VITAMIN SUPPLEMENTATION OF SOWS

by

NICHOLAS WILLIAM SHELTON

B.S., Tarleton State University, 2007
M.S., Kansas State University, 2010

AN ABSTRACT OF A DISSERTATION

submitted in partial fulfillment of the requirements for the degree

DOCTOR OF PHILOSOPHY

Department of Animal Sciences and Industry
College of Agriculture

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Abstract

A total of 701 pigs were used to evaluate effects of natural vitamin E relative to synthetic vitamin E in sow diets, late gestation feeding level on sow reproductive performance, dietary L-carnitine and chromium on sow reproductive performance, and experimental design on nursery pig trial interpretation. As D-α-tocopheryl acetate increased in the sow's diet, concentrations of α-tocopherol increased ($P < 0.03$) in sow plasma, colostrum, milk, pig plasma, and pig heart. Regression analysis indicated that the bioavailability coefficients for D-α-tocopheryl acetate relative to DL-α-tocopheryl acetate ranged from 2.1 to 4.2 for sow and pig plasma α-tocopherol, 2.9 to 3.0 for colostrum α-tocopherol, 1.6 for milk α-tocopherol, 1.8 for heart α-tocopherol, and 2.0 for liver α-tocopherol. Overall, this study indicates that the relative bioavailability for D-α-tocopheryl acetate relative to DL-α-tocopheryl acetate varies depending on the response criteria but is greater than the standard potency value of 1.36. Increasing sow gestation feeding level by 0.9 kg from d 90 of gestation through farrowing reduced ($P = 0.001$) daily lactation feed intake in gilts, but also resulted in improved conception rate in gilts, whereas increasing late gestation feeding level decreased conception rate in sows (interaction; $P = 0.03$). Increasing late gestation feed intake in gilts also increased ($P < 0.02$) pig weaning weights during the second parity. Increasing late gestation feeding levels did not improve performance of older sows. Adding L-carnitine and chromium from chromium picolinate to sow gestation and lactation diets reduced ($P = 0.01$) the amount of sow weight loss during lactation, however, did not improve ($P > 0.05$) litter size, pig birth weight, or the variation in pig birth weight. Blocking pens of nursery pigs by BW in a randomized complete block design (RCBD) did not improve the estimates for σ^2_{error} compared to a completely randomized design (CRD) where all pens were allotted to have similar means and variations of body weight. Therefore, the added degrees of freedom for the error term in the CRD allowed more power to detect treatment differences for the CRD compared to the RCBD.

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Major Professor
Jim Nelssen

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Nick

Chapter 1 - Effects of dietary vitamin E concentration and source on sow, milk, and pig concentrations of α-tocopherol

Abstract

A total of 126 gilts and sows (PIC 1050) and their litters were used to determine the effects of dietary vitamin E concentration and source on sow plasma, milk, and pig concentrations of α-tocopherol. Additionally, we estimated the bioavailability of D-α-tocopheryl acetate (D-α-TAc) relative to DL-α-tocopheryl acetate (DL-α-TAc) when fed in diets containing dried distillers grains with solubles (DDGS). The 6 dietary treatments included DL-α-TAc at 44 and 66 mg/kg and D-α-TAc at 11, 22, 33, and 44 mg/kg. From breeding to d 69 of gestation, sows were fed 2.0 kg/d of a diet containing 40% DDGS, 0.30 mg/kg added Se, and no added vitamin E. Vitamin E treatments were fed from d 70 of gestation through weaning. Plasma was collected from sows on d 69 and 100 of gestation, at farrowing, and at weaning. Colostrum and milk samples were also collected. Plasma from 3 pigs per litter and heart and liver samples from 1 pig per litter were collected at weaning. Plasma, milk, and tissues from 6 litters per treatment were analyzed for α-tocopherol. Although tissue, plasma, and milk concentrations of α-tocopherol were the primary response criteria of interest, sow and litter performance were measured. As expected, treatment effects were not observed for lactation feed intake, sow BW, or backfat measurements. A trend ($P = 0.085$) for a treatment effect on average pig BW at weaning was detected, with pigs nursing sows fed 44 mg/kg DL-α-TAc weighing less because of a younger weaning age. No other differences in litter performance were observed. As D-α-TAc increased in the diet, sow plasma, colostrum, milk, pig plasma, and pig heart concentrations of α-tocopherol increased (linear, $P < 0.03$). Sows fed diets with 44 mg/kg D-α-TAc had increased ($P < 0.03$) plasma, colostrum, and pig plasma concentrations of α-tocopherol compared with sows fed 44 mg/kg of DL-α-TAc. Sows fed 66 mg/kg DL-α-TAc also had greater ($P = 0.022$) plasma α-tocopherol at weaning than sows fed 44 mg/kg DL-α-TAc. Bioavailability coefficients for D-α-TAc relative to DL-α-TAc ranged from 1.9 to 4.2 for sow and pig plasma α-tocopherol, 2.9 to 3.6 for colostrum α-tocopherol, 1.6 for milk α-tocopherol, and 1.7 to 2.0 for pig heart and liver α-tocopherol. Overall, this study indicates the bioavailability for D-α-TAc relative to DL-α-TAc varies depending on the response criteria but is greater than the standard potency value of 1.36.

Key words: α-tocopherol, bioavailability, natural vitamin E, sow

Introduction

Vitamin E is a collective term referring to a group of 8 compounds (α-, β-, γ-, and δ tocopherols and α-, β-, γ-, and δ tocotrienols) that serve as antioxidants in plant and animal tissues. The α-tocopherol compound is the most bioactive form in the lipid component of animals (Traber, 2007). Eight stereoisomers of α-tocopherol have biological activities from 21 to 100% (Weiser et al., 1996). Synthetic vitamin E (DL-α-tocopherol; all rac-α-tocopherol) is a combination of the 8 stereoisomers, whereas natural vitamin E (D-α-tocopherol; RRR-α-tocopherol) comprises only the RRR stereoisomer.

The United States Pharmacopeia (1980) states that 1 IU of vitamin E is equivalent to 1 mg of DL-α-tocopheryl acetate (DL-α-TAc) or 0.735 mg of D-α-tocopheryl acetate (D-α-TAc). The 1.36 biopotency estimate for D-α-TAc relative to DL-α-TAc is based on a pregnant rat model (Harris and Ludwig 1949). The value has been extrapolated for use in other species; however, research suggests the bioavailability ratio for D-α-TAc relative to DL-α-TAc is greater than 1.36 in pigs (Mahan et al., 2000; Lauridsen et al., 2002; Yang et al., 2009).

Increasing colostrum α-tocopherol concentration is important because the newborn pig is vitamin E–deficient and colostrum is the only source for suckling pigs (Lauridsen et al., 2002; Mahan, 1991). Dried distillers grains with solubles (DDGS) based diets increase unsaturated fatty acid concentration compared with corn-soybean meal–based swine diets (Kim et al., 2012; Stein and Shurson, 2009). Unsaturated fatty acids negatively impact the vitamin E status of pigs (Hidiroglou et al., 1993). Therefore, adding DDGS may negatively impact the sow's and her litter's vitamin E status. The objectives of this study were to determine the α-tocopherol concentration in plasma, milk, and pig body tissues when sows were fed diets supplied with D-α-TAc or DL-α-TAc. We also estimated the bioavailability of D-α-TAc relative to DL-α-TAc when included in diets containing DDGS.

Materials and Methods

The protocol used in this experiment was approved by the Kansas State University Institutional Animal Care and Use Committee. The study was conducted at the Kansas State University Swine Teaching and Research Facility in Manhattan, KS.

Animals and diets

A total of 126 gilts and sows and their progeny (PIC 327 × 1050; Hendersonville, TN) were used. The 6 dietary treatments were DL-α-TAc at 44 and 66 mg/kg, and D-α-TAc at 11, 22, 33, and 44 mg/kg. The 44 and 66 mg/kg of DL-α-TAc were selected as treatment levels in this experiment because they reflect the estimated requirement of the sow (NRC, 1998) and the standard dietary level that is commonly used in the industry for vitamin E (PIC, 2008), respectively. The 4 levels of D-α-TAc were selected to evaluate a range of bioavailability for each DL-α-TAc level. Treatments were allotted to sows in a generalized block design with farrowing group as the blocking factor and parity balanced across treatment. Six farrowing groups were used and farrowed from November 2010 through May 2011.

Prior to beginning the experiment, all gilts and sows were fed diets containing 66 mg/kg DL-α-TAc. This level of vitamin E is an industry acceptable level for use in sow diets (PIC, 2008) and the standard inclusion for this particular farm. Beginning at breeding and continuing through d 69 of gestation, gilts and sows were fed 2.0 kg of a gestation diet containing no added vitamin E. On d 70 post coitum, gilts and sows were randomly allotted to dietary treatments and remained on their dietary vitamin E concentration and source through the end of lactation. The gestation and lactation diets were formulated to contain 0.71% and 1.10% total Lys, or 0.55% and 0.94% standardized ileal digestible Lys, respectively (Table 1-1). All diets were also formulated to include 0.30 mg/kg added Se from sodium selenite. All other nutrients were formulated to meet or exceed the NRC (1998) requirements. Gestation and lactation diets were also formulated to contain 40% and 20% DDGS, respectively. A sample of each lot of DDGS was analyzed for S content by inductively coupled plasma atomic emission spectroscopy (AOAC, 2000), and calcium sulfate was added to DDGS to maintain a constant S concentration of 0.80% in the DDGS. Dietary α-tocopherol analysis was also performed by Europhins Scientific Inc. (Des Moines, IA) using HPLC as outlined by AOAC procedure 971.30 (AOAC, 2000). For the first 3 d after farrowing, sows were gradually stepped up on feed, and after d 3, all sows were allowed ad libitum access to the lactation diet. Temperature in the farrowing facility was maintained at a minimum of 20°C, and supplemental heat was provided to litters through the use of heat lamps.

Data collection and analysis

Although they were not the primary response criteria for the experiment, sow BW and backfat thickness measurements were recorded at breeding, d 69 of gestation, postfarrowing, and at weaning. Individual pig BW, litter size, and total litter weight were recorded at birth, d 3 of lactation, d 17 of lactation, and at weaning. Lactation feed intake was measured. The primary response criteria for this trial included plasma, pig tissue, and milk α- tocopherol concentrations. Plasma was collected from sows via jugular venapuncture on d 69 and 100 of gestation approximately 4 h after feeding. Plasma was collected in tubes containing sodium heparin as an anticoagulant. Plasma was stored on ice for approximately 1 h, and then centrifuged at 1,600 × *g* for 20 min. Milk and sow plasma samples were also collected at 8 to 12 h postfarrowing and at weaning. Milk samples were obtained after an intravenous injection of oxytocin, and milk was collected with a minimum of 15 mL from each functional gland. At weaning, plasma was taken from the vena cava of 3 pigs per litter, and 1 pig per litter was stunned and killed to obtain heart and liver samples. Each heart and liver was immediately flash-frozen in liquid N to limit any oxidation in the tissue. Milk, plasma, and tissue samples were kept at -80°C from the time of collection until the completion of the live animal portion of the study. After all samples were collected, α-tocopherol concentrations were determined for all samples at the same time.

From each farrowing group, 1 sow and litter per dietary treatment were used for plasma, milk, and pig tissue analysis of α-tocopherol, and females from the same parity were selected for each dietary treatment within each farrowing group. In addition, litters with similar lactation lengths, number of piglets, litter weights, and sow lactation feed intakes across the 6 dietary treatments were selected for α-tocopherol analysis in order to limit any potential bias. Tissue samples were prepared for analysis as outlined by Willburn et al. (2008), and samples were analyzed for α-tocopherol by HPLC using the procedures of Zaspel and Csallany (1983). The samples were sent on dry ice overnight and were analyzed at The Ohio State University, Columbus, OH.

Statistical Analysis

Experimental data were analyzed initially using PROC MIXED in SAS (SAS Institute Inc., Cary, NC). Overall treatment significance was first determined by the overall treatment F-test. Contrast statements also were used to test for linear and quadratic effects associated with

increasing D-α-TAc and to compare the 44 mg/kg DL-α-TAc treatment separately to both the 44 mg/kg D-α-TAc and 66 mg/kg DL-α-TAc treatments. Farrowing group was used as a random effect, and sow or litter was used as the experimental unit for all data analysis. For sow performance, interactions between dietary treatments and farrowing group were found to be non-significant, and were therefore pooled with the error variance component for each response. For sow plasma, d-69 plasma α-tocopherol was used as a covariate. Statistics were considered significant at P values < 0.05 and tendencies at P values < 0.10.

Coefficients for the bioavailability of D-α-TAc relative to DL-α-TAc were also calculated based on both the formulated and analyzed concentrations of both DL-α-TAc treatments. The estimation of added α-tocopherol based on analyzed concentration was calculated by subtracting an estimate for the amount of indigenous α-tocopherol from the analyzed concentration for each dietary treatment. The estimated amounts of indigenous α-tocopherol were 11.4 and 8.9 mg/kg for gestation and lactation, respectively. Analyzed dietary concentrations of α-tocopherol in the gestation diets were used to calculate bioavailability estimates based on α-tocopherol concentrations in sow plasma on d 100 of gestation, sow plasma at farrowing, and colostrum. Analyzed dietary concentrations of α-tocopherol in the lactation diets were used to calculate bioavailability estimates based on α-tocopherol concentrations in sow plasma at weaning, sow milk, and pig tissues. To calculate estimates of bioavailability, linear regression was first conducted with PROC REG in SAS to relate the analyzed plasma, milk, and tissue concentrations of α-tocopherol to the dietary concentrations of added D-α-TAc. Based on the regression line, the D-α-TAc dietary concentration needed to achieve the same tissue concentration of α-tocopherol as each of the DL-α-TAc treatments was calculated. The ratio of the each dietary DL-α-TAc relative to the calculated D-α-TAc was used to estimate the relative bioavailability.

Results

The analyzed concentrations of α-tocopherol in each treatment's gestation and lactation diet are shown in Table 1-2. The analyzed α-tocopherol values were similar to the expected values with the exception of the lactation diet with 66 mg/kg DL-α-TAc, gestation diet with 44 mg/kg D-α-TAc, and lactation diet with 44 mg/kg D-α-TAc, which analyzed lower than expected.

No differences were observed in sow BW or backfat thickness measurements at any of the time points (Table 1-3). Also, no differences were observed in total or daily lactation feed intake. No litter size, average weight, or total litter weight differences were observed for total born, live born, d 3 of lactation, or d 17 of lactation (Table 1-4). A trend was observed (P = 0.085) for a difference in average pig BW at weaning, primarily due to the numerically lower average pig BW for sows fed the diet containing 44 mg/kg DL-α-TAc compared with other concentrations or sources of vitamin E. This lower average weight may be a function of the difference (P = 0.044) in weaning age, in addition to a numerically lower average pig birth weight for sows on that particular treatment, which was unexpected.

Sow plasma α-tocopherol was similar (P = 0.724) on d 69 when all sows were fed diets containing no added vitamin E, but it increased (linear, P < 0.01) with increasing D-α-TAc on d 100 of gestation, postfarrowing, and at weaning (Table 1-5). Sow plasma α-tocopherol was greater (P < 0.01) for sows fed 44 mg/kg D-α-TAc compared with those of sows fed either of the 2 DL-α-TAc concentrations at each time point. Sow plasma α-tocopherol also increased (P = 0.022) at weaning with increased dietary concentrations of DL-α-TAc. The calculated bioavailability estimates for sow plasma α-tocopherol concentrations on d 100 of gestation ~~of~~ were 2.1 and 2.4 for the 44 and 66 mg/kg DL-α-TAc treatments, respectively, based on the formulated concentrations of added DL-α-TAc and D-α-TAc (Table 1-6). Plasma α-tocopherol postfarrowing yielded bioavailability estimates of 4.2 and 3.0 for the 44 and 66 mg/kg DL-α-TAc treatments, respectively, based on the formulated concentrations of added DL-α-TAc and D-α-TAc. Estimates of bioavailability based on sow plasma α-tocopherol at weaning were 2.7 and 2.4 for the 44 and 66 mg/kg DL-α-TAc treatments, respectively, based on the formulated concentrations of added DL-α-TAc and D-α-TAc. Estimates of bioavailability based on sow plasma α-tocopherol in relation to analyzed dietary α-tocopherol were 1.9 to 3.9.

Sow colostrum and milk α-tocopherol increased (linear, P < 0.03) with increasing dietary D-α-TAc (Table 1-5). Sows fed 44 mg/kg D-α-TAc had greater (P = 0.003) colostrum α-tocopherol than sows fed 44 mg/kg DL-α-TAc. A numerical increase in colostrum α-tocopherol was also observed as DL-α-TAc increased in the sow's diet, but the increase was not significant (P = 0.456). The calculated bioavailability estimates based on colostrum α-tocopherol were 3.0 and 2.9 for the 44 and 66 mg/kg DL-α-TAc treatments, respectively, based on formulated concentrations of added DL-α-TAc and D-α-TAc (Table 1-6). The estimated bioavailability for

colostrum α-tocopherol was 3.6 for both DL-α-TAc treatments when using the analyzed dietary α-tocopherol concentrations. The estimates for bioavailability based on milk α-tocopherol were 1.6 and 7.3 for the 44 and 66 mg/kg DL-α-TAc treatments, respectively, based on formulated concentrations of added DL-α-TAc and D-α-TAc. The estimated bioavailability for milk α-tocopherol was 1.6 and 4.0 for the 44 and 66 mg/kg DL-α-TAc treatments, respectively, based on analyzed dietary concentrations of α-tocopherol.

Heart and plasma α-tocopherol in the suckling pigs increased (linear, $P < 0.01$) as the D-α-TAc increased in the sow's diet and tended to increase (linear, $P = 0.089$) in the pig's liver (Table 1-5). Pigs from sows fed 44 mg/kg D-α-TAc had greater ($P < 0.05$) plasma α-tocopherol compared with pigs from sows fed 44 mg/kg DL-α-TAc. Similar to sow's milk, a numerical decrease in plasma, heart, and liver α-tocopherol was observed as DL-α-TAc increased in the sow's diet; however, the differences were not significant. The estimated bioavailability from the suckling pigs with 44 and 66 mg/kg DL-α-TAc concentrations were 3.0 and 5.1 for plasma, 1.8 and 5.3 for heart, and 2.0 and 7.5 for liver, respectively, based on formulated concentrations of added DL-α-TAc and D-α-TAc (Table 1-6). Estimated bioavailability based on analyzed dietary concentration α-tocopherol for the suckling pigs were 2.0 and 3.4 for plasma, 1.7 and 3.4 for heart, and 1.9 and 4.2 for liver based on the 44 and 66 mg/kg DL-α-TAc concentrations, respectively.

Discussion

This experiment demonstrated no differences in sow or litter performance associated with varying concentrations of either D-α-TAc or DL-α-TAc. The main objective of the experiment was to determine the relative efficacy of the 2 sources of vitamin E on various biological parameters. Mahan et al. (2000) compared supplementing sow diets with 30 or 60 IU/kg of either D-α-TAc or DL-α-TAc and also observed no differences in lactation litter performance over 5 parities, so the lack of differences in our experiment were not unexpected. Several other studies have indicated that 30 to 60 IU/kg of added vitamin E is sufficient to optimize reproductive performance (Mahan, 1991, 1994; Nielsen et al., 1979).

