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EFFECT OF PORCINE SOMATOTROPIN, SOYBEAN OIL AND ENERGY INTAKE ON MAMMARY DEVELOPMENT IN GESTATING GILTS

By

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ABSTRACT

EFFECT OF PORCINE SOMATOTROPIN, SOYBEAN OIL AND ENERGY INTAKE ON MAMMARY DEVELOPMENT IN GESTATING GILTS

By

Kimberly Ann Howard

Three studies were conducted to determine the influence of porcine somatotropin, dietary soybean oil (SBO) and energy intake on gilt (n = 37, 27 and 34, respectively) mammary development during late gestation. In each study, gilts were killed and total mastectomies were performed on d 105 of gestation. Mammary tissue was separated into parenchymal and extra-parenchymal tissue. Parenchymal tissue was weighed and analyzed for DNA, RNA, water, protein and lipid. Treatments in the first study were dietary energy (6.1 or 10.5 Mcal ME·d^{-1}) and porcine somatotropin (0 or 8 mg·d^{-1} PST). In the second study treatments were additions of 0 or 5% SBO to the diet. Treatments were initiated on d 75. Compared with placebo-injected controls, gilts injected with PST had more ($P < .05$) parenchymal tissue (1609 g vs 1963 g; SEM = 136.5). Compared with placebo-injected controls fed adequate energy diets, PST-injected gilts fed adequate energy diets had more ($P < .05$) parenchymal DNA, RNA and protein. Feeding elevated energy inhibited the positive effect of PST on mammary secretory tissue growth. Parenchymal weight, DNA, RNA and protein were not altered ($P > .10$) by feeding 5% SBO from d 75 to 105 of gestation. Treatments in the third study were energy intakes of 6.1 or 10.5 Mcal ME·d^{-1} fed over two periods (d 0 to 74 and d 75 to 105 of gestation), to achieve variation in body composition over

the last third of gestation. Body composition was measured on d 75 and 105 of gestation. Total parenchymal weight, parenchymal DNA, parenchymal RNA and parenchymal protein were not altered by feeding elevated energy diets regardless of the gestational period in which elevated energy was fed. In addition, total DNA and RNA on d 105 were not related ($P > .10$) to empty body fat or protein on d 75 or 105, or to changes in empty body fat or protein from d 75 to 105. In conclusion, elevated dietary energy and 5% SBO additions in late gestation did not enhance or impair mammary development; however PST injections increased mammary secretory tissue. Results of these studies strengthen the potential for use of PST in commercial swine production to improve milk production.

DEDICATION

This dedication is to my family because without their love, support and guidance the completion of my dissertation would not have been possible. My mother, Joyce, has inspired me throughout my life to achieve the best that I am capable of. My brothers and sister-in-laws, Ricky, Bobby, Corinne and Alice, supported me in the good times as well as the bad. My father, Richard, had a determination to succeed which he shared with my brothers and myself. My niece, Leilani, and nephews, Brandon, Clinton and Nicholas, allow me to see through the eyes of a child and marvel at the joy it is to be alive.

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Introduction

The goal to increase sow productivity continues to be a high priority in commercial swine production. To increase the number of pigs weaned per sow per year and litter weaning weights, the sow must produce more milk. The association between nutrition and the growth of milk secretory tissue during gestation is not well understood, and must be in order to maximize milk production in the sow. Deoxyribonucleic acid is an indicator of mammary secretory cell number (Tucker, 1987), because the DNA content per mammary cell nucleus is constant during pregnancy and lactation (Tucker and Reece, 1962). Ribonucleic acid is a reflection of the functional state of the cell, and the RNA/DNA ratio is an indicator of protein synthesis activity per cell in the mammary gland. Total DNA ($r = .85$) and RNA ($r = .93$) in the rat mammary gland were highly correlated with total litter weight gains (Tucker, 1969). Due to the relationships between mammary DNA, mammary cell number and milk production, differences in mammary DNA can be used as an indicator of subsequent milk production differences.

Anatomy

The anatomy of the swine mammary gland has been known for decades (Turner, 1952). Swine mammary glands extend over the entire abdominal wall in the form of two parallel gland areas with each gland being separate and distinct from the other glands. Teat number usually ranges between 10 and 14 with each teat connecting to a pair of glands. The

arterial circulation supplies the posterior glands from the aortal branch, while the common carotid artery supplies the anterior glands. Anastomosis occurs between the second and fourth inguinal glands for the anterior and posterior mammary arteries and veins. Teats of swine usually have two streak canals which open into two distinct teat cisterns which lead into gland cisterns. Above the gland cisterns, ducts branch to all parts of the gland tissue. The ducts lead to lobes which contain lobules with alveoli (milk secretory cells).

Little change occurs in the amount of DNA in the swine mammary gland prior to d 50 of gestation (Hacker and Hill, 1972). Hacker and Hill (1972) studied mammary development in pigs at estrus, d 25, d 50 and d 100 of gestation. They found that approximately 100% of the increase in parenchymal tissue weight, parenchymal DNA and parenchymal RNA was accounted for in the last half of pregnancy. Kensinger et al. (1982) found similar results in swine with mammary DNA concentrations increasing four-fold between d 75 and d 90 of gestation. Amounts of parenchymal tissue and parenchymal DNA plateaued at d 90 of gestation while parenchymal RNA steadily increased through d 4 of lactation. The increase in RNA represents increases in protein synthetic capabilities of the mammary cell which is in agreement with Hacker and Hill (1972) who found that RNA:DNA ratios also increased through pregnancy.

Histological results in swine show progressive duct growth early in pregnancy followed by rapid lobulo-alveolar development between d 75 and d 90 of gestation to complete mammogenesis (Kensinger et al., 1982). Likewise, Weldon et al. (1991) demonstrated significant increases in mammary growth in the pig between d 75 and 105 of gestation. These results are further substantiated by Noblet et al. (1985) who

demonstrated that there was a 20% decrease in the amount of mammary tissue fat from mid-pregnancy to late pregnancy in swine. As the day of gestation and level of feed intake increased (d 75 to d 102; 1.8 kg/d to 2.5 kg/d, respectively), the weight of mammary tissue also increased (902 g to 2885 g). Mammary tissue weight and chemical composition increased exponentially with stage of gestation. In addition, increasing feeding level increased mammary tissue weight at d 102 of gestation. Thus, it seems that the majority of mammary secretory tissue growth occurs during late pregnancy in the pig.

Endocrinology

Conceptus number. Mammary development is stimulated by the presence of conceptuses, most likely due to the concurrent rise in estrogen which occurs with fetal growth. Dehoff et al. (1986) used psuedopregnancy to investigate the influence of conceptus number on mammary development. Psuedopregnancy was induced in non-gravid cycling gilts by injecting estradiol valerate from day 11 to 15 of the estrous cycle. Psuedopregnant gilts had 22% less mammary DNA than pregnant gilts by d 110 of gestation; however, mammary development did not differ when conceptus number ranged between 4 and 13 (Dehoff et al., 1986; Noblet et al., 1985). Dehoff et al. (1986) suggested that estrogen may be the primary conceptus-derived mammogenic hormone in gilts due to significant correlations between estrogen-sulfate or estrone concentrations in blood and maternal mammary DNA content. The major rise in circulating estrogens began between d 60 and 75 of gestation, which is associated with the major period of mammary growth.

Thyrotropin releasing hormone. Mammary secretory tissue growth in the pig may be related to changes in the concentrations of other hormones. Grieshop et al. (1991) reported that injecting thyrotropin releasing hormone (TRH) during a 28-d prepubertal period and a 50-d prepartum period (d 64 of gestation to parturition) increased DNA concentrations and 21-d piglet milk consumption. However, total DNA and the amount of mammary parenchymal tissue did not differ. Feed consumption during the lactation period increased as TRH levels increased which confounded the effects of feed consumption and TRH levels on 21-d piglet milk consumption. It is possible that the increase in milk production was related to the increase in feed consumption during lactation which could explain the lack of differences in total DNA and parenchymal tissue. Feed intake may be one of several other factors influencing milk production.

Growth hormone in the pregnant and lactating pig. Plasma concentrations of growth hormone (GH) remain fairly constant throughout pregnancy in swine (12 ng/ml), rising during the periparturient period (Dehoff et al., 1986). Fetal GH concentrations increase from d 60 and peak at d 90 (> 150 ng/ml) of gestation, however maternal concentrations of GH remain low, which indicates that GH may not cross the fetoplacental barrier (DeHoff et al., 1986).

Sows injected with growth hormone during gestation and lactation had higher IGF-I and glucose concentrations compared with non-injected controls (Baile et al., 1989; Smith et al., 1991). Okere and Hacker (1995) injected gestating gilts with 5 mg/d porcine somatotropin and .5 IU/kg insulin from d 30 to 70 of gestation and found that maternal serum IGF-I levels and maternal d 133 BW were increased but there was no

difference in piglet serum IGF-I levels suggesting that IGF-I production in maternal and fetal compartments are independent. Gregor and Burleigh (1985) have demonstrated in sows that there are receptors for IGF-I on mammary epithelial cells. Kvergas et al. (1986) found that pigs from GH-injected sows during late gestation had higher total body lipids, serum glucose concentrations, and liver glycogen concentrations at birth and tended to have increased mean free fatty acid concentrations.

The influence of growth hormone on swine mammary DNA and RNA has not been directly studied; however, there have been several studies conducted involving injections in late gestation (Baile et al., 1989; Boyd et al., 1984; Crenshaw et al., 1989; Cromwell et al., 1989b; Kvergas et al., 1986) and through lactation (Cromwell et al., 1989a; Harkins et al., 1989; Smith et al., 1991) to determine the influence of growth hormone on sow lactational performance. The studies produced conflicting results.

Although some reports show increased milk production for sows injected with growth hormone during gestation, the majority of reports indicate that milk production is not effected in sows injected as early as d 91 of gestation. Sows injected with an isolated porcine growth hormone preparation (USDA/NPA, 4.5 $IU \cdot mg^{-1}$, 24 $IU \cdot sow^{-1} \cdot d^{-1}$) from d 100 of gestation to d 21 of lactation produced 15% more milk by d 21 of lactation; however, milk yields were similar at d 14 of lactation (Boyd et al., 1984). Contradictory to this work, injecting sows with PST beginning as early as d 94 of gestation, with or without 8% added fat, did not increase milk yield or piglet body weight (Baile et al., 1989; Crenshaw et al., 1989; Cromwell et al., 1989b; Kvergas et al., 1986).

Similar to PST-injection during late gestation, injection during lactation has not resulted in consistent increases in milk production. Harkins et al. (1989) found that injecting lactating sows daily with porcine somatotropin (PST) from d 12 through d 29 of lactation increased milk production 22% and increased piglet weights (.34 kg/pig) at the end of the lactation period. However, milk composition was similar for sows regardless of PST injections. Smith et al. (1991) demonstrated increased weaning weights of pigs only if they were from heavier litters (averaged $\geq$ 2.6 kg·pig/BW) on d 7 of lactation when lactating sows were injected daily from d 7 to d 20 with PST. Weekly injections of PST (70 mg) on d 3, d 10, d 17 and d 24 of lactation does not increase milk yield or alter the average litter weaning weight (Cromwell et al., 1989a). In most of the studies, sows injected with PST during lactation lost more weight and had less backfat than control sows (Cromwell et al., 1989a; Harkins et al., 1989).

Sow deaths related to PST injections during gestation and lactation have been noted in several studies. Eighteen out of ninety-eight sows injected with PST during late gestation and lactation died due to heat stress (Cromwell et al., 1989b). Trials were conducted from May to July. Extreme respiratory distress was reported by Kvergas et al. (1986) just prior to, during or shortly after parturition. Daily injections of 4, 8 or 16 mg of recombinant PST to lactating sows from d 7 to d 20 caused 6, 20 and 90% mortality , respectively, due to hemorrhaging of ulcers that developed at the pars esophagea (Smith et al., 1991). Higher doses of PST had no apparent negative effects in growing pigs (average BW 50 to 100 kg), suggesting that lactating sows (average BW 202 to 270 kg) were more sensitive to exogenous PST (Smith

et al., 1991). The majority of the deaths related to PST injections occurred around farrowing time except in the case of Smith et al. (1991) who injected significantly more PST per sow (16 $mg \cdot sow^{-1} \cdot d^{-1}$ versus 10 $mg \cdot sow^{-1} \cdot d^{-1}$ or less) than other researchers. Therefore, injecting sows earlier in gestation and ceasing prior to parturition may not result in sow deaths to the PST injections.

Growth hormone injections from late gestation through lactation seem to produce inconsistent results with respect to subsequent increases in milk production. Day 91 of gestation is the earliest in the allometric growth phase of the mammary gland that exogenous GH has been administered to sows thus far. Since the allometric growth phase of the gestating pig mammary gland begins between d 75 and d 90 of gestation, it is possible that in the studies conducted previously, injections of PST were not administered early enough in gestation to achieve the optimal effect on mammary development. Therefore, it may be possible to increase mammary secretory tissue by injecting gilts from d 75 of gestation.

