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JSHAP SPOTLIGHT

Dr Yolande Seddon

University of Saskatchewan

Dr Yolande Seddon earned a BS ('05) from the University of Central Lancashire, an MS ('06) from the University of Essex, and a PhD ('11) from Newcastle University. Dr Seddon is an assistant professor and the NSERC Industrial Research Chair in Swine Welfare at the University of Saskatchewan where she conducts applied research to contribute knowledge to help develop solutions to swine welfare challenges. Dr Seddon joined the JSHAP Editorial Board to support publication of high quality science on the topic of swine behavior and welfare in the *Journal of Swine Health and Production*. She encourages JSHAP contributors to consider their audience and the message, adhere to high standards of work, follow the journal guidelines, and enjoy the process of communicating their work!

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Lessons from the crew

Over the past two years, I have had the opportunity to learn, observe, and become a fan of women's collegiate rowing. While traveling many miles across the country in recent months as a fan, the similarities between the sport and our preparation, response, and recovery to the introduction of a transboundary disease has become apparent. For most of these athletes, participation in the sport begins when they arrive at college with no prior experience. All of them had been successful athletes in other sports and now face a steep technical learning curve under more challenging physical conditioning than most had experienced in the past.

Similarly, most swine veterinarians have experience with the successful management of endemic diseases, but most have not experienced a transboundary disease or been directly involved in the response to an introduction. These veterinarians would face a similar steep learning curve to better understand the disease and learn from the experiences of others to prepare and plan. In both cases, the "I have never done this before, but I am going to continue to learn, practice, and prepare" philosophy is similar. The continued dedication to increasing technical knowledge as well as repeated practice and improvement leads to a better outcome during the race, or introduction of a transboundary disease.

Complacency destroys progress resulting in less favorable outcomes. As a profession, we must continue to challenge each other and our preparedness and response plans to assure that we, and the producers who we serve, are best prepared to keep these diseases from entering the country and respond rapidly and efficiently if they are introduced. Members of AASV have had, and continue to play, an integral role in the development of these plans including the Secure Pork Supply (SPS) Continuity of Business Plan, the Certified Swine Sample Collector (CSSC) training program, and the US Swine Health Improvement Plan (USSHIP).

In rowing, a boat consists of either 4 or 8 rowers plus a coxswain. While each of the rowers have slightly different technical roles, they must function as a single synchronous unit to efficiently propel the boat forward to be successful in competition. The rowers represent all the varied plans and participants in transboundary disease preparedness and response including producers, federal and state animal health officials, National Animal Health Laboratory Network, SPS, CSSC, and USSHIP. While all these groups and programs have different roles, they all must operate synchronously and to the best of their capability to assure exceptional preparedness and response. The coaches are critical in the preparation for the race, orchestrating technical training, physical conditioning, and nutrition through repeated practice and continual improvement. The coach's role is limited once the race starts, and success is dependent on the team in the boat to put all the practice and preparation into action against their competitors. The race is analogous to the response to a transboundary disease introduction, with the outcome being dependent on the ability to synchronously apply all the preparation and planning from many individuals and groups.

"In a sport like this—hard work, not much glory, but still popular in every century—well, there must be some beauty which ordinary men can't see, but extraordinary men do."

George Yeoman Pocock

The coxswain is the person who directs the team during the race and serves as the eyes, ears, and mouth of the boat. They become the "coach in the boat!" The swine veterinarian is similar to the coxswain, as they are the common link between the producer, animal health officials, biosecurity, disease surveillance, and regulated animal movements. The coxswain lets the rowers know where they are in relationship to the other boats and how much farther they have to go. A coxswain must know rowing techniques so that if a correction is necessary, he or she will know what to do and why to do it. Similarly, the swine veterinarian has the broad technical knowledge and involvement in all aspects of preparation and response that allows them to assess the current situation and provide coordination and guidance to adapt the response to the ever-changing situation. Like the coxswain, the veterinarian must be a good motivator, provide guidance, and facilitate coordination and cooperation to result in success. As we continue to prepare, it is important to know what seats are on the boat, and the role each seat plays. More importantly, it is critical that all the seats continue to communicate and coordinate to assure a positive outcome.

Mike Senn, DVM, MS
AASV President


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It's probably nothing

"ASF will likely be endemic by the time we identify its introduction." That was a statement made by the US Department of Agriculture (USDA) during a recent African swine fever (ASF) working group call. Although probably accurate, it was a little disheartening to hear it stated out loud. It is a statement of recognition of something we all already suspected - the swine industry is likely to have a huge problem on its hands from the start of an ASF outbreak.

That realization highlights the need to identify an ASF virus introduction as quickly as possible. The speed with which we identify those early cases depends on the robustness and effectiveness of the surveillance strategy in place prior to the outbreak. That strategy relies on two key factors: 1) the ability to detect the virus in samples submitted to the veterinary diagnostic laboratories (VDLs) as part of the routine USDA ASF surveillance program and 2) our ability to observe signs consistent with ASF in the field and report those observations to the appropriate animal health officials. The first factor operates mostly behind the scenes and routinely analyzes case-compatible samples submitted to the VDLs from a variety of resource streams. It is designed to work regardless of whether the submitter suspects ASF, and functions largely without relying on the observational skills of producers or veterinarians. It is the second factor I want to talk about in this article.

Much of our hopes to diagnose ASF as early as possible rely on the ability of producers and veterinarians in the field to observe something out of the ordinary and react to it. I have no doubt that any one of us that suspected they were facing a case of ASF would immediately reach out to the appropriate animal health official and report our suspicions. What worries me is that I am not sure we consider ASF as a possible cause for the oddities we see every day.

For months, I have participated in working group calls aimed at enhancing our ability to detect ASF in the field. All those efforts are based on people in the barns recognizing something is different from normal and reporting it. But, what is "normal" versus "different?" We all know that there is significant variability between farms, barns, groups, genetics, seasons, etc when evaluating production parameters like morbidity and mortality. Veterinarians and producers are busy people. "It's probably just PRRS or influenza and I really need to get home at a reasonable time tonight. I'll check on them tomorrow." How do you know when to pull the trigger and call the state or federal animal health official?

I understand that routine variability means that the folks in the field must make judgement calls. What is worrisome to me are the times we hear about very dramatic shifts in the norm and still no one reports it. Just recently, we have heard reports of significantly elevated rates of condemnations at a packing plant but no notification to the folks responsible for tracking disease for weeks. In another instance, high levels of mortality were observed in multiple barns in the Midwest without any report to animal health officials or initiation of a foreign animal disease (FAD) investigation. Fortunately, neither of these turned out to be an FAD but that is hindsight and exactly why ASF will be endemic upon detection.

If we are serious about doing everything we can to minimize the impact of an FAD introduction, everyone in the production chain must stop being afraid to report something abnormal. The USDA should be doing a lot more FAD investigations than they are currently doing. According to data on the USDA ASF/CSF Surveillance website, the Animal and Plant Health Inspection Service only conducted 27, 48, 32, and 53 swine-focused FAD investigations in Fiscal Years 2019, 2020, 2021, and 2022 respectively.[1] Since June 1, 2019, only 8190 specimens have been submitted to the VDLs from commercial herds as part of the ASF/CSF Surveillance Program.[1]

Those numbers are way too low. Do the math – an average of 40 investigations since 2019 on over 60,000 swine farms and only 8190 specimens from more than 330 million hogs marketed. Is that really the best we can do and claim to be concerned about finding that first case? Observational surveillance can be, and must be, a significant and effective tool in the surveillance strategy. Remember porcine epidemic diarrhea virus? It was a veterinarian in the field who was willing to stand up and ask the questions about why things were different that called attention to that outbreak. Everyone who sees pigs in the field must be empowered to raise the alarm when there are suspicions that things just aren't "normal."

Harry Snelson, DVM
Executive Director

Reference

*1. US Department of Agriculture. ASF & CSF Executive Summary. Accessed May 31, 2022. **https://www.aphis.usda.gov/aphis/dashboards/tableau/asf-csf-exec-summary-dashboard**

* Non-refereed reference.

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Bon voyage and welcome aboard!

This spring, the journal officially said "Happy retirement and bon voyage" to our publications manager, Karen Richardson.

I hope you had the chance to read Karen's message in the previous issue but in case you did not know, Karen has been with the journal since 2001.[1] I know many of you have had the chance to meet Karen in person at the AASV Annual Meeting over the years. And many of you have worked with Karen directly with some aspect of the journal, be it as an author, a reviewer, or both. I have had the privilege of working with Karen for over 10 years at the journal and at the University of Guelph. As Karen embarks on her retirement endeavors, I personally have shed many tears as I will miss working together. I also feel fortunate that I live "close-ish" to Karen and will be able to continue to see her in a casual capacity as friends.

Karen - may your retirement be filled with joy and happiness. On behalf of the journal, I would like to say thank you for all your dedication and love for the journal and all things AASV.

With retirements come new and exciting beginnings! I am also very happy to announce that Rhea Schirm will be joining the journal as the incoming publications manager. And with that I would like to say, "Welcome aboard, Rhea!"

You will all have the opportunity to meet Rhea at upcoming meetings and through email communications you have with the journal. I am looking forward to working with Rhea and will let her introduce herself in an upcoming JSHAP issue. In the meantime, she will be working with Karen to learn the ropes and I invite you to extend a warm welcome to Rhea!

The journal continues to have a healthy line-up of quality manuscripts that span a wide range of swine health and production issues. I hope you enjoy this issue.

Terri O'Sullivan, DVM, PhD
Executive Editor

"Karen - may your retirement be filled with joy and happiness. On behalf of the journal, I would like to say thank you for all your dedication and love for the journal and all things AASV"

Reference

*1. Richardson K. My life with AASV and JSHAP [Editorial]. *J Swine Health Prod*. 2022;30(3):129.

* Non-refereed reference.

ORIGINAL RESEARCH PEER REVIEWED

Effects of different parenteral iron treatment regimens on hematology characteristics, serum concentrations of hepcidin, and growth performance in pigs fed nursery diets supplemented with copper

Mark J. Estienne, PhD; Kimberly A. Williams, BS; Nima K. Emami, PhD; Sherri G. Clark-Deener, DVM, PhD; Rami A. Dalloul, PhD

Summary

Objective: To determine the effects of iron treatments on hematology, hepcidin, and growth in weaned pigs fed copper-supplemented diets.

Materials and methods: Pigs were allocated to a 3 × 2 factorial arrangement of treatments (4 pens/treatment combination, 3 pigs/pen) with factors being intramuscular iron (200 mg at birth; 100 mg at birth and weaning [22.4 days of age]; or 100 mg at birth and 14 days of age) and dietary copper (14 [control] or 250 ppm [supplemented]). Blood was sampled at days 0, 7, and 49 post weaning.

Results: Pigs receiving 100 mg iron at birth and weaning, but not pigs in the other groups, had hemoglobin concentrations consistent with iron deficiency at day 0 (iron treatment × day, $P < .001$). For pigs receiving 100 mg iron at birth and 14 days of age, hepcidin concentrations were greater in control pigs than copper-supplemented pigs (iron treatment × diet, $P = .06$). A diet × day interaction ($P = .07$) existed for hepcidin, with concentrations greater in control vs copper-supplemented pigs on day 49. Pigs receiving iron at day 14 of age had the greatest ($P = .01$) weaning weights. Gain from day 0 to 7 was enhanced ($P = .03$) by 250 ppm copper but nursery performance (day 0-49) was unaffected by iron treatment.

Implications: Pigs receiving 100 mg iron at birth were iron deficient at weaning. Treatment with iron at 14 days of age could improve weaning weights and prevent iron deficiency at weaning. Age-related increases in hepcidin were decreased by additional copper supplementation.

Keywords: swine, performance, iron, copper, hepcidin

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Resumen - Efectos de diferentes regímenes de tratamiento con hierro parenteral sobre las características hematológicas, las concentraciones séricas de hepcidina, y el rendimiento del crecimiento en cerdos alimentados con dietas de destete suplementadas con cobre

Objetivo: Determinar los efectos de los tratamientos con hierro sobre la hematología, la hepcidina, y el crecimiento en cerdos destetados alimentados con dietas suplementadas con cobre.

Materiales y métodos: Los cerdos fueron asignados en un acomodo factorial de tratamientos 3 × 2 (4 corrales/combinación de tratamiento, 3 lechones/corral) con factores que fueron hierro intramuscular (200 mg al nacimiento; 100 mg al nacimiento y al destete [22.4 días de edad]; o 100 mg al nacimiento y 14 días de edad) y cobre dietético (14 [control] o 250 ppm [suplementado]). Se tomaron muestras de sangre los días 0, 7, y 49 después del destete.

Resultados: Los cerdos que recibieron 100 mg de hierro al nacimiento y al destete, pero no los cerdos de los otros grupos tenían concentraciones de hemoglobina compatibles con deficiencia de hierro el día 0 (tratamiento con hierro por día, $P < .001$). En los cerdos que recibieron 100 mg de hierro al nacimiento y a los 14 días de edad, las concentraciones de hepcidina fueron mayores en los cerdos control que en los cerdos suplementados con cobre (tratamiento con hierro × dieta, $P = .06$). Existió una interacción de dieta por día ($P = .07$) para la hepcidina, con concentraciones mayores en el control que en los cerdos suplementados con cobre en el día 49. Los cerdos que recibieron hierro en el día 14 de edad tuvieron los mayores ($P = .01$) pesos al destete. La ganancia del día 0 al 7 mejoró ($P = .03$) con 250 ppm de cobre, pero el rendimiento del destete (día 0-49) no se vio afectado por el tratamiento con hierro.

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Estienne MJ, Williams KA, Emami NK, Clark-Deener SG, Dalloul RA. Effects of different parenteral iron treatment regimens on hematology characteristics, serum concentrations of hepcidin, and growth performance in pigs fed nursery diets supplemented with copper. *J Swine Health Prod*. 2022;30(4):210-222. **https://doi.org/10.54846/jshap/1288**

Implicaciones: Los cerdos que recibieron 100 mg de hierro al nacimiento tenían deficiencia de hierro al destete. El tratamiento con hierro a los 14 días de edad podría mejorar los pesos al destete y prevenir la deficiencia de hierro al destete. Los aumentos de hepcidina relacionados con la edad se redujeron con suplementos adicionales de cobre.

Résumé - Effets de différents régimes de traitement parentéral au fer sur les caractéristiques hématologiques, les concentrations sériques d'hepcidine, et les performances de croissance chez les porcs nourris avec des régimes de pouponnière enrichis en cuivre

Objectif: Déterminer les effets des traitements au fer sur l'hématologie, l'hepcidine, et la croissance chez des porcs sevrés nourris avec des régimes enrichis en cuivre.

Matériels et méthodes: Les porcs ont été répartis selon un arrangement factoriel 3 × 2 de traitements (4 enclos/combinaison de traitement, 3 porcs/enclos) les facteurs étant le fer intramusculaire (200 mg à la naissance; 100 mg à la naissance et au sevrage [22.4 jours d'âge]; ou 100 mg à la naissance et à 14 jours d'âge) et du cuivre alimentaire (14 [témoin] ou 250 ppm [supplémenté]). Du sang a été prélevé aux jours 0, 7, et 49 après le sevrage.

Résultats: Les porcs recevant 100 mg de fer à la naissance et au sevrage, mais pas les porcs des autres groupes, présentaient des concentrations d'hémoglobine compatibles avec une carence en fer au jour 0 (traitement au fer par jour, $P < .001$). Pour les porcs recevant 100 mg de fer à la naissance et à l'âge de 14 jours, les concentrations d'hepcidine étaient plus élevées chez les porcs témoins que chez les porcs supplémentés en cuivre (traitement au fer × alimentation, $P = .06$). Une interaction régime/jour ($P = .07$) existait pour l'hepcidine, avec des concentrations plus élevées chez les témoins que chez les porcs supplémentés en cuivre au jour 49. Les porcs recevant du fer au jour 14 avaient les poids au sevrage les plus élevés ($P = .01$). Le gain du jour 0 au jour 7 a été amélioré ($P = .03$) par 250 ppm de cuivre mais la performance en pouponnière (jour 0-49) n'a pas été affectée par le traitement au fer.

Implications: Les porcs recevant 100 mg de fer à la naissance présentaient une carence en fer au sevrage. Un traitement au fer à l'âge de 14 jours pourrait améliorer le poids au sevrage et prévenir la carence en fer au sevrage. Les augmentations d'hepcidine liées à l'âge ont été réduites par une supplémentation additionnelle en cuivre.

Iron deficiency anemia develops in suckling pigs unless exogenous iron is supplied early in life. On commercial sow farms, neonatal pigs are treated intramuscularly (IM) with iron dextran or gleptoferron, and doses of 100 to 200 mg have been used to prevent iron deficiency anemia.[1-3] Modern sows, however, produce large litters of pigs with capacity for rapid preweaning growth. Recent reports have indicated that despite iron supplementation given during the first week of life, many pigs, particularly the largest, fastest-growing animals in a litter, are anemic or iron deficient at weaning.[4-7] Pigs that are anemic at weaning are more susceptible to disease[8] and exhibit slower nursery growth rates.[5,9] The economic impact of iron deficiency on US pork production is estimated to be $46 to $335 million annually.[10]

Thus, there is renewed interest in the iron status of weaned pigs. This could be particularly important if growth-promoting levels of copper (200 to 250 ppm)[11-13] are used in nursery diets as pharmacological levels of copper may decrease iron absorption,[14,15] and perhaps exacerbate an iron deficient condition. Treatment with IM iron doses in excess of 200 mg could be toxic to some pigs,[16] encourage bacterial growth and susceptibility to infection,[17] or cause increased release of hepcidin, a hormone secreted by the liver that inhibits iron absorption.[18,19] Another strategy for increasing blood iron concentrations in nursery pigs is to alter the number and timing of injections of iron.[20] Thus, the objective of this study was to determine the effects of various iron treatment regimens on hematology, circulating hepcidin concentrations, and growth performance in nursery pigs fed copper-supplemented diets.

Animal care and use

The protocol for this experiment was reviewed and approved by the Institutional Animal Care and Use Committee at Virginia Tech (Blacksburg, VA).

Materials and methods

Study animals and housing

Eight Yorkshire × Landrace sows farrowed 75 Duroc-sired pigs, of which 72 pigs (38 males and 34 females) were used in this experiment. Three pigs were laid on by the sow prior to processing. Within 24 hours after birth, pigs were ear notched for identification, weighed, needle teeth were resected, and tails docked. No antibiotics were administered at processing or during the lactation and nursery periods. Pigs were transferred (n = 5) among litters so that sows were nursing an approximately equal number of pigs (9.0 ± 0.6 piglets). Boars were castrated at 7 days of age using a sterile scalpel. Pigs were not given creep feed during the suckling period.

The mean (SE) weaning age was 22.4 (0.2) days when pigs were moved to an environmentally controlled nursery facility. Each nursery pen measured 0.91 × 1.22 m over galvanized steel bar slats and contained a nipple drinker and a stainless-steel feeder with four feeding spaces.

Study design

Iron hydrogenated dextran (Iron-100; Durvet, Inc) was administered to pigs as an IM injection in the neck muscle behind the ear using a 20-gauge, 1.27-cm long needle. The following three iron treatment regimens were employed: 1) 200 mg iron at initial processing (birth); 2) 100 mg iron at birth and at weaning; and 3) 100 mg iron at birth and at 14 days of age.

Four blocks were created by placing 18 pigs in 6 pens (3 pigs/pen) in each block. Pens were balanced for body weight (BW), sex, and litter of origin. Pens within blocks were randomly allocated to a 3 × 2 factorial arrangement of treatments. The factors were 1) iron treatment (one of three treatments as previously described) and 2) level of dietary copper (14 [control] or 250 ppm [copper-supplemented]). There were four replicate pens per treatment combination (total of 24 pens). The sample size selected was needed to detect a 12.5% difference in performance with a coefficient of variation of 5%, assuming 80% power and a 5% significance level.

Experimental diets

Pigs were allowed *ad libitum* access to a phase feeding regimen with all diets meeting the requirements for the

Table 1: Composition of copper-supplemented and control diets fed to nursery pigs for 49 days*

	Dietary phase:	I	II	III
Ingredient, %	**Days fed relative to weaning:**	**0 - 7**	**8 - 21**	**22 - 49**
Ground corn		42.13	54.94	64.94
Soybean oil		3.00	3.00	3.00
Dried whey		25.00	10.00	0.00
Menhaden fish meal		4.00	2.00	0.00
Soycomil†		3.00	2.00	2.00
Soybean meal		19.85	24.90	26.65
Dicalcium phosphate		1.00	1.00	1.25
Calcium carbonate		0.70	1.00	1.00
Salt		0.20	0.20	0.20
Lysine-HCL		0.40	0.30	0.30
DL-methionine‡		0.12	0.06	0.06
Vitamin-trace mineral§		0.50	0.50	0.50
Copper sulfate or ground corn		0.10	0.10	0.10
Totals		**100.00**	**100.00**	**100.00**
Calculated analysis, %				
Crude protein		20.57	20.33	19.57
Lysine		1.53	1.37	1.27
Methionine		0.46	0.39	0.37
Calcium		0.88	0.83	0.74
Phosphorous		0.75	0.65	0.61

* Copper sulfate or control diets were prepared by mixing copper sulfate (Pestell Minerals and Ingredients) or ground corn, respectively, with basal diet consisting of the major portion of the ground corn and all other common ingredients. Control diets contained approximately 14.2 ppm copper, 113 ppm iron, and 113 ppm zinc.

† Archer Daniels Midland Co.

‡ Rhodimet NP 99.

§ ANS Swine Breeder Premix manufactured for Agri-Nutrition Services, Inc. Trace minerals in sulfate forms were in a polysaccharide complex.

various nutrients[21] and copper adjusted as previously indicated. For each of the three phases, a basal diet was first prepared containing most of the corn and all the common ingredients for each of the two experimental diets. Copper sulfate (Pestell Minerals and Ingredients) or an equal amount of ground corn was added to the basal diet to create the copper-supplemented or control diets, respectively (Table 1).

Data and sample collection and blood analyses

Pigs were weighed at weaning (day 0) and on days 7, 21, and 49 post weaning. Average daily gain (ADG) was determined for periods from day 0 to 7, day 8 to 21, day 22 to 49, and day 0 to 49. Feed additions were recorded so that for each period and the entire trial, average daily feed intake (ADFI) and the gain to feed ratio (G:F) could be determined.

A blood sample was collected from the barrow weighing closest to the mean pig weight in each pen at weaning (before receiving the weaning iron treatment), and at days 7 and 49 post weaning. The same pig was used on each collection day. Barrows were placed supine on a v-board and approximately 7 mL of blood was collected via jugular venipuncture (20-gauge, 2.54-cm long needle) into a vacutainer tube (Becton, Dickinson and Company) containing EDTA and a similar sized tube containing no anticoagulant.

Blood collected into tubes containing EDTA was used for hematology analyses using a Coulter Multisizer 3 cell counter (Beckman Coulter, Inc) by Animal Laboratory Services of the Virginia-Maryland College of Veterinary Medicine (Blacksburg, VA). The following hematological parameters were measured: number of red blood cells, reticulocytes, white blood cells, neutrophils, lymphocytes, monocytes, eosinophils, basophils, and platelets; hemoglobin concentration; hematocrit; mean corpuscular volume; mean corpuscular hemoglobin concentration; red blood cell distribution width; and mean platelet volume. Blood sample tubes containing no additive were allowed to clot for 24 hours at 4°C and serum was harvested following

30 minutes of centrifugation at 1820*g*. Serum concentrations of hepcidin were determined using a sandwich enzyme-linked immunoabsorbent assay kit (LS-F11619; LifeSpan BioSciences, Inc). Intra-assay coefficient of variation was 10% and assay sensitivity was 0.78 ng/mL.

Statistical analyses

Data were subjected to analysis of variance using the mixed-models procedure of SAS (SAS Institute Inc). Body weights, ADG, ADFI, and G:F were analyzed using a model that included iron treatment, diet, and iron treatment by diet interaction as possible sources of variation. Block was included as a random variable. Birth weight served as a covariate for BW at weaning (day 0) and BW at day 0 served as a covariate for BW at days 7, 21, and 49 post weaning. Pen was the experimental unit.

A repeated measures model was used for analyzing hematological characteristics and hepcidin. The model included iron treatment, diet, day, and all possible two- and three-way interactions as possible sources of variation. Block was included as a random variable and the individual pig was the experimental unit. Individual means were compared using the LSMEANS option of PROC MIXED and were adjusted using the Tukey-Kramer procedure. Effects were considered statistically significant at $P < .05$ with trends for significance at $P \leq .10$.

Results

Hematology characteristics

Table 2 reports hematology characteristics in nursery pigs as affected by the main effects of iron treatment, diet, and day post weaning. There were no effects of iron treatment by diet by day post weaning or iron treatment by diet for any hematology measure. The concentration of red blood cells ($P = .06$), hemoglobin concentrations ($P < .001$), hematocrit ($P < .001$), mean corpuscular volume ($P < .001$), mean corpuscular hemoglobin concentration ($P < .001$), red blood cell distribution width ($P < .001$), and reticulocyte percentage ($P = .01$) and number ($P = .03$) were affected by iron treatment by day post weaning (Figures 1 and 2A). Red blood cell concentration tended to increase ($P = .06$) from weaning to day 7 post weaning and then remained similar to day 49 in pigs receiving 100 mg iron at birth and weaning. In the other two iron groups, red blood cell concentrations were similar across days. On day 0, hemoglobin ($P < .001$), hematocrit ($P < .001$), mean corpuscular volume ($P < .001$), and mean corpuscular hemoglobin ($P = .02$) in pigs receiving 100 mg iron at birth and weaning were less compared with pigs from the other two iron groups. In contrast, red blood cell distribution width and the number and percentage of reticulocytes, were greater in pigs receiving 100 mg iron at birth and weaning compared to the other two iron groups on both day 0 ($P = .002$, $P = .03$, and $P = .07$, respectively) and day 7 ($P = .02$, $P = .03$, and $P = .03$, respectively). The injection of 100 mg iron in the iron-deficient pigs at weaning caused hemoglobin concentrations, hematocrit, and mean corpuscular volume to increase to normal levels by day 7 post weaning. However, these pigs had lower mean corpuscular hemoglobin and greater red blood cell distribution width and number and percentage of reticulocytes at 7 days post weaning than pigs in the other two iron groups.

There were tendencies for effects of diet by day post weaning for mean corpuscular volume ($P = .06$) and mean corpuscular hemoglobin concentrations ($P = .08$; Figures 3A and B). Mean corpuscular volume in pigs fed the control diet was similar on day 0 and day 7 and tended ($P = .06$) to increase from day 7 to day 49 post weaning. In contrast, mean corpuscular volume was similar among days in 250 ppm copper-fed pigs. Mean corpuscular hemoglobin concentrations in 250 ppm copper-fed pigs tended to be greater ($P = .08$) on day 49 versus day 0, with day 7 having an intermediate value not different from the other two days. In contrast, mean corpuscular hemoglobin concentrations in control pigs were similar across days. Finally, reticulocyte concentration was greater in pigs fed the copper-supplemented diet compared to controls (diet, $P = .03$; Table 2).

