received sufficient nutrients during gestation to support satisfactory pregnancy gain.

Table 21. Mean serum urea nitrogen (mg dl^{-1}) of gilts during gestation

Factor			Urea Nitrogen (mg dl^{-1})
Gestation Trt.			ns
		C	14.58 ● 0.38
		P	15.16 ● 0.44
Day			P = 0.0001
		1	$18.58^{a} \pm 0.43$
		30	$14.16^{b} \pm 0.43$
		60	$13.22^{b} \pm 0.45$
		90	$14.94^{b} \pm 0.45$
		109	$13.43^{b} \pm 0.47$
Gest.*Day			ns
	C	1	17.74 ± 0.58
		30	13.83 ± 0.58
		60	13.16 ± 0.58
		90	14.71 ± 0.58
		109	13.45 ± 0.61
	P	1	19.42 ● 0.64
		30	14.49 ± 0.64
		60	13.27 ± 0.70
		90	15.16 ± 0.70
		109	13.42 ± 0.70

Values are LS means ● SEM.
ns = non-significant, P>0.05.
$^{a-b}$ within columns, means with unlike superscripts differ, P<0.05.

Progesterone

Gestation treatment had no effect ($P>0.05$) on mean serum P_4 concentration during pregnancy (Table 22). Nutritionally-induced differences in P_4 concentration may affect reproductive performance of the gilt. These possible P_4-mediated negative effects on embryo survival occur in the very early stages of pregnancy (Jindal *et al.*, 1996; Jindal *et al.*, 1997). Due to the schedule of blood collection in this experiment, differences in serum P_4 concentrations due to gestation treatment, and potential influences on metabolic clearance of P_4 were not investigated.

All gilts exhibited a sharp rise in P_4 concentration by d 30 of gestation and a decrease in concentration between d 109 consistent with the pattern of secretion described by Dyck *et al.* (1980).

Table 22. Mean serum progesterone (P_4) (ng ml^{-1}) of gilts during gestation

Factor			P_4 (ng ml^{-1})
Gestation Trt.			ns
		C	15.25 ± 0.84
		P	14.44 ● 0.96
Day			P = 0.0001
		1	1.61^{a} ± 0.96
		30	19.25^{b} ± 0.96
		60	21.03^{b} ± 0.96
		90	17.52^{bc} ± 1.01
		109	14.81^{c} ± 1.03
Gest.*Day			ns
	C	1	1.42 ± 1.28
		30	19.96 ± 1.28
		60	21.40 ● 1.28
		90	17.68 ● 1.28
		109	15.78 ± 1.37
	P	1	1.81 ± 1.43
		30	18.53 ± 1.43
		60	20.66 ± 1.43
		90	17.37 ± 1.55
		109	13.84 ± 1.55

Values are LS means ± SEM.
ns = non-significant, P>0.05.
$^{a-c}$ within columns, means with unlike superscripts differ, P<0.05.

LACTATION

Lactation Feed Intake

Average Daily Feed Intake

Average daily feed intake (ADFI) of the gilts during lactation is shown in Table 23. Gestation treatment did not influence ADFI during the lactation period ($P>0.05$). In a study by Cromwell *et al.* (1989), additional feed from d 90 of gestation to farrowing, resulting in increased total gestation feed intake, did not influence ADFI during lactation. The influence of pattern of feed intake during gestation on feed intake in the subsequent lactation has not been extensively studied. Treatment similarities in feed intake during pregnancy, and the absence of treatment differences in sow weight or backfat depth at d 109, may explain why the negative relationship between gestation and lactation feed intake was not observed in this experiment. Although total gestation feed intake of gC gilts was 13.96 kg (5.8 %) greater than total feed intake of the gP group, this increase in feed intake was probably insufficient to promote a decrease in subsequent lactation feed intake.

Mean ADFI during lactation was similar for both lactation treatments ($P>0.05$) and ADFI increased with the progression of lactation for both treatments ($P<0.05$). The interaction of lactation treatment*stage of lactation was significant ($P<0.05$) (Figure 16). Gilts that were allowed *ad libitum* access to feed from the start of lactation (lc) had higher ADFI in early lactation than gilts that were restricted in feed intake (lp), resulting in a greater

difference between treatments in the first stage of lactation. This level of feed intake of the *ad libitum* group was maintained throughout lactation. In contrast, Koketsu *et al.* (1996a) characterized feed intake patterns of lactating sows and found that higher feed intake during early lactation was associated with the occurrence of drops in feed intake in lactation. Decreases in feed intake during the lactation period were not associated with *ad libitum* feeding during this trial. Moser *et al.* (1987) found that sows fed *ad libitum* from d 0 of lactation had low feed intake following parturition and reached maximum feed consumption by d 3 of lactation and consumed 10 % more feed during the lactation period than sows which were restricted (where *ad libitum* intake was attained by d 6 of lactation). The difference between lactation treatments (c and p) during the first stage of lactation (d 1 to d 6) was greater than the difference between treatments during the period from d 7 to d 12 of lactation. ADFI of the lc and lp lactation treatments was the same in mid-lactation, which would indicate that *ad libitum* intake was approximately 3.0 times maintenance. The imposed pattern of feed intake of lp gilts is reflected in Figure 16, with the difference between treatments for the final 6 d of lactation greater than the previous differences. ADFI of lp gilts increased throughout lactation, resulting in higher ADFI for lp gilts during the last stage of lactation compared to the *ad libitum* group.

Table 23. Lactation ADFI (kg) of gilts

Factor			Average Daily Feed Intake[f] (kg d^{-1})
Gestation Trt.			ns
		C	4.38 ± 0.16
		P	4.35 ± 0.19
Lactation Trt.			ns
		c	4.40 ± 0.18
		p	4.33 ● 0.18
Gest.*Lact.			ns
	C	c	4.41 ± 0.21
		p	4.35 ± 0.25
	P	c	4.39 ± 0.29
		p	4.32 ● 0.25
Stage[d]			P = 0.0001
		1	3.48[a] ± 0.19
		2	4.46[b] ± 0.19
		3	5.15[c] ± 0.19
Gest.*Stage[e]			ns
Lact.*Stage			P = 0.0001**
	c	1	4.20 ● 0.26
		2	4.57 ● 0.26
		3	4.42 ± 0.26
	p	1	2.76 ± 0.26
		2	4.36 ± 0.26
		3	5.87 ± 0.26
Gest.*Lact.*Stage[e]			ns

Values are LS means ● SEM.
ns=non-significant, P>0.05.
[a-c] within columns, means with unlike superscripts differ, P<0.05.
[d]Stage= lactation divided into 3 stages:
stage1 = d 1 to d 6, stage2 = d 7 to d 12, stage3 = d 13 to d 18.
[e]means not presented for these non-significant effects.
[f]LS means are average daily feed intake for each stage of lactation.
**See Figure 16.

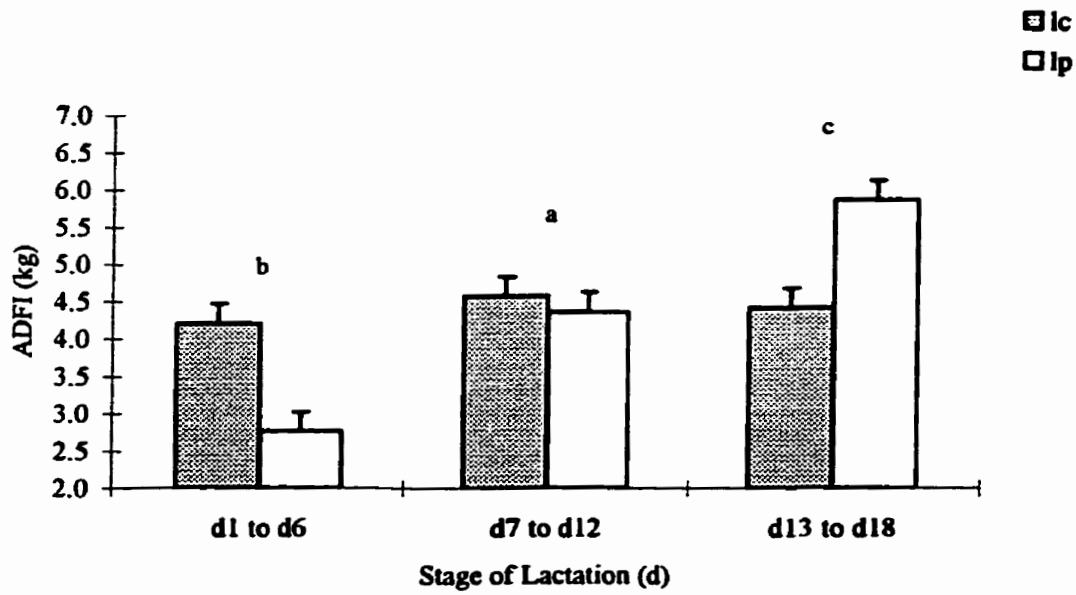

Figure 16.Lactation treatment*stage of lactation interaction for ADFI of gilts (LS means±SEM).
[a]different letters indicate that the difference between treatments in these time periods are not the same (P<0.05).

