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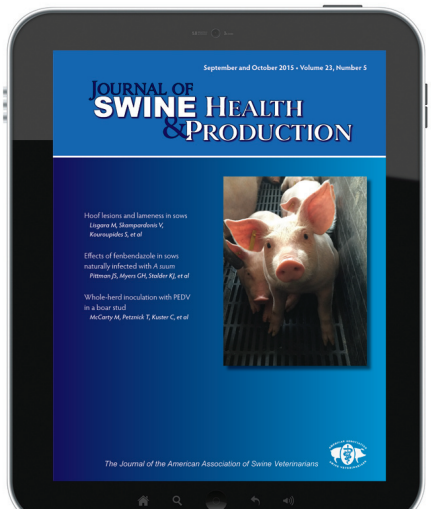

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About the cover…

My, what big ears you have!

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quoted from Why do you do what you do?, page 243

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PRRS **DIARRHEA** **MYCO**

The year 2015 – a time of fresh new opportunity

My daughter Anna graduated from high school in May – the class of 2015. For her, the preparation leading up to graduation day seemed like a few short weeks. For Joan and me, it seemed like preparation for this day had been going on for years. Well, of course it had! We had been following and helping to facilitate Anna and her classmates through sports, music, academics, and social activities since grade school. Now, for Anna, Joan and me, it seems like it is all finished – or is it? We've had a summer break, it's time to look ahead.

Summer in Nebraska has been filled with activities, including trips to the Missouri River, family reunions, and small-town celebrations. Anna is now at a "pivotal point" for the next stage of life. By the time you read this, she will be a college freshman participating in the Honors Academy within the University of Nebraska-Lincoln College of Business Administration. She will experience the many changes that college life offers. Along with those changes come responsibilities and some fresh new opportunities. As for all of us, her challenge will be to realize and capture opportunity when it presents itself.

This year also represents a pivotal point of change and a fresh new opportunity for veterinarians in private practice. In June 2015, the Federal Drug Administration (FDA) published their final rule for the updated version of the Veterinary Feed Directive (VFD),[1] which will go into effect October 1, 2015. The VFD rule change is the third of three core documents that FDA is using to announce and implement its policy framework for the judicious use of medically important antimicrobial drugs in food-producing animals. The first two rule documents are FDA Guidance 209[2] and FDA Guidance 213.[3] Together, the three documents eliminate over-the-counter use of most antibiotics in feed, provide rules for new labeling requirements, and direct the responsibility of legal oversight to veterinarians.

"I see the new FDA rules as an opportunity to get veterinarians on the farm to see things that may be overlooked."

The VFD is not a new instrument. It is similar to a veterinary prescription, but is defined differently to account for usage of antimicrobials in livestock feed. It was implemented in the year 2000 within the Animal Drug Availability Act.[4] The usage of VFDs has been limited because there have been only two new antibiotics approved for swine since then. Up until now, the frequency of VFDs issued by veterinarians has been relatively low. The new FDA antibiotic rules will necessitate a large increase in the number of VFDs.

What is particularly interesting in the new VFD rule is the reference to a veterinary-client-patient-relationship (VCPR). Until the new ruling was published, it was unclear what role the VCPR would have. The FDA has affirmed the VCPR is a requirement for all VFDs and defers to individual states regarding VCPR definitions. For most states, two portions of a VCPR present a challenge.

The first challenging portion of the VCPR is "The veterinarian assumes responsibility for making clinical judgments regarding the health and medical treatment of the animals, and the client has agreed to follow the instructions of the veterinarian." Historically, an agreement between veterinarians and their clients has not been required when blending over-the-counter antimicrobials into livestock feed. This represents a paradigm shift and will require a period of adjustment for both producers and veterinarians. I anticipate that veterinarians will ask producers to sign a written agreement to follow veterinary instructions before new VFDs are issued. This could potentially release veterinarians from liability of non-compliance.

The second challenging portion of the VCPR, to paraphrase, is that the veterinarian must be personally familiar with the care of the animals and the veterinarian has made medically appropriate and timely visits to the premises where the animals are kept. The phrase "timely visits to the premises" will lend itself to interpretation. This time period will be debated, but I speculate that no less than one visit per year will become standard. Yes, there will be added expense. And yes, more veterinarians will be employed.

I see the new FDA rules as an opportunity to get veterinarians on the farm to see things that may be overlooked. Health programs should be reviewed during farm visits. Animals and facilities should be observed. Site assessments can be included for Pork Quality Assurance (National Pork Board) site status. Biosecurity protocols can be reviewed. Preparations can be made for the next Common Industry Audit. All of these services allow veterinarians to enhance their relationship with producers of all herd sizes.

The new FDA rules place us veterinarians at our own pivotal point, providing a chance to

President's message continued on page 239

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Reference: 1. Buhr BL, Hurley T, Tonsor G, Zering K, DiPietre D. Comprehensive economic analysis of Improvest adoption by the US pork industry. *Am Assoc Swine Vet.* 2014;201-206.

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President's message continued from page 237

capture an opportunity – an opportunity to strengthen client relationships and enhance the public image of veterinarians. As with my daughter Anna, we too are entering a time with fresh new opportunity. We as veterinarians are being entrusted with the stewardship of antibiotics. Let's make good with it.

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Scope

Many journals establish and publish the criteria surrounding the scope of their journal. As an editor, I have a good understanding of what scope means in reference to the scope of a journal, but I wondered what the dictionary would say, so I dusted off my dictionary and looked it up. According to *Merriam-Webster's Collegiate Dictionary*, scope is derived from the Italian word "scopo," which means aim or purpose.[1] There is also the Latin root "skopeo, which means "to look out for." I consider the scope of a journal to be the extent of coverage of a subject area that the journal choses to focus on. The scope of the *Journal of Swine Health and Production* (JSHAP) has always been to have an applied focus as it relates to commercial swine health and production. Having an applied focus is what makes our publication accessible to the busy veterinary swine practitioner. Earlier this year the journal updated our Web site and included a more detailed description of the scope of JSHAP. If you haven't had a chance to see this description (available at **https://www.aasv.org/shap.html**), I have provided it here (emphasis added):

> The *Journal of Swine Health and Production* (JSHAP) is an open-access and peer-reviewed journal published by the American Association of Swine Veterinarians (AASV) since 1993. The aim of the journal is the timely publication of peer-reviewed papers with a scope that encompasses the many domains of applied swine health and production, including the diagnosis, treatment, management, prevention, and eradication of swine diseases, welfare and behavior, nutrition, public health, epidemiology, food safety, biosecurity, pharmaceuticals, antimicrobial use and resistance, reproduction, growth, systems flow, economics, and facility design. The journal provides a platform for researchers, veterinary practitioners, academics, and students to share their work with an international audience. The journal publishes information that contains an applied and practical focus and presents scientific information that is accessible to the busy veterinary practitioner, as well as to the research and academic community. Hence, manuscripts with an ***applied focus*** are considered for publication, and the journal publishes original research, brief communications, case reports/series, literature reviews, commentaries, diagnostic notes, production tools, and practice tips. All manuscripts published in JSHAP are peer-reviewed.

"The scope of the Journal of Swine Health and Production (JSHAP) has always been to have an applied focus as it relates to commercial swine health an production."

I occasionally have an author contact me to see if their work (and manuscript) is within the scope of our journal. Sometimes a brief description of the topic area and the general message in the manuscript is all that is needed for confirmation. But if it is still unclear whether their manuscript is within our scope, I usually encourage them to send it to the journal office for pre-screening. This pre-screen can save considerable time and effort that is appreciated by both the authors and potential reviewers. If the work is not applicable to our journal scope, then this saves the author time in waiting for a full review and allows them to instead send their work to a journal with a better fit. If the manuscript is within the scope of JSHAP, then the manuscript goes on for full peer review.

The description above of the scope of JSHAP also includes the terms "open access" and "peer-reviewed." The term open access, in reference to JSHAP, means that you do not need to buy a subscription to have access to the online scientific publications and that anyone can freely access our publications online. As members of the American Association of Swine Veterinarians (AASV) know, the print subscription is included with AASV membership or, alternatively, it can be purchased separately. To freely access JSHAP online follow the link **https://www.aasv.org/shap/issues/**. The JSHAP also does not charge author publications fees. For some journals, publication fees can be quite high, in the area of thousands of dollars, but the strength of the AASV membership and Industry Support Council allows us to keep publication fees transferred to authors at $0. In a previous editorial, I have described the peer-review process for manuscripts submitted to JSHAP.[2] The peer-review process at JSHAP is a thorough procedure that requires input from many individuals, such as numerous reviewers, editorial board members, and journal staff. As I have highlighted in another editorial,[2] this detailed oriented process is critical in order to maintain the integrity of peer-reviewed publications. For questions regarding the scope of JSHAP or to ask if your work is within our journal scope, please do not hesitate to contact the journal office at **pub_mgr@aasv.org**.