Growth hormone in other species. In cattle and sheep, high dietary energy and protein decrease serum GH concentrations and mammary growth (Sejrsen et al., 1983; Johnsson et al., 1985). Prepubertal heifers fed ad libitum had decreased GH concentrations compared with heifers fed restricted diets, which was associated with reduced mammary secretory cell numbers (Sejrsen et al., 1983). Total parenchymal DNA was positively correlated (r=.40) with plasma GH concentrations while extraparenchymal adipose tissue was negatively correlated (r = -.53) with serum GH concentrations. Similar to prepubertal heifers, pregnant heifers fed ad libitum as compared with those fed moderately during the

isometric (1 year of age to 3 months of gestation) and the allometric (3 months of gestation to 14 d before calving) growth phases of gestation had lower growth hormone concentrations, although concentrations of IGF-I were similar during both phases of gestation (Lacrasse et al., 1994). There were positive relationships between postpartum concentrations of growth hormone and both peak milk production and feed intake (Lacrasse et al., 1994). Prepubertal heifers injected with bovine somatotropin (bST) around puberty (8 months of age) had more parenchymal tissue than placebo-injected controls (Sejrsen et al., 1986). Furthermore, bST-injected prepubertal heifers had increased total DNA, total RNA and RNA/DNA ratios compared with non-injected controls (Radcliff et al., 1995).

Prepubertal lambs fed restricted diets had higher mean plasma concentrations of GH and reduced concentrations of prolactin and greater mammary development, with the impact of feeding level decreasing as age of the lamb increased (Johnsson et al., 1985). The stimulating effect of food intake restriction on endogenous secretion of GH declined markedly with age in the first 20 wk in female lambs. Mammary parenchymal DNA was positively correlated to mean plasma GH ($r = .95$) and the GH:insulin ratio ($r = .39$) and negatively correlated with the prolactin:GH ratio ($r = -.65$) in 20-wk-old lambs (Johnsson et al., 1986). Prepubertal lambs injected with GH and fed ad libitum had more parenchymal tissue and parenchymal DNA than those animals on a restricted diet or fed ad libitum (Johnsson et al., 1986). Growth hormone injections did not affect the proportions of epithelial, connective and fat tissues at 20 wk of age (Johnsson et al., 1986). In another study, prepubertal lambs injected with GH had a greater amount

of parenchymal tissue and parenchymal DNA; however, the differences were not significant because five out of ten lambs injected with GH died with no definitive cause of death (McFadden et al., 1990).

From this information, it appears that low GH concentrations decrease the magnitude of the mammary growth in prepubertal ruminants. In addition, administration of GH to prepubertal heifers and sheep increases mammary secretory tissue growth. Akers et al. (1985) speculated that the effects of GH on mammary growth may be indirect, mediated through production of somatomedin C by the liver.

Nutrition and Mammary Development

Swine. Manipulation of the gilt's diet during gestation may affect mammary secretory tissue growth based on limited studies conducted to date. In a study conducted previous to the experiments in this dissertation, excessive dietary energy intake (> 10.0 Mcal ME d^{-1}) in late gestation was detrimental to the development of mammary secretory tissue (Weldon et al., 1991) when the results were expressed per number of functional glands and per kg maternal body weight. Gilts receiving excessive energy intake had 27% less parenchymal weight, 30% less total parenchymal DNA, less parenchymal RNA and protein than gilts receiving adequate energy intake (5.76 Mcal ME/d). In the same study, differences in dietary protein intake between d 75 and 105 of gestation were not related to mammary development.

Other species. The relationship between nutrition and mammary growth has been studied more extensively in species other than swine. It is important to understand the similarities and differences in mammary

development among species to improve our understanding of swine mammary development.

In rats, cattle and sheep there is a period of prepubertal allometric growth of the mammary gland (Johnsson and Hart, 1985; Sinha and Tucker, 1969a; Sinha and Tucker, 1969b) which is adversely affected by a high plane of nutrition (Johnsson and Hart, 1985 and Sejrsen et al., 1982). The mammary tissue grows at a faster rate than the rest of the body during the allometric growth phase. Sinha and Tucker (1966) found that the allometric growth of the rat mammary fat pad occurs 1.56 and 1.96 times faster than body surface area based on total mammary gland fat pad DNA and specific mammary area, respectively. Cowie (1949) demonstrated that the rat allometric growth phase could be inhibited by ovariectomy; therefore, illustrating the necessity of estrogen and progesterone secretion for the allometric growth phase to occur. Prepubertal mammary growth has not been studied in swine; however, the prepubertal growth period in other species is similar to the growth of the pig mammary gland during late gestation.

Mammary secretory tissue weight and DNA content decreased 23% and 32% in prepubertal heifers fed ad libitum as compared with prepubertal heifers fed restricted diets (Sejrsen et al., 1982). In contrast, plane of nutrition had no affect on postpubertal heifer mammary secretory tissue growth (Sejrsen et al., 1982). Parenchymal tissue composition was not altered by plane of nutrition in either the pre- or postpubertal period. Parenchymal tissue consisted of 10% epithelial cells, 50% connective tissue, 30 to 40% fat cells and 2 to 3% ductal lumen. In contrast to restricted versus ad libitum feeding, mammary secretory tissue growth was similar for prepubertal Holstein heifers fed control

diets ad libitum or fed diets more concentrated in energy and protein ad libitum (Radcliff et al., 1995). Prepubertal sheep fed a restricted diet had more total parenchymal tissue and total parenchymal DNA than lambs fed ad libitum (Johnsson and Hart, 1985; Johnsson et al., 1986). Lambs fed restricted diets as compared with lambs fed ad libitum had higher relative growth coefficients for mammary parenchyma, estimated from the increase in DNA relative to that for live weight, 3.7 versus 2.4, respectively (Johnsson and Hart, 1985). A high plane of nutrition compared to restricted feeding during the allometric mammary growth period in prepubertal ruminants has a detrimental effect similar to that of high dietary energy during the allometric growth phase of the gestating pig. It is possible that similar mechanisms are involved with respect to nutrition and it's impact during the allometric growth phase of the mammary gland in the gestating pig.

A high plane of nutrition fed to heifers during gestation does not have a negative effect mammary gland development (Harrison et al., 1983; Valentine et al., 1987) or subsequent milk production (Ducker et al., 1985; Lacrasse et al. 1993). Harrison et al. (1983) found that mammary development was not impaired in dairy heifers which were reared rapidly during pregnancy but were moderately reared prior to one year of age. Likewise, Valentine et al. (1987) found mammary gland development and subsequent milk production to be similar in heifers fed 3 planes of nutrition during the last half of pregnancy. Lacrasse et al. (1993) reported that milk production in dairy heifers was not affected by plane of nutrition (ad libitum or moderate feeding (80 and 70% of ad libitum)) fed during the allometric growth phase of the mammary gland during pregnancy (3 months of gestation to 14 d before calving). Additionally,

Ducker et al. (1985) found that heifers fed a high plane of nutrition during the last 10 weeks of pregnancy had similar milk yields to heifers fed a lower plane of nutrition. Thus, it seems that there may be differences in cattle and swine mammary development during gestation with respect nutritional influences. Specifically, mammary development in swine may be inhibited by excessive energy intake during late gestation whereas mammary development is not influenced in cattle fed a high plane of nutrition during gestation.

Dietary fat. Dietary polyunsaturated fatty acids (PUFA) stimulated mammary parenchymal development in prepubertal sheep (McFadden et al., 1990a). Lambs fed a formaldehyde-protected sunflower seed supplement (by-pass fat, approximately 8.3% sunflower oil) had increased parenchymal weight and parenchymal DNA compared with lambs fed control diets without fat additions. In addition, PUFA-fed lambs also had more extraparenchymal tissue, which could subsequently result in future space for parenchymal growth to occur. Lambs fed diets with PUFA had twice as much linoleic acid in the parenchymal lipid compared with lambs which were not fed diets containing PUFA (McFadden et al., 1990b). Dietary PUFA increased the number of GH receptors in the liver and the number of prolactin receptors in the mammary parenchyma. Carrington and Hosick (1985) have proposed that increases in numbers of receptor sites may result from increased membrane fluidity caused by higher unsaturated fatty acid contents. Wahle (1983) demonstrated that accumulation of unsaturated fatty acids increases membrane fluidity, and Dave and Knazek (1980) demonstrated that modification of membrane fluidity can alter hormone binding.

Mammogenesis in mice is increased as a result of feeding dietary PUFA (Welsch et al., 1985). Mammary gland development increased as dietary corn oil increased from 0 to 5%. The difference between 5 or 20% corn oil on mammary gland development was not as great as the difference between 0 and 5%; however, differences between 5 and 20% corn oil when comparing whole-mount analyses of mammary glands reached the 7% level of statistical probability. In-vitro responsiveness of mammary epithelial area to estradiol and progesterone also increased as the fat content of the diet increased. The response increased from 5 to 20% but it was not as pronounced as the increase from 0 to 5%. Therefore, the responsiveness of the mammary epithelial area to estradiol and progesterone was greatest when the levels of corn oil supplied provided adequate essential fatty acids to meet the requirements for essential fatty acids. There was a linear increase in the number of [^{3}H]-thymidine-labeled mammary epithelial cells as fat increased from 0 to 5 to 20%.

Diets fed to mice containing 20% coconut oil (92.8% saturated fatty acids) inhibited growth of normal endogenous mammary epithelial cells (Carrington and Hosick, 1985). Normal rat mammary and tumorgenic cell growth in vitro was stimulated by the addition of PUFA whereas the addition of saturated free fatty acids inhibited cell growth (Wicha et al., 1979). Linoleic and linolenic acids were the most stimulatory while stearic acid inhibited cell growth at all concentrations that were tested. The optimal concentration of unsaturated free fatty acids in culture roughly paralleled the percentage distribution of these fatty acids (free plus esterified) in the mammary gland and serum.

The effects of PUFA on mammary development in swine has not been studied to date. Due to similarities which exist between swine mammary development and mammary development of other species, it is possible that mammary secretory tissue growth will increase as a result of feeding dietary PUFA. In addition, dietary PUFA may be synergistic with exogenous growth hormone on mammary development due to possible increases in receptor availability for growth hormone.

Soybean oil fed at a rate of 1-5% of the diet is commonly used by commercial swine producers to improve energy intake in hot weather and control dust levels in swine facilities. The use of soybean oil for these practical reasons may also result in increased mammary development in gestating gilts.

Body Composition

Techniques. Various methods have been used to determine body composition in pigs. Chemical analysis of the entire ground animal is the most accurate method; however, it is time consuming, very expensive and requires killing the animal. The sawdust-residue analysis procedure was the most accurate technique in growing swine (birth to 145 kg weight) when compared to using specific gravity or backfat thickness measurements for prediction of empty body water, protein and fat (Shields et al., 1983b). The sawdust-residue analysis involves chemical analysis of the sawdust-residue accumulated from cross-sectional sawing of the frozen carcass. Similar results were found in reproducing swine in which the sawdust residue technique was more accurate than the specific gravity technique to determine carcass or maternal body composition (Shields and Mahan, 1983). The methods used to determine

body composition mentioned thus far are laborious and require death of the animal. Therefore, these techniques are not suitable to study changes in vivo body composition. Researchers have developed several indirect methods to evaluate body composition which do not require killing the animal, thus these methods are suitable to study in vivo body composition. The deuterium oxide dilution technique has been used to estimate in vivo body composition in neonatal pigs (BW = 4 kg), growing pigs (BW = 7 kg to 145 kg), sexually mature (nongravid) gilts, and pregnant and lactating sows (Rudolph et al., 1988; Shields et al., 1983a; Rozeboom et al., 1994; Shields et al., 1984). Shields et al. (1984) found that predicted fat, ash, protein and water values developed from maternal body equations were similar to actual values regardless of stage of pregnancy (d 0, d 57 or d 105). Prediction equations for protein (R^2= .25 and .23, empty body protein and maternal body protein, respectively) and ash had the lowest accuracy because they involved the composite error of estimation of the other body components. Rozeboom et al. (1994) found empty body protein had an R^2 of .99 for Yorkshire x Landrace gilts and .87 for Duroc x (Yorkshire x Landrace) gilts. These researchers concluded that prediction equations using D_2O space, live weight and backfat thickness were accurate in estimating body composition of animals only when the equations were derived from animals that physiologically resembled the population in which the equations were derived.

Body composition and mammary development. Relative to body composition, Head and Williams (1991) and Head et al. (1991) found that DNA concentrations and estimated mammary cell numbers were less in fat versus lean gilts at d 112 of gestation. Body weight was similar.

Backfat levels were 36 mm and 25 mm for fat and lean gilts, respectively. Fat gilts had half as many aveolar (milk secretory) cells as lean gilts based on the numerical density of aveolar cells per gram of mammary gland. Parenchymal weights and parenchymal fat did not differ between fat and lean gilts. To summarize, high energy intake during late gestation, which would lead to fatter gilts, may have a negative impact on mammary growth in swine. The mechanism by which this may occur remains unclear.

Conclusion

Studies conducted to-date indicate that a high plane of nutrition during gestation does not influence mammary development in heifers; however, ad libitum as compared with restricted feeding decreases mammary growth in prepubertal heifers and sheep. Additionally, elevated energy intake during late gestation in gilts may be detrimental to mammary secretory tissue growth which contradicts the findings in pregnant heifers. The effect of energy intake on gilt mammary development and potential mechanisms whereby excessive energy intake may elicit it's response needs to be determined. Two potential mechanisms whereby excessive energy intake may decrease mammary secretory tissue growth include decreased GH concentrations and increased body fat. Exogenous GH administered to ruminants increases mammary secretory tissue growth. In addition, polyunsaturated fatty acids stimulate mammary development in other species and may be synergistic with growth hormone. In conclusion, the effect of energy intake, body composition, PUFA and growth hormone on mammary development in gestating swine warrants further research.