The diets used in this experiment contained DDGS. Adding DDGS to swine diets results in increased amounts of dietary unsaturated and polyunsaturated fatty acids (Stein and Shurson, 2009). Increasing the amount of dietary PUFA in sow diets has been shown to lower the pig's vitamin E status (Hidroglou et al., 1993). The PUFA found in DDGS are susceptible to lipid

peroxidation (NRC, 1998). The amount of peroxidation is related to duration and amount of heat to which PUFA are exposed during the drying process; therefore, the amount of oxidized lipids in DDGS can vary among ethanol plants as well as individual production lots.

Adding DDGS to swine diets also increases the dietary S concentration. The S content in DDGS is greater than other ingredients due to the addition of sulfuric acid during the ethanol production process and corn protein's greater proportion of the sulfur-containing amino acids compared with soy protein (Kim et al., 2012). Sulfur is a component in many bioactive compounds, such as Met, Cys, and glutathione, which all have antioxidant properties (Atmaca, 2004). Sulfur also has been shown to compete with Se for absorption in ruminants (Spears, 2003). Therefore, feeding elevated sulfur amounts may have both positive and negative effects on the oxidative stress on the animal. Each incoming load of DDGS was analyzed for S content, and calcium sulfate was added to achieve a constant S level of 0.80% to standardize any impacts of S in our experiment. The similar bioavailability estimates from our experiment compared with other studies that did not use DDGS (Lauridsen et al., 2002; Mahan et al., 2000) suggests that dietary DDGS inclusions did not alter the bioavailability of D-α-TAc relative to DL-α-TAc.

Hanson et al. (2013) demonstrated that feeding sows a diet containing DDGS with a high concentration of oxidized lipids resulted in a decreased concentration of α-tocopherol in the serum of weaned pigs compared with pigs nursing sows fed a diet with no DDGS and a lower concentration of oxidized lipids. In contrast, Song et al. (2013) indicated that feeding DDGS with a high concentration of oxidized lipids to pigs for 8 wk post weaning increased the α-tocopherol concentration in serum compared with pigs fed diets without DDGS and a decreased concentration of oxidized lipids. The authors suggest that this observation is a result of a sparing effect of α-tocopherol by the elevated S-containing antioxidants associated with DDGS supplementation in the diet. One possible theory that combines the results of these 2 studies is that feeding sows diets with DDGS containing oxidized fatty acids will be more detrimental to the α-tocopherol status of the nursing pigs than to the sow, possibly due to the elevated S-containing antioxidants' ability to spare α-tocopherol in the sow but not transfer through the milk to the nursing pig. Additional research is needed to validate this hypothesis.

All sows were fed a non-vitamin E–fortified diet for the first 69 d of the experiment. This model was used to reduce the sows' storage capacity of vitamin E and appeared effective because plasma concentrations of α-tocopherol increased across dietary treatments from d 69 of

gestation to d 100. A similar model was used by Yang et al. (2009) for finishing pigs, in which increasing amounts of D-α-TAc resulted in increases in plasma concentrations of α-tocopherol. Although both the gestation and lactation diets containing the 44 mg/kg level of added D-α-TAc analyzed approximately 5 to 10 mg/kg less than expected, the numerical increase in plasma α-tocopherol associated with that particular treatment suggests that the lower analyzed levels may have been due to analytical variation. Increasing the amount of DL-α-TAc resulted in increased sow plasma concentrations of α-tocopherol at weaning. Similar increases in sow plasma α-tocopherol have been observed with increasing D-α-TAc or DL-α-TAc (Mahan, 1991; Mahan et al., 2000). The estimates of bioavailability based on plasma concentrations of α-tocopherol were 1.9 to 4.2, with similar estimates calculated using formulated and analyzed dietary levels of α-tocopherol. Lauridsen et al. (2002) administered deuterated labeled forms of D-α-TAc and DL-α-TAc to compare the bioavailability of the 2 sources by supplementing both simultaneously. Based on ratios of the isotopes in plasma, they estimated the bioequivalence of D-α-TAc to be 2 times that of DL-α-TAc, which is similar to our conclusions. Mahan et al. (2000) also demonstrated that when sow diets contained similar vitamin E concentrations from the 2 sources on an IU basis, D-α-TAc increased plasma α-tocopherol concentrations compared with DL-α-TAc.

The newborn pig is vitamin E–deficient, and colostrum is the only source of α-tocopherol for suckling pigs (Lauridsen et al., 2002; Mahan, 1991). In the present study, increasing the amount of added D-α-TAc resulted in increased α-tocopherol concentrations in both colostrum and milk. Mahan et al. (2000) demonstrated increased α-tocopherol concentrations for colostrum and milk when increasing concentrations of DL-α-TAc or D-α-TAc were provided. They also indicated that when diets contained similar concentrations of vitamin E on an IU basis, D-α-TAc increased in the colostrum and milk concentrations of α-tocopherol compared with DL-α-TAc. In our experiment, milk α-tocopherol was numerically lower for the 66 mg/kg DL-α-TAc treatment than for the 44 mg/kg treatment. This response was also observed for α-tocopherol concentrations in the plasma, liver, and hearts of suckling pigs. One explanation for the decrease in milk and pig α-tocopherol concentrations is that sows consuming the diet containing 66 mg/kg of added DL-α-TAc did not transfer greater quantities of α-tocopherol into the milk as compared to sows consuming the diet with 44 mg/kg of added DL-α-TAc despite consuming a greater daily amount of DL-α-TAc and having greater plasma α-tocopherol concentrations at weaning. While

numerically lower, the differences in milk and pig α-tocopherol concentrations were not significantly different between the DL-α-TAc treatments and could simply have been due to sampling variation. Another possible explanation for this observation might be related to the 66 mg/kg of added DL-α-TAc lactation diet analyzing approximately 10 mg/kg of α-tocopherol below the expected level; however, it still analyzed with a greater concentration of α-tocopherol than the 44 mg/kg of added DL-α-TAc treatment. As a result of this observation, the bioavailability estimate for milk is based on the 44 mg/kg DL-α-TAc treatment; therefore, the estimated bioavailability for D-α-TAc relative to DL-α-TAc was 3.0 for colostrum α-tocopherol and 1.6 for milk α-tocopherol based on formulated dietary α-tocopherol concentrations. Similar bioavailability estimates were calculated when using analyzed dietary α-tocopherol concentration. The estimated bioavailability based on milk α-tocopherol concentrations was similar to the estimate of 1.54 by Mahan et al. (2000). Lauridsen et al. (2002) showed a 2:1 ratio for D-α-TAc relative to DL-α-TAc based on milk concentrations of labeled vitamin E sources.

It is important that the concentrations of α-tocopherol increase in pigs while they are nursing the sow because the neonate pig is born vitamin E deficient (Mahan, 1991) and plasma α-tocopherol drops post weaning (Lauridsen and Jensen, 2005). The plasma α-tocopherol concentrations observed from all treatments in this study would be above the threshold of 0.4 to 1.0 μg/mL, which separates deficiency from sufficiency (Jensen et al., 1988; Van Vleet, 1980). Concentrations of α-tocopherol in pig plasma, livers, and hearts followed patterns similar to milk α-tocopherol concentrations. Increasing D-α-TAc resulted in increased α-tocopherol in pig plasma, hearts, and livers. Mahan et al. (2000) demonstrated increased α-tocopherol concentrations in pig livers and plasma when increasing the amount of D-α-TAc or DL-α-TAc. Similar to milk, the bioavailability estimates are based on the 44 mg/kg DL-α-TAc treatment, so the estimated bioavailability for D-α-TAc relative to DL-α-TAc was 3.0 for pig plasma α-tocopherol, 1.8 for heart α-tocopherol, and 2.0 for liver α-tocopherol based on formulated concentrations of added D-α-TAc and DL-α-TAc; similar bioavailabilities were estimated using analyzed dietary α-tocopherol concentrations.

Previous research has established that the biological activity of D-α-TAc and DL-α-TAc differ due to the differences in confirmation of the stereoisomers in DL-α-TAc (Blatt et al., 2004). The potency estimate of 1.36 used in conversion of IU (United States Pharmacopia, 1980) was based on data using a rat fetal absorption model (Harris and Ludwig, 1948). That potency

value has been extrapolated for use in other livestock species as well as in humans. Recent findings related to the metabolism and transport of α-tocopherol stereoisomers suggests that the bioavailability is different in pigs and humans, particularly when dietary vitamin E concentrations are fed at the requirement. In addition, the affinity of different tissues can result in different bioavailability estimates. Understanding the metabolism associated with α-tocopherol may help explain differences from the original potency value.

The hepatic α-tocopherol transfer protein (α-TTP) is responsible for regulating plasma concentrations of α-tocopherol. The α-TTP-α-tocopherol complex will move α-tocopherol to the plasma membrane and release the α-tocopherol so it can be taken up by very low-density lipoproteins and released into the blood stream (Horiguchi et al., 2003). Hepatic α-TTP has varying affinities for different forms of vitamin E; RRR-α-tocopherol is 100%, SRR-α-tocopherol is 10.5%, and other tocopherols vary from 1.5% to 38% (Hosomi et al., 1997). This suggests that the α-TTP preferentially transports stereoisomers of α-tocopherol with the 2-R confirmation in comparison to the 2-S confirmation (Leonard et al., 2002; Traber et al., 1990). In addition to hepatic tissue, the α-TTP also has been detected in the pregnant mouse uterus (Jishage et al., 2001). In the rat fetal absorption model, rats were depleted of vitamin E for an extended period prior to beginning test diets (Harris and Ludwig, 1948). Due to the depletion of vitamin E in rats, the α-TTP may have transported greater quantities of the 2-S stereoisomers of α-tocopherol to the uterus than if the rats were not in a deficient state.

Studies have also indicated differences in elimination of α-tocopherol with D-α-TAc compared with DL-α-TAc. Traber et al. (1998) dosed 150 mg of deuterated D-α-TAc and DL-α-TAc and showed that plasma contained twice as much α-tocopherol from D-α-T as DL-α-T; however, the urinary metabolite 2,5,7,8-tetramethyl (2'-carboxyethyl)-6-hydroxychroman (α-CEHC) from DL-α-TAc was 3 to 4 times greater than α-CEHC from D-α-TAc. Clifford et al. (2006) also performed a crossover study in humans and demonstrated that the degradation and elimination of α-tocopherol from DL-α-TAc was 2 to 3 times greater than for D-α-TAc.

Meglia et al. (2006) looked at the relative proportions of α-tocopherol stereoisomers in the milk and plasma of dairy cows during late gestation and early lactation. Essentially all of the α-tocopherol in plasma and milk were the RRR-confirmation when cows were fed diets containing D-α-T or D-α-TAc; however, about 90% of the α-tocopherol was in the RRR conformation in plasma and milk when cows were fed diets contained DL-α-TAc. The remaining

10% was primarily made up of the other 2-R stereoisomers (RRS, RSR, and RSS) and a small portion (1%) of the 2-S stereoisomers (SSS, SRR, SRS, and SSR). Jensen and Lauridsen (2006) performed a similar experiment with sows in late gestation and lactation and determined that when DL-α-TAc was used as the source of α-tocopherol, approximately 35% of the α-tocopherol in milk was in the RRR-configuration, 55 to 65% was in the other 2-R configuration, and 5 to 8% were in the 2-S configuration. The relative proportion of different stereoisomers of α-tocopherol in suckling pig plasma was similar to that of the milk. These findings agree with hypothesis that the 2-S stereoisomers are metabolized rapidly and not circulated in the bloodstream at concentrations equal to the 2-R stereoisomers.

A great deal of controversy arises from conflicting terminology in publications. The bioavailability of a nutrient has been defined as the proportion of ingested nutrient that is absorbed in its chemical form and available for use in metabolic pathway (Ammerman et al., 1995). This can be difficult to determine because the chemical forms of D-α-TAc and DL-α-TAc are not identical (Hoppe, 2010). In a letter to the editor, Hoppe (2010) points out that the potency for D-α-TAc relative to DL-α-TAc can be determined only by measuring a physiological activity reflective of vitamin E activity as was accomplished in the rat fetal absorption model. Hoppe (2010) also states that no true potency tests have been conducted in pigs or cattle, and it is therefore premature to conclude that the potency ratio is different than 1.36 as was determined in the rat fetal absorption assay (Harris and Ludwig, 1948). Hoppe (2010) claims that measurements of bioavailability based on concentrations or ratios of labeled forms of α-tocopherol in various tissues pools are not valid estimates to replace the potency value of 1.36 as determined in the rat fetal absorption model. The retention of α-tocopherol also needs to be considered because they differ greatly.

Although estimates of bioavailability presented in this experiment may not be true estimates of potency, they do provide insight into the various tissue concentrations of α-tocopherol and how vitamin E sources affect those pools of α-tocopherol. It should be noted that this experiment was performed with concentrations of DL-α-TAc either at or above the sows' requirement (NRC, 1998). True potency can be quantified only in a deficient state, and it has been suggested that the bioavailability changes with different dosages and durations (Hoppe and Krennrich, 2000; Hoppe, 2010). Therefore, the potency of D-α-TAc relative to DL-α-TAc may underestimate the sows' ability to utilize D-α-TAc relative to DL-α-TAc when concentrations at

or slightly above the requirement are fed due to differences in retention and elimination of the various α-tocopherol stereoisomers.

In conclusion, this experiment demonstrated a range of bioavailability estimate coefficients. The bioavailability coefficients for D-α-TAc relative to DL-α-TAc ranged from 1.9 to 4.2 for sow and pig plasma α-tocopherol, 2.9 to 3.6 for colostrum α-tocopherol, 1.6 for milk α-tocopherol, 1.8 for heart α-tocopherol, and 2.0 for liver α-tocopherol. Overall, this study suggests that the bioavailability for D-α-TAc relative to DL-α-TAc varies depending on the response criteria but is greater than the standard potency of 1.36 when sows are fed diets close to the requirement of vitamin E.

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Table 1-1 Composition of diets (as-fed basis)

Feed ingredient	Gestation	Lactation
Corn	51.98	51.96
Soybean meal (46.5% CP)	4.15	24.24
DDGS[1]	40.00	20.00
Monocalcium P (21% P)	0.70	1.00
Limestone	1.75	1.45
Salt	0.50	0.50
Vitamin premix[2]	0.25	0.25
Trace mineral premix[3]	0.15	0.15
L-Lys HCl	0.18	0.10
Phytase[4]	0.10	0.10
Vitamin E premix[5]	0.25	0.25
Calculated analysis		
ME, kcal/kg	3,302	3,293
CP, %	17.4	21.2
Total Lys, %	0.71	1.10
SID[6] amino acids, %		
Lys	0.55	0.94
Thr	0.49	0.66
Met	0.28	0.32
Trp	0.11	0.20
Ile	0.51	0.74
Leu	1.67	1.79
Val	0.66	0.86
Ca, %	0.84	0.84
P, %	0.61	0.66
Available P, %[8]	0.50	0.49

[1]Dried distillers grains with solubles.
[2]Vitamin premix provided per kilogram of diet: 11,022 IU of vitamin A, 1,378 IU of vitamin D_3, 4.41 mg of vitamin K, 0.04 mg of vitamin B_{12}, 49.60 mg of niacin, 27.56 mg of pantothenic acid, 8.26 mg of riboflavin, 0.22 g of biotin, 1.65 mg of folic acid, 4.96 mg of pyridoxine, 551.14 mg of choline, 49.6 mg of carnitine, and no vitamin E.
[3]Trace mineral premix provided per kilogram of diet: 16.5 mg Cu from $CuSO_4$, 200 µg of Cr from $Cr(C_6H_4NO_2)_3$,0.30 mg I from $Ca(IO_3)_2$, 165 mg Fe from $FeSO_4$, 40 mg Mn from $MnSO_4$, 0.305 mg Se from $NaSeO_3$, and 165 mg Zn from $ZnSO_4$.
[4]Phyzyme 600 (Danisco Animal Nutrition, St Louis, MO) provided 600 phytase units (FTU)/ kg of diet.
[5]Vitamin E premixes were generated for each treatment by combining appropriate amounts of DL-α-tocopheryl acetate or D-α-tocopheryl acetate and rice hulls. For the depletion diet used in gestation, the vitamin E premix was replaced with corn starch.
[6]Standardized ileal digestible.
[7]Phytase provided 0.11% available P to the gestation and lactation diets.

Table 1-2 Dietary concentration of α-tocopherol, mg/kg[1]

Source of vitamin E:	DL-α-TAc[2]		D-α-TAc[3]			
Formulated added vitamin E:	44	66	11	22	33	44
Analyzed concentrations of α-tocopherol						
Gestation diet	54.4	85.7	23.0	33.4	46.0	45.7
Lactation diet	54.9	66.3	23.0	33.2	47.6	48.4
Estimation of added α-tocopherol based on analyzed concentration[4]						
Gestation diet	43.0	74.3	11.6	22.0	34.6	34.3
Lactation diet	46.0	57.4	14.1	24.3	38.7	39.5

[1]Samples were collected from each batch of feed manufactured. From the samples, a composite for each dietary treatment and phase (gestation and lactation) was used for analysis of α-tocopherol.
[2]DL-α-tocopheryl acetate (all-rac-α-tocopheryl acetate).
[3]D-α-tocopheryl acetate (RRR-α-tocopheryl acetate).
[4]The estimation of added α-tocopherol based on analyzed concentration was calculated by subtracting an estimate for the amount of indigenous α-tocopherol from the analyzed concentration for each dietary treatment. The estimated amounts of indigenous α-tocopherol were 11.4 and 8.9 mg/kg for gestation and lactation, respectively.

Table 1-3 Effects of vitamin E concentration and source on sow backfat, sow body weights, and lactation feed intake[1]

Source of vitamin E:	DL-α-TAc[2]		D-α-TAc[3]					Significance level, *P* =		
									D-α-TAc	
Added vitamin E, mg/kg:	44	66	11	22	33	44	SEM	Trt	Linear	Quadratic
n	21	21	21	21	21	21				
Backfat measurements, mm[4]										
Breeding[5]	15.7	16.0	16.0	16.0	15.6	15.9	0.70	0.997	0.839	0.794
d 69 of gestation[6]	16.2	16.4	16.2	16.0	15.9	16.3	0.76	0.998	0.952	0.717
Postfarrowing	15.8	15.7	15.6	15.9	15.7	16.0	0.59	0.995	0.682	0.933
Weaning	12.5	12.3	12.4	12.0	11.9	13.0	0.57	0.778	0.466	0.194
Sow BW, kg										
Breeding[5]	187	181	192	189	189	192	7.6	0.862	0.956	0.692
d 69 of gestation[6]	207	203	209	206	209	212	7.2	0.933	0.703	0.562
Postfarrowing	213	208	218	213	214	216	6.7	0.886	0.860	0.562
Weaning	206	202	210	205	204	209	6.8	0.934	0.867	0.358
Daily lactation feed intake, kg										
d 0 to 17	6.09	5.98	5.92	5.89	5.78	5.90	0.335	0.978	0.881	0.779
d 0 to weaning	6.20	6.11	6.05	6.01	5.89	6.12	0.336	0.975	0.926	0.611

[1]A total of 126 sows and litters were used over 6 farrowing groups to determine the effects of supplemental vitamin E concentration and source on sow, milk, and pig concentrations of α-tocopherol.
[2]DL-α-tocopheryl acetate (all-rac-α-tocopheryl acetate).
[3]D-α-tocopheryl acetate (RRR-α-tocopheryl acetate).
[4]Backfat measurements were determined by averaging both sides at the P2 position (last rib and approximately 65 mm off the midline).
[5]From breeding until d 70 of gestation, all sows were fed a deficient diet containing no supplemental vitamin E.
[6]On d 70, sows were allotted to treatment diets; sows remained on the same vitamin E concentration throughout the remainder of gestation as well as lactation.

