RESEARCH REPORT
SWINE HEALTH

Title: Effects of enteric disease on the prevalence of fallback pigs and profitability in a commercial setting – **NPB #11-084**

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Industry summary: Pigs can be born as fallbacks, in that they have a lighter birth weight and decreased capacity for postnatal growth. However, pigs with a normal or heavy birth weights can become fallback pigs due to a number of compromising factors, including poor nutrition, environmental conditions, or disease. Only 70% of a pig's genetic potential for growth is reached when reared in commercial conditions. Disease is thought to be a primary cause of this, because the immune system requires nutrients that could otherwise be available for growth. Pathogen elimination would be the most ideal way to control disease. However, this is often unfeasible in today's industry. Removing predisposing factors associated with pathogens is often the most practical method to control disease. Fallback pigs may be one of these predisposing factors as they have been thought to harbor pathogens that assault healthy pigs sharing the same space. However, we need to characterize if fallback pigs are, indeed, sources of increased enteric disease within a barn. Therefore, the objectives of this experiment were to: 1) identify the effects of piglet birth weight and transition average daily gain on subsequent growth, mortality, and carcass composition; and 2) determine if light birth weight pigs or those from the bottom 10th percentile of transition ADG in a commercial setting have a greater incidence of disease or gastrointestinal lesions. A total of 1,054 pigs were farrowed at a commercial sow farm, weighed at birth, and tagged individually. At 16 or 17 days of age, 1,054 pigs were weaned and moved to a commercial wean-to-finish barn. Mortalities were recorded on a daily basis. Pigs originated from a PRRSV negative herd, but broke with the disease in week 2 post-weaning. Pigs were weighed individually at weeks 0, 3, 6, and 22 post-weaning. Average daily gain from weeks 0 to 3 post weaning was termed transition ADG. One pig from each of the 10th, 30th, and 70th percentiles was used to create a 'set' of three pigs with the same gender, litter size and parity. Forty such sets were created, for a total of 120 pigs. On each of weeks 3 and 22 post-weaning, 20 sets of pigs were necropsied at the Iowa State University Diagnostic Laboratory. Lung, lymph node, and digesta were analyzed for presence of various pathogens, and many organs were scored for lesion incidence or severity by a licensed veterinary pathologist. Birth weight was a good predictor of overall post-weaning growth performance and final

weight, except for the period immediately after weaning. However, this lack of effect may have been affected by the PRRSV outbreak diagnosed one week prior. Transition ADG was an excellent predictor of subsequent post-weaning growth and a good predictor of post-weaning mortality. There was a significant birth weight × transition ADG interaction for mortality, where pigs from the 10th percentile of transition ADG and birth weight heavier than 1.51 kg had greater mortality than those with birth weights from 1.26 to 1.50 kg. Transition ADG did not affect carcass composition, but pigs with birth weights heavier than 1.76 had more backfat than all other pigs and larger loin eyes at 22 weeks post-weaning than pigs with birth weights lighter than 1.25 kg. There was no correlation between transition ADG and pathogen presence at either 3- or 22-weeks post-weaning. Incidence and severity of lesions in the large intestine decreased with increasing transition ADG at 3-weeks post-weaning. Lesion incidence and severity were also affected by transition ADG at 22-weeks post-weaning. Birth weight affected *Haemolytic E. coli* and *Salmonella spp. B* incidence at 3-weeks post-weaning, as well as *Brachyspira spp.* incidence at 22-weeks post-weaning. In summary, we determined that birth weight did not affect transition ADG or mortality, but greatly affected subsequent performance. Increasing transition ADG decreased mortality substantially, but did not affect carcass composition. Both birth weight and transition period ADG affect lesions in some organs and *Brachyspira spp.* infection incidence decrease with increasing birth weight. However, poor transition ADG in pigs is not correlated with disease incidence during a PRRSV outbreak. For more information, contact John Patience at jfp@iastate.edu or at 515-294-5132.

Keywords: birth weight, disease, fallback, nutrition, pig, transition, weaning

Scientific abstract: The objectives of this experiment were to: 1) identify the effects of piglet birth weight and transition average daily gain (tADG) on subsequent growth, mortality, and carcass composition; and 2) determine if light birth weight pigs or those from the bottom 10th percentile of tADG in a commercial setting have a greater incidence of disease or gastrointestinal lesions. A total of 1,054 pigs (Danbred 600 × Newsham NC32) were farrowed at a commercial sow farm, weighed at birth (BRW), and tagged individually. At 16 or 17 days of age, 1,054 pigs were weaned and moved to a commercial wean-to-finish barn. Mortalities were recorded on a daily basis. Pigs originated from a PRRSV negative herd, but broke with the disease in week 2 post-weaning. Pigs were weighed individually at weeks 0, 3, 6, and 22 post-weaning. Average daily gain from weeks 0 to 3 post weaning was termed transition ADG (tADG). One pig from each of the 10th, 30th, and 70th percentiles was used to create a 'set' of three pigs with the same gender, litter size and parity. Forty such sets were created, for a total of 120 pigs. On each of weeks 3 and 22 post-weaning, 20 sets of pigs were harvested to determine nutrient digestibility, carcass composition, and organ system tissue evaluation. Lung, lymph node, and digesta were analyzed for presence of various pathogens by PCR and culture methods. Data were analyzed using the GENMOD, GLIMMIX, and REG procedures of SAS. Birth weight was a good predictor ($P < 0.02$; $^{R2} > 0.97$) of overall post-weaning growth performance and final weight, except for the period immediately after weaning ($P = 0.99$). Transition ADG was an excellent predictor of subsequent post-weaning growth ($P < 0.0001$; $R^2 > 0.98$) and a good predictor of post-weaning mortality ($P < 0.0001$; $R^2 = 0.82$). There was a significant BRW × tADG interaction for mortality, where pigs from the 10th percentile of transition ADG and BRW heavier than 1.51 kg had greater mortality than those with birth weights from 1.26 to 1.50 kg ($P < 0.05$). Neither birth weight nor transition ADG affected apparent total tract digestibility of nutrients or energy ($P > 0.15$), but these values were substantially lower than other values in the literature. Transition ADG did not affect carcass composition ($P > 0.11$), but pigs with BRW heavier than 1.76 had more backfat than all other pigs and larger longissimus muscle area at 22 weeks post-weaning than pigs with BRW lighter than 1.25 kg ($P < 0.05$). There was no correlation ($P > 0.12$) between tADG and pathogen presence at either 3- or 22-weeks post-weaning. Incidence and severity of microscopic lesions in the large intestine decreased linearly with increasing tADG (incidence: $P = 0.01$; 65, 55, 25% for 10th, 30th, and 70th percentiles, respectively; severity: $P = 0.01$; 1.15, 0.75, 0.16 for 10th, 30th, and 70th percentiles, respectively) at 3-weeks post-weaning. Lesion incidence

and severity were also affected ($P < 0.04$) by tADG at 22-weeks post-weaning. Birth weight affected (P = 0.02) *Haemolytic E. coli* and *Salmonella spp. B* isolation at 3-weeks post-weaning, as well as *Brachyspira spp.* isolation at 22-weeks post-weaning (P = 0.05). There were no effects (P > 0.21) of BRW or tADG on serum or ileum mucosa scraping immune markers. Thus, our data suggest that tADG, but not BRW is related to post-weaning mortality. Both BRW and tADG are indicative of subsequent growth performance, but not due to differences in nutrient digestibility. Finally, poor tADG is not correlated with pathological or immunological markers of enteric disease.

Introduction: Transition average daily gain (tADG), or ADG during the first 3 weeks post-weaning, quantifies success during the weaning process and is affected by a multitude of factors. Transitioning pigs to solid diets during the weaning process involves environmental, psychological, and nutritional changes that cause stress to the animal. Weaning-related stress instigates villous atrophy and subsequently decreases absorption of nutrients, resulting in depressed growth. However, these stressors affect some pigs to a greater extent than others, as evidenced by the increased variation that occurs at weaning. This increased variation may be due to intrinsic factors, such as birth weight, gender, or dam parity; or extrinsic factors, such as pathogen exposure. It is well established that birth weight and weaning weight are related to subsequent growth performance. However, the effect of periweaning growth performance on overall growth improvement is variable. In addition, there are limited data available on the effects of periweaning growth performance on nutrient digestibility, mortality, or carcass composition.

There is also little data regarding the effect of disease on the prevalence of fallback pigs, particularly within a commercial setting. This is troubling because only 70% of a pig's genetic potential for growth is reached when reared in commercial conditions. Both disease and inflammation have been hypothesized to be the primary sources of this reduction in performance. A commercial environment has been shown to produce continuous immune stimulation that diverts nutrients that would otherwise be available for growth. The number of stressors is highly correlated with decreases in feed intake, gain, and tissue deposition; and stress at weaning has been hypothesized to be the primary driver of peri-weaning failure to thrive syndrome. Pathogen elimination would be the most ideal way to control disease. However, this is often unfeasible in today's industry. Removing predisposing factors associated with pathogens is often the most practical method to control disease. Fallback pigs may be one of these predisposing factors as they may harbor pathogens that assault healthy pigs sharing the same space. Rearing these fallback pigs separately from their contemporaries may improve the overall enteric health and performance of the growing-pig population. However, we first need to characterize if fallback pigs are sources of increased enteric disease within a barn.