CHAPTER 1. EFFECTS OF EXOGENOUS PORCINE SOMATOTROPIN AND DIETARY ENERGY ON MAMMARY DEVELOPMENT OF SWINE DURING LATE GESTATION

ABSTRACT

Fifty gilts were used to determine effects of dietary energy and exogenous porcine somatotropin (PST) on mammary development during late gestation. Ten gilts were randomly chosen and slaughtered on d 75 of gestation to describe mammary growth prior to treatment implementation. On d 75 of gestation, the remaining forty gilts were randomly assigned in a 2 x 2 factorial arrangement to dietary regimes of adequate (A) or elevated (E) energy (6.1 or 10.5 Mcal ME/d, respectively) and injections of 0 or 8 mg/d PST. These gilts were killed on d 105 of gestation. Total mastectomies were performed on all carcasses following exsanguination. Mammary tissue was separated into parenchymal (P) and extraparenchymal stromal (EPS) tissue and analyzed for DNA, RNA, protein and lipid. Tenth rib backfat on d 105 of gestation was less ($P < .05$) for gilts injected with PST regardless of energy level (1.99 cm vs 2.85 cm). There was no effect of dietary energy level or exogenous PST on number of fetuses; however, average fetal weight was greater ($P < .05$) for gilts fed E diets (1.1 kg vs .9 kg). Amount of parenchymal tissue, total parenchymal DNA, RNA and protein did not differ with dietary energy level. Neither dietary energy level or exogenous PST influenced the amount of EPS. However, gilts injected with PST had more ($P < .10$) P tissue (1609 g vs 1963 g). Total parenchymal DNA, RNA and protein were greater ($P < .05$) for A gilts injected with PST (4,406 mg, 7,516 mg and 176 g, respectively) than in A gilts not injected with PST (2,636 mg, 4,250 mg and 105 g, respectively). Feeding elevated energy may have inhibited the positive effect of PST on mammary secretory tissue growth,

although how this takes place is unclear at this time. Results of the present study strengthen the potential for use of PST in commercial swine production to improve sow milk yields.

Key Words: Swine, Mammary development, Growth hormone

Introduction

Each year greater production demands are put on the sow in commercial swine production. As producers strive to improve their competitiveness there is increased pressure on the sow to produce more milk and increase the kilograms of pigs weaned per sow per year. To maximize milk production, feeding and management strategies must provide for proper growth of the mammary gland.

In sheep, cattle and rats there is a period of prepubertal allometric growth of the mammary gland (Johnsson et al., 1985; Sinha and Tucker, 1969a; Sinha and Tucker, 1969b), which is adversely affected by a high plane of nutrition (Johnsson and Hart, 1985; Sjersen et al., 1982). In ruminants, ad libitum as compared with restricted feeding decreases serum growth hormone (GH) concentrations (Sjersen et al., 1982; Johnsson et al., 1985). These decreases in GH concentrations are associated with reduced mammary cell numbers (as measured by DNA content) and a decrease in subsequent milk production. In prepubertal lambs fed ad libitum injections of GH restore DNA content of mammary glands to levels similar to those of animals on a restricted diet (Johnsson et al., 1986). From this information, it appears that GH concentrations have a positive effect on the magnitude of the prepubertal mammary gland growth in ruminants.

Prepubertal mammary growth has not been studied in swine; although,

research has been conducted in gestating gilts. Little change occurs in the DNA content of the mammary gland prior to d 50 of gestation (Hacker and Hill, 1972). However, DNA concentrations increase four-fold between d 75 and d 90 of pregnancy (Kensinger et al., 1982). Excess dietary protein fed to gestating gilts from d 75 to 105 of gestation has no effect on mammary tissue development while excess energy intake seems to be detrimental to the development of mammary secretory tissue (Weldon et al., 1991).

It is possible that GH concentrations and mammary development could be related in swine, therefore GH may play a role in mammary development during pregnancy but how is not known. Initial research suggest that during pregnancy, plasma concentrations of GH remain fairly constant in swine (Dehoff et al., 1986). The objective of this experiment was to assess the interaction between dietary energy level and exogenous porcine somatotropin (PST) on mammary tissue development in pregnant gilts.

Material and Methods

Animals. Fifty Yorkshire or Yorkshire x Landrace gilts, weighing 161 kg, were initially selected for use in this study. Two gilts were removed from the study because they were not pregnant, and one gilt died during the study. Cause of death was unknown. Ten gilts were used to measure mammary development at d 75 of gestation. After being bred on their second estrus, gilts (n = 47) were moved into an environmentally controlled building (minimum temperature 18.3°C, 16 hr light) and housed in individual stalls (stall size .6 m x 2.1 m). All gilts were fed 1.82 kg/d of a diet formulated to meet NRC (1988) requirements for protein,

energy and all other nutrients (designated Adequate energy; Table 1) to d 75 of gestation.

Treatments. A 2 x 2 randomized factorial design was employed. Main effects included either feeding elevated (10.5 Mcal ME/d [E]) or adequate (6.1 Mcal ME/d [A]) dietary energy and injecting gilts with 0 or 8 mg porcine somatotropin (PST), both from d 75 to 105 of gestation. Desired dietary energy intake was achieved by changing concentrations of corn and soybean meal, by the addition of cornstarch to the diet, and by altering feed intake. Daily intake of supplemental vitamins and minerals was held constant across all treatments. Lysine was held constant across all treatments by the addition of L-lysine-HCL. PST was injected immediately following feeding of the gilts. The gilts receiving 0 mg PST/d were injected with a placebo saline solution.

Slaughter. On d 75 (10 gilts) and d 105 (37 gilts) of gestation, animals were electrically stunned and exsanguinated. After exsanguination, total mastectomies were performed and the mammary glands were immediately frozen in a mixture of dry ice and acetone. Glands were stored at -20^{o}C until analyses were performed. Individual fetuses were euthanized, counted and weighed. In addition, hot carcass weight, backfat and loin eye area at the 10th rib were obtained.

Mammary Dissection and Chemical Analysis. The right side of the frozen mammary glands was separated into parenchymal (P) and extraparenchymal stromal (EPS) tissue (Weldon et al., 1991). Tissues were weighed and stored at -20^{o}C until they were homogenized.

Mammary P and EPS were analyzed for DNA, RNA and lipid content as described by Tucker (1964). Protein was determined using the method of Lowry et al. (1951), with bovine serum albumin used as the standard.

Statistical Analysis. Data were subjected to ANOVA using the general linear model of SAS (1985). Due to the presence of interactions ($P < .10$) between dietary energy level and PST on the dependent variables, four conditional contrasts using the Bonferonni t-test, were used to test treatment effects. The conditional contrast tested the effect of elevated energy on mammary development (A0 vs E0), the effect of PST on mammary development for gilts fed diets with adequate energy (A0 vs A8), the effect of PST on mammary development for gilts fed elevated energy diets (E0 vs E8) and the effect of dietary energy on mammary development for gilts injected with exogenous PST (A8 vs E8). Interactions did not exist between dietary energy level and PST with respect to mammary parenchymal and extraparenchymal stromal tissue; therefore, results were pooled by main effects rather than using conditional contrasts. Data reported are least squares treatment means with corresponding conditional contrasts.

Results and Discussion

Sow Performance. Gilts fed the elevated energy diets were heavier at the end of the trial ($P < .05$) and had heavier hot carcass weights ($P < .05$; Table 2). Similar to the findings reported by Harkins et al. (1989) and Cromwell et al. (1989a), tenth rib backfat was less ($P < .05$) for gilts injected with PST regardless of energy intake. Weight of uterus plus conceptus was not affected by dietary energy level or PST injections; however, there was a tendency ($P < .10$) for heavier uterine weights without fetuses for gilts fed adequate energy and receiving PST as compared with gilts fed adequate energy and receiving placebo saline injections. There was no effect of dietary energy or PST on number of

fetuses or mummified fetuses (10.5, .30 [A0]; 10.7, .33 [E0]; 10.2, .33 [A8]; 9.7, .44 [E8]; SEM = .86 and .20). However, the average fetal weight (904 g [A], 1,061 g [E]; SEM = 44) was increased ($P < .05$) by feeding diets with elevated energy.

Mammary development. Characteristics of total mammary tissue of gilts sacrificed at d 75 and 105 of gestation are listed in Table 3. Gilts killed on d 75 had less extraparenchymal stromal tissue, parenchymal tissue, parenchymal DNA, parenchymal RNA and parenchymal protein but had a greater concentration of parenchymal lipid than gilts killed on d 105 of gestation. This agrees with previous work with pregnant swine (Hacker and Hill, 1972; Kensinger et al., 1982; Weldon et al., 1991). On d 105 of gestation, mammary tissue contained more total parenchymal tissue, DNA, RNA and protein but had a greater concentration of parenchymal lipid. It is apparent that from d 75 to 105 of gestation there is a dramatic increase in milk secretory tissue capacity.

Mammary extraparenchymal stromal weights (656 vs 651 g; SEM = 61) were not different ($P > .10$) for gilts fed adequate energy diets as compared with gilts fed elevated energy diets from d 75 to 105 of gestation. Likewise, mammary parenchymal weight (1820 g vs 1753 g; SEM = 136) was not different ($P > .10$) for gilts fed adequate energy diets as compared with gilts fed elevated energy diets from d 75 to 105 of gestation. These results contradict those of Weldon et al. (1991) who found that gilts fed diets with adequate energy levels had increased parenchymal weights and similar extraparenchymal stromal weights compared with gilts fed elevated energy diets. Compared with placebo-injected controls, gilts injected with PST had more ($P < .10$) parenchymal tissue (1609 g vs 1963 g; SEM = 136.5) while

extraparenchymal stromal weight (612 g vs 695 g; SEM = 42.5) did not differ ($P > .10$).

The effect of dietary treatment and PST injections on the amount of DNA, RNA, protein and lipid, as well as RNA/DNA ratios in parenchymal tissue are shown in Table 4. Parenchymal lipid was not affected by PST injections or dietary energy levels ($P > .10$). In contrast, parenchymal lipid was decreased in prepubertal sheep fed ad libitum and injected with exogenous growth hormone compared to sheep fed ad libitum and not injected with exogenous growth hormone (McFadden et al., 1990). Parenchymal DNA, RNA and protein, as well as RNA/DNA ratios were similar for gilts provided with elevated energy as compared with gilts provided with adequate energy. Thus, contradictory to the results of Weldon et al. (1991) elevated dietary energy intake in late gestation did not have a detrimental effect on gilt mammary development. Parenchymal RNA was greater ($P < .10$) in gilts injected with PST regardless of dietary energy level fed.

Various energy x PST interaction were identified. Compared with placebo-injected controls fed adequate energy diets, gilts injected with PST and fed adequate energy diets had more ($P < .05$) parenchymal DNA and protein. Additionally, parenchymal DNA was higher ($P < .10$) in PST-injected gilts fed adequate energy diets as compared with PST-injected gilts fed elevated energy diets. Although, PST increased mammary secretory tissue in gilts fed adequate energy diets, feeding elevated energy diets seemed to inhibit the positive effect of PST. The mechanism by which this took place is unclear at this time and warrants further studies to determine the mechanism whereby excess energy is exerting it's influence. Based on the mammary growth data from this

experiment, the present NRC (1988) recommendations for dietary energy seem satisfactory for gilts injected with PST from d 75 to d 105 of gestation.

Little is known about the effect of exogenous growth hormone on porcine mammary development. However, the increase in parenchymal weight, DNA, RNA and protein found in the present study are similar to the observations found in prepubertal sheep (Johnsson et al., 1986; McFadden et al., 1990) and prepubertal heifers (Sejrsen et al., 1986; Radcliff et al., 1995) injected with exogenous growth hormone.

In the present study, mammary secretory tissue growth was similar ($P > .10$) for gilts fed adequate or elevated energy diets from d 75 to d 105 of gestation when the results were expressed on a total basis, or corrected for number of teats and kg maternal BW. In contrast, Weldon et al. (1991) found that feeding elevated energy diets during the same period was detrimental to mammary development. Gilts in both the present study and that of Weldon et al. (1991) were housed in similar environments, fed similar diets and were from similar genetic backgrounds. Body weight of gilts used in the present study were lighter than those gilts used by Weldon et al. (1991). Although not known for sure, it is possible that differences in body fat between gilts in these studies altered the response to differing dietary energy levels. Head and Williams (1992) found that fat versus lean gilts had reduced numbers of mammary secretory cells.

Results of Weldon et al. (1991) were expressed on a per teat and per kg maternal BW basis. This may be another reason for the differences in results when compared to the present study. Since gilts fed elevated energy diets were heavier and number of teats were controlled between

gilts, this correction may not be valid. Furthermore, gilts were heavier due to dietary energy intake, thus body weight is confounded with treatment. We have analyzed the data of Weldon (1988), and expressed DNA and RNA on a total basis. Total parenchymal DNA, RNA, protein and lipid were similar ($P > .10$) for gilts fed adequate and elevated energy diets. The amount of parenchymal tissue tended ($P = .10$) to be less for gilts fed elevated energy diets as compared with gilts fed adequate energy diets. Thus results from the present study as well as from Weldon et al. (1991) demonstrate that elevated energy fed from d 75 to d 105 of gestation is not detrimental to mammary growth when mammary constituents are expressed on a total basis.