Table 1-4 Effects of vitamin E concentration and source on sow lactation performance[1]

Source of vitamin E:	DL-α-TAc[2]		D-α-TAc[3]					Significance level, *P* =		
									D-α-TAc	
Added vitamin E, mg/kg:	44	66	11	22	33	44	SEM	Trt	Linear	Quadratic
n	21	21	21	21	21	21				
Litter size, n										
Total born	14.1	13.2	12.6	12.6	13.2	14.0	0.82	0.645	0.172	0.601
Live born	13.7	13.0	12.0	12.0	12.8	13.2	0.81	0.652	0.235	0.793
d 3	11.7	11.8	11.4	11.4	12.0	11.9	0.35	0.487	0.088	0.747
d 17	11.5	11.3	11.0	10.9	11.3	11.1	0.34	0.748	0.564	0.781
Weaning	11.5	11.3	11.0	10.8	11.3	11.1	0.34	0.651	0.460	0.913
Total litter wt, kg										
Total born	18.6	19.5	17.1	17.5	17.9	19.0	0.98	0.464	0.152	0.728
Live born	18.3	19.2	16.5	17.0	17.5	18.2	0.97	0.428	0.205	0.896
d 3	20.7	22.6	20.9	21.1	21.5	21.4	0.78	0.540	0.561	0.801
d 17	55.8	58.7	57.3	57.4	56.7	58.4	2.40	0.953	0.800	0.719
Weaning	60.1	66.2	62.6	62.6	64.9	65.6	2.62	0.523	0.326	0.883
Pig wt, kg										
Total born	1.33	1.52	1.41	1.45	1.39	1.40	0.053	0.170	0.690	0.781
Live born	1.34	1.53	1.44	1.47	1.40	1.42	0.053	0.230	0.632	0.927
d 3	1.76	1.91	1.84	1.85	1.79	1.79	0.056	0.371	0.397	0.954
d 17	4.87	5.19	5.29	5.27	4.99	5.26	0.189	0.302	0.611	0.348
Weaning	5.24	5.87	5.75	5.81	5.72	5.89	0.208	0.085	0.671	0.751
Lactation length, d	19.1	20.0	19.2	19.5	20.0	20.2	0.31	0.044	0.010	0.816

[1]A total of 126 sows and litters were used over 6 farrowing groups to determine the effects of supplemental vitamin E concentration and source on sow, milk, and pig concentrations of α-tocopherol.
[2]DL-α-tocopheryl acetate (all-rac-α-tocopheryl acetate).
[3]D-α-tocopheryl acetate (RRR-α-tocopheryl acetate).

Table 1-5 Effects of vitamin E concentration and source on sow plasma, milk and pig tissue concentrations of α-tocopherol[1]

								Significance level, *P* =				
Source of vitamin E:	DL-α-TAc[2]		D-α-TAc[3]						D-α-TAc		44 DL-α-TAc vs.	
Added vitamin E, mg/kg:	44	66	11	22	33	44	SEM	Trt	Lin.	Quad.	44 D-α-TAc	66 DL-α-TAc
No. of samples	6	6	6	6	6	6						
Tissue concentrations of α-tocopherol, μg/mL												
Sow plasma												
d 69 of gestation[4]	1.00	0.85	0.89	0.89	0.95	0.98	0.082	0.724	0.383	0.849	0.828	0.177
d 100 of gestation[5]	1.32	1.51	1.09	1.28	1.64	1.99	0.187	0.003	0.001	0.556	0.003	0.372
Post-farrowing[5,6]	0.72	0.87	0.75	0.86	1.01	1.19	0.120	0.018	0.003	0.717	0.002	0.289
Weaning[4]	1.41	1.88	1.15	1.75	2.02	2.53	0.139	0.001	0.001	0.739	0.001	0.022
Sow colostrum[6]	8.19	10.31	7.62	11.39	9.4	17.76	2.165	0.017	0.004	0.258	0.003	0.456
Sow milk[7]	3.25	2.51	2.36	3.22	3.75	3.63	0.458	0.145	0.030	0.260	0.524	0.232
Pig[7]												
Plasma	2.47	2.38	2.11	3.03	3.51	3.78	0.376	0.024	0.004	0.395	0.022	0.861
Heart	4.84	3.93	3.60	4.75	5.93	6.00	0.619	0.014	0.002	0.301	0.128	0.225
Liver	4.18	3.39	2.99	4.88	4.96	5.12	1.063	0.339	0.089	0.301	0.423	0.499

[1]A total of 126 sows and litters were used over 6 farrowing groups to determine the effects of supplemental vitamin E concentration and source on sow, milk, and pig concentrations of α-tocopherol.
[2]DL-α-tocopheryl acetate (all-rac-α-tocopheryl acetate).
[3]D-α-tocopheryl acetate (RRR-α-tocopheryl acetate).
[4]Prior to beginning dietary treatments.
[5]Adjusted with d 69 as a covariate.
[6]Collected 8 to 12 h after the completion of farrowing.
[7]Collected at the time of weaning.

Table 1-6 Bioavailability estimates based on tissue concentrations of α-tocopherol[1]

	Calc. bioavailability of D-α-TAc[2] relative to DL-α-TAc[3]			
Formulated synthetic vitamin E, mg/kg:	44		66	
Based on dietary concentrations:	Formulated	Analyzed[4]	Formulated	Analyzed[4]
Sow plasma				
d 100 of gestation	2.1	2.1	2.4	2.9
Postfarrowing	4.2	3.9	3.0	3.6
Weaning	2.7	2.4	2.4	1.9
Sow colostrum	3.0	3.6	2.9	3.6
Sow milk	1.6	1.6	7.3	4.0
Piglets				
Plasma	3.0	2.5	5.1	3.4
Heart	1.8	1.7	5.3	3.4
Liver	2.0	1.9	7.5	4.2

[1]A total of 126 sows and litters were used over 6 farrowing groups to determine the effects of supplemental vitamin E concentration and source on sow, milk, and piglet concentrations of α-tocopherol.
[2]D-α-tocopheryl acetate (RRR-α-tocopheryl acetate).
[3]DL-α-tocopheryl acetate (all-rac-α-tocopheryl acetate).
[4]Analyzed dietary concentrations of α-tocopherol for gestation diets were used to calculate bioavailability estimates based on α-tocopherol concentrations in sow plasma on d 100 of gestation, sow plasma at farrowing, and colostrum. Analyzed dietary concentrations of α-tocopherol for lactation diets were used to calculate bioavailability estimates based on α-tocopherol concentrations in sow plasma at weaning, sow milk, and piglet concentrations.

Chapter 2 - Effects of Increasing Feed Intake During Late Gestation on Sow and Litter Performance

Abstract

A total of 108 gilts and sows and their litters (PIC 327 × 1050) were used over 2 gestation and lactation periods to determine the effect of increasing late gestation feed intake on sow and litter performance. Treatments were structured as a 2 × 2 factorial with main effects of feeding allowance (0 or 0.9 kg of extra feed from d 90 to farrowing) and parity group (gilts or sows). The trial was conducted for 2 successive parities, with gilts and sows remaining on the same treatment for both parities. Increasing late gestation feed intake increased ($P < 0.001$) BW gain from d 90 to 112 in both gilts and sows. Increasing the amount of feed in late gestation resulted in gilts consuming less feed during lactation and resulted in sows consuming more feed during lactation (intake × parity interaction, $P < 0.04$).

A feed intake × parity interaction was observed ($P < 0.04$) for the average birth weight of total born and live born pigs. Increasing feeding intake in late gestation increased pig birth weight in gilts but decreased pig weight in sows. A late gestation feed intake × parity interaction was also observed ($P < 0.03$) for conception rate. Gilts that received increased late gestation feed had a greater conception rate than those offered no extra feed, whereas a decrease in conception rate was observed when parity 2 and older sows received increased late gestation feed.

During the subsequent lactation period, a feed intake × parity interaction was observed ($P < 0.02$) for lactation backfat and BW loss. This interaction was a result of an increase in backfat and BW loss in parity 2 (P2) sows as the late gestation feed intake was increased and a decrease in backfat and BW loss in parity 3 and older (P3+) sows. A late gestation feed intake × parity interaction was observed ($P < 0.01$) for average weight of both total born and live born pigs due to an increase in pig birth weight as P2 sows were supplemented with 0.9 kg of additional feed in late gestation and a slight numeric decrease in P3+ sows. Additional feed in late gestation increased ($P < 0.02$) average pig weaning weight. This trial indicates that adding 0.9 kg of feed in late gestation offered no benefit in sow performance. In gilts, individual birth weight was increased and conception rate and litter weaning weight were increased during the second parity, but no other benefits were found.

Key words: birth weight, gestation feeding, lactation, sow

Introduction

During gestation, gilts and sows are commonly fed to meet a variety of nutritional requirements. The dietary energy requirement during gestation is partitioned to meet the female's maintenance energy requirement, fetal and uterine energy requirement, as well as to achieve maternal growth (NRC, 1998). In order to achieve the nutritive needs during gestation, gilts and sows were historically fed a consistent amount of nutrients throughout gestation (Elsley et al., 1971). However, Cole et al. (1990) observed that when providing a constant nutrient level through gestation, sows can become catabolic and mobilize fat in late gestation. This period of gestation is also when the greatest amount of fetal growth occurs (McPherson et al., 2004). Depending on a gilt or sow's tissue reserves, a catabolic status in the later stages of gestation can impact her performance during lactation and potentially in subsequent parities. Increasing nutrient intake through increasing the feeding amount in late gestation has been shown to reduce the catabolic status during late gestation (Miller et al., 2000).

Birth weight has been shown to affect subsequent performance of pigs (Schinckel et al., 2007; Fix et al., 2010; Bergstrom et al., 2010). Cromwell et al. (1989) observed that sows receiving an additional 1.36 kg from d 90 of gestation through parturition had increased numbers of live born pigs and increased birth weight for pigs compared with sows receiving no added feed in late gestation. However, other research studies have shown no differences in birth weight with supplementing additional nutrients in late gestation (Pond et al., 1981; Sterling and Cline, 1986; Miller et al., 2000). Therefore, the goal of this experiment was to re-evaluate the impact of increased nutrients through increased feed amounts during late gestation on pig birth weight and sow backfat mobilization with current genetics over 2 lactation periods.

Materials and Methods

The protocol used in this experiment was approved by the K-State Institutional Animal Care and Use Committee. The study was conducted at the K-State Swine Teaching and Research Center in Manhattan, KS.

Animals and housing

A total of 108 gilts and sows (PIC 1050; Hendersonville, TN) and their litters were used in this study over 2 lactation periods. Treatments were structured as a 2 × 2 factorial with main effects of feed intake (0 or 0.9 kg of additional feed from d 90 to farrowing) and parity group

(gilts and sows). The trial was conducted for 2 successive parities. Thus, data are presented comparing gilts to sows for the first farrowing and then comparing parity 2 (P2) vs. parity 3 and greater (P3+) for the second farrowing. Treatments were allotted to gilts and sows in a generalized block design with farrowing group as the blocking factor. Four farrowing groups of approximately 27 gilts and sows were used to obtain the 108 gilts and sows used for the trial. Temperature in the farrowing facility was maintained at a minimum of 20°C, and supplemental heat was provided to the pigs with heat lamps.

First Parity

On d 35 of gestation, gilts and sows were confirmed pregnant using real-time ultrasound and designated as candidates for the study. Sows used in this study were primarily second (n=28) and third parity (n=22) with some fourth parity sows (n=15). At the time of assignment, gilts and sows were weighed and backfat thickness was measured. Backfat thickness was measured at the P2 position (last rib and approximately 65 mm off the midline) using Lean-Meater (Renco Corp.; Minneapolis, MN). Feed boxes were used to feed to gilts and sows once daily at 0800 and the boxes were calibrated to the current diet to ensure appropriate gestation feeding amounts.

Gestating feed amounts were set based on BW, backfat thickness, and predicted energy requirements. Each gilt or sow was sorted into an initial BW and backfat thickness category for determination of predicted energy requirement. Initial BW categories included 115 to 150 kg, 150 to 180 kg, 180 to 215 kg, 215 to 250 kg, and 250 to 300 kg. Initial backfat thickness categories included less than or equal to 11 mm, 12 to 14 mm, 15 to 17 mm, and greater than or equal to 18 mm. Gilts and sows were targeted to achieve a backfat thickness of 19 mm at farrowing. Increasing backfat thickness by 9, 6, 3, 0 mm required BW gains of 35.0, 27.5, 20.0, and 12.7 kg, respectively (Aherne, 1999; Young et al., 2004). Predicted energy requirements were set by summing the ME requirement for maintenance (Noblet and Etienne, 1987), ME required for maternal gain (Dourmad et al., 1996, 1997, 1998), and ME required for conceptus and uterine gain (Noblet et al., 1985). Once the total gestation ME requirement was estimated, the daily feed allowance was calculated based on the number of days remaining in gestation (80 d in first parity and 115 d in second parity) and gestation diet energy concentration (3,267 Kcal/kg).

The gestation and lactation diets were formulated to contain 0.66% and 1.10% total lysine, or 0.57% and 0.97% standardized ileal digestible lysine, respectively (Table 2-1). Diets

were formulated to meet or exceed all requirements (NRC, 1998) using nutrients profiles from the NRC (1998). For the first 3 d after farrowing, sows were gradually stepped up on feed, and after d 3, all sows were allowed ad libitum access to the lactation diet. Lactation feed disappearance was determined weekly to calculate ADFI and total feed intake for lactating sows.

On d 90, gilts and sows were weighed, backfat thickness was determined, and treatments were assigned. On d 112 of gestation, gilts and sows were weighed, backfat thickness was measured, and animals were moved to the farrowing facility. From d 112 until farrowing, gilts and sows remained on the same feeding level as offered from d 90 to 112. Upon completion of farrowing, pigs were individually weighed and processed and mummified pigs and stillbirths were recorded. From these records, the number of pigs, total weight, and average weight were calculated for total born and live born pigs. Gilts and sows were weighed and backfat thickness was determined approximately 6 to 8 h after farrowing. Cross-fostering was performed within 24 h after farrowing to standardize litter size within late gestation feeding level treatments. Total pigs, average birth weight, and total birth weight were also calculated for the pigs remaining on the sow at cross-fostering. Pigs were individually weighed at weaning to determine number weaned, average weaning weight, total litter weight, pig weight gain, pig daily weight gain, litter weight gain, and pre-weaning mortality. Gilts and sows were weighed and backfat thickness was measured at weaning.

Second parity

For the second parity, sows remained on the same late gestation feed intake treatment as the previous parity. Conception rate was calculated as number of sows confirmed pregnant on d 28 divided by number of sows bred. Gilts were then considered P2 sows and analyzed separately from P3+ sows. Gestating feed amounts were set based on sow BW and backfat thickness at weaning. Similar to the first gestation and lactation period, sow weight, backfat thickness measurements, and litter performance criteria were determined at similar days of pregnancy and lactation.

Statistical Analysis

Data were analyzed as a generalized block design with parity designation and late gestation feeding amount as fixed effects and farrowing group as a random effect. Numerical analysis of continuous responses was performed using the MIXED procedure in SAS (SAS

Institute, Inc., Cary, NC). Interactions between the fixed treatment effects and farrowing groups were pooled together with the error term because no significant interaction effects with farrowing group were detected. Conception rates were analyzed with a binomial distribution using the PROC GENMOD procedure in SAS and return to estrus was analyzed using a X^2 analysis. For all responses, sow or litter was used as the experimental unit.

Results and Discussion

For the initial gestation and lactation period, no feeding amount × parity interactions or feeding amount differences were observed ($P > 0.10$) for backfat thickness or sow weight measurements on any particular day of gestation or lactation (Table 2-2). Gilts had increased ($P < 0.001$) backfat depth on d 35, 90, and 112 of gestation and at farrowing compared with sows. A trend was also observed for gilts to have increased ($P < 0.09$) backfat at weaning in comparison to sows. Gilts also had increased ($P < 0.001$) lactation backfat loss, increased ($P < 0.001$) lactation BW loss, and decreased ($P < 0.02$) late gestation backfat gain compared with sows. Sows were heavier ($P < 0.02$) on d 35 of gestation, after farrowing, and at weaning compared with gilts. Gilts and sows that were fed 0.9 kg of extra feed in late gestation had increased ($P < 0.001$) weight gain from d 90 to 112 compared with those that did not have their feed intake increased. Also, increasing late gestation feed intake resulted in numerical improvements in backfat change from d 90 to 112 of gestation; however, the differences were not statistically different ($P > 0.10$). Cromwell et al. (1989) examined supplementing 1.36 kg of additional feed from d 90 of gestation through farrowing and observed similar increases in gilt and sow BW. The diet used by Cromwell et al. (1989) had similar ME concentrations to the current experiment. Therefore, they would have supplemented a greater energy quantity in late gestation than in the current experiment. Miller et al. (2000) also observed increases in sow BW and backfat thickness gain during late gestation with increased feed amounts. It should also be noted that sow feeding levels were set in order to achieve a BF level at farrowing of approximately 19 mm. Gilts began above the threshold and farrowed relatively close to our target; however, sow BF levels only changed slightly from d 35 and did not reach the target.

For the initial lactation, feed intake× parity interactions were observed ($P < 0.04$) for ADFI and total feed intake for each week in lactation, as well as for the overall lactation period (Table 2-3). This interaction was due to an increase in lactation feed intake when sow intake was

increased in late gestation and a decrease in lactation feed intake when gilt intake was increased in late gestation. The interaction was of greater magnitude in wk 1 than in other weeks. Sows had greater ($P < 0.001$) ADFI each week and greater lactation feed intake during wk 2 and 3 and overall than gilts. Miller et al. (2000) and Cromwell et al. (1989) observed no differences in lactation feed intake with increased late gestation feeding amounts. In the present study, gilts had significantly greater backfat than sows which may have been one of the reasons for the reduction in lactation feed intake. Several researchers have demonstrated the negative relationship in gilts between elevated body fat composition and lactation feed intake (Dourmad, 1991; Revell et al., 1998a; Weldon et al., 1994a). Weldon et al. (1994a) demonstrated that gilts that were overfed in late gestation had lower lactation feed intake due to fewer daily meals and no difference in meal size. Weldon et al. (1994a, b) also demonstrated that gilts that were overfed in late gestation had reduced sensitivity to insulin and lower peripheral glucose utilization. This would suggest that the reduction in lactation feed intake for gilts in our study was due to excess feeding in late gestation which resulted in reduced insulin sensitivity.

For litter performance during the first lactation period, a feeding amount × parity interaction was observed ($P < 0.04$) for average weight of total born and live born pigs (Table 2-4). Increased late gestation feed intake led to increased pig birth weight in gilt litters and decreased pig weight in sow litters. Gilts also had increased ($P < 0.02$) number and total weight of the total born, live born, and number after fostering and had an increased ($P < 0.05$) percentage of mummified pigs compared with sows. No difference was observed ($P > 0.10$) in the percentage of stillbirths. In similar research, Pond et al. (1980) observed no increases in pig birth weight when doubling energy intake for the last 14 d of gestation in multiparous sows. Also, similar to the present study, Cromwell et al. (1989) observed no improvements in pig birth weight with supplementation of additional feed in late gestation to gilts or multiparous sows for the first cycle. Soto et al. (2011) observed increased pig birth when sows were fed 1.82 kg of additional feed from d 100 to 114 of gestation as compared with sows fed 0 or 0.91 kg of additional feed in late gestation. These three published studies made no reference to the body composition or condition of the animals used which could be an important factor in interpreting the results of each study.