Objectives: 1) Identify the effects of piglet birth weight and transition average daily gain (ADG) on subsequent growth performance, mortality, nutrient digestibility, and carcass composition; and 2) determine if light birth weight pigs or those from the bottom 10th percentile of tADG have a greater incidence of enteric pathogen presence, gastrointestinal lesion presence or severity, and immunological or oxidative stress markers in ileal mucosa and serum in a commercial setting.

Materials & Methods: All experimental procedures adhered to the ethical and humane use of animals for research, and were approved by the Iowa State University Institutional Animal Care and Use Committee (#2-11-7095-S).

Animals and housing. Over a 3.5-d period, 1,500 pigs (Danbred 600 × Newsham NC32) were farrowed at a commercial sow farm located in southern Iowa (Iowa Select Farms, LLC). Prior to suckling, pigs were weighed to obtain individual birth weights and tagged with an identification number. Source sow parity and

litter size were recorded. All procedures from birth until weaning were carried out according to normal procedures at the source farm, including cross-fostering among litters. At 16 or 17 days of age, a total of 1,054 pigs were randomly selected, weaned, and transported to a commercial curtain-sided wean-to-finish barn with slatted floors and a deep pit in central Iowa (Iowa Select Farms, LLC). Pigs were sorted by sex and randomly allotted to 40 pens with 26 or 27 pigs per pen.

Experimental design and diets. Pigs were weighed individually at birth, at weaning, and 3, 6, and 22-weeks post-weaning. Transition ADG (tADG) was calculated as the average daily gain between weeks 0 and 3 post-weaning. One pig from each of the 10th, 30th, and 70th percentiles of tADG was used to create one set of three pigs with the same litter size and from the same parity sow. This allowed for the direct comparison of tADG category without the confounding factors of litter size or sow parity. Forty sets were created in this manner, for a total of 120 pigs. Pigs remained in original pens after initial placement, so a single set of pigs may have been located in multiple pens. Data were analyzed as a 5 × 3 factorial, with 5 birth weight categories: < 1.00 kg, 1.00 to 1.25 kg, 1.26 to 1.50 kg, 1.51 to 1.75 kg, and > 1.76 kg and 3 transition ADG categories: 10th, 30th, or 70th percentiles. Fallback pigs were identified as those with tADG in the 10th percentile. Pigs remained in original pens after initial placement, so a single set of pigs may have been located in multiple pens. All pigs were fed the same diets in the phase-feeding program utilized by the commercial producer. However, 0.40% titanium dioxide was included at the expense of corn in diets at weeks 3 and 22 post-weaning to serve as an indigestible marker for nutrient digestibility analyses. Feed samples were collected from the hopper of each feeder and pooled at weeks 3 and 22 post-weaning. Feed-grade antibiotics were included in the feeding program. From weaning until the barn average weight was approximately 9.3 kg, pigs received diets containing 50 ppm carbodox. When pigs were approximately 9.3 kg to 12 kg, diets contained 400 ppm chlortetracycline and 35 ppm tiamuline hydrogen fumarate. When pigs weighed approximately 12 to 18 kg, diets contained 500 ppm oxytetracycline. When pigs were approximately 18kg to 23 kg, diets contained 400 ppm chlorotetracycline. When pigs were approximately 23 kg until the end of the experiment, pigs received diets containing 25 ppm zinc bacitracin. As previously reported, overall ADG from weaning until 22 weeks post-weaning was 599, 618, and 649 g/d ($P < 0.0001$; SEM = 9.7 g/d), while week 22 body weight was 100.0, 102.9, and 107.7 kg ($P < 0.0001$; SEM = 1.55 kg) for pigs from the 10th, 30th, and 70th percentiles, respectively (Jones and Patience, 2012).

Pig health status. Pigs originated from a herd negative for Porcine Reproductive and Respiratory Syndrome virus (PRRSV). However, a mixed PRRSV and Influenza A virus outbreak was confirmed week 2 post-weaning in the wean-to-finishing barn where this study was conducted.

Necropsy and sample collection. Twenty sets of pigs (20 sets × 3 pigs per set = 60 total pigs) were transported to the Iowa State University Veterinary Diagnostic Laboratory (ISU-VDL) at each of weeks 3 and 22 post-weaning. After transport, blood was collected from live pigs via jugular venipuncture; serum was collected and stored at -80°C for later analyses. Pigs were euthanized via captive-bolt stunning and exsanguination (week 3 post-weaning) or electrocution (week 22 post-weaning) and necropsied by veterinary pathologists. A 15-cm segment of the ileum was collected exactly 1-m proximal to the ileal-cecal junction immediately after euthanasia. The ileum was immediately split along the mesenteric border placed on a cold metal tray, flushed with cold saline, and mucosa was collected by gently scraping the luminal surface with a glass microscope slide. Mucosa scrapings were flash-frozen in liquid nitrogen and stored at -80°C for later analyses. Rectal contents were collected from each pig to determine the apparent total tract digestibility of energy and nutrients. Appropriate fresh tissue samples and digestive contents were collected for pathogen

identification. Sections of brain, heart, kidney, large intestine, liver, lung, lymph node, nasal turbinate, pancreas, small intestine, spleen, stomach, and thymus were collected, fixed in 10% neutral-buffered formalin for 48 hours, routinely processed, and stained with hematoxylin and eosin for microscopic examination.

Incidence of pathogens. The incidence of both the North American and European genotype of PRRSV (TaqMan® NA and EU PRRSV, Applied Biosystems, CA, USA) in lung samples and the incidence of Porcine Circovirus 2 (PCV2) in lymph nodes (tracheobronchial and mesenteric) were determined by PCR. Additionally, PCR was utilized to determine the incidence of rotavirus serogroups A, B, and C and *Lawsonia intracellularis* in colon content samples. Lung, spleen, small intestine and colon were cultured for *Arcanobacterium pyogenes*, *Brachyspira spp.*, *Escherichia coli haemolytic* (Hemolytic E. coli), *Haemophilus parasuis*, *Pasteurella multocida A*, *Salmonella spp.*, and *Streptococcus suis*.

Incidence and severity of lesions. Presence and severity of microscopic lesions were analyzed in a blinded fashion by a certified veterinary pathologist at the Iowa State University Veterinary Diagnostic Laboratory. All collected tissues were examined for inflammation, necrosis, degeneration, and atrophy that could be associated with a disease process, and scored from 0 to 3 (0 = no lesion, 1 = mild, 2 = moderate, 3 = severe).

Liver mineral concentration. Fresh liver samples were quantitatively assessed for concentrations of cadmium, calcium, cobalt, copper, chromium, iron, phosphorus, potassium, magnesium, manganese, molybdenum, selenium, sodium, and zinc by Inductively Coupled Plasma/Mass Spectrometry (ICP/MS).

Immune marker concentration. Approximately 500 mg of mucosa scrapings was placed in 4 mL phosphate-buffered saline, homogenized, and centrifuged at 10,000 × g for 15 minutes. The supernatant was extracted, subsampled, and stored at -80°C for subsequent analyses. Total protein concentration of hydrolyzed mucosa scrapings was quantified using a Pierce bicinchoninic acid (BCA) Protein Assay kit (Thermo Scientific). Using porcine ELISA kits, serum and mucosa concentrations of IgA (Bethyl Laboratories, Inc.), IL-1β (R&D Systems), IL-8 (R&D Systems), and total GSH (Cayman Chemical Company) were determined. All analyses were completed in duplicate, and repeated when intra-duplicate coefficient of variation exceeded 5%. Mucosa concentrations were standardized from a per milliliter of supernatant basis to a per gram of mucosa scraping basis by multiplying the immune marker concentration of each sample by its total protein concentration.

Nutrient digestibility and carcass composition data collection. Rectal content and feed samples were oven-dried, ground through a 0.5-mm screen, and analyzed for DM, ash, GE, N, and titanium concentration. Percentage DM and ash were determined according to modified methods 930.15 and 942.05 (AOAC Int., 2007), respectively, where samples were dried at 105˚C or 600˚C, respectively to a constant weight. Benzoic acid was used as the standard for calibration (6,318 ± 18 kcal/kg) for gross energy using bomb calorimetry, and was determined to be 6,321 ± 13 kcal/kg. Nitrogen content was determined by Kjeldahl according to method 981.13 (AOAC Int., 2007). Calibration was conduction with a glycine standard (N content 18.7 ± 0.1%). Upon analysis, N content of the glycine standard was determined to be18.7 ± 0.08%. Crude protein was expressed as nitrogen × 6.25. Titanium was analyzed according to Leone (1973). All chemical analyses were carried out in duplicate, and repeated when intra-duplicate coefficient of variation exceeded 1%. Dry matter and apparent total tract digestibility of nutrients were calculated according to the equation: DM Apparent Digestibility (%) = 100% - [(Diet Marker Concentration ÷ Feces Marker Concentration) × 100] or Nutrient ATTD Coefficient (%)

= 100% - {[(Diet Index Marker Concentration ÷ Feces Index Marker Concentration) × (Feces Nutrient Concentration ÷ Diet Nutrient Concentration)] × 100}.