Total EPS DNA and RNA tended ($P < .10$) to be less in gilts injected with PST and fed adequate energy diets as compared with placebo-injected controls fed adequate energy diets (Table 5). Weldon et al. (1991) reported that EPS DNA and RNA were not affected by dietary energy level. Likewise, Sjersen (1981) reported that mammary extraparenchymal DNA, RNA and lipid were not affected by restricted or ad libitum feeding in the prepubertal heifer. Total EPS protein was greater for gilts fed adequate energy and injected with PST compared to those gilts fed adequate energy and receiving placebo injections. Total EPS lipid was less ($P < .05$) in gilts injected with PST and fed adequate energy relative to gilts injected with saline and fed adequate energy diets.

Implications

Injecting gilts with exogenous growth hormone combined with feeding diets which meet NRC (1988) recommendations for dietary energy (6,100 kcal ME/d) during late gestation enhances mammary growth, which could

subsequently lead to increased milk production. Feeding regime or energy intake seems to be less critical for mammary development and potential milk production.

Table 1. Composition and calculated analysis of experimental diets

Ingredient, %	Adequate energy	Elevated energy
Corn	85.05	71.30
Soybean meal (44%)	10.22	2.00
Mono-dicalcium phosphate	1.88	.93
Ground limestone	1.35	.90
Vit-TM premix [a]	.60	.48
Vit E-Se premix [b]	.50	.30
Salt	.40	.25
Cornstarch		23.75
L-Lysine HCL		.09
Calculated analysis		
ME, Mcal/kg	3.2	3.27
Crude protein, %	11.7	6.9
Lysine, %	.5	.3
Calcium, %	.9	.53
Phosphorus, %	.7	.41
Daily intake		
Feed, kg	1.90	3.21
ME, Mcal	6.1	10.5
Crude protein, g	228	228
Lysine, g	9.50	9.63

[a]Concentration per kg premix: vitamin A, 661,380 IU; vitamin D, 132,276 IU; menadione, .66g; riboflavin, .66g; niacin, 3.53g; d-pantothenic acid, 2.64 g; choline chloride, 88.18 g; vitamin B12, 3.96 mg; zinc, 7.5 g; iodine, .11 g; copper, 2 g; iron, 12 g.

[b]Concentration per kg premix: vitamin E, 3310 IU; selenium, 19.8 mg.

Table 2. Effects of dietary energy and porcine somatotropin on sow weight and carcass composition

Energy level (mcal/d)	6.1 (A)	10.5 (E)	6.1 (A)	10.5 (E)						
PST (mg/d)[a]	0	0	8	8		Contrast[d]				
	(n = 10)	(n = 9)	(n = 9)	(n = 9)	SEM	A0 vs E0	A0 vs A8	E0 vs E8	A8 vs E8
d 75 wt, kg	160.2	162.7	160.5	160.9	4.7				
d 105 wt, kg	172.3	192.1	186.3	190.9	5.1	*	†		
d 75 to 105 wt gain,kg	14.8	31.4	26.8	31.8	1.7	*	*		*
Utero-placental full wt, kg[b]	17.89	21.30	20.51	18.51	1.80				
Utero-placental empty wt, kg[c]	8.31	10.00	10.43	9.15	.87		†		
10th rib backfat	2.69	3.00	1.95	2.02	.26		*	*	
Loineye area, cm^2	38.99	44.82	43.71	41.92	2.54				
Carcass wt, kg	100.7	110.5	102.3	105.4	3.1	*			

[a]Porcine somatotropin.
[b]Weight including fetuses and fluid.
[c]Weight without fetuses.
[d]†P > .10, *P < .05, **P < .01

Table 3. Characteristics of mammary tissue from gilts at d 75 or 105 of gestation

Item	d 75	SD	d 105[a]	SD
Number of gilts	10		37	
Average wt, kg	155	15	161	13
Extraparenchymal stroma, g	57	20	93	24
Parenchyma				
Weight, g	102	248	249	69
DNA, mg/g	.7	.4	1.9	.2
Total DNA,mg	73	53	476	181
RNA, mg/g	1.2	.3	3.3	1.0
Total RNA, mg	130	56	831	386
Protein, mg/g	42	12	76	14
Total protein, g	5	2	20	8
Lipid, mg/g	609	125	350	97
Total lipid, g	61	9	83	18

[a]Mean of all treatment groups.

Table 4. Effects of dietary energy and porcine somatotropin from d 75 to 105 of gestation on mammary parenchymal tissue[a]

Energy level (mcal/d)	6.1 (A)	10.5 (E)	6.1 (A)	10.5 (E)		Contrast[c]			
PST (mg/d)[b]	0	0	8	8					
	(n=10)	(n=9)	(n=9)	(n=9)	SEM	A0 vs E0	A0 vs A8	E0 vs E8	A8 vs E8
DNA									
µg/g	1,724	1,986	2,046	1,772	131.5		†		
mg	2,636	3,418	4,406	3,270	456.2		*		†
mg/teat	391	460	606	465	57.0		*		†
mg·teat^{-1}·kg BW^{-1}	2.20	2.40	3.24	2.41	.29		*		†
RNA									
µg/g	2,743	3,050	3,611	3,731	309.3		*		
mg	4,250	5,242	7,516	7,112	973.4		*	†	
mg/teat	620	700	1,037	991	120.3		*	†	
mg·teat^{-1}·kg BW^{-1}	3.57	3.64	5.58	5.11	.60		*	†	
RNA/DNA	1.66	1.56	1.81	2.22	.21			*	
Protein									
mg/g	66.68	81.62	82.03	76.30	4.29	*	*		
g	105	140	176	143	19.9		*		
g/teat	15.3	18.7	24.5	20.0	2.38		*		
g·teat^{-1}·kg BW^{-1}	.09	.10	.13	.10	.01		*		†
Lipid									
mg/g	417.2	350.7	281.0	342.1	29.48		*		
g	603	573	580	574	46.8				
g/teat	87.7	79.0	80.3	82.7	6.15				
g·teat^{-1}·kg BW^{-1}	.51	.41	.43	.43	.03	*	†		

[a]Based on right side teats which were associated with secretory tissue.
[b]Porcine somatotropin.
[c]†P < .10, *P < .05.

Table 5. Effects of dietary energy and porcine somatotropin from d 75 to 105 of gestation on mammary extraparenchymal stromal tissue[a]

Energy Level (mcal/d)	6.1 (A)	10.5 (E)	6.1 (A)	10.5 (E)		Contrast[c]			
PST (mg/d)[b]	0	0	8	8					
	(n=10)	(n=9)	(n=9)	(n=9)	SEM	A0 vs E0	A0 vs A8	E0 vs E8	A8 vs E8
DNA									
µg/g	147.7	148.7	191.9	190.1	26.63				
mg	86.3	96.9	123.9	125.8	14.91		†		
mg/teat	12.38	13.70	17.33	17.87	2.09		†		
mg·teat^{-1}·kg BW^{-1}	.07	.07	.09	.09	.01				
RNA									
µg/g	731.6	913.0	861.5	814.1	51.86	*	†		
mg	425.7	591.3	624.6	532.9	61.63	†	*		
mg/teat	61.85	84.31	86.94	75.98	8.97	†	*		
mg·teat^{-1}·kg BW^{-1}	.37	.44	.46	.40	.05				
RNA/DNA	5.24	6.25	5.67	4.40	.51			*	†
Protein									
mg/g	27.8	28.0	31.9	24.4	1.19		*	*	*
g	15.9	18.1	23.2	16.0	1.80		**	**	
g/teat	2.31	2.57	3.21	2.28	.25		*		*
g·teat^{-1}·kg BW^{-1}	.01	.01	.02	.01	.002				†
Lipid									
mg/g	674.5	626.9	471.6	581.6	35.70		*		*
g	391.6	401.4	326.9	377.2	34.26				
g/teat	57.21	57.57	45.24	54.23	5.38				
g·teat^{-1}·kg BW^{-1}	.34	.30	.24	.28	.03		*		

[a]Based on right side teats associated with secretory tissue.
[b]Porcine somatotropin.
[c]†P < .10, *P < .05, **P < .01.

CHAPTER 2. DIETARY SOYBEAN OIL DOES NOT INFLUENCE MAMMARY DEVELOPMENT OF GILTS WHEN FED FROM DAY 75 TO DAY 105 OF GESTATION

ABSTRACT

Twenty-seven pregnant gilts were used to determine if supplemental polyunsaturated fat fed during late gestation influences mammary development. On d 75 of gestation, gilts were randomly assigned to either a corn-soybean meal control diet with 0% added soybean oil or a corn-soybean meal diet containing 5% added soybean oil. Animals in both treatments received 6.1 Mcal ME, 244 g crude protein, and similar amounts of lysine, vitamins and minerals daily. On d 105 of gestation, gilts were slaughtered and total mastectomies were performed. Right-side mammary tissue was separated into parenchymal and extra-parenchymal stromal tissue. Parenchymal tissue was weighed and analyzed on a wet-tissue basis for DNA, RNA, water, protein and lipid. Students t-test was used to detect treatment differences. Parenchymal tissue water tended to be less ($P = .09$) for gilts fed 5% soybean oil. Parenchymal weight, DNA, RNA and protein were not altered ($P > .10$) by feeding 5% soybean oil. The number of fetuses ($P > .10$) and fetal weights ($P = .09$) were similar regardless of soybean oil level fed to the gilts. Dietary soybean oil did not affect mammary development of gilts when fed at 5% of the diet from d 75 to 105 of gestation.

Key Words: Swine, Mammary development, Soybean oil

Introduction

Maximum litter size and weaning weights are dependent on the sow's ability to secrete milk. Therefore, as producers strive to increase the number of pigs and pounds of pigs weaned per sow per year, there is increased pressure on the sow to produce more milk. To maximize milk production of the sow, feeding strategies must be employed which ensure optimal mammary gland development of the gilt.

The mammary gland consists of parenchymal tissue (milk secretory tissue) and extra-parenchymal tissue (primarily fat and connective tissue). Amounts of parenchymal deoxyribonucleic acid (DNA) and ribonucleic acid (RNA) can be used as indicators of mammary development and growth. The amount of DNA is used to assess milk secretory cell number and may be an indicator of subsequent milk production. The amount of RNA is an indicator of the functional state of mammary secretory cells. The RNA to DNA ratio is an indicator of the capacity of the cells to produce protein. Prior to d 50 of gestation, little change occurs in the DNA content of the mammary gland (Hacker and Hill, 1972). However, DNA concentrations increase 4-fold between d 75 and 90 of pregnancy (Kensinger et al., 1982). Therefore, late gestation appears to be the critical period of mammary development.

Mammary parenchymal growth is stimulated in mice and sheep by feeding diets containing high concentrations of polyunsaturated fats (McFadden et al., 1990; Welsch et al., 1985), whereas parenchymal growth is inhibited in mice and rats fed diets containing high concentrations of saturated fats (Wicha et al., 1979; Carrington and Hosick, 1985; Welsch, 1987). The effect of dietary polyunsaturated fat on mammary development in swine has not been reported.

Soybean oil (SBO) contains 79.5% unsaturated fatty acids of which 61.5% are polyunsaturated fatty acids (Maynard et al., 1979). It is commonly used by commercial swine producers at 1 to 5% of the diet to improve energy intake in hot weather and to reduce dust levels in swine facilities. In the present study, 5% soybean oil was added to the diet from d 75 to 105 of gestation to determine if polyunsaturated fat influences mammary development in gilts.

Materials and Methods

General Procedure. A completely randomized block design with 14 gilts per treatment was used. All gilts (initial average weight 155 ± 3 kg) were F1, crossbred Yorkshire x Landrace or at least half Yorkshire with a minimum of 12 functional teats. Relocation was used to stimulate and synchronize estrus among gilts. Gilts were bred by natural service on two consecutive days when they expressed estrus following the location change. Day 0 of gestation was considered to be the first day of mating. Gilts were housed in individual stalls (stall size = .6 m x 2.1 m) following d 2 of mating in an environmentally controlled (minimum temperature = 18.3^{O}C, 16 h light) building for the remainder of the trial and fed a fortified corn-soybean meal diet formulated to meet NRC (1988) requirements for all nutrients from d 0 to d 75 of gestation (Table 6, Control diet). Beginning on d 75 of gestation, gilts were assigned to experimental diets. Experimental diets contained either 0 or 5% soybean oil (SBO) and were formulated so that animals in both treatment groups received similar daily intakes of metabolizable energy, crude protein, lysine, vitamins and minerals (Table 6). One gilt was removed from the study because she was not pregnant.

Slaughter. Gilts were slaughtered on d 105 of gestation at the Michigan State University Meats Laboratory, under USDA supervision, according to standard operating procedures. Immediately after exsanguination, mammary glands, uterus, and fetuses were removed and weighed. Mammary glands were quickly frozen in a mixture of acetone and dry ice and stored at -20°C until analyzed. In addition, hot carcass weight, backfat and loin eye area at the 10th rib were obtained.