Total Feed Intake

Total feed intake during each stage of lactation is presented in Table 24. Pattern of feed intake during gestation had no significant effect on total feed intake during any stage of lactation. Research demonstrating a significant negative relationship between gestation and lactation feed intake had high ADFI throughout gestation (Dourmad, 1991) or *ad libitum* feed intake during late gestation (Weldon *et al.*, 1994a) resulting in differences in total gestation feed intake between treatments. Yang *et al.* (1989) found that the relationship between gestation feed intake and lactation feed intake was very weak for gilts.

Lactation treatment significantly influenced total feed intake during the first stage of lactation. The restricted feeding pattern (lp) resulted in a 34 % (8.61 kg) reduction in feed consumption compared to *ad libitum* gilts (lc) from d 1 to d 6 of lactation ($P<0.05$). Both lactation treatments consumed similar total amounts of feed during mid-lactation (d 7 to d 12) ($P>0.05$). The third stage of lactation (d 13 to d 18) resulted in *ad libitum* gilts consuming 25 % less feed (8.69 kg) than gilts fed at 4.1 times their maintenance requirement (lp) ($P<0.05$). Total feed intake for the entire lactation interval was not affected by lactation treatment ($P>0.05$). This is in contrast to work by Moser *et al.* (1987) who found that for a 28-day lactation period, restricted sows consumed 10 % less feed than *ad libitum* sows, and that this difference was approximately equal to that seen during the period of restriction. However, in this study the restriction in feed intake was not as severe as in the experiment carried out by Moser *et al.* (1987) where sows were restricted to 0.45 kg on the day following parturition with daily increases of 0.91 kg until *ad libitum* intake was reached on

Table 24. Total feed intake (kg) of gilts during each stage of lactation

		Stage of Lactation			
Factor		**d 1 to d 6**	**d 7 to d 12**	**d 13 to d 18**	**d 1 to d 18**
Gestation Trt.		ns	ns	ns	ns
	C	21.13 ± 1.47	26.60 ± 0.97	31.03 ± 1.98	78.77 ± 2.94
	P	20.64 ± 1.74	26.96 ± 1.15	30.74 ± 2.35	78.34 ± 3.48
Lactation Trt.		P = 0.0023	ns	P = 0.0143	ns
	c	25.19 ± 1.61	27.42 ± 1.07	26.54 ± 2.17	79.15 ± 3.22
	p	16.58 ± 1.61	26.14 ± 1.07	35.23 ± 2.17	77.96 ± 3.22
Gest.*Lact.[a]		ns	ns	ns	ns

Values are LS means ± SEM.
ns=non-significant, P>0.05.
[a]means not presented for these non-significant effects.

d 6. The difference between lc and lp total feed intake during the final stage of lactation (8.69 kg) is approximately equal to the difference between restricted and *ad libitum* animals in early lactation (8.61 kg difference).

Body Composition

Backfat

Mean P2 backfat measurements for lactation are shown in Table 25. Gestation treatment, lactation treatment, and the interaction of these main effects had no significant effect on mean P2 backfat level during lactation. Gilt backfat at the beginning of lactation was similar for both treatments and with the common lactation treatment feed intake levels, differences in mean P2 backfat depth would not be expected.

Table 25. P2 backfat (mm) and weight (kg) of gilts during lactation

Factor			P2 Backfat (mm)[e]	Weight (kg)
Gestation Trt.			ns	ns
		C	15.81 ± 0.81	162.83 ● 3.64
		P	16.62 ± 0.98	160.29 ● 4.25
Lactation Trt.			ns	ns
		c	16.09 ± 0.90	165.00 ± 3.98
		p	16.35 ± 0.89	158.12 ● 3.94
Gest.*Lact.			ns	ns
	C	c	15.44 ± 1.01	163.07 ± 4.69
		p	16.19 ± 1.26	162.59 ± 5.57
	P	c	16.74 ± 1.49	166.92 ± 6.43
		p	16.51 ± 1.26	153.66 ± 5.57
Day			P = 0.0003	P = 0.0032
	farrow (0)		$16.97^{a} \pm 0.37$	$164.35^{a} \pm 1.20$
		6	$17.08^{a} \pm 0.33$	$163.28^{a} \pm 1.17$
		12	$15.81^{ac} \pm 0.34$	$160.30^{ab} \pm 1.17$
		18	$15.01^{bc} \pm 0.34$	$158.31^{b} \pm 1.17$
Gest.*Day[d]			ns	ns
Lact.*Day			ns[d]	P = 0.0165**
	c	0		167.14 ± 1.73
		6		168.50 ± 1.66
		12		165.59 ± 1.66
		18		158.75 ± 1.66
	p	0		161.56 ± 1.66
		6		158.06 ± 1.66
		12		155.00 ± 1.66
		18		157.87 ± 1.66
Gest.*Lact.*Day[d]			ns	ns

Values are LS means ± SEM.
ns=non-significant, P>0.05.
[a-c] within columns, means with unlike superscripts differ, P<0.05.
[d]means not presented for these non-significant effects.
[e]P2 values on d0 of lactation = actual measurements taken on d109 of gestation.
**See Figure 17.

Weight

Mean body weight of gilts during lactation is presented in Table 25. Similar to the P2 backfat measurements, the effects of gestation treatment, lactation treatment and their interaction were non-significant.

The 2-way interaction of lactation treatment*day of lactation was significant (Figure 17). An initial difference in gilt weight between treatments existed at the start of lactation (d 0). Gilts assigned to the *ad libitum* lactation treatment (lc) were heavier ($P<0.05$) at the start of lactation than gilts that were to be fed at a restricted level (lp). The initial difference between treatments continued throughout lactation, and the difference between treatments was greater in mid-lactation than the difference between treatments on d 18. Although the difference in weight between treatments on d 18 of lactation was the same as the initial difference, mean body weight of the two gestation treatments did not appear to be significantly different at weaning. The lc treatment maintained weight during early- to mid-lactation, followed by a decrease in body weight during the last stage of lactation. Gilts that received the lp treatment exhibited a similar pattern of weight change during early- to mid-lactation and seemed to maintain (or gain) weight during the final stage of lactation. Weight change during lactation is presented in Appendix 8. Overall, lactation treatment had no effect on gilt weight change from farrowing to weaning ($P>0.05$) and weight losses of 8.77 $\pm$ 2.55 and 3.69 $\pm$ 2.47 kg (lc and lp, respectively) did not exceed the maximum of 10 kg of lactation weight loss cited by Aherne and Williams (1992).

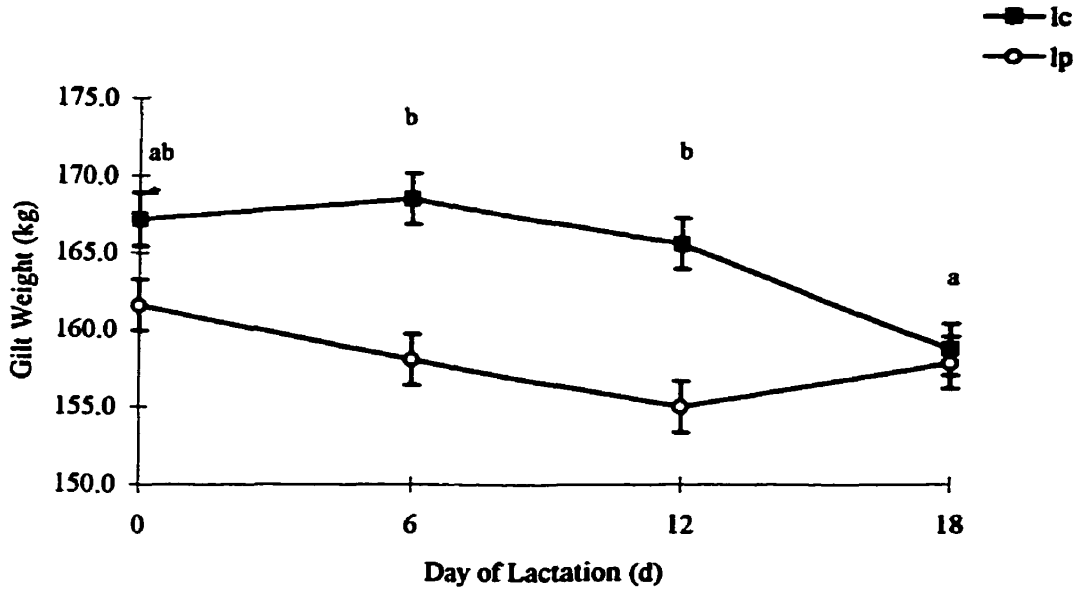

Figure 17. Lactation treatment*day of lactation interaction for gilt weight (LS means ± SEM).