Eosinophil concentration was affected ($P = .05$) by an interaction of iron treatment and day (Figure 2B). For pigs receiving 100 mg iron at birth and 100 mg at either day 14 of age or weaning, eosinophil concentrations were greater ($P = .05$) on day 49 than on days 0 and 7; however, they were similar across days in pigs receiving 200 mg iron at birth. There was also a tendency for an effect of diet by day for eosinophil concentrations ($P = .06$; Figure 3C). Eosinophil concentrations in pigs from both dietary treatment groups were similar on days 0 and 7, and then increased to day 49 post weaning; however, concentrations on day 49 tended to be greater ($P = .06$) in copper-supplemented pigs versus controls.

Overall, white blood cell concentrations, as well as the various populations of white blood cells, were affected by day post weaning. White blood cell concentrations were greater ($P < .001$) on day 49 than on day 0 or day 7, which did not differ. Concentrations of lymphocytes increased ($P < .001$) from day 0 to day 7, and then remained similar to day 49 post weaning. Monocyte concentrations ($P < .001$) increased from day 0 to day 7 and further increased to day 49. The concentration of basophils ($P = .04$) was greater on day 49 compared to day 0 post weaning, with values on day 7 being intermediate and not different from the other two days.

The concentrations of platelets ($P = .09$) and mean platelet volume ($P = .09$) tended to be affected by iron treatment by day (Figures 2C and D). Platelet concentration tended ($P = .09$) to be greater on day 0 versus day 7 or day 49 post weaning in pigs receiving 200 mg iron at birth, decreased from day 0 to day 7, and further decreased to day 49 in pigs receiving 100 mg iron at birth and weaning. Platelet concentration in pigs receiving 100 mg iron at birth and day 14 of age tended to be less on day 49 than either day 0 or day 7. Mean platelet volume tended to increase ($P = .09$) from day 0 to 7 and further increased to day 49 in pigs receiving 100 mg iron at birth and 100 mg at either day 14 of age or weaning. In contrast, mean platelet volume increased from day 0 to day 7 and then remained similar to day 49 post weaning in pigs receiving 200 mg iron at birth. There was also an effect of diet by day post weaning for platelet concentrations ($P = .03$; Figure 3D). For pigs fed the control diets, platelet concentration decreased ($P = .03$) from day 0 to day 7 and further decreased ($P = .03$) to day 49 post weaning. In copper-supplemented pigs, however, platelet concentrations decreased ($P = .03$) from day 0 to 7 and remained similar ($P = .21$) to day 49.

Serum hepcidin concentrations

There was an effect of day post weaning ($P < .001$) on hepcidin concentrations on days 0, 7, and 49, which were 16.5, 44.0, and 177.0 ng/mL, respectively. Hepcidin concentrations tended to be affected by iron treatment ($P = .06$) with pigs receiving 100 mg at birth and day 14 having the greatest concentration (88.8 ng/mL) and

Table 2: Hematology characteristics in pigs receiving different iron treatment regimens and fed control or copper-supplemented (250 ppm) diets during the 49-day nursery phase of production

	Iron treatment*‡					Diet				Day post-weaning				
Characteristics	1 (n = 8)	2 (n = 8)	3 (n = 8)	SE	P†	Control (n = 12)	Copper (n = 12)	SE	P†	0 (n = 24)	7 (n = 24)	49 (n = 24)	SE	P†
Red blood cells, × 10^6/μL‡	6.98	7.21	7.17	0.24	.60	7.11	7.13	0.20	.96	6.89^{a}	7.29^{b}	7.18^{a,b}	0.13	.002
Hemoglobin, g/dL‡	12.62^{a,b}	11.71^{a}	12.64^{a}	0.39	.03	12.28	12.37	0.31	.76	11.88^{a}	12.33^{a,b}	12.76^{b}	0.24	.005
Hematocrit, %‡	42.15	40.37	42.75	1.13	.10	41.68	41.83	0.91	.87	40.49^{a}	41.78^{a,b}	42.99^{b}	0.86	.02
Mean corpuscular volume, fL‡§	60.48^{a}	56.2^{b}	59.80^{a}	1.15	.001	58.66	58.99	0.93	.72	58.79^{a}	57.43^{b}	60.25^{c}	0.60	< .001
Mean corpuscular hemoglobin, g/dL‡§	29.93^{a}	28.94^{b}	29.58^{a,b}	0.30	.008	29.44	29.53	0.25	.73	29.24	29.53	29.68	0.21	.10
Red blood cell distribution width, %‡	16.77^{a}	21.06^{b}	17.82^{a}	1.12	.001	18.69	18.41	0.91	.76	20.12	19.48	16.05	0.41	< .001
Reticulocytes, %‡	3.33^{a}	4.87^{b}	3.37^{a}	0.36	< .001	3.63	4.08	0.25	.13	5.65	3.25	2.67	0.34	< .001
Reticulocytes, × 10^3/μL‡	229.68^{a}	342.56^{b}	237.57^{a}	20.00	< .001	251.54^{a}	288.34^{b}	16.65	.03	385.31^{a}	234.83^{a,b}	189.67^{b}	24.23	< .001
White blood cells, × 10^3/μL	17.64	16.18	15.74	1.30	.53	16.79	16.25	1.04	.71	14.01^{a}	15.74a	19.79^{b}	1.40	< .001
Neutrophils, × 10^3/μL	6.96	6.36	5.83	1.29	.68	6.54	6.23	0.75	.77	6.54	5.57	7.04	0.71	.10
Lymphocytes, × 10^3/μL	9.42	8.60	8.63	0.90	.58	9.04	8.74	0.73	.68	6.72^{a}	8.95^{b}	10.99^{b}	0.89	< .001
Monocytes, × 10^3/μL	0.84	0.74	0.71	0.11	.44	0.79	0.74	0.09	.55	0.40^{a}	0.75^{b}	1.13^{c}	0.12	< .001
Eosinophils, × 10^3/μL‡§	0.35	0.36	0.40	0.07	.78	0.32	0.42	0.06	.06	0.22^{a}	0.25^{a}	0.65^{b}	0.05	< .001
Basophils, × 10^3/μL	0.11	0.10	0.08	0.02	.31	0.10	0.10	0.02	.94	0.07^{a}	0.10^{a,b}	0.12^{b}	0.02	.04
Platelets, × 10^3/μL‡§	296.61	365.22	315.07	36.69	.16	328.22	323.05	29.68	.86	499.00^{a}	308.73^{b}	169.17^{c}	28.28	< .001
Mean platelet volume, fL‡	10.11	10.48	10.53	0.37	.46	10.49	10.25	0.30	.42	8.06^{a}	10.44^{b}	12.60^{c}	0.36	< .001

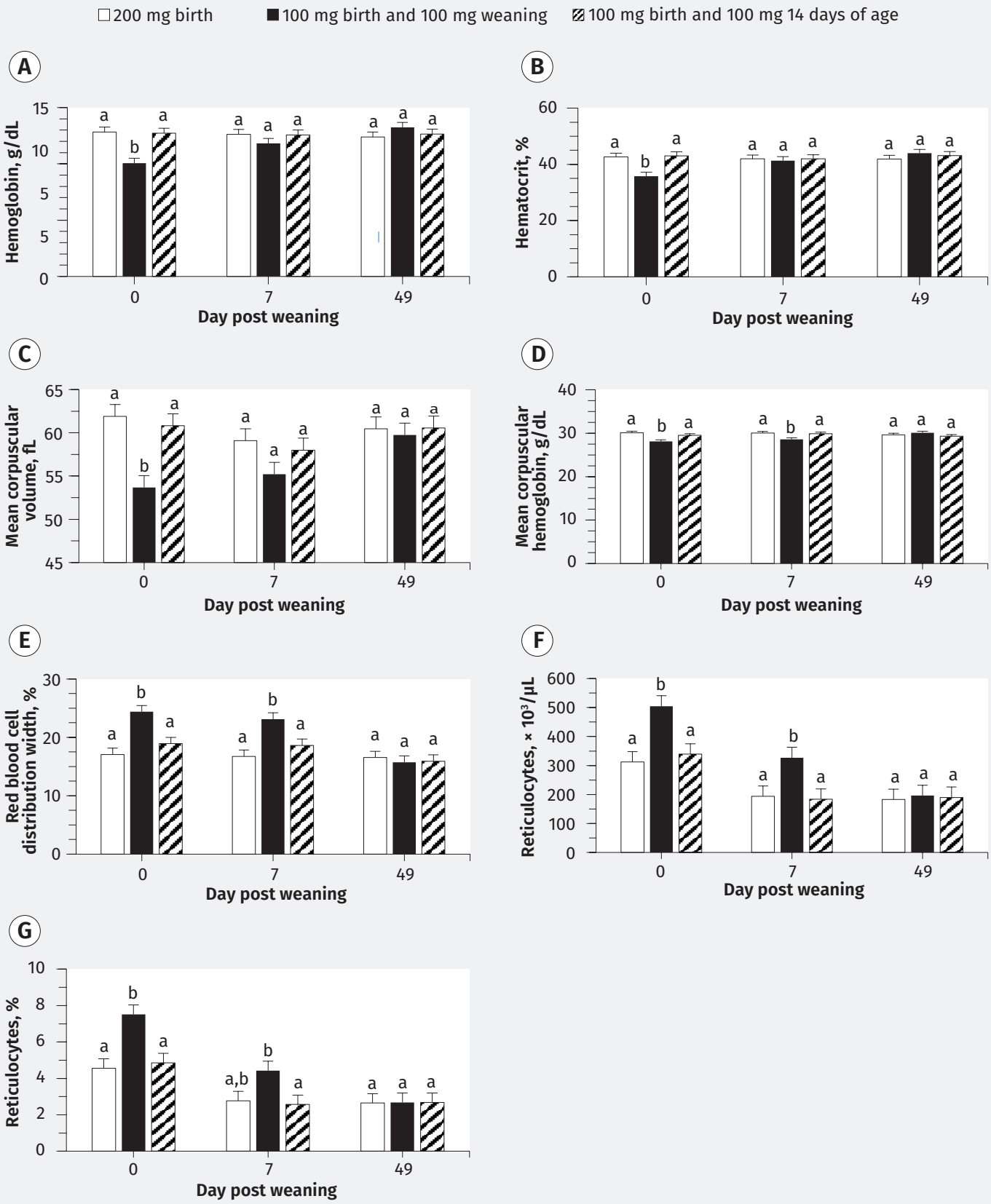
* Treatment 1 = 200 mg iron at birth; Treatment 2 = 100 mg iron at birth and 100 mg iron at weaning (22.4 days of age); and Treatment 3 = 100 mg iron at birth and 100 mg iron at day 14 of age.

† Data were subjected to ANOVA. The model included iron treatment, diet, and day and all two- and three-way interactions as possible sources of variation. For main effects of treatment, and day, values within a row with different superscripts (^{a,b,c}) differ ($P < .05$).

‡ Affected by an interaction, or tendency for an interaction, between iron treatment and day (red blood cells, $P = .06$; hemoglobin, hematocrit, mean corpuscular volume, mean corpuscular hemoglobin, and red blood cell distribution width, $P < .001$; reticulocytes, %, $P = .01$; reticulocyte number, $P = .03$; eosinophils, $P = .05$; platelets, $P = .09$; and mean platelet volume, $P = .09$).

§ Affected by an interaction, or tendency for an interaction, between diet and day post weaning (mean corpuscular volume, $P = .06$ mean corpuscular hemoglobin $P = .08$; eosinophils, $P = .06$; and platelets, $P = .03$).

Figure 1: Hematology characteristics of pigs receiving 200 mg of iron at birth, or 100 mg iron at both birth and day 14 of age, or 100 mg iron at both birth and weaning (22.4 days of age) by intramuscular injection and a control or copper-supplemented diet (14 or 250 ppm copper, respectively). A) Hemoglobin, B) hematocrit, C) mean corpuscular volume, D) mean corpuscular hemoglobin, E) red blood cell distribution width ($P < .001$), F) reticulocytes number ($P = 0.03$), and G) reticulocyte percentage ($P = 0.01$) were affected by the interaction of iron treatment and day. Data were subjected to ANOVA for repeated measures with a model that included iron treatment, diet, day, and all two- and three-way interactions as possible sources of variation. Within day post weaning, columns with different superscripts (^{a,b}) differ.



pigs receiving 200 mg at birth the least (70.0 ng/mL); pigs receiving 100 mg at birth and weaning had an intermediate concentration (79.8 ng/mL) not different from either of the other two groups. There was no effect of diet ($P = .11$) on hepcidin concentrations.

Iron treatment by diet by day ($P = .19$), and iron treatment by day ($P = .43$) did not affect concentrations of hepcidin in serum. There were tendencies, however, for hepcidin concentrations to be affected by iron treatment by diet ($P = .06$; Figure 4) and diet by day ($P = .07$; Figure 5). Hepcidin concentrations tended to be greater ($P = .06$) in control pigs receiving 100 mg iron at both birth and 14 days of age, compared to similarly treated copper-supplemented pigs (Figure 4). This dietary relationship did not exist for pigs treated with 200 mg of iron at birth ($P = .99$) or with 100 mg at both birth and weaning ($P = .99$). Hepcidin concentrations were similar on day 0 ($P = .99$) and day 7 ($P = .99$) post weaning between diets but tended to be greater ($P = .07$) on day 49 post weaning in control compared to copper-supplemented pigs (Figure 5).

BW and growth performance

There were no effects of treatment by diet on BW at weaning (day 0) or day 7, 21, or 49 post weaning (Table 3). Body weights at weaning were affected by iron treatment ($P = .01$). The mean (SE) BW of pigs that received 100 mg iron doses at birth and at day 14 of age (7.75 [0.53] kg) were greater ($P = .01$) than BW of pigs that received 100 mg iron doses at both birth and at weaning (7.29 [0.53] kg), with pigs that received 200 mg iron at birth having an intermediate value (7.47 [0.7] kg) that did not differ from the other two groups. In contrast, BW at days 7, 21, and 49 were not affected by iron treatment (Table 3). Diet affected BW at day 7 only with copper-supplemented pigs being heavier ($P = .03$) than their control counterparts.

Growth performance measures including ADG, ADFI, and G:F were not affected by iron treatment by diet for the periods from day 0 to 7, day 8 to 21, day 22 to 49, or day 0 to 49. Table 3 summarizes growth performance in nursery pigs as affected by the main effects of treatment and diet. Growth performance measures were similar among pigs receiving various iron treatment regimens for each period and the overall trial. Average daily gain was affected by diet for the period from weaning to day 7 only, with pigs consuming the copper-supplemented diet gaining faster ($P = .03$) than controls (139.4 [6.3] g/d versus 118.2 [6.3] g/d, respectively). All other growth performance measures were not affected by diet (Table 3).

Discussion

Iron is a critical component of hemoglobin, a protein molecule that allows red blood cells to carry oxygen from the lungs to bodily tissues and return carbon dioxide from tissues to the lungs. Anemia occurs when iron levels in the body are inadequate to maintain normal circulating concentrations of hemoglobin. Thus, the hemoglobin concentration in blood is a reliable indicator of iron status in swine.[21] Pigs with hemoglobin concentrations less than 9.0 g/dL are anemic and those with hemoglobin levels above 9.0 g/dL, but less than 11.0 g/dL, are iron-deficient.[4,22] In the current investigation, three different strategies for increasing blood iron concentrations in young pigs were compared in terms of hematology, hepcidin concentrations, and nursery growth performance.

Pigs receiving 100 mg iron injections at birth and weaning (after blood samples were collected) displayed a mean hemoglobin concentration (approximately 10 g/dL) consistent with iron deficiency. In contrast, pigs receiving 200 mg of iron at birth or 100 mg at both birth and day 14 of age, had sufficient iron stores available for hemoglobin synthesis. Similar to these results, Williams et al[2] reported that pigs administered 100 mg of gleptoferron 3 days after farrowing had mean hemoglobin concentrations at 21 days of age indicative of iron deficiency (approximately 9.3 g/dL). In that experiment, pigs receiving 150 or 200 mg of iron at 3 days of age or 200 mg of iron at both 3 and 11 days of age had weaning hemoglobin concentrations of 11.3, 12.0, and 12.8 g/dL, respectively. Chevalier et al[3] reported that pigs receiving 200 or 300 mg of iron at birth had normal levels of hemoglobin at weaning, but pigs that were injected with 100 mg of iron had mean hemoglobin concentrations indicative of anemia as early as 14 days of age.

Other hematological measures at approximately 22 days of age (weaning) in pigs receiving 100 mg iron at birth and at weaning in the current study, were also consistent with iron deficiency. Consonant with a previous report,[7] decreased hemoglobin concentrations were associated with decreased red blood cell concentration, hematocrit, mean corpuscular volume, and mean corpuscular hemoglobin concentrations, and increased red blood cell distribution width, a measure of variability in cellular size. The elevated levels of reticulocytes (immature red blood cells) seen in this study are consistent with increased production of these cells from bone marrow as a response to decreased iron levels.[23] Injection of an additional 100 mg of iron in these pigs restored hemoglobin, hematocrit, and mean corpuscular volume, but not mean corpuscular hemoglobin, red blood cell distribution width, and reticulocyte count to values similar to the other two treatment groups on day 7 post weaning. By day 49 post weaning, however, there were no differences in these measures among iron treatment regimens.

In general, the various hematological measures at weaning were similar for pigs receiving 200 mg of iron at birth and pigs receiving 100 mg of iron at both birth and day 14 of age. The responses observed here are consistent with that reported in a previous study during which hemoglobin concentrations at weaning were similar in pigs receiving 300 mg iron injections at birth or 200 mg iron at birth and 100 mg at 10 days of age, with animals in both treatment groups having greater hemoglobin levels than pigs receiving only 200 mg of iron at birth.[24]

Hepcidin, a protein hormone secreted by the liver, tightly controls iron availability in the body. In response to iron loading, hepatocytes release hepcidin. This hormone negatively affects the efflux of iron from duodenal enterocytes, and the release of iron from hepatocytes and macrophages. Collectively, these mechanisms prevent iron toxicity. In contrast, hepcidin expression is down regulated during iron deficiency, increasing iron availability. By controlling iron homeostasis, hepcidin strongly influences erythropoiesis.[18]

Lipiński et al[19] reported that administration of 200 mg of iron to neonatal pigs caused protracted increases in circulating concentrations of hepcidin, and elevated concentrations were still evident until at least 21 days of age. Starzyński et al[20] prevented iron deficiency anemia without affecting hepcidin concentrations by injecting pigs at 3 and 14 days of age with reduced amounts of iron dextran (37.5 mg/kg body weight). In the current experiment, hepcidin

Figure 2: Hematology characteristics of pigs receiving 200 mg of iron at birth, 100 mg iron at both birth and day 14 of age, or 100 mg iron at both birth and weaning (22.4 days of age) by intramuscular injection and fed control or copper-supplemented diets (14 or 250 ppm copper, respectively). A) Red blood cells (P = .06), B) eosinophils (P = .05), C) platelet number (P = .06), and D) platelet volume (P = .09) were affected by an interaction between iron treatment and day. Data were subjected to ANOVA for repeated measures with a model that included iron treatment, diet, day, and all two- and three-way interactions as possible sources of variation. Within iron treatment, columns with different superscripts (^{a,b,c}) differ.

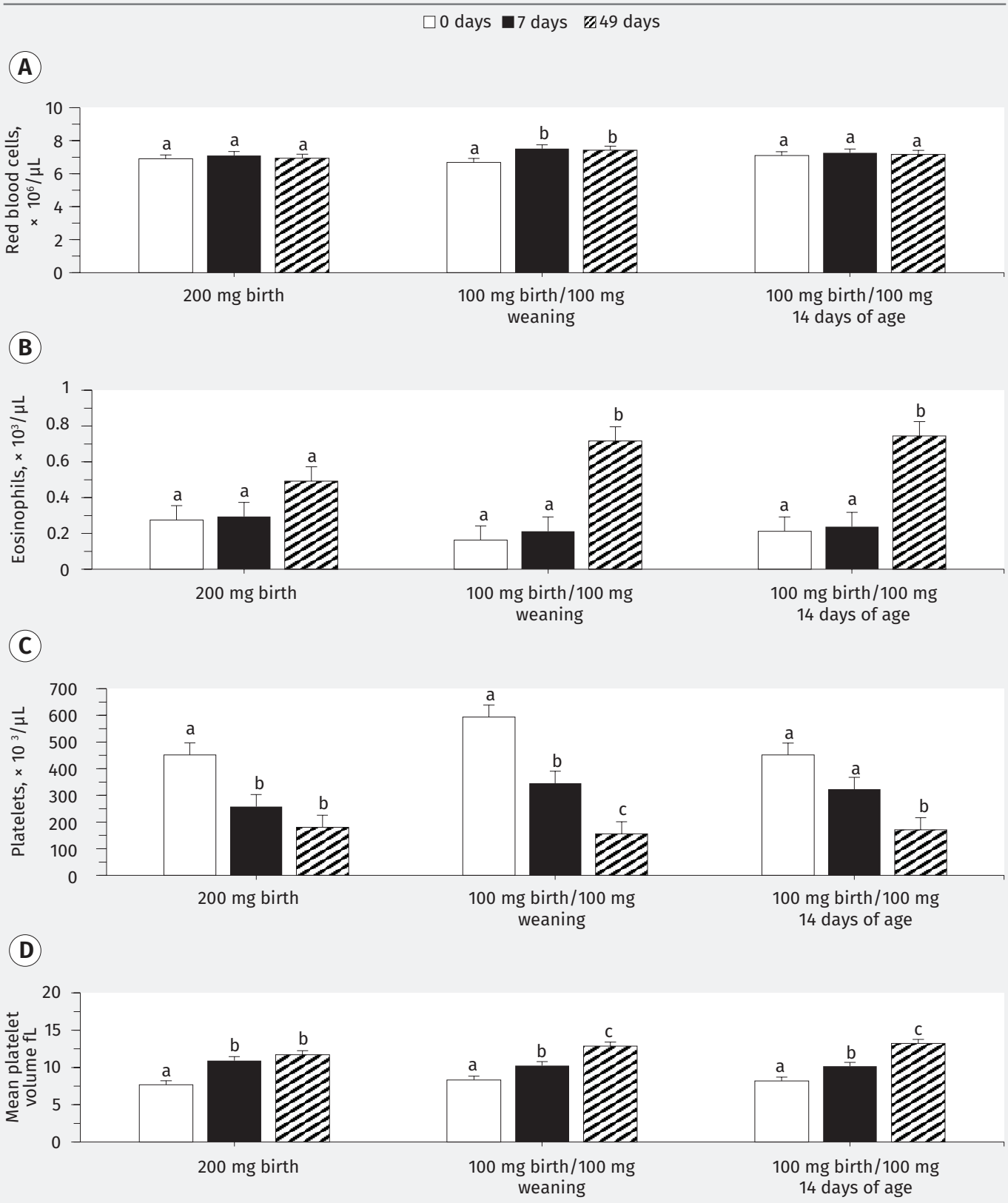
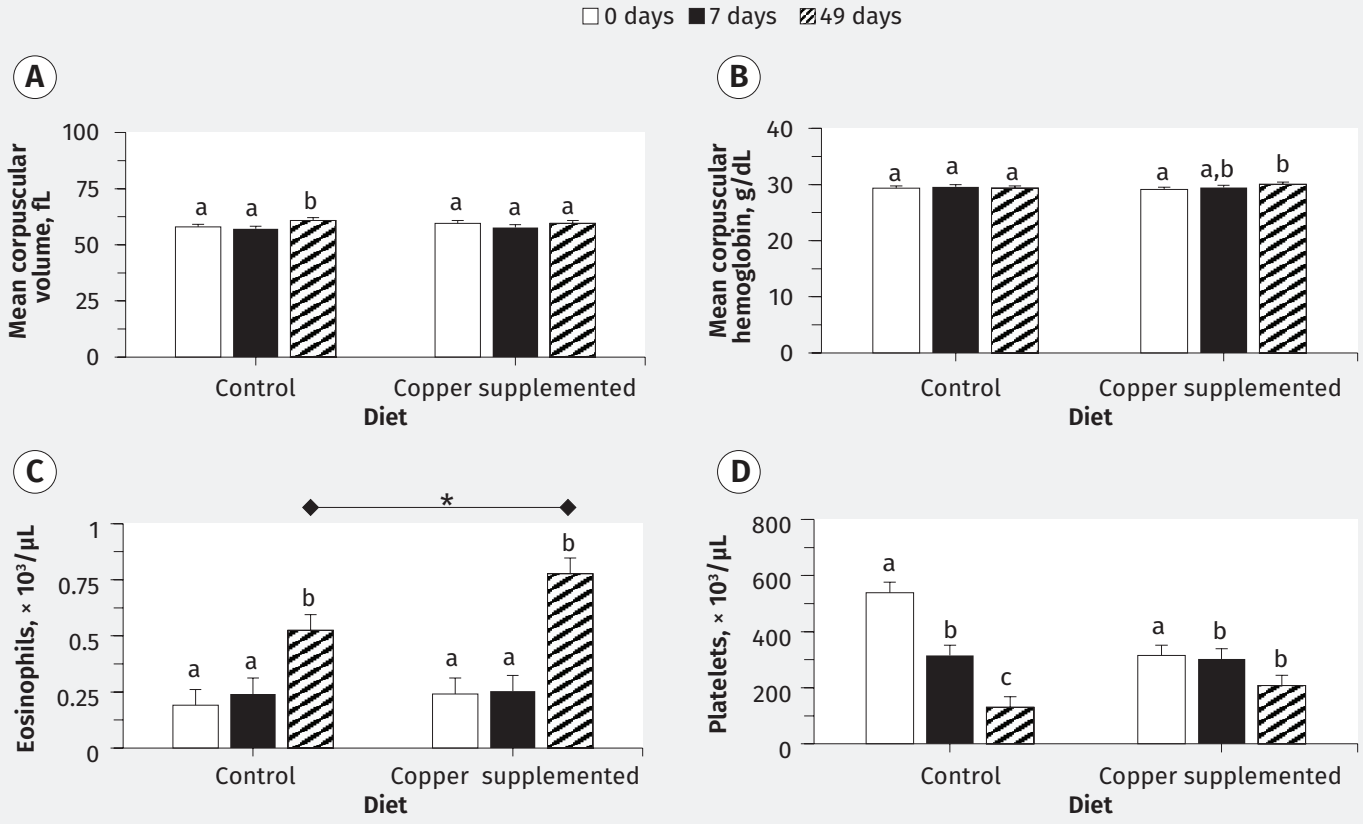


Figure 3: Hematology characteristics of pigs given 200 mg of iron at birth, 100 mg iron at both birth and day 14 of age, or 100 mg iron at both birth and weaning (22.4 days of age) by intramuscular injection and fed control or copper-supplemented diets (14 or 250 ppm copper, respectively). A) Mean corpuscular volume (P = .06), B) mean corpuscular hemoglobin (P = .08), C) eosinophil concentration (P = .06), and D) platelet number (P = .03) were affected by an interaction between diet and day. Data were subjected to ANOVA for repeated measures with a model that included iron treatment, diet, day, and all two- and three-way interactions as possible sources of variation. Within diet, columns with different superscripts (^{a,b,c}) differ. For eosinophils, concentrations tended to be greater (P = .06) on day 49 in copper-supplemented pigs versus controls (indicated with horizontal bar and *).



concentrations increased robustly with time post weaning. The values on day 49 post weaning, however, were undoubtedly influenced by consumption of dietary iron, in addition to the effects of the various iron injection regimens. Although there was no significant interaction of iron treatment and day post weaning across time points, hepcidin was greatest in the pigs that received 100 mg of iron at both birth and 14 days of age, and least in pigs receiving 200 mg of iron at birth only. In pigs receiving 100 mg iron at both birth and 14 d of age, hepcidin concentrations were greater in control versus copper-supplemented individuals. Additionally across iron treatments, hepcidin concentrations were greater in control versus copper-supplemented pigs on day 49 post weaning. To our knowledge, this is the first report of the effects of pharmacological levels of dietary copper on hepcidin concentrations in pigs. Dietary supplementation with copper has been demonstrated to decrease iron absorption.[14] Perhaps hepcidin concentrations decreased in copper-supplemented pigs as a mechanism to increase iron availability. Our finding that reticulocyte numbers were increased in pigs fed the copper-supplemented diet provides hematological support for this concept.