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Terri O'Sullivan, DVM, PhD
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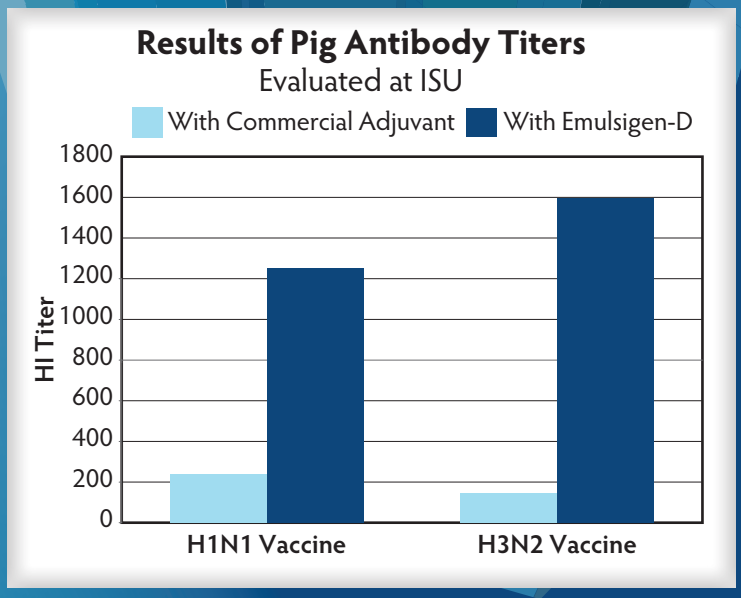

B.C. Lin, et al., AASV March 2006

Why I do what I do

In the hectic world of busy veterinary practice and family life, I was not looking forward to sitting down and writing a piece for the *Journal of Swine Health and Production* (JSHAP) after Dr Burkgren asked! In hindsight, it has been an interesting exercise in self-evaluation and self-awareness, and a reminder of some life lessons learned so far! There was a time, in early practice life, that I described myself as having a professional identity crisis. The romantic idealism of a young veterinarian had worn thin, and being welcomed into swine practice many years ago has allowed me many opportunities to grow personally and professionally. I will try to describe why I do what I do.

In previous articles, under similar titles, Drs Burkgren,[1] Holman,[2] and Starke[3] have eloquently outlined their passion and drive for a profession and industry that I believe many AASV members share. I share this passion as well, although I didn't start out that way, rather developing a passion for pigs, people, and problem-solving by accident.

I was a farm kid, raised on a small dairy farm in central Manitoba. We worked hard as youngsters, pitching square hay bales, mucking out calf pens, feeding calves, and occasionally getting to milk cows if Dad was away. My passion was cows. Veterinary medicine never really featured in my plans. I had no intentions of spending any more time in school than necessary, as I was going to be a 4th generation dairy farmer. Besides, most of the veterinarians I had ever seen visit our farm were a bit strange, always in too much of a rush to spend time teaching a farm kid how to recognize problems or the contributing factors creating those problems. Those were missed opportunities for those veterinarians, and I have tried in my career not to miss teachable moments.

"My swine-practice career...allows me to find balance between veterinary science, the art of reading people, problem solving, and critical thinking."

Obviously, I am not a 4th generation dairy farmer today! Life's path does seem to twist and turn. In my pursuit to farm with my family, having received my college agricultural diploma, I quickly realized two things. Milking cows "24/7" is hard, relentless work, and working with family members can be even more difficult and relentlessly frustrating! Another life lesson, looking for compromise when it can be found, will often be more productive and save time. Pick your battles wisely: again, something I try to remember every day in veterinary practice.

Well, farming wasn't in my cards, but with a passion for farm animals, I had talked myself into a veterinary career. Veterinary school was great. I met some great people and made some life-long friendships. I had little interest in the "pet-ables," but had a real passion for the edibles. Fast-forward 4 years, and I was a single father of two little kids, starting out in a busy multi-person practice with a large component of dairy-farm clients. I had the world by the tail and was living the dream!

About 10 months into practice I was tired, grumpy, and not intellectually stimulated. My kids were usually first to day care in the morning and last to be picked up in the evening. My practice style had turned into pattern recognition, not problem solving. Life lesson number three: you must set priorities at each stage of your life, because nobody else will do that for you. While I realized a change was needed, a serendipitous meeting with Dr Brad Chappell, my current practice partner, gave me the opportunity to join a busy, progressive swine practice. I had landed in a place that encouraged personal growth, family balance, and lifelong learning.

Thirteen years later, I absolutely no longer have a professional identity crisis! My kids are no longer the last to leave day care (...not every day, anyway!) and I have the privilege of working alongside extremely talented veterinarians and practice staff. I take pride in trying to provide mentorship to our younger veterinarians and students that come through our practice, as I was mentored. I am routinely reminded of the important role veterinarians play as teachers and leaders while on-farm, in our communities, and within our profession. I have huge esteem for my veterinary colleagues within the AASV membership and the seemingly ceaseless drive to advance swine medicine, welfare, and the production of safe abundant food. My swine-practice career placed me among colleagues who share these values and allows me to find balance between veterinary science, the art of reading people, problem solving, and critical thinking. This is really why I do what I do.

Not all AASV members will have the opportunity to contribute an article like this one to the JSHAP; however, this exercise has been personally gratifying and has further helped me find self-awareness. I would suggest, if you have not done so yet, that you write down why *you* do what you do!

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Blaine Tully, DVM
Swine Health Professionals, Ltd

ORIGINAL RESEARCH PEER REVIEWED

Hoof lesions and lameness in sows in three Greek swine herds

Marina Lisgara, DVM; Vassilis Skampardonis, DVM, PhD; Stelios Kouroupides, DVM; Leonidas Leontides, DVM, MPVM, PhD

Summary

Objectives: To characterize foot lesions, estimate their frequency and severity, and investigate their association with parity and lameness in three Greek farrow-to-finish swine herds.

Materials and methods: The studied sows, which had been individually stalled during previous gestations, were examined for foot lesions upon entry into the lactation facilities. Lesions scored included heel hyperkeratinization, erosions or cracks, and toe and dew claw overgrowths. When exiting the farrowing facilities, the sows were observed while walking along an alley and their degree of lameness was scored.

Results: The proportion of sows with at least one lesion on any foot was very high and similar among herds, with 121 of 125 (96.8%), 123 of 125 (98.4%), and 377 of 386 (97.7%) sows affected in herds A, B, and C, respectively. The most frequent lesions were those located on the heel, and overgrown toes and dew claws. For these sites, lesion severity increased with sow parity. The concurrent presence of lesions on more than one foot site, on the same or different feet or both, had a multiplicative effect on the likelihood of lameness.

Implications: Under the conditions in the herds participating in this study, sow foot lesions are extremely common, with older sows more likely than younger sows to have lesions on the heel and overgrown toes and dew claws. The degree of lameness may be affected by a causal interface among foot lesions.

Keywords: swine, hoof lesions, lameness

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Resumen - Lesiones de pezuña y cojera en hembras en tres hatos porcinos de Grecia

Objetivos: Caracterizar las lesiones de pata, estimar su frecuencia y severidad, e investigar su asociación con paridad y cojera en tres hatos porcinos de ciclo completo en Grecia.

Materiales y métodos: Las hembras estudiadas permanecieron en jaulas individuales en gestaciones previas y fueron examinadas en busca de lesiones al entrar a las instalaciones de maternidad. Las lesiones calificadas, incluyeron erosiones hiperqueratinizadas o grietas de talón, y el crecimiento excesivo en las uñas traseras o delanteras. Al salir de las instalaciones de maternidad, las hembras fueron observadas al caminar por el pasillo y se calificó su grado de cojera.

Resultados: La proporción de hembras con al menos una lesión en cualquier pata fue muy alta y similar entre los hatos, con 121 de 125 (96.8%), 123 de 125 (98.4%), y 377 de 386 (97.7%) hembras afectadas en los hatos A, B, y C, respectivamente. Las lesiones más frecuentes fueron aquellas localizadas en el talón, y las uñas delanteras o traseras con crecimiento excesivo. Para estos sitios, la severidad de la lesión se incrementó con la paridad de la hembra. La presencia simultánea de lesiones en más de un área de la pata, en la misma pata o en pata diferente o en ambas, tuvo un efecto multiplicativo en la probabilidad de cojera.