[a]different letters indicate that the difference between treatments in these time periods are not the same (P<0.05).

*denotes a significant difference between treatments within day (P<0.05).

Body Protein

Predicted maternal body protein content is presented in Table 26. Gestation treatment, lactation treatment, and their interaction did not significantly affect body protein content ($P>0.05$).

The interaction of lactation treatment*day of lactation was significant, and the pattern of change in maternal body protein content is illustrated in Figure 18. The predicted body protein content of gilts at the start of lactation differed between treatments ($P<0.05$), with gilts assigned to the *ad libitum* lactation treatment beginning lactation with higher body protein content than lp gilts. The difference between lactation treatments increased by the end of the second stage of lactation (d 12), and the difference at weaning was smaller than the previous differences. Figure 18 shows a relatively constant level of body protein during early lactation and a subsequent decline in protein levels to d 18 of lactation for *ad libitum* (lc) gilts consistent with the constant level of feed intake and increased demand by the piglets in late lactation (Patience, 1993). Restricted (lp) gilts lost body protein until late lactation and displayed an increase in body protein levels to weaning.

The consistent level of body protein reserves maintained by control (lc) gilts during lactation reflected the pattern of ADFI. The pattern of feed intake of lp gilts is also evident in maternal body protein levels during lactation. The differences in body protein between treatments are similar to the differences observed for gilt weight during lactation. Whittemore and Yang (1989) cite a strict relationship between maternal body protein and weight.

Body Lipid

Mean predicted maternal body lipid content during lactation is presented in Table 26. There was no effect of gestation treatment, lactation treatment, or their interaction on predicted maternal body lipid ($P>0.05$).

The interaction of lactation treatment*day of lactation was significant for predicted maternal body lipid content (Figure 19). Lipid content on d 0 of lactation was the same for both treatments. The difference in body lipid content between treatments at weaning (d 18) was similar in magnitude to the initial difference (d 0), but the direction of response of the lactation treatments had changed. *Ad libitum* (lc) gilts had larger lipid stores than lp gilts during early- to mid lactation and maintained these levels early in lactation. However, their body lipid reserves declined to d 18. Restriction of feed intake during early lactation (lp) resulted in utilization of lipid reserves beginning immediately after farrowing until late lactation. However, lp gilts maintained body lipid levels during the final stage of lactation compared to *ad libitum* gilts. Change in maternal body lipid reserves is presented in Appendix 10. There was no effect of lactation treatment on total lipid loss during the lactation period ($P>0.05$).

Table 26. Predicted maternal body protein and lipid content (kg) of gilts during lactation

Factor			Body Protein (kg)[d]	Body Lipid (kg)[d]
Gestation Trt.			ns	ns
		C	25.09 ± 0.66	37.64 ± 1.65
		P	24.39 ± 0.80	38.11 ± 2.00
Lactation Trt.			ns	ns
		c	25.40 ± 0.74	38.45 ± 1.84
		p	24.07 ± 0.73	37.29 ± 1.82
Gest.*Lact.			ns	ns
	C	c	25.24 ± 0.83	37.34 ± 2.07
		p	24.94 ± 1.04	37.93 ± 2.58
	P	c	25.56 ± 1.23	39.57 ± 3.06
		p	23.21 ± 1.04	36.66 ± 2.58
Day			ns	P = 0.0001
	farrow (0)		24.97 ● 0.24	39.73[a] ± 0.65
		6	24.97 ● 0.21	39.52[a] ± 0.57
		12	24.58 ● 0.22	36.92[b] ± 0.61
		18	24.43 ± 0.22	35.34[b] ± 0.61
Gest.*Day[c]			ns	ns
Lact.*Day			P = 0.0360**	P = 0.0369**
	c	0	25.39 ± 0.37	39.77 ± 1.01
		6	25.88 ± 0.30	41.11 ± 0.81
		12	25.66 ± 0.30	38.25 ± 0.81
		18	24.67 ± 0.30	34.69 ± 0.81
	p	0	24.56 ± 0.30	39.67 ± 0.81
		6	24.05 ± 0.30	37.92 ± 0.81
		12	23.49 ± 0.33	35.59 ± 0.90
		18	24.19 ± 0.33	35.99 ± 0.90
Gest.*Lact.*Day[c]			ns	ns

Values are LS means ● SEM.
ns=non-significant, P>0.05.
[a-b] within columns, means with unlike superscripts differ, P<0.05.
[c]means not presented for these non-significant effects.
[d]Prediction equations of Whittemore and Yang (1989).
**See Figures 18, 19.

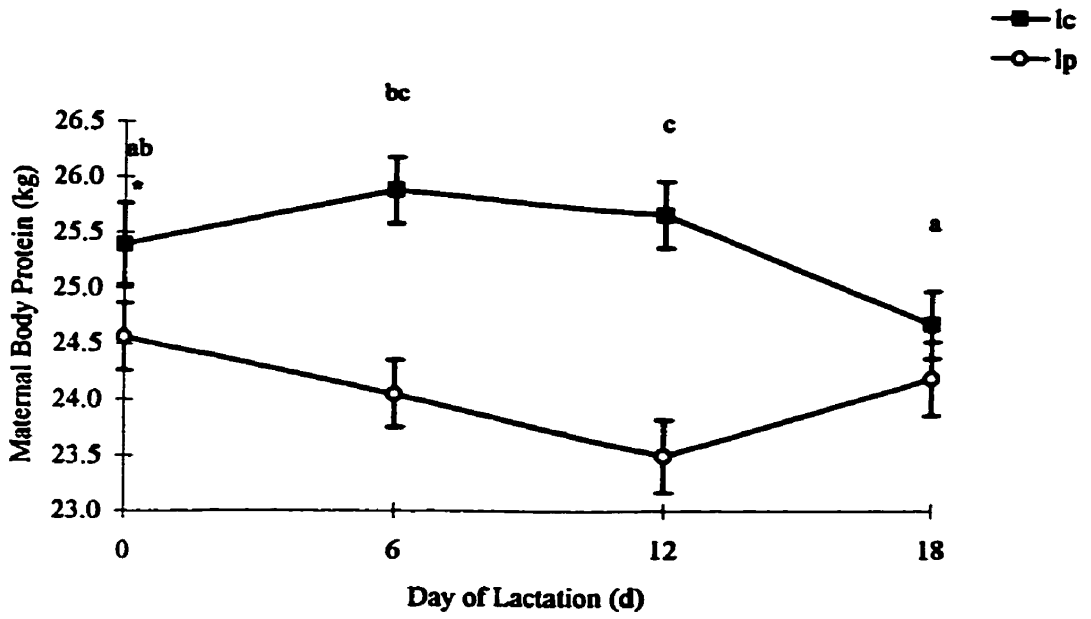

Figure 18. Lactation treatment*day of lactation interaction for predicted maternal body protein content of gilts (LS means ± SEM).

[a]different letters indicate that the difference between treatments in these time periods are not the same (P<0.05).

*denotes a significant difference between treatments within day (P<0.05).

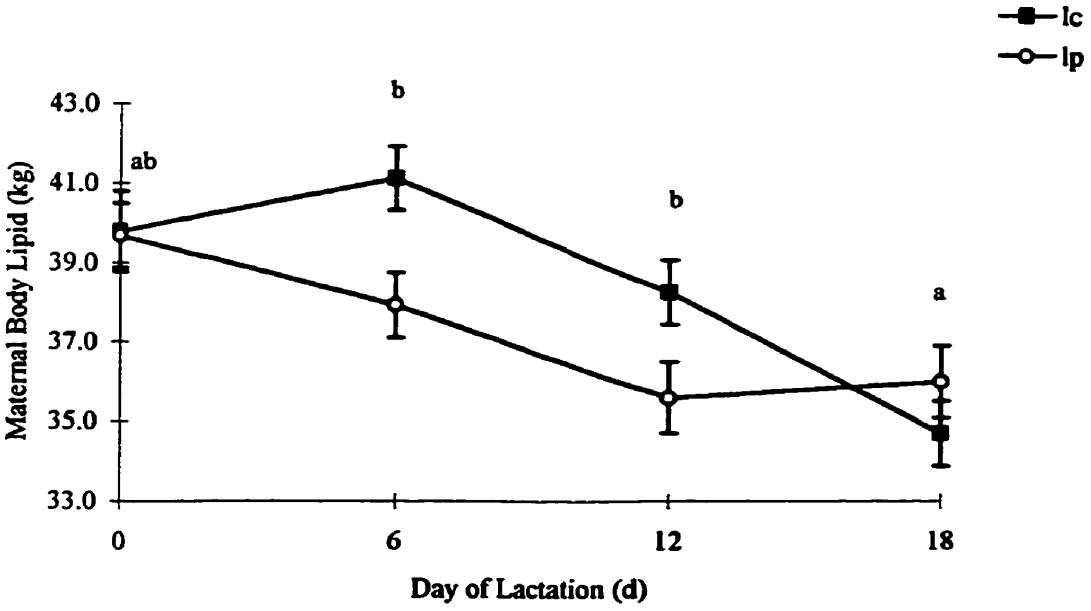

Figure 19. Lactation treatment*day of lactation interaction for predicted maternal body lipid content of gilts (LS means ± SEM).