In addition to digestibility analyses, carcass composition was analyzed at week 3 post-weaning. All blood was collected following exsanguination. Contents from the stomach, intestines, gallbladder, and bladder were emptied. Whole carcasses, plus head, feet, blood, and organs were frozen, ground, homogenized, and subsampled. Sub-samples were then freeze dried, ground through a 1-mm screen, and analyzed for percentage DM, ash, crude fat, N, and GE as described above.

At 22 weeks post-weaning, carcass composition of the remaining 20 sets of pigs was determined by real-time ultrasound. Off-midline backfat and longissimus muscle area were measured from a cross-sectional image taken at the 10th rib as described by Newcom et al. (2002). Pigs were then transported to the Iowa State University Diagnostic Laboratory and slaughtered. Samples of rectal contents were collected from each pig to analyze the apparent total tract digestibility of energy and nutrients. Rectal content and feed samples were oven-dried, ground through a 0.5-mm screen, and analyzed for DM, ash, GE, N, and titanium concentration as described above. All other pigs were reared according to normal procedures of the commercial producer.

Statistical analyses. Pig was the experimental unit for all analyses. The fixed effects were BRW and tADG categories, while the random effects were pen and set. For carcass data analyses at week 22 post-weaning, the model also included the fixed effect of sex. Any non-significant interactions ($P > 0.05$) were removed from the model. The mortality and the incidence of bacterial or viral pathogens, or lesions were binary variables (0 = mortality or pathogen/lesion not present, 1 = alive or pathogen/lesion present) and had Bernoulli distributions. Thus, the incidence of mortality, bacterial or viral pathogens, or lesions was analyzed using the GENMOD procedure of SAS (Version 9.2, SAS Institute, Cary, NC), which utilized a logit model with categorical independent variables (Kaps and Lamberson, 2009). Results were considered significant if $\chi^2 < 0.05$. Predicted proportions for each independent variable were calculated using the PREDICTED statement, and differences ($\alpha = 0.05$) between least squares means were calculated using the LSMEANS DIFF statement. If lesion incidence was significant ($P < 0.05$), lesion severity score was calculated using the GLIMMIX procedure of SAS. The GLIMMIX procedure was also used to determine growth, nutrient digestibility, carcass composition, liver mineral concentration, and immune marker concentrations. Immune marker concentrations were log transformed prior to analyses in order to meet the assumptions of ANOVA. Reported immune marker concentration *P* -values represent transformed values, but means and standard errors of the means represent untransformed values. The LSMEANS and DIFFS statements provided estimates of least-squares means and differences ($\alpha = 0.05$) between them, respectively. Effects were considered trends if $0.05 < P < 0.10$. Pearson correlation coefficients were calculated using the CORR procedure of SAS. Results were considered significant if $P < 0.05$.

Results: Birth weight was significantly different among pigs within each birth weight category ($P < 0.05$; Table 1). However, the differentiation among pigs with heavy birth weights decreased over time, which was a reflection of CV decreasing with age (CV = 29.8, 17.0, 10.2, and 5.2% at birth, weaning, 3-weeks post-weaning, and 22-weeks post-weaning, respectively). Pigs with birth weights from 1.51 to 1.75 kg had statistically similar weights at weaning and week 3 post-weaning as pigs with birth weights heavier than 1.76 kg ($P > 0.05$). By 6 and 22 weeks post-weaning, there were no significant weight differences among pigs in different birth weight categories if birth weights were heavier than 1.26 kg ($P > 0.05$). While subsequent weight differences among pigs from the heavy birth weight categories diminished, pigs with birth weights lighter than

1.00 kg were still significantly lighter than all other pigs throughout the experiment ($P < 0.05$); and the magnitude of difference was noteworthy.

Surprisingly, birth weight was not associated with transition ADG ($P = 0.99$). We had hypothesized pigs from light birth weights would have a more difficult transition period, and thus depressed ADG. However, this was not apparent (Pearson correlation $P = 0.49$). Although pigs from the 10th percentile ADG had substantially more variation in transition ADG, there was similar variation in birth weight among the transition ADG categories (Figure 1). We did observe the expected relationship between birth weight and subsequent growth rate after the transition period. Pigs with birth weights lighter than 1.00 kg had depressed growth performance compared to pigs with birth weights heavier than 1.26 kg from 3 weeks to 22 weeks post-weaning ($P < 0.05$).

Mortality incidence was not different among different birth weight categories (Main effect $P = 0.43$; Pearson Correlation $P = 0.63$). Birth weight differed among gender, where barrows were slightly heavier than gilts ($P = 0.01$; 1.40 vs. 1.38 kg). However, gender did not affect any other growth performance variable or mortality ($P > 0.07$), including weight at week 22 post-weaning ($P = 0.48$; 90.9 vs. 89.4 kg for barrows vs. gilts, respectively). There were significant interactions between birth weight and gender for weights at weaning and 3-weeks post-weaning ($P < 0.04$; Figure 2), as well as transition period ADG ($P = 0.004$; Figure 3). In general, barrows from lighter birth weights were lighter or slower growing compared to gilts, while barrows from heavier birth weights were of similar weight but faster growing than gilts.

Transition ADG differed significantly among pigs within each category ($P < 0.0001$; Table 2). Weight among the three transition ADG categories differed from one another at weeks 3 and 6 post-weaning ($P < 0.05$). By week 22, however, weight of pigs from the 10th and 30th percentiles of transition ADG were not different ($P > 0.05$). Similarly, ADG was different among pigs in each category from weeks 3 to 6 post-weaning ($P < 0.05$); but ADG between the 10th and 30th percentiles did not differ from weeks 6 to 22 or overall ($P > 0.05$). Again, the magnitude of difference between the 10th and 70th percentiles was surprising.

In contrast to birth weight, slow transition ADG was associated with increased mortality ($P < 0.0001$). In support of our hypothesis, pigs from the 10th percentile of transition ADG had substantially greater mortality than those from the 30th or 70th percentiles ($P < 0.05$; 30.5 vs. 11.4 and 8.6%, respectively). In fact, 82% of the variation in mortality could be explained by transition ADG category. There were no transition ADG × gender or transition ADG × gender × birth weight interactions. There were also no transition ADG × birth weight interactions for growth performance, ($P > 0.18$), but there was an interaction with mortality ($P = 0.001$; Table 3). Pigs from the 10th percentile ADG and birth weights from the median category (1.26 to 1.50 kg) had lower mortality than pigs in all other categories in the 10th percentile ($P < 0.05$).

At both 3- and 22-weeks post-weaning, the apparent total tract digestibility of energy or nutrients did not differ among pigs from different transition ADG categories ($P > 0.11$), or among pigs from different birth weight categories or genders ($P > 0.07$; Table 4). There were also no interactions among transition ADG, birth weight, or gender for apparent total tract digestibility of nutrients ($P > 0.06$). In agreement with our nutrient digestibility data, the body composition of pigs did not differ by birth weight category, transition ADG category, or gender at 3-weeks post-weaning ($P > 0.07$). Transition ADG did not affect carcass composition at 22-weeks post-weaning ($P > 0.13$); however, there were effects due to both birth weight and gender ($P < 0.03$; Table 5). Pigs with birth weights heavier than 1.76 kg had greater backfat than pigs in all other birth weight categories ($P < 0.05$). Meanwhile, pigs with birth weights heavier than 1.76 kg had larger longissimus muscle area than pigs in all other birth weight categories ($P < 0.05$). Pigs with birth weights lighter than 1.00 kg had a substantial numerical decrease in longissimus muscle area, but it was only significantly different from pigs with birth weights from 1.26 to 1.50 kg or greater than 1.76 kg ($P < 0.05$). In this experiment, we also saw the

expected difference in longissimus muscle area according to gender ($P = 0.001$; 42.8 vs. 47.0 cm^2 for barrows vs. gilts, respectively). However, gender did not affect backfat ($P = 0.43$).