Laboratory Analyses. Mammary glands were dissected into parenchymal and extra-parenchymal stromal tissue (Weldon et al., 1991). Parenchymal DNA, RNA, and lipid content were determined according to methods described by Tucker (1964), and protein was determined by the method of Lowry et al. (1951).

Statistical Analysis. Analysis of variance using the general linear model of SAS (1985) was used to test the effect of dietary SBO on mammary development. Students t-test was used to detect mean differences.

Results and Discussion

The live weight of gilts at d 105 of gestation did not differ ($P > .33$) for gilts fed 0 or 5% SBO, 165 kg vs 170 kg; SEM = 4, respectively. Carcass weight (95 kg vs 98 kg; SEM = 2.1), loineye area (41.4 cm^2 vs 42.4 cm^2; SEM = 1.5) and 10th rib backfat (2.15 cm vs 2.14 cm; SEM = .19) likewise were not different ($P > .10$) for gilts fed 0 or 5% SBO, respectively. Regardless of dietary treatment, gilts had similar ($P = .09$) numbers of fetuses and fetal weights (Table 7). The similarity in gilt carcass composition and fetal numbers between treatments was not surprising because daily consumption of energy, lysine and protein were

also similar between treatments.

Parenchymal tissue weight, DNA, RNA and protein were not changed by feeding 5% SBO (Table 8). Parenchymal tissue water tended to be less ($P < .10$) for gilts fed 5% SBO. The lack of difference in parenchymal tissue weight, DNA, RNA and protein between gilts fed 0 or 5% SBO is not consistent with studies in mice (Welsch et al., 1985) and sheep (McFadden et al., 1990) which had increased parenchymal tissue weights and parenchymal DNA when fed polyunsaturated fat. However, this inconsistency may be related to species differences, the type of fat fed as well as differences in dietary energy intake that existed between experiments. It is possible that PUFA might increase mammary secretory tissue in gestating swine if the PUFA were fed at higher levels or was a greater percentage of the total energy intake than existed in the current study. Diets fed to mice contained corn oil which was added at the expense of carbohydrate (sucrose), resulting in differences in daily caloric intake between treatments (Welsch et al., 1985). Diets fed to sheep, containing sunflower oil, were isocaloric and isonitrogenous, but daily feed consumption differed among treatments, resulting in differences in energy intake (McFadden et al., 1990). It is also possible that SBO was not fed over a long enough period of time to elicit a response in mammary development. Carrington and Hosick (1985) proposed that increased deposition of unsaturated fatty acids may unmask hormone receptor sites through increased membrane fluidity, it is possible that the PUFA in this study were not fed long enough for this to occur. Feeding PUFA over a longer period of time may also result in higher concentrations of lipid-derived stimulatory factors, such as prostaglandins, which may also influence mammogenesis (Welsch, 1987).

Implications

Supplemental dietary polyunsaturated fat (soybean oil) does not affect mammary development of gilts when fed at 5% of the diet from d 75 to 105 of gestation. Thus, using 5% dietary soybean oil to improve environmental conditions in swine facilities or increase dietary energy should not impair or improve subsequent milk production.

Table 6. Composition and calculated analysis of experimental diets

Ingredient, %	Control	Soybean oil
Corn	82.80	74.40
Soybean meal (44%)	12.65	15.85
Mono-dicalcium phosphate	1.85	2.00
Ground limestone	1.30	1.35
Vit-TM premix[a]	.60	.60
Vit E-Se premix[b]	.30	.30
Salt	.50	.50
Soybean oil	.00	5.00
Calculated analysis		
ME, Mcal/kg	3.2	3.4
Crude protein, %	12.6	13.3
Lysine, %	.56	.64
Calcium, %	.88	.93
Phosphorus, %	.70	.73
Daily intake		
Feed, kg	1.97	1.86
ME, Mcal	6.1	6.1
Crude protein, g	245	244
Lysine, g	10.95	11.65

[a]Composition per kg premix: vitamin A, 661,380 IU; vitamin D, 132,276 IU; menadione, .66 g; riboflavin, .66 g; niacin, 3.53 g; d-pantothenic acid, 2.64 g; choline chloride, 88.18 g; vitamin B_{12}, 3.96 mg; zinc, 7.5 g; iodine, .11 g; copper, 2 g; iron, 12 g.

[b]Composition per kg premix: vitamin E, 3,310 IU; selenium, 19.8 mg.

Table 7. Effect of soybean oil on fetal and gilt mammary characteristics

Item	Soybean oil, % 0 (n = 14)	5 (n = 13)	SEM	*P* value
No. of fetuses	9.40	9.90	.55	.54
Fetus wt, kg	1.08	1.00	.03	.09
No. of mummies	.08	.14	.09	.60
No. of teats				
Right	6.85	6.79	.18	.81
Left	7.00	7.14	.15	.51
Total udder wt, g	5859	5483	326	.41
Parenchymal wt, g[a]	1306	1237	93	.93
Extraparenchymal, g[b]	609	679	47	.30
Utero placental full wt[c]	19.2	18.5	.97	.58
Utero placental empty wt[d]	10.1	9.7	.5	.63

[a]Based on right side mammary glands, weight of secretory tissue minus skin, muscle and lymph nodes.

[b]Based on right side mammary glands, weight of nonsecretory tissue minus skin, muscle and lymph nodes.

[c]Weight including fetuses and fluid.

[d]Weight minus fetuses and fluid.

Table 8. Composition of mammary parenchymal tissue in gestating gilts fed diets with soybean oil[a]

Item	Soybean oil, % 0 (n = 14)	5 (n = 13)	SEM	*P* value
Wt, g	1306	1237	93	.59
Fat, %	28.9	32.4	1.7	.16
Water, %	59.1	55.7	1.4	.09
Protein, mg/g	81.5	79.3	3.4	.64
DNA				
µg/g	2084	2060	152	.91
mg	2594	2485	168	.64
mg/teat	373	350	26	.52
RNA				
µg/g	3281	3438	236	.64
mg	4251	4187	376	.90
mg/teat	610	591	55	.80
RNA/DNA	1.6	1.7	.12	.84

[a]Based on right side teats associated with secretory tissue.

CHAPTER 3. NUTRITION, BODY TISSUE ACCRETION AND MAMMARY DEVELOPMENT IN PREGNANT GILTS

ABSTRACT

Thirty-four pregnant Hampshire x (Yorkshire x Landrace) gilts were used to determine if dietary energy intake and body composition changes in late gestation (d 75 to 105) were related to mammary development. Gilts were provided one of two energy intakes (6.1 [Adequate] or 10.5 [Elevated] Mcal ME/d) over two periods (d 0 to 74 and d 75 to 105 of gestation). Two periods were used to achieve body compositional differences at d 75 and d 105, and differences in body tissue accretion rates between d 75 and d 105 of gestation. Empty body composition was estimated using BW, day of gestation and deuterium oxide space as predictor variables in equations developed for this experiment. On d 0, 23, 40, 75, and 105 of gestation, gilts were slaughtered and total mastectomies were performed. Right-side mammary tissue was separated into parenchymal and extra-parenchymal stromal tissue. Parenchymal tissue was weighed and analyzed for total DNA, RNA protein and lipid. Analysis of variance was carried out using the GLM procedure of SAS to determine the effect of dietary energy level from d 0 to 74 of gestation and from d 75 to 105 of gestation on mammary development. Parenchymal tissue DNA (3428 mg vs 3199 mg; SEM = 195), RNA (7494 mg vs 6478 mg; SEM = 479) and protein (132 g vs 116 g; SEM = 13.2) on d 105 of gestation for gilts fed adequate and elevated energy, respectively, were not related ($P > .10$) to dietary energy level fed regardless of the gestational period that it was fed. Parenchymal lipid (389 g vs 507 g; SEM = 29.6) was increased ($P < .001$) at d 105 of gestation when gilts were fed elevated energy diets from d 0 to d 74 of gestation regardless

of the level of energy fed from d 75 to d 105 of gestation. Empty body fat from the four treatment groups ranged from 40.7 to 47.9 kg and 44.4 to 52.6 kg on d 75 and 105, respectively. Empty body protein from the four treatment groups ranged from 23.7 to 26.4 kg and 28.3 to 30.9 kg on d 75 and 105, respectively. Between d 75 and 105, gilts gained 3.16 to 7.74 kg of fat and 3.25 to 5.70 kg of protein. Regression analysis was used to evaluate the relationship between body composition changes and mammary development. DNA and RNA on d 105 were not related ($P > .10$) to empty body fat or protein on d 75 or 105, or to changes in empty body fat or protein from d 75 to 105. In conclusion, elevated dietary energy intake from d 0 to d 74 of gestation or from d 75 to 105 of gestation did not affect mammary secretory tissue growth. The gilts ability to milk in her first lactation was not related to body compositional changes occurring in late gestation.

Key Words: Swine, Gilt, Mammary development, Body composition

Introduction

The goal to increase sow productivity continues to be a high priority in commercial swine production. To increase the number of pigs weaned per sow per year and litter weaning weights, the sow must produce more milk. The association between nutrition and growth of milk secretory tissue during gestation is not well understood and must be, in order to maximize milk production in the sow.

Parenchymal deoxyribonucleic acid (DNA)is an indicator of mammary secretory cell number. Ribonucleic acid (RNA) is a reflection of the functional state of the cell, and the RNA/DNA ratio is an indicator of protein synthesis activity per cell in the mammary gland. Little change

occurs in the amount of DNA in the mammary gland prior to d 50 of gestation (Hacker and Hill, 1972). Mammary DNA concentrations increase fourfold between d 75 and 90 of gestation (Kensinger et al., 1982). Excessive dietary energy intake (> 10.0 Mcal ME/d) in late gestation was shown to be detrimental to development of mammary secretory tissue in an experiment by Weldon et al. (1991). In a subsequent study however, there was no detrimental effect on mammary development when gilts were fed excessive energy in late gestation (Howard et al., 1995 companion paper see Chapter 1).

In experiments conducted by Weldon et al. (1991) and Howard et al., (1995 companion paper see Chapter 1), gilts were housed in similar environmental conditions, provided with similar nutrient intakes daily and selected from a similar genetic pool of animals. However, in the second study gilts were heavier on d 75 of gestation and subsequently may have been fatter than gilts in the first study. It is possible that differences in body composition among gilts when entering late gestation influenced the mammary growth response to excessive dietary energy intake. Our objective was to determine if dietary energy intake or body composition changes during gestation influence total mammary secretory tissue development in the gilt.

Materials and Methods

Animals. Forty Hampshire x (Yorkshire x Landrace) gilts (average weight 117 kg, average backfat depth 20.6 mm) with a minimum of 12 functional teats were selected initially. Gilts were checked for estrus two times per day and bred naturally on their second estrus using year-old Hampshire boars. Matings were permitted every 12 h for a maximum of

three times. The first day of mating was considered d 0 of gestation. Following breeding, gilts were moved into an environmentally controlled (minimum temperature = 18.3° C, 16 h light) building and housed individually (stall size = .6 m x 2.1 m). Gilts were of similar ages (average 218 d ± 12 d) to remove any possible age effect. Six gilts were removed from the study because they were not pregnant; therefore, 34 gilts were eventually used.

Treatments. A randomized design was used with a 2 x 2 factorial arrangement of treatments. Ten gilts were assigned to each treatment initially. The main effects included gestational period (d 0 to d 74 [P1] or d 75 to d 105 [P2]) and dietary energy level, either adequate energy (6.1 Mcal ME/d, [A]) or elevated energy (10.5 Mcal ME/d,[E]). Dietary energy level was altered by changing concentrations of corn and soybean meal, adding cornstarch to the elevated energy diet and changing the daily allotment of feed per gilt (Table 9). Daily intake of supplemental vitamins, minerals and lysine were held constant across all treatments and met NRC requirements (1988).

Body Composition. Amounts of body fat and protein on d 75, d 105 and changes occurring from d 75 to d 105 of gestation was varied by providing adequate or elevated amounts of dietary energy from d 1 to d 74 and d 75 to d 105 of gestation. Empty body composition of all gilts at d 75 and 105 of gestation was estimated using the deuterium oxide (D_2O) dilution method as described by Shields et al. (1984) and Rozeboom et al. (1994). Prediction equations (Table 10) were developed specifically for this population of gilts. Animals were slaughtered and chemically analyzed at various stages of gestation, d 0 (n = 7), d 23 (n = 3), d 40 (n = 2), d 75 (n = 6) and d 105 (n = 7). Carcass, viscera,

uterus with fetuses and blood samples were analyzed in triplicate for moisture (oven drying), ether extract (Soxhlet apparatus), protein (Kjeldahl method, AOAC, 1980) and ash (AOAC, 1980). Appropriate procedures (Rozeboom et al., 1993) were followed to collect measurements used in body composition determinations.

Body composition equations were developed on both an empty body and maternal body basis. Empty BW was calculated by subtracting digesta from live weight (Shields et al., 1984 and Rozeboom et al., 1994). Maternal BW was calculated by subtracting digesta and uterus with fetus from live weight (Shields et al., 1984).

Mammary Growth. To assess mammary growth gilts were slaughtered on d 0 (n = 10), d 23 (n = 3), d 40 (n = 3), d 75 (n = 7) and d 105 (n = 34) of gestation at the Michigan State Meats Laboratory, under USDA supervision, according to standard operating procedures. All of the gilts slaughtered for body composition prediction were also used to evaluate mammary development. Immediately after exsanguination, mammary glands, uterus and fetuses were removed and weighed. Right side mammary glands were quickly frozen in a mixture of acetone and dry ice and stored at $^{-}20^{o}C$ until analyzed. The number of corpora lutea were counted and recorded for both ovaries.