[a]different letters indicate that the difference between treatments in these time periods are not the same (P<0.05).

Piglet Weight

Gestation treatment, lactation treatment, and their interaction did not significantly affect mean piglet weight during the lactation period (Table 27). The interaction of gestation treatment*day was of lactation was significant (Figure 20). The difference in average piglet weight between treatments was the same on d 0, d 1, d 6, and d 12 of lactation. The difference in average piglet weight between gestation treatments was larger on d 18 than on d 0, through d 6 of lactation. Average piglet weight from gilts that had received the gP treatment during gestation increased at a greater rate during late lactation, and these piglets were heavier at weaning, than piglets from gilts that were fed at 1.4 times maintenance (gC) during gestation. Pattern of feed intake (Aherne, 1996) and particularly additional feed intake from d 90 of gestation (Cromwell *et al.*, 1980) increased birth weight and may influence postnatal growth. Nutrition during mid- to late gestation may influence mammary gland development in the young sow (Weldon *et al.*, 1991). The period from d 75 to d 105 of gestation is the period of growth of mammary tissue, specifically milk secretory tissue. It is possible that the increased level of feed intake of gP gilts during this time affected mammary gland development and subsequent milk production and piglet growth. As well, muscle fiber number development of the fetus occurs during mid- to late gestation. Specifically it is the number of secondary muscle fibers that are responsive to maternal nutrition (Dwyer *et al.*, 1994) and that may have potential effects on postnatal growth.

Table 27. Piglet weight (kg) during lactation

Factor			Mean Piglet Weight (kg)
Gestation Trt.			ns
		C	2.94 ± 0.13
		P	3.13 ± 0.15
Lactation Trt.			ns
		c	3.18 ± 0.15
		p	2.89 ± 0.14
Gest. *Lact.			ns
	C	c	3.18 ± 0.18
		p	2.71 ± 0.18
	P	c	3.17 ± 0.23
		p	3.08 ± 0.20
Day			P = 0.0001
		birth (0)	1.47^{a} ± 0.044
		1	1.58^{a} ± 0.043
		6	2.54^{b} ± 0.043
		12	4.02^{c} ± 0.043
		wean (18)	5.57^{d} ± 0.043
Gest.*Day**			P= 0.0040
	C	0	1.45 ± 0.054
		1	1.55 ± 0.055
		6	2.49 ± 0.056
		12	3.90 ± 0.056
		18	5.34 ± 0.056
	P	0	1.48 ± 0.070
		1	1.62 ± 0.067
		6	2.59 ± 0.064
		12	4.15 ± 0.065
		18	5.79 ± 0.065
Lact.*Day[c]			ns
Gest.*Lact.*Day[c]			ns

Values are LS means ● SEM.
ns=non-significant, P>0.05.
[a-d] within columns, means with unlike superscripts differ, P<0.05.
[c]means not presented for these non-significant effects.
**See Figure 20.

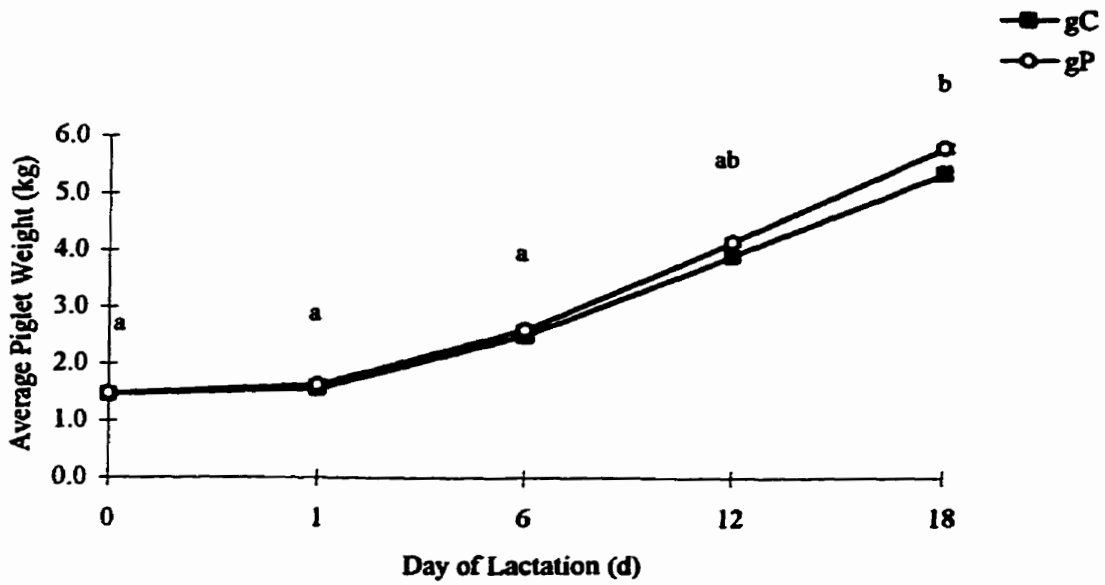

Figure 20. Gestation treatment*day of lactation interaction for piglet weight (LS means ± SEM).
[a]different letters indicate that the difference between treatments in these time periods are not the same ($P<0.05$).

Litter Size

Gilt litter size at birth and at weaning is presented in Table 28. There was no effect of gestation treatment on litter size at birth (total born or born alive). Feeding 2.5 kg d^{-1} versus restriction to maintenance (approximately 1.25 kg d^{-1}) for 10 d in early gestation had no effect on litter size at birth in a study by Dyck *et al.* (1991). Embryo survival (and litter size) was not decreased by feeding gilts 2.5 kg d^{-1} during gestation in the study by Dyck *et al.* (1991). In contrast, Jindal *et al.* (1996) found that feeding gilts greater than 2.5 kg d^{-1} (~2 times maintenance) compared to 1.5 times maintenance (~NRC recommended levels) caused a decrease in embryo survival in early gestation. Average daily feed intake for the gilts in this trial in early gestation (d 1 to d 30) was 2.06 ± 0.02 and 1.61 ± 0.02 kg d^{-1} for gC and gP treatments, respectively. Results of this study suggest that feed intake in early gestation was not high enough to cause a decrease in embryo survival, consistent with the observed similarity in total litter size at birth.

The number of pigs weaned was not affected by gestation treatment, lactation treatment, or their interaction ($P>0.05$). Cromwell *et al.* (1989) found that additional feed in late gestation, although resulting in increased piglet birth weight, did not result in increased survival at weaning. A previous study by Pettigrew (1981) improved piglet survival at weaning by feeding supplemental fat to the sows in late gestation. However, survival was only improved if average survival from birth to weaning was less than 80%. The lack of response to additional feed in the study by Cromwell *et al.* (1989) may also be due to the

Table 28. Gilt litter size at birth and weaning

Factor			Total Born	Born Alive	Weaned
Gestation Trt.			ns	ns	ns
		C	8.40 ● 1.01	8.30 ± 0.99	7.83 ± 0.83
		P	8.00 ± 1.20	8.00 ± 1.19	8.33 ● 0.98
Lactation Trt.			**	**	ns
		c			7.92 ± 0.92
		p			8.25 ± 0.92
Gest.*Lact.			**	**	ns
	C	c			7.17 ± 1.28
		p			8.50 ± 1.29
	P	c			8.67 ● 1.49
		p			8.00 ● 1.29

Values are LS means ± SEM.
ns=non-significant, P>0.05.
**Lactation treatment not included in the model for variable born alive.

relatively high survival (84%). Piglet survival to weaning in this trial (97%) may also explain the absence of treatment effects on litter size at weaning.

Serum Urea Nitrogen

Mean serum urea nitrogen values are presented in Table 29. There was no effect of gestation treatment, lactation treatment, or their interaction on serum urea nitrogen concentrations during lactation ($P>0.05$). Serum urea nitrogen did increase for all gilts from the day of farrowing (d 0) to d 6 of lactation ($P<0.05$), and remained high until weaning. The increase in serum urea nitrogen concentration for all sows early in lactation supports the concept of amino acid catabolism during lactation associated with mobilization of body reserves (Nelssen *et al.*, 1985). However, distinction between altered serum urea nitrogen levels due to exogenous or endogenous sources is not possible in this experiment, and the increase in urea levels may be due to feed intake levels.