The transfer of weaned pigs to new surroundings in the nursery undoubtedly increased the antigenic load as evidenced by increases in indicators of both innate and acquired immunity. These temporal changes in white blood cell counts and the concentrations of neutrophils, monocytes, eosinophils, basophils, and lymphocytes are consistent with previous reports in the literature.[3,25-27] Moreover, decreases in platelet concentrations in pigs during the nursery phase of production have been previously shown.[7,27]

Numerous studies have demonstrated positive growth responses in nursery pigs provided concentrations of dietary copper in excess of nutritional requirements.[11-13] Consistent with previous reports, during the first week post weaning in this experiment, pigs fed the copper-supplemented diet exhibited greater weight gain and tendencies for greater feed intake and feed conversion efficiency compared to control pigs. In a previous study, ADG, ADFI, and G:F were enhanced by dietary copper in pigs that received 100 mg iron dextran at both birth and weaning but not in pigs receiving 100 mg iron at birth only, suggesting that an adequate iron status is requisite for copper to enhance growth performance in nursery pigs.[27] However, no measure of growth performance was influenced by the interaction of iron treatment regimen and diet in the current investigation. Thus, it appears that all 3 iron treatment regimens employed in

Figure 4: Hepcidin concentrations in pigs receiving 200 mg of iron at birth, 100 mg iron at both birth and day 14 of age, or 100 mg iron at both birth and weaning (22.4 days of age) by intramuscular injection and fed control or copper-supplemented diets (14 or 250 ppm copper, respectively). Blood was sampled on days 0, 7, and 49 post weaning. Hepcidin tended to be greater in control compared to copper-supplemented only in pigs that received 100 mg of iron at both birth and 14 days of age ($P = .06$; *). This dietary relationship did not exist in the other two groups. Data were subjected to ANOVA for repeated measures with a model that included iron treatment, diet, day, and all two- and three-way interactions as possible sources of variation.

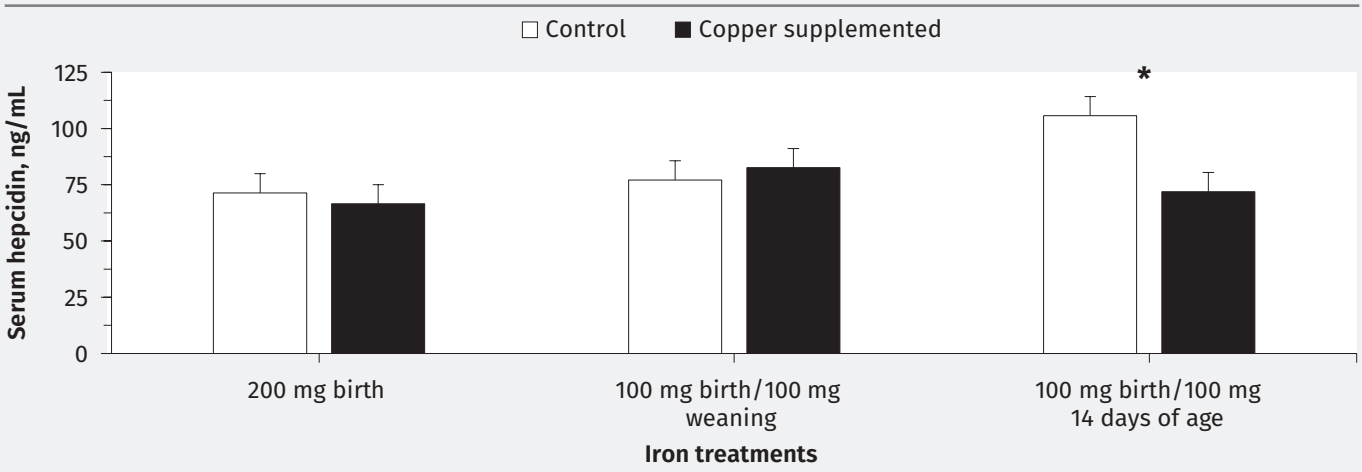
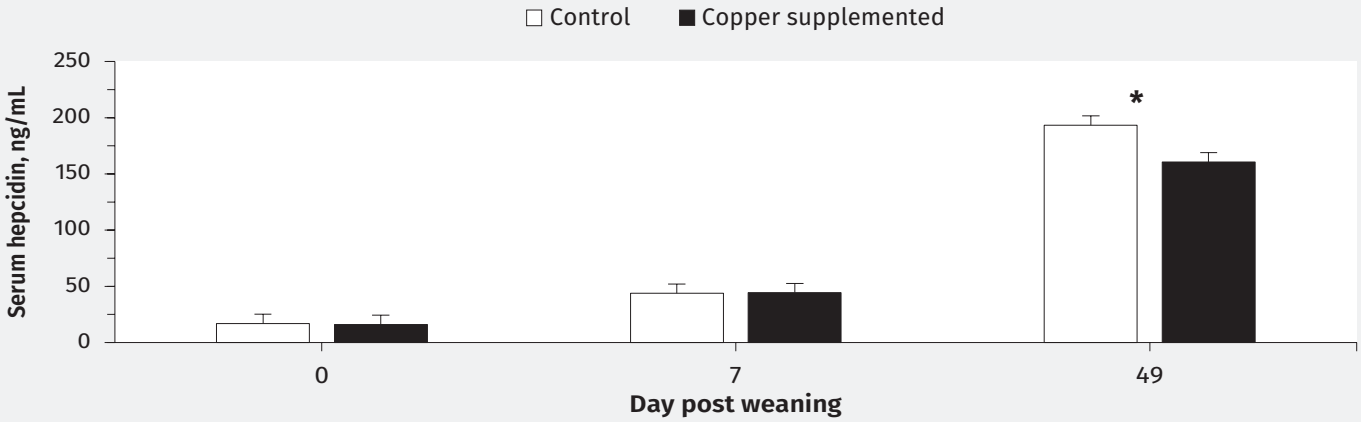


Figure 5: Hepcidin concentrations in pigs receiving 200 mg of iron at birth, 100 mg iron at both birth and day 14 of age, or 100 mg iron at both birth and weaning (22.4 days of age) by intramuscular injection and fed control or copper-supplemented diets (14 or 250 ppm copper, respectively). Blood was sampled on days 0, 7, and 49 post weaning. Data were subjected to ANOVA for repeated measures with a model that included iron treatment, diet, day, and all two- and three-way interactions as possible sources of variation. Concentrations of hepcidin tended to be affected ($P = .07$) by an interaction of diet and day post weaning with concentrations between diets tending to be different ($P = .07$; *) on day 49 only.



this study resulted in an iron status that allowed the weaned pigs to respond similarly to the supplemented copper.

Chevalier et al[3] administered increasing levels of iron at birth (0, 50, 100, 200, or 300 mg) and reported that pig weaning weights (day 22 of age) increased in both linear and quadratic fashions. Similarly, Williams et al[2] demonstrated that increasing amounts of iron (0, 50, 100, 150, and 200 mg) injected at day 3 of age resulted in linear and quadratic increases in ADG from day 3 to day 21 of age (weaning), with no increase in the response for doses greater than 100 mg. In the current study, pigs that received 100 mg of iron at both birth and day 14 of age had weaning BW that were greater than pigs receiving 100 mg of iron at both birth and weaning. Pigs that received 200 mg iron only at birth had weaning BW that were intermediate and not statistically different from the other two groups. Consistent with this finding, pigs receiving injections of 200 mg iron at both day 3 of age and 7 days prior to weaning at 28 days of age, had increased preweaning growth rates compared to pigs receiving 200 mg iron at birth only.[28] In contrast, preweaning ADG was not affected by an additional injection of 200 mg of iron 14 days prior to weaning at 34 days of age[29] or 100 mg of iron 10 days before weaning at 21 days of age.[2] Growth prior to weaning at approximately 17 days of age was similar among pigs treated with single doses of 200 or 300 mg of iron at birth or a 200 mg dose at birth followed by a 100 mg dose 10 days later.[24]

Table 3: Body weights and growth performance of pigs receiving different iron dextran treatment regimens and control (14 ppm) or copper-supplemented (250 ppm) nursery diets for 49 days

	Iron Treatment*									$P^{\dagger}$		
	1			2			3					
	Control (n = 4)	Copper (n = 4)	SE	Control (n = 4)	Copper (n = 4)	SE	Control (n = 4)	Copper (n = 4)	SE	Treatment	Diet	Treatment × Diet
Body weights, kg												
Weaning, D 0	7.49	7.46	0.54	7.19	7.39	0.54	7.67	7.83	0.54	.01	.37	.64
D 7	8.29	8.58	0.08	8.28	8.48	0.08	8.41	8.37	0.08	.75	.03	.12
D 21	12.63	12.84	0.44	12.31	13.25	0.45	13.10	13.18	0.45	.61	.26	.56
D 49	29.51	30.07	1.93	29.72	30.94	1.95	32.56	30.91	1.95	.60	.98	.74
D 0 to 7												
Gain, g/d	112.5	153.4	10.8	112.5	139.8	10.8	129.5	125.0	10.8	.80	.03	.13
Feed intake, g/d	247.7	288.6	14.5	244.3	256.8	14.5	262.5	262.5	14.5	.39	.11	.29
Gain:Feed, g/g	0.46	0.53	0.03	0.46	0.55	0.03	0.50	0.47	0.03	.75	.11	.16
D 8 to 21												
Gain, g/day	305.7	305.7	30.4	278.4	338.6	30.4	340.9	352.3	30.4	.30	.31	.54
Feed intake, g/d	504.5	504.5	48.4	534.1	560.2	48.4	559.1	558.0	48.4	.34	.79	.92
Gain:Feed, g/g	0.61	0.60	0.03	0.52	0.61	0.03	0.61	0.63	0.03	.15	.18	.19
D 22 to 49												
Gain, g/day	601.1	615.9	57.4	615.9	631.8	57.4	697.7	638.6	57.4	.57	.84	.76
Feed intake, g/d	1134.1	1172.7	114.9	1280.7	1227.2	114.9	1280.7	1354.5	114.9	.30	.73	.89
Gain:Feed, g/g	0.54	0.51	0.04	0.50	0.52	0.04	0.55	0.47	0.04	.81	.33	.31
Overall, D 0 to 49												
Gain, g/day	446.6	460.2	39.1	446.6	478.4	39.1	514.8	483.0	39.1	.49	.89	.71
Feed intake, g/d	828.4	855.7	80.0	903.2	897.7	80.0	929.5	970.5	80.0	.32	.72	.94
Gain:Feed, g/g	0.55	0.53	0.04	0.50	0.54	0.04	0.56	0.50	0.04	.71	.52	.17

* Treatment 1 = 200 mg iron at birth; Treatment 2 = 100 mg iron at birth and 100 mg iron at weaning (21 days of age); and Treatment 3 = 100 mg iron at birth and 100 mg iron at day 14 of age.

† Data were subjected to ANOVA. The model included iron treatment, diet, and the iron treatment by diet interaction as possible sources of variation.

In contrast to previous work[27] demonstrating a positive growth response to a second injection of 100 mg iron at weaning in pigs fed copper, post-weaning growth performance in the current investigation was similar among pigs receiving 200 mg of iron either as a single dose at birth or in equally divided doses given at birth and at day 14 of age or at weaning. Equivocal responses to a second iron injection before or at weaning on post-weaning growth performance have been reported. For example, pigs receiving injections of 200 mg iron at birth and 200 mg iron at 7 to 14 days prior to weaning had increased ADG compared to pigs receiving 200 mg iron at birth only.[28,29] In contrast, nursery growth performance after weaning was not dramatically affected by increasing the dosage of iron given at birth from 200 to 300 mg,[24,30] or by injecting 200 mg at birth and 100 to 200 mg at day 17 of age or at weaning.[24,31] Finally, increasing iron (0, 50, 100, or 200 mg) increased ADG and ADFI during the nursery phase of production with no effect of an additional injection of 100 mg at day 11 of age in pigs that received 200 mg at day 3 of age.[2] It is likely that any beneficial effects of additional iron treatment before or at weaning on growth performance is dependent on herd to herd factors such as iron status and diets.

Implications

Under the conditions of this study:

- Pigs receiving 100 mg of iron IM at birth were iron deficient at weaning.
- Additional 100 mg of iron given at 14 days of age increased weaning weights.
- Age-related increases in post-weaning hepcidin were dampened by copper supplementation.

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Conflict of interest

None reported.

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Effects of lameness on productivity and longevity for sows in pen gestation

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Summary

Objective: To determine the impact of lameness on sow productivity and longevity and evaluate the effects of housing management on the removal of lame sows in herds using pen gestation.

Materials and methods: Retrospective production records and information on housing methods were collected from 23 farms using pen gestation and analyzed for the removal of 214,254 sows from 2014 through 2020. Statistical analyses were performed to evaluate differences in longevity, productivity, and the impact of housing methods.

Results: Lameness was the third most reported cause of removal for sows in the study (13.7%). Sows culled for lameness spent significantly fewer days in the herd ($P < .001$), resulting in fewer litters ($P < .001$). The odds of removal for lameness were increased by several farm level factors including using dynamic groups and decreasing square footage ($P < .05$).

Implications: Lameness is one of the top 3 reasons reported for sow removal and those sows are costly as they leave the herd earlier, are less productive, and are more likely to die or be euthanized versus culled. Housing methods play a role in the odds of removal for lameness and should be further investigated.

Keywords: swine, lameness, survival analysis, welfare, group housing

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Resumen - Efectos de la cojera en la productividad y la longevidad de las cerdas en gestación en corrales

Objetivo: Determinar el impacto de las cojeras en la productividad y longevidad de las cerdas y evaluar los efectos del manejo del alojamiento en la eliminación de las cojeras en hatos con gestación en corrales.

Materiales y métodos: Se recopilaron registros de producción retrospectivos e información sobre los métodos de alojamiento de 23 granjas que utilizan la gestación en corrales y se analizaron para la eliminación de 214,254 cerdas desde 2014 hasta 2020. Se realizaron análisis estadísticos para evaluar las diferencias en la longevidad, la productividad y el impacto del sistema de alojamiento.

Resultados: En el estudio, la cojera fue la tercera causa más reportada de desecho de las cerdas (13.7%). Las cerdas descartadas por cojera pasaron significativamente menos días en la piara ($P < .001$), lo que resultó en menos camadas ($P < .001$). Las probabilidades de eliminación por cojera aumentaron por varios factores a nivel de granja, incluido el uso de grupos dinámicos y la disminución de los pies cuadrados ($P < .05$).

Implicaciones: La cojera es una de las 3 razones principales reportadas de desecho de las cerdas, estas cerdas son costosas ya que se retiran de la piara antes de tiempo, son menos productivas y es más probable que mueran o sean sacrificadas en lugar de ser desechadas. Los métodos de alojamiento juegan un papel en las probabilidades de ser desechadas por cojera y deben investigarse más a fondo.

Résumé - Effets de la boiterie sur la productivité et la longévité des truies en gestation en enclos

Objectif: Déterminer l'impact de la boiterie sur la productivité et la longévité des truies et évaluer les effets de la gestion du logement sur le retrait des truies avec boiterie dans les troupeaux utilisant la gestation en enclos.

Matériels et méthodes: Rétrospectivement, les dossiers de production et des informations sur les méthodes de logement ont été recueillis auprès de 23 fermes utilisant la gestation en enclos et analysés pour le retrait de 214,254 truies de 2014 à 2020. Des analyses statistiques ont été effectuées pour évaluer les différences de longévité, de productivité et l'impact des méthodes de logement.

Résultats: La boiterie était la troisième cause de retrait la plus signalée chez les truies dans l'étude (13.7%). Les truies réformées pour boiterie ont passé beaucoup moins de jours dans le troupeau ($P < .001$), ce qui a entraîné moins de portées ($P < .001$). Les probabilités d'élimination pour boiterie ont été augmentées par plusieurs facteurs au niveau de la ferme, notamment l'utilisation de groupes dynamiques et la diminution de la superficie en pieds carrés ($P < .05$).

Implications: La boiterie est l'une des trois principales raisons signalées pour le retrait des truies et ces truies sont coûteuses car elles quittent le troupeau plus tôt, sont moins productives et sont plus susceptibles de mourir ou d'être euthanasiées que réformées. Les méthodes de logement jouent un rôle dans les probabilités de retrait pour boiterie et devraient faire l'objet d'étude supplémentaires.

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Lameness is a serious welfare and economic issue on sow farms and gains importance as the use of pen gestation increases. Cross-sectional studies have detected the prevalence of lameness in sows in pen gestation between 4.5% and 16.9% and the National Animal Health Monitoring System reported that 15.2% of all sows culled in the United States, are culled for lameness.[1-3] Given the prevalence of lameness, it is not surprising that lameness can be costly for swine farmers and has been shown to result in increased labor needed to manage lame sows[4] and higher veterinary costs for treatment.[4,5] Lameness has been associated with decreased reproductive performance due to premature removal of sows from the herd[5,6] and reduced salvage value attributed to on-farm euthanasia.[4,7]

Engblom et al[8,9] conducted large scale studies of sow removal reasons for 21 herds in Sweden, all using pen gestation,[8] and examined survival time[9] finding that lameness accounted for 8.6% of removals[8] and that lameness as a removal reason was more common in younger animals.[9] Anil et al[6,10] collected records from 11 farms in Canada using gestation stalls and found that the risk of removal for lameness varied by time in the production cycle and the productivity of the sow.[10] They later examined survival time for 674 animals and found that lame sows had fewer piglets due to less time in the herd.[6] Studies that examine the number of sows and gilts removed for lameness, as well as the impact on their lifetime productivity, in US herds using pen gestation is lacking.

When sows are housed in groups during gestation, there are a multitude of options for managing feeding and mixing, thus bringing a unique challenge to understanding the impact of these housing management strategies on lameness. Previous studies have identified that feeding method, group size, space allowance, group structure (dynamic or static), and time of group formation (immediately post breeding or 28 to 35 days post breeding) influences animal-animal aggression which may lead to stress, injuries, and lameness for animals housed in pen gestation.[11,12] In a study of 8 Belgian pig herds, Pluym et al[4] found that there was no difference in the percentage of lame sows in farms using electronic sow feeders (ESF) compared to farms using free access stalls. Having a larger area and a higher stocking density, both increased the risk of lameness for sows on English sow farms.[1] There is limited research that evaluates the associations between housing management strategies and removal of sows for lameness in pen gestation in US herds. Understanding the link between group housing methods and lameness could help producers understand how much they can invest in alterations to housing management as well as make decisions on the best housing and management strategies for pen gestation.

The first objective of the present study was to examine retrospective data to determine the effects of lameness on sow productivity and longevity in herds using pen gestation. We predict that in comparison to other removal reasons, lameness will be associated with sows spending less time in the herd, ultimately producing fewer litters. Additionally, we hypothesize that lame sows are more likely to be involuntarily removed (death or euthanasia) from the herd compared to non-lame sows. Secondly, we examined the effects of feeding method, group size, space allowance, group structure, and time of group formation on the odds of removal for lameness. We hypothesize that lame sows fed with an ESF, housed in dynamic pens, contained in smaller groups, with less space allowance, and mixed immediately post breeding are more likely to be removed for lameness compared to other housing practices.

Animal care and use

Production records were used for this study, so no Institutional Animal Care and Use Committee approval was needed. This retrospective study was carried out using production records from commercial farms certified in the Pork Quality Assurance Plus program. The program guidelines directed animal care on the farms and the study was conducted without changing animal care routines.

Materials and methods

Experimental design

Retrospective data from 23 farms using pen gestation were examined for the removal of 214,254 sows from June 2014 through July 2020. Farms were enrolled in the study if they removed at least parity 2 animals and recorded reasons for at least 80% of removals. Farms shared the feeding method, timing of group formation, group structure, square meters per sow, group size, and farm size for each farm included in the study. Management techniques differed for gilts compared to sows on 4 farms so gilt removals on those farms were categorized by the management factors in place for gilts. Housing management techniques are given in Table 1. There were 3 different feeding systems represented in the data: ESF (n = 16), small pens using drop feeding (n = 6), and free access feeding stalls (n = 1). Farmers were also asked to submit production records that included sow identification number, entry date, removal date, removal parity, removal type, and removal reason. Total lifetime parameters for the number of litters, piglets born, piglets born alive, and piglets weaned were included in the production records.

Removal types included transfer, cull, euthanasia, and death with 96% of all removals accompanied by a reason for their removal. All sows identified as transfers were dropped from the study (n = 14). Removal reasons were broadly grouped into the following categories: age, body condition and structure, disease, lameness, injury other than lameness, reproduction, sudden death, prolapse, and other. The category of age combined any reasons that an animal was removed from the herd due to age (eg, old age and high parity). The category of body condition and structure included reasons of body size, off feed, poor condition, poor structure, and unthrifty. The category of disease included specific infectious diseases (eg, erysipelas, influenza, *Glaesserella parasuis*, porcine reproductive and respiratory syndrome, and *Streptococcus suis*), infections (eg, discharge, mastitis, cutaneous infection, urinary infection, vaginal discharge, and abscess in the body cavity), health conditions (eg, heart attack/failure, constipation, ileitis, twisted gut, stomach ulcer, scours, respiratory disease, post-farrowing illness, hemorrhage, heat stress/trauma, and cancer), and depopulation. The category of lameness combined any reasons representing locomotor problems (eg, lame, nonambulatory, unsound, joint problem/infection, bad legs, downer, hooves, chronically lame, swollen extremities, and septic from severe infection of the leg) since reasons for the removal of the sow were recorded by herd personnel and were not necessarily based on diagnosis as determined by a veterinarian or necropsy.[10] The category of injury other than lameness included reasons of abscess, accident, rupture, hernia, injury, ulcer, udder trauma, nonhealing shoulder sore, and broken back. The

Table 1: Housing description from 23 farms* using pen gestation that shared retrospective data for the removal of 214,254 sows from June 2014 through July 2020

Feeding method	Time of group formation	Group structure		Range of space allowance, m^2/sow, (median [IQR])	Range of group size, No. of sows, (median [IQR])	Range of farm size, No. sows, (median [IQR])
		Dynamic, No. of farms (No. of removals)	Static, No. of farms (No. of removals)			
Electronic sow feeding (n = 16)†	Immediately post breeding	3 (17,789)	6 (68,046)	1.45-2.04 (1.86 [0.13])	66-290 (130 [98])	2400-6300 (255 [2600])
	28-35 d post breeding	4 (13,667)	12 (57,389)			
Drop feeding (n = 6)	28-35 d post breeding	0	6 (50,261)	1.58-1.86 (1.83 [0])	10-20 (10 [2])	250-5600 (5150 [1775])

* Farms were enrolled in the study if removing at least parity 2 animals and recording reasons for at least 80% of removals. One participating farm used free access stalls and is not included in this table to preserve anonymity.

† On some farms, gilts were housed in a different group structure than the sows, so farms may be included in both group structure types.

category of reproduction combined any reasons involving poor reproductive performance or productivity (eg, farrowing difficulty/dystocia, open, unable to conceive, no estrus, poor milker, poor mothering, abortion, low born alive/total born, low weaned, low weaning average, low born alive average, poor litter sizes, small/weak pigs, abnormal pigs, and dead/mummified litter). The category of sudden death included animals that were found dead. The category of prolapse combined rectal prolapse, uterine prolapse, and vaginal prolapse. Reasons that did not appropriately fit under a specific category and accounted for less than 1% of total removal reasons were categorized as other. The other category included reasons of poor underline, behavior, inventory adjustment, market conditions/taxes, testing, and genetics.

Piglet mortality rate was determined by the differences between the piglets born alive per litter and the piglets weaned per litter and represents the number of piglets that died per litter. The time in the herd for each sow was determined as the interval between the entry date and the removal date. Nonproductive days were calculated as the total number of days in the herd minus the total number of gestation and lactation days. The proportion of nonproductive days was calculated as the total nonproductive days divided by the number of days a sow remained in the herd. Removal type was condensed to a binary variable of voluntary (sows were culled) and involuntary (sows were euthanized or died).

Statistical analyses

Statistical analyses were performed using Stata IC v.16. Sows were categorized as lame or non-lame based on whether they were removed for lameness or another reason. The sow was treated as the statistical unit for all analysis and $P < .05$ was treated as significant. Production measures including total litters, piglets born alive per litter, piglets weaned per litter, piglet mortality rate, and proportion of nonproductive days were analyzed with mixed effect linear regression models. Each model included removal for lameness and year of removal as factor variables as fixed effects. Farm served as the random effect. Survival time for sows reported as removed for lameness compared to sows removed for other reasons was analyzed using a mixed effect survival analysis model with an exponential distribution. Lameness and farm were included as factor variables as fixed effects and year was included as a random effect. Removal type was examined using a mixed effect logistic regression model. Lameness and year were included as factor variables as fixed effects and farm was included as a random effect. Housing management factors were analyzed for their impact on the odds of a sow being removed for lameness using a mixed effect logistic regression model with feeding system, timing of mixing, pen structure, and year included as factor variables and standard deviation of group size and square meters per sow included as continuous variables. Farm was included as a random effect.

Results

Productivity and longevity

In this study of 214,254 sow removals, 29,334 (13.7%) were reported as removed for lameness, the third most reported removal reason behind reproduction (114,961 sows, 53.7%) and age (30,809 sows, 14.4%). Lame animals were significantly less productive compared to non-lame sows (Table 2). Lame sows spent significantly fewer days in the herd (mean [SE] = 323.1 [18.7]) compared to sows removed for other reasons (489.2 [28.2]; $P < .0001$; Figure 1). The odds of being removed involuntarily were significantly higher for lame sows compared to non-lame sows (odds ratio = 10.6; 95% CI, 10.3-10.9; $P < .001$).

Housing management

Feeding method did not have a significant effect on the odds of removal for lameness ($P = .51$). Odds ratios associated with housing management are provided in Table 3 where dynamic groups increased odds of removal for lameness and increasing group size and increasing square footage decreased odds of removal for lameness.

Discussion

In this production data from 23 US sow herds, we found that lameness was the third most common reason reported for removal from the herd. This is higher than reported by Engblom et al[8] who found that lameness was the fifth most common reason for removal from Swedish herds preceded by reproductive

Table 2: Least squares means (SE) of productivity of sows from 23 farms using pen gestation removed for lameness (n = 29,344) compared to sows removed for other reasons (n = 114,961)

	Lame	Non-lame*	*P***
Total litters†	1.8 (.22)	3.0 (.27)	< .001
Piglets born alive/litter‡	12.9 (.15)	12.9 (.13)	.29
Piglets weaned/litter	10.3 (.29)	10.8 (.25)	< .001
Piglet mortality rate§	2.6 (.19)	2.2 (.19)	< .001
Proportion nonproductive days¶	0.49 (.04)	0.40 (.04)	< .001

* Removed for reasons other than lameness such as age, body condition and structure, disease, injury other than lameness, reproduction, sudden death, prolapse, and other.