Right side mammary glands were dissected into parenchymal and extraparenchymal stromal tissue (Weldon et al., 1991). Parenchymal DNA, RNA and lipid were determined according to Tucker (1964), and protein was determined by the method of Lowry et al. (1951) for gilts slaughtered at d 105 of gestation. Prior to d 105 of gestation the right side mammary glands were only analyzed for parenchymal weight,

parenchymal protein, parenchymal lipid, parenchymal water and extraparenchymal weight.

Constituents of mammary tissue were expressed on four different bases in the tables: 1) concentration, 2) total, 3) corrected for the number of teats associated with secretory tissue and 4) corrected for the number of teats associated with secretory tissue and kg maternal BW (live weight minus fetal weight).

Statistical Analysis. Analysis of variance was carried out using the general linear model procedure of SAS (1986) to determine the effect of dietary energy level and the gestational period in which the dietary energy level was fed on mammary development. The interaction between dietary energy level and gestational period was included in the model. Pair-wise t-tests were used to detect differences between treatments.

Linear regression models predicting empty and maternal body components using all combinations of independent variables (D_2O space, BW, day of gestation, and backfat depth) were fitted using SAS (1986) procedures. Selection of variables for inclusion in prediction models followed criteria described by Rozeboom et al., 1994.

Regression analysis (SAS, 1986) was used to determine if mammary parenchymal DNA and RNA on d 105 of gestation were related to changes in body composition occurring during late gestation.

Results and Discussion

Body Composition Prediction Equations. Equations for estimating body composition for this population of gilts were developed with good prediction accuracy. Rozeboom et al. (1994) found EBPRO and EBFAT had R^2 values of .99 and .96 for Yorkshire x Landrace gilts and .87 and .97

for Duroc x (York x Landrace) gilts, respectively. The equations developed by Shields et al. (1984) had R^2 values of .35 and .86 for EBPRO and EBFAT, respectively. The equation developed in this study for empty body protein (R^2 = .66) was not as powerful as that developed by Rozeboom et al. (1994) but more powerful than that of Shields et al. (1984). Furthermore, the equation developed in this study for empty body fat (R^2 = .95) was as powerful as that developed by Rozeboom et al. (1994) and more powerful than that of Shields et al. (1984).

Growth Performance and Body Composition. During P1 gilts fed elevated energy compared with gilts fed adequate energy were heavier, had more backfat, EBFAT ($P < .01$) on d 75 and on d 105 of gestation and EBPRO ($P < .10$) on d 75 of gestation (Table 11). Gilts provided with elevated as compared with adequate dietary energy intake during P2 regardless of dietary energy intake during P1 were heavier, had more EBFAT ($P < .01$) and tended ($P < .10$) to have more EBPRO on d 105 of gestation. Interactions did not exist between period and dietary energy level on d 105 of gestation for gilt BW, backfat or empty body composition ($P > .10$), therefore the response to energy intake in P2 was not dependent on energy intake in P1.

Change in EBFAT from d 75 to 105 of gestation was influenced ($P < .01$) by energy intake from d 0 to 74 of gestation. More specifically, change in EBFAT between d 75 and 105 of gestation was less ($P < .05$) for gilts with elevated dietary energy intake during P1 and greater ($P < .01$) for gilts fed elevated energy diets during P2. EBFAT at d 105 of gestation increased 214% for gilts fed adequate energy during P1 (7.18 kg vs 3.36 kg) but only 143% for gilts fed elevated energy during P1 (4.76 kg vs 3.33 kg) when gilts were fed elevated energy diets during

P2. Change in EBFAT from d 75 to 105 of gestation was similar for gilts fed adequate energy diets during P2 regardless of diet fed during P1 (3.36 kg vs 3.33 kg). There are a couple of reasons why these results were observed. First, the gilts provided with adequate energy during P1 were lighter (157 kg vs 181 kg) at d 75 of gestation than gilts provided with elevated energy. Thus, these gilts would require less maintenance energy and may have also utilized energy more efficiently in P2. Subsequently, when both groups were switched to elevated energy diets during P2, the gilts fed adequate energy during P1 had greater fat accretion rates during P2 compared with gilts provided with elevated energy intake during P1. Secondly, since the majority of fetal growth occurs during late gestation, more energy would be utilized for fetal growth as compared with maternal deposition during late gestation.

Reproductive measurements. Gilts fed elevated energy diets during P1 had a greater ($P < .05$) number of fetuses compared to gilts fed adequate energy diets during P1 (Table 12). Results from a previous study indicate that conspectuses may stimulate mammary development (Kensinger et al., 1986) if differences are extreme (≤ 4 versus ≥ 11). The difference among treatment groups in the present study was only 2 pigs. Thus, fetal number differences in the present study are not thought to have affected overall mammary growth. Fetal weight was similar across treatments. The numbers of corpora lutea did not differ among treatments; consequently, percent survival was higher ($P < .05$) in gilts fed elevated energy compared to gilts fed adequate energy during P1.

Mammary development. Mammary development at d 0, d 23, d 40 and d 75 of gestation was characterized by evaluating mammary glands from gilts slaughtered for development of body composition prediction equations.

The mammary glands were analyzed only for amount of extraparenchymal tissue, parenchymal tissue, parenchymal protein, parenchymal fat and parenchymal water. Parenchymal tissue weight did not change from d 0 to d 40 of gestation, but it increased ($P < .01$) 63 percent by d 75 of gestation (Table 13). Similarly, total parenchymal tissue water and protein were greater ($P < .01$) at d 75 of gestation than at d 0, d 23 or d 40 of gestation whereas total parenchymal fat did not change between d 0 and d 75 of gestation. Parenchymal DNA and RNA at d 75 were not measured in this study. However, a positive relationship at d 105 of gestation was identified between parenchymal DNA and RNA and parenchymal weight ($R^2 = .81; .80$) and protein ($R^2 = .78; .83$) shown in Table 14. Thus, based on the assumption that the same relationship occurs at d 75 of gestation between parenchymal DNA and RNA and parenchymal protein and parenchymal weight, it is likely that parenchymal DNA and RNA were also increased at d 75 of gestation. Hacker and Hill, (1972) found minimal changes in the amount of DNA and RNA in the gilt mammary gland during the first 50 days of pregnancy, whereas between the 50th and 100th days of pregnancy the amount of parenchymal tissue, parenchymal DNA and parenchymal RNA were increased dramatically. Kensinger and coworkers (1982) found that although concentration of mammary DNA numerically increased from d 0 to 30 and d 45 of gestation, the change was not statistically significant. However, between d 75 and 90 of gestation mammary DNA increased significantly. Ribonucleic acid continued to increase from d 90 of gestation through d 4 of lactation. The previous studies support the findings in this study that there is minimal growth of the mammary gland prior to d 75 of gestation.

Mammary parenchymal weight and protein increased by d 75 of gestation; however, the magnitude of increase was small in comparison to the increase in parenchymal weight (68%) and protein (626%) by d 105 of gestation. The increase in mammary growth from d 75 to 105 of gestation is in agreement with Weldon et al. (1991) and Howard et al. (1992) who found that the majority of mammary growth occurs between d 75 and 105 of gestation.

Since the majority of mammary secretory tissue growth during gestation occurs between d 75 and d 105, it is not surprising that feeding elevated energy from d 0 to d 74 of gestation does not influence the amount of parenchymal tissue or parenchymal tissue composition on d 75 of gestation. Feeding elevated versus adequate dietary energy to gilts from d 0 to 75 of gestation increased ($P < .01$) extraparenchymal tissue weight (814 g vs 367 g; SEM = 54) without affecting ($P > .10$) parenchymal tissue weight (698 g vs 591 g; SEM = 64), parenchymal tissue water (225 g vs 237 g; SEM = 28), parenchymal tissue fat (433 g vs 321 g; SEM = 50) or parenchymal tissue protein (35.8 g vs 31.7 g; SEM = 3.6) on d 75 of gestation.

Gilts were provided with adequate or elevated dietary energy intake over two periods to determine the influence of energy intake during late gestation on mammary growth at d 105 of gestation. The amount of extraparenchymal tissue and parenchymal fat were increased ($P < .01$) in gilts provided with elevated energy as compared with gilts provided with adequate energy intakes during P1 (Table 15). However, the amount of parenchymal tissue, extraparenchymal tissue, parenchymal fat and parenchymal protein were not different ($P > .10$) among gilts provided with elevated energy intake compared with gilts provided with adequate

energy intakes during P2 (Table 15). Total parenchymal DNA and RNA did not differ with dietary energy level fed, regardless of the gestational period in which it was fed (Table 16). Therefore, gilts provided with excessive energy intake (10.5 Mcal/d) from d 0 to d 74 or d 75 to d 105 of gestation had similar mammary secretory tissue growth as gilts provided with adequate energy intake (6.1 Mcal/d) during the same periods. These findings contradict those of Weldon et al. (1991) who found that gilts fed elevated energy diets from d 75 to 105 of gestation had decreased parenchymal DNA and RNA compared with gilts fed adequate energy diets. However, Weldon et al. (1991) corrected parenchymal DNA and RNA results for the number of functional glands (teats associated with secretory tissue) and per kg maternal BW. Since dietary energy influences both BW and mammary growth, BW is confounded with treatment. In addition, the number of teats in the present study and that of Weldon et al. (1991) were controlled within experiment (12 to 14 per gilt), so a statistical correction or adjustment is not necessary. Therefore presentation and interpretation of mammary growth data on a total basis is believed to be the most valid. When we expressed the data of Weldon (1988) on a total basis, the amounts of parenchymal DNA and RNA were not influenced by dietary energy level in late gestation ($P > .10$). These results are now in agreement with similar data collected in the present study.

Similar to swine, Lacrasse et al. (1993) found that plane of nutrition during gestation did not influence milk production in heifers. Likewise, work with prepubertal heifers by Radcliff et al. (1995), has shown that feeding excess energy and protein does not influence parenchymal DNA, RNA or RNA/DNA ratios. In contrast, however ad libitum

as compared with restricted feeding during the prepubertal period has been shown to be detrimental to mammary growth in sheep and cattle (Sejrsen et al., 1982; Johnsson and Hart, 1985). Thus, the influence of nutrition on mammary growth in sheep and cattle is still thought to have an impact during the prepubertal period on total mammary secretory cell number.

Parenchymal DNA and RNA at d 105 of gestation were not related (R^2 < .07, P > .10) to EBFAT or EBPRO at d 75 or 105 of gestation or empty body weight at d 105 of gestation. Also, changes in EBFAT or EBPRO from d 75 to 105 of gestation were not related (R^2 < .04, P > .33) to parenchymal DNA or RNA at d 105 of gestation. Thus, fat and protein accretion in gilts from d 75 to 105 of gestation has no detrimental affect on potential milk yield of the sow. These results contradict those of Head and Williams (1991) who found that DNA concentrations and estimated cell numbers were decreased at d 112 of gestation for fat versus lean gilts of the same BW. The discrepancies between studies may be attributed to differences in total body fat. Head et al. (1991) reported backfat thickness of 36 mm and 25 mm, for fat and lean gilts, respectively. Gilts in this study had backfat thickness of 31 mm and 25 mm for gilts fed elevated and adequate energy diets from d 0 to d 74 of gestation, respectively. In our study feed intakes by gilts on elevated energy treatments were maximized therefore it would have been difficult to achieve greater amounts of backfat and total body fat. Yet, in the studies conducted by Head et al. (1991) there may have been greater amounts of body fat which may have had a greater influence on mammary secretory tissue growth.

Implications

Feeding excessive energy during gestation does not influence mammary secretory tissue growth. Likewise, there seems to be no relationship between mammary development and body fat or protein accretion rates between d 75 and 105 of gestation. The period when the majority of mammary growth occurs in the pig. Thus, feeding excessive energy to primiparous females during gestation should not impair milk production.

Table 9. Composition and calculated analysis of experimental diets

Ingredient, %	Adequate energy	Elevated energy
Corn	85.05	71.30
Soybean meal (44%)	10.22	2.00
Mono-dicalcium phosphate	1.88	.93
Ground limestone	1.35	.90
MSU Vit-TM premix[a]	.60	.48
Vit E-Se premix [b]	.50	.48
Salt	.40	.25
Cornstarch		23.75
L-Lysine HCL		.09
Calculated Analysis		
ME, Mcal/kg	3.2	3.27
Crude protein, %	11.7	6.9
Lysine, %	.5	.3
Calcium, %	.9	.53
Phosphorus, %	.7	.41
Daily Intake		
Feed, kg	1.94	3.27
ME, Mcal	6.1	10.5
Crude protein, g	228	228
Lysine, g	9.71	9.81

[a]Concentration per kg premix: vitamin A, 661,380 IU; vitamin D, 132,276 IU; menadione, .66 g; riboflavin, .66 g; niacin, 3.53 g; d-pantothenic acid, 2.64 g; choline chloride, 88.18 g; vitamin B_{12}, 3.96 mg; zinc, 7.5 g; iodine, .11 g; copper, 2 g; iron, 12 g.

[b]Concentration per kg premix: vitamin E, 3310 IU; selenium, 19.8 mg.