Neither birth weight nor transition ADG greatly affected the bacteria or viruses burden in the pigs used in this study. At week 3 post-weaning, there was no effect ($P > 0.08$; Table 6) of BRW on the incidence of *Arcanobacterium pyogenes, Haemophilus parasuis,* PCV2, PRRSV, rotavirus, or *Streptococcus suis* infection. However, the incidence of hemolytic *E. coli* and *Salmonella spp. B* was affected ($P = 0.02$) by BRW, but in different manners. Pigs from BRW categories less than 1.25 kg had decreased ($P < 0.05$) incidence of hemolytic *E. coli* than those pigs in the 1.26 to 1.50 kg or > 1.76 kg categories. This resulted in hemolytic *E. coli* incidence increasing linearly with BRW ($P = 0.04$). Meanwhile, pigs with BRW from 1.00 to 1.50 had a decreased ($P < 0.05$) incidence of *Salmonella spp. B* compared to pigs with BRW > 1.76, resulting in a quadratic effect ($P = 0.02$).

There was no effect ($P > 0.31$) of tADG on the presence of bacteria or viruses at 3-weeks post-weaning. While this may be the case at 3-weeks post-weaning, BRW and tADG may affect pathogen incidence during the finishing period. Pigs with BRW < 1.00 kg had a greater ($P < 0.05$) incidence of colonic *Brachyspira spp.* infection than those with BRW 1.51 kg or heavier. Increasing birth weight tended to decrease ($P = 0.08$) colonic *Brachyspira spp.* infection, and there was a strong negative correlation between the two ($P = 0.05$; Correlation = - 0.881; Table 7). There were no ($P > 0.14$; Table 6) other effects of BRW on bacteria or viral presence. *Brachyspira spp.* infection was also affected ($P = 0.01$) by tADG, although in an unexpected manner. Pigs from the 30th percentile had greater ($P < 0.05$) incidence *Brachyspira spp.* infection than those from the 10th percentile of tADG, an effect for which we cannot explain.

There was no effect ($P > 0.32$) of tADG on the incidence of *Haemophilus parasuis, Lawsonia intracellularis, Pasteurella multocida A,* PRRSV, *or Salmonella spp. B.* However, tADG affected ($P = 0.03$) PCV2 incidence, where pigs from the 10th percentile of tADG had increased ($P < 0.05$) PCV2 prevalence compared to those from the 30th or 70th percentiles. Still, there were no significant correlations ($P > 0.12$) between tADG and pathogen presence at either week 3 or 22 post-weaning, including PCV2 incidence at 22 weeks post-weaning ($P = 0.43$; Table 7).

At week 3 post-weaning, there was no effect ($P > 0.24$; Table 8) of BRW on lesion incidence in the brain, heart, kidney, large intestine, liver, lung, lymph nodes, nasal turbinate, pancreas, small intestine, or spleen. Both stomach inflammation ($P = 0.05$) and thymic atrophy ($P = 0.02$) lesion incidence was affected by BRW, but in opposite manners. Pigs with BRW < 1.00 kg had increased ($P < 0.05$) stomach inflammation compared to those with BRW greater than 1.51 kg. This resulted in stomach lesion incidence decreasing ($P = 0.03$) linearly with increasing BRW. However, the severity score of stomach lesions was not affected ($P = 0.21$) by BRW. Meanwhile, pigs with BRW > 1.76 kg had increased ($P < 0.05$) thymic atrophy incidence compared to pigs with BRW < 1.00 kg and from 1.26 to 1.75 kg. In addition, the severity of thymic atrophy was significantly affected ($P = 0.01$) by BRW, as lesions from pigs with BRW > 1.76 kg had greater ($P < 0.05$) severity compared to lesions from all other pigs. Following the trend of *E. coli* incidence, thymic atrophy increased ($P = 0.03$) linearly with BRW. Although we reported a strong correlation between BRW and prevalence of *Brachyspira spp.* infection above, the extent of the disease was not reflected in lesion incidence. In fact, there were no effects ($P > 0.12$) of BRW on lesion incidence at week 22 post-weaning.

While there was no effect of tADG on pathogen presence at week 3 post-weaning, it did affect the incidence of lesions in the large intestine ($P = 0.03$) and their severity ($P = 0.02$).Incidence and severity were greater ($P < 0.05$) in pigs from the 10th and 30th percentiles compared to those in the 70th percentiles. Lesion incidence was not affected ($P > 0.10$) by tADG in any other measured organs at 3-weeks post-weaning.

At 22-weeks post-weaning, tADG affected both lung ($P = 0.03$) and stomach ($P = 0.04$) lesion incidence, but again in opposite directions. Pigs from the 10th percentile tADG had decreased ($P < 0.05$) lung lesion incidence compared to pigs from the 30th or 70th percentiles. This effect is similar to that of the incidence of *Brachyspira spp.* infection. Severity score of lung lesions were not affected ($P = 0.60$) by tADG. Meanwhile, Pigs from the 10th percentile tADG had greater ($P < 0.05$) stomach inflammation incidence and severity compared to those from the 30th or 70th percentiles.

There was no link ($P > 0.20$; Table 9) between BRW or tADG and serum of ileum mucosa IgA, IL-1β, IL-8, or total GSH. There were some numerical differences, such as light BRW pigs and pigs from the 10th percentile tADG generally had numerically decreased IgA concentrations in both the serum and mucosal scrapings compared to heavier or faster growing pigs. However, there was too much variation to draw any conclusions.

There was no effect ($P > 0.11$; Table 10) of BRW or tADG on liver cadmium, calcium, chromium, cobalt, copper, magnesium, manganese, molybdenum, phosphorus, potassium, selenium, sodium, or zinc concentrations. However, hepatic iron concentrations were affected by both BRW ($P = 0.004$) and tADG (P 0.0004). Pigs with BRW < 1.00 kg had greater ($P < 0.05$) iron concentrations than those with BRW from 1.51 to 1.75 kg. Additionally, pigs from the 10th percentile had greater ($P < 0.05$) iron concentrations than those from the 70th percentile of tADG. Iron concentrations decreased linearly ($P < 0.01$) with both increasing BRW and tADG.

Discussion: Fallback pigs may simply be those born with a light birth weight. Perhaps caused by intrauterine growth retardation, prenatal programming may restrict subsequent performance as suggested by the thrifty phenotype hypothesis. However, fallback pigs can also be those with similar birth weights or weaning weights, such as with porcine periweaning failure-to-thrive syndrome (PFTS). Typically, PFTS is associated with pigs that have normal weaning weights and no sign of infectious or nutritional factors that would predispose them to poor post-weaning growth, but that have anorexia noticeable within one week post-weaning and extreme lethargy in the following 1-2 weeks. While similar to the pigs in this experiment, pigs with PFTS have a common weaning weight, which is not a requirement of fallback pigs.

In addition to light BRW or PFTS, disease may cause pigs to fall back from normal performance. The classification of fallback pigs may encompass pigs with intrauterine growth retardation, pigs with PFTS, pigs with increased pathogen presence, or pigs that fall back for other unknown reasons. Within the confines of this experiment, we wanted to determine if disease presence and severity is associated with fallback pigs by studying the effects of both birth weight and periweaning or transition period ADG (tADG). Furthermore, this study was conducted in commercial facilities using commercial diets and handling conditions.

It is important to note that the PRRSV outbreak at week 2 post-weaning likely influenced the results of this experiment, particularly those at week 3 post-weaning. The PRRS virus is highly associated with secondary infections. Although the incidence of PRRS viremia was equal among all pigs at 3-weeks post-weaning, these results provide valuable insight into the prevalence of pathogens and immunological markers during and after a PRRSV outbreak.

The 1.1-kg difference at birth between the two extreme birth weight categories (< 1.00 kg vs. > 1.76 kg) resulted in a 12.3 kg difference by week 22 post-weaning (170 d of age). Birth weight substantially influenced subsequent weight. Over 95% of all variation in weight at weaning, 3, 6, or 22-weeks post-weaning could be explained by birth weight. These very high R^2 values were quite surprising, because a number of other factors affect weight variation, including environment, health, and diet. Still, our data agrees with previous research suggesting that birth weight is the foundation of final weight variation.

It was surprising that tADG was not affected by BRW. Perhaps the PRRSV outbreak during the transition period confounded the effect of birth weight on ADG. We were unable to find other research regarding the effect of birth weight during a PRRSV outbreak. However, the high disease load likely influenced the transition period ADG by inhibiting nutrient digestibility. The PRRSV as well as the secondary infections of Streptococcus suis and SIV, likely induced B-cell proliferation. The primary site of B-cell differentiation in pigs is in the Peyer's patches of the ileum, the primary site of nutrient absorption in the pig.

Interestingly, the observed differences in body weight and post-weaning growth performance among pigs from different birth weight categories were not reflected in mortality. This is in contrast to our hypothesis that pigs with light birth weights would have decreased survivability. However, the current experiment evaluated mortality beginning at weaning, and thus the effects of preweaning mortality were not considered. This is important to note because the majority of mortality related to birth weight occurs during the preweaning period In the 1,500 pigs farrowed for this experiment, preweaning mortality differed significantly among different birth weight categories ($P < 0.0001$; < 1.00 kg: 25.9%, 1.00 to 1.25 kg: 9.0%; 1.26 to 1.50 kg: 6.8%; 1.51 to 1.75 kg: 6.1%; > 1.76 kg: 1.5%). Our experimental design began at weaning with 1,054 pigs, thus preventing the inclusion of preweaning mortality in our postweaning mortality data, but it is important to note the distinction, particularly when comparing to data sets that consider preweaning mortality.