† Total litters for a sow for their lifetime.

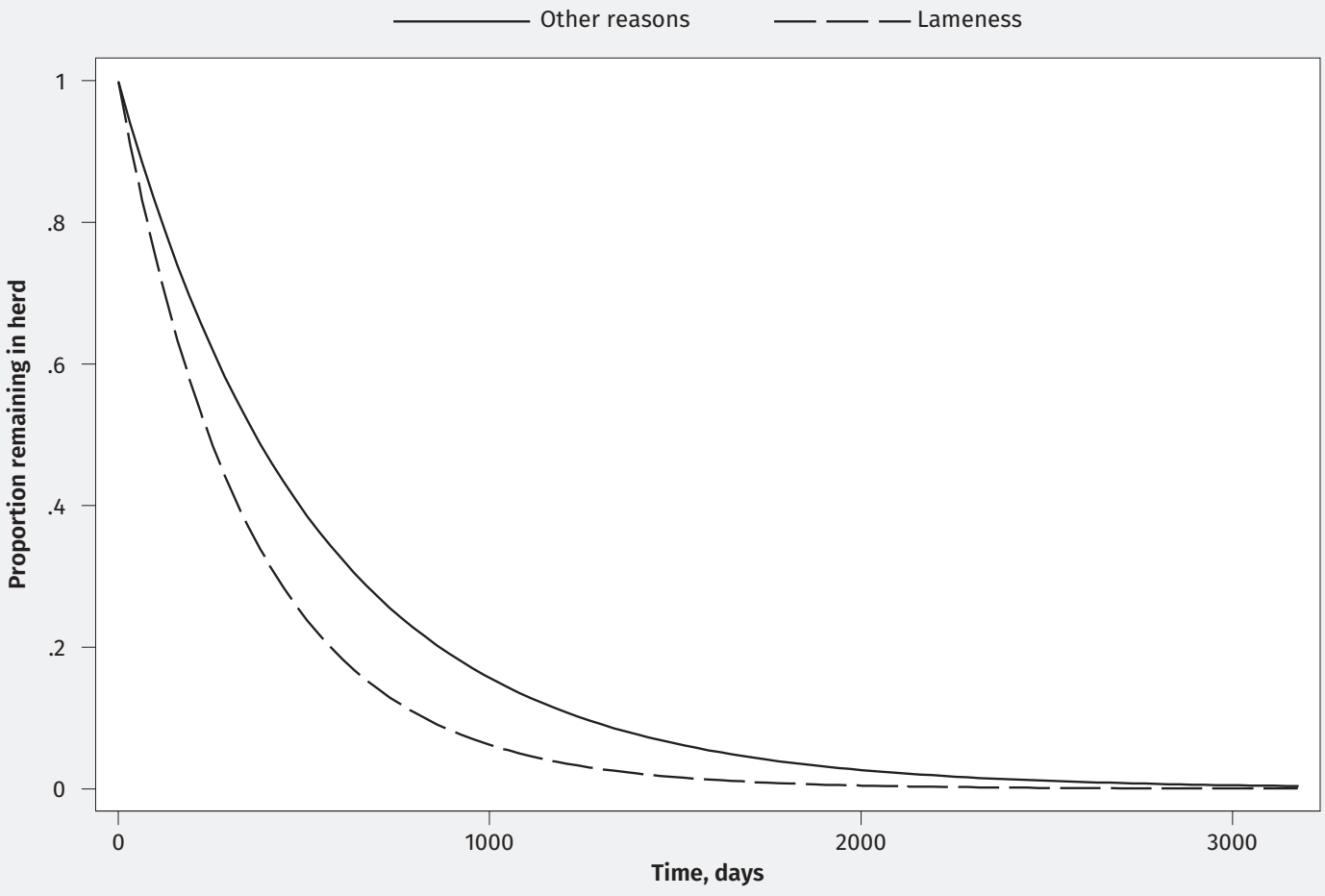
‡ Piglets that were not mummified or stillborn when born.

§ Number of piglets that died/litter calculated by number born alive minus number weaned.

¶ Days the sow is neither pregnant nor nursing a litter as a proportion of her days in the herd.

** Significance was determined by mixed effect linear regression models for each production outcome. Each model included removal for lameness and year of removal as factor variables as fixed effects. Farm served as the random effect.

Figure 1: The predicted time to removal from the herd for sows removed for lameness (n = 29,344) and sows removed for other reasons (n = 114,961) for 23 farms using pen gestation that shared entry dates and removal dates. Day zero is the day that the sow entered the herd. Sows removed for lameness spent significantly fewer days (323 d) in the herd compared to sows removed for other reasons (489 d; *P* < .001).



reasons, age, udder issues, and low productivity, which could be attributable to different farm practices or genetics. Our results showed a smaller number of removals for lameness than several other US-based studies. A case study on a large US farm conducted by Sanz et al[13] found that lameness was responsible for the majority of animal removals with 23.4% of the animals dying or being removed for locomotor issues during their study period. Irwin et al[14] found that the largest contributor to sow mortality during their study was locomotor issues, which was responsible for 44% of the mortalities across 6 different farms. Our results may undercount lameness, as a sow may have been lame but removed for another primary reason such as reproduction. In the context of these case studies, our results confirm that in US herds specifically, locomotor issues are a significant percentage of sow removals.

Sows removed for lameness in our study spent significantly less time in the herd compared to sows removed for other reasons. This is similar to other studies that have looked at reasons for sow removal. In Engblom et al[9] they found that lameness posed the greatest risk in determining when gilts left the herd compared to other parities. Likewise, Anil et al[10] found that the percent removed for lameness was highest in parity 0 and parity 1 sows compared to sows that were parity 2 or greater. This is of concern because when lame sows are removed before they attain their expected life in the herd, the economic performance of the herd can be adversely affected.[10] When a sow remains in the breeding herd for fewer parities, the animal is likely to produce fewer piglets in her lifetime compared to a sow that remained in the breeding herd for a longer period. Lame sows in this study did have fewer litters producing 1.8 litters in their lifetime while non-lame sows produced 3 litters. This reduces the opportunity for a sow to be sufficiently productive and for a farmer to achieve a profit from the investment in that animal since sows reach peak production between the third and sixth parity and do not produce a profit for the farmer until their third parity.[15]

Not only did sows removed for lameness generate fewer total litters in their lifetime, but the proportion of nonproductive days were higher for such sows compared to sows removed for other reasons. There was no difference in the piglets born alive per litter for sows removed for lameness however, there were fewer piglets weaned per litter and a higher piglet mortality rate compared to sows removed for other reasons. These results are similar to the higher preweaning piglet losses reported by Anil et al.[6] This decrease in number of litters may be related to pain caused by lameness creating stress, which has a negative influence on sow reproductive performance through inhibition of ovulation or by hindering the expression of estrus behavior.[16] The pain of lameness may also affect the piglet mortality rate as it may influence the ability of a sow to make postural changes within a farrowing crate or lead to uncontrolled lying-down behavior and consequently may cause death of baby pigs as a result of crushing.[5,6]

Another impact increasing the cost of lameness is the cost associated with losing the salvage value of the sow when she is removed involuntarily. The data collected in this study indicate that the odds of being removed involuntarily were significantly higher for lame sows compared to non-lame sows. Kirk et al[7] similarly found that the largest cause of euthanasia in Danish herds was related to musculoskeletal and locomotor issues. In the case study by Sanz et al,[13] 38.5% of the animals removed from breeding were removed for locomotor reasons and 59.1% of those were euthanized. In gestation, 64% were removed for locomotor reasons and 56.8% of those were euthanized[13] supporting the conclusion of our data that lameness is even more costly for farmers as it results in animals being disposed of on the farm.

Certain housing and feeding methods may predispose herds to increased locomotor issues. In our data, the feeding method did not impact the odds of removal for lameness when comparing sows fed with ESF to those using drop feeding and free access feeding stalls. Though studies of sow removals are rare, studies of lameness prevalence are more common, and we would expect an association between the two. A comparative study that assessed individual feeding methods concluded that group-housed sows fed using ESF and trickle systems had higher incidences of locomotion disorders and hoof lesions compared with sows fed in free access stalls.[17] Though Zurbrigg and Blackwell[18] did not analyze the feeding system in their study of 4 farms, the farm using the ESF feeding method did have the highest percentage of lameness, which is in contrast to our study. Like our study, a study of 8 Belgian herds found the prevalence of lameness was not different between the sows fed with ESF compared to the sows housed in free access stalls.[4] Different feeding systems on the same farm, though challenging, would be a way to isolate the impact of the feeding system in future work while controlling for other factors.

The results of this study indicated that increasing group size in farms decreased the odds of removal for lameness. The literature shows mixed results on the impact of group size on aggression in sows. In some studies, aggression does

Table 3: Odds of removal for lameness associated with housing management factors for 23 farms that shared housing management information*

	Odds ratio	SE	*P*	95% CI
Dynamic vs static	1.37	0.08	< .001	1.22-1.54
Immediately post breeding vs 28-35 d post breeding	1.59	0.66	.27	0.71-3.60
m^2/sow	0.26	0.02	.02	0.08-0.80
SD of group size	0.74	0.03	< .001	0.68-0.81

* Results are presented as the odds ratio generated from a mixed effect logistic regression model which included the standard deviation of group size, feeding type, time of group formation (immediately post breeding or 28-35 days post breeding), and group structure (static or dynamic) as factor variables and square meters as a continuous variable.

not increase with increasing group size as shown in a study by Turner et al[19] where inter-sow aggression decreased in larger groups. However, other studies show an increase in lesions as group size increases[20] indicating more aggression. Like many aspects of housing, there are other factors, such as mixing strategy, which may influence aggression. It is also worth noting that many of these studies have not looked at the impact of group size specifically on lameness.

There was a decrease in the odds of being removed for lameness with increasing square footage. This idea is in contrast to the findings of Salak-Johnson et al[21] where an increase in square meters increased lameness in small static groups. In other studies, decreasing space allowance led to more agonistic interactions when ESF were being used[22] and lesion scores increased when space allowance was decreased when using feeding stalls.[23] Such lesions and aggressive interactions could correlate with an increased odds of lameness. Space allowance on the study farms included here only varied from 1.45 to 2.04 m^2/sow which is not a wide range and less than the square meters mandated in the European Union.[24]

Group structure also had an impact on the odds of removal on farms using ESF where sows in dynamic pens had 1.4 times greater odds of removal for lameness compared to sows in static groups. Group structure influences animal behavior and thus may influence the occurrence of lameness. A review by Bench et al[25] described static groups as more consistent compared to dynamic groups as mixing only occurs once and then stable subgroups can form. Bos et al[26] compared prevalence, incidence, and mean scores of lameness in static versus dynamic group housed sows at different stages of gestation and found that static groups demonstrated lower lameness scores at the end of gestation when compared to dynamic groups. Anil et al[27] similarly detected that pregnant sows housed in dynamic systems with ESF had a significantly higher total injury score which could lead to an increase in lameness. These increases in aggression and lameness in dynamic groups are consistent with our findings of increased odds of removal for lameness in farms with such groups.

The timing of group formation is yet another aspect of sow housing that could be expected to influence the amount of lameness in a herd. Unlike our study, where time of mixing did not have an effect, Strawford et al[28] found fewer aggressive encounters occurred at the feeder when sows were mixed later in gestation. Like our study however, Knox et al[29] found there was no difference in the amount of leg inflammation in sows that were mixed between 7- and 35-days post weaning. The research is therefore equivocal on whether timing of mixing has an impact on the odds of lameness in pen gestated sows.

Our data is a sample of US herds using pen gestation that shared records for a large number of sow removals. As sows may not be correctly categorized by farm staff as to the reason for their removal[10] and sows may be removed for multiple reasons that were not captured in our data, we may be undercounting lameness by looking only at removal reasons. Associations between housing types and lameness are challenging as the relationships are not necessarily casual and should be considered carefully within each production system. More research is needed to investigate the direct relationship between housing management strategies and the risk of removing lame sows in US herds using pen gestation. The impact of group size on lameness is difficult to assess since it is related to the feeding methodology and thus isolating group size as its own factor to understand the association with the risk of removal for lameness is important. Additional studies are needed to determine whether time of mixing has an impact on the risk of removal for lame sows since there have been conflicting results from previous research. Based on the information provided by the farms in this study, housing systems and mixing methods that promote the formation of stable groups may have an impact on decreasing odds of removal for lameness. These data indicate the importance of lameness as a reason for removal and highlights the cost of lameness due to its impact on productivity and the removal of younger animals from the herd. Ultimately, understanding the link between housing methods and lameness removals and the high costs associated with lameness could help producers make decisions on best housing strategies for pen gestation and how much they can invest in alterations to housing management.

Implications

Under the conditions of this study:

- Lameness was the third most commonly reported cause of sow removal.
- Lame sows were removed earlier, less productive, and more likely removed by death or euthanasia.
- More work is needed to assess impacts of housing methods on risk of lameness.

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Conflict of interest

None reported.

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ORIGINAL RESEARCH PEER REVIEWED

Viral load, lung lesions, and average daily gain in a porcine reproductive and respiratory syndrome virus-2 challenge model

Kimberly Ann Vonnahme, PhD; Manuel Alexander Vasquez-Hidalgo, DVM, PhD; Jose Angulo, DVM; Deborah Amodie, MS; Martha A Mellencamp, PhD; Lucina Galina Pantoja, DVM, PhD

Summary

Objective: To determine viremia, percentage lung lesions, average daily gain (ADG), and their associations after a porcine reproductive and respiratory syndrome virus-2 (PRRSV-2) lineage 1 (open reading frame 5 restriction fragment length polymorphism 1-7-4 [ORF5 RFLP 1-7-4]) challenge in pigs vaccinated with either a PRRSV-2 lineage 8 modified live virus (MLV) vaccine, a PRRSV-2 lineage 1 MLV vaccine, or not vaccinated.

Materials and methods: Pigs were vaccinated with either Fostera PRRS (n = 52), Prevacent PRRS (n = 50), or sterile water (nonvaccinated; n = 47). Four weeks after vaccination, all animals were challenged with PRRSV-2 lineage 1 ORF5 RFLP 1-7-4. Viremia was determined at 3-, 6-, and 12-days post challenge. Body weights were recorded to determine ADG throughout the experiment. Percentage lung lesions were assessed on day 40 (12 days post challenge).

Results: Vaccination with either vaccine reduced ($P < .001$) lung lesions, increased ($P < .001$) ADG post challenge, and better controlled viremia ($P < .001$) compared to nonvaccinated pigs.

Implication: A commercially available PRRSV-2 lineage 8 vaccine was as effective as a PRRSV-2 lineage 1 vaccine against a heterologous PRRSV-2 lineage 1 viral challenge.

Keywords: swine, average daily gain, lung lesions, porcine reproductive and respiratory syndrome virus-2, viremia

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Resumen: Carga viral, lesiones pulmonares y ganancia diaria promedio en un modelo de desafío del virus del síndrome reproductivo y respiratorio porcino-2

Objetivo: Determinar la viremia, el porcentaje de lesiones pulmonares, la ganancia diaria promedio (GDP) y sus asociaciones después del reto con un virus del síndrome reproductivo y respiratorio porcino-2 (PRRSV-2) linaje 1 (marco abierto de lectura 5 polimorfismo de longitud de fragmentos de restricción 1-7- 4 [ORF5 RFLP 1-7-4]) en cerdos vacunados con una vacuna de virus vivo modificado (MLV) de linaje 8 de PRRSV-2, una vacuna de MLV de linaje 1 de PRRSV-2, o no vacunados.

Materiales y métodos: Los cerdos fueron vacunados con Fostera PRRS (n = 52), Prevacent PRRS (n = 50) o agua esterilizada (no vacunados; n = 47). Cuatro semanas después de la vacunación, todos los animales se expusieron al PRRSV-2 ORF5 RFLP 1-7-4 linaje 1. La viremia se determinó a los 3-, 6-, y 12-días después de la exposición. Se registraron los pesos corporales para determinar la GDP durante todo el experimento. El porcentaje de lesiones pulmonares se evaluó el día 40 (12 días después de la exposición).

Resultados: La vacunación con cualquiera de las vacunas redujo ($P < .001$) las lesiones pulmonares, aumentó ($P < .001$) ADG después del desafío y controló mejor la viremia ($P < .001$) en comparación con los cerdos no vacunados.

Implicación: Una vacuna de PRRSV-2 de linaje 8 comercialmente disponible fue tan eficaz como una vacuna de PRRSV-2 de linaje 1 contra un desafío viral heterólogo de PRRSV-2 de linaje 1.

Résumé - Charge virale, lésions pulmonaires et gain quotidien moyen dans un modèle d'infection-défi par le virus-2 du syndrome reproducteur et respiratoire porcin

Objectif: Déterminer la virémie, le pourcentage de lésions pulmonaires, le gain quotidien moyen (GMQ) et leurs associations après une infection-défi avec le virus du syndrome reproducteur et respiratoire porcin-2 (PRRSV-2) lignée 1 (cadre de lecture ouvert 5 polymorphisme de longueur des fragments de restriction 1-7-4 [ORF5 RFLP 1-7-4]) chez des porcs vaccinés avec soit un vaccin à virus vivant modifié (MLV) PRRSV-2 lignée 8, un vaccin PRRSV-2 lignée 1 MLV, ou non vaccinés.

Matériels et méthodes: Les porcs ont été vaccinés soit avec Fostera PRRS (n = 52), Prevacent PRRS (n = 50), soit

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Vonnahme KA, Vasquez-Hidalgo MA, Angulo J, Amodie D, Mellencamp MA, Galina Pantoja L. Viral load, lung lesions, and average daily gain in a porcine reproductive and respiratory syndrome virus-2 challenge model. *J Swine Health Prod*. 2022;30(4):230-237. **https://doi.org/10.54846/jshap/1257**

avec de l'eau stérile (non vaccinée; n = 47). Quatre semaines après la vaccination, tous les animaux ont été inoculés avec du PRRSV-2 lignée 1 ORF5 RFLP 1-7-4. La virémie a été déterminée 3-, 6-, et 12-jours après l'inoculation. Les poids corporels ont été enregistrés pour déterminer l'ADG tout au long de l'expérience. Le pourcentage de lésions pulmonaires a été évalué au jour 40 (12 jours post-inoculation).

Résultats: La vaccination avec l'un ou l'autre des vaccins a réduit ($P < .001$) les lésions pulmonaires, augmenté le GMQ ($P < .001$) après l'infection-défi et permis de mieux maitriser la virémie ($P < .001$) par rapport aux porcs non vaccinés.

Implication: Un vaccin PRRSV-2 lignée 8 disponible dans le commerce était aussi efficace qu'un vaccin PRRSV-2 lignée 1 contre une provocation virale hétérologue PRRSV-2 lignée 1.

Porcine reproductive and respiratory syndrome virus (PRRSV)-2 is one of the most important infectious agents affecting the swine industry worldwide. It causes several forms of clinical and subclinical disease that presents with various symptoms including anorexia, fever, respiratory distress, lung lesions, abortion, general weakness, and a decrease in valuable production traits such as feed intake and average daily gain (ADG).[1-3] The economic burden of the disease is due mainly to the effects of the virus in post-weaning pigs, especially through the reduction of ADG[2] with approximately 55% of losses from PRRSV occurring during the growing phase of production.[4]

The PRRSV is a single stranded RNA virus characterized by rapid mutation rates and extensive genetic divergence.[2,5] The PRRSV is classified as two species: PRRSV-1 (*Betaarterivirus suid 1;* formerly European PRRSV) and PRRSV-2 (*Betaarterivirus suid 2;* formerly North American PRRSV). The PRRSV-2 is widely spread throughout North America and Asia, and is further divided into 9 distinct lineages based on open reading frame 5 (ORF5) sequences.[5] Over the past 20 years the lineage distribution and prevalence has varied greatly with lineage 1 being the most common strain of the virus currently in the United States.[6,7] This genetic diversity is a challenge for sustained efficacy of current vaccines.[2,6,8] Nevertheless, studies of many commercially available vaccines have reported heterologous protection against the newest strains currently in US swine production systems.[9] For instance, a PRRSV-2 lineage 8 modified live virus (MLV) vaccine has been proven effective in protecting pigs from lung lesions and maintaining production parameters when challenged with PRRSV-2 lineages 1, 3, 5, 8, and 9.[5,10]

Other studies have shown that preventative vaccination with several commercially available vaccines, including MLV vaccines, reduce lesions and other clinical signs following a PRRSV-2 challenge with similar or different lineage than that of the derived vaccine.[2,11] This protective effect has been described as the ability of the vaccines to reduce lung lesions and hinder the ADG decrease that these new PRRSV-2 strains cause.[2,3,9] Lung lesions caused by various diseases have been associated with decreased ADG,[1,12] with viral load usually correlated to the severity of the lesions.[13] However, since some vaccines seem to show similar degrees of protection against clinical signs and lung lesions in pigs with widely divergent viremia,[13] there is a possibility of a more direct negative relationship between PRRSV viral load and ADG. Certainly, viral load has also been negatively correlated with feed efficiency in PRRSV-infected pigs.[14]

Our hypothesis was that lung lesions and viremia would be similar between the two vaccinated groups and that both groups would have less viremia and fewer lung lesions than nonvaccinated pigs. We also hypothesized that vaccinated animals would have similar post-vaccination shedding and would be significantly protected against a PRRSV-2 challenge, regardless of the lineage from which the MLV vaccine was derived. The objective of this study was to investigate and compare viremia in nonvaccinated pigs and in pigs vaccinated with either a lineage 8 MLV vaccine (Fostera PRRS) or a lineage 1 MLV vaccine (Prevacent PRRS) for vaccine shedding prior to challenge, and lung lesions score and viremia post challenge.

Animal care and use

The study was conducted at Swine Services Unlimited, Inc (SSUI) and was approved by the SSUI Animal Care and Use Committee.

Materials and methods

Animals

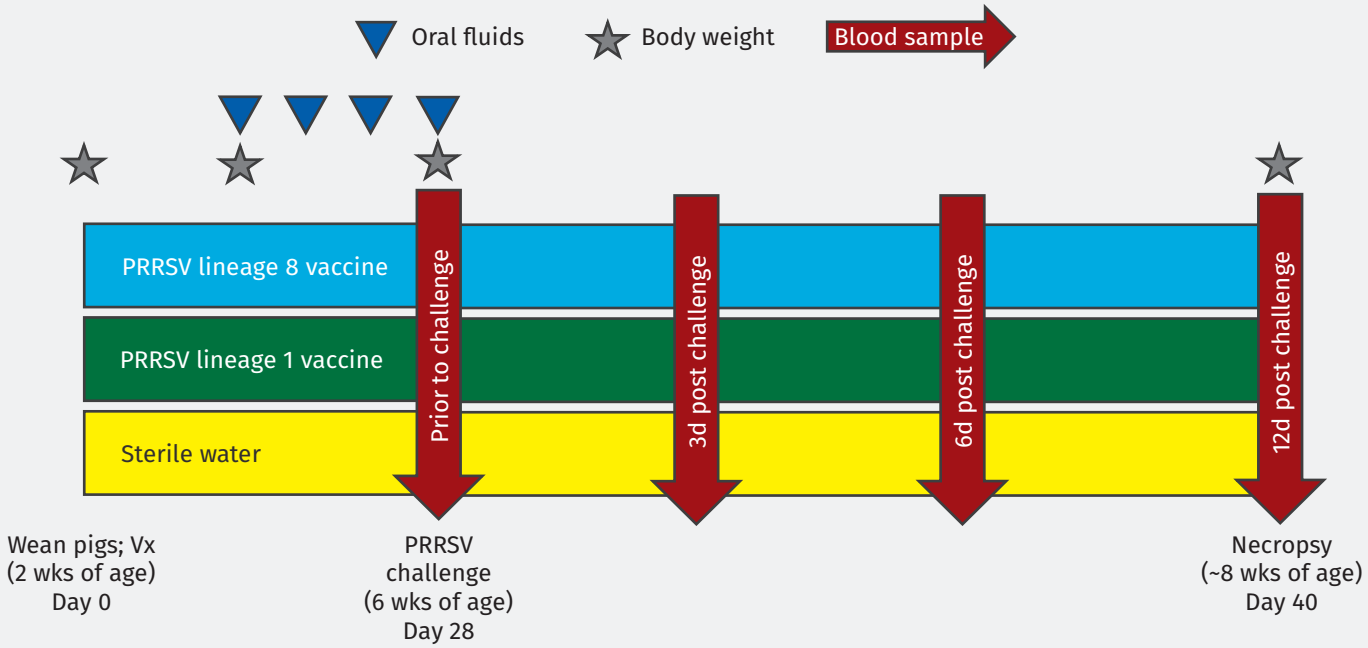
All pigs originated from a single PRRSV-naive sow farm. Piglets on study were from litters born within 4 days of each other from second parity sows. The health status of the farm was high (ie, negative for *Mycoplasma hyopneumoniae* and influenza A virus, and stable for porcine circovirus type 2). Sows were vaccinated for porcine parvovirus, erysipelas, and leptospirosis (FarrowSure Gold; Zoetis) prebreeding and for rotavirus, enterotoxemia, and colibacillosis (ProSystem RCE; Merck) prior to farrowing. Piglets were given 1 mL of injectable iron (Uniferon; Pharmacosmos) the day after birth. Pigs on study were weaned at 2 weeks of age. Special nutritional care was provided for the piglets. Upon arrival at the research site, all piglets were weighed and tagged.

Experimental design

Using SAS 9.4 (Cary, NC), randomization within sex occurred by ranking pigs in descending order using their Day 0 weight. Starting with the heaviest males, each consecutive sequence of 3 animals were grouped together to form a block. The 3 pigs within each block were then randomly allocated to 1 of 3 treatment groups: 1) an experimental serial (L0817LW02; 4.36 Log_{10} $TCID_{50}$/2 mL) of a PRRSV lineage 8 MLV vaccine (2 mL; Fostera PRRS; Zoetis; n = 52), which was unique among commercial live PRRSV-2 vaccines in that it was attenuated by serial passing on cells expressing the porcine CD163 gene; 2) a PRRSV lineage 1 MLV vaccine (1 mL; Prevacent PRRS; Elanco; n = 50); or 3) sterile water (2 mL; n = 47). After the males were allocated, the same allocation procedure was used for females. Pigs were then placed into 1 of 3 rooms to prevent cross-contamination of vaccine virus (Figure 1). There were 8 pigs placed per pen prior to challenge. Once in their respective rooms, pigs were vaccinated according to their assigned treatment group. Pigs were observed for 15 minutes following vaccination for any adverse reactions (ie, anaphylactic shock and injection site reactions), but none were observed. One week post vaccination, individual body weight was recorded to determine if vaccination had an impact on growth.