Gilts weaned larger ($P < 0.002$) litters and had increased ($P < 0.03$) total litter weaning weight and litter weight gain comparison with sows. However, providing gilts and sows with

extra feed in late gestation feed offered no benefit ($P > 0.10$) in number weaned, weaning weight, pig weight gain, or litter weight gain compared with maintaining a constant gestation feeding amount. Sow and litter gain also increased ($P < 0.03$) in sows as compared to gilts. The increase in pig birth weights for gilts fed additional feed in late gestation was not maintained throughout lactation. This could simply be due to lowered milk production from reduced lactation feed intake in gilts that were fed additional feed in late gestation. It could also be a function of impaired mammary development. Weldon et al. (1991) showed that feeding excess energy in late gestation was detrimental to the mammary development of gilts. In addition, Revell et al. (1998b) observed that leaner gilts at parturition produced approximately 15% additional milk yield than their over conditioned counterparts.

Upon weaning, sows had decreased ($P < 0.002$) days to estrus than gilts, and sows fed increased amounts of feed during late gestation showed a trend for lower ($P < 0.10$) wean to breed intervals compared with sows maintained on no extra feed. A late gestation feed intake × parity interaction was detected ($P < 0.03$) for conception rate. Gilts that received increased feed in late gestation had a greater conception rate than those given no extra feed, whereas a decrease in conception rate was observed when sows received extra feed in late gestation. The response in conception rates for gilts supplemented with additional feed may be reflective of improved sow metabolic status prior to farrowing. Follicles that ovulate after weaning may first emerge during the late gestation stage and if sows are in a catabolic state the integrity of emerging follicles may be reduced (Foxcroft et al., 1995).

All females remained on the original late gestation feeding amount treatment for the subsequent gestation and lactation period. The sharp difference in conception rate of gilts between different late gestation feeding intakes generated a substantial difference in number of gilts that could be used for subsequent performance (Table 2-5). For the subsequent gestation and lactation period, no differences in sow weight and backfat thickness were observed ($P > 0.10$) between late gestation feeding amounts or parity. A feed intake × parity interaction was observed ($P < 0.005$) for lactation backfat and sow BW loss. This interaction was reflective of an increase in backfat and sow BW loss in P2 sows as the late gestation feed intake was increased and a decrease in backfat and sow BW loss in P3+ sows with increasing late gestation feed intake. In addition, P3+ sows were heavier ($P < 0.02$) at farrowing and at weaning that P2 sows. Cromwell et al. (1989), Miller et al. (2000), and the first parity in this study observed that

increasing the late gestation feeding amount resulted in increased ($P < 0.001$) sow BW gain from d 90 to 112 of gestation for both P2 and P3+ sows.

For subsequent lactation feed intake, no interactions or feeding amount differences were observed ($P > 0.10$) for total or daily sow feed intake (Table 2-6). In addition, P2 sows had decreased ($P < 0.05$) total and daily feed intake for wk 1 compared with P3+ sows and tended to have decreased ($P < 0.09$) overall total and daily lactation feed intake.

For the subsequent lactation, P2 sows had increased ($P < 0.006$) pig numbers for total born, live born, and at weaning compared with P3+ sows (Table 2-7). Parity 2 sows also increased ($P < 0.03$) average pig birth weight for total born, live born, and cross-fostered pigs and increased ($P < 0.004$) litter weight for total born, live born, cross-fostered, and at weaning compared with P3+ sows. These changes also allowed for P2 sows to have greater ($P < 0.02$) daily and total litter weight gain as compared to P3+ sows. A late gestation feed intake × parity interaction was observed ($P < 0.01$) for average weight of both total born and live born pigs, and a similar trend was observed ($P < 0.07$) at cross-fostering. These interactions were reflective of increased pig birth weight as P2 sows were fed the additional 0.9 kg of feed in late gestation, and a slight numeric decrease in P3+ sows. The cause of this increase in average weight could be related to the numeric decrease in the number of pigs born. Despite the interaction, providing additional feed in late gestation tended to increase ($P < 0.07$) average pig weight for total born, live born, and those remaining at cross-fostering. Average pig weight at weaning also increased ($P < 0.02$) with supplementation of additional feed in gestation, with a large improvement observed in P2 sows. Pre-weaning mortality, sow and litter gain were unaffected ($P > 0.10$) by increasing late gestation feed intake. The increased birth weight and weaning weight of pigs from P2 sows fed more in late gestation would be in agreement with Cromwell et al. (1989) in that the response to additional late gestation feed is achieved after multiple parties.

Other researchers have evaluated improving sow performance by changing specific nutrient concentrations in the late gestation diet. Heo et al. (2008) examined whether the lysine or energy quantity in the diet would impact pig birth weights of gilt litters. Dietary energy concentration did not impact birth weight; however, they observed an increase in pig birth weight when sows were fed a diet containing 0.80% total lysine compared with 0.60% total lysine during late gestation. The low lysine level of 0.60% fed by Heo et al. (2008) would be similar to the 0.66% lysine in our gestation diet; however, due to differences in feed allowance,

gilts on their low lysine treatment would have consumed approximately 30% more lysine on a per day basis than gilts in our study that did not receive extra feed in late gestation. This suggests that the positive responses observed with increasing the late gestation feeding amount in gilts may be a function of lysine intake as compared to energy intake. In addition, it has been demonstrated that the amino acid requirements of gilts and sows vary in different stages of gestation (Levesque et al., 2011; Samuel et al., 2012). Adding extra feed in late gestation may a means by meet the sow's amino acid requirements on a daily basis instead of utilizing a different diet formulation in late gestation.

It is interesting to note that although gestation feeding amounts were set to achieve a maternal backfat thickness of 19 mm, the mean backfat thickness of sows did not reach 19 mm target at farrowing. Young et al. (2004) validated the use of this method for feeding sows in gestation compared with feeding to a body condition score; however, they also observed a high proportion of sows that did not reach their target backfat gain. The ability to reach the target backfat thickness gain or BW gain appears to be reduced in older parity sows (Cooper et al., 2001).

Overall, there was benefit to increasing late gestation feed intake for older sows. In gilts, increasing late gestation feed by 0.9 kg increased pig birth weight, improved conception rate, and increased litter weaning weight during the subsequent parity, but no other benefits were found.

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Table 2-1 Composition of diets (as-fed basis)

Item	Gestation	Lactation
Ingredient, %		
Corn	80.75	65.28
Soybean meal (46.5% CP)	14.95	30.80
Monocalcium P (21% P)	1.70	1.45
Limestone	1.35	1.20
Salt	0.50	0.50
Vitamin premix[1]	0.25	0.25
Mineral premix[2]	0.15	0.15
Sow add pack[1,2]	0.25	0.25
Phytase[3]	0.10	0.10
Total	100	100
Calculated analysis		
SID[4] amino acids, %		
Lys	0.57	0.97
Thr	0.43	0.65
Met	0.21	0.29
Trp	0.13	0.21
Ile	0.48	0.75
Leu	1.22	1.60
Val	0.57	0.49
ME, kcal/kg	3,267	3,273
CP, %	13.8	19.9
Total Lys, %	0.66	1.10
Ca, %	0.90	0.85
P, %	0.69	0.70
Available P, %[5]	0.52	0.48

[1]Vitamin premix and sow add pack provided per kg of diet: 11,022 IU of vitamin A, 1,378 IU of vitamin D_3, 66.14 IU of vitamin E, 4.41 mg of vitamin K, 0.04 mg of vitamin B_{12}, 49.60 mg of niacin, 27.56 mg of pantothenic acid, 8.26 mg of riboflavin, 0.22 g of biotin, 1.65 mg of folic acid, 4.96 mg of pyridoxine, 551.14 mg of choline, and 49.6 mg of carnitine.

[2]Trace mineral premix and sow add pack provided per kg of diet: 16.5 mg Cu from $CuSO_4$, 200 µg of Cr from $Cr(C_6H_4NO_2)_3$,0.30 mg I from $Ca(IO_3)_2$, 165 mg Fe from $FeSO_4$, 40 mg Mn from $MnSO_4$, 0.305 mg Se from $NaSeO_3$, and 165 mg Zn from $ZnSO_4$.

[3]Phyzyme 600 (Danisco Animal Nutrition, St Louis, MO) provided 600 FTU/ kg of diet.

[4]Standardized ileal digestible.

[5]Phytase provided 0.11% and 0.10% available P to the gestation and lactation diets, respectively.

Table 2-2 Effects of late gestation feed intake and parity designation on sow weight and backfat[1]

	Gilt		Sow			Probability, *P* <		
Late gestation feed intake[2]:	Normal	+ 0.9 kg	Normal	+ 0.9 kg	SED	Intake × parity	Parity	Intake
n	22	21	33	32	---	---	---	---
Gestation length, d	114.9	115.4	115.5	116.0	---	---	---	---
Lactation length, d	20.8	20.6	19.9	19.4	---	---	---	---
Backfat measurements, mm[3]								
Gestation d 35	20.0	20.1	13.5	13.7	0.78	0.94	0.001	0.83
Gestation d 90	20.3	20.4	14.9	14.9	0.91	0.96	0.001	0.93
Gestation d 112	19.0	19.9	14.9	15.3	0.77	0.70	0.001	0.39
Farrowing	18.4	18.7	14.8	15.4	0.69	0.77	0.001	0.51
Weaning	15.1	14.5	13.4	13.9	0.75	0.38	0.09	0.94
Backfat change, mm								
d 90 to 112	-1.3	-0.5	0.0	0.3	0.62	0.57	0.02	0.18
Farrowing to weaning	-3.4	-4.3	-1.3	-1.4	0.57	0.30	0.001	0.22
Sow BW, kg								
Gestation d 35	188.6	187.2	196.4	197.2	5.18	0.76	0.02	0.94
Gestation d 90	225.4	226.0	229.6	228.9	6.06	0.89	0.40	0.99
Gestation d 112	239.9	245.8	245.8	250.2	6.01	0.87	0.27	0.25
Farrowing	220.2	222.8	235.9	239.4	5.70	0.92	0.001	0.44
Weaning	206.6	204.1	228.2	232.4	6.58	0.40	0.001	0.83
Weight change, kg								
d 90 to 112	14.6	19.9	16.1	21.1	2.09	0.92	0.36	0.001
Farrowing to weaning	-13.7	-18.7	-7.6	-6.9	2.17	0.12	0.001	0.23

[1]A total of 108 gilts and sows (PIC 1050) were used to determine the effect of increasing feed intake in late gestation.
[2]Late gestation intakes were set at d 90 of gestation. Normal = the same amount as designated at d 35 by BW and last rib backfat; +0.9 kg = 0.9 kg more than the d 35 amount.
[3]Backfat measurements were determined by averaging both sides at the last rib approximately 65 mm off the midline.

Table 2-3 Effects of late gestation feed intake and parity designation on lactation feed intake[1]

	Gilt		Sow			Probability, *P* <		
Late gestation feed intake[2]:	Normal	+ 0.9 kg	Normal	+ 0.9 kg	SED	Intake × parity	Parity	Intake
n	22	21	33	32	---	---	---	---
Gestation d 35 feed amount, kg/d	2.1	2.0	2.6	2.6	---	---	---	---
Gestation d 90 feed amount, kg/d	2.1	2.9	2.6	3.5	---	---	---	---
Lactation ADFI, kg								
wk 1	4.49	3.10	4.83	5.26	0.404	0.001	0.001	0.03
wk 2	5.50	4.77	6.23	6.39	0.210	0.007	0.001	0.09
wk 3	6.01	5.51	6.34	6.57	0.367	0.04	0.001	0.43
Overall	5.33	4.53	5.84	6.13	0.224	0.001	0.001	0.10
Lactation total intake, kg								
wk 1	29.9	21.7	27.9	28.5	2.21	0.02	0.17	0.03
wk 2	38.5	33.4	43.6	44.8	1.47	0.007	0.001	0.09
wk 3	42.1	38.5	44.4	46.0	2.57	0.04	0.001	0.43
Overall	110.6	94.0	115.7	119.0	4.25	0.004	0.001	0.06

[1]A total of 108 gilts and sows (PIC 1050) were used to determine the effect of increasing feed intake in late gestation.
[2]Late gestation intakes were set at d 90 of gestation. Normal = the same amount as designated at d 35 by BW and last rib backfat; + 0.9 kg = 0.9 kg more than the d 35 amount.

Table 2-4 Effects of late gestation feed intake and parity designation on pig performance[1]

	Gilt		Sow			Probability, $P <$		
Late gestation feed intake[2]:	Normal	+ 0.9 kg	Normal	+ 0.9 kg	SED	Intake × parity	Parity	Intake
n	22	21	33	32	---	---	---	---
Litter size, n								
Total born[3]	14.6	14.0	11.9	12.9	0.82	0.20	0.004	0.70
Live born	13.8	12.9	11.2	12.3	0.73	0.13	0.02	0.82
Cross-fostering[4]	12.5	12.4	11.2	11.5	0.34	0.58	0.001	0.63
Weaning	11.5	11.5	10.6	10.5	0.32	0.91	0.002	0.98
Total litter weight, kg								
Total born[3]	20.1	19.8	17.4	17.7	0.92	0.74	0.004	0.99
Live born	19.5	19.1	16.7	17.0	0.88	0.67	0.002	0.96
Cross-fostering[4]	18.2	18.3	16.6	16.6	0.44	0.79	0.001	0.89
Weaning	69.2	69.7	64.2	63.2	2.09	0.69	0.003	0.89
Average pig weight, kg								
Total born[3]	1.41	1.49	1.53	1.42	0.059	0.04	0.55	0.80
Live born	1.42	1.50	1.54	1.43	0.057	0.04	0.67	0.78
Cross-fostering[4]	1.46	1.48	1.49	1.44	0.032	0.18	0.93	0.53
Weaning	6.1	6.1	6.1	6.0	0.14	0.82	0.98	0.70
Mummies, %	1.86	3.95	1.25	0.84	1.075	0.18	0.05	0.36
Stillbirths, %	3.40	3.35	4.53	4.25	1.538	0.93	0.40	0.89
Litter wt gain, kg								
Daily	2.5	2.5	2.4	2.4	0.10	0.99	0.36	0.83
Overall	51.1	51.4	47.6	46.7	2.00	0.72	0.03	0.86
Pre-weaning mortality	7.35	7.05	5.65	8.28	2.117	0.40	0.90	0.50
Sow and litter wt gain, kg[5]	37.4	32.6	40.0	39.8	3.02	0.28	0.03	0.23
Subsequent performance								
Wean to breed, d	5.15	4.71	4.47	4.40	0.171	0.24	0.002	0.10
Conception rate, %	77.27	95.24	96.97	87.50	6.521	0.03	0.32	0.48

[1]A total of 108 gilts and sows (PIC 1050) were used to determine the effect of increasing feed intake in late gestation.
[2]Late gestation intakes were set at d 90 of gestation. Normal = the same amount as designated at d 35 by BW and last rib backfat; +0.9 kg = 0.9 kg more than the d 35 amount.
[3]Weights of total born reflect only pigs born alive or stillbirths and not mummified pigs.
[4]Cross-fostering weights reflect the total and mean birth weights of pigs that survived until fostering, which occurred at approximately 24 h.
[5]Sow and litter wt gain = (sow weaning wt - sow farrowing wt) + (litter wt gain).

Table 2-5 Effects of late gestation feed intake and parity designation on sow weight and backfat of subsequent performance[1]

	Parity 2		Parity 3+			Probability, P <		
Late gestation feed intake[2]:	Normal	+ 0.9 kg	Normal	+ 0.9 kg	SED	Intake × parity	Parity	Intake
n	14	19	26	25	---	---	---	---
Gestation length, d	115.9	115.9	115.8	116.3	---	---	---	---
Lactation length, d	19.2	19.5	19.8	19.4	---	---	---	---
Backfat measurements, mm[3]								
Gestation d 90	15.4	16.5	14.7	15.5	1.32	0.88	0.32	0.25
Gestation d 112	15.2	16.8	15.0	16.1	1.34	0.77	0.63	0.12
Farrowing	14.8	16.2	14.9	15.8	1.35	0.79	0.87	0.20
Weaning	14.4	14.4	13.7	15.5	1.25	0.22	0.90	0.27
Backfat change, mm								
d 90 to 112	-0.2	0.3	0.3	0.6	0.48	0.78	0.29	0.31
Farrowing to weaning	-0.5	-1.9	-1.1	-0.1	0.67	0.005	0.21	0.58
Sow BW, kg								
Gestation d 90	223.5	231.4	235.9	239.7	8.62	0.72	0.08	0.30
Gestation d 112	248.1	256.7	254.4	262.1	9.90	0.95	0.35	0.19
Farrowing	234.4	241.9	250.0	254.9	9.88	0.82	0.02	0.29
Weaning	228.8	227.5	241.1	249.2	9.05	0.40	0.003	0.54
Weight changes, kg								
d 90 to 112	18.2	25.1	18.3	22.6	1.62	0.20	0.27	0.001
Farrowing to weaning	-5.3	-14.3	-7.3	-5.7	2.42	0.02	0.12	0.08

[1]A total of 84 of the original 108 gilts and sows (PIC 1050) were used to determine the effects of late gestation sow intake on a subsequent lactation period.

[2]Late gestation feeding treatments were set at d 90 of gestation. Normal = the same amount as designated at breeding; 0.9 kg = 0.9 kg higher than that particular amount.

[3]Backfat measurements were determined by averaging both sides at the last rib approximately 65 mm off the midline.

Table 2-6 Effects of late gestation intake and parity designation on lactation feed intake of subsequent farrowing[1]

	Parity 2		Parity 3+			Probability, *P* <		
Late gestation intake[2]:	Normal	+ 0.9 kg	Normal	+ 0.9 kg	SED	Intake × parity	Parity	Intake
n	14	19	26	25	---	---	---	---
Gestation d 0 feed amount, kg/d	2.6	2.5	2.6	2.6	---	---	---	---
Gestation d 90 feed amount, kg/d	2.6	3.4	2.6	3.5	---	---	---	---
Lactation ADFI, kg								
wk 1	5.1	5.2	5.3	5.9	0.30	0.30	0.05	0.18
wk 2	6.4	6.1	6.2	6.6	0.25	0.13	0.46	0.96
wk 3	7.2	6.8	7.3	7.5	0.39	0.21	0.14	0.78
Overall	6.3	6.1	6.3	6.7	0.23	0.10	0.09	0.73
Lactation total intake, kg								
wk 1	26.2	28.1	31.7	32.5	2.60	0.81	0.03	0.54
wk 2	44.9	42.7	43.7	46.0	1.74	0.13	0.46	0.96
wk 3	50.5	47.6	50.9	52.8	2.74	0.21	0.14	0.78
Overall	121.4	118.5	126.2	131.2	5.34	0.39	0.06	0.82

[1]A total of 84 of the original 108 gilts and sows (PIC 1050) were used to determine the effects of late gestation sow intake on a subsequent lactation period.

[2]Late gestation feeding treatments were set at d 90 of gestation. Normal = the same amount as designated at breeding; + 0.9 kg = 0.9 kg higher than that particular amount.