Implicaciones: Bajo las condiciones de los hatos participantes en este estudio, las lesiones de pata de las hembras son extremadamente comunes, teniendo las hembras más viejas mayor probabilidad que las hembras más jóvenes de tener lesiones en el talón y crecimiento excesivo de uñas. El grado de cojera puede ser afectado por una interrelación causal entre las lesiones de pata.

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This article is available online at http://www.aasv.org/shap.html.

Lisgara M, Skampardonis V, Kouroupides S, et al. Hoof lesions and lameness in sows in three Greek swine herds. *J Swine Health Prod.* 2015;23(5):244–251.

Résumé - Lésions aux sabots et boiterie chez des truies dans trois troupeaux porcins en Grèce

Objectifs: Caractériser les lésions aux pieds, estimer leur fréquence et sévérité, et évaluer leur association avec la parité et les boiteries dans trois troupeaux porcins de type naisseur-finisseur en Grèce.

Matériels et méthodes: Les truies à l'étude, qui étaient logées individuellement lors des gestations antérieures, furent examinées pour la présence de lésions aux pieds lors de leur entrée dans les installations d'allaitement. Les lésions notées incluaient l'hyperkératinization du talon, les érosions ou les fendillements, et la croissance exagérée des onglons des orteils et des ergots. Lors du départ des installations de mise-bas, les truies étaient observées lorsqu'elles marchaient dans l'allée et leur degré de boiterie notée.

Résultats: La proportion de truies avec au moins une lésion à un des pieds était très élevée et semblable parmi les troupeaux avec 121 des 125 (96,8%), 123 de 125 (98,4%), et 377 de 386 (97,7%) des truies affectées dans les troupeaux A, B, et C, respectivement. Les lésions les plus fréquemment observées étaient celles localisées au talon, et la croissance exagérée des orteils et des ergots. Pour ces sites, la sévérité des lésions augmentait avec le nombre de parité de la

truie. La présence simultanée de lésions à plus d'un site, sur le même pied, sur des pieds différents, ou les deux, avait un effet multiplicateur sur la probabilité de boiterie.

Implications: Dans les conditions de la présente étude, les lésions aux pieds des truies sont extrêmement fréquentes, les truies plus âgées plus enclin que les plus jeunes à avoir des lésions sur les talons et a une croissance exagérée des orteils et des ergots. Le degré de boiterie peut être affecté par l'interaction entre les lésions aux pieds.

Hoof lesions, an important underlying cause of locomotor disorders in pigs,[1] have been associated with lameness and culling or euthanasia.[2-4] Lameness is an animal-based welfare indicator.[5] From an economic point of view, lameness reduces the productivity of a pig unit by reducing sow longevity and the number of pigs produced per sow per year due to increased involuntary culling rate of sows, increased expenses as a result of sow replacement costs, increased work load and treatment expenses, and fewer finisher pigs reaching the slaughterhouse.[6-8]

In studies conducted in modern herds in the United States and Belgium, almost every sow had at least one foot lesion.[9,10] However, not all of these sows were lame. Approximately 5% to 20% of lameness cases in sows were attributable to foot lesions.[1,11] Location[9,12] and severity[10,13] of the lesions might determine whether a sow shows overt lameness or not. Furthermore, hoof injuries may serve as possible ports of entry for infections which may ascend and spread through the body, affecting joints and other tissues, causing stress and pain. Hence, infected hoof lesions can cause severe lameness that reduces the sow's appetite and increases her susceptibility to other diseases, through alteration of the immunological response.[14]

One of the major causes of injuries to the foot at the time of mixing in pens is fighting on concrete or slatted flooring or on combinations of concrete and slatted flooring. Even after the dominance hierarchy is established, grouped sows will continue to fight if they are overstocked, have to compete for access to feed, or are stressed by a perpetual feeling of hunger.[15,16] Previously, fighting was controlled by the use of individual stalls for pregnant sows. However, the European Union (EU) Directive 2001/88/EC, implemented since January 2013 in all 25 member states, requires that sows and gilts be kept in groups during a period starting 4 weeks after service and until 1 week before the expected time of farrowing. Without managerial adjustments, it is reasonable to expect that the importance of foot lesions and associated lameness to longevity and productivity of grouped sows will increase.[9,17] In this study, conducted in three Greek swine herds during the first 6 months of 2013, sow foot lesions were characterized, their frequency and severity were estimated, and their associations with parity and lameness were investigated. The results depict the baseline prevalence and severity of foot lesions before implementation of the directive for group housing in these herds.

Materials and methods

This study was conducted in farms that complied with the current laws concerning the protection of animals kept for farming in the European Union.[18] Approval of the study protocol by an animal care committee was not required because taking part in the study was in no way painful or invasive for the animals.

Study population

The studied herds were indoor, farrow-to-finish herds with 330 (Herd A), 160 (Herd B), and 800 sows (Herd C), respectively, with Danbred (herds A and B) and Hermitage (Herd C) genotypes. Before finalizing the necessary reconstruction of the dry-sow units to meet the requirements of the EU Directive 2001/88/EC, all herds kept pregnant sows in individual stalls. Herd C finalized the reconstruction at the end of 2012 and was inspected and granted compliance with the directive by the veterinary authorities in January 2013, whereas herds A and B finalized the reconstructions in the spring and were granted compliance in June 2013.

In the reconstructed units, the animals were loose housed in groups of eight to 12 on combinations of concrete and slatted flooring, as described in the directive. All herds operated on weekly farrowing schedules. Transition to group housing was accomplished gradually, within 4 months after compliance was granted, by grouping the pregnant sows that had been inseminated a month before. Therefore, all sows in the study population had been individually housed during their previous gestations. For participation in the study, the only criterion was the owners' written consent. Neither the health status of the sows' feet nor the frequency of locomotor disorders was considered for herd selection.

Study design

Three farm employees examined the sows' feet for lesions when they entered the lactation facilities. Sow lameness was evaluated upon exit of the animals from the lactation facilities, when managers decided whether a weaned sow would be re-bred or culled, considering reproductive performance, age, and locomotor soundness. Two of the authors (LL and ML) trained the employees to recognize, characterize, and score foot lesions and lameness. An initial training session was held at the clinics of the School of Veterinary Medicine, University of Thessaly (Karditsa, Greece), where the different anatomical sites of the foot were identified, and representative foot lesions in feet collected at slaughter were characterized and scored. Lameness identification and scoring were demonstrated in a video of sows with normal or abnormal gait and posture. Training was repeated on each farm, and employees were provided with a collection of photographs and the video of the training material. Each sow's data was recorded on especially developed paper data-capture forms. The primary author visited all farms once a month, collected the completed data-capture forms, and cross-checked the data by re-examining a random sample of 20% of the sows with the responsible farm employee. The medial and lateral toes of each foot were individually examined for lesions and scored both when sows were lying down (the ventral surface) and standing up (the dorsal surface) in the farrowing crate before farrowing. Lesions included heel hyperkeratinization, erosions or cracks, and toe and dew claw overgrowth. Specifically, five hoof anatomical sites were examined: the heel (soft keratinized epidermis on the ventral surface of the hoof towards the caudal end); the sole (hard keratinized epidermis cranial to the heel on the ventral surface of the hoof, including the junction between heel and sole); the white line (junction between sole and wall); the wall (hard keratinized epidermis on the dorsal surface of the hoof); and the coronary band. The scoring system applied (Table 1) was based on "Zeugenklauwencheck," a scoring system developed in the Netherlands,[10] and the Zinpro Foot First method,[19] with some modifications. Epidermal lesions and

length of toes and dew claws were scored on a severity scale ranging from 0 to 2, with the exception of the coronary band lesion, where the score was 0 when healthy and 1 when any lesion was observed. On exiting the farrowing facilities, sows were observed from the front and rear while walking down an alley, and their difficulty in bearing weight on one or more feet was scored. Sows exhibiting normal gait were assigned lameness score 0 (non lame); those with alteration or shortening of stride, without serious locomotion impairment or reluctance to move, showing partial inability to bear weight on one or more feet, were assigned score 1; and those with serious locomotion impairment and reluctance to move, showing complete inability to bear weight on one or more feet, were assigned score 2.