Four cotton ropes were hung per room on a weekly basis (ie, days 7, 14, 21, and 28) and after approximately 20 minutes, fluids were collected into a plastic 50 mL conical tube. Oral fluids (n = 4/room) were shipped on wet ice on the day of collection to the Iowa State University Veterinary Diagnostic Laboratory (ISU VDL) to determine vaccine shedding by quantitative polymerase chain

Figure 1: Timeline of vaccination and PRRSV 1-7-4 challenge. At 2 weeks of age, pigs were weaned (day 0), weighed, randomized into 1 of 3 treatments by day 0 weights, placed into 3 rooms, and vaccinated per treatment assignment. Day 7 weights were used as an indicator of vaccination setback. To determine vaccine shedding, weekly oral fluids were collected prior to challenge. The PRRSV 1-7-4 challenge occurred on day 28 with blood samples occurring prior to challenge (day 28) and 3 (day 31), 6 (day 34), and 12 (day 40) days later. Pigs were euthanized and lungs assessed for lesions on day 40. PRRSV = porcine reproductive and respiratory syndrome virus; Vx = vaccination.



reaction (qPCR) for PRRSV. Four weeks after vaccination (study day 28; 6 weeks of age), all animals were challenged with PRRSV-2 ORF5 restriction fragment length polymorphism (RFLP) 1-7-4 ($TCID_{50}$: 1×10^4/4 mL dose; 2 mL intranasally and 2 mL intramuscularly; Zoetis). After the individual challenge, pigs were co-mingled within their designated block. There were 6 pigs placed per pen (2 from each treatment; 1 male and 1 female). Body weights and blood samples were taken on the day of, but prior to, challenge and co-mingling. Thereafter, blood samples to test for viremia were obtained on days 3, 6, and 12 post challenge. Blood was collected using one needle and one vacutainer tube per pig. Blood was transported in a cooler on wet ice back to the laboratory. Blood samples were processed by centrifugation for 10 minutes at 1800*g*. Serum was stored at -70°C until shipped on dry ice as one shipment to ISU VDL. Twelve days post challenge (study day 40), body weights were recorded, and necropsies performed to determine percentage of lung lesions. Each individual lobe of the lungs was assessed for gross surface lesions by Dr Mueller and lesion score recorded. The calculation to determine percentage lung lesions was as follows: Percentage of total lung with lesions = 100 × (0.10 × left cranial lobe) + (0.10 × left middle lobe) + (0.25 × left caudal lobe) + (0.10 × right cranial lobe) + (0.10 × right middle lobe) + (0.25 × right caudal lobe) + (0.10 × accessory lobe). All serum samples from the study were shipped to ISU VDL where qPCR for PRRSV was performed.

To determine potential post-vaccination shedding, PRRSV reverse transcriptase-qPCR (RT-qPCR) was performed on oral fluids. Moreover, due to positive PCR cycle threshold (Ct) values on day 28 (just prior to challenge), ORF5 genomic sequencing was completed by ISU VDL on 6 pigs (3 pigs/room) to confirm that it was vaccine virus and not wild-type virus.

qPCR analysis

All samples were sent at the conclusion of the experiment and processed by ISU VDL. Briefly, nucleic acids were extracted using a Thermo Electron KingFisher Flex automated magnetic particle processor system. The 5X MagMAX™ Pathogen RNA/DNA Kit (Applied Biosystems) was used with the Thermo Electron KingFisher Flex according to manufacturer specifications.

The PRRSV RT-qPCR was performed using the 10X PRRSV Primer Probe Mix V2 from the VetMAX PRRSV NA and EU kit. The assay was modified from the original kit to use TaqMan Fast Virus 1-Step Master Mix (4×) along with the addition of Amplitaq 360 DNA Polymerase. Each reaction consisted of 6.5µL of TaqMan Fast Virus 1-Step Master Mix (4×), 0.8µL Amplitaq 360 DNA Polymerase (5U/µL), 2.7µL of nuclease-free water, 2.0µL of the 10× PRRSV Primer Probe Mix V2, and 8.0µL of nucleic acid template. The assay was run on the ABI-7500 Fast system, using the 7500 Fast System SDS Software Version 1.4.0.27. All samples were assayed within two days, with each day on separate plates. To control for plate-to-plate variation, a positive extraction control and negative extraction control were included on each extraction plate, which went through the entire process, as well as a negative amplification control that went through just the PCR step. Statistical Process Control (SPC) charting of the Cts of the positive controls were plotted to ensure Ct values were within allowed ranges. If they were not, the testing was repeated. The assay also included an internal positive control added to each sample at the time of extraction. This internal positive control needed to be detected in every sample to verify the process was performed correctly.

Statistical analysis

This study was conducted as a split-plot design with sex as the whole-plot factor and treatment as the split-plot factor. Individual animal was the experimental unit. Tests for normality and goodness of fit (Shapiro-Wilk test) were run for all data via Proc Univariate (SAS 9.4). Percentage lung lesions was transformed by the arcsine (square-root [%]) transformation and analyzed by a generalized linear mixed model (GLMM) approach (SAS Proc Mixed Procedure; SAS 9.4). Initial weight, day 40 weight (end of study) and ADG from day 28 (time of co-mingling) to day 40 were analyzed with a GLMM approach. For all variables, the model consisted of the fixed effects of treatment, sex, and the interaction of treatment × sex, and the random effects of room, pen (room), block (room × pen × sex) and the residual error. Body weights and ADG before co-mingling were summarized but not statistically analyzed.

For viremia, Ct values were transformed by natural log transformation prior to statistical analysis, as they were not normally distributed. Transformed values were analyzed using a GLMM approach for repeated measures. Using the Proc Mixed Procedure, transformed data were analyzed with a model that considered the fixed effects of treatment, sex, day, treatment × sex, treatment × day, sex × day, and treatment × sex × day and the random effects of room, pen (room), block (room × pen × sex) and the residual error. Day was the repeated factor. Pig was the subject. The covariance structure in the repeated measures analysis was investigated using six structural assumptions: compound symmetry, heterogeneous compound symmetry, spatial power, first order autoregressive, heterogeneous first order autoregressive, and unstructured. The assumption giving the minimum value of the Akaike's Information Criterion was selected in the final analysis. Unstructured was the selected covariance structure. Because the treatment × day interaction was significant, LSMeans comparisons were assessed for each study day. For all variables of interest, treatment and all interactions were assessed at the 5% level.

Results

Prior to co-mingling, oral fluids analyzed for PRRSV demonstrated that there was potential shedding of vaccine virus in pigs from both vaccinated treatment groups (Figure 2). There was no evidence of PRRSV within the samples collected from the nonvaccinated room indicating that there was no contamination of vaccine virus to the control pigs. Moreover, weights obtained on day 7 were numerically similar across treatment groups (Table 1).

All nonvaccinated control pigs were negative for PRRSV prior to challenge, and all pigs became viremic after challenge (Figure 3). Pigs from both vaccinated treatment groups had similar viremia levels on day 28 (just prior to challenge), and sequencing data indicated that these positive values were vaccine virus, not wild-type PRRSV-2 (data not shown). At 3- and 6-days post challenge (study days 31 and 34), PRRSV-2 Ct values for pigs vaccinated with the lineage 1 vaccine were decreased ($P < .001$ on day 3; $P = .02$ on day 6) indicating a greater viral load compared to lineage 8 vaccinated and nonvaccinated control pigs, which did not differ (Figure 3A). By day of necropsy (day 40; 12 days post challenge), pigs vaccinated with the lineage 1 vaccine had significantly greater ($P = .005$) Ct values compared to pigs vaccinated with the lineage 8 vaccine, that in turn exhibited significantly greater ($P < .001$) Ct values compared to nonvaccinated control pigs (Figure 3A). To remove the effect of vaccine virus from the data (Figure 3A), the percentage change from time 0 (day 28) was analyzed for each pig. On day 31 and 34 (3- and 6-days post challenge), nonvaccinated pigs experienced the greatest ($P < .001$) decrease in Ct values (Figure 3B) from day 28. Lineage 1 MLV vaccinated pigs had a greater decrease in Ct values compared to lineage 8 vaccinated pigs on days 31 ($P < .001$) and 34 ($P = .01$). By day 40 (day of necropsy), the percentage change was significantly greater in the nonvaccinated control pigs compared to either vaccinated group ($P < .001$), which did not differ (Figure 3B).

On day 40 (12 days post challenge) the GMean (SEM) percentage of lung lesions in the nonvaccinated group (20.03% [2.16%]) was significantly greater than in either of the vaccinated groups ($P < .001$). The GMean (SEM) percentage lung lesions in the lineage 8 vaccinated group (2.55% [0.82%]) and in the lineage 1 vaccinated group (1.60% [0.66%]) were not significantly different from each other ($P = .36$).

There was no effect of treatment ($P = .34$) or treatment × sex interaction ($P = .94$) on initial weight for animals in any of the 3 treatment groups (Table 2). As expected, males were heavier ($P < .001$) than females (data not shown). On day 40, nonvaccinated pigs were lighter ($P < .001$) compared to the pigs in either of the vaccinated groups, which did not differ significantly from each other (Table 2). Similarly, nonvaccinated pigs had a decreased ($P < .001$) ADG from days 28 to 40 compared to pigs in the vaccinated treatment groups, which did not differ significantly from each other (Table 2). Statistical analyses were only performed once pigs were co-mingled (day 28). A summary of all pig weights is reported in Table 1.

Discussion

We fail to reject our hypothesis that viremia and lung lesions that are induced by a PRRSV-2 lineage 1 challenge are controlled by a PRRSV-2 lineage 8 vaccine, as well as a homologous vaccine (ie, a PRRSV-2 lineage 1 vaccine). Viremia (measured by qPCR) has been negatively correlated with feed efficiency in PRRSV-infected animals and negatively correlated with ADG in PCV2-infected animals.[15] Vaccines evaluated in this study have fundamentally different methods of attenuation, but further investigations are needed to determine if this influences onset of immunity. Indeed, other studies have reported a negative relationship between lung lesions and production traits such as ADG and average daily feed intake.[1,12] Moreover, viremia has repeatedly been negatively correlated with ADG in PRRS[16] and other respiratory diseases.[13]

Lung lesion scoring, viremia, and production trait measurements are the gold standards to assess protection against PRRSV.[2,3,9,17] In this study, both vaccines were similar in their ability to protect against a PRRSV-2 lineage 1 challenge. Pigs vaccinated with either product demonstrated significant protection compared to the nonvaccinated pigs. It has been reported that vaccines derived from more contemporary viral lineages may be more protective compared to vaccines derived from older lineages.[2,11,18] However, this study adds to the reports that a lineage 8 vaccine was just as effective as a lineage 1 vaccine at protecting against a PRRSV 1-7-4 (lineage 1) challenge. Protection against a PRRSV challenge cannot be accurately predicted by the percentage sequence identity between the virus from which the vaccine was made and the virulent PRRSV-2 in circulation.[19,20] Strains of PRRSV are often described based on RFLP patterns, which are calculated from ORF5 sequences.[6,20] However,

Figure 2: Cycle threshold (Ct) values from oral fluid sample collected post vaccination. Upon arrival at the study site, pigs from each treatment group were placed into individual rooms and vaccinated according to their assigned treatment. Oral fluids from 4 ropes/room were collected weekly until co-mingling and challenge with porcine reproductive and respiratory syndrome virus (PRRSV) 1-7-4. Each oral fluid sample was submitted for detection of PRRSV via polymerase chain reaction. Individual samples are shown by a circle. The × indicates the average Ct value for that room at each week. Some values overlap.

RFLP designations have shortcomings as the genetic relationship between different RFLP types is not obvious, and there are many examples of two distantly related viruses sharing the same RFLP pattern.[6] The RFLP nomenclature is most useful for distinguishing between a new virus and a limited number of resident field and vaccine viruses in a small geographic region and over a short period of time. For long term global classification of PRRSV-2, it is much more useful to use the entire ORF sequence to phylogenetically organize the genetic diversity into lineages and sublineages (or subtypes in the case of PRRSV-1). Even though many contemporary viruses are lineage 1, this does not necessarily mean they are closely related to each other or to lineage 1 vaccines. Lineage 1 is the most diverse of the PRRSV-2 lineages, and there is as much variability within lineage 1 as there is between certain other lineages.[6,20] In this study, the percentage of lung lesions in nonvaccinated animals was decreased to a similar degree in animals vaccinated with either the lineage 8 vaccine or the lineage 1 vaccine. This further confirms the efficacy of the lineage 8 vaccine against PRRSV-2 lineage 1 challenges.

Our dataset adds to the scientific literature that a PRRSV-2 lineage 8 vaccine is effective against a PRRSV-2 lineage 1 viral challenge. Both vaccines proved to be similar in protecting against lung lesions, weight loss, and ADG reduction. Moreover, the lineage 8 vaccinated pigs had reduced wild-type viremia compared to lineage 1 vaccinated and nonvaccinated control animals at 3- and 6- days post challenge with an PRRSV-2 ORF5 RFLP 1-7-4 lineage 1 virus.

Implication

Under the conditions of this study:

- The PRRSV-2 lineage 8 and lineage 1 vaccines were equally effective in a PRRSV-2 lineage 1 challenge.

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Table 1: Mean (SD) body weight and ADG of pigs vaccinated with a PRRSV lineage 8 vaccine, PRRSV lineage 1 vaccine, or sterile water at 2 weeks of age*

	PRRSV lineage 8 vaccine (n = 52)	PRRSV lineage 1 vaccine (n = 50)	Nonvaccinated control (n = 47)
Weight, mean (SD), kg			
Day 0	5.31 (0.99)	5.33 (0.88)	5.40 (0.93)
Day 7	6.98 (1.10)	6.86 (0.98)	6.92 (1.00)
Day 28	13.82 (2.35)	13.64 (1.75)	14.21 (2.19)
Day 40	18.90 (2.75)	19.18 (2.81)	16.40 (2.95)
ADG, mean (SD), kg/d			
Day 0 to 7	0.24 (0.04)	0.22 (0.03)	0.22 (0.03)
Day 0 to 28	0.30 (0.06)	0.30 (0.04)	0.32 (0.06)
Day 0 to 40	0.33 (0.05)	0.34 (0.05)	0.27 (0.06)

* Day 0 = start of trial and vaccination; Day 7 = determine any vaccination setback; Day 28 = day of challenge and co-mingling; Day 40 = end of project.

ADG = average daily gain; PRRSV = porcine reproductive and respiratory disease virus.

Conflict of interest

Authors Vonnahme, Angulo, Amodie, Mellencamp, and Galina Pantoja are employed by Zoetis, Inc, the manufacturer of Fostera PRRS and have ongoing financial interest in the sale of Fostera PRRS. All authors contributed to the design of the study. Authors Vonnahme and Vasquez-Hidalgo were the primary authors; Vasquez-Hidalgo and Amodie performed the statistical analyses; all authors reviewed the manuscript prior to submission.

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Figure 3: Viremia of pigs vaccinated with either a PRRSV lineage 8 vaccine (blue), PRRSV lineage 1 vaccine (green), or sterile water (nonvaccinated; yellow) at 2 weeks of age. Challenge with PRRSV 1-7-4 occurred on day 28, and blood samples were collected on day 0 (study day 28; prior to challenge), 3-, 6-, and 12-days post challenge. A) The cycle threshold (Ct) values for wild-type PRRSV for all pigs. The positive values observed on day 0 from vaccinated pigs was determined to be their respective vaccine virus via sequencing (data not shown). B) The percentage change in Ct value was calculated to determine the proportion of Ct value that was due to the vaccine virus. LSMeans (SEM) within a day and panel with different superscripts (^{a,b,c}) differ; $P \leq .05$.

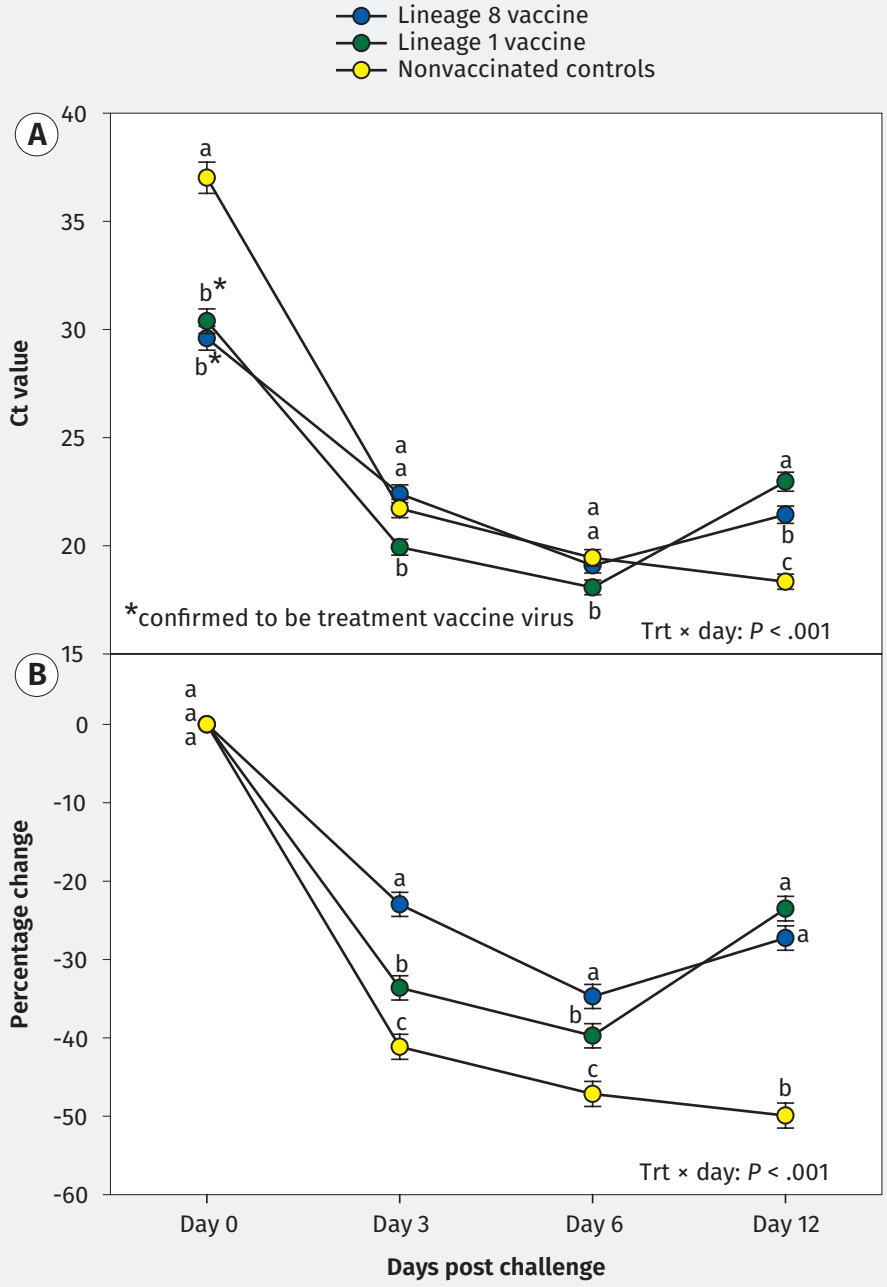

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Table 2: LSMeans (SEM) of body weight and ADG from day 28 (day of co-mingling) to end of study (day 40) of pigs vaccinated with PRRSV lineage 8 vaccine, PRRSV lineage 1 vaccine, or sterile water at 2 weeks of age (day 0)

	PRRSV lineage 8 vaccine (n = 52)	PRRSV lineage 1 vaccine (n = 50)	Nonvaccinated control (n = 47)	*P*
D 0 wt, kg	5.37 (0.76)[a]	5.35 (0.76)[a]	5.39 (0.76)[a]	.34
D 40 wt, kg	18.97 (1.58)[a]	19.22 (1.58)[a]	16.49 (1.58)[b]	< .001
ADG (Day 28 to 40), kg/d	0.41 (0.03)[a]	0.46 (0.03)[a]	0.19 (0.03)[b]	< .001

[ab] LSMeans (SEM) within a row with different superscripts statistically differ.

ADG = average daily gain; PRRSV = porcine reproductive and respiratory syndrome virus.

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The sow microbiome: Current and future perspectives to maximize the productivity in swine herds

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Summary

The development of new generation sequencing methods and the reduction in the cost per base sequenced over the past few years is drawing the attention of the pig industry to microbiome understanding and modulation. In recent years, there has been an increase in the number of articles published related to microbiome studies in swine. With respect to sows, microbiome studies mainly focused on the gut, with some studies evaluating the reproductive tract and mammary microbiome. However, studies about urinary microbiome are still lacking. The present literature indicates that the microbiome in the sow's gut can affect the microbiome in other body parts. Moreover, the understanding of the dynamics and interactions among microbial populations within the sow or the herd has led to improvements in animal health and reproductive performance. This review provides new insights related to sow intestinal, urinary, mammary, and reproductive microbiomes and their relationships with reproductive outcomes, diseases, and early colonization in offspring by gathering the most recent work in this field as well as pinpoints information gaps that require further investigation. This literature review also sheds light on the knowledge regarding the role of microbiomes in the reduction of antimicrobial use.

Keywords: swine, dam, microbiota, reproduction, diseases

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Resumen - El microbioma de la cerda: Perspectivas actuales y futuras para maximizar la productividad en las piaras porcinas

En los últimos años el desarrollo de métodos de secuenciación de nueva generación y la reducción en el costo por base secuenciada está atrayendo la atención de la industria porcina hacia la comprensión y modulación del microbioma. En los últimos años, ha habido un aumento en el número de artículos publicados relacionados con estudios del microbioma en cerdos. Con respecto a las cerdas, los estudios del microbioma se centraron principalmente en el intestino, con algunos estudios que evaluaron el tracto reproductivo y el microbioma mamario. Sin embargo, todavía faltan estudios sobre el microbioma urinario. La literatura actual indica que el microbioma en el intestino de la cerda puede afectar el microbioma en otras partes del cuerpo. Además, la comprensión de la dinámica y las interacciones entre las poblaciones microbianas de la cerda o de la piara han llevado a mejoras en la salud animal y el rendimiento reproductivo. Esta revisión de los trabajos más recientes en esta área proporciona nueva información relacionada con los microbiomas intestinales, urinarios, mamarios, y reproductivos de las cerdas, su relación con los resultados reproductivos, las enfermedades, y la colonización temprana de su progenie e indica también la falta de información que requiere mayor investigación. Esta revisión de la literatura también se expone el conocimiento del rol de los microbiomas en la reducción del uso de antimicrobianos.

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Monteiro MS, Poor AP, Muro BBD, Carnevale RF, Leal DF, Garbossa CAP, Moreno AM, Almond G. The sow microbiome: Current and future perspectives to maximize the productivity in swine herds. *J Swine Health Prod*. 2022;30(4):238-250. **https://doi.org/10.54846/jshap/1277**

Résumé - Le microbiome de la truie: Perspectives actuelles et futures pour maximiser la productivité des troupeaux porcins

Le développement de méthodes de séquençage de nouvelle génération et la réduction du coût par base séquencée ces dernières années attirent l'attention de la filière porcine sur la compréhension et la modulation du microbiome. Au cours des dernières années, il y a eu une augmentation du nombre d'articles publiés liés aux études sur le microbiome chez le porc. En ce qui concerne les truies, les études sur le microbiome se sont principalement concentrées sur l'intestin, certaines études évaluant l'appareil reproducteur et le microbiome mammaire. Cependant, les études sur le microbiome urinaire font encore défaut. La littérature actuelle indique que le microbiome dans l'intestin de la truie peut affecter le microbiome dans d'autres parties du corps. De plus, la compréhension de la dynamique et des interactions entre les populations microbiennes au sein de la truie ou du troupeau a permis d'améliorer la santé et les performances de reproduction des animaux. Cette revue fournit de nouvelles informations sur les microbiomes intestinaux, urinaires, mammaires, et reproducteurs des truies et leurs relations avec les résultats de la reproduction, les maladies, et la colonisation précoce de la progéniture en rassemblant les travaux les plus récents dans ce domaine et en identifiant les lacunes en matière d'informations qui nécessitent une recherche plus approfondie. Cette revue de la littérature met également en lumière les connaissances concernant le rôle des microbiomes dans la réduction de l'utilisation des antimicrobiens.

Productivity of the sow herd is traditionally measured by the number of pigs weaned[1] or kilograms of piglets weaned per sow per year.[2] Longevity is another factor that can impact herd productivity and is directly affected by disease.[3] Antimicrobials are used in all production phases of pig production; and with respect to the sow, they are more frequently used during the lactation phase.[4] Reproductive failures and diseases frequently associated with polymicrobial organisms are traditionally controlled with use of in-feed, broad-spectrum antimicrobials.[5,6] It is estimated that a sow will be treated with at least one active antimicrobial ingredient for an average 3.2 days/year,[7] however this is often underestimated in treatment records.[8] The category of antibiotics used in sows varies greatly between herds, but it was reported that 26% of all herds use antibiotics to treat sows.[9] Rosengren et al[10] reported an incidence of 7.84 sows treated with antibiotics per 1000 sows/day, while Sjölund et al[11] reported an incidence of 42 sows treated with antibiotics per 1000 sows/day. In some herds, all sows were routinely injected with an antimicrobial agent after farrowing.[10] The majority of antimicrobials used in swine herds are classified as critically important or highly important by the World Health Organization.[12] Rosengren et al[10] reported that some herds routinely use ceftiofur for treating sows. The use of third-generation cephalosporins has increased since 2001 and an increase in bacterial isolates from healthy swine showing extended-spectrum, beta-lactamases was observed in the same period.[13] Ceftiofur is restricted to use in animals but is similar to ceftriaxone, which is widely used in human medicine. Therefore, ceftiofur should not be used as a first-choice antimicrobial for sows.[12] The use of antimicrobials in animal production is a public health matter, as it engenders selection pressure for resistance to antimicrobials. Of all swine, sows are the pigs least treated with antimicrobials.[9,11] Attention should be paid to antimicrobial administration to sows as they can act as a reservoir for transferal of resistant bacteria to their offspring.[4] Due to recent concerns about antimicrobial resistance and the subsequent restrictions on the use of antimicrobials in animal production, researchers are looking for new alternatives to prevent and treat disease. One possible alternative relies on unveiling the mechanisms by which the microbiome interacts with the host and its relationship with health and productivity.[14-16]

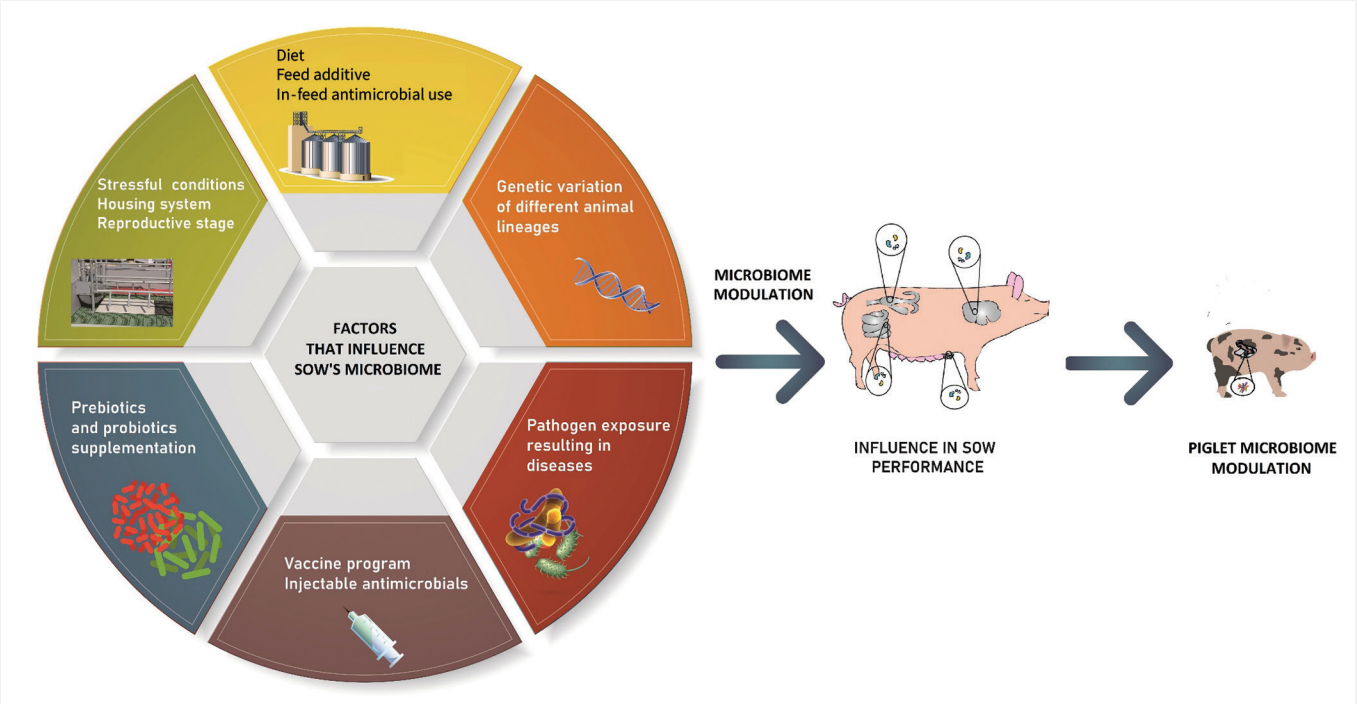
The microbiome is defined as a characteristic microbial community occupying a well-defined habitat which has distinct physio-chemical properties and includes the whole spectrum of molecules produced by the microorganisms, their structural elements, metabolites, and molecules produced by the host and are influenced by the surrounding environmental conditions. The microbiome is prone to change in time and scale and is essential for multicellular organism health.[17,18]

Studies associating the microbiome with disease have been carried out in various species, including humans.[16,19-23] Alterations in vaginal and intestinal microbiomes can reduce urinary tract infections and gut infections in humans.[19,24,25] This new knowledge opens possibilities for new studies to provide a better understanding about microbiome relationships with diseases and reproductive performance. In sows, several factors may alter the microbiome composition. It was reported that antimicrobials used,[26] reproductive stage,[27] genetic line,[28] feed additives, probiotic and prebiotic supplementation,[29] pathogen exposure, vaccines to prevent disease,[23] and stressful conditions[30] can affect the microbiome. Some of these factors are being studied to increase sow productivity by microbiome modulation[14,15,28] alongside studies investigating the possibility of modulating the offspring microbiome through sow microbiome modulation.[29,31,32] These factors are presented in Figure 1.