Table 2-7 Effects of late gestation intake and parity designation on pig performance in a subsequent litter[1]

	Parity 2		Parity 3+			Probability, *P* <		
Late gestation intake[2]:	Normal	+ 0.9 kg	Normal	+ 0.9 kg	SED	Intake × parity	Parity	Intake
n	14	19	26	25	---	---	---	---
Litter size, n								
Total born[3]	15.1	13.5	12.3	12.2	0.89	0.29	0.006	0.28
Live born	14.0	12.7	11.2	11.4	1.07	0.27	0.004	0.42
Cross-fostering[4]	12.0	11.8	11.1	11.4	0.55	0.57	0.08	0.87
Weaning	11.2	11.2	10.2	10.1	0.56	0.81	0.004	0.82
Total litter weight, kg								
Total born[3]	21.4	21.9	16.6	16.9	1.05	0.87	0.001	0.65
Live born	20.2	21.1	15.7	16.0	1.09	0.75	0.001	0.53
Cross-fostering[4]	17.8	19.5	16.0	16.6	0.75	0.39	0.001	0.07
Weaning	66.6	74.0	61.8	62.8	3.56	0.22	0.004	0.11
Average pig weight, kg								
Total born[3]	1.44	1.67	1.44	1.40	0.066	0.01	0.02	0.07
Live born	1.44	1.68	1.46	1.42	0.066	0.01	0.03	0.05
Cross-fostering[4]	1.49	1.65	1.46	1.46	0.051	0.07	0.009	0.06
Weaning	5.92	6.59	6.11	6.26	0.200	0.14	0.67	0.02
Mummies, %	0.94	1.26	1.71	0.77	0.796	0.35	0.84	0.65
Stillbirths, %	6.60	4.26	6.07	6.18	1.960	0.46	0.68	0.50
Litter wt gain, kg								
Daily	2.5	2.8	2.3	2.4	0.14	0.38	0.004	0.12
Overall	48.8	54.5	45.8	46.1	3.19	0.24	0.02	0.19
Pre-weaning mortality, %	6.09	5.16	7.26	11.02	3.500	0.30	0.13	0.53
Sow and litter gain, lb[5]	43.1	39.8	38.6	40.7	3.10	0.31	0.48	0.82

[1]A total of 84 of the original 108 gilts and sows (PIC 1050) were used to determine the effects of late gestation sow intake on a subsequent lactation period.

[2]Late gestation feeding treatments were set at d 90 of gestation. Normal = the same amount as designated at breeding; + 0.9 kg = 0.9 kg higher than that particular amount.

[3]Weights of total born reflect only pigs born alive or stillbirths and not mummified pigs.

[4]Cross-fostering weights reflect the total and mean birth weights of pigs that survived until fostering, which occurred at approximately 24 h.

[5]Sow and litter gain during lactation = (sow weaning wt – sow farrowing wt) + litter wt gain.

Chapter 3 - Effects of dietary L-carnitine and chromium picolinate on sow reproductive performance

Abstract

A total of 211 sows and litters (PIC) were used to compare the effects adding L-carnitine (Carn) and chromium picolinate (CrPic) on sow reproductive performance. Two dietary treatments were used including a control with no added Carn or CrPic and a test diet with 25 mg/kg of added Carn and 200 μg/kg of added Cr from CrPic. Gilts and sows received treatment diets for at least 24 d before breeding in either gilt development or in lactation. Females remained on treatment concentrations of Carn and CrPic throughout loading, gestation, lactation, and up until breeding for the subsequent parity.

Sows fed diets with Carn and CrPic had greater ($P = 0.02$) BW at breeding compared with sows not fed Carn and CrPic. Sows fed diets with Carn and CrPic had decreased ($P < 0.01$) gestation weight gain and lactation weight loss as compared to sows fed diets with no Carn and CrPic. A trend for an interaction between parity and maternal treatment was observed ($P = 0.06$) for BCS. This interaction was reflective of a decrease in sow BCS when Carn and CrPic were added to the diets of gilts and sows bred to carry their 2nd litter and an increase in sow BCS when Carn and CrPic were added to the diets of parity 3 and older (P3+) sows. Sows fed diets with Carn and CrPic had lower ($P < 0.03$) BCS at farrowing and weaning in comparison with those receiving diets with no added Carn and CrPic. Sows fed diets containing Carn and CrPic had increased ($P < 0.02$) lactation feed intake compared with sow consuming diets without Carn and CrPic.

Interactive responses between parity and maternal treatment were observed ($P < 0.04$) for litter size and weight at d 7 of lactation. Gilts and parity 2 sows fed Carn and CrPic nursed larger litters on d 7 of lactation compared to those receiving no Carn and CrPic, while no differences were observed in P3+ sows. Gilts and parity 2 sows fed diets with added Carn and CrPic had increased litter weight on d 7 of lactation as compared to those fed diets without Carn and CrPic and P3+ sows fed diets with Carn and CrPic had decreased litter weights as compared to those that were fed diets without Carn and CrPic.

Farrowing rate, total born, and born alive on the subsequent performance were unaffected ($P > 0.10$) by adding Carn and CrPic to sow diets. This experiment showed little differences in sow performance with the addition of Carn and CrPic. Overall, adding Carn and CrPic to sow diets did not impact litter size, pig birth weight, or the amount of variation in pig birth weight.

Key words: birth weight, L-carnitine, chromium, litter size, sow

Introduction

Carnitine is a water soluble compound that serves as a cofactor in the transport of fatty acids across the mitochondrial membrane for energy production through β-oxidation. Supplementing sow gestation diets with L-carnitine (Carn) has been shown to increase litter size (Ramanau et al., 2004; Birkenfeld et al., 2005); however, that response has not been consistent (Musser et al., 1999b; Real et al., 2008). Adding Carn to sow diets has also been shown to increase neonatal pig birth weight and improve nutrient utilization (Musser et al., 1999b; Eder et al., 2001). The mechanisms associated with improved litter performance and nutrient utilization have not been completely explained; however adding dietary Carn has been shown to increase sow plasma concentrations of IGF-I during mid-gestation (Musser et al., 1999b; Doberenz et al., 2006), increase plasma leptin concentrations during gestation (Woodworth et al., 2004), and decrease messenger RNA for IGF-II and myogenin in porcine embryonic muscle cells (Waylan et al., 2005).

Chromium is a trace mineral that impacts insulin sensitivity and results in improved intracellular carbohydrate and lipid metabolism (Lindemann et al., 1995). Adding dietary Cr from chromium picolinate (CrPic) has been shown to increase litter size in several studies (Lindemann et al., 1995; Hagen et al., 2000). Similar to Carn, the response in litter size with CrPic supplementation has not been consistent (Campbell, 1998; Real et al., 2008). Combining both Carn and CrPic has been shown to improve farrowing rate and, thus, increase sow productively (Real et al., 2008). Some of the trials that compared adding Carn and/or CrPic had limited sow numbers which will impact the ability to detect differences in sow performance. Therefore, the goal of this experiment was to evaluate the combination Carn and CrPic in sow diets on a commercial sow farm where adequate sow numbers can be achieved, and focus on comparing litter size, pig birth weight, and variation in birth weight.

Materials and Methods

The protocol used in this experiment was approved by the Kansas State University Institutional Animal Care and Use Committee. The study was conducted at a commercial sow farm in western Illinois.

Animals and feeding

A total of 211 gilts and sows (PIC 1050, C22, and C29; Hendersonville, TN) and their litters were used to evaluate the effects of dietary Carn and CrPic on sow and litter performance. Gilts and first parity sows were all PIC line 1050. Second parity and older sows were one of the following PIC lines: 1050, C22, or C29. The two dietary treatments were a control with no added Carn or CrPic and a test diet formulated to contain 25 mg/kg of Carn and 200 μg/kg of Cr from CrPic. Gilts and sows were allotted to treatments in a generalized block design with parity serving as the blocking factor. Genetic lines were also balanced between maternal treatments. The trial took place between August 2011 and May 2012 with sows farrowing in December 2011 for the initial period and May 2012 for the subsequent period.

Prior to beginning the experiment, all gilts and sows were fed diets that contained no supplemental Carn or CrPic. Sows were allotted to dietary treatments for a loading period, at least 24 d, before the initial gestation period began. Sows were allotted at arrival into the farrowing rooms and remained on test diets through the lactation period and pre-breeding. Gilts expected to be in estrus during the breeding period were allotted during gilt development. All females were fed ad libitum during this loading period with sows receiving the pre-trial lactation diet and gilts receiving the gestation diet. Upon artificial insemination (PIC line 327), gilts and sows were moved into gestation stalls and segregated by parity groups (as per farm protocol) and dietary treatments. Sows were fed in gestations crates once daily at 0430 by dropping feed into a continuous feed trough. Females were group together by treatment on different feed lines to prevent any cross contamination of diets.

All females remained in gestation stalls until pregnancy was confirmed on d 35 of gestation. After pregnancy was confirmed, static groups of females were moved into pens for the remainder of gestation with approximately 1.57 m^2 of floor space allowed per sow. Groups were segregated by dietary treatment to prevent any cross contamination of feed as well as by parity to reduce the amount of variation in size differences and limit aggressive behavior. Gilts and first

parity sows were grouped together and all older sows were grouped together. Feeding in pens was accomplished by the NEDAP electronic sow feeder (Velos, Netherlands). While in gestation pens, sows were allowed to consume their daily feed allowance throughout the day as they traveled through the feeding station. Sows that needed to be removed from pens due to health, lameness, or poor body condition were removed from the study with eleven and eight females being removed from the control and test groups, respectively.

Feeding amounts throughout gestation were set based on parity as well as BCS. For gilts and sows in ideal body condition, the maximum feeding amounts were set at 2.05 and 2.27 kg, respectively, up until d 100 of gestation. From d 100 of gestation until sows were moved into farrowing rooms, maximum feed allowances for gilts and sows in ideal body condition were set at 3.25 and 3.15 kg, respectively. Adjustments were also made based on body condition score. Females considered thin or very thin were given 20% and 40% more feed, respectively, than the amount for a sow in ideal body condition. Females considered slightly fleshy were restricted to 10% less than an ideal sow would be allowed, and gilts and sows considered fat were restricted to 20% and 25% less than an ideal sow would be allowed, respectively. Body condition was evaluated at mating, as gestation pens were loaded, every 14-d while in pens, at farrowing, and at weaning. The amount of gestation feed dropped by the feeding stations was recorded for part of the time while sows were in pens. Unexpectedly, technical difficulties across the entire farm made recording the true late gestation feed intake impossible. Therefore, in order to estimate the amount of gestation feed intake, it was assumed that each sow consumed her maximum allotment each day when actual amounts were not recorded.

Gilts and sows were moved to farrowing rooms on d 112 of gestation. Farrowing rooms consisted of 56 crates. Maternal treatments were balanced within farrowing rooms so that each treatment had equal representation within each room. Two of the farrowing rooms were equipped with an electronic feed drop system (Howema; Holland, MI) and the remaining rooms were equipped with SowMax feeders (AP; Assumption, IL) and were hand fed. Sows were fed once daily throughout lactation. Prior to farrowing, sows received 1.81 kg of lactation feed. Upon farrowing, sow feed allowance was gradually increased at 1.81, 2.72, and 3.63 kg for the first 3-d. Sow feed intake was recorded throughout lactation and daily feed allowances were limited to 6.12 kg per d for all sows. Upon weaning, sows remained on treatment lactation diets until rebred.

At birth, individual pig weights were obtained. Cross fostering occurred daily at 1300- and pigs were cross fostered within dietary treatment within 24 h of birth. Functional teats were counted for each sow and litters were fostered up to the number of functional teats available. Litters were evaluated daily for fall behind pigs and those pigs were removed and placed on nurse sows. At the time of the trial, this farm was experiencing a higher prevalence of scours in the farrowing house, leading to increases in pig mortality and removal rate. Litter weights were taken on d 7 and individual pig weights were recorded on d 18. An estimated litter weaning weight was determined by calculating daily litter growth rate from d 7 to 18 of lactation and using that value to extrapolate from d 17 until weaning.

The compositions of the basal diets used in this trial are shown in Table 3-1. The pre-trial lactation, gestation, and trial lactation diets were formulated to contain 1.35%, 0.66%, and 1.25% total lysine, or 1.17%, 0.51%, and 1.07% standardized ileal digestible lysine, respectively. Gestation and lactation diets were also formulated to contain 40% and 30% dried distiller's grains with solubles (DDGS), respectively. Diets were formulated to meet or exceed all of the sow's requirements (NRC, 1998) using ingredient profiles from the NRC (1998). A portion of corn was mixed with Carn and CrPic (DSM; Parsippany, New Jersey) to make a more dilute premix that could be added at the expense of corn to generate the Carn and CrPic diet. Diet samples were obtained weekly in gestation and from each 3 ton batch of feed in lactation. At the conclusion of the trial, composite samples for each dietary treatment were generated for the following categories: pre-trial lactation, gestation, and lactation. Diet samples from each treatment and category underwent analysis for Carn using a radioisotopic enzymatic method (Parvin and Pande, 1977) and analysis for Cr using atomic absorption spectroscopy (AOAC, 2000).

Statistical Analysis

Sow and litter performance were analyzed using the PROC MIXED procedure of SAS (SAS Institute Inc., Cary, NC). Main and interactive effects for parity and maternal diets were determined by the overall treatment F-test. Due to the low sample size, gilts and parity 2 sows were combined for analysis and P3+ sows were grouped. Sow was used as the experimental unit for all data analysis.

Categorical responses for BCS were given numerical scores of 1 to 5 for very thin, thin, ideal, fleshy, and fat sows, respectively. The numerical scores were then analyzed. Tests for normality using the PROC UNIVARIATE procedure in SAS suggested that the values did not depart from normality. Return to estrus was analyzed using a X^2 analysis and farrowing rate was analyzed as binomial response using the PROC GENMOD procedure of SAS.

In addition to looking at birth weight with litter performance, birth weights of all pigs (total born) were examined separately. For this analysis, individual pig was used as the experimental unit. A means model for the birth weight of pigs from control or Carn and CrPic fed sows was developed using the MIXED procedure of SAS. Parity was not included in this model. Separate variances were estimated for pigs from control and Carn and CrPic fed sows. The variance estimates were compared using Hartley's F-Max test. The comparison of the variance estimates used to test for differences in the amount of variation in pig birth weights from sows fed control or Carn and CrPic diets. All statistics were considered significant at P values < 0.05 and were considered tendencies at P values < 0.10.

Results

Diet samples for both control and test diets were assayed for proximate analysis, Cr, and Carn. The proximate analysis results were similar for both control and test diets within both the gestation and lactation phases (data not shown). The analyzed concentrations of Carn and Cr in each treatment's gestation and lactation diets are shown in Table 3-2. While there was some variation, the test diets appear to contain approximately 25 mg/kg more Carn and 200 μg/kg more Cr than the control diets.

No interactions between maternal treatment and parity were observed ($P > 0.10$) for sow BW, BW change, or lactation feed intake (Table 3-3). Unexpectedly, sows fed diets with Carn and CrPic had greater ($P = 0.02$) BW at breeding compared with sows not fed Carn and CrPic. This was driven primarily by the increase in BW from older sows randomly allotted to the Carn and CrPic treatment. Sow BW was not affected ($P > 0.10$) by adding Carn and CrPic to maternal diets on d 35 of gestation, d 112 of gestation, weaning, as well for estimated post-farrowing BW. Parity 3 and older sows had greater ($P = 0.001$) BW at breeding, d 35 of gestation, d 112 of gestation, post-farrow, and weaning as compared with gilts and parity 2 sows. Sows fed diets with Carn and CrPic had decreased ($P < 0.01$) gestation weight gain and lactation weight loss as

compared to sows fed diets with no added Carn and CrPic. Gilts and parity 2 sows had greater ($P = 0.001$) gestation weight gain and lactation weight loss as compared to P3+ sows.

Sow body condition scores at the beginning of the pre-trial lactation were similar ($P > 0.10$) for sows consuming diets with and without Carn and CrPic. Gilts that would be in their second parity for the trial period also had reduced ($P < 0.003$) BCS at the beginning of the loading period compared with older sows. A trend for an interaction between parity and maternal treatment was observed ($P = 0.06$) for breeding BCS. This interaction is reflective of a decrease in sow BCS when Carn and CrPic were added to the diets of gilts and sows bred to carry their 2nd litter and an increase in sow BCS when Carn and CrPic were added to the diets of P3+ sows. Gilts and sows bred to carry their 2nd litter had lower BCS ($P < 0.03$) scores at the breeding compared with P3+ sows. No differences in sow BCS were detected ($P > 0.10$) between maternal treatments or parities on d 35 or d 70 of gestation. Sows fed diets with Carn and CrPic had lower ($P < 0.03$) BCS at farrowing and weaning in comparison with those receiving diets with no added Carn and CrPic.

An interaction between parity and maternal treatment was observed ($P = 0.05$) for gestation feed intake from d 0 to 35. This interaction is reflective of an increase in feed intake when Carn and CrPic were added to the diets of gilts and sows bred to carry their 2nd litter and a decrease in intake when Carn and CrPic were added to the diets of P3+ sows. No treatment or parity effects were observed ($P > 0.10$) for gestation intake from d 35 to farrowing or over the entire gestation period. Sows fed diets containing Carn and CrPic had increased ($P < 0.02$) daily and total lactation feed intake compared with sow fed diets without Carn and CrPic. Gilts and parity 2 sows also had decreased ($P < 0.002$) daily and total lactation feed intake compared with P3+ sows.

No maternal treatment effects were observed ($P > 0.10$) in total number of pigs born, born alive, cross fostered, or remaining on d 18 of lactation (Table 3-4). An interactive response between parity group and maternal treatment was observed ($P < 0.03$) for pig number at d 7 of lactation. Gilts and parity 2 sows fed Carn and CrPic nursed larger litters compared to those receiving no Carn and CrPic, while no differences were observed in P3+ sows. In addition, gilts and parity 2 sows nursed larger litters ($P = 0.001$) at cross-fostering, d 7 of lactation, and d 18 of lactation compared with P3+ sows.

No interactive effects or maternal treatment differences were detected ($P > 0.10$) for average pig weight. No parity effects were observed ($P > 0.10$) for average pig weight with the exception that P3+ sows had greater ($P < 0.05$) individual pig weight on d 18 of lactation and pig weight gain as compared with gilts and parity 2 sows. Litter weight for total born, born alive, or cross fostered were unaffected ($P > 0.10$) by maternal treatment. An interaction between parity and maternal treatment was observed ($P = 0.04$) for litter weight on d 7 of lactation and with similar tendencies observed ($P < 0.09$) on d 18 of lactation and for calculated weaning weight. These interactions are reflective of an increase in litter weight when Carn and CrPic were added to the diets of gilts and parity 2 sows and a decrease in litter weight when Carn and CrPic were added to the diets of P3+ sows. The interaction for litter weight was driven primarily by the interaction for litter size. Gilts and parity 2 sows produced larger ($P < 0.01$) litter weight at cross fostering and d 7 of lactation and tended to have heavier ($P = 0.08$) litter weight on d 18 of lactation as compared with P3+ sows.

Adding Carn and CrPic to sow diets made no impact ($P > 0.10$) on return to estrus (Table 3-5). Subsequent farrowing rate was also unaffected ($P > 0.10$) by parity or adding Carn and CrPic to sow diets. Total born and born alive were unaffected ($P > 0.10$) by supplementing sow diets with Carn and CrPic. Females that were gilts or sows in their second parity during the trial period had larger ($P < 0.05$) litters as compared with females that were P3+ during the trial period.