Statistical analysis

All statistical analyses were performed using Stata 13.1 (Stata Statistical Software, College Station, Texas). The total score for the four feet for each anatomical site was obtained by adding the respective scores of hooves, toes, and dew claws. Therefore, for each anatomical site except the coronary band, the total score for the four feet could range from 0 to 16; for the coronary band, the total score could range from 0 to 8. The total score for each foot was obtained by adding the scores for each anatomical site considered. Therefore, the total score for each foot could range from 0 to 13.

Subsequently, descriptive statistics of the data were calculated. The Wilcoxon signed-rank test was used to compare the medians of total scores of lesions in each anatomical site between front and rear feet, in each herd. McNemar's χ^2 test for symmetry was used to compare the proportion of sows with at least one lesion in front and rear feet. Pearson's χ^2 test was used to compare the proportion of sows with lesions on each site scored among the three herds, whereas the medians of scores for each site were compared among herds with the Kruskal-Wallis test. Multiple comparisons were interpreted at Bonferroni-adjusted *P* values.

Three ordered logistic regression models were fitted to estimate the association between parity and the total score on all feet, one for each of the three most frequently recorded lesions, which were heel lesions, overgrown toes, and overgrown dew claws. In each model, parity was the dependent variable, while the total lesion score was the explanatory variable. Parity was characterized in one of three categories (parity groups) (PGs) comprising parities 1 or 2 (PG1), 3 to 5 (PG2), and ≥ 6 (PG3). A dummy variable coding for "herd" was forced in all models because it controlled for variation in the outcome due to different herd-parity distribution and unmeasured factors associated with it, as well as different sampling frequency. The assumption of proportionality in the odds did not hold for herd in the models associating parity with heel lesions and dew-claw length, and for toe length in the model associating parity with this lesion. Thus, partial proportional odds models were fitted using the gologit2 command. These models are less restrictive than

Table 1: Scoring system applied for evaluation of lesions on seven foot sites of 636 sows in three Greek farrow-to-finish herds*

Foot site	Score 0†	Score 1‡	Score 2§
Sole	No lesions or very small superficial cracks in the epidermis	Serious lesions in the epidermis not extending into the corium, heel-sole separation, or both	One or more deep cracks extending into the corium, severe heel-sole separation, or both
Heel	No lesions or very small superficial cracks in the epidermis	Hyperkeratinization and erosions in the epidermis not extending into the corium	Hyperkeratinization, deep cracks extending into the corium, and often necrosis
White line	No lesions or very small superficial cracks in the epidermis	Wall-sole separation not extending into the corium	Wall-sole separation extending into the corium
Wall	No lesions or very small superficial cracks in the epidermis	Cracks not extending into the corium, often accompanied by bruising	Cracks extending into the corium, separation of the keratin, or both
Coronary band	No lesions or very small superficial cracks in the epidermis	Edema with purulent exudate, hemorrhage and necrosis, or both	NA
Toe	Normal length	Overgrown toes	Overgrown and twisted or cracked toes
Dew claw	Normal length	Overgrown dew claws, touching the floor when the animal is standing	Overgrown and twisted or crushed dew claws

* Based on a Dutch scoring system (Zeugenklauwencheck)[10] and the Foot First Method[19] with some modifications.

† Corresponding to "score 1 or 2" in the Dutch scoring system or "mild" in the Foot First system.

‡ Corresponding to "score 3" in the Dutch system or "moderate" in the Foot First system. For the coronary band, the score applied in this study corresponds to "score 3 or 4" in the Dutch system.

§ Corresponding to "score 4" in the Dutch scoring system or "severe" in the Foot First system.

NA = not applicable; for the coronary band, lesion score was 0 when healthy and 1 when any lesion was observed.

the parallel-lines models, but more parsimonious and interpretable than those fitted by a non-ordinal method, such as multinomial logistic regression.[20]

In herds A and B, the recorded frequency of lame sows was very low, and therefore their data was not considered in the analysis of the association between lameness and severity of foot lesions. Scoring of lesions at the seven foot sites considered resulted in 56 variables for each sow examined. The major problem to be dealt with in analyzing this data set was multicollinearity, ie, predictor variables were closely related to each other (highly correlated) because they referred to the same animal or foot, or even to the same claw. The available techniques to deal with multicollinearity include either exclusion of highly correlated variables after screening for associations among the independent variables, or creation of indices or scores which combine data from multiple factors into a single variable, or creation of a smaller set of independent variables through use of multivariable techniques such as principal components or factor analysis.[21] We opted to conduct factor analysis to consolidate the information contained in all the original variables into a new smaller set of uncorrelated variables (factors). In factor analysis, the original variables are assumed to be a linear combination of the factors with weights (factor loadings) plus an error term.[21]

Extraction of the factors was accomplished by using the method of principal components.[22] The suitability of individual variables for use in the factor analysis was evaluated by using the Kaiser-Meyer-Olkin measure of sampling adequacy. Determination of the number of factors to keep for interpretation was a compromise between parsimony, interpretability, and the total amount of variation in the original variables that was explained by the factors in the model.[22] Kaiser's criterion (initial eigenvalue ≥ 1), a scree-test plot, and the number of factors that are required to account for a given proportion of the variance observed in the original variables[23] were considered in the analysis to determine which factors to retain for interpretation. Orthogonal and oblique factor rotations were both evaluated, but ultimately an orthogonal rotation (varimax option) was selected for the final analysis because it resulted in a relatively simple and interpretable structure while maintaining factor independence.[22] Factor loadings > 0.40 were used in the interpretation of rotated factors. Sixteen factors had an eigenvalue ≥ 1, suggesting that they should be kept for interpretation according to Kaiser's criterion, while use of the scree method suggested that 15 or 16 factors should be retained. After consideration of the amount of variance explained, we retained 16 factors, cumulatively accounting for almost 70% of the variance in the original variables. Then, for these 16 factors, the regression method was used to produce standardized factor scores.[22]

Subsequently, the produced standardized factor scores were evaluated as predictors of lameness score in an ordinal logistic regression model. Adjustment for the likely parity effect was accomplished by forcing parity into the model.[24] Because the assumption of proportionality did not hold for all predictors examined, we fitted partial proportional odds models.[20] To identify partial proportional odds models that fitted our data best, we used the autofit option, which is a built-in option of gologit2. When this option is specified, gologit2 goes through an iterative process. Initially it fits a totally unconstrained model and then performs a series of Wald tests on each variable to determine whether its coefficients differ across equations, eg, whether the variable meets the parallel-lines assumption. If the test is significant for one or more variables, the variable with the least significant value is constrained to have equal effects across equations. The model is then refitted with constraints, and the process is repeated until there are no more variables that meet the parallel-lines assumption. Finally, a global Wald test is done, which compares the final model with constraints to the original unconstrained model and, if the Wald test is statistically insignificant, the final model does not violate the parallel-lines assumption.[20]

For factor score selection for the final regression model, we initially fitted bivariable models, including each factor score and parity. Factor scores significant at $P < .25$ were candidates for the final model.[25] The initial full model fitted included parity and all standardized factor scores previously identified as significant. It was then reduced by backward elimination of factor scores with $P \geq .05$.[26] When only those with $P < .05$ remained, factor scores previously eliminated were offered one at a time to the model. This ensured that factor scores excluded earlier, during backward elimination, but adding significantly to the final model, were not missed. Lastly, all possible two-way interactions between factor scores in the model were created and tested for significance one by one. The fit of the final model to the data was assessed by comparing the observed to model-predicted probabilities of occurrence of each lameness score.[27]

Results

Foot lesions

A total of 636 sows were scored, of which 125 were in Herd A, 125 in Herd B, and 386 in Herd C (Table 2). The proportion of sows with at least one lesion on any foot was very high and similar among herds with 121 of 125 (96.8%), 123 of 125 (98.4%), and 377 of 386 (97.7%) affected sows in herds A, B, and C, respectively. In Herd C, the proportion of sows with at least one lesion on the front feet (338 of 386; 87.6%) was lower ($P < .001$) than the proportion of sows with at least one lesion on the rear feet (378 of 386; 97.9%). However, these proportions did not differ in Herd A or Herd B.

The most frequent and severe foot lesions observed in each herd separately are shown in Table 2. There was among-herd variation in the frequency and severity of these lesions. Heel lesions were less frequent ($P < .001$) in Herd A than in Herd B or Herd C. Frequency of heel lesions did not differ ($P = .10$) between Herd B and Herd C. The total score of heel lesions differed ($P < .001$) among the three herds, being lowest in Herd A and highest in Herd C. Both the frequency and severity of overgrown toes differed among the herds ($P \leq .001$ in each comparison), being more frequent and severe in Herd A and least frequent and severe in Herd C. Similarly, the frequency and severity of overgrown dew claws differed among the three herds ($P < .001$ in each comparison), being more frequent in Herd A and more severe in Herd C, and least frequent and severe in Herd B. In general, within herds, the median scores of the heel lesions, toe, and dew-claw length were higher ($P < .02$ in each comparison) for the rear than for the front feet, with the exception of the toe length in Herd A sows, which did not differ between front and rear feet ($P = .29$).