Like the large effects we saw in early birth weight differences, a 41% improvement in transition ADG resulted in a 7.7-kg difference in weight at 22 weeks post-weaning. One can calculate that, if using ADG from 6 to 22 weeks post-weaning and marketed to a common weight, pigs from the 70th percentile would have likely reached market six days earlier than those from the 10th percentile.

Although our hypothesis regarding decreasing mortality with increasing tADG was proven true, this was not the case when evaluating the significant mortality interaction between tADG and BRW. Numerically, pigs from the 10th percentile ADG and heaviest birth weight categories had the highest mortality, which disproved our hypothesis. We had expected mortality to be greatest in pigs from the 10th percentile transition ADG and lightest birth weight categories. Perhaps mortality was greatest among those with heavy birth weights because the weaker light birth weight pigs died during the suckling period. We may have proven our hypothesis if we had considered both preweaning and post-weaning mortality.

The lack of effect on digestibility was surprising, particularly for pigs in different transition ADG categories. Growth modeling has shown us that birth and weaning weights are highly correlated with weights at finishing. However, pigs with heavier birth or weaning weights are thought to have a physiological advantage over lighter weight pigs of the same age beyond that of simple differences in weight. It is thought that pigs with heavier WW pigs adapt more quickly to a solid diet during weaning than lighter weight pigs because those with heavier WW have a more developed digestive tract than pigs with lighter weaning weights, even at a similar age. The heavier pig is likely earlier maturing, and thus its digestive enzyme production is more attuned to grain-based diets at an early weaning age, such as the one used in this experiment. If differences in digestive tract maturity existed in this experiment, we would have likely seen these differences reflected in the apparent total tract digestibility and nutrients. However, we saw no such affect at either week 3 or week 22 post-weaning. The nutrient and energy digestibility values in our experiment ranged from 55.6% to 72.5%, which are substantially lower than expected from NRC (1998). We had expected slightly lower digestibility values because our experiment was in a commercial barn, which would assumedly have that had more stressors compared to the confines of a typical digestibility experiment. Our values were substantially lower than those seen in another commercial experiment. While impossible to determine within the confines of this experiment,

we hypothesize that the low nutrient digestibility values were due to high disease incidence. We found no research regarding the direct implications of PRRSV-infection on nutrient digestibility. However, PRRSV has been shown to induce serum IL-1, IFN-ɣ, IgM, IgG, and IgA concentrations. A PRRSV infection results in the production of a variety of monoclonal antibodies, and the primary site of PRRSV replication occurs in macrophages within mucosal surfaces. Taken together, we can suspect that the PRRSV and secondary infections induced the production of proinflammatory cytokines within the gut mucosa. Cytokines are known to decrease feed efficiency and inhibit nutrient transport by acting on the somatotropic axis. In addition to decreased nutrient transport, immune stimulation suppresses growth by stimulating anabolic growth factors. Immune stimulation likely resulted in decreased nutrient digestibility across all pigs, but additional research is needed to corroborate these findings.

Previous research relating birth weight and carcass composition has been mixed. However, pigs with lighter birth weights have been shown to have fewer myofibrils, which are larger in size. Muscle fiber number is an important factor in growth rate, particularly after 70 d of age, and fewer myofibrils are indicative of lower carcass yield. This was likely the effect of body weight, as we chose not to use final body weight as a covariate. Although not statistically significant, pigs from the lighter birth weight categories had numerically greater backfat than those from the median birth weight categories. If backfat measurements had been taken at a common weight, as opposed to a common age, these differences may have become statistically significant.

We were very surprised to see such little effect of either birth weight or tADG on pathogen incidence. The direction of both the *E. coli haemolytic* and *Salmonella spp. B* incidence in regard to BRW was unexpected and in contrast with previous human research. For example, very light birth weight infants are more prone to *E. coli* pathogen incidence than normal weight infants because the growth of anaerobic organisms in a healthy gut inhibits the proliferation of gram-negative organisms. We would have expected *Salmonella spp. B* incidence to decrease with increasing BRW, as has been described in human infants. For example, a 250-g increase in infant BRW resulted in increased protection from *Salmonella* Infantis.

Our hypothesis that fallback pigs have a greater incidence of pathogens than their faster-growing contemporaries was disproven. Unreported data from our lab demonstrates a difference between the theoretical and actual ME intake of fallback pigs compared to their contemporaries. One of the possible explanations for this difference is a variation in the maintenance requirement of pigs, which may fluctuate due to disease prevalence. However, this study suggests that disease incidence, as measured by pathogen prevalence, does not differ among pigs of varying tADG. We have found no other data regarding the effect of pathogen presence on the prevalence fallback pigs. However, other researchers have reported that PFTS is not caused by common pathogens, including *Clostridium perfringens,* pathogenic *E. coli,* attaching and effacing *E. coli, Pasteurella mutocida, Streptococcus suis, Haemophilus parasuis, Bordetella bronchiseptica, Brachyspira hyodysenteriae, Brachyspira pilosicoli,* PCV2, PRRSV, Influenza A virus, Alphacoroanvirus 1, rotavirus A, Porcine enteric calicivirus, Suid herpesvirus 2, Betacoronavirus 1, Torque teno virus 1, Torque teno virus 2, or Coccidia. This is the only controlled experiment of pigs with PFTS in the literature, and consisted of an investigation at a single farm. However, taken together with data from this experiment, it appears that neither PFTS nor fallback during the periweaning period were influenced by the infectious disease agents evaluated.

While PCR and digestive content samples allow us to determine the incidence of pathogens, we must also consider the magnitude of their effect. One manner in which this can be quantified is through the incidence and severity score of histopathological lesions on various organs. Again, neither BRW nor tADG greatly affected lesion incidence or severity. However, thymic atrophy appeared to follow the direction and magnitude of *Salmonella spp. B* incidence at 3-weeks post-weaning and stomach lesion incidence mimicked PCV2 incidence at 22-weeks post-weaning, while.

Another method to determine severity of pathogen load is to determine the concentration of markers of immunological or oxidative stress, such as immunoglobulins, cytokines, chemokines, and glutathione. Gram-negative bacteria, such as the *E. coli* and *Salmonella spp. B* that were present in this experiment produce endotoxins, which increases cell sensitivity to an immune response and the ensuing production of pro-inflammatory cytokines. Epithelial wall integrity can be compromised during an immune response, allowing for increased translocation of pathogens. Additionally, there are recent reports that pigs with PFTS have decreased epithelial barrier function and altered mucosal morphology that were not only explained by low feed intake.

Because pathogen and lesion incidence were low, the lack of effect of immunological markers was expected. Our values appear similar to those reported previously in the literature. However, there is very limited immune marker data with respect to mucosa scrapings, particularly during a disease challenge. Thus, our data are among the first to report mucosa immunoglobulin and cytokine concentrations during a natural PRRSV outbreak.

Because the immune system has a high demand for trace elements to serve as cofactors in various metabolic pathways, we determined liver mineral concentrations. However, because there were few pathological differences between pigs of different BRW or tADG categories, we were not surprised to see the same lack of effect in liver mineral concentrations.

The differences in iron concentration among pigs with both different BRW and tADG was initially surprising, but upon further review we concluded that this effect was likely due to the residual iron injection (200 mg/mL Fe dextran) given within 48-hr of birth. Pigs have relatively low body Fe reserves at birth, and there is little Fe transfer in sow's milk. Rapid growth and corresponding blood and hemoglobin development quickly utilizes Fe reserves, leaving the pig vulnerable to anemia. Historically, pigs have overcome this anemia by rooting in Fe-containing soil, but confinement conditions restrict a pig's contact with soil. Thus, instigating the need for an Fe-dextran injection to prevent anemia. The stored iron from the Fe-dextran injection is primarily utilized by pigs between 14-21 d of age. However, the iron stores may be available in the pig up to 8 weeks of age, particularly if pig growth is slow.

In summary, we determined that birth weight was not indicative of transition ADG or mortality, but greatly affected subsequent performance. Increasing transition ADG decreased mortality substantially, but did not affect nutrient digestibility or carcass composition. Both birth weight and transition period ADG result in gastrointestinal lesions in some organs to various severities, and that *Brachyspira spp.* infection incidence is negatively correlated with BRW. However, poor tADG in pigs is not correlated with pathogen incidence or immunological markers of enteric disease during a PRRSV outbreak.

Table 1. Effect of birth weight category on post-weaning (PW) growth performance and mortality.