Table 10. Prediction equations developed to estimate body composition in gravid and nongravid gilts

Item	b_0	Regression coefficients (bi)[a]			Mean square		R^2	PRESS[b]	Mean	PE[c]
		D_2O space	Live wt	Day	Model	Error				
Empty body[d]										
Weight	5.9 ± 5.0		.461 ± .030		6,068	26.43	.91	727.3	80.3	5.39
Ash	-1.67 ± 1.22	.072 ± .013			37.6	1.144	.59	30.66	5.21	1.11
Water	-.99 ± 5.63	.370 ± .170	.286 ± .085		3,088	22.74	.93	639.1	80.3	5.06
Protein	-2.8 ± 4.3	.297 ± .045			633	14.48	.66	391.7	25.4	3.96
Fat	-.27 ± 2.4		.279 ± .017	-.036 ± .014	887	4.31	.95	120.9	42.8	2.20
Maternal body[e]										
Ash	2.80 ± 1.11	.011 ± .011			.95	.944	.04	25.24	3.89	1.00
Water	30.4 ± 5.4		.250 ± .033		1,788	30.86	.72	837.5	70.7	5.79
Protein	14.7 ± 4.6	.059 ± .057		-.021 ± .023	5.23	9.42	.05	259.0	19.1	3.22
Fat	2.01 ± 2.82		.262 ± .021	-.053 ± .016	690	6.04	.91	171.4	41.4	2.62

[a]Model was $Y = b_0 + b_1x_1 + \ldots + b_px_p + e$, where Y was weight of chemical component (kilograms) and coefficients (b_i; least squares estimate ± standard error) are of variables D_2O space [kg], live weight [kg] and day of gestation, as indicated.

[b]Predicted residual sum of squares.

[c]Average prediction error of mean = (predicted residual sum of squares/n)$^{1/2}$.

[d]Empty body consisted of live weight minus ingesta.

[e]Maternal body consisted of live weight minus ingesta, uterus and products of conception.

Table 11. Body weights, backfat and empty body composition for gilts fed adequate or elevated energy over two feeding periods (d 0 to 74 and d 75 to 105 of gestation)

	Energy Intake[a]					Energy Effect		
Item	A:A (n = 10)	A:E (n = 8)	E:A (n = 8)	E:E (n = 8)	MSE	P1	P2	P1*P2
Body wt, kg								
d 1	119	117	118	114	76.43	.53	-	-
d 30	134	132	149	143	70.42	.0001	-	-
d 75	156	157	181	181	97.05	.0001	-	-
d 105	174	188	197	203	93.42	.0001	.003	.23
Δ 75-105, kg	17.3	31.5	15.2	20.7	13.57	.0001	.0001	.002
Backfat, mm								
d 1	21.0	20.6	20.3	20.6	8.196	.74	-	-
d 30	21.6	20.3	24.4	22.4	9.689	.03	-	-
d 75	23.0	22.1	29.9	27.2	9.335	.0001	-	-
d 105	23.7	26.1	31.3	30.1	13.126	.0001	.57	.18
Δ 75-105, mm	.4	4.0	1.4	2.9	7.741	.89	.0002	.12
Carcass wt, kg	98	102	104	107	168.04	.20	.44	.89
d 75								
EBFAT,kg	40.7	40.8	47.7	47.9	7.56	.0001	-	-
EBPRO, kg	25.1	23.7	25.7	26.4	6.37	.08	-	-
EBWAT, kg	78.5	77.0	86.4	87.6	32.35	.0001	-	-
EBASH,kg	5.14	4.81	5.29	5.47	.378	.08	-	-
d 105								
EBFAT,kg	44.1	48.0	51.0	52.7	8.33	.0001	.007	.26
EBPRO, kg	27.8	30.4	29.8	32.0	14.968	.16	.09	.86
EBWAT, kg	86.6	93.7	95.8	100.7	50.388	.002	.02	.65
EBASH,kg	5.86	6.18	6.28	7.13	.976	.04	.10	.43
d 75 to 105 Δ								
EBFAT,kg	3.36	7.18	3.33	4.76	1.417	.02	.0001	.007
EBPRO, kg	2.7	6.7	4.2	5.5	8.896	.74	.01	.22
EBWAT, kg	8.1	16.7	9.4	13.1	20.747	.66	.0003	.13
EBASH,kg	.72	1.36	.99	1.66	.876	.33	.05	.96

[a]Energy intake during the first period (P1):energy intake during the second period (P2); A = adequate energy (6.1 Mcal ME/d), E = elevated energy (10.5 Mcal ME/d).

Table 12. Reproductive measurements for gilts fed adequate or elevated energy over two feeding periods (d 0 to 74 and d 75 to 105 of gestation)

Item	Energy Intake[a] A:A (n = 10)	A:E (n = 8)	E:A (n =8)	E:E (n = 8)	MSE	Energy Effect P1	P2	P1*P2
No. of fetuses	8.0	8.0	8.0	10.1	3.279	.07	.16	.14
Fetus wt, kg	1.0	1.1	1.1	1.1	.028	.20	.41	.32
No. of mummified fetuses	.3	.0	.1	.1	.128	.74	.21	.23
Corpora Lutea	12.4	11.0	10.3	11.1	8.359	.28	.75	.26
Survival, %	68.5	75.9	82.6	91.7	329.88	.02	.20	.90
Udder wt, g	5,554	6,208	7,241	6,642	1,266,856	.01	.88	.12
No. of teats[b]	7.0	6.8	7.0	7.0	.250	.52	.45	.47
Uterine Complex uterine tissue, fetus & fluid, kg	16.4	18.9	18.5	22.7	4.777	.07	.05	.61
uterine tissue, kg	5.6	5.9	5.8	6.9	1.684	.17	.14	.40
uterine fluid, kg	2.8	3.9	3.5	4.6	1.666	.09	.02	.95

[a]Energy intake during the first period (P1):energy intake during the second period (P2); A = adequate energy (6.1 Mcal ME/d), E = elevated energy (10.5 Mcal ME/d).

[b]No. of right side teats associated with secretory tissue.

Table 13. Extraparenchymal and parenchymal tissue constituents of gilts at various stages of gestation[a]

Item	Day of gestation 0 (n = 10)	23 (n = 3)	40 (n = 3)	75 (n = 7)	MSE	P
Extraparenchyma[b]						
g	443	334	378	623	49,719	NS
g/teat[c]	62.8	46.7	52.3	94.6	1,173	NS
g·teat^{-1}·kg BW^{-1}d	.462	.387	.367	.544	.0390	NS
Parenchyma[f]						
g	401^{f}	430^{f}	473^{f}	652^{g}	19,724	.004
g/teat[c]	56.5^{f}	58.7^{f}	64.7^{f}	97.6^{g}	449	.09
g·teat^{-1}·kg BW^{-1}d	.403^{f}	.487^{f}	.457^{f}	.580^{g}	.0094	.001
Parenchymal water						
percent	27.0^{f}	29.8^{fg}	27.8^{fg}	35.6^{g}	32.35	.004
g	112^{f}	128^{f}	128^{f}	230^{g}	2,122	.004
g/teat[c]	15.7^{f}	17.4^{f}	17.5^{f}	34.4^{g}	44.32	.001
g·teat^{-1}·kg BW^{-1}d	.111^{f}	.145^{f}	.124^{f}	.207^{g}	.0018	.05
Parenchymal fat						
percent	68.5^{f}	64.7^{fg}	67.8^{f}	58.7^{g}	40.88	.004
g	271	278	325	385	10,293	NS
g/teat[c]	38.2^{f}	37.8^{f}	44.1^{fg}	57.9^{g}	236.5	.07
g·teat^{-1}·kg BW^{-1}d	.273	.315	.311	.341	.0047	NS
Parenchymal protein						
percent	4.09^{f}	4.50^{fg}	4.33^{fg}	5.26^{g}	.492	.002
g	16.8^{f}	19.1^{f}	20.0^{f}	34.0^{g}	45.19	.006
g/teat[c]	2.3^{f}	2.6^{f}	2.7^{f}	5.1^{g}	.99	003
g·teat^{-1}·kg BW^{-1}d	.017^{f}	.022^{f}	.019^{f}	.031^{g}	.00003	.04

[a]Based on right side mammary glands.
[b]Weight of non-secretory tissue less skin, muscle and lymph nodes.
[c]Mammary data are expressed per functional teat (teats associated with secretory tissue).
[d]Mammary data are expressed per functional teat (teats associated with secretory tissue) per kg maternal BW.
[e]Weight of secretory tissue less skin, muscle and lymph nodes.
[fg]Different superscripts within rows differ by the *P* value indicated in that row.

Table 14. Regression analysis of parenchymal DNA and RNA on parenchymal constituents and extraparenchymal percentage at d 105 of gestation[ab]

Item	b_o			b_1			R^2	P
DNA, mg								
Protein, g	1,046	±	230.7	18.07	±	1.77	.77	.0001
Water, g	682	±	181.2	3.84	±	.255	.88	.0001
Fat, g	1,517	±	512.7	4.0	±	1.13	.29	.001
Parenchyma, g	241	±	237.5	2.4	±	.19	.85	.0001
Extra-parenchyma, %	4,837	±	502.1	-4,082	±	1,308.2	.23	.004
RNA, mg								
Protein, g	1,069	±	508.3	47.19	±	3.89	.82	.0001
Water, g	266	±	411	9.80	±	.58	.90	.0001
Fat, g	3,414	±	1,387.6	8.0	±	3.05	.18	.01
Parenchyma, g	-530	±	687.7	5.9	±	.53	.80	.0001
Extra-parenchyma, %	10,447	±	1,302.3	-9,266	±	3,393	.19	.01
mg DNA·teat^{-1}·kg BW^{-1}[c]								
Protein, g·teat^{-1}·kg BW^{-1}[c]	.949	±	.186	.017	±	.002	.78	.0001
Water, g·teat^{-1}·kg BW^{-1}[c]	.55	±	.18	.004	±	.0003	.85	.0001
Fat, g·teat^{-1}·kg BW^{-1}[c]	1.33	±	.585	.004	±	.0015	.17	.03
Parenchyma, g·teat^{-1}·kg BW^{-1}[c]	.010	±	.2431	2.60	±	.220	.81	.0001
Extra-parenchyma, %	4.31	±	.397	-4.0	±	1.03	.31	.0006
mg RNA·teat^{-1}·kg BW^{-1}[c]								
Protein, g·teat^{-1}·kg BW^{-1}[c]	1.10	±	.409	.05	±	.004	.83	.0001
Water, g·teat^{-1}·kg BW^{-1}[c]	.134	±	.379	.01	±	.0006	.89	.0001
Fat, g·teat^{-1}·kg BW^{-1}[c]	3.01	±	1.53	.008	±	.004	.11	.06
Parenchyma, g·teat^{-1}·kg BW^{-1}[c]	-1.05	±	.628	6.5	±	.57	.80	.0001
Extra-parenchyma, %	9.3	±	1.04	-8.9	±	2.71	.25	.002

[a]Model was $Y = b_o + b_1x_1 + e$, where Y was mammary DNA and RNA variables and coefficients (b_1; least squares estimate + standard error) are of mammary parenchymal protein, parenchymal water, parenchymal fat, parenchymal tissue and extra-parenchymal tissue.

[b]Based on right side mammary gland.

[c]Mammary data are expressed per functional teat (teats associated with secretory tissue) per kg maternal BW.

Table 15. Extraparenchymal and parenchymal tissue constituents at d 105 of gestation for gilts fed adequate or elevated energy over two feeding periods (d 0 to 74 and d 75 to 105 of gestation)[a]

Item	Energy Intake[b] A:A (n = 10)	A:E (n = 8)	E:A (n = 8)	E:E (n = 8)	MSE	Energy Effect P1	P2	P1*P2
Extraparenchyma[c]								
g	543	753	936	907	52,823	.001	.23	.14
g/teat[d]	78	112	134	131	1,205	.003	.17	.13
g·teat^{-1}·kg BW^{-1}[e]	.51	.67	.75	.72	.040	.03	.32	.17
Parenchyma								
g	1,219	1,161	1,481	1,307	82,927	.10	.41	.79
g/teat[d]	174	171	204	186	1,383	.10	.44	.56
g·teat^{-1}·kg BW^{-1}[e]	1.13	1.02	1.15	1.02	.050	.96	.14	.90
Parenchymal water								
%	56.5	53.5	53.5	49.7	15.451	.01	.02	.75
g	695	627	763	656	35,415	.50	.19	.76
g/teat[d]	99	93	110	93	614.8	.56	.19	.57
g·teat^{-1}·kg BW^{-1}[e]	642	551	617	513	22,069	.48	.07	.90
Parenchymal fat								
%	31.6	35.2	34.9	40.4	25.89	.01	.02	.67
g	376	402	496	519	7,017	.0002	.40	.96
g/teat[d]	53.8	59.4	71	74	115.23	.0001	.25	.73
g·teat^{-1}·kg BW^{-1}[e]	349	354	401	407	4,130	.02	.81	.96
Parenchymal protein								
%	10.6	9.9	9.7	8.7	1.691	.02	.07	.82
g	132	116	138	115	1,394	.90	.14	.81
g/teat[d]	18.8	17.1	19.9	16.3	26.24	.97	.15	.61
g·teat^{-1}·kg BW^{-1}[e]	122	102	112	90	971.5	.28	.06	.93

[a]Based on right side mammary glands.
[b]Energy intake during the first period (P1):energy intake during the second period (P2); A = adequate energy intake (6.1 Mcal ME/d), E = elevated energy intake (10.5 Mcal ME/d).
[c]Weight of non-secretory tissue less skin, muscle and lymph nodes.
[d]Mammary data are expressed per functional teat (teats associated with secretory tissue).
[e]Mammary data are expressed per functional teat (teats accociated with secretory tissue) per kg maternal BW.