Table 29. Mean serum urea nitrogen (mg dl^{-1}) of gilts during lactation

Factor			Urea Nitrogen (mg dl^{-1})
Gestation Trt.			ns
		C	16.08 ± 0.71
		P	17.69 ± 0.87
Lactation Trt.			ns
		c	16.26 ± 0.81
		p	17.51 ± 0.79
Gest.*Lact.			ns
	C	c	15.28 ± 0.87
		p	16.88 ± 1.13
	P	c	17.24 ± 1.36
		p	18.14 ± 1.09
Day			P = 0.0003
		farrow (0)	14.07^{a} ± 0.69
		6	17.27^{b} ± 0.65
		12	18.44^{b} ± 0.62
		18	17.76^{b} ± 0.62
Gest.*Day[c]			ns
Lact.*Day[c]			ns
Gest. x Lact. x Day[c]			ns

Values are LS means ± SEM.
ns=non-significant, P>0.05.
[a-b] within columns, means with unlike superscripts differ, P<0.05.
[c]means not presented for these non-significant effects.

Luteinizing Hormone

Luteinizing hormone secretion is a key component in the return to estrus after weaning (Koketsu *et al.*, 1996), and nutrition during lactation can alter LH profiles (Tokach *et al.*, 1992; Zak *et al.*, 1997).

Mean serum LH concentration and pulse frequency are presented in Table 30. Gestation treatment had no effect on mean LH concentration, LH baseline concentration and pulse frequency of samples collected during the 4-h sampling period ($P>0.05$).

The effects of lactation treatment and the interaction of gestation treatment*lactation treatment interaction were also non-significant. Mean LH concentrations and LH baseline concentrations were the same on all sampling days during lactation and on the day of weaning for all gilts ($P>0.05$). However, on d 1 post-weaning, mean LH concentration was higher ($P<0.05$) than concentrations on d 6 and d 12 of lactation, but was not significantly different from d 18 and the day of weaning (d 19). Shaw and Foxcroft (1985) noted a significant increase in LH concentration in a 12-h period after weaning. The lack of treatment-induced differences in LH profiles treatment may be due to the short sampling interval utilized in this study (4 h). Tokach *et al.* (1992) proposed that the 6-h sampling period used in their study was insufficient to observe a post-weaning rise in LH secretion. The small sample size of this study may also have contributed to the lack of observable differences in LH concentration or pulsatility.

Table 30. Luteinizing Hormone (LH) concentrations (ng ml^{-1}) and pulse frequency of gilts during lactation and the post-weaning period

Factor			Mean LH (ng ml^{-1})	LH baseline[f] (ng ml^{-1})	Pulse Frequency[g]
Gestation Trt.			ns	ns	ns
		C	0.46 ± 0.07	0.44 ± 0.07	0.23 ● 0.07
		P	0.35 ± 0.10	0.35 ● 0.10	0.03 ● 0.10
Lactation Trt.			ns	ns	ns
		c	0.44 ● 0.07	0.44 ± 0.07	0.11 ± 0.07
		p	0.36 ± 0.09	0.35 ● 0.10	0.15 ± 0.10
Gest.*Lact.			ns	ns	ns
	C	c	0.49 ± 0.08	0.48 ± 0.08	0.15 ± 0.08
		p	0.43 ± 0.11	0.41 ± 0.11	0.31 ± 0.11
	P	c	0.40 ± 0.12	0.39 ± 0.12	0.07 ± 0.12
		p	0.30 ± 0.16	0.30 ± 0.16	0.00 ± 0.16
Day			P = 0.0066	P = 0.0065	ns
		6	0.17^{a} ± 0.14	0.16^{a} ± 0.14	0.15 ± 0.09
		12	0.09^{a} ± 0.14	0.08^{a} ± 0.14	0.13 ± 0.10
		18	0.46^{ac} ± 0.13	0.44^{ac} ± 0.13	0.25 ± 0.09
		wean	0.31^{ab} ± 0.17	0.31^{ab} ± 0.17	0.03 ± 0.12
		d1pw[e]	0.98^{bc} ± 0.19	0.98^{bc} ± 0.19	0.03 ± 0.13
Gest.*Day[d]			ns	ns	ns
Lact.*Day[d]			ns	ns	ns
Gest.*Lact.*Day[d]			ns	ns	ns

Values are LS means ● SEM.
ns=non-significant, P>0.05.
[a-c] within columns, means with unlike superscripts differ, P<0.05.
[d]means not presented for these non-significant effects.
[e]d1pw = day 1 post-weaning.
[f]LH baseline: average LH concentration when points >3s.d. removed.
[g]pulse frequency = number of pulses in 4-hr. sampling period.

When LH data was analyzed as a two-way factorial, a significant effect of lactation treatment was observed on d 6. Mean LH concentration on this day was higher for *ad libitum* gilts (0.19 ng ml^{-1}) than for restricted gilts (0.06 ng ml^{-1}). LH baseline concentration was also significantly affected (0.18 ng ml^{-1} versus 0.05 ng ml^{-1} for lc and lp gilts, respectively). Koketsu *et al.* (1996) found that energy restriction during any week of lactation reduced LH secretion during the lactation period. Alterations in LH concentration and pulsatility as early as d 14 of lactation were associated with the length of the WEI (Tokach *et al.*, 1992). However, no treatment effects were observed for LH profiles during the remaining sampling periods or for WEI during this experiment.

Weaning-to-Estrus Interval

The length of the WEI, particularly for gilts, is influenced by feed intake in lactation and loss of weight and backfat depth during the lactation period (King and Williams, 1984; Mullan and Williams; Dourmad *et al.*, 1994).

The WEI was not significantly affected by gestation treatment, lactation treatment, or their interaction ($P>0.05$) as illustrated in Table 31. Loss of body protein and lipid during lactation, as well as the absolute levels of these constituents at parturition and at weaning, influence the WEI (Vesseur *et al.*, 1996; Cosgrove *et al.*, 1997). The relative losses of maternal body reserves of lipid and protein during lactation and their levels at weaning were not different in this experiment.

Table 31. Weaning-to-estrus interval (WEI) (d) of gilts

Factor			WEI (d)
Gestation Trt.			ns
		C	10.77 ± 2.38
		P	9.75 ● 2.98
Lactation Trt.			ns
		c	11.85 ± 2.72
		p	8.67 ± 2.66
Gest.*Lact.			ns
	C	c	14.20 ● 2.91
		p	7.33 ● 3.76
	P	c	9.50 ● 4.61
		p	10.00 ● 3.76

Values are LS means ● SEM.
ns=non-significant, P>0.05.

Ovulation Rate

Ovulation rate and the number of normal appearing corpora lutea (CL) are presented in Table 32. Ovulation rate was not significantly affected by gestation treatment, lactation treatment, or their interaction ($P>0.05$). Information relating lactation feed intake to subsequent ovulation rate is not consistent. Aherne and Williams (1992) in a review of research findings state that ovulation rate is not influenced by lactation feed intake. King and Williams (1984) found no significant difference in ovulation rate due to lactation feed intake when comparing *ad libitum* versus restricted lactation feed intake (2 kg d^{-1}). Conversely, Zak *et al.* (1997a) found that feed restriction imposed for a one week period in late lactation, or for the first 21 days of a 28-day lactation, resulted in a lower ovulation rate compared to sows fed *ad libitum* throughout lactation.

Gestation treatment did have a significant effect ($P<0.05$) on the number of normal CL. Gilts that were fed an increasing pattern of intake (gP) had 45 % more normal CL counted at slaughter. Follicular imprinting resulting from changes in nutrition during late gestation (Cosgrove *et al.*, 1997) or lactation (Zak *et al.*, 1997) may affect ovulation rate.

Table 32. Ovulation rate of gilts[a]

Factor			Ovulation Rate[b]	Number of normal CL[c]
Gestation Trt.			ns	P = 0.0319
		C	18.50 ± 1.39	14.03 ● 1.55
		P	22.17 ± 1.74	20.33 ± 1.94
Lactation Trt.			ns	ns
		c	20.00 ± 1.59	16.70 ● 1.78
		p	20.67 ● 1.55	17.67 ● 1.73
Gest.*Lact.			ns	ns
	C	c	16.00 ± 1.70	12.40 ● 1.90
		p	21.00 ± 2.20	15.67 ± 2.45
	P	c	24.00 ± 2.69	21.00 ● 3.00
		p	20.33 ● 2.20	19.67 ± 2.45

Values are LS means ● SEM.
ns = non-significant, P>0.05.
[a]Ovulation rate determined 5- to 10-d post-estrus.
[b]total number of CL counted (normal + abnormal).
[c]total number of normal CL counted.