The distribution and cumulative proportions for several total born birth weight categories of pigs from sows consuming diets with and without Carn and CrPic are shown in Figure 1. Parity groups were combined within each maternal treatment for the distribution. The histogram shows similar normal distributions of pig birth weight for each maternal diet treatment. It appears that the right tail of the distribution decreases more slowly for pigs born to sows fed diets without any Carn and CrPic as compared to those born to sows fed diets with Carn and CrPic. However, Hartley's F-Max test for homogenous variances detected no differences (data not shown; $P > 0.10$) in the estimated variance components between pigs born to control or Carn and CrPic fed sows. Therefore, the variation in birth weight was not different between maternal control and Carn and CrPic groups.

Discussion

For several of the responses, parity effects were observed as expected. It should be noted that differences in genetic lines would also be confounded with parity as gilts and parity 2 sows were all of the same genetic line. Parity 3 and older sows would be made up of 3 genetic lines from the same genetic supplier. While, the response to dietary Carn and CrPic was not expected to vary based on sow genetic line; the genetic lines were balanced among dietary treatments.

At breeding, all females supplemented with Carn and CrPic during lactation were heavier and P3+ sows were in better body condition than control fed sows. This was not expected and could be reflective of improvements in lactation feed intake and utilization during the loading period for sows fed supplemental Carn and CrPic or possibly due to a randomization error where heavier sows were used for the Carn and CrPic treatment by chance. During the loading period, lactation feed intake was not measured and sows were not weighed prior to allotment. The true reason for the difference cannot be definitively known; however, the fact that sow BCS were similar for control and Carn and CrPic sows at the beginning of the loading period and improvements in lactation feed intake were observed during the trial lactation period suggests that the differences were not due to a randomization error.

Irregardless of parity group, females fed diets with Carn and CrPic had less gestation BW gain than sows not fed Carn and CrPic. This in contradiction to the improvements in gestation BW gain observed by Musser et al. (1999) where a constant gestation feeding amount was used and by Birkenfeld et al. (2005) where gestation feed was provided ad libitum. In the current study, sows feeding levels were adjusted regularly to account for the animal's current BCS in order to strive to achieve or maintain an ideal BCS.

Sow BCS on d 35 and 70 of lactation were similar between sows fed diets with and without Carn and CrPic. Therefore, the mean restricted feed allowance should have been similar for sows receiving diets with and without Carn and CrPic. Sows fed diets with Carn and CrPic had worse BCS at farrowing compared with sows receiving diets without Carn and CrPic. This suggests that during late gestation sows fed diets Carn and CrPic lost body condition for an unknown reason. Overall, the estimated gestation feed intake did not differ for maternal treatments or parity groups; however, it should be noted that technical difficulties dictated the need to estimate daily gestation feed intakes for the later portions of gestation. During lactation, sows fed diets with Carn and CrPic had greater lactation feed intake and less BW loss compared

with sows receiving the diet without Carn and CrPic. This may be a result of Carn and CrPic supplementation as Ramanau et al. (2004) observed increases in lactation feed intake for sows supplemented with Carn. The fact that sows fed Carn and CrPic were in poorer body condition could also be a factor as leaner sows will consume more feed in lactation (Mullan and Williams, 1989).

Supplementing sow diets with Carn and CrPic did not impact the number of total pigs at birth, live pigs at birth, or on d 18 of lactation. Real et al. (2008) also observed no differences in total born or live born pigs for sows fed diets containing Carn, CrPic, both or neither with a large sample size (N=600). Other experiments have shown decreases in the number of stillbirths (Musser et al., 1999b; Birkenfeld et al., 2006), increases in total born (Ramanau et al., 2004; Birkenfeld et al., 2005), and increases in the number of pigs born alive (Ramanau et al., 2004, 2008; Birkenfeld et al., 2005) for sows fed diets with Carn compared with sows fed diets without Carn. Adding 200 μg/kg of Cr from CrPic has also been shown to increase the total number of pigs born as well as the number of live born pigs (Lindemann et al., 1995, 2004).

No differences were observed in litter or pig weight for total born or born alive between sows supplemented with or without Carn and CrPic. No published trials have shown increases in pig weight with adding CrPic to sow diets; however, several studies have shown advantages in litter size with adding 200 μg/kg of Cr from CrPic resulting in greater litter birth weight (Lindemann et al., 1995, 2004). Supplementing Carn to sows during gestation has been shown to increase pig birth weights (Musser et al., 1999b; Eder et al., 2001; Ramanau et al. 2008). In contrast, Ramanau et al. (2004) observed a decrease in pig birth weight for 2 consecutive parities with supplementing sows with Carn; however, they used a small size (n=13 and 10 sows per treatment for the first and second parity, respectively) and they observed a large litter size response which resulted in increased total litter weight. Ramanau et al. (2004) also observed increases in litter growth rate while pigs were nursing the sow particularly in the early part of lactation. The authors speculated that the increased growth of suckling pigs was a result of increased total milk production and improved nutrient utilization in the sucking pig. Suckling pigs can only produce small amounts of Carn (Coffey et al., 1991) and adding Carn to sows diets has been shown to increase Carn in sow's milk (Musser et al., 1999a; Ramanau et al., 2004). In addition, the current study observed no differences in the amount of variation of pig birth weight

between pigs born to sows fed diets with or without Carn and CrPic. This was similar to the finding of Musser et al. (1999b).

For subsequent performance no differences were observed with Carn and CrPic supplementation. However, Musser et al. (1999a) observed a numerical increase in subsequent total born when adding Carn to sow diets during lactation while Ramanau et al. (2004) observed improvements in the second farrowing when Carn was fed to the sows continuously over both parities. Real et al. (2008) showed increased farrowing rates with adding Carn or CrPic to sow diets, but the response was not additive when both were fed in combination. The improved farrowing rate led to greater total production of pigs when Carn or CrPic were added to sow diets.

Improvements in nutrient utilization have been observed when supplementing Carn to gestating sows (Musser et al., 1999b), lactating sows (Ramanau et al., 2004), nursery pigs (Owen et al., 1996; Real et al., 2001; Rincker et al., 2003) and finishing pigs (Owen et al., 1993). Supplemental Cr from CrPic has also been shown to improve feed utilization in growing and finishing pigs (Lindemann et al., 1995). The improvements in feed utilization suggest the Carn and/or CrPic may impact energy metabolism through one of several potential mechanisms.

Several experiments have been conducted to investigate the energy metabolism mechanisms that explain the associated benefits in sow performance with Carn and/or CrPic supplementation. Owen et al. (1996) showed that supplementing Carn to weanling pigs resulted in decreased lipid accretion rates possibly due to the potential increase in β-oxidation of fatty acids. This could be valid in the lactating sow where a significant amount of the dietary energy is going to milk production and sparing energy from lipid accretion may benefit litter growth rates. In contrast, Musser et al. (1999) observed increases in backfat in gestating sows supplemented with Carn thereby suggesting that any reduction any improvements in feed utilization in gestation are not due to reduced lipid accretion rates. Also, Young et al. (2004) compared the effect of Carn and CrPic on energy balance of sows and determined no differences in the components of heat production during early, mid, or late gestation with supplementing Carn and CrPic.

Woodworth et al. (2007) examined the effect of Carn and/or CrPic on blood hormones and metabolite concentrations. They determined that supplementing Carn appeared to reduce mean non-esterified fatty acids (NEFA) concentrations, increase mean IGF-I concentrations in

the plasma, and increase mean IGF-binding protein 3 (IGF-BP3) concentration in plasma, while adding CrPic reduced insulin and glucose concentrations in plasma immediately after feeding. These finding would suggest that supplementing CrPic to sow diets improves energy utilization immediately after the meal and during the fasting state with Carn. Musser et al. (1999) also observed similar increases in maternal plasma concentrations of IGF-I during gestation with adding dietary Carn; however, Waylan et al. (2005) and Brown et al. (2007) observed no differences in circulating IGF-I concentrations s during mid-gestation between sows receiving diets with or without added Carn. Supplementing Carn in sow gestation diets has also been shown to increase mean leptin concentrations in plasma (Woodworth et al., 2004). Leptin plays an important role in food intake regulation and energy metabolism (Houseknecht et al., 1998) and elevated concentrations of leptin have been correlated with elevated concentrations of IGF-I and IGF-BP3 in lean human subjects (Seck et al., 1998). These trials suggest that Carn and/or CrPic may improve energy metabolism in gestating sows and do so independently of each other.

Contrary to the results of the current study, other studies have shown increases in pig and litter birth weights (Musser et al., 1999b; Eder et al., 2001) and/or suckling pig weight gain with adding Carn to sow diets (Ramanau et al., 2004). The elevated maternal circulating IGF-I and IGF-BP3 concentrations associated with adding dietary Carn (Musser et al., 1999bb; Woodworth et al., 2007) may have increased the amount of nutrients delivered to the fetus leading to increases in birth weight. At the same point, consistent increases in blood IGF-I have not been observed and it appears that adding Carn to sow diets does not increase IGF-I concentrations in the fetal liver or skeletal muscle tissue during middle portion of gestation (Waylan et al., 2005; Brown et al., 2008). Supplementing Carn to sow diets has also been shown to reduce the amount of IGF-II in the fetus (Brown et al., 2008) and reduce the amount of mRNA for IGF-II and myogenin in porcine embryonic myoblasts (Waylan et al., 2005). Insulin-like growth factor II is considered an embryonic growth factor effecting the proliferation and differentiation of myogenic precursor cells (Moses et al., 1980) and it has been negatively correlated with fetal weights (Waylan et al., 2005). Waylan et al. (2005) suggests that the changes in mRNA concentrations for IGF-II and myogenin could delay differentiation of porcine embryonic myoblasts allowing for increased proliferation and ultimately increasing muscle fiber numbers. This is in agreement with the results of Musser et al. (2001) who showed that supplementation of Carn to sows diets tended to increase the number of muscle fibers in the suckling pig.

Another factor that may be impacting birth weight and suckling performance associated with Carn supplementation is increases in fetal concentrations of Carn and carnitine palmitoyltransferases (CPT). The CPT1 enzyme is located on the outer membrane of the mitochondria and is associated with the binding of Carn to fatty acids in the cytosol of cells for transport into the mitochondria, and CPT2 is in the mitochondrial matrix and cleaves the bond between carnitine and a fatty acid. This is how the CPT and carnitine assist in β-oxidation of fatty acids. Adding Carn to sow diets has been shown to increase carnitine in fetal tissues and stimulate CPT activity in the in the liver (Lin et al., 2008). This increase in Carn and CPT activity may increase the suckling pig's ability to utilize fat.

While several studies have shown improvements in litter size and pig birth weight with supplementing Carn to sow diets and/or increases in litter size with adding CrPic, the current study shows no difference. One potential explanation for this difference may be due to lower levels of Carn used in the current study (25 mg/kg). Studies showing responses to Carn have supplemented diets with 50 mg/kg of Carn or 90 to 100 mg of Carn per day in gestation and 250 mg of Carn per day in lactation. The concentrations in this study were approximately ½ those used in many other published studies. Ramanau et al. (2008) showed increases in born alive and pig BW with supplementing diets with 25 mg/kg of Carn compared with diets containing no added Carn; however, the improvements were smaller in magnitude in comparison to sows supplemented with 50 mg/kg of Carn in the diet. In addition, long term trends over time have shown increases in litter size for current genetics. Improvements in litter size and birth weight associated with Carn and CrPic supplementation to sow diets may be limited in current genetics due to the uterine capacity of sows (Foxcroft, 2007).

Overall, minimal differences in sow performance were observed with the addition of Carn and CrPic to sow diets. Adding Carn and CrPic to sow diets did increased lactation feed intake, reduced lactation weight loss, and increased litter weight on d 7 of lactation in gilts and parity 2 sows. Overall, this trial found that adding Carn and CrPic to sow diets did not affect litter size, pig birth weight, or the variation in pig birth weight.

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Table 3-1 Composition of diets (as-fed basis)

Item	Pre-trial lactation[1]	Gestation	Lactation
Ingredient, %			
Corn[2]	42.66	56.89	42.86
Soybean meal (46.5% CP)	22.13	---	23.88
DDGS[3]	30.00	40.00	30.00
Choice white grease	2.00	0.00	0.25
Monocalcium P (21% P)	0.55	0.43	0.53
Limestone	1.38	1.68	1.48
Salt	0.45	0.45	0.45
Vitamin premix[4]	0.05	0.05	0.05
Trace mineral premix[5]	0.15	0.15	0.15
Sow add pack[4]	0.025	0.025	0.025
L-Lys HCl	0.43	0.26	0.24
L-Thr	0.10	---	---
Choline Cl	0.10	0.10	0.10
Total			
Calculated analysis			
ME, kcal/kg	3,413	3,325	3,329
CP, %	22.6	16.0	23.1
Total Lys, %	1.35	0.66	1.25
SID[6] amino acids, %			
Lys	1.17	0.51	1.07
Thr	0.78	0.43	0.71
Met	0.34	0.27	0.35
Trp	0.20	0.08	0.21
Ile	0.76	0.44	0.79
Leu	1.90	1.58	1.96
Val	0.89	0.59	0.92
Ca, %	0.72	0.73	0.76
P, %	0.60	0.53	0.61
Available P, %[7]	0.45	0.44	0.44

[1]The pre-trial lactation diet was used for the loading period for sows in lactation (Parity 2 and older) and the gestation diet was used for loading gilts.

[2]A portion of the corn was mixed and replaced with L-carnitine and chromium picolinate (DSM; Parsippany, New Jersey) to make the test diets.

[3]Dried distillers grains with solubles

[4]Provided the following per kg of complete diet: 9,920 IU of vitamin A, 1,590 IU of vitamin D_3, 66 IU of vitamin E, 3.97 mg of vitamin K, 0.03 mg of vitamin B_{12}, 39.70 mg of niacin, 29.80 mg of pantothenic acid, 8.25 mg of riboflavin, 0.25 g of biotin, 1.52 mg of folic acid, 3.81 mg of pyridoxine, 500.00 mg of choline, and 625 FTU from Optiphos 2000 (JBS United; Sheridan, IN).

[5]Provided the following per kg of complete diet: per lb of diet: 6.82 mg Cu from hydroxyl-methylthio-butyric acid (HMTBa; Novus International; St. Louis, MO), 6.78 mg Cu from CuSO4, 0.31 mg I from $Ca(IO_3)_2$, 92.59 mg Fe from $FeSO_4$, 20.09 mg Mn from Mn HMTBa, 19.84 mg Mn from $MnSO_4$, 0.15 mg Se from $NaSeO_3$, 0.15 mg Se from Se yeast (Novus International), 55.20 mg Zn from Zn HMTBa, 57.54 mg Zn from Zn0, and 38.36 mg Zn from $ZnSO_4$.

[6]Standardized ileal digestible.

[7]Phytase provided 0.11% available P to the gestation and lactation diets.

Table 3-2 Analyzed dietary concentrations of L-carnitine and chromium

	Control	Test diet
Analyzed concentrations of L-carnitine, mg/kg		
Pre-trial lactation[1]	2.3	26.4
Gestation diet[2]	2.0	22.5
Lactation diet[1]	4.9	21.5
Analyzed concentrations of chromium, μg/kg		
Pre-trial lactation[1]	17.1	198.3
Gestation diet[2]	32.3	264.0
Lactation diet[1]	25.8	253.5

[1]Samples were collected from each batch of feed manufactured. From all of the samples, a composite sample for each dietary treatment was analyzed.
[2]Samples of each gestation diet were collected each week. From all of the samples, a composite sample for each dietary treatment was analyzed.

Table 3-3 Effect of adding L-carnitine and chromium picolinate to sow diets on sow performance[1]

	Parity 1 and 2		Parity 3+			Observed significance level, *P* <		
	Control	Carn & CrPic[2]	Control	Carn & CrPic	SEM	Diet × Parity	Diet	Parity
Sow number	19	22	83	87				
Sow BW, kg								
Breeding	153	157	196	213	5.9	0.21	0.02	0.001
d 35 of gestation	174	173	211	224	6.4	0.20	0.27	0.001
d 112 of gestation[3]	220	212	249	244	6.9	0.77	0.24	0.001
Post-farrow[4]	201	194	229	225	6.7	0.70	0.25	0.001
Weaning	186	181	218	222	6.5	0.32	0.99	0.001
Gestation wt gain, kg[5]	67	54	52	31	3.9	0.18	0.001	0.001
Lactation wt loss, kg	15	12	10	2	2.4	0.18	0.01	0.001
Sow BCS[6,7]								
Loading[8]	3.18	3.23	3.52	3.69	0.243	0.75	0.55	0.03
Breeding	3.37	3.18	3.65	3.97	0.175	0.06	0.60	0.001
d 35 of gestation	3.05	3.09	3.01	3.07	0.174	0.95	0.72	0.82
d 70 of gestation	2.68	2.68	2.74	2.85	0.154	0.66	0.67	0.33
Farrowing	4.00	3.64	3.96	3.59	0.212	0.97	0.03	0.79
Weaning	3.11	2.64	2.88	2.55	0.199	0.64	0.01	0.30
Gestation feed intake, kg								
Breeding to d 35 of gestation	73.9	76.6	74.6	70.9	2.00	0.05	0.73	0.11
d 35 of gestation to farrowing[9]	205.8	206.9	212.8	211.8	5.01	0.79	0.99	0.13
Breeding to farrowing[9]	279.8	283.5	287.4	282.7	6.37	0.40	0.93	0.49
Lactation feed intake, kg								
Daily	5.1	5.2	5.3	5.5	0.09	0.75	0.02	0.002
Total	100.0	105.3	106.4	111.5	2.43	0.97	0.01	0.001

[1]A total of 211 sow and litters were used to determine the effects of supplemental L-carnitine and Cr on sow performance.
[2]L-carnitine (Carn) was added at 25 mg/kg and Cr was added at 200 μg/kg from chromium picolinate (CrPic).
[3]Entry into farrowing rooms.
[4]Post farrow wt was estimated by subtracting the total born litter wt from the pre-farrow wt.
[5]Gestation wt gain was measured as the difference from breeding up through d 112 of lactation.
[6]Body condition scores (BCS).
[7]Sow body condition scores of very thin, thin, ideal, fleshy, and fat were given numerical scores of 1 through 5, respectively, for analysis.
[8]Loading BCS only reflect animals in lactation for the loading period.
[9]Due to technical difficulties, actual gestation feed intake during the latter portions of gestation had to be estimated.

Table 3-4 Effect of adding L-carnitine and chromium picolinate to sow diets on litter performance[1]

	Parity 1 and 2		Parity 3+			Observed significance level, *P* <		
	Control	Carn & CrPic[2]	Control	Carn & CrPic	SEM	Diet × Parity	Diet	Parity
Sow number	19	22	83	87				
Pig number								
Total born	13.7	13.7	14.1	14.4	0.74	0.80	0.81	0.39
Born alive	13.2	13.0	12.8	12.8	0.70	0.85	0.86	0.62
Cross-fostering[3]	13.0	13.5	12.1	12.0	0.36	0.27	0.50	0.001
d 7	10.8	11.9	10.2	9.8	0.41	0.03	0.21	0.001
d 18	10.4	10.9	9.8	9.4	0.38	0.14	0.92	0.001
Pig wt, kg								
Total born	1.35	1.36	1.40	1.35	0.050	0.49	0.58	0.67
Born alive	1.38	1.37	1.42	1.38	0.050	0.59	0.52	0.51
Cross-fostering[3]	1.39	1.37	1.42	1.37	0.045	0.57	0.29	0.58
d 7	2.64	2.58	2.68	2.55	0.082	0.62	0.12	0.98
d 18	5.42	5.35	5.70	5.53	0.145	0.66	0.26	0.04
Litter wt, kg								
Total born	18.4	18.3	19.2	18.8	0.84	0.79	0.69	0.32
Born alive	17.9	17.6	17.9	17.3	0.84	0.83	0.46	0.82
Cross-fostering[3]	17.8	18.5	17.1	16.4	0.67	0.16	0.89	0.01
d 7	28.4	30.5	27.4	25.0	1.35	0.04	0.93	0.001
d 18	56.2	57.9	56.0	51.9	2.26	0.09	0.51	0.08
Calculated weaning[4]	60.4	63.2	62.0	57.0	2.85	0.08	0.63	0.29
Wt gain at d 18, kg								
Pig	3.99	3.90	4.22	4.09	0.127	0.74	0.38	0.05
Litter	41.1	42.4	41.2	38.3	1.73	0.12	0.52	0.14
Lactation length, d	19.7	20.2	20.2	20.4	0.29	0.57	0.13	0.11

[1]A total of 211 sow and litters were used to determine the effects of supplemental L-carnitine and Cr on litter performance.
[2]L-carnitine (Carn) was added at 25 mg/kg and Cr was added at 200 μg/kg from chromium picolinate (CrPic).
[3]Cross-fostering was performed daily at 1300 with pigs cross-fostered within 24 hr of birth. Cross-fostering wt reflect the birth wt of pig remaining at fostering.
[4]Litter weaning wt was estimated for each litter by calculating the daily litter growth rate from d 7 to 18 of lactation and using that value as an estimate for the expected daily litter gain for each day from d 18 of lactation until weaning.