	Birth Weight Category, kg							
	< 1.00	1.00 to 1.25	1.26 to 1.50	1.51 to 1.75	> 1.76	Pooled SEM	*P* =	R^2 with Birth Weight
n =	131	244	320	256	103			
Growth Performance								
Weight, kg								
Birth	0.82[e]	1.16[d]	1.40[c]	1.63[b]	1.88[a]	0.024	< 0.0001	-
Weaning	3.65[d]	4.23[c]	4.88[b]	5.38[a]	5.60[a]	0.278	< 0.0001	0.98
3 Week PW	6.64[d]	7.21[c]	7.84[b]	8.37[a]	8.49[a]	0.282	< 0.0001	0.97
6 Week PW	10.28[c]	12.00[b]	12.96[a]	13.38[a]	13.96[a]	0.730	< 0.0001	0.95
22 Week PW	83.4[c]	88.1[b]	90.9[ab]	92.8[a]	95.7[ab]	3.56	0.002	0.99
ADG, g/d								
0 – 3 Week PW	161	161	160	161	162	4.3	0.99	0.17
3 – 6 Week PW	175[b]	231[a]	247[a]	238[a]	257[a]	33.0	0.003	0.78
6 – 22 Week PW	616[b]	650[ab]	669[a]	680[a]	697[a]	29.6	0.02	0.98
0 – 22 Week PW	501[b]	527[ab]	541[a]	549[a]	562[a]	22.3	0.02	0.98
Mortality, %	21.4	16.4	14.4	18.4	18.5	-	0.43	0.09

[abcde]Means within a row that do not share a common superscript differ $P < 0.05$.

Table 2. Effect of transition ADG category on post-weaning (PW) growth performance and mortality.

	Transition ADG Category					
	10th	30th	70th	Pooled SEM	*P* =	R^2 with Transition ADG
n =	105	105	105			
Growth Performance						
Weight, kg						
3 Week PW	6.75[c]	7.77[b]	8.97[a]	0.093	< 0.0001	0.99
6 Week PW	11.49[c]	13.53[b]	15.59[a]	0.293	< 0.0001	0.99
22 Week PW	100.0[b]	102.9[b]	107.7[a]	1.55	< 0.0001	0.99
ADG, g/d						
0 – 3 Week PW	90[c]	151[b]	219[a]	1.4	< 0.0001	-
3 – 6 Week PW	224[c]	270[b]	316[a]	12.2	< 0.0001	0.98
6 – 22 Week PW	796[b]	821[b]	862[a]	12.8	< 0.0001	0.99
0 – 22 Week PW	599[b]	618[b]	649[a]	9.7	< 0.0001	0.99
Mortality, %	30.5[a]	11.4[b]	8.6[b]	-	< 0.0001	0.82

[abc]Means within a row that do not share a common superscript differ $P < 0.05$.

Table 3. Means of significant transition ADG category × birth weight category interaction on post-weaning (PW) mortality.

Transition ADG Category:	10th Percentile					30th Percentile					70th Percentile				
Birth Weight Category, kg:	< 1.00	1.00 to 1.25	1.26 to 1.50	1.51 to 1.75	> 1.76	< 1.00	1.00 to 1.25	1.26 to 1.50	1.51 to 1.75	> 1.76	< 1.00	1.00 to 1.25	1.26 to 1.50	1.51 to 1.75	> 1.76
n =	25	29	29	15	7	18	35	27	16	9	13	26	33	27	6
Total Mortality, %	28.0^{abc}	31.0^{ab}	17.2^{bcd}	46.7^{a}	57.1^{a}	16.7^{abcd}	14.3^{bcd}	3.7^{d}	12.5^{bcd}	11.1^{abcd}	7.7^{bcd}	7.7^{cd}	9.1^{cd}	11.1^{bcd}	0.0^{e}

^{abcde}Means within a row that do not share a common superscript differ $P < 0.05$.

Table 4. Main effects and interactions on apparent total tract digestibility (ATTD) of energy and nutrients and carcass composition.

	P =						
	Birth weight	Transition ADG	Gender	Birth weight × Transition ADG	Birth weight × Gender	Transition ADG × Gender	Birth weight × Transition ADG × Gender
Week 3 post-weaning							
ATTD[1], %							
Dry matter	0.65	0.72	0.64	0.77	0.98	0.90	0.75
Gross energy	0.66	0.44	0.98	0.48	0.92	0.15	0.43
Nitrogen	0.16	0.64	0.61	0.24	0.47	0.84	0.58
Ash	0.15	0.52	0.28	0.72	0.19	0.63	0.43
Carcass composition[2], %							
Water	0.32	0.33	0.85	0.75	0.60	0.59	0.73
Protein	0.62	0.11	0.07	0.55	0.17	0.06	0.65
Lipid	0.18	0.18	0.70	0.99	0.14	0.16	0.69
Ash	0.48	0.76	0.71	0.54	0.15	0.95	0.44
Week 22 post-weaning							
ATTD[3], %							
Dry matter	0.65	0.67	0.26	0.85	0.63	0.15	0.31
Gross energy	0.44	0.87	0.91	0.18	0.64	0.46	0.26
Nitrogen	0.18	0.997	0.88	0.86	0.98	0.65	0.77
Ash	0.27	0.22	0.32	0.45	0.50	0.80	0.51
Carcass composition[4]							
Backfat, mm	0.03	0.43	0.37	0.09	0.54	0.14	0.17
Longissimus muscle area, cm^2	0.01	0.14	0.001	0.39	0.95	0.79	0.67

[1]Overall means (mean ± SEM): dry matter = 72.2% ± 0.60%, gross energy = 72.5% ± 0.64%, nitrogen = 67.9% ± 0.65% and ash = 72.1% ± 0.66%.
[2]Overall means (mean ± SEM): water = 68.0% ± 0.66%, protein = 15.8% ± 0.12%, lipid = 13.3% ± 0.76% and ash = 2.9% ± 0.35%.
[3]Overall means (mean ± SEM): dry matter = 65.6% ± 0.60%, gross energy = 59.3% ± 0.94%, nitrogen = 55.6% ± 0.48% and ash = 62.0% ± 0.52%.
[4]Overall means (mean ± SEM): backfat = 16.8 mm ± 0.62 mm and longissimus muscle area = 44.9 cm^2 ± 0.86 cm^2.

Table 5. Effect of birth weight category on carcass composition at 22 weeks post-weaning.

	Birth Weight Category, kg					
	< 1.00	1.00 to 1.25	1.26 to 1.50	1.51 to 1.75	> 1.76	Pooled SEM
Backfat, mm	16.8[b]	16.0[b]	15.5[b]	15.4[b]	20.2[a]	0.61
Longissimus muscle area, cm^2	41.7[c]	46.7[bc]	45.5[ab]	44.5[bc]	49.0[a]	1.26

[abc]Means within a row that do not share a common superscript differ $P < 0.05$.

Table 6. Effects of birth weight or transition ADG category on pathogen incidence[1]

	Birth Weight Category, kg						Transition ADG Category			
	< 1.00	1.00 to 1.25	1.26 to 1.50	1.51 to 1.75	> 1.76	*P* =	10th	30th	70th	*P* =
Week 3 post-weaning										
n =	7	10	20	15	8		20	20	20	
Incidence, %										
Arcanobacterium pyogenes	0.0	10.0	5.0	0.0	0.0	0.54	0.0	5.0	5.0	0.44
Escherichia coli haemolytic[2]	0.0[b]	0.0[b]	35.0[a]	20.0[ab]	37.5[a]	0.02	25.0	25.0	15.0	0.66
Haemophilus parasuis	14.3	10.0	30.0	20.0	25.0	0.73	20.0	15.0	30.0	0.51
PCV2[3]	0.0	0.0	5.0	6.7	0.0	0.69	0.0	5.0	5.3	0.44
PRRSV (NA)[4]	100.0	100.0	100.0	100.0	100.0	-	100.0	100.0	100.0	-
PRRSV (EU)[5]	-	-	-	-	-	-	-	-	-	-
Rotavirus serogroup A	14.3	30.0	0.0	13.3	25.0	0.08	5.0	15.0	20.0	0.32
Rotavirus serogroup B	0.0	0.0	0.0	6.7	12.5	0.38	5.0	5.0	0.0	0.44
Rotavirus serogroup C	0.0	20.0	10.0	13.3	0.0	0.38	10.0	5.0	15.0	0.56
Salmonella spp. B[6]	42.9[ab]	10.0[b]	15.0[b]	40.0[ab]	62.5[a]	0.02	25.0	30.0	35.0	0.79
Streptococcus suis	0.0	20.0	15.0	0.0	0.0	0.11	10.0	5.0	10.0	0.79
Week 22 post-weaning										
n =	7	16	17	13	7		20	20	20	
Incidence, %										
Brachyspira spp.	57.1[a]	12.5[ab]	17.6[ab]	7.7[b]	0.0[b]	0.05	0.0[b]	30.0[a]	20.0[ab]	0.01
Haemophilus parasuis	0.0	6.3	0.0	0.0	0.0	0.61	0.0	0.0	5.0	0.33
Lawsonia intracellularis	0.0	0.0	0.0	0.0	0.0	-	0.0	0.0	0.0	-
Pasteurella multocida A	0.0	6.3	5.9	0.0	0.0	0.65	5.0	5.0	0.0	0.44
PCV2[3]	0.0	18.8	17.6	30.8	0.0	0.15	35.0[a]	5.0[b]	10.0[b]	0.03
PRRSV (NA)[4]	42.9	31.3	35.3	15.4	0.0	0.14	25.0	20.0	35.0	0.55
PRRSV (EU)[5]	0.0	0.0	0.0	0.0	0.0	-	0.0	0.0	0.0	-
Salmonella spp. B	14.3	0.0	11.8	7.7	0.0	0.37	5.0	10.0	5.0	0.78