Association of lesions with parity

For heel lesions and for overgrown toes and dew claws, which were the most common lesions, there were associations of parity with the total score (Table 3). These associations were adjusted for the herd effect, which was included in the models as a confounder. For

each unit increase in the total score of heel lesions and dew-claw length, a sow was 1.10 times ($P < .001$) and 1.20 times ($P < .001$) more likely, respectively, to belong to PG2 or PG3 than to PG1. Additionally, for each unit increase in the total score of toe length, a sow was 1.15 and 1.26 times more likely ($P < .001$) to belong to PG2 or PG3 than to PG1 and to PG3 than to PG2 or PG1, respectively.

Association of lesions with lameness

In Herd C, the proportion of sows with locomotor disorders was 81 of 386 (21.0%). Specifically, 53 of 386 (13.7%) and 28 of 386 (7.3%) sows had lameness scores 1 and 2, respectively. In herds A and B, three of 125 and one of 125 sows, respectively, had lameness score 1, whereas none had lameness score 2.

All variables examined were suitable for inclusion in the factor analysis, since their Kaiser-Meyer-Olkin values were > 0.5, suggesting an acceptable fit with the structure of the other variables. Most variables loaded high on only a single factor, the exception being Factor 1. For this factor, three different groups of variables loaded: variables describing scores of white-line and sole lesions and of toe length of the rear foot. During final model building, five factor scores were found significant after backward elimination, and another during forward selection. None of the examined interactions were significant. Thus the final model included factor scores 1, 2, 5, 7, 11, and 13 as independent variables (Box 1).

According to the final model, lameness was associated with lesions on five foot sites. For all but one site, lameness severity increased with increasing lesion score, the exception being the wall of the front hoof, factor score 11 (Table 4). The likelihood of lameness score being ≥ 1 compared to 0 was almost three times higher ($P < .001$) per one unit increase in factor score 1, whereas it was almost two times higher ($P < .001$) for lameness score 2 compared to ≤ 1. For one unit increase in factor score 2, the odds were 1.90 times higher ($P = .004$) that lameness score would be 2 rather than ≤ 1. It was 1.70 times more likely ($P < .001$) that a sow would have a higher lameness score for a unit increase in

Table 2: Frequency of sows with at least one foot lesion and median (range) of the total score* for all feet by site and herd in a study conducted in three Greek farrow-to-finish herds

	Lesion frequency by site on the foot						
Herd	**Sole (%)**	**Heel (%)**	**White line (%)**	**Wall (%)**	**Coronary band (%)**	**Toe length (%)**	**Dew-claw length (%)**
A n = 125	55 (44.00)	65 (52.00)	58 (46.40)	67 (53.60)	21 (16.80)	115 (92.00)	114 (91.20)
B n = 125	70 (56.00)	112 (89.60)	24 (19.20)	84 (67.20)	12 (9.60)	96 (76.80)	83 (66.40)
C n = 386	207 (53.63)	362 (93.78)	148 (38.34)	212 (54.92)	63 (16.32)	162 (41.97)	322 (83.42)
	Median of total score (range)						
A	0 (0-13)	1 (0-12)	0 (0-8)	1 (0-11)	0 (0-8)	4 (0-14)	3 (0-15)
B	1 (0-7)	2 (0-10)	0 (0-6)	1 (0-4)	0 (0-3)	2 (0-7)	2 (0-9)
C	1 (0-12)	7 (0-10)	0 (0-11)	1 (0-10)	0 (0-5)	0 (0-10)	5 (0-16)

* Scores defined in Table 1. The total score for the four feet for each anatomical site was obtained by adding the respective scores of hooves, toes, and dew claws.

Table 3: Odds ratios (OR) and 95% confidence intervals (CI) for herd-adjusted associations between sow parity group (PG)* and total lesion score on heel, overgrown dew claws, and overgrown toes.

Foot site	**PG ≥ 2 versus PG1 and PG3 versus PG ≤ 2**	**PG ≥ 2 versus PG1**	**PG3 versus PG ≤ 2**
	OR (95% CI)	**OR (95% CI)**	**OR (95% CI)**
Heel†	1.10 (1.06-1.14)	NA	NA
Overgrown dew claws†	1.20 (1.19-1.26)	NA	NA
Overgrown toes‡	NA	1.15 (1.06-1.20)	1.26 (1.07-1.40)

* PG1, parity 1 or 2; PG2, parities 3-5; and PG3, parities ≥ 6.

† The assumption of proportionality in the odds is valid.

‡ The odds ratios are not constant across PGs because the assumption of proportionality in the odds is not valid.

NA = not applicable.

factor score 5. It was 1.40 times more likely ($P = .005$) that a sow would have a higher lameness score for a unit increase in factor score 7. It was 1.50 times more likely ($P = .001$) that a sow would have a higher lameness score for a unit increase in factor score 13. It was 0.60 times less likely ($P = .006$) for a sow to be lame for a unit increase in factor score 11.

Box 1: Factor scores* included in the final model for lameness, representing the lesion scores† for the foot sites examined in 386 sows in Herd C

Factor score 1, for toe length and white-line and sole lesions, rear hooves
Factor score 2, for dew-claw length, front hooves
Factor score 5, for dew-claw length, rear hooves
Factor score 7, for toe length, front hooves
Factor score 11, for wall lesions, front hooves
Factor score 13, for white-line lesions, front hooves

* Scoring of lesions at the seven foot sites considered (Table 1) resulted in 56 variables for each sow examined. From these multicollinear variables, a smaller set of independent variables (factors) were extracted using factor analysis. The regression method was used to produce factor scores for these factors.

Discussion

This study is part of a greater project aiming to characterize foot health and improve sow longevity in Greek swine herds with managerial and nutritional interventions. In the first part of the project, presented here, we estimated the frequency and severity of foot lesions and associated lameness in three herds with general management and housing typical of that in most Greek herds. We initially developed and documented a scoring system for lesions and lameness which was similar to those used in previous reports, with some modifications. Almost every sow examined in the three herds had at least one lesion, and the most frequent and severe were the heel lesions and the overgrown toes and dew claws. Likewise, other studies also recorded an extremely high frequency of foot lesions in sows.[9,10] Heel lesions and hoof wall cracks were the most common,[9,28-30] whereas the most severe lesions were detected on the heel and the dew claws.[10] We found a positive association between parity and severity of lesions. Older sows were more likely to have severe heel lesions and overgrown toes and dew claws. Hoof abnormalities occurred more frequently and were more severe in older sows,[1,10,17] although a reverse effect has also been reported,[9] probably due to the differential culling rate of affected sows. Since the heel bulb, mainly of the lateral digits, carries most of the sow's weight,[31] and high-parity sows, on average, weigh more than younger sows, the heel area is stressed more in older than younger sows. Furthermore, the mean rate of hoof horn growth in sows was recently estimated at approximately 6.3 mm and the mean wear rate at approximately 5.1 mm per month.[32] Therefore, toe overgrowth may occur simply as a function of age, especially when sows are not provided with enough space for exercise. Formation of hoof horn is a complex and structured process of cellular changes that transform living, highly functional epidermal cells into mechanically very stable horn cells. This process of horn formation is sensitive to nutritional influences, hormones, and environmental factors.[33,34]