Table 3-5 Effect of adding L-carnitine and chromium picolinate to sow diets on subsequent farrowing performance[1]

	Parity 2 and 3		Parity 4+			Observed significance level, $P <$		
	Control	Carn & CrPic[2]	Control	Carn & CrPic	SEM	Diet × Parity	Diet	Parity
Sow number	19	22	75	72				
Return to estrus, d	5.2	6.0	5.2	5.0	0.43	0.13	0.42	0.13
Farrowing rate, %	94.7	90.5	89.9	90.0	6.46	0.68	0.70	0.62
Pig Number								
Total born	12.8	13.4	11.2	12.1	0.96	0.80	0.34	0.05
Born alive	12.1	12.1	9.6	10.3	0.97	0.63	0.69	0.01

[1]A total of 188 sow and litters were used to determine the effects of supplemental L-carnitine and Cr on subsequent sow performance.

[2]L-carnitine (Carn) was added at 25 mg/kg and Cr was added at 200 μg/kg from chromium picolinate (CrPic).

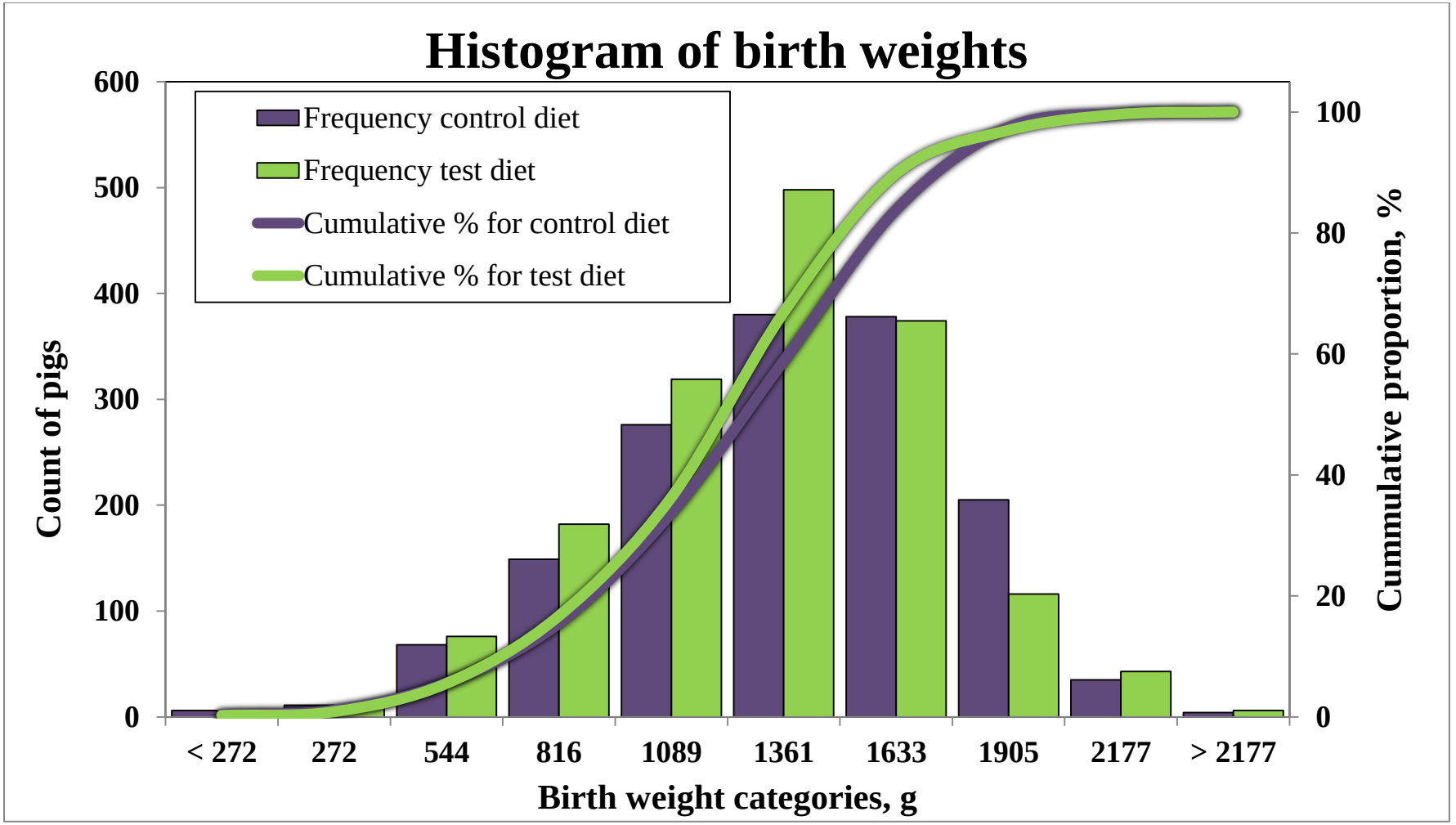

Figure 3.1 Distribution of birth weights of pigs born to sows fed either the control diet or the test diet with 25 mg/kg of added L-carnitine and 200 µg/kg of added Cr.

Chapter 4 - Effects of experimental design and its role in interpretation of results

Abstract

A total of 256 weanling pigs (PIC TR4 × 1050, initially 6.3 kg) were used in a 28-d growth trial to compare the allotment methods of a completely randomized design (CRD) and a randomized complete block design (RCBD). Two treatments were used to compare these designs: including a negative control with no added antimicrobials and a positive control with 38.6 mg/kg of Denagard (tiamulin; Novartis Animal Health, Greensboro, NC), 440.9 mg/kg of chlortetracycline (Alpharma, Eagle Grove, IA), and zinc from zinc oxide at 3,000 and 2,000 mg/kg from d 0 to 14 and d 14 to 28, respectively. Weaned pigs were allotted to the 2 designs such that each design would have equal mean and variation of BW for all pigs. Pigs assigned to the CRD were allotted so that average BW and within-pen variation of BW were similar between all pens. Pigs in the RCBD were blocked by BW and placed in location blocks. With the exception of pens of pigs in the CRD having a trend for improved ($P = 0.08$) G:F from d 0 to 14 compared with pens in the RCBD, no other design or design × antimicrobial treatment differences were detected ($P > 0.10$) for any response variables. The CV for BW within-pen remained the same in the CRD from d 0 to 28 at approximately 20%; however, increased from 3% on d 0 to 10% on d 28 for the RCBD. The within pen CV for ADG was similar ($P > 0.10$) for pens on the CRD and RCBD for the first 14-d post-weaning, but was increased ($P = 0.05$) for pens on the CRD compared with pens on the RCBD for the last 14-d.

Adding antimicrobials to nursery diets increased ($P < 0.04$) ADG, ADFI and G:F in both the CRD and RCBD from d 0 to 14. From d 14 to 28, the CRD detected an increase ($P < 0.001$) in ADG and ADFI with dietary antimicrobials, and the RCBD detected an increase ($P < 0.001$) only in ADFI. Over the entire 28-d trial, growth promoters increased ($P < 0.03$) ADG, ADFI, and G:F in the CRD but only increased ($P < 0.02$) ADG and ADFI in the RCBD. There were lower estimates for the standard errors of the difference for ADG and G:F in the CRD than in the RCBD from d 0 to 28.

The variance ratios of the CRD to RCBD from d 0 to 28 were 0.67 for ADG, 1.70 for ADFI, and 0.25 for G:F, suggesting that the CRD offered estimates for σ^2_{error} similar to those of

the RCBD. With similar estimates for σ^2_{error}, the increase in degrees of freedom for the error term would lead to greater power to detect differences in the CRD compared with the RCBD.

Key words: allotment, experimental design, data interpretation, nursery pigs

Introduction

Experimental design is a major factor that must be considered when planning research trials. The primary designs used in swine production and nutrition research include the completely randomized design (CRD) and the randomized complete block design (RCBD). Modifications or additions to these designs can be performed to generate more complex designs, such as a Latin square, that typically are used in specific instances when experimental units are limited. Each experimental design dictates the process of how treatments are allotted to experimental units (EU). The CRD is the simplest of all designs, and treatments are allotted to EU independently of any factors. In the RCBD, EU are grouped together into homogenous blocks with each treatment having one EU per block. One of the assumptions in this design is that treatments respond similarly in each block or that there were no true block × treatment interactions because the mean square calculated as the block × treatment term estimates the error variance structure for the model.

Statistical power is the probability of rejecting a null hypothesis when the null hypothesis is truly false (Leventhal, 2009). Power calculations are useful during the planning stages of an experiment to determine the sample size needed to detect effects if they truly exist (Lenth, 2007). Experimental designs can impact statistical power due to changes in degrees of freedom (DF) and estimates of variation. It is common to use blocking factors such as BW in nursery pig studies to achieve a reduced estimate for the error component (σ^2_{error}) of an experiment; however, blocking also reduces the error DF for the testing term. Therefore, blocking may increase or decrease the power depending on the individual blocking factor. Therefore, the main objective of this trial was to determine the impact of a RCBD by blocking pens of pigs by BW and pen location has on statistical interpretation compared to using a CRD where within pen variation in BW is similar but high for all pens of pigs.

Materials and Methods

The procedures used in this experiment were approved by the Kansas State University Institutional Animal Care and Use Committee. The study was conducted at the Kansas State University Swine Teaching and Research Center in Manhattan, KS.

Animals and diets

A total of 256 weanling pigs (PIC TR4 × 1050, Hendersonville, TN; initially 6.3 kg and 21 d of age) were used in a 28-d growth trial to compare allotment methods of a CRD and a RCBD. Two treatments were used to compare these designs: a negative control with no antimicrobials and a positive control with growth promoting concentrations of antibiotics and pharmacological concentrations of zinc (Zn). The positive control contained 38.6 mg/kg of Denagard (tiamulin; Novartis Animal Health, Greensboro, NC), 440.9 mg/kg of chlortetracycline (Alpharma, Eagle Grove, IA), and Zn from zinc oxide at 3,000 and 2,000 mg/kg in Phases 1 and 2, respectively. Experimental diets were fed in 2 phases: Phase 1 from d 0 to 14 and Phase 2 from d 14 to 28 (Table 4-1). Phase 1 and 2 diets were fed in meal form and formulated to contain 1.41% and 1.31% standardized ileal digestible lysine, respectively. Phase 1 diets contained 15% spray-dried whey and 3.75% fish meal, and Phase 2 diets were based on corn and soybean meal. Diets were formulated using ingredient profiles from the NRC (1998) and formulated to meet or exceed all requirements (NRC, 1998). Eight pens of 8 pigs were used for each dietary treatment and experimental design combination. Each pen contained a 4-hole dry self-feeder and a nipple waterer to provide ad libitum access to feed and water. Pens had wire-mesh floor and allowed for approximately 0.25 m^2 per pig.

Allotment and data collection

For the allotting of pens, a group of 4 pens located in the same location were randomized such that 2 pens would be used in the CRD, and 2 pens would be used in the RCBD. The 2 RCBD pens would constitute a location block and be randomized to the 2 dietary treatments. This was performed 8 times throughout barn generating 8 blocks for the RCBD. At the conclusion of allotting pens to designs, all pens on the CRD were randomized to treatments with 8 pens per treatment. For the allotting of pigs to pens, initially weaned pigs were split to each of the 2 designs such that each design would have equal initial BW and variations of BW for all

pigs. In addition, to reduce any bias, both gender and litter were balanced between experimental designs. Pigs in the RCBD were sorted into 8 different BW categories based on weaning weight. The 16 pigs from a BW category were split among each of the two pens in a location block generating a weight and location block. Pigs assigned to the CRD were allotted to pens so that the average BW and within-pen variation of BW were similar between all pens. This was accomplished by selecting one pig from each of BW categories for each pen. Individual pig weights and pen feed disappearance were measured every 14 d to determine ADG, ADFI, and G:F.

Statistical analysis

Three different SAS (SAS Institute Inc., Cary, NC) models were used to describe the effects of experimental design on trial interpretation. For each model, pen was used as the EU and ANOVA was conducted using an effects model in the MIXED procedure in SAS. The first model used data combined from the CRD and RCBD and was analyzed as a 2 × 2 factorial with the 2 experimental designs (CRD or RCBD) and the 2 dietary treatments (no added antimicrobials or added antimicrobials) treated as fixed factors with no random effects. This analysis was performed to evaluate any differences in pig performance between the two designs, evaluate any potential interactions between experimental design and dietary treatments, as well as compare the within pen variation of BW and ADG. Variation of BW and ADG within-pen was examined by comparing the CV for each pen.

Next, separate models were used to analyze each of the 2 designs independently. The model for the CRD used the dietary treatment as a fixed effect with a random effect of pen within dietary treatment (error term). For the RCBD, dietary treatment was again used as a fixed effect, block was used as a random effect, and block × antimicrobial treatment was used as a random effect to estimate the error variance component. Statistical significance was determined using F-tests and significance was determined at P values < 0.05 and was considered tendencies at P values > 0.05 but < 0.10.

After statistics were analyzed for each design, uncorrected and corrected relative efficiencies (RE) were calculated from the RCBD for the growth performance responses (Cochran and Cox 1957; Kuehl, 2000; Lentner et al., 1989). The uncorrected RE was determined by the following formula:

Uncorrected Relative Efficiency = s^2_{CRD} / s^2_{RCBD}

where s^2_{CRD}= calculated estimate for the sample variance if the data had been analyzed as a CRD and s^2_{RCBD}= estimate for the sample variance. The following equation is used to calculate s^2_{CRD}:

$$s^2_{CRD} = [SS_{block} + b(t-1)MS_{(Treatment \times Block)}] / (bt-1)$$

where SS_{block}= sums of squares associated with the blocking factor, b= number of blocks, t= number of treatments, and $MS_{(Treatment \times Block)}$= mean square for treatment × block (error term). A correction factor may also be applied to convert the uncorrected RE for inferences about the population variance (σ^2) instead of the sample variance. The corrected RE is calculated as follows:

$$\text{Corrected RE} = \text{Uncorrected RE} \times \{[(f_{RCBD}+1) \times (f_{CRD}+3)] / [(f_{RCBD}+3) \times (f_{CRD}+1)]\}$$

where f_{RCBD}= number of degrees of freedom associated with the treatment × block (error) term for the RCBD and f_{CRD}= number of degrees of freedom that would be associated with EU with treatment (error) term if the data had been analyzed as a CRD. In addition to the RE, a ratio of the actual CRD error variance component to the RCBD error variance component was computed. Ratios of variances were interpreted using the critical limits of 0.3 and 4.6 based on a 2-tailed *F-distribution*.

Results and Discussion

The results from the first model used data sets from both designs. This model examined dietary treatment, experimental design (CRD or RCBD), and the design × dietary treatment as fixed factors with no blocking factors. Equal variance was assumed for both experimental designs. The main focus of this model was to determine if the treatments means behaved similarly in each design and if overall performance differed in each experimental design. With the exception of pens in the CRD having a trend for improved (P = 0.08) G:F from d 0 to 14 compared with pens in the RCBD, no other design or design × antimicrobial treatment differences were detected (P > 0.10) for any responses variables (Table 4-2). On the basis of these results, it appears that antimicrobial treatment means reacted similarly in each of the experimental designs.

In addition to growth performance, the CV for ADG and BW within pens was examined (Table 4-3). No interactions were detected (P > 0.10) between experimental designs and antimicrobial treatments for the CV of ADG or BW within pens. As expected, blocking pigs by

initial BW in the RCBD reduced (P = 0.05) the CV for BW within pens at each time point compared with pens in the CRD. The difference in BW variation between the 2 designs is reflective of the allotment of pigs to pens. However, as time progressed, the difference in within pen variation between the CRD and RCBD was reduced. In the CRD, variation of BW within pen remained the same from d 0 to 28 at approximately 20% but increased from 3% to 10% for the RCBD. Unexpectedly, the CV for within pen variation of ADG from d 0 to 14 was similar ($P > 0.10$) for pens of pigs in both the CRD and RCBD. From d 14 to 28 and overall, the CV for within pen variation of ADG was greater ($P = 0.02$) for pens of pigs in the CRD as compared to pens of pigs in the RCBD. Also, adding antimicrobials to nursery diets reduced ($P = 0.05$) the CV for ADG from d 0 to 14 compared with diets containing no antimicrobials.

While it was not the primary objective of this experiment, if one assumes that location does not have an effect on data interpretation, this model would also compare the impact of sorting pigs by BW. Pens of pigs in the RCBD would be indicative of pens sorted by initial BW and pens of pigs in the CRD would be suggestive of a random assortment or gate cut filling of pens. Data from this experiment would suggest that sorting pigs by BW has no impact on nursery performance. Also, sorting pigs by BW decreases the initial variation of BW within pens compared with a random assortment; however, that difference is reduced over time. Similar results have been observed with nursery (Tindsley and Lean, 1984) and grow-finishing pigs (Hastad et al., 2005; O'Quinn et al., 2001). It appears that when finishing pigs are sorted by BW upon entrance, the variation in BW at the end of the finishing period will be similar to random assortment pens (O'Quinn et al., 2001). If the current study had lasted longer, the variation in BW may have become similar for the CRD and RCBD. In agreement with this experiment, it appears that sorting pigs to pens by initial BW within a single barn does not help reduce the amount of variation or improve the performance of pigs.

After determining that performance was similar between antimicrobial treatments in each of the experimental designs, models were generated to evaluate the effects of each design separately. Basic ANOVA tables for both a CRD and RCBD with a single fixed treatment factor and equal replications are shown in Tables 4-4 and 4-5, respectively. For a CRD, the only variance component would be the experimental unit (EU) within treatment term which is commonly referred to as the error term for a CRD. This error term is used as the testing term for the overall *F-test* for treatment effects. In the RCBD, variances are estimated for both the

blocking and block × treatment terms. One of the main assumptions associated with a RCBD is that treatments do not interact with blocking factors; and therefore, the block × treatment term can be used as the testing (error) term for treatment effects in a RCBD. If interactions between treatments and blocking factors are expected, a generalized block design would be appropriate. There are two main differences in the CRD and RCBD that will impact statistical analyses. First, using a RCBD will decrease the degrees of freedom (DF) for the error term by a factor of one less than the number of blocks compared to a CRD. The error DF are used as the denominator DF in the ANOVA *F-test*, and decreasing the DF will decrease the power to detect differences, all other things being equal. Second, a different σ^2_{error} is estimated for the RCBD compared to the CRD. Ideally the RCBD estimate for σ^2_{error} would be reduced in comparison to the CRD estimate for σ^2_{error}, thereby increasing statistical power for the RBCD in comparison to the CRD. These two differences can impact the statistical power of tests.