[1]There were no ($P > 0.38$) birth weight category × transition ADG category interactions.
[2]Linear effect for birth weight, $P = 0.04$.
[3]Porcine Circovirus Type 2
[4]Porcine Reproductive and Respiratory Syndrome Virus, North American genotype
[5]Porcine Reproductive and Respiratory Syndrome Virus, European genotype
[6]Quadratic effect for birth weight, $P = 0.02$.
[ab]Means within a row that do not share a common superscript differ $P < 0.05$.

Table 7. Correlations between pathogen incidence and birth weight or transition ADG.

	Correlations with Birth Weight		Correlations with Transition ADG	
Variable	Coefficient	*P* =	Coefficient	*P* =
Week 3 post-weaning				
Arcanobacterium pyogenes	-	0.65	-	0.51
Escherichia coli haemolytic	-	0.09	-	0.94
Haemophilus parasuis	-	0.27	-	0.73
PCV2[1]	-	0.58	-	0.35
PRRSV (NA)[2]	-	-	-	-
PRRSV (EU)[3]	-	-	-	-
Rotavirus serogroup A	-	0.91	-	0.14
Rotavirus serogroup B	-	0.06	-	0.12
Rotavirus serogroup C	-	0.93	-	0.50
Salmonella spp. B[3]	-	0.46	-	0.12
Streptococcus suis	-	0.68	-	0.34
Week 22 post-weaning				
Brachyspira spp.	-0.881	0.05	-	0.65
Haemophilus parasuis	-	0.63	-	1.00
Lawsonia intracellularis	-	-	-	-
Pasteurella multocida A	-	0.71	-	0.26
PCV2[1]	-	0.77	-	0.43
PRRSV (NA)[2]	-	0.07	-	0.17
PRRSV (EU)[3]	-	-	-	-
Salmonella spp. B	-	0.36	-	0.25

[1]Porcine Circovirus Type-2
[2]Porcine Reproductive and Respiratory Syndrome Virus, North American genotype
[3]Porcine Reproductive and Respiratory Syndrome Virus, European genotype

Table 8. Effects of birth weight or transition ADG category on lesions incidence[1]

	Birth Weight Category, kg						Transition ADG Category			
	< 1.00	1.00 to 1.25	1.26 to 1.50	1.51 to 1.75	> 1.76	*P* =	10th	30th	70th	*P* =
Week 3 post-weaning										
n =	7	10	20	15	8		20	20	20	
Lesion incidence, %										
Brain	0.0	0.0	5.0	6.7	0.0	0.69	5.0	5.0	0.0	0.44
Heart	14.3	20.0	15.0	33.3	25.0	0.74	20.0	30.0	15.0	0.51
Kidney	0.0	0.0	0.0	0.0	0.0	-	0.0	0.0	0.0	-
Large intestine[2]	71.4	40.0	40.0	53.3	50.0	0.64	65.0[a]	55.0[a]	25.0[b]	0.03
Liver	85.7	70.0	55.0	80.0	62.5	0.43	75.0	65.0	65.0	0.73
Lung	100.0	100.0	100.0	100.0	100.0	-	100.0	100.0	100.0	-
Lymph nodes	85.7	70.0	90.0	93.3	100.0	0.30	85.0	90.0	90.0	0.86
Nasal turbinate	12.9	60.0	30.0	60.0	25.0	0.24	45.0	55.5	30.0	0.27
Pancreas	0.0	0.0	0.0	0.0	0.0	-	0.0	0.0	0.0	-
Small intestine	71.4	50.0	55.0	53.3	75.0	0.73	65.0	65.0	45.0	0.34
Spleen	0.0	0.0	5.0	6.7	12.5	0.64	5.0	5.0	5.0	1.00
Stomach[3]	28.6[a]	10.0[ab]	15.0[ab]	0.0[b]	0.0[b]	0.05	10.0	15.0	5.0	0.56
Thymus[4]	0.0[b]	10.0[ab]	0.0[b]	0.0[b]	37.5[a]	0.02	15.0	5.0	0.0	0.10
Week 22 post-weaning										
n =	7	16	17	13	7		20	20	20	
Lesion incidence, %										
Brain	0.0	0.0	0.0	0.0	0.0	-	0.0	0.0	0.0	-
Heart	0.0	0.0	0.0	7.7	0.0	0.54	0.0	0.0	5.0	0.33
Kidney	14.3	18.8	17.6	7.7	14.3	0.92	15.0	20.0	10.0	0.67
Large intestine	100.0	81.3	58.8	61.5	71.4	0.12	60.0	85.0	70.0	0.20
Liver	28.6	18.8	17.6	0.0	14.3	0.26	10.0	25.0	10.0	0.33
Lung[5]	100.0	93.8	94.1	92.3	100.0	0.79	85.0[b]	100.0[a]	100.0[a]	0.03
Lymph nodes	14.3	0.0	0.0	0.0	14.3	0.20	5.0	0.0	5.0	0.44
Nasal turbinate	14.3	12.5	17.6	7.7	28.6	0.80	10.0	15.0	20.0	0.67
Pancreas	42.9	6.3	17.6	23.1	14.3	0.34	15.0	20.0	20.0	0.89
Small intestine	0.0	0.0	0.0	0.0	0.0	-	0.0	0.0	0.0	-
Spleen	0.0	0.0	0.0	0.0	0.0	-	0.0	0.0	0.0	-
Stomach[6]	14.3	12.5	5.9	7.7	0.0	0.74	20.0[a]	0.0[b]	5.0[b]	0.04
Thymus	0.0	0.0	0.0	0.0	0.0	-	0.0	0.0	0.0	-

[1]There were no ($P > 0.39$) birth weight category × transition ADG category interactions. If lesion incidence was significant, lesion severity was analyzed according to the following scale: 0 = no lesion, 1 = mild, 2 = moderate, 3 = severe.

[2]Linear effect for transition ADG, $P = 0.01$. Severity scores of lesions were significant for ADG ($P = 0.02$; 1.15, 0.75, 0.16 for 10th, 30th, and 70th percentiles, respectively; SEM = 0.239).

[3]Linear effect for birth weight, $P = 0.03$. Severity scores of lesions were not significant ($P = 0.21$) for birth weight.

[4]Severity scores of lesions were significant for birth weight ($P = 0.01$; 0.00, 0.07, 0.00, 0.00, 0.33 for < 1.00, 1.00 to 1.25, 1.26 to 1.50, 1.51 to 1.75, and >1.76, respectively; SEM = 0.081).

[5]Severity scores of lesions were not significant ($P = 0.60$) for transition ADG.

[6]Severity scores of lesions were significant for ADG ($P < 0.0001$; 0.49, 0.00, 0.04 for 10th, 30th, and 70th, respectively; SEM = 0.069).

[ab]Means within a row that do not share a common superscript differ $P < 0.05$.

Table 9. Effects of birth weight or transition ADG category on immune marker concentrations at 3 weeks post-weaning[1,2]

	Birth Weight Category, kg							Transition ADG Category			
	< 1.00	1.00 to 1.25	1.26 to 1.50	1.51 to 1.75	> 1.76	Pooled SEM	*P* =	10th	70th	Pooled SEM	*P* =
n =	6	6	14	9	5			20	20		
Serum											
IgA, ng/mL	482	564	558	592	539	83.2	0.54	532	571	68.7	0.94
IL-1β , ng/mL	0.126	0.115	0.158	0.091	0.104	0.0550	0.84	0.129	0.109	0.3542	0.70
IL-8, ng/mL	0.452	0.426	0.570	0.558	0.607	0.1385	0.77	0.508	0.538	0.0894	0.66
Total GSH, nM/mL	6,247	6,432	6,340	6,984	6,543	722.0	0.94	6,833	6,201	468.7	0.45
Ileal mucosa											
IgA, ng/g	15,236	30,947	32,220	25,102	50,434	12,761.7	0.64	24,594	36,879	8,254.3	0.70
IL-1β, ng/g	48	54	53	53	53	3.3	0.81	54	50	2.2	0.21
IL-8, ng/g	1370	1460	1410	1520	1450	97.0	0.86	1500	1390	63.0	0.22
Total GSH, nM/g	578	530	543	530	517	33.4	0.80	523	554	21.5	0.36

[1]There were no ($P > 0.15$) birth weight category × transition ADG category interactions.
[2]Analysis was performed after log transformation, but means and standard errors represent untransformed values.

