

Poor Weaning Transition ADG in Pigs is not Correlated with Pathological or Immunological Markers of Enteric Disease during a PRRSV Outbreak

A.S. Leaflet R2808

Cassandra Jones, Research Associate;
John Patience, Professor;
Nicholas Gabler, Assistant Professor,
Department of Animal Science;
Rodger Main, Veterinary Diagnostic Laboratory
Director of Operations;
Darin Madson, Assistant Professor,
Veterinary Diagnostic & Production Animal Medicine

Summary and Implications

Previous research suggests that enteric disease and gut health interact to decrease pig performance. Our objective was to determine if light birth weight (BRW) pigs or those from the bottom 10th percentile of transition ADG (tADG) have a higher incidence of pathogen presence or enteric lesions than heavier or faster-growing contemporaries. A total of 1,500 pigs were weighed at birth and divided into 5 BRW categories: <1 kg, 1-1.25 kg, 1.26-1.5 kg, 1.51-1.75 kg, >1.76 kg. At weaning, 1,054 random pigs were moved to a commercial wean-to-finish barn. Pigs were weighed individually at 0 and 3 weeks post-weaning. Gain from 0 to 3 weeks post-weaning was calculated and termed tADG. Pigs from 3 tADG percentiles were of interest: 10th, 30th, and 70th. Forty pigs from each of the 3 tADG percentiles were matched for sex, litter size, and sow parity, but not BRW to create 20 matched sets totaling 60 pigs. 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. At 3- and 22-weeks post-weaning, pigs were euthanized for organ system tissue evaluation. Lung, lymph node, and digesta were analyzed for presence of various pathogens by PCR and culture methods. Serum and ileal mucosa were analyzed for markers of immunological or oxidative stress. Data were analyzed using PROC CORR, GENMOD, and GLIMMIX, where pig served as the experimental unit. There was no correlation ($P > 0.12$) between tADG and pathogen presence at either 3- or 22-weeks post-weaning. However, *Brachyspira* spp. was negatively correlated ($P = 0.05$; $\text{Corr.} = -0.881$) with birth weight. Neither birth weight ($P > 0.54$) nor tADG ($P > 0.20$) affected markers of immunological or oxidative stress in either serum or ileal mucosa at 3- or 22-weeks post-weaning. In summary, poor tADG is not correlated with the pathogens or immunological markers of enteric disease measured in this study.

Introduction

Disease still poses significant economic and animal welfare concerns to the swine industry. Pathogen elimination would be the most ideal way to control disease. However, this is often unfeasible. 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 hypothesized to harbor pathogens that assault healthy pigs sharing the same space. If this is true, rearing fallback pigs separately from their contemporaries may improve the overall enteric health and performance of the population. However, we first need to determine if fallback pigs are sources of disease within a barn. Our objectives were to determine if, compared to their heavier or faster-growing contemporaries, light birth weight pigs or those from the bottom 10th percentile of tADG have a greater incidence of 1) pathogen presence or 2) gastrointestinal lesion presence or severity.

Materials and Methods

This study was conducted at a commercial sow farm and wean-to-finish facility in Iowa under the approval of the Institutional Animal Care and Use Committee (#2-11-7095-S). In a commercial sow farm, a total of 1,500 pigs were weighed immediately at birth, individually tagged, and divided into 5 birth weight categories: <1 kg, 1-1.25 kg, 1.25-1.5 kg, 1.5-1.75 kg, >1.75 kg. At weaning, 1,054 random pigs were moved to a commercial wean-to-finish research barn with 40 pens. All pigs were weighed individually at 0 and 3-weeks post-weaning. Gain during this period was calculated and termed 'transition ADG.' Pigs from 3 transition ADG percentiles were of interest: 10th, 30th, and 70th.

Forty pigs from each of these three transition ADG categories were matched for sex, litter number born alive, and sow parity to create 20 matched sets for 60 total pigs. At each of 3- and 22-weeks post-weaning, 20 sets of pigs were humanely euthanized and necropsied by veterinary pathologists. Fresh tissue samples and digestive contents were collected and analyzed by PCR or culture for incidence of common North American porcine pathogens. Tissues were also fixed in formalin, routinely processed, and evaluated for lesion incidence and severity by examining inflammation, necrosis, degeneration, and atrophy.

Data were analyzed using the CORR, GENMOD, and GLIMMIX procedures of SAS (SAS Inst. Inc., Cary, NC), with pig as the experimental unit. The fixed effects were birth weight and transition ADG. There were no birth

weight \times transition ADG interactions ($P > 0.15$). Results were considered significant if $P < 0.05$.

Results and Discussion

Birth weight and transition ADG had little effect on the bacteria or viruses burden in the pigs used in this study. At week 3 post-weaning, there was no effect ($P > 0.08$) 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 ($P = 0.04$). The direction of this effect was unexpected. Meanwhile, pigs with BRW from 1.00 to 1.50 kg had a decreased ($P < 0.05$) incidence of *Salmonella spp. B* compared to pigs with BRW > 1.75 , resulting in a quadratic effect ($P = 0.02$). We would have expected *Salmonella spp. B* incidence to decrease with increasing BRW.

There was no effect ($P > 0.31$) of tADG on the presence of bacteria or viruses at 3-weeks post-weaning. This was unexpected, as we had hypothesized that fallback pigs had a greater incidence of pathogens than their faster-growing contemporaries. We have found no other data regarding the effect of pathogen presence on the prevalence fallback pigs. However, Huang et al. (2012) reported that PFTS was not ($P > 0.05$) caused by any pathogens tested in their experiment, including pathogenic *E. coli*, attaching and effacing *E. coli*, *Streptococcus suis*, *Haemophilus parasuis*, *Brachyspira hyodysenteriae*, PCV2, PRRSV, Influenza A virus, rotavirus A, 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 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. *Brachyspira spp.* was strongly and negatively correlated with BRW ($P = 0.05$; Correlation = - 0.881). We have been unable to find a similar response in the literature, but increasing birth weight is associated with improving intestinal flora development. There were no ($P > 0.14$) 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. We did not expect a response in PCV2 prevalence because all pigs were vaccinated at 6 weeks of age (4 weeks post-weaning). However, the direction of the response was expected because poor post-weaning growth is characteristic of both PCV2 and the related post-weaning multisystemic wasting syndrome. 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 ($P > 0.43$).

Another method to determine severity of pathogen load is to determine the concentration of markers of immunological or oxidative stress. An immune response results in the production of pro-inflammatory cytokines, the production of which require energy and nutrients that may have otherwise been used for growth. Bacteria, such as the *E. coli* and *Salmonella* that were present in this experiment produce endotoxins, which increase cell sensitivity to an immune response and the ensuing production of pro-inflammatory cytokines. Additionally, Moeser et al. (2012) has recently reported that pigs with PFTS have decreased epithelial barrier function and altered mucosal morphology that were not only explained by low feed intake. Thus, the mucosal immune system is an integral part of immunity. However, there was no link ($P > 0.20$) between BRW or tADG and serum of ileum mucosa IgA, IL-1 β , 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. This lack of effect was expected, given the poor correlation between pathogen incidence and BRW or tADG. Our values appear similar to those reported previously in the literature. However, there is very limited immune marker data with respect to mucosal 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.

In summary, our data suggest that *Brachyspira spp.* infection is negatively correlated with BRW. However, poor tADG in pigs is not correlated with pathogens or immunological markers of enteric disease during a PRRSV outbreak.

Acknowledgements

We gratefully acknowledge the Iowa Pork Producers Association for funding this experiment (Project # 11-084) and Iowa Select Farms, LLC for other in-kind contributions.