In general, lameness is considered a multifactorial phenomenon with several physiological causes (infectious and non-infectious) affecting various tissues and anatomical structures.[35] There is evidence that some types of foot lesions cause lameness and poor reproductive performance.[10,17] The link between foot lesions and lameness is believed to be pain mediated.[36] Typically, the location[9] and severity of lesions[13] are important factors. However, several relevant studies have either failed to demonstrate a significant association[37] or identified few specific foot lesions (ie, white-line lesions, overgrown toes) associated with lameness.[9,38,39] In our attempt to associate foot lesions with lameness, we employed factor analysis, which handled the limitations and complications involved in the simultaneous evaluation of a large number of variables, many of which were correlated. We were able to identify a causal interface between various foot lesions and lameness scores. Some lesions affected lameness scores when they were combined (factor score 1), whereas others had a discerned effect according to their location. Lesions located on five sites of the foot, namely the white line, sole, wall, and overgrown toes and dew claws, were associated with lameness. Furthermore, the concurrent presence of lesions on more than one foot site, on the same or a different foot had a multiplicative effect on the likelihood of lameness. It is understandable that severe white-line and sole lesions can affect some gait parameters in sows.[30] Since the white line is the junction of wall and sole horn, injuries on that site may easily facilitate the invasion of bacteria into the corium, causing pain and inflammation. This can lead to locomotor disorders in sows[9] and in cows – white-line disease.[40] According to the experience obtained in this study, lesions on the white line of a hoof were frequently accompanied by lesions on the sole, since these two sites are adjoined. The prominent clinical sign of locomotion disorder associated with long toes was a gait abnormality that has been described as "goose-stepping of rear legs."[39] Severe overgrowth of toes and dew claws was associated with lameness[10,38] and was reported to be the most common foot lesion responsible for culling.[1] When sows are kept on fully or partially slatted floors, overgrown toes and dew claws may be caught between slats. When the animal attempts to move they may be cracked, and dew claws especially may be completely ripped off. Furthermore, overgrown dew claws may be concave and extend beneath the heel bulb, which is thereby traumatized. Thus, bacteria can enter the corium, causing infection and pain. These observations may explain why sows with long dew claws were more likely to be lame. Therefore, regular trimming of dew claws, which grow along with the toes but do not normally touch the ground to wear, may be a valuable measure to mitigate the risk of lameness.[41]

The results of our study are limited to the extent that recording and scoring of lesions and lameness were conducted by farm personnel. Although there were training sessions for lesion characterization and

Table 4: Odds ratios (OR) and 95% confidence intervals (CI) for parity-adjusted associations between factor scores and lameness for 386 sows in one Greek farrow-to-finish herd (Herd C)*

Factor scores	Lameness score ≥ 1 versus 0 and score 2 versus score ≤ 1	Lameness score ≥ 1 versus score 0	Lameness score 2 versus score ≤ 1
	OR (95% CI)	OR (95% CI)	OR (95% CI)
1†	NA	2.90 (2.10-4.00)	1.80 (1.30-2.40)
2†	NA	1.20 (0.90-1.60)	1.90 (1.02-3.00)
5‡	1.70 (1.30-2.30)	NA	NA
7‡	1.40 (1.10-1.70)	NA	NA
11‡	0.60 (0.40-0.80)	NA	NA
13‡	1.50 (1.20-1.90)	NA	NA

* Factor scores defined in Box 1. Lameness scored from 0 (non-lame) to 2 (complete inability to bear weight on one or more feet).

† The odds ratios are not constant across lameness scores because the assumption of proportionality in the odds is not valid.

‡ The assumption of proportionality in the odds is valid.

NA = not applicable.

lameness diagnosis by the personnel, and the validity of a subsample of the recordings was verified by one of us (ML), there were differences among herds. These differences were due not only to the unavoidable imperfect validity and repeatability of personnel scorings, but also to the existing variations in management, productivity, and genetic lines of sows. In two of the three herds (herds A and B), primarily managerial decisions for quick culling of sows with locomotor problems, and secondarily limited ability to detect lame sows, resulted in very low frequencies of lame sows. Using the data from the third herd, we identified significant associations between several foot lesions and lameness score. Our analytical approach was able to identify groups of closely related foot lesions among a larger set of 56 variables describing lesions on the feet of each sow, without losing any important information, and minimizing the possibility of finding associations "due to chance alone."[21] We showed that the degree of lameness was affected by a causal interface among various foot lesions. Although generalization of these results is risky, since the data originated from one herd, when combined with the results of other studies[10] they point out the need for general improvement in foot health. Though housing conditions and management on the farm are crucial as immediate causes for development of foot lesions,[37] trace-mineral nutrition should also be considered a predisposing factor, because it is vital in developing foot structure and integrity.[42] Proper nutrition with supplementation of proteinated trace mineral may improve the quality of the hoof horn tissue and reduce its susceptibility to chemical, physical, or microbial damage from the environment.[42] It should, therefore, very likely be part of managerial changes required for transition from individual to loose housing of pregnant sows.

Implications

- Under the conditions of this study in three Greek herds, sow foot lesions are extremely common.
- Older sows are more likely than younger sows to have heel lesions and overgrown toes and dew claws.
- The degree of lameness in sows may be affected by a causal interface among foot lesions.

Conflict of interest statement

There are no conflicts of interest professionally or financially with this manuscript, to the knowledge of the co-authors.

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Effect of fenbendazole on shedding and embryonation of *Ascaris suum* eggs from naturally infected sows

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Summary

Objectives: To determine reduction of *Ascaris suum* egg shedding and ovicidal effects in naturally infected commercial female breeding swine treated with fenbendazole.

Materials and methods: Five shedding and three embryonation experiments across three commercial sow farms were conducted. *Ascaris suum*-infected sows were allocated to four treatments: untreated controls; 545.5 mg fenbendazole, 1 day (Treatment 1); 545.5 mg fenbendazole, 3 consecutive days (1636.5 mg total) (Treatment 2); and 1636.5 mg fenbendazole, 1 day (Treatment 3). Fecal samples were collected and evaluated by a standard flotation method (shedding study) or eggs were isolated and incubated (embryonation study) to determine embryonation rates. Groups were compared for time-to-negative (Kaplan-Meier survival analysis); percent negative (chi-square analysis); environmental burden (analysis of variance); and embryonation rates (analysis of variance).

Results: Time-to-negative ranges were 9.3-13.1, 8.9-13.1, and 9.8 days post treatment (DPT) for treatments 1, 2, and 3, respectively; control ranges were 13.4-28.2 DPT. Treatment sows were 90%-100% negative, compared to 0.0%-28.6% of controls. Environmental burden ranges were 7.0%-60.9%, 13.9%-60.8%, and 29.3% (treatments 1, 2, and 3, respectively) and 60.4%-219.0% for controls. All treatment values differed from controls ($P < .05$), but not from each other. Embryonation rates were lower for treatments than controls at 6 and 8 DPT ($P < .001$).

Implications: Fenbendazole at various dosages is effective against *A suum* infections in sows. Treatment should begin 14 days prior to movement into clean farrowing facilities. Under the conditions of this study, fenbendazole demonstrates ovicidal activity against *A suum* at 4-8 DPT.

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Resumen - Efecto del fenbendazol en la excreción de huevos del *Ascaris suum* y la formación del embrión en hembras naturalmente infectadas

Objetivos: Determinar la reducción de la excreción de huevos *Ascaris suum* y de los efectos ovicidas en hembras comerciales infectadas tratadas con fenbendazol.

Materiales y métodos: Se realizaron cinco experimentos de excreción y tres de formación de embrión en tres granjas comerciales de hembras. Las hembras infectadas con el *Ascaris suum* fueron asignadas a cuatro tratamientos: control sin tratamiento; 545.5 mg de fenbendazol por 1 día (Tratamiento 1); 545.5 mg de fenbendazol por 3 días consecutivos (1636.5 mg en total) (Tratamiento 2); y 1636.5 mg de fenbendazol por 1 día (Tratamiento 3). Se recolectaron muestras fecales y se evaluaron por medio del método de flotación estándar (estudio de excreción) o se aislaron los huevos y se incubaron (estudio de formación de embrión) para determinar los índices de formación de embrión. Se compararon los grupos de tiempo a negativo (análisis de supervivencia Kaplan-Meier); porcentaje negativo (análisis de chi-cuadrado); carga medioambiental (análisis de varianza); e índices de formación de embrión (análisis de varianza).

Resultados: Los rangos de tiempo a negativo fueron 9.3-13.1, 8.9-13.1, y 9.8 días post tratamiento (DPT por sus siglas en inglés) para los tratamientos 1, 2, y 3, respectivamente; los índices de control fueron 13.4-28.2 DPT. Las hembras en tratamiento fueron 90%-100% negativas, comparadas con un 0.0%-28.6% del grupo control. Los índices de carga medioambiental fueron 7.0%-60.9%, 13.9%-60.8%, y 29.3% para los tratamientos 1, 2, y 3, respectivamente, y 60.4%-219.0% para control. Todos los valores de los tratamientos difirieron del control ($P < .05$), pero no entre ellos. Los índices de formación de embrión fueron más bajos en los tratamientos que en control a 6 y 8 DPT ($P < .001$).

Implicaciones: El fenbendazol en diferentes dosis es efectivo contra las infecciones de *A suum* en hembras. El tratamiento debe comenzar 14 días antes del cambio a instalaciones de maternidad limpias. Bajo las condiciones de este estudio, el fenbendazol demuestra actividad ovicida contra el *A suum* entre 4-8 DPT.