In pigs, microbiome modulation can prevent disease and reduce the use of antimicrobials.[33] Pathogen exposures can cause dysbiosis,[23] which can result in an unstable microbiome and increase susceptibility to diseases caused by opportunistic organisms.[34] Both factors contribute to development of disease in sows and impair productivity. Development of a stable microbiome by administration of *Lactobacillus* to newborn piglets has been shown to reduce diarrhea and improve weaning weight.[35,36] Similarly, probiotic supplementation to weaned piglets had a positive effect on average daily gain and reduced diarrhea[37-39] and *Salmonella* shedding.[37] Other studies in swine indicate interaction between the microbiome and other areas of the body. It was observed that *Enterococcus faecalis* EC-12 increased the response of *ex vivo* tissue to immunostimulants such as porcine reproductive and respiratory syndrome virus (PRRSV) modified live virus vaccine.[40] A fecal microbiota transplant (FMT) had beneficial effects in pigs challenged against *Mycoplasma hyopneumoniae*, reducing gross lung pathology.[41]

In sows, there is evidence that changes in the local microbiome (eg, intestinal and vaginal microbiome) may have led to effects in different systems and,

Figure 1: Factors that may influence the sow reproductive, urinary, and digestive tracts, colostrum, and milk microbiomes and, consequently, sow performance and the microbiome of their offspring.



consequently, several biomarkers for productivity and optimal health were found.[14,15,30] It was observed that symbiotic supplementation in sows improved their litter performance.[42] It is possible to modulate the sow's microbiome through microbiome transplantation, altering endometrial glands, circulating hormones, and improving reproductive efficiency.[28,43] Research to date has focused mainly on piglet microbiomes, so there is a lack of information regarding the use of probiotics to prevent vaginal discharge, cystitis, mastitis, and diseases that have a great economic impact in sow herds.

For decades, microbiology research has focused on culture methods or detection of individual microbial species or polycultures that may not represent the full bacterial population and diversity since most microorganisms could not be grown by traditional culture methods.[44] The seminal work of Woese and Fox[45] in the 1970s using ribosomal RNA (rRNA) as a bacterial evolutionary marker, mainly with the 16S rRNA gene, revolutionized microbiology research. This and the development of new generation sequencing (NGS) methods have made it possible to characterize the bacterial community in all its richness, diversity, and relative abundance, even in tissues believed to be sterile.[46] Recently, technological advances have allowed a drastic reduction in sequencing costs, mainly due to the emergence of commercial high-throughput sequencing platforms,[47] and research involving the assessment of the microbiome in swine has gained importance.

Despite the increase of microbiome analysis research, there is a lack of studies correlating the microbiome with its impact on sow productivity. Furthermore, studies that perform organism-based metabolic analysis, identify microbe-microbe interactions, and identify microbe-host interactions are even more scarce. The microbiome is complex, and studies focused on system-based approaches would probably provide more valuable information.[48,49] Thus, this review aims to compile information related to modifications or alterations in the microbiome to improve reproductive performance, as well as to point out topics that require further investigation.

The reproductive tract microbiome

The number of studies analyzing the vaginal microbiome of sows has increased, especially in the last four years.[14,15,50-54] Studies have focused on identifying possible biomarkers related to increased productivity,[14] infectious diseases in target sites, ie, endometritis,[16] and immune responses against systemic diseases, such as PRRS. The vaginal microbiome was also studied to identify possible biomarkers for diseases that have an ill-defined biological factor, such as prolapses.[51]

Endometritis has a major impact on the reproductive efficiency of sows[55-58] and its main cause is bacterial infection.[59,60] Common clinical manifestations include purulent vulvar secretion, reproductive failure, abortion, anestrus, reduced farrowing rates, inappetence, and poor body condition which often leads to sow culling.[60,61] This condition could also predispose the sow to other diseases such as postpartum dysgalactia syndrome (PDS) and cystitis.[62,63] Vaginal discharge is the reported reason for 20.5% of culled sows, and endometritis was the most common postmortem lesion (14.5%) in sows culled due to anestrus and repeated breeding.[61]

The application of culture methods associated with biomolecular techniques, notably polymerase chain reaction (PCR), has identified several organisms in purulent vaginal discharge, such as *Escherichia coli*, *Staphylococcus*, *Streptococcus*, *Trueperella pyogenes*,[60,63] *Arcobacter*,[64]

Chlamydia,[65] *Proteus*, *Pseudomonas*, and *Corynebacterium*.[63,66] The most common organism found was *E coli*, which was isolated in more than 30% of endometritis cases.[60] Despite the great potential of extraintestinal pathogenic *E coli* to cause metritis, it can also be part of the vaginal microbiome in samples from healthy sows.[16,52-54] The NGS-based studies have corroborated the importance of some of these organisms previously identified by traditional methods, such as *E coli, Staphylococcus,* and *Streptococcus*.[16,52] However, NGS metagenomic techniques allow the identification of microbes at a whole community level, in addition to allowing the comparison of relative abundances of each microbe type. This allows for greater resolution to identify organisms which are difficult to identify with traditional methods and may be important in dysbiosis such as low-abundance or fastidious microbes (eg, *Bacteroides*, *Clostridium*, and *Fusobacterium*) recently identified in metagenomic approaches as important pathogenic causes of endometritis.[16,52,53]

Previous studies demonstrated that the vaginal microbiome may act as biological barrier by secreting antimicrobial components such as lactic acid, bacteriocin, and hydrogen peroxide to maintain the health of the reproductive tract.[67,68] Therefore, a sow's vaginal microbiome is complex and even potentially pathogenic bacteria can be part of the community, suggesting that urogenital diseases may arise from dysbiosis.

Wang et al[16] analyzed sow vaginal samples classified as either affected or not affected by endometritis. The Firmicutes phylum was the most abundant (40%-60%) in the vaginal microbiome followed by Proteobacteria (20%-32%) and Bacteroidetes (9%-13%). However, the Firmicutes phylum had the greatest relative abundance in healthy sows, while Proteobacteria and Bacteroidetes were more abundant in samples of sows affected by endometritis. At the genus level, Wang et al[16] found that *Bacillus* and *Paenibacillus* were relatively more abundant in the healthy sows, while *Escherichia-Shigella* and *Bacteroides* were relatively more abundant in sows affected by endometritis. Wang et al[16] observed that one sow with endometritis had a great abundance of *Staphylococcus* during the metagenomic analysis, although the microbial species within the *Staphylococcus* genus was not classified. Experimental inoculation with *Staphylococcus hyicus* caused endometritis in sows in a previous study, as did *E coli*.[59]

Similarly to Wang et al[16], Zhang et al[52] found that sows with endometritis had a higher relative abundance of *Porphyromonas, Clostridium sensu stricto 1, Streptococcus, Fusobacterium, Actinobacillus,* and *Bacteroides* in the birth canal. *Escherichia-Shigella* and *Bacteroides* were higher in the intestines of sows suffering from endometritis, suggesting a link between the onset of endometritis and the increase of these organisms in intestinal microbiota. Xu et al[53] also found the phyla Proteobacteria, Firmicutes, and Bacteroidetes among the most abundant in sow vaginal samples; at the genus level, the most abundant were *Escherichia, Streptococcus, Enterococcus, Bacillus, Clostridium sensu stricto 1, Staphylococcus, Acinetobacter, Lactobacillus,* and *Proteus*. Although *Escherichia-Shigella*, *Clostridium sensu stricto 1,* and *Streptococcus* relative abundance were related to endometritis in the other studies,[16,52] no sow had endometritis in the Xu et al[53] study. However, the small number of females evaluated in these two studies (n = 8) precludes stronger conclusions.

Furthermore, Xu et al[53] showed that the addition of lysozyme, an antimicrobial enzyme that occurs naturally in the mucosal barrier of mammals, to the diet of sows affected the vaginal bacterial community by decreasing the relative abundance of *Escherichia-Shigella* and increasing *Lactobacillus*. Members of the Lactobacillaceae family are most abundant in the birth canal of healthy women and are considered protective against infection by other organisms and probiotic candidates.[69] The metagenomic studies related to the vaginal microbiome did not observe a higher prevalence of *Lactobacillus* in healthy sows[16,52] and that even healthy sows carried a higher prevalence of potential pathogenic or opportunistic organisms.[16,52] These results indicate that the sow vaginal microbiome is more complex than what is observed in humans, which contributes to the difficulty of describing a core vaginal microbiome in sows since even discrete changes can impair sow health. Therefore, these authors suggested lysozyme as a candidate for the maintenance of a beneficial vaginal microbiome and consequently reduce the necessity of antimicrobial use to prevent or treat vaginal discharge in the sow herd. Further studies should elucidate the ability of lysozyme to modulate the sow vaginal microbiome for only beneficial microbes.

Sanglard et al[14] evaluated the vaginal microbiome of sows with low and high reproductive performance after PRRSV vaccination. Sows with low reproductive performance had a higher abundance of noxious bacteria such as *Phascolarctobacterium, Filifactor, Treponema,* and *Bacteroides* compared to sows with high reproductive performance. *Phascolarctobacterium* was negatively correlated with litter weight at day 21 of lactation[27] and *Filifactor* has been associated with metritis in dairy cows.[70] In addition, discriminant linear analysis using the specific genera *Campylobacter, Bacteroides, Porphyromonas,* unclassified Lachnospiraceae*, Prevotella,* and *Phascolarctobacterium* was able to differentiate animals with high and low farrowing performance, indicating that these could serve as potential biomarkers.[14] Understanding the vaginal microbiome and potential biomarkers of high reproductive performance may guide improvements in genetic selection at an early age, even prior to breeding. Sanglard et al[14] verified that this method is minimally invasive and can be performed at early ages, such as 4 and 52 days after PRRSV vaccination (132 ± 12 days of age).

Another study[50] investigated the relationship between the vaginal microbiome and sow genetics and the impact on immune response and farrowing traits in commercial gilts. It was found that the genotype was able to explain up to 33% of the immune response variation to vaccination and 14% of the total microbial variation of the vaginal microbiome. The results indicated that the microbiome can be modulated by genetic selection for beneficial microbes, which may indirectly improve reproductive performance, and the possibility to genetically select sows for a better immune response.[50]

The diversity of the vaginal microbiome has been discussed in recent years. Laguardia-Nascimento et al[71] found great variability in the vaginal microbiome of cows, which contradicted previous studies that used culture methods. Sanglard et al[14] found that the microbiome of sows with low reproductive performance had greater vaginal microbial diversity compared to sows with high reproductive performance.

Another factor that contributes to impaired herd productivity is pelvic organ prolapse. Prolapses are more prevalent during late gestation and early lactation and contributes to approximately 21% of sow mortalities annually.[51,72] Sow mortality during the peripartum period is economically critical because

it increases nonproductive days and impairs neonatal nutrition. Despite the great impact of prolapses, prevention is in part neglected due to an ill-defined biological factor. Kiefer et al[51] observed that alpha diversity revealed no significant differences between samples for species richness, community evenness, and diversity. But when analyzed with linear discriminant analysis, there was abundant differences in 89 total operational taxonomic units between sows with high and low prolapse risk. A higher abundance of Prevotellaceae, *Treponema*, and *Streptococcus dysgalactiae* was observed in high prolapse risk sows. However, principal coordinate analyses revealed no distinct clustering of sows with high or low prolapse risk and the putative markers identified in this work will require determination of causality.[49] While the Sanglard et al,[14,50] Wang et al,[16] Kiefer et al,[51] Zhang et al,[52] and Xu et al[53] studies were not designed to describe a core vaginal microbiome community associated with better reproductive outcomes in sows, they do show that some changes in bacterial composition may influence a sow's disease response and reproductive performance. Further studies focusing on system-based approaches are required to understand the role of the microbiome in reproductive performance.

The urinary tract microbiome

Urinary tract infections (UTIs) have great prevalence in swine herds and cause economic losses due to reproductive failures, increased sow culling, and mortality.[73,74] It was reported that more than 90% of sows with some reproductive disorder also were diagnosed with a UTI.[75] Additionally, UTIs during gestation are reported to reduce litter size by 0.6 piglets/litter.[76] Sows diagnosed with a UTI had 3.5 times higher risk of developing endometritis compared with healthy animals.[63] Furthermore, UTIs are associated with other diseases, such as mastitis metritis agalactia.[77-79]

The UTI etiology is complex, polymicrobial, and may feature rotation or changes in etiological pathogens. Among the possible organisms, *E coli* was the predominant microbiological organisms isolated in single (71%) and mixed (85%) UTIs in sows.[80] For a long period, the urine within the urinary tract was generally considered sterile.[81,82] This was due to insensitive identification for most bacterial species using traditional microbiological cultures.[83-87] However, a growing list of studies using DNA methods (PCR, NGS, and genome sequencing) detected a wide range of microbiological species in urine samples from diseased and healthy humans and animals.[22,84] Furthermore, it was observed that not only was DNA present, but that the bacterial strains were viable.[86] Therefore, the urinary bladder has an active and functional microbiome and may affect the onset of a UTI. The microbiome role in UTIs was demonstrated by a study in humans that administered *Lactobacillus crispatus* in vaginal suppositories after completion of a full course of antibiotic therapy, which reduced the recurrence of UTIs by 50% in UTI-prone women.[19] This is of particular importance in pigs because UTIs are prevalent in swine herds, and are usually treated with in-feed, broad-spectrum antimicrobials.[88,89] Another alternative for reducing the prevalence of UTIs, and consequently antibiotic use, is the use of urine acidifiers in the diet. The use of acidifiers affects the acid-base balance of the sow diet and is correlated with urinary pH and reduced total bacteria colony-forming units in the urine.[90] Similar results were found in a mouse model with the reduction of uropathogenic *E coli*.[91] Kluge et al[92] showed that supplementation with 1% benzoic acid in the diet reduced the urinary pH of sows by up to one unit when compared to the nonsupplemented group.

Few studies in animal science have analyzed the urinary tract microbiome. One study using dogs as a model identified a urinary tract microbiome in these animals.[22] There seems to be a relationship between vaginal and urinary tract microbiomes in animals and humans.[19,22] Similarly, a positive correlation between UTI and endometritis was observed in pigs.[63,75] Overlap between vaginal and urinary microbiota exists in dogs and humans, but more research is needed to determine if this overlap also exists in sows.[19,22]

However, there are no studies to our knowledge that have evaluated the urinary tract microbiome in sows and its relationship with the use of nutritional management strategies (eg, probiotics and acidifiers). Nevertheless, Xu et al[53] observed that lysozyme administration in sow feed altered vaginal microbiota. Other literature indicates that nutritional changes led to a reduction in urinary pH and a reduction in some potential pathogens in sow urine.[90,92] If gut microbiome can be modulated to prevent dysbiosis, perhaps similar strategies can be used to prevent or even treat UTIs and consequently reduce the use of antibiotics. However, further investigation is necessary to understand the microbiome role in the sow bladder during cystitis and to develop new technologies and strategies to modulate the microbiome, minimizing dysbiosis and diseases.

Colostrum and milk microbiomes

Besides their nutritional value, colostrum and milk are essential to stimulate immune system development of piglets.[32,93-95] Postpartum dysgalactia syndrome is commonly associated with infectious pathogens and is classified as having a multifactorial etiology. Postpartum dysgalactia syndrome compromises milk production and is triggered by associations between risk factors such as management, feeding, and hygiene.[77-79]

It was observed that a lack of sufficient milk production resulted in an increase in piglet preweaning mortality, especially during the first week of age where mortality can be up to 38.6%.[79,96] The infection of mammary glands may lead to their lack of function and impairment of pregnancy rate.[79] Mastitis has a complex treatment and, consequently, it was observed that a high percentage (23%-33%) of antimicrobials used were classified as highest priority or critically important for human medicine by the World Health Organization.[12,97] Moreover, Jenny et al[97] showed that for antibiotic treatment of sow mastitis, duration was shorter and dosage was lower than recommended in 54% and 19%, respectively, which can influence antibiotic resistance selection.[96] Based on the negative impact of PDS on reproductive performance and antimicrobial resistance, alternative tools are essential to reduce the occurrence of this syndrome.

The origin of colostrum and milk microbiomes is complex and not fully elucidated.[98] The high percentage of anaerobic intestinal microorganisms in milk samples indicates that part of the milk bacterial community originates from the maternal gastrointestinal tract through the bacterial entero-mammary pathway[99] or ascending colonization of the udder via the teat canal (galactogenic route).[77,78,100] Other studies indicate that the skin may also be a source for the colostrum and milk microbiome.[101,102] Bacteriological

analysis of colostrum and samples from mammary gland skin from healthy sows showed that all skin samples were bacteriologically positive with Staphylococcaceae as the most frequently isolated (96.9%) followed by Streptococcaceae (63.5%). In addition, 66.7% of all skin samples had species from the Enterobacteriaceae family, with *E coli* the dominant species. Similarly, 79.2% of colostrum samples were bacteriologically positive with Staphylococcaceae as the most frequently isolated (54.1%) followed by Streptococcaceae (30.3%) and Enterobacteriaceae (3.9%). Again, *E coli* was the dominant species among the Enterobacteriaceae family.[102]

Despite not fully understanding the makeup of the mammary gland microbiome, it was observed that sow milk contained Enterobacteriaceae[102] and anaerobic gut-associated genera such as *Bacteroides*, *Blautia*, *Ruminococcus*, and *Bifidobacterium* indicating that the gut has an essential role in the mammary microbiome composition.[95] Gerjets et al[103] studied the virulence genes most frequently detected in milk samples from healthy sows and sows with coliform mastitis. Although sows with coliform mastitis had significantly more specific virulence genes in their samples, healthy sows showed frequencies close to and even higher of some virulence coding genes.[103] Furthermore, no pattern was found in the virulence profile comparing sick and healthy animals.[103] These findings raise the question whether the presence of virulence genes alone is sufficient for bacteria to cause disease. There is no doubt that virulence genes are determinant for bacteria to attach, invade, and colonize the host resulting in illness.[104] However, it also indicates that there is a complex interaction among pathogenic and opportunistic organisms, the environment, and animal genetics. The disruption of one of these factors by stressful handling, mixing of animals from different origins, or the entry of a new infectious pathogen in the naive herd can affect the microbiome allowing the multiplication of pathogenic bacteria causing dysbiosis and disease.

Chen et al[95] analyzed the bacterial 16S rRNA gene sequences from sow colostrum and milk, and the predominant phyla were Firmicutes and Proteobacteria with a counter-balanced relationship between them. The relative abundance of these two phyla significantly fluctuated throughout lactation, while total proportions between them remained at a certain level (75.9%-80.9%).[95] The predominant genera observed during a microbiome assay was different between sow colostrum and milk. The most predominant genus in the colostrum was *Streptococcus*, while transitional and mature milk samples were dominated by unclassified Ruminococcaceae, *Bifidobacterium*, *Staphylococcus*, and *Acinetobacter*, which are lactose-utilizing genera.[95] The six most predominant genera in sows' milk were Ruminococcaceae, *Streptococcus*, unclassified Clostridiales, *Lactobacillus, Corynebacterium*, and unclassified Lachnospiraceae.[95] Analysis from bacteriological isolation[102] and 16S rRNA sequences[95] indicates that *Staphylococcus* and *Streptococcus* are generally the predominant genera in sow colostrum and milk. Moreover, it was reported that microbiome changes in the mammary gland can be the cause for some nutritional alterations from colostrum to transitional and mature milk.[95]

It was observed that microbiome in the gut is related to diseases in other organs[41,53] and a probiotic/prebiotic or symbiotic supplementation may reduce the shedding of potential opportunistic organisms.[37,53] The bacterial entero-mammary pathway is being established[99] and this interconnection indicates that gut microbiome modulation may affect colostrum and milk microbiome composition. In this context, lysozyme feed supplementation altered fecal microbiome and decreased some proinflammatory and increased anti-inflammatory cytokines. These inflammatory cytokines may play a role in PDS development.[105] Based on this, the mammary gland microbiome and its interaction with the gastrointestinal microbiome would constitute an alternative strategy to prevent mammary disorders through gut microbiome modulation and consequently reduce the use of antimicrobials to treat mastitis. Another possibility to reduce the occurrence of mastitis is the development of probiotics for topical application to the sow udder to exclude opportunistic organisms from colonizing the mammary gland. Similar strategies using probiotics in the form of biofilm, spray, or intramammary inoculation to prevent mastitis have been developed and have shown promising results *in vitro*[106] and in dairy cows.[107,108] Furthermore, formulations to be applied in sows should also be beneficial to piglet gut health.

Finally, the sow colostrum and milk microbiome can also influence piglet gut development and innate immune response. The maternal milk microbiome is primarily responsible for the colonization of the piglet gut contributing approximately 90% of the bacteria throughout the first 35 days of life.[32] *Lactobacillus reuteri, Lactobacillus mucosae*, and *Akkermansia muciniphila* are present in sow milk and can act as potential probiotic bacteria.[109,110] An increase of these organisms in the milk was observed during the lactation period.[95] Conversely, potentially pathogenic bacteria such as *Staphylococcus epidermidis*, *Helcococcus, Corynebacterium, Actinobacillus,* and *Haemophilus* are also present in sow milk, but these organisms generally decreased during lactation in healthy sows.[95,111,112] The *Helcococcus* genus was negatively correlated with the abundance of the most bacteria genera in sow milk[95] and its increase in the milk may affect sow and piglet health.

Further studies exploring the sow milk microbiome are necessary to determine a microbial core. More research is also needed to evaluate the influence of environmental characteristics and the gut microbiome on the colostrum and milk microbiome and the subsequent impacts on the offspring.

Fecal microbiome and reproduction

The increased number of piglets born with lower birth weights and the greater within-litter weight variation leads to concerns about the ability of the sow to satisfactorily raise the piglets until weaning. In recent years, numerous studies were developed to understand the impact of the sow gut microbiome and the effects of microbiome modulation on offspring performance. Moreover, the gut microbiome has being studied to find possible biomarkers for productivity, and studies related to FMT were conducted to observe the impact of microbiome of different genetic lines on productivity.

The colonization of the piglet gut is initiated during the farrowing process and immediately after birth. This early colonization plays a crucial role in intestinal maturation. The developmental process of the intestinal microbiome is similar for humans and most animals.[113] The earliest colonizers in the gut are facultative anaerobes, which are responsible for the creation of a favorable environment for anaerobe establishment.[114,115]

Chen et al[115] demonstrated that the core microbiome of piglet feces in the first days post partum is determined by surrounding environmental factors such as floor microorganisms and the microbiomes of the sow's vagina, teats, mammary secretions (colostrum and milk), and feces. Also, several studies demonstrated that the process of immune maturation is influenced by the microbiome that colonizes the gut during the early stage of life.[116,117] The piglet gut microbiome is influenced by milk oligosaccharides (MOS). The MOS decrease intestinal pH and increase cecal and colonic butyrate in the piglet gut and have prebiotic activity, anti-adhesion effects, and anti-inflammatory properties. These characteristics stimulate the growth of beneficial microbes and inhibit possible pathogens.[118,119] It was observed that sows fed with chitooligosaccharide supplement had altered MOS with increasing trisaccharide and tetrasaccharide, but the impact on the piglet gut microbiome was not evaluated.[119] Although a plethora of preweaning and postweaning factors (eg, tail docking, teeth clipping, antibiotic treatment, weaning-associated stressors, and diet composition) may affect the gut microbiome of piglets, a maternal influence on the piglet microbiome was observed for up to 63 days of age.[120]

Dysbiosis in the intestinal microbiome may increase gut permeability and plasma endotoxin concentrations leading to sow metabolic disorders and exacerbated inflammatory status during early lactation.[121] Wang et al[27] found that differences in the intestinal microbiome of sows resulted from oxidative stress during the peripartum period. The authors observed that the relative abundance of *Bacteroides* was correlated to a reduced dam oxidative stress status and higher litter weight on day 21 of lactation. In contrast, *Phascolarctobacterium* and *Streptococcus* were associated with increased oxidative stress and lower litter weight at 21 days post partum.[27]

In highly productive sows, the gut microbiome at 3 days before farrowing was mainly enriched in genera belonging to the Prevotellaceae and Ruminococcaceae families and a relative abundance of gram-negative bacteria in comparison to sows classified with low productivity.[4] Sows classified as high performing during gestation[15,122] and lactation[27] had lower microbiome diversity. Uryu et al[15] also identified that sows with high reproductive performance had an increase in the relative abundances of 43 bacterial genera, markedly the short-chain fatty acid (SCFA)-producing bacteria.

One important factor to evaluate during gut microbiome manipulation is SCFA production. The SCFAs play a role in sow metabolism, immune regulation, and gut homeostasis[31,122-124] and act as precursor of colostrum and milk fat.[125] Moreover, the SCFA-producing bacteria were negatively correlated with porcine epidemic diarrhea virus infection[23] and heat stress.[30] Brutsaert[126] indicates that feeding the sow with a nutritional additive (phenolic compound, slow release C12, target release butyrate, medium-chain fatty acids, and organic acids) has the potential to stabilize the sow gut microbiome during parturition, increase feed intake, and increase the proportion of females that produce heavier piglets at weaning.