Post-weaning Progesterone

Progesterone concentrations from samples collected post-weaning are presented in Table 33. The pattern of feed intake in gestation influenced P_4 concentration post-weaning ($P<0.05$). Gilts that were fed at a constant level with respect to metabolic body weight during pregnancy (gC) had lower P_4 values post-weaning than gilts fed at increasing levels of feed intake (gP). Lactation treatment and the interaction of gestation treatment*lactation treatment had no effect on post-weaning P_4 concentration ($P>0.05$). Progesterone concentrations at weaning and on the day of estrus were not different ($P>0.05$). However 5 days post-estrus (d5pe), P_4 levels had increased significantly for all gilts.

The interaction of gestation treatment*day post-weaning is illustrated in Figure 21. The difference in P_4 concentration between gC and gP treatments on the day of weaning was small and the same as the difference between treatments at estrus. The difference in P_4 concentration between treatments on d5pe was greater than on previous days. The increase in P_4 concentration from the day of weaning until 5 days following estrus was greater for gilts which had received the gP treatment. Higher concentrations of progesterone on d5pe for the gP group are in agreement with the gestation treatment effect on the number of normal CL, although there were no differences in ovulation rate. Feed intake during late gestation may influence the subsequent ovulation (Cosgrove *et al.*, 1997) by influencing the development of preovulatory follicles (Zak *et al.*, 1997b).

Table 33. Mean post-weaning progesterone (P_4) (ng ml^{-1}) of gilts

Factor			P_4 (ng ml^{-1})
Gestation Trt.			P = 0.0155
		C	3.53 ± 0.53
		P	5.75 ± 0.54
Lactation Trt.			ns
		c	4.44 ± 0.42
		p	4.84 ± 0.62
Gest.*Lact.			ns
	C	c	3.44 ± 0.51
		p	3.61 ± 0.92
	P	c	5.43 ● 0.68
		p	6.08 ± 0.83
Day			P = 0.0001
	wean (w)		0.55[a] ● 0.62
	estrus (e)		0.28[a] ● 0.58
	d5pe[c]		13.09[b] ● 0.68
Gest.*Day**			P = 0.0237
	C	w	0.11 ± 0.58
		e	0.19 ± 0.82
		d5pe	10.27 ● 1.08
	P	w	0.98 ± 1.09
		e	0.37 ± 0.83
		d5pe	15.91 ± 0.83
Lact.*Day[d]			ns
Gest.*Lact.*Day[d]			ns

Values are LS means ± SEM.
ns=non-significant, P>0.05.
[a-b] within columns, means with unlike superscripts differ, P<0.05.
[c]d5pe= 5d post-estrus
[d]means not presented for these non-significant effects.
**See Figure 21.

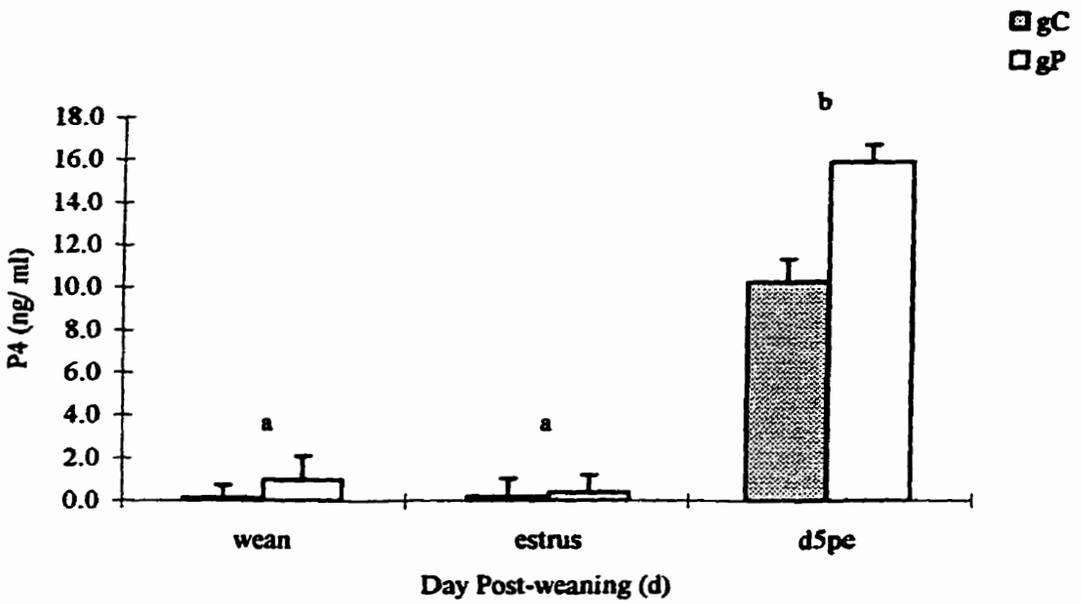

Figure 21. Gestation treatment*day post-weaning interaction for gilt serum progesterone (LS means + SEM).

*different letters indicate that the difference between treatments in these time periods are not the same ($P<0.05$).

CHAPTER 5

GENERAL DISCUSSION

Modification of the feed intake patterns of gilts and first parity sows during gestation and lactation improved aspects of sow reproductive performance. Gilts responded differently than first parity sows to the altered patterns of feed intake during gestation and lactation in specific cases. In addition, the response to feed intake patterns may have been influenced by genotype of the sow.

Gestation

The feed intake patterns during gestation resulted in similar ADFI for both treatments in Experiments I and II, while total feed intake differed between treatments in Experiment II only.

Modifying the feed intake pattern in gestation to provide stepwise increases in feed intake (gP) did not result in improved weight or backfat depth at the end of gestation. The experimentally imposed patterns of feed intake altered gilt and sow body weight and backfat thickness during the individual stages of gestation, and these fluctuations in body condition reflected the changing nutrient supply. Early work by Cromwell *et al.* (1980) showed that gestation weight gain was influenced by total feed intake rather than pattern of feed intake. Subsequent research has confirmed that increased total nutrient supply positively affects weight gain (Young *et al.*, 1990; Xue *et al.*, 1997b).

Data from the first experiment indicates that first parity sows responded to feed intake patterns as expected in terms of weight gain. Gilts however, exhibited a lower rate of weight gain throughout pregnancy when receiving treatment gP in Experiment I and II. The reason for this decreased weight gain even when feed intake was increased above the level of the gC group is unclear. Perhaps the higher requirements for growth of the gilts resulted in a poorer response to increased feeding levels in late gestation following feed restriction in early pregnancy. The variability associated with backfat measurement may explain why gestation treatment effects on backfat depth were not observed in either experiment.

Nutrient utilization was not improved by altering the pattern of feed intake in pregnancy. Differences in nutrient retention between treatments during gestation in Experiment II were induced by the different patterns of nutrient intake. However, when nutrient retention values were expressed as a percent of nutrient intake, treatment differences were not significant. Therefore, nutrient utilization by gilts was similar at differing levels of intake. The absence of treatment-induced differences in nitrogen retention in Experiment II are supported by the treatment similarities in serum urea nitrogen concentration.

Gestation treatment did not influence serum P_4 concentrations during pregnancy in Experiments I and II. Nutritionally-mediated changes in P_4 concentration, and possible influences on embryo survival occur in early gestation (Jindal *et al.*, 1996, 1997). The periods of blood sample collection in Experiments I and II (d 1 and d 30) would not allow for observation of possible treatment-induced changes in P_4 concentration as related to embryo survival and subsequent litter size.

Restriction of feed intake in early gestation followed by a gradual increase in intake as pregnancy progressed (gP), improved some aspects of gilt performance during the lactation period as well as subsequent reproductive performance. Beneficial effects of pattern-feeding (gP) during gestation, included an improvement in the number of piglets born alive to gilts and larger litter size at weaning in Experiment I. Pattern-feeding during gestation did not, however, result in a greater proportion of piglets born alive or weaned by first parity sows in Experiment I or gilts in Experiment II. Maintaining gilt feed intake at a constant level times maintenance (gC) in Experiment II resulted in poorer piglet growth in late lactation and lower weaning weights, compared to piglets from gilts that had received treatment gP. Subsequent ovulation rate was not influenced by the adjustment of feed intake during gestation and lactation in Experiment II, however, the number of normal corpora lutea were greater for gilts which had received the pattern treatment during gestation (gP), and this difference was reflected in a greater increase in P_4 concentration post-weaning.

Lactation

Comparison of the lactation portions of the two experiments will not be made due to the different feeding levels associated with the lactation control treatment groups.

Average daily feed intake during lactation was not influenced by gestation treatment in Experiments I and II. ADFI and total feed intake during the lactation period differed between lactation treatments in Experiment I. These measures of feed intake were greater for the lp treatment than the lc treatment group during the lactation period. In contrast, ADFI and total feed intake were not affected by the pattern of lactation feed intake in Experiment II. In both experiments, the lp treatment consumed more feed during the final stage of lactation (d 13 to weaning) than the lc treatment group. Specifically, feed consumption of the lp treatment in Experiment I was greater in mid-lactation, which may have contributed to the greater total feed intake of this treatment during the lactation period. Lactation treatment differences in feed consumption between the two experiments are due to the feeding regimen associated with each experiment. The control treatment was fed at true *ad libitum* intake in Experiment II, while c sows and gilts in Experiment I were assigned to a full-feeding system during lactation.