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Résumé - Effet du fenbendazole sur l'excrétion et l'embryonnation d'œufs d'*Ascaris suum* provenant de truies naturellement infectées

Objectifs: Déterminer la réduction d'excrétion d'œufs d'*Ascaris suum* et les effets ovicides chez des truies d'élevages commerciaux naturellement infectées traitées avec du fenbendazole.

Matériels et méthodes: Cinq essais en lien l'excrétion et trois avec l'embryonnation ont été réalisés dans trois fermes commerciales de reproduction. Des truies infectées par *A suum* ont été réparties dans quatre groupes de traitement: témoin non-traité; 545,5 mg de fenbendazole pour 1 journée (Traitement 1); 545,5 mg de fenbendazole pour 3 jours consécutifs (1636,5 mg au total) (Traitement 2); et 1636,5 mg de fenbendazole pour 1 journée (Traitement 3). Des échantillons de fèces furent prélevés et évalués par la méthode standard de flottaison (étude sur l'excrétion) ou les œufs furent isolés et incubés (étude sur l'embryonnation) afin de déterminer les taux d'embryonnation. Les groupes ont été comparés quant au délai pour devenir négatif (analyse de survie Kaplan-Meier); le pourcentage d'animaux négatifs (chi-carré); la charge environnementale (analyse de variance); et taux d'embryonnation (analyse de variance).

Résultats: L'écart des délais pour devenir négatif étaient de 9,3-13,1; 8,9-13,1; et 9,8 jours post-traitement (JPT) pour les traitements 1, 2, et 3, respectivement; l'écart pour le groupe témoin était de 13,4-28,2 JPT. Les taux de négativité variaient entre 90%-100% pour les truies traitées, comparativement à 0%-28,6% pour les témoins. Les charges environnementales variaient entre 7,0%-60,9%; 13,9%-60,8%; et 29,3% pour les traitements 1, 2, et 3 respectivement, et 60,4%-219,0% pour les témoins. Les valeurs des groupes traitées différaient toutes significativement ($P < 0,05$) de celles du groupe témoin mais pas entre elles. Les taux d'embryonnation étaient moindres à 6 et 8 JPT pour les groupes traités comparativement aux témoins ($P < 0,001$).

Implications: L'administration de fenbendazole à différents dosages est efficace contre une infection par *A suum* chez les truies. Le traitement devrait débuter 14 jours avant le déplacement dans des installations de mise-bas propres. Dans les conditions expérimentales de cette étude, le fenbendazole a démontré une activité ovicide contre *A suum* à 4-8 JPT.

Ascaris suum, the large roundworm of swine, is the most common and important parasite of swine, with worldwide distribution.[1,2] The main economic impacts of *A suum* on swine are reduced feed efficiency,[3,4] decreased average daily gain,[5-7] liver condemnations at slaughter due to larval migration and organ pathology ("milk spots"),[8-10] and increased medication costs related to treatment.[11,12]

Additional *A suum* impacts include suboptimal vaccine efficacy associated with migrating larvae;[13,14] organ damage from larval migration, predisposing the host to secondary diseases;[15-24] morbidity and mortality associated with acute infection;[25-27] and impacts on carcass and meat quality.[28,29] *Ascaris suum* may be a zoonotic infection in areas where humans and swine cohabitate.[30-33]

Significant changes in the way most modern swine are managed, such as housing pigs indoors, separation of ages and production phases, improved sanitation, and effective anthelmintic use, have either eliminated or significantly reduced parasite incidence and severity. Due to reduced prevalence and overdispersion within populations,[34] parasites cause mainly a subclinical disease and not often considered to be of major importance in modern swine production.[12,35] However, due to several *A suum* characteristics, the parasite still persists in modern production systems.[1,36-39] Those characteristics are a highly fecund adult female (estimated to produce 1 to 2 million eggs per day for up to 55 weeks),[40,41] eggs that are highly resistant to environmental conditions and disinfectants,[42-44] a direct life cycle involving extra-intestinal migration,[45] and a relatively short pre-patent period.[45] The high environmental contamination level and egg resistance make it nearly impossible to eradicate *A suum* from contaminated facilities, thus continuous monitoring and implementation of control measures are required.[35,44]

Recent husbandry requirements driven by concerns other than parasite control, such as group sow housing, required provision of nesting or bedding materials, and drug use restrictions, are re-introducing known risk factors and creating exposures that promote *A suum* transmission, resulting in increased prevalence and clinical severity within those farm types.[46-49] In addition, niche production systems (eg, organic, pasture-raised, differentiated markets) and swine associated with regional and national exhibition show circuits provide environments for the parasite's maintenance.[50-54]

The goal for most parasite control programs is to minimize clinical disease and production impact through preventing transmission and reducing environmental contamination.[35,55-58] This is accomplished by improved sanitation, management of pig flow, and use of anthelmintics at key times in the parasite's life cycle and host's production cycle. On breeding farms, sanitizing the farrowing facilities before loading sows and removal of organic material on the skin of sows by washing prior to farrowing are often implemented as control measures.[44,55-57] Another common practice in the swine industry is to administer anthelmintics to pregnant sows prior to entering clean farrowing facilities.[35,44] This reduces transmission of eggs to the farrowing environment (eg, stall, pasture) and from the sow to piglets. Treatment timing prior to movement to farrowing may vary considerably, and if the period between treatment and moving into farrowing area is too short, egg shedding into the farrowing environment is not prevented.

Fenbendazole is a broad-spectrum benzimidazole (Class I) anthelmintic approved for use in swine in North America and elsewhere around the world. Fenbendazole has a high safety margin[59,60] and is highly effective against the adult and larval stages of *A suum*.[60-65] The benzimidazole class of anthelmintics has ovicidal activity against parasites of a number of species.[66-70] This activity results from the benzimidazole molecule binding with embryonic tubulin at the leading edge of polymerization, which prevents microtubule formation.[68,69,71]

Fenbendazole is currently available in North America as a feed additive (Safe-Guard Medicated Dewormer for Swine; Merck Animal Health, Summit, New Jersey) or as an individual feed top-dress (Safe-Guard

EZ Scoop; Merck Animal Health). The product is available for other species in North America and for swine in the other countries under the trade name Panacur (MSD Animal Health, Summit, New Jersey). The individual top-dress formulation enables treatment of individual animals or subpopulations (ie, weekly batches) without necessity of simultaneously medicating the entire population served by the feed system, and eliminates the need for on-farm feed mixing, which may not be available.

The use of fenbendazole in swine has been studied extensively, and its use in sow herds is common.[72] However, little information is available regarding fenbendazole's impact on shedding dynamics and embryonation of *A suum* eggs post treatment under common commercial conditions. Judicious drug use would be supported by evidence of the magnitude of impact on shedding, shedding duration, and time to negative shedding post treatment under commercial conditions. Understanding the dynamics of reduced environmental contamination is of benefit in developing effective control measures for *A suum* in commercial swine operations worldwide. Further knowledge on controlling *A suum* in swine may be used to model *Ascaris lumbricoides* control in humans.[31,73]

The series of experiments presented herein were conducted to characterize the impact of fenbendazole (Safe-Guard EZ Scoop) on *A suum* egg shedding and embryonation rates for *A suum* eggs that were shed from naturally infected gestating sows under commercial conditions that included commonly used anthelmintic protocols.

Materials and methods

All animals were cared for in accordance with the Guide for the Care and Use of Agricultural Animals in Research and Teaching (**http://www.fass.org/docs/agguide3rd/Ag_Guide_3rd_ed.pdf**) and Pork Quality Assurance Plus (PQA Plus) guidelines (**http://www.pork.org/Certification/2341/pqaPlusMaterials.aspx**).

Farms

The farms in which all experiments (five shedding and three embryonation experiments) were conducted were sow farms in a commercial swine production company, using Large White-Landrace cross maternal genotypes, between 2010 and 2013. Farms A, B, and C were selected because they had each been previously diagnosed with *A suum* infection in breeding sows as part of a system surveillance study reported by Pittman et al.[37] Farm A was a 1000-sow, one-site, breed-to-market operation located in southeastern Virginia. Farms B and C were each 2000 breed-to-feeder-pig operations (feeder pigs 10 weeks of age), both located in northeastern North Carolina. All farms were managed with weekly batches of gestating, multiparous sows in which sows were housed in individual gestation and farrowing stalls, weaning pigs at approximately 3 weeks of age. Replacement gilts, purchased from an off-site multiplication flow, were housed in groups of four to six until bred, when they were moved into individual gestation stalls. All farms utilized fenbendazole (Safe-Guard EZ scoop) as a feed top-dress weekly for gestating sow groups 2 weeks prior to farrowing. All routine fenbendazole use was suspended prior to starting and for the duration of the experiments. Animals were fed individually through automated drop boxes once daily, and individual water nipples were available for each animal at all times.