In both the CRD and the RCBD, pig weights were increased ($P < 0.003$) with supplementation of antimicrobials on d 14 and 28 (Table 4-6). Dietary addition of antimicrobials increased ($P < 0.04$) ADG, ADFI, and G:F ($P < 0.04$) in both the CRD and RCBD from d 0 to 14. The observed significance levels were lower for the CRD compared to the RCBD because of the increase in denominator DF for the CRD; however, similar or numerically lower standard error for differences in means (SED) were observed for the RCBD compared to the CRD. From d 14 to 28, the CRD detected an increase ($P < 0.001$) in ADG and ADFI with dietary addition of pharmacological Zn and antibiotics, and the RCBD detected an increase ($P < 0.001$) only in ADFI. The primary reason for the difference in response in ADG between the CRD and RCBD is due to the magnitude of change. Adding dietary antimicrobials increased ADG by 13.9% for the CRD and only 8.0% for the RCBD. Also the SED for ADG was numerically greater for the RCBD compared to the CRD. Over the entire 28-d trial, adding dietary antimicrobials increased ($P < 0.001$) ADG and ADFI and improved ($P = 0.03$) G:F in the CRD. In comparison, adding antimicrobials in the RCBD only resulted in increased ($P < 0.02$) ADG and ADFI. For the entire 28-d trial, reduced SED were also estimated for ADG and ADFI in the CRD compared with the RCBD.

The antimicrobial effect model was selected for use in this experiment as it has been shown to be one of the more consistent responses in nursery pigs. Dritz et al. (2002) conducted a meta-analysis looking at adding in-feed antibiotics in multisite production systems and

determined a 5% increase in ADG for nursery pigs fed diets with added antimicrobials compared with pigs receiving diets with no antimicrobials. Additional studies have shown additive effects of pharmacological Zn and in-feed antimicrobials on pig performance (Hill et al., 2001). Similar to the current results, Steidinger et al. (2009) observed improvements in growth, intake, and efficiency of nursery pigs fed diets containing Denagard and chlortetracycline, and Shelton et al. (2011) observed increased ADG and ADFI when pharmacological concentrations of Zn were added to the diets of weanling pigs in the same facility as the current experiment.

The effects of experimental design on the variance components and RE for each of the performance responses are shown in Table 4-7. It should be noted that the σ^2_{error} for the CRD and RCBD are not truly the same as those models have different variance and covariance structures; however, both estimates are used for testing treatment effects in the overall *F-test* and therefore can be compared. The RE are estimates as to the improvement in estimation of the σ^2_{error} for the RCBD compared to analyzing that particular data set as a CRD based on calculated estimates. It does not reflect the estimates actually observed by the CRD allotment. The uncorrected RE ranged from 0.65 to 10.63, and the corrected RE ranged from 0.59 to 9.64 for each of the growth responses. The corrected RE uses a correction factor to estimate the population variance from the sample variance; and therefore, is more useful as compared to the uncorrected RE (Kuehl, 2000). Each of the three response criteria seemed to follow a similar pattern for RE regardless of the time period with the greatest RE for ADFI, lowest RE for G:F, and the RE for ADG were intermediate. The gain and intake values suggest that the added variation explained by blocks in the RCBD was beneficial for achieving a more reduced estimate of σ^2_{error} compared to analyzing that particular data set as a CRD. It is to be expected that when pigs are sorted by BW, blocking by those BW categories would be beneficial in estimating σ^2_{error}. Relative efficiency is an accurate and effective tool that can be used to evaluate blocking factors. Although there is not an actual test that can be performed on those values, they do provide some insight and can be used to evaluate the efficacy of using a RCBD.

When a different allotment scheme was performed in the actual CRD, the variance ratio of the CRD to the RCBD ranged from 0.17 to 3.50. The ratios from d 0 to 28 depict the different responses well, with ADG at 0.67, ADFI at 1.70, and G:F at 0.25. The variance ratios can be compared with an *F-distribution* and the critical limits for a 95% confidence interval would be 0.30 and 4.60. Observed values greater than the upper limit, would suggest that the RCBD had a

reduced estimate for σ^2_{error}. No values were near in proximity to the upper limit. However, ratios for G:F from d 14 to 28 and d 0 to 28 were below the lower limit, suggesting the CRD had reduced estimates for σ^2_{error} compared with the RCBD. If blocking had been effective, it should be expected to observe the variance ratios above the upper critical limit.

Statistical power for an overall treatment *F-test* can be computed with the following values; acceptable type I error (probability of rejecting a null hypothesis that is actually true), numerator DF (DF for treatment), denominator DF (DF for error), and a function of the non-centrality parameter (Kuehl, 2000). The function of the non-centrality parameter is indicative of the number of replications, magnitude of treatment effect, and estimate for σ^2_{error}. Power will increase with an increase in each of the following items independently: acceptable type I error rate, denominator DF, number of replications, or magnitude of treatments differences. Power will decrease as the numerator DF or the estimate of σ^2_{error} increase. When comparing the two experimental designs as in this experiment, the two items that will change, in regards to power, are the denominator DF and the estimate for σ^2_{error}. The CRD will have greater denominator DF than the RCBD because DF in the RCBD are lost to the blocking factor. Therefore, the RCBD will need a lower estimate for σ^2_{error} compared to the CRD to obtain a similar level of power. The ratios of the variances did not show reduced estimates of σ^2_{error} for the RCBD compared the CRD, which lead to less power to detect treatment differences.

The ability to obtain homogenous EU may be the main factor as to the similar estimates for σ^2_{error} for both the CRD and RCBD. In this research facility, researchers can make pens homogenous for BW and variation of BW at the beginning of the period. However, location cannot be homogenized after randomization in the barn. Location effects may vary depending on room size. This trial was conduct in a small nursery room (approximately 300 pigs); and therefore, it would be easier to keep a constant environment across the entire room as compared to a large commercial research nursery (approximately 1,000 to 2,000 pigs per room). In a large commercial nursery, there may be instances were blocking by location may be beneficial. In addition, other factors have been shown to be influential in determining responses for other trials. Depending on the particular investigation, it may be beneficial to block by some factor. Some of more common blocking factors used for nursery pigs would be location, BW, gender, litter of origin, weaning age, genetic line, and/or pig density. Also, if researchers feel that their treatment structure may interact with a blocking factor, a generalized block design would be advantageous.

A generalized block design contains more than 1 replication of each treatment per block and will allow researchers to evaluate for any potential interactions between treatments and blocking factors.

In conclusion, researchers who typically block pigs by BW or some other factor can use the RE to determine whether blocking offers better estimates for σ^2_{error} than a CRD. Relative efficiency is a quick method of quantifying the benefit received from a blocking factor. This single study suggests that for this nursery facility in which researchers can control the homogeneity of the BW and variation of BW within pens, the CRD estimates for σ^2_{error} are similar to those in a RCBD. With the same estimate for σ^2_{error}, the function of the non-centrality parameter for each design would be similar, and therefore, the increase in DF for the error term would lead to greater power to detect differences among treatments. Additional studies are needed to verify these results as well as to compare designs in different facilities and stages of production to determine whether blocking is an efficient use of error DF.

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Table 4-1 Composition of diets (as-fed basis)

	Phase 1[1]		Phase 2[2]	
Antimicrobials[3]	No	Yes	No	Yes
Ingredient, %				
Corn	49.19	48.15	61.07	60.17
Soybean meal (46.5% CP)	28.98	29.06	34.97	35.03
Spray-dried whey	15.00	15.00	---	---
Select menhaden fish meal	3.75	3.75	---	---
Monocalcium P (21% P)	1.05	1.05	1.60	1.60
Limestone	0.70	0.70	1.10	1.10
Salt	0.33	0.33	0.33	0.33
Vitamin premix[4]	0.25	0.25	0.25	0.25
Trace mineral premix[5]	0.15	0.15	0.15	0.15
L-Lys HCl	0.30	0.30	0.30	0.30
DL-Met	0.175	0.175	0.125	0.125
L-Thr	0.125	0.125	0.110	0.110
Zinc oxide	---	0.384	---	0.256
Antibiotic	---	0.575	---	0.155
Total	100.00	100.00	100.00	100.00
Calculated analysis				
SID[6] amino acids, %				
Lys	1.41	1.41	1.31	1.31
Ile:Lys	60	60	63	63
Leu:Lys	120	120	129	129
Met:Lys	36	36	33	33
Met & Cys:Lys	58	58	58	58
Thr:Lys	62	62	62	62
Trp: Lys	17	17	18	18
Val:Lys	65	65	69	69
Total Lys, %	1.55	1.55	1.45	1.45
ME, kcal/kg	3,296	3,296	3,296	3,296
SID Lys:ME, g/Mcal	4.28	4.28	3.97	3.97
CP, %	22.3	22.3	21.9	21.9
Ca, %	0.88	0.88	0.85	0.85
P, %	0.78	0.78	0.75	0.75
Available P, %	0.50	0.50	0.42	0.42

[1]Pigs were fed Phase 1 from d 0 to 14.
[2]Pigs were fed Phase 2 from d 14 to 28.
[3]Antimicrobials included zinc from zinc oxide at 3,000 mg/kg in phase 1 and 2,000 mg/kg in phase 2, Denagard (tiamulin; Novartis Animal Health, Greensboro, NC) at 38.6 mg/kg, and chlortetracycline (Alpharma, Eagle Grove, IA) at 440.9 mg/kg.
[4]Vitamin premix provided per kg of complete feed: 11,023 IU of vitamin A, 1,377 IU of vitamin D, 44.1 IU of vitamin E, 4.4 mg of vitamin K, 0.04 mg of vitamin B_{12}, 50.0 mg of niacin, 27.6 mg of pantothenic acid, and 8.3 mg of riboflavin.
[5]Trace mineral premix provided per kg of complete feed: 16.5 mg of Cu from $CuSO_4 \cdot 5H_20$, 0.30 mg of I as $C_2H_2(NH_2)_2 \cdot 2HI$, 165 mg of Fe as $FeSO_4H_2O$, 39.7 mg of Mn as $MnSO_4 \cdot H_20$, 0.30 mg of Se as Na_2SeO_3, and 165 mg of Zn as $ZnSO_4$.
[6]Standardized ileal digestible.

Table 4-2 Effects of experimental design on nursery performance[1]

	Design			Significance Level, $P <$		
Item	CRD[2]	RCBD[3]	SED	Design × Antimicrobial	Design	Antimicrobial
d 0 to 14						
ADG, g	221	214	7.0	0.45	0.44	0.001
ADFI, g	263	263	8.6	0.65	1.00	0.001
G:F	0.84	0.81	0.011	0.60	0.08	0.001
d 14 to 28						
ADG, g	485	485	11.7	0.44	0.99	0.006
ADFI, g	709	704	14.7	0.85	0.81	0.001
G:F	0.69	0.69	0.007	0.16	0.58	0.13
d 0 to 28						
ADG, g	353	349	8.8	0.39	0.73	0.001
ADFI, g	486	482	11.1	0.72	0.83	0.001
G:F	0.73	0.72	0.006	0.11	0.72	0.38
BW, kg						
d 0	6.3	6.3	0.23	1.00	0.99	0.99
d 14	9.4	9.3	0.29	0.80	0.79	0.04
d 28	16.2	16.1	0.44	0.70	0.92	0.02

[1]A total of 256 weanling pigs (PIC TR4 ×1050, initially 6.3 kg and 21-d of age) were used in a 28-d study with 8 pigs per pen to determine the effect of experimental design on trial interpretation.
[2]Completely randomized design.
[3]Randomized complete block design.

Table 4-3 Effects of experimental design on variation in nursery performance[1]

	Design			Significance Level, $P <$		
Item	CRD[2]	RCBD[3]	SED	Design × Antimicrobial	Design	Antimicrobial
CV for pig weight with each pen						
d 0	20.6	3.1	0.59	0.70	0.001	0.70
d 14	20.6	9.9	1.02	0.83	0.001	0.58
d 28	18.5	9.9	1.04	0.84	0.001	0.71
CV for ADG within each pen						
d 0 to 14	28.2	28.6	3.08	0.27	0.90	0.05
d 14 to 28	20.0	14.8	1.83	0.29	0.008	0.55
d 0 to 28	20.1	15.9	1.67	0.86	0.02	0.36

[1]A total of 256 weanling pigs (PIC TR4 ×1050, initially 6.3 kg) were used in a 28-d study with 8 pigs per pen to determine the effect of experimental design on trial interpretation.
[2]Completely randomized design.
[3]Randomized complete block design.

Table 4-4 Analysis of variance table for a completely randomized design[1]

Source	DF[2]	SS[3]	MS[4]	EMS[5]
Treatment	t-1	$r\sum_{i=1}^{t}(\bar{y}_{i.}-\bar{y}_{..})^2$	$SS_{treatment}$ / (t-1)	$\sigma_e^2+r(\sum_{i=1}^{t}(\mu_i-\overline{\mu}_{.})^2)/(t-1)$
EU(Treatment)[6]	t(r-1)	$\sum_{i=1}^{t}\sum_{j=1}^{r}(y_{ij}-\bar{y}_{i.})^2$	$SS_{EU(Treatment)}$ / (t(r-1))	σ_e^2
Corrected total	N-1	$\sum_{i=1}^{t}\sum_{j=1}^{r}(y_{ij}-\bar{y}_{..})^2$		

[1]Example ANOVA table for a completely randomized designed experiment with balanced replication and a one-way organization of treatments: t= number of treatments, r=number of replications, y_{ij}=estimate for the j^{th} replicate of the i^{th} treatment, μ_i=population mean for the i^{th} treatment, σ_e^2=population variation component, and N= total number of observations.
[2]Degrees of freedom.
[3]Sums of squares.
[4]Mean square.
[5]Expected mean square.
[6]Experimental unit within treatment (error).

Table 4-5 Analysis of variance table for a randomized complete block design[1]

Source	DF[2]	SS[3]	MS[4]	EMS[5]
Treatment	t-1	$b\sum_{i=1}^{t}(\bar{y}_{i.}-\bar{y}_{..})^2$	$SS_{treatment}$ / (t-1)	$\sigma_e^2+b(\sum_{i=1}^{t}(\mu_i-\bar{\mu}_{.})^2)/(t-1)$
Block	b-1	$t\sum_{j=1}^{b}(\bar{y}_{.j}-\bar{y}_{..})^2$	SS_{block} / (b-1)	$\sigma_e^2+\sigma_b^2$
Treatment × Block[6]	(t-1)(b-1)	$\sum_{i=1}^{t}\sum_{j=1}^{b}(y_{ij}-\bar{y}_{i.})^2$	$SS_{(Treatment \times Block)}$/ (t-1)(b-1)	σ_e^2
Corrected total	N-1	$\sum_{i=1}^{t}\sum_{j=1}^{b}(y_{ij}-\bar{y}_{..})^2$		

[1]Example ANOVA table for a completely randomized designed experiment with balanced replication and a one-way organization of treatments: t= number of treatments, b=number of blocks, y_{ij}=estimate for the j^{th} block and the i^{th} treatment, μ_i=population mean for the i^{th} treatment, σ_e^2=population variation component, σ_b^2=block variation component, and N= total number of observations.
[2]Degrees of freedom.
[3]Sums of squares.
[4]Mean square.
[5]Expected mean square.
[6]Treatment × block (error).

Table 4-6 Effects of experimental design on interpretation of the growth effects of addition of growth promters[1]

	Completely randomized design				Randomized complete block design			
Antimicrobials[2]:	No	Yes	SED	Significance level, *P* <	No	Yes	SED	Significance level, *P* <
d 0 to 14								
ADG, g	185	258	13.1	0.001	184	243	8.8	0.001
ADFI, g	230	296	15.6	0.001	235	291	12.7	0.004
G:F	0.81	0.87	0.022	0.009	0.79	0.84	0.019	0.04
d 14 to 28								
ADG, g	454	517	13.6	0.001	467	504	19.8	0.11
ADFI, g	662	755	20.2	0.001	661	746	10.8	0.001
G:F	0.69	0.68	0.008	0.90	0.71	0.68	0.019	0.14
d 0 to 28								
ADG, g	319	387	12.3	0.001	326	372	15.0	0.02
ADFI, g	446	526	16.9	0.001	448	516	12.9	0.002
G:F	0.72	0.74	0.008	0.03	0.73	0.72	0.014	0.68
BW, kg								
d 0	6.3	6.3	0.01	0.87	6.3	6.3	0.01	0.64
d 14	8.9	9.9	0.18	0.001	8.9	9.7	0.12	0.001
d 28	15.2	17.1	0.34	0.001	15.4	16.8	0.31	0.003

[1]A total of 256 weanling pigs (PIC TR4 × 1050, initially 6.3 kg and 21-d of age) were used in a 28-d study with 8 pigs per pen to determine the effect of experimental design on trial interpretation.

[2]Antimicrobials included zinc from zinc oxide at 3,000 mg/kg in Phase 1 and 2,000 mg/kg in Phase 2, Denagard (Tiamulin; Novartis Animal Health, Greensboro, NC) at 38.6 mg/kg, and chlortetracycline (Alpharma, Eagle Grove, IA) at 440.9 mg/kg.

Table 4-7 Effects of experimental design on the variance components and estimation of the error terms[1]

Design:	CRD[2]	RCBD[3]				
Variance components:	σ^2_{error}	σ^2_{block}	σ^2_{error}	Uncorrected RE[4]	Corrected RE[5]	Variance ratio CRD:RCBD[6]
d 0 to 14						
ADG, g	685.6	556.5	311.1	2.67	2.42	2.20
ADFI, g	973.8	738.2	645.5	2.07	1.87	1.51
G:F	0.0019	0.0003	0.0015	1.19	1.08	1.27
d 14 to 28						
ADG, g	742.3	2038.1	1566.0	2.21	2.01	0.47
ADFI, g	1626.0	4788.9	463.9	10.63	9.64	3.50
G:F	0.0003	-0.0005	0.0018	0.74	0.67	0.17
d 0 to 28						
ADG, g	600.3	967.0	894.2	2.01	1.82	0.67
ADFI, g	1136.1	2160.5	669.2	4.01	3.64	1.70
G:F	0.0003	-0.0005	0.0012	0.61	0.55	0.25

[1]A total of 256 weanling pigs (PIC TR4 × 1050, initially 6.3 kg and 21-d of age) were used in a 28-d study with 8 pigs per pen to determine the effect of experimental design on trial interpretation.
[2]Completely randomized design.
[3]Randomized complete block design.
[4]Uncorrected relative efficiency = estimated σ^2_{error} for CRD / σ^2_{error} for RCBD and estimated σ^2_{error} for CRD = $(SS_{block}+b(t-1)MS_{error})/(bt-1)$ where b = the number of blocks and t = the number of treatments.
[5]Corrected relative efficiency = uncorrected relative efficiency × degrees of freedom correction, and the degrees of freedom correction = (df_{error} for RCBD + 1)(df_{error} for CRD + 3) / (df_{error} for RCBD + 3)(df_{error} for CRD + 1).
[6]Variance ratio CRD: RCBD = σ^2_{error} for CRD / σ^2_{error} for RCBD.