The fermentation of dietary fiber, notably soluble fiber, by the hindgut microbiome leads to high production of SCFA[124] and improves piglet development,[127] reduces pathogenic bacteria in the gut,[123,124] reduces digesta transit time, and may prevent colonization by opportunistic organisms and lipopolysaccharide absorption.[128] According to Jiang et al,[43] sows that received a diet with 7.5% crude fiber throughout the reproductive cycle, as compared to sows that received 2.5%, had an increased litter size (3.57 piglets/litter), increased proportion of genera considered beneficial to the intestinal microbiome (*Ruminococcus*, *Butyrivibrio*, *Lactobacillus*, and *Fibrobacter*), and decreased potentially pathogenic genera such as *Clostridium*, *Streptococcus*, *Bacteroides*, and *Escherichia-Shigella*. When the level of dietary fiber was the same, a higher soluble fiber vs insoluble fiber inclusion improved enzymes with antioxidant capacity and decreased proinflammatory factors in the sows and their offspring.[129] The authors also reported that soluble fiber in sow diets increased the proportion of *Romboutsia*, *Sediminibacterium*, *Bifidobacterium*, unidentified Lachnospiraceae, unidentified Ruminococcaceae, *Subdoligranulum*, *Bacillus*, *Blautia*, *Bacteroides*, and *Parabacteroides* and reduced the proportion of *Acinetobacter*, *Vagococcus*, and *Streptococcus* in sow feces and piglet colons.[129] The microbial organisms reduced in the piglet colon were already characterized as opportunistic organisms.[130-132] Similarly, Cheng et al[133] observed that increasing soluble fiber to 2% in the sow gestation diet resulted in piglets with greater growth rate and lower diarrhea rate during the lactation period. Furthermore, the inclusion of dietary fiber in sow diets may contribute to maintenance of proper satiety throughout gestation,[134] reduced constipation,[128] decreased farrowing duration,[127] and reduced stillbirth rate.[125]

Supplementing the diet with functional foods capable of altering the intestinal microbiome has also been an area of research in recent years. Hasan et al[29] showed that the supplementation of yeast hydrolysate in sow diets changed the composition of the fecal microbiome of pregnant sows at the phylum level, reduced farrowing duration, and increased colostrum production, which resulted in a 13% increase in colostrum consumption by piglets. In addition, a lower relative abundance of the phylum Proteobacteria was observed in the supplemented group, which can be considered beneficial since the increased prevalence of this phylum is a marker of dysbiosis associated with intestinal diseases and inflammation.

It is well established that nutrition during the rearing period may affect the performance of future gilts[135] but there is a lack of information regarding the gut microbiome role in this aspect. Emerging evidence in rats suggests that the gut microbiome may affect reproductive function since estrogens interact with the commensal microbiome through the estrogen-gut microbiome axis.[136,137] Wang et al[138] observed that the gut of gilts showing failure to enter estrus before 210 days of age was enriched with Ruminococcacea, Lachnospiraceae, *Ruminococcus, Coprococcus*, and *Oscillospira*. In contrast, gilts showing a normal heat cycle had higher abundance of *Prevotella, Treponema, Faecalibacterium, Oribacterium, Succinivibrio*, and *Anaerovibrio.* In the same study, the authors found that the abundance of both *Sphaerochaeta* and *Treponema* was associated with specific periods of the estrus cycle in which estrogen is high (estrus and proestrous).

Some studies showed that most of the afore mentioned genera may be increased in the gut microbiome of sows and gilts by including fiber in the diet.[123,133] The high inclusion of fiber, predominantly soluble (50% beet pulp), between the 1st and 19th day of the 3rd post-puberty estrous cycle resulted in improved oocyte quality and embryo development *in vitro* and *in vivo*.[139,140] Also, the inclusion of 350 g/kg of lupine

(rich in insoluble fiber and a moderate amount of soluble fiber) in the diet of prepubertal gilts improved oocyte quality and embryonic survival at 28 days of age. Moreover, a recent study showed that highly prolific Meishan sows have increased fecal microbiome diversity and levels of fecal steroid hormones (estradiol and progesterone) than less prolific sows, which may contribute to the improvement of sow reproductive performance.[29] Xu et al[141] observed that the gut of sows with a short wean-to-estrus interval had lower *Prevotella* and *Bacteroides* at the genus level, whereas Firmicutes and Lentisphaerae are greater at the phylum level.

The uterus of Meishan gilts secrete more endometrial proteins than the uterus of white crossbred gilts and that the secretion of endometrial proteins is positively correlated with endometrial gland development before 60 days of age. Xu et al[28] designed a study to evaluate the role of the gut microbiome on endometrial gland development through an FMT from Meishan to Landrace × Yorkshire gilts from 90 days of age until puberty. Fecal microbiome transplantation explained 60.49% of the variation in gut microbiome and increased concentrations of SCFAs, endometrial gland area, insulin-like growth factor 1 (IGF-1) concentration in plasma and uterine tissue, and mRNA expression level of estrogen receptor 1 gene in ovary tissue. The authors also observed that Lentisphaerae, *Bifidobacterium*, and *Fibrobacter* were positively correlated with endometrial gland area; Bacteroidetes was negatively correlated with estradiol and IGF-1 concentration; Firmicutes and *Fibrobacter* were positively correlated with estradiol concentration; and Bacteroidetes was positively correlated with progesterone concentration while Fibrobacteres, Firmicutes, *Bifidobacterium*, and *Fibrobacter* were negatively correlated.

Conclusion and future approaches

The microbiome composition is very sensitive and influenced by diverse environmental, management, and nutritional events. Recent studies indicate that in some cases correlations are insufficient to understand the microbiome complexity. The productivity of offspring may also be affected by sow microbiome modulation. Sow microbiome modulation with probiotics, prebiotics, symbiotics, or other feed additives or nutritional management may constitute a new tool to increase productivity and reduce disease in swine herds and consequently reduce antimicrobial use. Some biomarkers for productivity and disease have been identified, but further investigation using different herds are necessary to determine causality and repeatability of these findings. Future studies should focus on system biology approaches to understand the microbial-microbial and microbial-sow interactions as well as the effect of microbial metabolic production on reproductive outcomes and disease. Randomized blinded clinical trials are necessary to determine if it is possible to increase or decrease target microbial genera previously identified as biomarkers in metagenomics studies and their impact on reproductive outcomes and disease. The decrease in cost per base sequenced over the past few years is encouraging further research in this area. With an increase in metagenomics studies, future research may be aimed at the development of more specific and useful commercial products and to guide future genetic selections.

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Conflict of interest

None reported.

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* Non-refereed references.

PEER REVIEWED

CASE REPORT

An outbreak of splayleg and congenital tremors in piglets farrowed by a newly populated sow herd

Robert Desrosiers, DVM, DABVP; Élisabeth Carrière, DVM; André Broes, DVM, PhD

Summary

A newly populated sow herd suffered an outbreak of splayleg and congenital tremors in the offspring. Some piglets were affected by one or the other condition, others by both. The problem lasted for about 9 months and was associated with significant losses, mainly because of the splayleg component. Most piglets with only congenital tremors were able to survive and their condition improved as they got older. Piglets with congenital tremors had histological lesions consistent with this condition, and pestivirus K (formerly atypical porcine pestivirus) was identified from their nervous tissues.

Keywords: swine, splayleg, congenital tremors, pestivirus K

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Resumen - Un brote de patas abiertas y temblores congénitos en lechones paridos en una piara de cerdas recién poblada

Una piara de cerdas recién poblada sufrió un brote de patas abiertas y temblores congénitos en las crías. Algunos lechones se vieron afectados por una u otra condición, otros por ambas. El problema duró alrededor de 9 meses y estuvo asociado con pérdidas significativas, principalmente por el componente patas abiertas. La mayoría de los lechones con solo temblores congénitos sobrevivieron y su condición mejoró a medida que aumentaron de edad. Los lechones con temblores congénitos tenían lesiones histológicas compatibles con esta condición y se identificó el pestivirus K, anteriormente llamado pestivirus porcino atípico, a partir de sus tejidos nerviosos.

Résumé - Une épidémie de 'splayleg' (porcelets nageurs) et de tremblements congénitaux chez des porcelets mis bas par un troupeau de truies nouvellement peuplé

Un troupeau de truies nouvellement peuplé a souffert d'une épidémie de splayleg et de tremblements congénitaux chez la progéniture. Certains porcelets étaient atteints de l'une ou l'autre affection, et d'autres des deux. Le problème a duré environ 9 mois et a été associé à des pertes importantes, principalement à cause de la composante splayleg. La plupart des porcelets qui n'avaient que des tremblements congénitaux ont pu survivre et leur état s'est amélioré avec l'âge. Les porcelets atteints de tremblements congénitaux présentaient des lésions histologiques compatibles avec cette affection et le pestivirus K, anciennement appelé pestivirus porcin atypique, a été identifié à partir de leurs tissus nerveux.

The recent identification of pestivirus K (PK), previously known as atypical porcine pestivirus, and piglets born with congenital tremors (CT) after pregnant animals were inoculated with the virus have been major steps in our understanding of this disease.[1-4] Nevertheless there is still limited information concerning the transmission, pathogenesis, carriage, and epidemiology of the virus.[5] Splayleg (SL) is another congenital problem for which questions remain, including possible etiologies. This case report describes an outbreak involving both conditions where losses were significant and lasted longer than what is commonly seen in the field.

Animal care and use

The animals in the case herd were adequately housed, and humanely cared for.

Case description

A 1400-sow herd using a 4-week batch farrowing system was populated in 2019, with the first weaning on December 18. In the first batch, 270 litters were farrowed and many piglets were affected with SL, CT, or both. In that batch, 5.81% (187 piglets) of the total live born pigs were reported to have died because of SL. Most piglets affected with CT appeared to survive. As opposed to SL, mortality records did not include CT as a cause so the exact number of pigs that died because of that condition is unknown. Similarly, the number of pigs affected with both conditions was not compiled. The problems persisted in subsequent batches. When the attending veterinarian visited in June 2020, 7 months after the first litters

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presented with these conditions, many litters were still affected. Table 1 summarizes the observations made during that visit. That particular batch had 13.6 liveborn piglets/sow and was weaned on July 1.

There was a total of 259 sows in lactation. A piglet with both conditions was recorded as a CT piglet and an SL piglet. Of the 55 affected litters, 52 were from parity 2 females and 3 were from parity 1 females. In litters with both conditions, the mean number of piglets affected with SL was 2.69 times greater than in litters where only this condition was observed. This increase in dually affected litters was not seen with CT, where the mean number of affected pigs were similar (5.87 and 6.0 piglets/litter, respectively). The mortality associated with SL in that batch was 3.82% (134 of 3511 pigs born live). It decreased further in the next batch (2.32%) and stabilized at about 1% in subsequent batches. Table 2 shows the mortality associated with SL in the first 7 batches following population (December 2019 – June 2020), and in the last 7 batches for which data are available (March 2021 – August 2021).

Two submissions were made to the diagnostic laboratory in January 2020. In the first submission, two 2-week-old piglets with clinical signs of CT were submitted. Histological lesions consistent with CT, including hypomyelination, were observed. A pool of spinal cord samples from both piglets was positive for PK by polymerase chain reaction (PCR) with a cycle threshold value of 28.53. The second laboratory submission included two 4-week-old piglets weaned the week before. One of them showed slight trembling and had histological lesions consistent with CT. A pool of nervous tissue from that piglet also came back positive for PK with a cycle threshold value of 28.41.

Because losses persisted, an attempt was made to inoculate gilts with serum from piglets affected with CT prior to their introduction into the sow herd. Blood was collected from 20 piglets with CT at 2 to 3 days of age, centrifuged, and serum collected and stored at -20°C. Seven of the serum samples were positive for PK by PCR. Serum samples from the 20 piglets were pooled (total of 47 mL). Two 1-mL vials of the pooled sample were sent to Iowa State University Veterinary Diagnostic Laboratory for quantification and came back with cycle threshold values of 34.8 and 33.5. Ninety-seven milliliters of phosphate-buffered saline and 2 mL of ceftiofur (Excenel, Zoetis) were added to the remaining 45 mL of serum for a total volume of 144 mL. Seventy-seven gilts weighing 120 kg were received on July 14. On July 17, 10 gilts were inoculated intramuscularly with a 2 mL dose of the pooled piglet serum. Since there were no adverse events observed, 62 gilts were inoculated on July 20, and the remaining 5 gilts were kept as controls. The gilts were inseminated 5- or 9-weeks post inoculation. Using quantitative PCR (qPCR), it was estimated that each gilt received a dose of approximately 1500 genomic copies of PK.

No clinical signs were noted following inoculation. Paired sera from 10 inoculated gilts and from the 5 control gilts were evaluated for PK titers using an enzyme-linked immunosorbent assay (ELISA) under development at Iowa State University (Figure 1). Four of the five control gilts had virtually no antibodies at the first sampling. Three of the control gilts remained negative and the fourth gilt strongly seroconverted. One control gilt initially had a relatively high titer and remained about the same through the second sampling. Of the 10 inoculated gilts, 3 had almost no antibodies initially but did seroconvert. The 4 inoculated gilts with intermediate titers saw their titers decline by the second sampling, and the 3 gilts with high titers at the first sampling had approximately the same titer levels at the second sampling.

Table 1: Incidence of CT and SL piglets from 259 litters farrowed 7 months (June 2020) after observation of the first cases

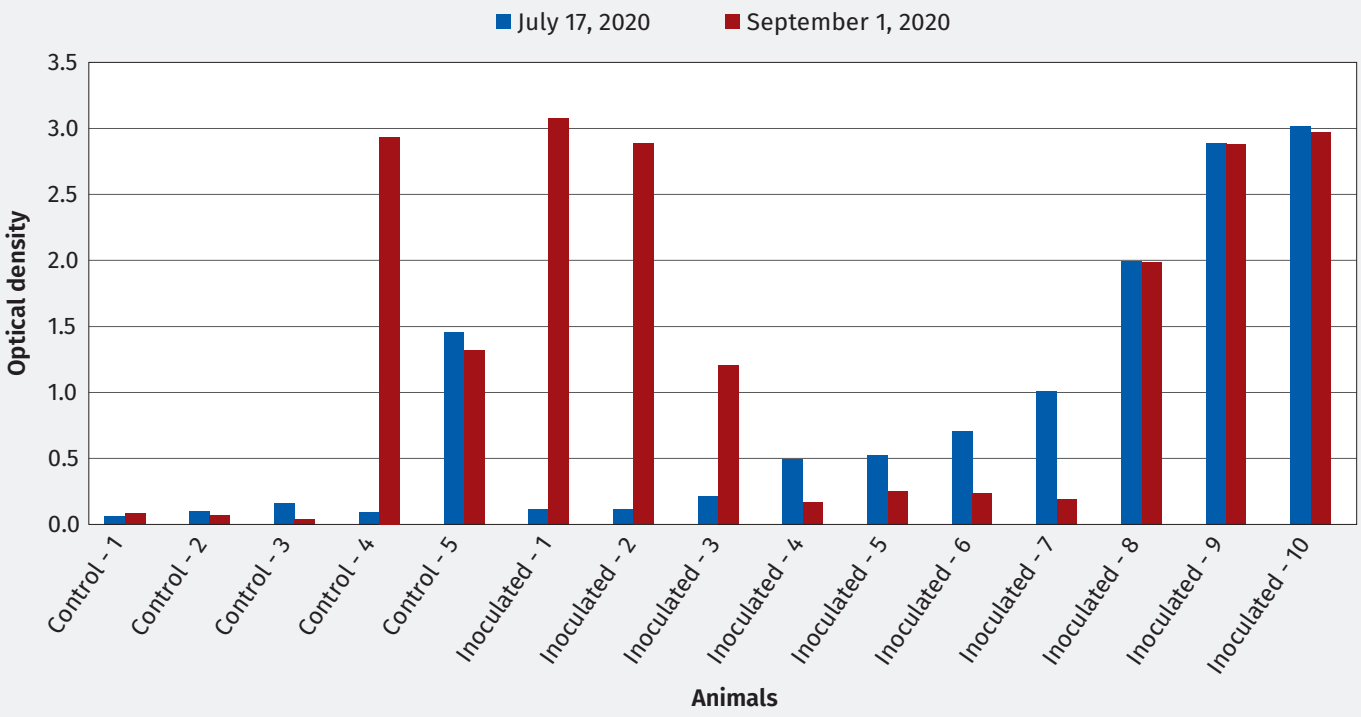
		Piglets with CT		Piglets with SL	
	Litters, No.	Total	Mean	Total	Mean
Unaffected	204	0	0	0	0
CT only	8	48	6.0	NA	NA
SL only	32	NA	NA	51	1.59
Both	15	88	5.87	64	4.27
Total	259	136	NA	115	NA

CT = congenital tremors; SL = splayleg; NA = not applicable.

Table 2: Preweaning mortality associated with splayleg during the first and last 7 batches of weaned pigs

					Splayleg	
Batches	Weaning dates	Total piglets born live	Piglets born live/litter, mean	Total preweaning mortality, %	Mortality, %	% of total mortality
First 7	Dec 2019 - Jun 2020	23,157	12.74	17.87	5.39	30.16
Last 7	Mar 2021 - Aug 2021	24,144	13.99	18.18	1.06	5.83

Figure 1: Paired serological titers of 5 control gilts and 10 gilts inoculated with sera from pestivirus K-positive piglets.



The first batch of inoculated gilts (31) farrowed in late December 2020. At that time, the losses associated with CT and SL were becoming minimal, which made it more difficult to determine if the inoculation strategy had an impact or not. One gilt had 5 piglets with CT, while 5 gilts had a total of 7 piglets with SL.

Discussion

A clear association between CT and PK has been made in previous studies.[3-5] However, the association between SL and this virus is not as clear. Different causes or factors have been proposed to explain the occurrence of SL including slippery floors, large litters, low birth weight, choline or methionine deficiency, mycotoxins, genetics, short gestation lengths, and inducing farrowing too early.[5] Madsen et al[5] did identify CT as a condition to which SL can be associated without specifying if PK could be considered as a causal agent. In this case, as seen in Table 1, more litters (32) had only SL piglets compared to only CT piglets (8). Thus, the incidence of SL was not conditional to the presence of CT in a litter. Nevertheless, litters with both problems had a higher mean number of SL piglets (4.27) than litters with only SL piglets (1.59), so there appeared to be a predisposition to SL in litters with CT piglets.

The role PK played in the occurrence of SL in the case herd cannot be confirmed. But, there is increasing evidence that the virus, while not the sole cause, may be associated with this condition. When inoculating sows with PK on day 45 or 62 of gestation, Arruda et al[3] reported that 75% and 17.5% of the piglets were affected with CT and SL, respectively. In one litter, all piglets (10 of 10) had CT and 4 of them also were splaylegged. In another study where 3 gilts were experimentally infected with the virus on day 32 of gestation, 2 of them produced piglets with CT (11 of 13 and 13 of 15) and SL (3 of 13 and 7 of 15), with some piglets affected with both conditions.[4] Under field conditions, Sutton et al[6] described a case on a high-health research farm in the United States where the prevalence of SL was 33% in pigs with CT, and 0.8% in unaffected pigs. All tested litters with CT (41) had pigs positive for PK by qPCR, while the litters without CT (50) had no PK-positive pigs. Similarly on two Brazilian farms with an abrupt increase of CT, 29.7% (102 of 343) and 44.2% (19 of 43) of the piglets with this condition also had SL. Pestivirus K was identified by PCR in all 13 piglets with CT that were tested and in 1 of 6 unaffected piglets.[7] Schwarz et al[8] reported that a fatal combination of CT and SL was observed in an Austrian herd, but in single piglets. When other herds with CT were investigated, 3 of 5 herds reported concomitant problems with SL. Finally, White[9] stated that it was common for CT pigs to also show SL.

The chronological association between CT and SL problems in field situations, coupled with the experimental reproduction of both CT and SL in gilts inoculated with PK during pregnancy, seems to leave little doubt as to a possible association between PK and SL. This is not to say that PK will necessarily produce SL pigs or that other causes or factors cannot be associated with it. Two of five herds investigated in the Schwarz et al[8] study did not report concomitant SL issues. In 4 Swedish farms in 2017-2018, 13 piglets with SL were tested and all were found to be PK-negative by PCR.[10] In Denmark, no reports of concomitant SL were mentioned in 10 herds with CT problems where all affected piglets tested (55) were found to be PK positive.[11]

What is currently known seems to suggest that there are situations where PK infection may be associated with SL problems, but not necessarily in others. Differences between PK strains have been identified.[12,13] It could be that some strains may be more likely to be associated with SL than others. In the case herd described here, the mean number of SL piglets in litters that also had CT

piglets was 2.69 times higher (4.27 vs 1.59) than in litters with only SL piglets. This suggests that litters in that herd with CT were more likely to also have SL problems.

The reason why the case farm broke with these 2 conditions and why it lasted so long is unknown. The current understanding is that for CT type A-II, nonimmune females that come in contact with the virus at a certain time in gestation may produce affected piglets. After infection, long-term immunity seems to develop as it appears rare for the same female to produce more than one affected litter.[4] In herds that have been established for a while, the condition can affect litters of different parities, but is more often seen in gilts.[4,7,9] New herds are particularly at risk in terms of losses that can be associated with CT.[9]

Seven months after the first clinical signs were observed in the case herd, 52 of the 55 litters affected with CT, SL, or both were from parity 2 sows. It is hypothesized that these females had not come in contact with the virus before their second gestation, and that they had not produced an affected litter during their first parity. Changes in farm personnel resulted in difficulties to compile accurate data. While uncommon, long-term problems with CT have been reported where the condition was present for several months and sometimes more than a year.[4,6]

It is believed that most herds are likely infected with PK. In a collection of sera from multiple US states, 94% of samples were found to be seropositive for PK using an ELISA. Further sampling from 3 farms revealed that 2 farms had 96% and 100% seropositive sera, while the third farm had none.[1] Consequently, introduction of PK-naive gilts into infected herds is a possibility that needs to be considered. Similarly, the virus has been detected by PCR in semen coming from different commercial US boar studs, and the role this could play in the epidemiology of the infection needs to be assessed.[14]

The case farm was populated from 5 different gilt developer units filled with gilts from 6 sow herds, but the source of the gilts could not be identified once introduced into the sow herd. Thus, it is plausible that gilts coming from one or more of these gilt developers had not come in contact with PK before their introduction into the sow herd being populated. This is supported by the serological data (Figure 1). Of the 15 tested gilts, 7 had few or no antibodies at the first sampling. All 3 inoculated gilts with initially few or no antibodies showed a strong increase at the second sample, suggesting that these animals had not been exposed to the virus before being introduced into the newly populated herd. Conversely, the 3 inoculated gilts with high titers at the first sampling basically maintained the same titer levels after inoculation. Ideally, efforts should be made so that gilts come from only one source, but in cases where it is not possible, mixing the gilts from different sources early before their introduction into the sow herd would seem to increase their chances of coming in contact with the virus and becoming immune before their first gestation. White[9] suggested that placing gilts in contact with 8- to 12-week-old pigs for 4 weeks and ending at least 2 weeks before service appeared to provide satisfactory exposure.

Following initial cases of CT, Sutton et al[6] orally exposed 91 gilts to an inoculate obtained from fetal fluids and membranes collected from sows that had produced CT-affected litters. This was done 54 days prior to insemination with the goal to immunize the gilts before they became pregnant. Yet 45.0% of the litters produced and 30.8% of all piglets were affected by CT. Thirty- three percent of the piglets affected with CT had SL, compared to 0.8% in unaffected piglets. The inoculation strategy used in the herd described in this case report did not seem to have a significant impact on the condition and losses. The clinical situation had already vastly improved when inoculated gilts farrowed, which made interpretation difficult. Still, 1 inoculated gilt produced 5 piglets with CT, and there was no difference between the number of SL piglets from the gilts administered the presumably infected serum and in the two batches that preceded the inoculated batch. It is also perplexing to see that 52 of the 55 females with affected litters in July 2020 (7 months after the first cases were observed) were of parity 2 and had been in the herd for about a year. This should have been enough time for gilts to become infected and immune before producing affected litters. More work is needed to identify procedures that can be applied to effectively prevent these conditions, particularly for new herds that need to use more than one gilt source. Given the differences between PK strains, one area that needs clarification and that can have an impact on control measures is the level of cross protection that is obtained against different strains following infection with a single strain.[12,13,15]

Serologic assays have been developed, but their usage is recent. Once more is known about what is to be expected under field conditions from these assays, they could become useful tools to determine if interventions are needed or not. In the case described here, the serological results obtained following the inoculation protocol are difficult to interpret and do not allow for conclusions to be made on its efficacy. The 3 inoculated gilts with very low initial titers did strongly seroconvert, but so did 1 control gilt that did not receive the serum from infected piglets. Whether the seroconversion was associated with virus shed by the inoculated gilts, or by contact with already infected animals is unknown. It is possible that the serum used to inoculate the gilts was not infectious and did not influence the results obtained. A few inoculated animals had lower titers at their second sampling, a situation that can be observed in animals with declining maternal immunity. Limited information is known about the duration of maternal immunity to PK. In 2 studies where this was investigated, it varied between 3 and 8 weeks which would seem to eliminate the possibility for declining maternal immunity to be involved in the current case given that the gilts were approximately 26 weeks of age at the time of inoculation.[8,16] The declining titers could also be a reflection of animals that had been exposed to the organism in the past and were towards the end of the detectable antibody duration. In one study that evaluated the duration of antibodies in a CT-affected herd using an ELISA, healthy piglets from a healthy litter were positive after birth and became negative at 3 to 6 weeks of age when maternal immunity waned. Following infection, the piglets were positive again at 70 days of age and were still strongly positive at 160 days of age. In that case, duration of actively acquired antibodies lasted at least 3 months.[16] The assay used in the current case report was under development at the time and had not yet been fully validated. Thus, more work is needed before the strengths and limitations of the assay are determined.

A few weaknesses of this case report are readily acknowledged. First, the number of pigs with both conditions and the associated mortality should have been

compiled. The litter mortality records used by the personnel included SL as a cause, but not CT. Second, necropsy of a few SL pigs could have helped to clarify the role of PK in that condition. The simultaneous appearance of both SL and CT suggested a common cause, and it was initially felt that the problems would be temporary and not persist as long as they did. Thus, there was no plan at the time to report the findings. The duration of the conditions and their significance, particularly that associated with SL, later suggested that reporting what was observed could be of value.

Finally, losses associated with CT can be significant. In a small new herd of 400 sows, it was estimated that 1000 piglets were lost.[9] Schwarz et al[8] reported that in 5 Austrian herds affected with CT, the losses went from almost none to the equivalent of 4.9 to 7.3 pigs/sow/year. In the case described here, the number of piglets that died because of CT could not be quantified but was estimated to be low. However, the losses associated with SL alone were estimated at more than 1000 pigs.

Implications

Under the conditions of this case report:

- Pestivirus K may be associated with both CT and SL.
- Losses associated with PK can be significant and last for several months.
- More work is needed to identify preventive methods, particularly for new herds.