Evaluation of parameters related to body condition during lactation showed that there were no differences in body weight or estimated levels of maternal body protein or lipid at the end of lactation due to lactation feeding level in Experiments I and II. It is possible that the

variability associated with backfat measurement and the small sample size for each treatment combination in Experiment II resulted in the lack of response to feed intake pattern.

However, in Experiment I, gestation feed intake pattern as well as the interaction of gestation treatment*lactation treatment, resulted in differences in body condition at the end of lactation. Pattern-feeding during gestation (gP) resulted in greater backfat loss during lactation. Conversely, maintaining feed intake at a constant level times maintenance (gC) during gestation resulted in greater utilization of body protein reserves during lactation. The combination of feeding at a constant level times maintenance during gestation (gC) and full-feeding during lactation (lc) resulted in larger backfat and lipid losses during the lactation period, and consequently the lowest levels of backfat and body lipid at d 17 compared to the other treatment combinations. Backfat depth during lactation was maintained and lipid loss was decreased through feeding at a constant level times maintenance during gestation (gC) followed by the pattern treatment (lp) in lactation. Backfat loss was evident in the other treatment combinations during lactation, but was not greatly affected by the pattern of feed intake during lactation when gilts and sows were fed at increasing levels times maintenance during gestation (gP).

Serum urea N concentrations during lactation were not affected by pattern of feed intake during any stage of the reproductive cycle in Experiment II.

Gilts fed the c level during lactation weaned more piglets than gilts restricted in feed intake during lactation, while lactation treatment did not seem to influence the number of piglets weaned by first parity sows in Experiment I. The number of piglets weaned by gilts

in treatment gP may be a reflection of the greater litter size born alive of this treatment group. The number of piglets weaned in Experiment II was not affected by feed intake patterns of the gilts during gestation or lactation.

In the second experiment, LH concentration and pulsatility were not affected by treatment during gestation or lactation. However, baseline and mean LH concentrations on d 6 of lactation were higher for gilts that were fed *ad libitum* during the first stage of lactation than for gilts that were restricted to 1.9 times maintenance during this time period.

The length of the WEI was not altered by pattern of feed intake during gestation or lactation in Experiment II. In Experiment I, the WEI of gilts was prolonged for the lp treatment during lactation compared to first parity sows of the same treatment. The effects of gestation treatment and lactation treatment interacted to alter the length of the WEI. The treatment combination Cp resulted in an extended WEI for gilts relative to first parity sows.

CHAPTER 6

SUMMARY AND CONCLUSIONS

Results of the two studies indicate that modifying the feed intake patterns of young sows improves some aspects of reproductive performance. Specifically, altering the pattern of gestation feed intake to provide increasing levels of feed times maintenance with the progression of pregnancy, improved litter size born alive, litter size at weaning, and piglet growth during late lactation for gilts, but not for first parity sows. The number of normal corpora lutea present on the ovaries of gilts following the post-weaning estrus was greater for gilts that received the pattern (gP) treatment during gestation. Altering the pattern of feed intake during lactation did not result in improved piglet growth or post-weaning sow reproductive performance.

Recommendations to improve the level of lactation voluntary feed intake can be made based on the observed differences in ADFI and total feed intake between treatments at the commercial facility. The feeding method currently employed resulted in significantly lower lactation feed intake than the pattern feeding method, particularly during late lactation. In situations where feed intake is restricted in early lactation (such as Experiment I), pattern-feeding during lactation may increase lactation feed intake.

Feeding gilts and first parity sows the combination of NRC recommended levels during gestation (C) and altering the pattern in lactation (p), resulted in improved body fat reserves at the end of lactation. However, the benefits of increased body fat resrves during lactation were not realized in terms of reducing the length of the WEI.

Further research is suggested to:

1) compare the pattern-feeding method (gP) utilized during gestation to industry standard gestation feed intake levels for the pregnant sow.

2) assess the impact of modified feed intake patterns on sow performance in successive parities.

3) refine the nutrient requirements of the young sow, specifically energy or protein requirements, with potential development of phase-feeding types of systems to more closely match the requirements of the sow with the stages of the production cycle.

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Experiment I:

Appendix 1. P2 backfat change (mm) of gilts and first parity sows during each stage of gestation

			Stage of Gestation				
Factor			**d 1 to d 30**	**d 31 to d 60**	**d 61 to d 90**	**d 91 to d 109**	**d 1 to d 109**
Gestation Trt.			ns	ns	ns	ns	P = 0.0328
		C	1.00 ± 0.45	-0.03 ± 0.36	0.07 ± 0.42	-0.50 ± 0.41	0.47 ± 0.53
		P	1.34 ± 0.47	0.55 ± 0.37	0.19 ± 0.43	0.01 ± 0.40	2.10 ± 0.53
Parity[a]			ns	P = 0.0337	ns	ns	P = 0.0065
		0	0.94 ± 0.45	-0.30 ± 0.36	-0.23 ± 0.41	-0.14 ± 0.39	0.24 ± 0.51
		1	1.41 ± 0.47	0.81 ± 0.38	0.50 ± 0.43	-0.35 ± 0.42	2.33 ± 0.55
Gest.*Parity	C		ns (0.0620)[b]	ns[b]	ns[b]	P = 0.0490	ns[b]
		0	0.16 ± 0.62			-0.97 ± 0.54	
		1	1.85 ± 0.67			-0.04 ± 0.60	
	P	0	1.72 ± 0.65			0.69 ± 0.56	
		1	0.96 ± 0.67			-0.67 ± 0.58	

Values are LS means ± SEM.
ns=non-significant at P>0.05.
[a]parity 0 (gilt), parity 1 (first parity sow).
[b]means not presented for these non-significant effects.

Experiment I:

Appendix 2. Weight change (kg) of gilts and first parity sows during each stage of gestation

		Stage of Gestation				
		d 1 to d 30	**d 31 to d 60**	**d 61 to d 90**	**d 91 to d 109**	**d 1 to d 109**
Gestation Trt.		P = 0.0104	ns	ns	P = 0.0196	ns
	C	15.65 ± 0.76	17.10 ± 0.73	21.05 ± 0.77	16.89 ± 0.93	70.03 ± 1.44
	P	12.80 ± 0.78	16.19 ± 0.75	22.19 ± 0.79	20.00 ± 0.93	71.19 ± 1.43
Parity[a]		P = 0.0001	ns	ns	ns	ns
	0	11.84 ± 0.75	16.83 ± 0.72	22.55 ± 0.75	17.80 ± 0.90	68.84 ± 1.38
	1	16.60 ± 0.79	16.46 ± 0.76	20.70 ± 0.80	19.10 ± 0.96	72.38 ± 1.49
Gest.*Parity		ns[b]	ns[b] (0.0604)	ns[b]	ns[b]	P = 0.0355
C	0		18.28 ± 0.99			70.43 ± 1.93
	1		15.92 ± 1.08			69.64 ± 2.14
P	0		15.37 ± 1.04			67.25 ± 1.99
	1		17.00 ± 1.08			75.12 ± 2.06

Values are LS means ± SEM.
ns=non-significant, P>0.05.
[a]parity 0 (gilt), parity 1 (first parity sow).
[b]means not presented for these non-significant effects.

Experiment I:

Appendix 3. P2 backfat (mm) and weight (kg) changes of gilts and first parity sows during lactation

			P2 Backfat Change[c] (mm)	Weight Change[c] (kg)
Factor			**d 0 to d 17**	**d 0 to d 17**
Gestation Trt.			P = 0.0320	ns
		C	-1.08 ± 0.31	-9.25 ± 1.04
		P	-2.03 ± 0.30	-6.98 ± 0.99
Lactation Trt.			ns	ns
		c	-1.88 ± 0.30	-8.62 ± 1.02
		p	-1.23 ± 0.31	-7.61 ± 1.02
Parity[a]			ns	P = 0.0002
		0	-1.76 ± 0.29	-5.37 ± 0.98
		1	-1.35 ± 0.32	-10.85 ± 1.06
Gest.*Lact.			P = 0.0058	ns[b]
	C	c	-2.02 ± 0.41	
		p	-0.15 ± 0.47	
	P	c	-1.74 ± 0.45	
		p	-2.32 ± 0.41	
Gest.*Parity[b]			ns	ns
Lact.*Parity[b]			ns	ns
Gest.*Lact.*Parity[b]			ns	ns

Values are LS means ± SEM.
ns=non-significant at P>0.05.
[a]parity 0 (gilt), parity 1 (first parity sow).
[b]means not presented for these non-significant effects.
[c]adjusted to 17-d lactation length.