Table 16. Parenchymal tissue DNA and RNA at d 105 of gestation for gilts fed adequate or elevated energy over two feeding periods (d 0 to 74 and d 75 to 105 of gestation)[ab]

Item	Energy Intake[b] A:A (n = 10)	A:E (n = 8)	E:A (n = 8)	E:E (n = 8)	MSE	Energy Effect P1	P2	P1*P2
DNA								
µg/g	2,718	2,666	2,512	2,542	68,280	.07	.88	.65
mg	3,298	3,056	3,557	3,342	607,420	.34	.40	.96
mg/teat[c]	472	453	511	474	10,643	.42	.45	.80
mg·teat^{-1}·kg BW^{-1}[d]	3.1	2.7	2.9	2.6	.412	.48	.16	.81
RNA								
µg/g	5,738	5,435	5,610	5,025	441,053	.22	.07	.54
mg	7,020	6,303	7,968	6,653	3,666,178	.36	.14	.65
mg/teat[c]	1,004	934	1,147	943	67,646	.43	.15	.46
mg·teat^{-1}·kg BW^{-1}[d]	6.5	5.6	6.5	5.2	2.456	.64	.05	.79
RNA/DNA	2.12	2.04	2.23	1.98	.042	.82	.03	.24

[a]Based on right side mammary glands.
[b]Energy intake during the first period (P1):energy intake during the second period (P2); A = adequate energy intake (6.1 Mcal ME/d), E = elevated energy intake (10.5 Mcal ME/d).
[c]Mammary data are expressed per teat (teats associated with secretory tissue).
[d]Mammary data are expressed per teat (teats associated with secretory tissue) per kg maternal BW.

General Discussion

Milk production is related to the amount of mammary secretory tissue in rats and heifers. Studies conducted in heifers during gestation indicate that ad libitum feeding does not influence mammary secretory tissue growth; however, ad libitum feeding in prepubertal sheep and heifers decreases mammary secretory tissue growth. Exogenous growth hormone administered to sheep and cattle increases mammary secretory tissue growth. Polyunsaturated fatty acids stimulate mammary development in other species and may be synergistic with growth hormone. Little research has been conducted on swine mammary development. Earlier research suggested that elevated energy intake during late gestation in swine may have a negative influence on mammary secretory tissue growth. But this conclusion is contested by research conducted herein. The effects of body composition, PUFA, growth hormone and energy intake on mammary development in pregnant swine were studied.

Body compositional changes during gestation did not influence mammary secretory tissue growth. Similarly, feeding PUFA to gilts from d 75 to 105 of gestation did not influence mammary secretory tissue. However, mammary secretory tissue was increased in PST-injected gilts fed adequate energy diets. The increase in mammary secretory tissue in PST-injected gilts fed adequate energy needs to be confirmed along with subsequent milk production measured for gilts injected with PST during the same gestational period. If the increase in mammary growth for PST-injected gilts fed adequate energy can be repeated and excessive energy intake inhibits the positive effect of growth hormone on mammary secretory tissue, as shown in experiment one, then the mechanism whereby

excessive energy inhibits the positive effect of growth hormone should be elucidated.

Weldon et al. (1991) reported that gilts fed diets which provided 10.5 mcal ME/d from d 75 to 105 of gestation had less mammary secretory tissue as compared with gilts fed diets containing 5.7 mcal ME/d. More specifically, gilts fed excessive energy had less parenchymal tissue, parenchymal DNA, parenchymal RNA and parenchymal protein as compared with gilts fed diets which met the NRC (1988) requirement for energy when the data were corrected for the number of functional glands and kg maternal BW. Similarly in the present study, gilts in experiment three, fed elevated energy diets from d 75 to 105 of gestation, had more parenchymal RNA·teat^{-1}·kg BW^{-1} and parenchymal protein teat^{-1}·kg BW^{-1} compared with gilts fed adequate energy diets.

We, in experiment three, and Weldon et al. (1991), corrected parenchymal tissue constituents, such as parenchymal DNA, for the number of teats (teats associated with secretory tissue) and kg maternal BW. This correction is believed to be invalid for the following reasons. First, the number of teats is a selection criteria used in all of the experiments, thus the number of teats are controlled across treatments. Secondly, BW is confounded with treatment in that it is influenced by energy intake; gilts fed excessive energy were heavier ($P < .001$) as compared with gilts fed adequate energy (Table 17).

Further evidence that the data need not be corrected for BW is shown in the regression of BW on parenchymal DNA and RNA for gilts treated similarly prior to mammary gland analysis. No relationship ($R^2 = .004$) existed between gilt BW (130 kg to 200 kg) and parenchymal DNA (1000 mg to 4900 mg) for gilts fed similar diets, illustrated in Figure 1.

Heavier gilts will not necessarily have more DNA than lighter gilts simply because they are heavier.

When the data in experiment three (present study) was expressed on a total basis rather than correcting for the number of teats and maternal BW, total parenchymal RNA and parenchymal protein were not influenced by feeding elevated energy diets. Likewise, when we reanalyzed the data of Weldon (1988) on a total basis, we found that excessive energy did not decrease mammary secretory tissue growth. In experiment one (present study) feeding excessive dietary energy (10.5 mcal ME/d) as compared with feeding adequate dietary energy (6.1 mcal ME/d) from d 75 to 105 of gestation did not influence mammary secretory tissue, regardless of how the data were expressed. Thus, the results from all three experiments lead to the same conclusion, feeding 10.5 mcal ME/d from d 75 to 105 of gestation does not impair mammary secretory tissue growth. These findings are in agreement with studies conducted in prepubertal and pregnant heifers fed a high plane of nutrition (Radcliff et al., 1995; Harrison et al., 1983; Valentine et al., 1987). But are in contrast to conclusions reached in other studies conducted with prepubertal heifers and lambs (Sejrsen et al., 1982; Johnsson and Hart, 1985).

To further confirm that feeding elevated energy from d 75 to 105 of gestation does not impair mammary secretory tissue development in swine, the results from experiment one, experiment three and Weldon (1988) were pooled and statistically analyzed for differences in parenchymal weight, extraparenchymal weight, parenchymal DNA, parenchymal RNA, parenchymal protein and parenchymal fat (Table 17). The model included experiment, energy intake (adequate or elevated) and the interaction between experiment and energy intake. Although, most variables tested had

significant experimental effects ($P < .05$), dietary energy level did not influence total parenchymal weight, parenchymal protein, parenchymal fat, parenchymal DNA, parenchymal RNA or the parenchymal RNA/DNA ratio ($P > .10$). The extraparenchymal tissue weight was higher for gilts fed elevated energy diets as compared with gilts fed adequate energy diets ($P < .10$). Results from this combined analysis confirm the conclusion that mammary secretory tissue is not influenced by feeding elevated energy from d 75 to 105 of gestation.

To confirm the relationship between mammary development and milk production in swine, studies need to be conducted in which mammary development and milk production from the same animals can be correlated. Subsequent to these studies areas of future research include prepubertal and hormonal influences on swine mammary development. Since, little is known about swine prepubertal mammary development and the impact of diet during this period it is an area which warrants further investigation.

Table 17. Composition of mammary tissue in gestating gilts fed diets with adequate or elevated energy levels[a]

Item	Energy level (EL)[b] 6,100 (n = 31)	10,500 (n = 23)	SEM	P value Exp[c]	EL	Exp*EL
Extraparenchymal wt, g	613	714	35.7	.16	.05	.30
Parenchymal wt, g	1314	1270	80	.0006	.75	.31
Parenchymal fat						
mg/g parenchyma	345	289	25.5	.003	.12	.19
g	470	377	44.9	.0009	.14	.16
g/teat	66.7	53.0	5.65	.0001	.09	.14
$g \cdot teat^{-1} \cdot kg\ BW^{-1}$	.41	.29	.037	.008	.03	.19
Parenchymal protein						
mg/g parenchyma	129	100	21.1	.04	.33	.29
g	180	123	37.3	.32	.28	.20
g/teat	25.0	17.1	4.59	.29	.22	.17
$g \cdot teat^{-1} \cdot kg\ BW^{-1}$	.16	.10	.031	.17	.16	.20
Parenchymal DNA						
µg/g parenchyma	2450	2477	95.7	.0001	.84	.44
mg	3089	3048	195.3	.85	.88	.09
mg/teat	448	425	25.5	.68	.51	.08
$mg \cdot teat^{-1} \cdot kg\ BW^{-1}$	2.79	2.42	.15	.13	.09	.12
Parenchymal RNA						
µg/g parenchyma	4038	3946	110	.0001	.55	.19
mg	5195	4916	343	.0001	.56	.16
mg/teat	752	686	43.6	.0001	.28	.19
$mg \cdot teat^{-1} \cdot kg\ BW^{-1}$	4.64	3.87	.26	.0001	.04	.24
Parenchymal RNA/DNA	1.69	1.62	.063	.0001	.46	.91

[a]Based on right side teats associated with secretory tissue.
[b]kcal/kg.
[c]Experimental data pooled from Weldon (1988), experiment 1 and experiment 3.

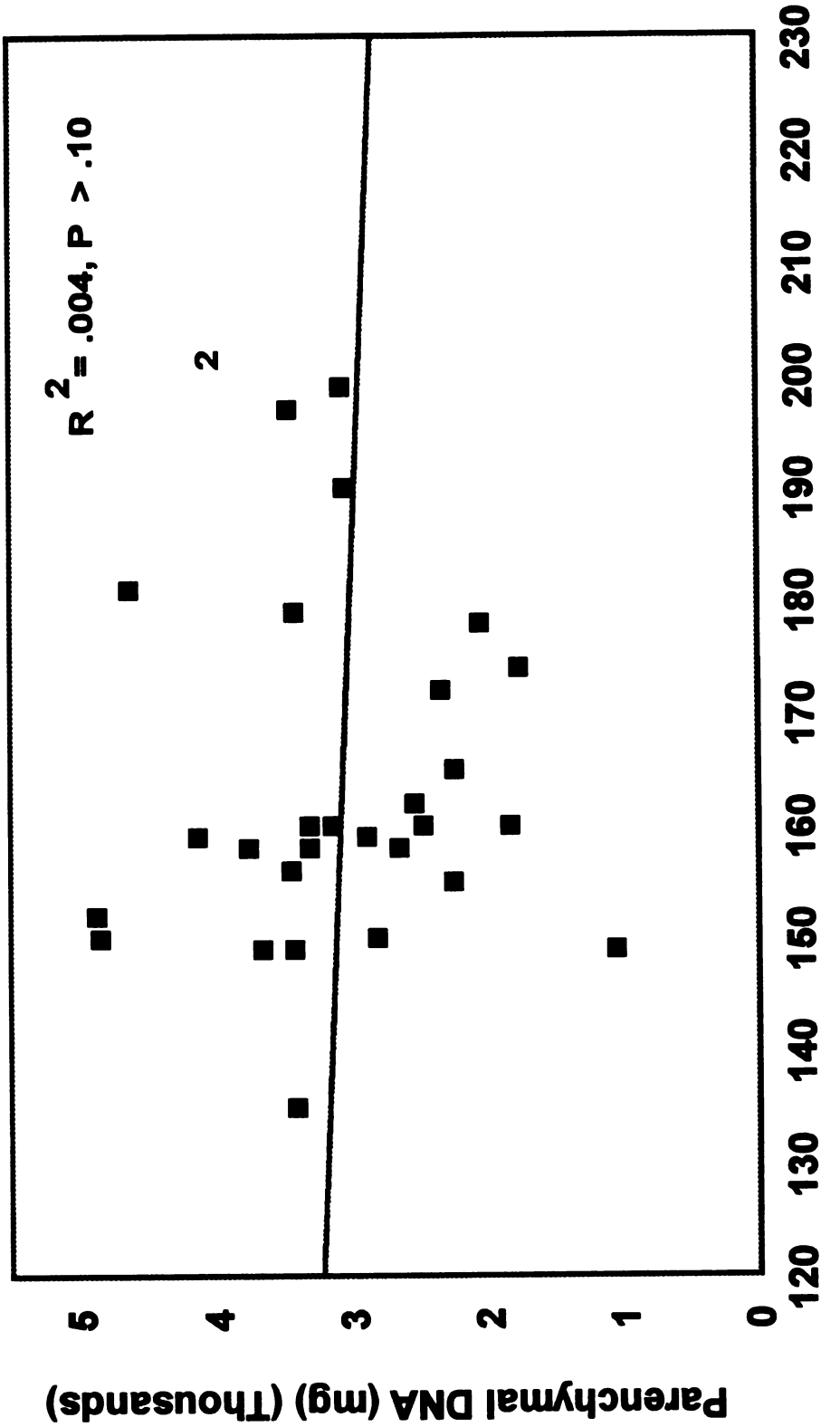

Figure 1. Relationship of parenchymal DNA to gilt body weight

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