Acknowledgments

Conflicts of interest

None reported

Disclaimer

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Conversion tables

Weights and measures conversions

Common (US)	Metric	To convert	Multiply by
1 oz	28.35 g	oz to g	28.35
1 lb (16 oz)	0.45 kg	lb to kg	0.45
2.2 lb	1 kg	kg to lb	2.2
1 in	2.54 cm	in to cm	2.54
0.39 in	1 cm	cm to in	0.39
1 ft (12 in)	0.3 m	ft to m	0.3
3.28 ft	1 m	m to ft	3.28
1 mi	1.6 km	mi to km	1.6
0.62 mi	1 km	km to mi	0.62
1 in^2	6.45 cm^2	in^2 to cm^2	6.45
0.16 in^2	1 cm^2	cm^2 to in^2	0.16
1 ft^2	0.09 m^2	ft^2 to m^2	0.09
10.76 ft^2	1 m^2	m^2 to ft^2	10.8
1 ft^3	0.03 m^3	ft^3 to m^3	0.03
35.3 ft^3	1 m^3	m^3 to ft^3	35.3
1 gal (128 fl oz)	3.8 L	gal to L	3.8
0.26 gal	1 L	L to gal	0.26
1 qt (32 fl oz)	0.95 L	qt to L	0.95
1.06 qt	1 L	L to qt	1.06

Temperature equivalents (approx)

°F	°C
32	0
50	10.0
60	15.5
61	16.1
65	18.3
70	21.1
75	23.8
80	26.6
82	27.7
85	29.4
90	32.2
102	38.8
103	39.4
104	40.0
105	40.5
106	41.1
212	100.0

°F = (°C × 9/5) + 32
°C = (°F - 32) × 5/9

Conversion calculator available at: amamanualofstyle.com/page/si-conversion-calculator

Conversion chart, kg to lb (approx)

Pig size	Lb	Kg
Birth	3.3-4.4	1.5-2.0
Weaning	7.7	3.5
	11	5
	22	10
Nursery	33	15
	44	20
	55	25
	66	30
Grower	99	45
	110	50
	132	60
Finisher	198	90
	220	100
	231	105
	242	110
	253	115
Sow	300	136
	661	300
Boar	794	360
	800	363

1 tonne = 1000 kg
1 ppm = 0.0001% = 1 mg/kg = 1 g/tonne
1 ppm = 1 mg/L

Producers and students receive hands-on FAD response practice

National Pork Board staff recently facilitated a foreign animal disease (FAD) exercise to practice and troubleshoot a simulated response to a mock disease outbreak. Over 40 people from academia, production, the US Department of Agriculture, the veterinarian community, and Iowa Pork Producers Association teamed up during the full-day event hosted by the Swine Medicine Education Center staff at Iowa State University.

"The value of this exercise is continuous practice since regulation, technology, and stakeholder awareness evolve. The more we prepare, the quicker we can respond to an actual incident," said Dr Tyler Bauman, herd veterinarian for The Maschhoffs, LLC.

Participants practiced every procedure in the coordinated response plan based on location and the outbreak's status to identify, understand, and address their knowledge gaps.

"The buildup of the mock incident and protocols instructed were cohesive since each stakeholder at the drill shared a wealth of knowledge in their role, rather than filling an unknown position. Plus, there were real-time insights from the Highly Pathogenic Avian Influenza outbreak that state veterinarians could share," Bauman said.

To help your producer clients be better prepared for an FAD outbreak, urge them to participate in the Secure Pork Supply plan and get an AgView account. More information is available at **securepork.org** and **porkcheckoff.org/agview**, respectively.

Cybersecurity tips for your business

As part of the National Pork Board's recent Pork Management Conference in Nashville, Tennessee, attendees heard from several security experts who are urging everyone involved in agri-business to make cybersecurity a top priority for their businesses. This includes pork producers and swine veterinarians. They advised that attendees should set up multifactor authentication on your accounts; update your software and turn on automatic updates; and think before you click since more than 90% of successful cyber attacks start with a phishing email. Finally, they advised assigning strong passwords and using a password manager.

The Federal Bureau of Investigation has warned the food and agriculture sector that ransomware actors may be more active now than ever. Agricultural cooperatives are of particular concern during planting and harvest seasons, which can disrupt operations, cause economic loss, and negatively impact the food supply chain.

As senior vice president of management liability and client experience for Marsh & McLennan Agency's Upper Midwest Region, Dan Hanson says cybersecurity is about 80% to 90% of what they do for client risk mitigation today. "Cyber attacks are a crime of opportunity," Hanson says. "They are looking for weakness wherever they can find it. That can make for a systemic impact such as the food chain. It is important to take steps today to protect yourself because it is not a matter of if they will strike, but when."

For more information on these plans, visit **fcc.gov/sites/default/files/cyberplanner.pdf**.

Producers can now be trained to become Certified Swine Sample Collectors

Producers can now be trained and certified to properly collect samples for diagnostic and surveillance purposes through a classroom and hands-on curriculum developed with funding from the US Department of Agriculture's National Animal Disease Preparedness and Response Program. Spanish translations of the materials are also available thanks to funding from the Pork Checkoff.

Category II accredited veterinarians provide training as part of the Certified Swine Sample Collector Training Program. Training for proper sample collection is an important step in preparing for a potential foreign animal disease outbreak. Recertification is required annually. For more information, visit **secureporksupply.org/cssc** or contact Dr Pam Zaabel at **pzaabel@pork.org**.

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Highlights: Board of Directors 2022 spring meeting

The AASV Board of Directors met on April 21 in Perry, Iowa to conduct official business.

The board welcomed newly elected District Directors Stephen Patterson (District 3) and Maryn Ptaschinski (District 7), who began their terms at the conclusion of the meeting. The board thanked outgoing District Directors Greg Cline and Megan Potter for their service. The board also welcomed newly elected Vice-President Angela Baysinger and the new Alternate Student Delegate Hunter Everett.

The board took several actions during the business section of their meeting.

- **Nutrition Committee mission change:** The board approved a request to revise the committee's mission. The new mission is online at **aasv.org/members/only/committee/NutritionCommittee.php**.

- **Student Recruitment Committee name change:** The board approved a request to change the committee's name to Student Engagement Committee.

- **Position statement: Raising Pigs without Antibiotics:** The board approved changes to the position as recommended by the Pig Welfare and Pharmaceutical Issues Committees. See **aasv.org/aasv/positions**.

- **US Swine Health Improvement Program (SHIP):** The board passed a motion to support the US Animal Health Association's resolution urging USDA APHIS to expand support for the US SHIP.

- **Diversity, Equity, and Inclusion Committee request:** The board voted to support the attendance of 2 AASV members at the 2023 Minorities in Agriculture, Natural Resources, and Related Sciences (MANRRS) Conference, with a cap of $5000.

- **Operation Main Street (OMS):** The board approved a request to provide up to $10,000 for costs associated with OMS presentations at veterinary schools in 2022-2023.

- **Swine Medicine Talks:** The board approved the Student Recruitment Committee's request for $2500 to support the 2022-2023 series of Swine Medicine Talks webinar broadcasts to US veterinary schools.

- **Boar Stud Committee requests:** The board approved a request to allow public access to the AASV document, *Health, Hygiene and Sanitation Guidelines for Boar Studs Providing Semen to the Domestic Market*, available at **aasv.org/documents/boarstudguidelines.pdf**. The board also authorized the Boar Stud Committee to work with the US Department of Agriculture and state animal health officials to develop standardized requirements for shipping semen across state lines.

- **Depopulation nomenclature:** The board approved a recommendation from the Pig Welfare Committee to revise the nomenclature associated with depopulation as follows:

  Emergency depopulation. Defined as "the rapid and efficient destruction of a complete population of animals in response to urgent circumstances" (AVMA, 2019). Urgent circumstances include but are not limited to disease control, natural disasters, and supply chain disruptions.

  Herd repopulation. This event encompasses the management and eradication of unfavorable conditions on farm by removing and replacing the whole herd to improve health, productivity, and welfare. These events are not urgent in nature and may occur over a significant period of time (ie, porcine reproductive and respiratory syndrome virus [PRRSV] and *Mycoplasma hyopneumoniae* eradication and genetic rollovers). Unlike emergency depopulation, animals are removed from the herd primarily through currently available market channels.

- **PRRSV survey:** The board approved the PRRS Task Force's request to conduct a 5-question survey of AASV members on PRRSV nomenclature.

- **Nutrition survey:** The board approved a request from the Nutrition Committee to survey members for topics of interest to be presented in a 2024 preconference seminar at the AASV Annual Meeting.

- **AASV bylaws:** After considering proposed changes to the AASV bylaws recommended by legal counsel, the board made additional revisions and approved the revised bylaws. The bylaws are online at **aasv.org/aasv/bylaws**.

- **Amicus brief:** The board passed a motion that AASV prepare an amicus brief to supply facts regarding the California Proposition 12 lawsuit scheduled to be heard by the US Supreme Court in 2022.

Read all AASV position statements at **aasv.org/aasv/positions**. View each committee's plan of work at **aasv.org/aasv/committee**. Members of AASV can read complete Board and Executive Committee meeting minutes at **aasv.org/aasv/board**.

Interested in joining a committee? Contact the AASV office by email, **aasv@aasv.org**, or phone, 515-465-5255.

AASV news continued on page 261

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AASV news continued from page 259

Call for abstracts - Research Topics

Plans are underway for the 54th Annual Meeting of the American Association of Swine Veterinarians to take place March 4-7, 2023 in Aurora (Denver), Colorado.

As part of the meeting, there will be a session highlighting research projects related to swine health and production. Abstracts are now being accepted to be considered for presentation during the Research Topics session, which will be held Sunday, March 5.

Those interested in making a 15-minute oral presentation must submit 2 copies of a 1-page abstract on applied research related to swine health and production issues (virology, bacteriology, parasitology, environment, food safety, odor, welfare, etc) to **aasv@aasv.org** by **August 15, 2022**. One copy will be used for review purposes and should contain the abstract title but must omit the authors' names and affiliations. Provide the presenting author's name, mailing address, phone number, and email address within the email message accompanying each submission.

Abstracts not selected for oral presentation will be considered for poster presentation. All submitting authors will be notified of the selection results in September. Authors of abstracts selected for oral or poster presentation must provide a paper, formatted for publication in the conference proceedings, by November 15, 2022.

PLEASE NOTE: Participation in the Research Topics oral and poster session is at the presenter's expense. No speaking stipend or travel expense reimbursement is paid by the AASV. **The presenting author is required to register for and attend the meeting in person to make the presentation.** Recorded/virtual presentations will not be accepted unless the meeting converts to an entirely virtual event.

It is not necessary to be an AASV member to submit an abstract for consideration or participate if selected. Nonmember participants may register for the meeting at the AASV regular member rate. Qualifying full-time graduate students must join AASV if they wish to register at the graduate student member rate.

Call for submissions - Industrial Partners

The American Association of Swine Veterinarians is making plans for the 2023 AASV Annual Meeting, to be held March 4-7, 2023 in Aurora (Denver), Colorado.

The AASV invites submissions for the Industrial Partners oral and poster sessions at the 54th AASV Annual Meeting. This is an opportunity for commercial companies to make brief presentations of a technical, educational nature to members of the AASV.

The oral sessions consist of a series of 15-minute presentations scheduled from 1:00 to 5:00 PM on Sunday, March 5. A poster session takes place the same day. Poster authors will be required to be stationed with their poster from noon until 1:00 PM, and the posters will remain on display throughout the afternoon and the following day for viewing.

SUBMISSION PREREQUISITE: All companies submitting topics for presentation during the Industrial Partners sessions must register to participate in the AASV Technical Tables Exhibit before September 30.

SUBMISSION LIMIT: Restricted program space necessitates a limit on the number of presentations per company. Companies that are a member of the *Journal of Swine Health and Production* Industry Support Council **and** sponsor the AASV e-Letter may submit 3 topics for oral presentation. Companies that are **either** a member of the JSHAP Industry Support Council **or** sponsor the AASV e-Letter may submit up to 2 topics. All other companies may submit 1 topic for oral presentation. In addition, every company may submit 1 topic for poster presentation, but the topic must not duplicate the oral presentation. All topics must represent information not previously presented at the AASV Annual Meeting or published in the meeting proceedings.

SUBMISSION REQUIREMENTS:
To participate, send the following information to **aasv@aasv.org** by **September 30, 2022**:
1) Company name
2) Presentation title
3) Brief description of the presentation content
4) Presenter name (one only) and contact details (mailing address, telephone number, and email address)
5) Whether the submission is intended for oral or poster presentation

Receipt of submissions will be confirmed by email. Presenters will be notified of their acceptance by October 15 and must submit a paper by November 15 for publication in the meeting proceedings. Failure to submit the paper in a timely manner will jeopardize the company's future participation in these sessions.

The presenting author is required to register for and attend the meeting in person to make the presentation. Recorded/virtual presentations will not be accepted unless the meeting converts to an entirely virtual event.

Presenters may register for the meeting either as a Tech Table representative, or as an individual registrant (nonmember oral and poster presenters are eligible to register at the AASV regular member rate). The AASV does not provide a speaking stipend or travel reimbursement to Industrial Partners presenters.

Call for papers – AASV 2023 Student Seminar Veterinary Student Scholarships

The American Association of Swine Veterinarians announces an opportunity for veterinary students to make a scientific presentation at the AASV Annual Meeting in Aurora (Denver), Colorado on Sunday, March 5, 2023. Interested students are invited to submit a one-page abstract of a research paper, clinical case study, or literature review for consideration. The submitting student must be a current (2022-2023) student member of the AASV at the time of submission and must not have graduated from veterinary school prior to March 5, 2023. Submissions are limited to 1 abstract per student.

Abstracts and supporting information must be submitted online at **cmt3.research.microsoft.com/AASV2023**. Submissions must be completed before **11:59 PM Central Daylight Time** on **Wednesday, September 14, 2022** (firm deadline). Late submissions will not be considered. Students will receive an email confirmation of their submission. If they do not receive the confirmation email, they must contact Dr Andrew Bowman (**bowman.214@osu.edu**) by Friday, September 16, 2022 with supporting evidence that the submission was made in time; otherwise the abstract will not be considered for judging.

The abstracts will be reviewed by an unbiased, professional panel consisting of private practitioners, academicians, and industry veterinarians. Fifteen abstracts will be selected for oral presentation in the Student Seminar at the AASV Annual Meeting. Students will be notified of the review results by October 15, 2022, and those selected to participate will be expected to provide the complete paper or abstract, reformatted for publication in the conference proceedings, by November 15, 2022.

Student Seminar

The **Zoetis Foundation** has provided a grant for a total of $20,000 for awards and the top student presenter scholarship. The grant will go towards a $750 award for the student presenter of each paper selected for oral presentation when they present at the meeting. These students also compete for one of several scholarships awarded through the AASV Foundation. The oral presentations will be judged to determine the amount of the scholarship awarded. As part of the Zoetis Foundation grant, the AASV Foundation will award a $5000 scholarship for the student whose paper, oral presentation, and supporting information are judged best overall.

Elanco Animal Health provides $20,000 in additional funding, enabling the AASV Foundation to award scholarships of $2500 each for 2nd through 5th place, $1500 each for 6th through 10th place, and $500 each for 11th through 15th place.

Student Poster Session

Abstracts that are not selected for oral presentation in the Student Seminar will be considered for presentation in a poster session at the Annual Meeting. The **Zoetis Foundation** grant, combined with direct support from AASV, will provide each student poster presenter at the meeting with a $250 award. Students selected to make a poster presentation will be expected to supply a brief paper, formatted for publication in the conference proceedings, by November 15. The guidelines for preparing posters for the display are available at **aasv.org/annmtg/2023/posters.php**.

Veterinary Student Poster Competition

The presenters of the top fifteen poster abstracts compete for scholarship awards ranging from $200 to $500 in the Veterinary Student Poster Competition, sponsored by **United Animal Health**. See **aasv.org/annmtg/2023/postercomp** for poster judging details.

In all cases, the student presenter is required to attend the meeting in person to make the presentation. Recorded/virtual presentations will not be accepted unless the meeting converts to an entirely virtual event.

Complete information for preparing and submitting abstracts is available at **aasv.org/annmtg/2023/studentseminar**. The rules for submission should be followed carefully. For more information, contact the AASV office by phone, 515-465-5255, or email, **aasv@aasv.org**.

Early Career Committee podcasts

The AASV Early Career Committee has been developing a new podcast series highlighting topics for early career swine veterinarians. Podcasts are available to download as .mp3 audio files from the AASV Podcast Library at **aasv.org/podcast/**.

AASV Foundation presents Heritage Award

Dr Max Rodibaugh received the American Association of Swine Veterinarians Foundation's Heritage Award during his retirement ceremony from AMVC Swine Health Services in Indiana on April 19, 2022.

Dr Rodibaugh received his DVM from Purdue University in 1977. He has been recognized as a distinguished alumnus of both the Purdue School of Agriculture and Purdue College of Veterinary Medicine.

With a lifetime of service to the AASV, Dr Rodibaugh has served on multiple committees, the Board of Directors, and as the association's president in 1995. In 2001, he was recognized as the AASV Swine Practitioner of the Year. He presented the Howard Dunne Memorial Lecture at the 2001 AASV Annual Meeting. His personal and inspirational story, "Life upside down: Is it possible to be prepared for a personal crisis?," received the top prize during the practice tip session at the 2021 AASV Annual Meeting. His service to the swine industry has truly been selfless.

Dr Rodibaugh is a member of the American Veterinary Medical Association, Indiana Veterinary Medical Association, and Indiana Pork Producers. He is also an adjunct faculty member at Purdue University College of Veterinary Medicine.

Many colleagues from across the country nominated Dr Rodibaugh for the award, citing his dedication to the swine industry, care for pigs and people, and genuine personality. While Dr Rodibaugh certainly loves working with pigs, it is the people who raise them – his clients – who matter the most to him.

Max and his wife, Carol, have three children and seven grandchildren. He enjoys volunteering through his church, county chamber of commerce, and United Way.

Dr Rodibaugh becomes only the sixth recipient of the Heritage Award, which recognizes individuals who have lifelong outstanding achievements in swine veterinary medicine. The award is made on an irregular basis and only when a deserving individual has been nominated and selected. Awardees have demonstrated their eligibility through their membership in the AASV, service to the AASV, and service to the North American swine industry.

New Heritage Video featuring Max Rodibaugh

A new Heritage Video, featuring Dr Max Rodibaugh, is now available. The Heritage Video Series is an ongoing project of the AASV Communications Committee, with support from the AASV Foundation and the creativity of Dr Sarah Probst Miller and AgCreate Solutions, to record and preserve AASV history through the recollections of its members. The video is available for viewing by AASV members at **aasv.org/members/only/video/**.

Sadly, Dr Rodibaugh passed away May 19th after battling brain cancer for more than two years.

AASV Foundation news continued on page 265

COST OF BUSINESS OR REVENUE OPPORTUNITY?

ARE YOU LEAVING $5/GILT ON THE TABLE?

The performance gap between gilts and barrows is well recognized. We all know that barrows grow faster and reach desirable carcass weights more quickly. Yet the economic impact of the gilt gap has not been broadly communicated to the industry until now.

New research by Dr. Ben Bohrer, meat scientist at The Ohio State University, and Dr. Jason Woodworth, nutritionist at Kansas State University, quantifies the differences between barrows and gilts in growth performance, carcass composition and meat quality. Their extensive research review offers the first clear definition of the gilt gap and the revenue implications for your operation.

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BASED ON HCW

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See the proof at BuiltForTheGilt.com

[1]Woodworth, J., et al. (2021). Characterizing the differences between barrow and gilt growth performance, carcass composition, and meat quality. KSU Applied Swine Nutrition Department.
*Assuming market price = $75/cwt, gilts HCW = -2.2%, premium price = +15% over base price

zoetis

AASV Foundation news continued from page 263

Golfers: Tee up!

It is time to recruit and register your golf team to support the AASV Foundation!

Registration is now open for the annual AASV Foundation Golf Outing, to be held **Wednesday, August 31**. The event is returning to Veenker Memorial Golf Course in Ames, Iowa, where past participants have enjoyed lovely weather, great food, and a well-groomed course with just the right amount of challenge. There is plenty of room for additional golfers -- so practice your swing and register to spend a relaxing day with your colleagues in support of the foundation.

Everyone is welcome! AASV members, industry stakeholders, clients, staff, family, and friends are all invited to register a 4-person team for this fun, 18-hole best-ball tournament. Individual golfers and pairs are also welcome and will be assigned to a team. The registration fee ($125 per golfer/$500 per team) includes 18 holes of best-ball golf, cart, lunch, beverages, awards dinner, and prizes. Preregistration is required by August 17.

Golfer check-in begins at 11:00 AM and a shotgun start at noon kicks off the event. Golfers compete as a foursome against the challenges of the course in addition to participating in individual contests along the way. Using Scrolf electronic scoring, golfers can check their progress against the other teams as they make their way around the course.

Boxed lunches will be sponsored by **APC** and **Zoetis** will keep golfers hydrated with beverages throughout the afternoon. At the conclusion of the golfing, event coordinator Dr Josh Ellingson announces the team and individual contest winners during the pork dinner sponsored by **Boehringer Ingelheim Animal Health**. Contests and giveaways hosted at the golf holes by additional sponsors add to the fun with prizes!

Funds raised by the event support AASV Foundation programs, including research grants, travel stipends for students attending the AASV annual meeting, swine externship grants, scholarships for veterinarians pursuing board certification in the American College of Animal Welfare, student debt relief scholarships, AASV heritage videos, and more. Thanks to strong sponsorship support and golfer participation, last year's outing raised more than $15,000 for the foundation!

For a sneak peek at the golf course, visit **veenkergolf.com**. For more information or to register, see **aasv.org/foundation/golf/**, or contact AASV by phone, 515-465-5255, or email, **aasv@aasv.org**.

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A: $1,889

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1: Perri A et al. An investigation of iron deficiency and anemia in piglets and the effect of iron status at weaning on post-weaning performance. JSHAP. 2016;24:10–20.

2: Fredericks L et al. Evaluation of the impact of iron dosage on post- weaning weight gain, and mortality. AASV. 2018;315

3: Olsen, C. (2019) The economics of iron deficiency anemia on US swine production: An annual impact of 46-335 million US dollars. American Association of Swine Veterinarians. Orlando. Florida.

* Industry Standards for Blood Hb Levels (g/L)

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What goes around, comes around

When I began graduate school in 2003, one of the major research studies I worked on was evaluating the impacts of 3 different group-housing space allowances on gestation sow welfare as compared to housing gestating sows in individual stalls. That study was one of many studies funded by the Pork Checkoff at the time. While the first ballot initiative restricting the use of gestation stalls had passed in Florida the year prior, the issue of gestation housing had landed on the US swine industry's radar a decade prior when the European Union had passed a directive restricting gestation stall use in 1991 (updated in 2008).

Three separate reviews of the scientific literature were conducted to compare group and individual housing systems and the impact they have on sow welfare. All three literature reviews came to similar conclusions – the advantages and disadvantages of gestation housing systems are qualitatively different and, therefore difficult to compare overall welfare.[1-4] One review added, "In fact, the focus on housing systems may have been to the detriment of recognizing the relative importance of another feature of the commercial pig's environment, that is the stockperson."[1] The conclusions of these reviews served as the basis for the development of both the AASV (**aasv.org/aasv/position-sowhousing.php**) and AVMA (**avma.org/resources-tools/avma-policies/pregnant-sow-housing**) position statements related to sow housing.

Fast forward 2 decades to 2022. Millions of dollars have been invested in public and private research of gestation sow housing. Gestation sow housing resources have been developed and experiences shared through numerous outlets. Pig farmers and equipment manufacturers are often featured speakers at industry meetings, including the AASV Annual Meeting, to share their experiences with building, implementing, and managing gestation sow housing systems. A few examples of available sow housing resources include:

- A series of factsheets that address the key decisions to be considered when choosing a housing system (**lms.pork.org/Tools/View/sow-housing-options**)
- A series of guides to assist caretakers in successfully managing each type of housing system (**lms.pork.org/Tools/View/sow-housing-management**).
- A financial comparison tool that enables producers to economically compare group sow housing systems (**canr.msu.edu/resources/sow_housing_options_tool**)
- Outputs from Canada's National Sow Housing Conversion Project (**groupsowhousing.com**)

No matter what gestation sow housing system a client may elect to use, veterinarians have opportunities to protect pig health and welfare by providing science-informed guidance. These opportunities may come in the form of interactions with individual clients, marketplace stakeholders, or state/federal policy makers. California's Proposition 12 is the latest opportunity for veterinarians to provide science-informed guidance on sow housing issues. The AASV submitted comments in response to the California Department of Food and Agriculture's proposed rulemaking for Proposition 12. As the legal challenge to Proposition 12 advances to the US Supreme Court, AASV has submitted an Amicus Brief that will emphasize the AASV's position on sow housing and share scientific evidence supporting the use of various types of housing systems to protect pig health and welfare, food safety, and public health.

Like all things in swine production, there is no one-size-fits-all solution for gestation sow housing. Ultimately, producers must do what is best for their animals, their employees, their facilities, and their marketplace. Veterinarians can support producers by helping them make science-informed decisions.

Sherrie Webb, MSc
Director of Swine Welfare

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For more information and to register:
Amanda Chipman
502 E 9th St
Des Moines, IA 50319
Tel: 515-422-4083
Email: **amanda.chipman@iowaagriculture.gov**
Web: **docs.google.com/forms/d/e/1FAIpQLSe5I2Z4tjt_ahsmOQZFVDDzfug1LyNfddkJ0lvjGyGDlp8ecw/viewform**

LIV AMVEC National Congress

July 12 - 15, 2022 (Tue-Fri)
Monterrey, Mexico

For more information:
Mexican Association of Veterinary Specialists in Swine
Plan de Adobes 2051 Calle Plan de Adobes
47600 Tepatitlán de Morelos, JAL
Mexico
Email: **administracion@amvec.com**
Web: **amvec.com/event/liv-congreso-nacional-2022-2022-07-12-2022-07-15-7/page/introduccion-liv-congreso-nacional**

2022 Annual Therio Conference

July 20 - 23, 2022 (Wed-Sat)
Bellevue, Washington

Hosted by the Society for Theriogenology and the American College of Theriogenologists

For more information:
Web: **theriogenology.org**

Allen D. Leman Swine Conference

September 17 - 20, 2022 (Sat-Tue)
Saint Paul, Minnesota

Hosted by the University of Minnesota College of Veterinary Medicine

For more information:
Web: **lemanconference.umn.edu**

126th US Animal Health Association Annual Meeting

October 5 - 12, 2022 (Wed-Wed)
Hyatt Regency Minneapolis
Minneapolis, Minnesota

For more information:
Web: **usaha.org/meetings**

North American PRRS/NC229 International Conference on Swine Viral Diseases

December 2 - 4, 2022 (Fri-Sun)
Chicago, Illinois

For more information:
Web: **go.illinois.edu/NAPRRSSymposium**

AVMA Leadership Conference

January 5 - 7, 2023 (Thu-Sat)
Chicago, Illinois

Hosted by the American Veterinary Medical Association

Web: **avma.org/events/veterinary-leadership-conference**

American Association of Swine Veterinarians 54th Annual Meeting

March 4 - 7, 2023 (Sat-Tue)
Gaylord Rockies Resort and Convention Center
Aurora, Colorado

For more information:
American Association of Swine Veterinarians
830 26th Street
Perry, Iowa
Tel: 515-465-5255
Email: **aasv@aasv.org**
Web: **aasv.org/annmtg**

For additional information on upcoming meetings: aasv.org/meetings

AASV INDUSTRY SUPPORT COUNCIL

The *Journal of Swine Health and Production* is made possible by the generous support of these Industry Support Council members:

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