Experiment I:

Appendix 4. Predicted maternal body protein and lipid changes (kg) of gilts and first parity sows during lactation

Factor		Body Protein Change (kg) d 0 to d 17	Body Lipid Change (kg) d 0 to d 17
Gestation Trt.		P = 0.0265	ns
	C	-1.48 ± 0.19	-3.61 ± 0.59
	P	-0.88 ± 0.18	-4.51 ± 0.56
Lactation Trt.		ns	ns
	c	-1.19 ± 0.19	-4.67 ± 0.58
	p	-1.17 ± 0.19	-3.45 ± 0.57
Parity		P = 0.0001	ns[b]
	0	-0.62 ± 0.18	-3.86 ± 0.55
	1	-1.74 ± 0.20	-4.26 ± 0.60
Gest.*Lact.		ns[b]	P = 0.0181
C	c		-5.19 ± 0.81
	p		-2.02 ± 0.86
P	c		-4.14 ± 0.83
	p		-4.87 ± 0.74
Gest.*Parity[b]		ns	ns
Lact.*Parity[b]		ns	ns
Gest.*Lact.*Parity[b]		ns	ns

Values are LS means ± SEM.
ns=non-significant at P>0.05.
[a]parity 0 (gilt), parity 1 (first parity sow).
[b]means not presented for these non-significant effects.

Experiment II:

Appendix 5. Gilt P2 backfat change (mm) during each stage of gestation

	Stage of Gestation				
Factor	**d 1 to d 30**	**d 31 to d 60**	**d 61 to d 90**	**d 91 to d 109**	**d 1 to d 109**
Gestation Trt.	ns	ns	ns	ns	ns
C	2.10 ± 0.41	0.90 ± 0.59	0.10 ± 0.57	0.19 ± 0.38	3.44 ± 0.47
P	1.06 ± 0.46	1.00 ± 0.66	1.00 ± 0.64	0.67 ± 0.44	3.08 ± 0.54

Values are LS means ± SEM.
ns = non-significant, P>0.05

Experiment II:

Appendix 6. Gilt weight change (kg) during each stage of gestation

	Stage of Gestation				
Factor	**d 1 to d 30**	**d 31 to d 60**	**d 61 to d 90**	**d 90 to d 109**	**d 1 to d 109**
Gestation Trt.	P = 0.0009	ns	ns (0.0569)	P = 0.0012	ns
C	7.09 ± 1.34	12.46 ± 1.27	15.50 ± 0.85	14.39 ± 1.12	50.39 ± 3.47
P	-1.08 ± 1.50	11.44 ± 1.41	18.12 ± 0.95	21.29 ± 1.28	49.34 ± 3.93

Values are LS means ± SEM.
ns = non-significant, P>0.05.

Experiment II:

Appendix 7. Gilt P2 backfat change (mm) during each stage of lactation

		Stage of Lactation			
Factor		**d 109 to d 6**	**d 7 to d 12**	**d 13 to d 18**	**d 109 to d 18**
Gestation Trt.		ns	ns	ns	ns
	C	0.34 ± 0.40	-1.42 ± 0.63	-0.58 ± 0.74	-1.95 ± 0.82
	P	-0.00 ± 0.52	-0.98 ± 0.68	-1.08 ± 0.84	-2.00 ± 1.11
Lactation Trt.		P = 0.0245	ns	ns	ns
	c	1.02 ± 0.50	-1.50 ± 0.63	-1.42 ± 0.72	-1.95 ± 1.02
	p	-0.69 ± 0.42	-0.89 ± 0.68	-0.25 ± 0.84	-2.00 ± 0.93
Gest.*Lact.		ns	ns	ns	ns
C	c	1.80 ± 0.54	-2.17 ± 0.72	-0.33 ± 0.84	-0.90 ± 1.09
	p	-1.12 ± 0.60	-0.67 ± 1.02	-0.83 ± 1.18	-3.00 ± 1.22
P	c	0.25 ± 0.85	-0.83 ± 1.02	-2.50 ± 1.18	-3.00 ± 1.72
	p	-0.25 ± 0.60	-1.12 ± 0.89	0.33 ± 1.18	-1.00 ± 1.40

Values are LS means ± SEM.
ns=non-significant, P>0.05.

Experiment II:

Appendix 8. Gilt weight change (kg) during each stage of lactation

		Stage of Lactation			
Factor		**d 0 to d 6**	**d 7 to d 12**	**d 13 to d 18**	**d 0 to d 18**
Gestation Trt.		ns	ns	ns	ns
	C	-2.69 ± 1.43	-1.62 ± 2.14	-2.34 ± 2.26	-7.41 ± 2.34
	P	0.94 ± 1.64	-4.35 ± 2.53	-1.62 ± 2.68	-5.04 ± 2.67
Lactation Trt.		P = 0.0331	ns	P = 0.0158	ns
	c	1.75± 1.57	-2.91 ± 2.34	-6.84 ± 2.48	-8.77 ± 2.55
	p	-3.50 ± 1.52	-3.06 ± 2.34	2.87 ± 2.48	-3.69 ± 2.47
Gest.*Lact.		ns	ns	ns	ns
C	c	-0.50 ± 1.92	-1.48 ± 2.70	-4.68 ± 2.86	-8.20 ± 3.12
	p	-4.87 ± 2.14	-1.75 ± 3.31	0.00 ± 3.50	-6.62 ± 3.49
P	c	4.00 ± 2.48	-4.33 ± 3.82	-9.00 ± 4.04	-9.33 ± 4.03
	p	-2.12 ± 2.14	-4.37 ± 3.31	5.75 ± 3.50	-0.75 ± 3.49

Values are LS means ± SEM.
ns=non-significant, P>0.05.

Experiment II:

Appendix 9. Gilt body protein change (kg) during each stage of lactation

			Body Protein Change (kg)			
Factor			**d 0 to d 6**	**d 7 to d 12**	**d 13 to d 18**	**d 0 to d 18**
Gestation Trt.			P = 0.0142	ns	ns	ns
		C	-0.50 ± 0.22	-0.16 ● 0.44	-0.13 ● 0.54	-0.85 ● 0.45
		P	0.56 ± 0.29	-0.62 ● 0.47	-0.01 ± 0.62	-0.43 ● 0.61
Lactation Trt.			P = 0.0118	ns	P = 0.0473	ns
		c	0.56 ± 0.28	-0.22 ± 0.44	-0.99 ● 0.54	-0.84 ± 0.56
		p	-0.51 ± 0.23	-0.57 ● 0.47	0.85 ● 0.62	-0.44 ● 0.51
Gest.*Lact.			ns (0.0701)	ns	ns	ns
	C	c	-0.31 ± 0.29	0.19 ± 0.50	-0.82 ± 0.62	-1.10 ± 0.60
		p	-0.68 ± 0.33	-0.52 ± 0.72	0.56 ± 0.88	-0.60 ± 0.67
	P	c	1.45 ± 0.47	-0.64 ± 0.72	-1.16 ± 0.88	-0.57 ± 0.95
		p	-0.35 ± 0.33	-0.61 ± 0.61	1.13 ● 0.88	-0.29 ● 0.77

Values are LS means ● SEM.
ns=non-significant, P>0.05.

Experiment II:

Appendix 10. Gilt body lipid change (kg) during each stage of lactation

			Stage of Lactation			
Factor			**d 0 to d 6**	**d 7 to d 12**	**d 13 to d 18**	**d 0 to d 18**
Gestation Trt.			ns	ns	ns	ns
		C	-0.66 ± 0.81	-2.65 ● 1.14	-1.16 ± 1.14	-5.38 ± 1.20
		P	0.62 ● 1.04	-2.29 ± 1.24	-1.90 ● 1.31	-3.96 ± 1.63
Lactation Trt.			P = 0.0227	ns	P = 0.0396	ns
		c	1.73 ± 1.01	-2.86 ● 1.14	-3.56 ± 1.14	-5.37 ± 1.50
		p	-1.77 ± 0.85	-2.08 ± 1.24	0.50 ● 1.32	-3.98 ● 1.38
Gest.*Lact.			ns	ns	ns (0.0761)	ns
	C	c	1.40 ● 1.08	-3.56 ● 1.32	-1.48 ● 1.31	-4.87 ● 1.60
		p	-2.71 ± 1.20	-1.73 ● 1.87	-0.83 ● 1.86	-5.89 ± 1.79
	P	c	2.05 ± 1.70	-2.16 ● 1.87	-5.64 ● 1.86	-5.86 ± 2.53
		p	-0.82 ± 1.20	-2.42 ● 1.62	1.83 ± 1.86	-2.06 ± 2.07

Values are LS means ± SEM.
ns=non-significant, P>0.05.