Table 10. Effects of birth weight or transition ADG category on liver mineral concentration at 3 weeks post-weaning[1]

	Birth Weight Category, kg							Transition ADG Category				
	< 1.00	1.00 to 1.25	1.26 to 1.50	1.51 to 1.75	> 1.76	Pooled SEM	*P* =	10th	30th	70th	Pooled SEM	*P* =
n =	7	10	20	15	8			20	20	20		
Concentration, µg/g												
Cadmium	0.005	0.005	0.006	0.006	0.005	0.0006	0.63	0.005	0.005	0.006	0.0004	0.67
Calcium	72	98	95	76	75	45.0	0.16	83	91	82	6.3	0.49
Chromium	0.10	0.10	0.10	0.08	0.06	0.027	0.74	0.12	0.07	0.08	0.018	0.12
Cobalt	0.006	0.006	0.006	0.005	0.006	0.0004	0.15	0.006	0.005	0.006	0.0012	0.16
Copper	8	12	13	12	10	2.2	0.56	13	10	11	1.6	0.45
Iron[2]	195[a]	146[ab]	127[ab]	85[b]	107[ab]	18.7	0.004	165[a]	122[ab]	84[b]	13.4	0.0004
Magnesium	199	208	213	198	204	7.2	0.42	209	203	205	5.4	0.77
Manganese	2.3	2.4	2.8	2.6	2.7	0.15	0.11	2.5	2.5	2.8	0.11	0.09
Molybdenum	0.46	0.54	0.63	0.48	0.52	0.066	0.25	0.53	0.54	0.55	0.049	0.99
Phosphorus	2,790	2,886	3,115	2,814	2,899	126.9	0.21	3,028	2,926	2,841	94.5	0.38
Potassium	3,207	3,390	3,388	3,287	3,239	113.1	0.70	3,325	3,348	3,292	82.71	0.89
Selenium	0.49	0.53	0.56	0.48	0.50	0.036	0.43	0.53	0.51	0.51	0.027	0.86
Sodium	636	782	755	679	670	52.9	0.23	699	774	671	38.5	0.16
Zinc	246	256	265	247	213	27.4	0.74	253	241	256	20.0	0.85

[1]There were no ($P > 0.26$) birth weight category × transition ADG category interactions.
[2]Linear effects for both birth weight and transition ADG, $P < 0.01$.
[ab]Means within a row that do not share a common superscript differ $P < 0.05$.

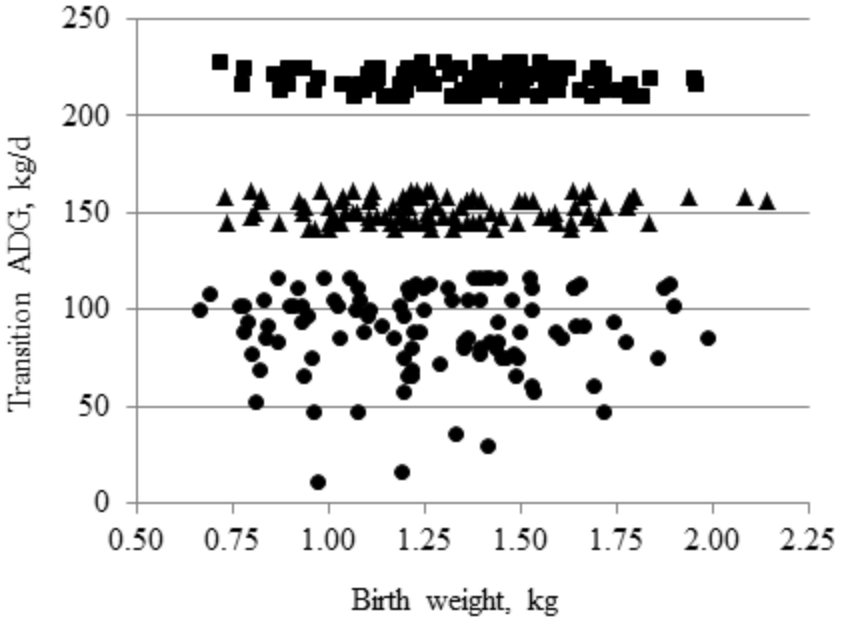

Figure 1. Birth weight × transition ADG interaction. Pigs were from the 10th (●), 30th (▲), or 70th (■) percentile of transition ADG.

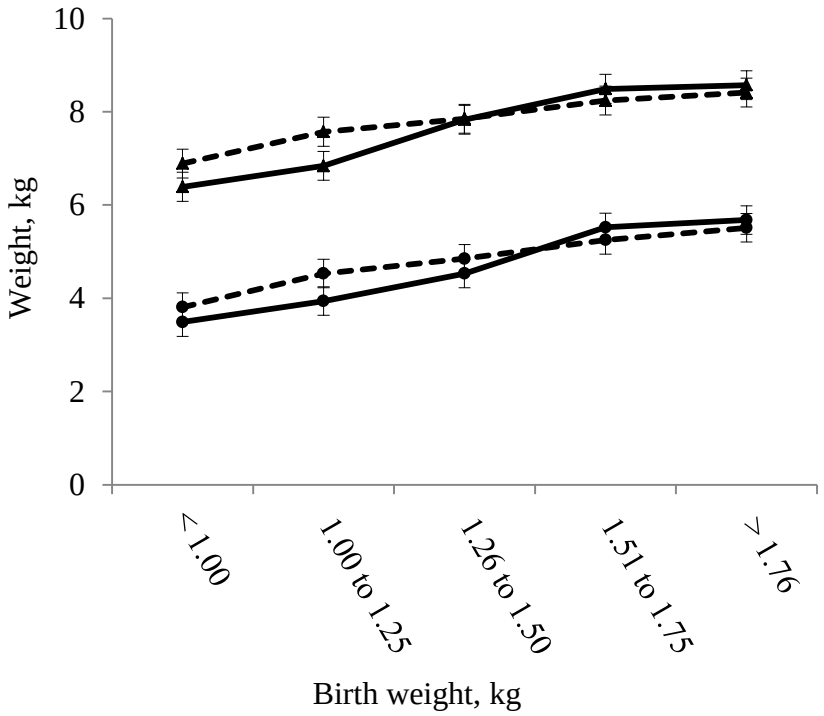

Figure 2. Differences in mean weight at 0 or 3 weeks post-weaning according to birth weight category × gender interaction. Pigs were barrows (solid line) or gilts (dashed line). Lines represent weights at week 0 (●) or 3 (▲) post-weaning. Error bars = SEM.

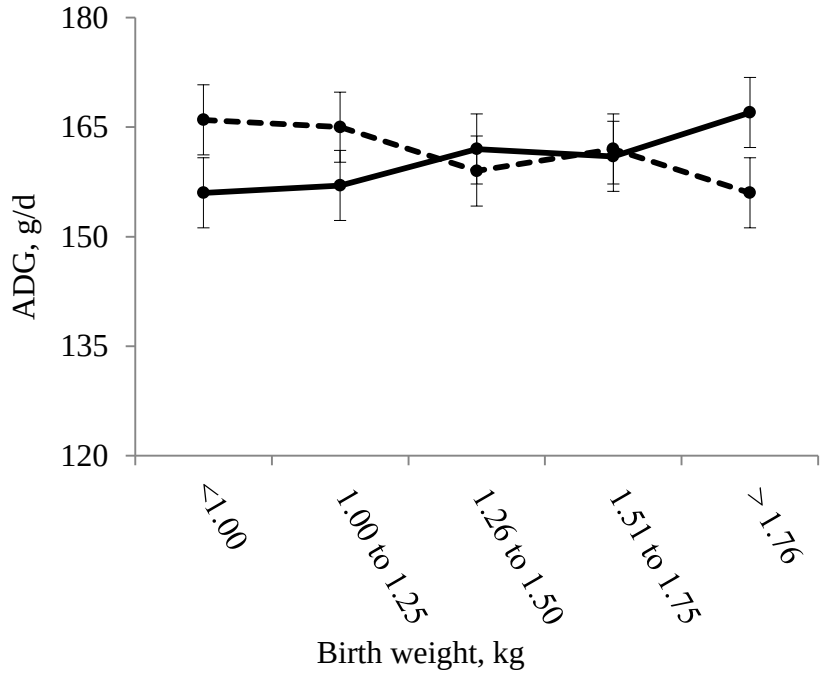

Figure 3. Differences in transition ADG according to birth weight category × gender interaction. Pigs were barrows (solid line) or gilts (dashed line). Error bars = SEM.