Experiments

Egg shedding. Five separate shedding experiments were conducted among the three farms between February 2011 and December 2013: one each at Farm A and Farm B and three at Farm C. Each experiment differed in number of subjects, sample collection dates, and treatments, as detailed in Table 1. All subject enrollment, sample collection, sample processing, and application of treatments were consistent among shedding experiments. No animals were used in more than one shedding experiment.

Embryonation. Three separate embryonation experiments were conducted between January 2012 and December 2013. Each experiment differed in number of subjects, sample collection dates, and treatments, as detailed in Table 2. All subject enrollment, treatment applications, sample collection, sample processing, and embryonation evaluation were consistent among experiments. No animals were used in more than one embryonation experiment.

Sow inclusion and exclusion criteria. Inclusion and exclusion criteria were consistent for all experiments. On the basis of reported literature and previous observations in the herd, younger animals (gilts through second-parity sows) were initially screened for *A suum* infection status, because of a higher expected prevalence of infection.[74-76] The goal was to identify gestating adult females shedding *A suum* eggs in their feces. In order to screen a large number of animals in an efficient and rapid manner, a modified fecal flotation method was utilized. Approximately 1 gram of feces was collected directly from the rectum of selected gilts and sows. A new clean nitrile glove was used for each animal to prevent sample cross-contamination. Each fecal sample was placed in a 15-mL centrifuge tube, pre-filled with 5 mL of a concentrated sugar solution (Sheather's solution).[77] Samples were processed immediately on site. Fecal samples and sugar solution were manually homogenized within the 15-mL tube by vigorous shaking. Supplemental homogenization, if required, was accomplished using a disposable wooden stirrer, discarded after a single use. A volume of sugar solution was added to each tube sufficient to create a reverse meniscus at the top of the tube. A 22 × 22-mm glass coverslip was placed on top of each tube and allowed to sit for 10 minutes minimum. Coverslips were removed, placed on glass microscope slides, and examined under 40× magnification for *A suum* eggs. Each coverslip was examined until confirmation of at least one egg was observed or no ova were visualized on the entire coverslip, ie, the sample was determined to be negative. In all experiments, animals providing positive test samples were enrolled in the study and randomly allocated using a random number generator to treatment and control groups, while animals with negative test samples were excluded from enrollment. For all enrolled animals, a day 0 fecal sample was collected and eggs per gram were quantified using the modified Wisconsin sugar flotation technique.[78] Animals with a negative day 0 fecal test were considered to have been "false positives" on initial screening and were excluded from the remainder of the experiment.

Treatments. Treatments were applied in a manner consistent with existing on-farm processes and were consistent for all experiments. Safe-Guard EZ Scoop was used for all fenbendazole treatments, and the scoop provided in the package was used for subject dose allotments. The scoop when level full provides approximately 545.5 mg of fenbendazole, sufficient to provide 3 mg per kg bodyweight to a 181.8-kg animal. In common field application, a level full scoop is provided to each sow, regardless of bodyweight, and treatments were based on this

Table 1: Five shedding experiments comparing fenbendazole treatment dosages and treatment periods for *Ascaris suum* in naturally infected gestating sows*

Experiment	Farm	Treatment	n	Fecal sample collection days	No. negative (%)	Time-to-negative (days)			Mean BURD (%)
						Mean	SE	Range	
1	A	CNT	4	0, 4, 5, 6, 8, 10, 12, 14, 16, 18, 20	1 (25.0)[a]	18.0[a]	NA	18-20	136.6[a]
		TX1	7		7 (100.0)[b]	13.1[b]	0.9	10-16	46.4[b]
		TX2	10		10 (100.0)[b]	11.0[b]	1.2	4-18	36.1[b]
2	B	CNT	5	0, 8, 10, 14	0 (0.0)[a]	14.0[a]	NA	14	79.9[a]
		TX1	6		6 (100.0)[b]	9.3[b]	1.0	8-14	60.9[b]
		TX2	10		9 (90.0)[b]	10.6[b]	0.8	8-14	60.8[b]
3	C	CNT	10	0, 8, 10, 12, 14, 21, 24, 31	3 (30.0)[a]	22.3[a]	1.6	10-24	219.0[a]
		TX1	13		13 (100.0)[b]	11.7[b]	0.4	10-14	44.4[b]
		TX2	12		11 (91.7)[b]	11.3[b]	0.5	8-31	33.1[b]
4	C	CNT	14	0, 4, 8, 10, 14, 22, 30, 37	3 (21.4)[a]	28.2[a]	1.5	14-37	60.4[a]
		TX1	29		28 (96.6)[b]	11.5[b]	0.7	4-22	7.0[b]
		TX2	26		26 (100.0)[b]	13.1[b]	1.0	4-22	13.9[b]
5	C	CNT	7	0, 4, 6, 8, 10, 14, 21	2 (28.6)[a]	13.4[a]	0.8	10-21	118.6[a]
		TX1	12		12 (100.0)[b]	9.5[b]	0.6	6-14	32.4[b]
		TX2	9		9 (100.0)[b]	8.9[b]	0.8	6-14	20.6[b]
		TX3	11		11 (100.0)[b]	9.8[b]	0.7	8-14	29.3[b]

* In five studies across three commercial sow farms, gestating sows (F1 Large White × Landrace, multiparous; n = 185) positive for *A suum* were each randomly allocated to one of four treatment groups: untreated controls (CNT); 545.5 mg fenbendazole (Safe-Guard EZ Scoop; Merck Animal Health, Summit, New Jersey) given on a single day (TX1); 545.5 mg fenbendazole given on each of 3 consecutive days (1636.5 mg fenbendazole total) (TX2); and 1636.5 mg fenbendazole given on a single day (TX3). Fecal samples were collected at various time points within each experiment for evaluation by fecal flotation and egg quantification. Treatment groups were compared by number negative at end of study period; time-to-negative (time to stop shedding) by survival analysis; and environmental burden (BURD), a measure of total egg shedding during the study.

[a,b] Values with different superscripts within a category and within an experiment are statistically significant ($P < .05$): number negative, chi-square with Fisher's exact test; mean time-to-negative, Kaplan-Meier log-rank test; mean environmental burden, analysis of variance.

n = number of sows by experiment-treatment; SE = standard error; NA = not applicable.

methodology. Therefore, a single scoop was estimated to provide 545.5 mg fenbendazole for each animal.

Treatment groups for all experiments were as follows: non-treated control; a single 545.5-mg scoop of fenbendazole for 1 day (Treatment 1); a single 545.5-mg scoop of fenbendazole daily for 3 consecutive days, total 1636.5 mg (Treatment 2); or three 545.5-mg scoops of fenbendazole on a single day, total 1636.5 mg (Treatment 3).

Treatment was applied to sows' individual feed drop boxes the day prior to feed being dispensed the following morning. Anthelmintic intake by each sow was monitored as complete intake of feed prior to the next feeding, and all animals in the studies consumed their entire daily ration.

Sample collection and processing

Egg shedding experiments. At each sample day, approximately 5 grams of feces was collected per rectum of each enrolled sow. If a fecal collection from a sow failed after two attempts, that sow was excluded from the day's collection. After collection, fecal samples were placed into 50-mL screw-top centrifuge tubes and held at 4°C until processing within 24 to 72 hours after collection. Samples were processed using the modified Wisconsin sugar flotation method.[78] Slides were evaluated using 40× magnification, and the number of *A suum* eggs on the entire slide was counted. A maximum egg count per slide ("cutoff") of 500 eggs was utilized.

Embryonation experiments. Samples in Experiment 1 were collected on day 8 after the start of treatment. Samples in Experiment 2 were collected prior to treatment (day 0) and on day 8 after the start of treatment. Samples in Experiment 3 were collected on day 0, and on days 2, 4, and 6 after the start of treatment.

On each collection day, feces (approximately100 to 500 g) was collected per rectum in a clean plastic sample bag in a manner consistent with the egg-shedding experiment. Samples were held at 4°C when not being processed. Samples were processed in a manner to obtain a large number of *A suum* eggs, not for fecal quantification. Processing and sample embryonation was based on several reported methodologies[34,70,79-81] and equipment availability. *Ascaris suum* eggs were isolated using a method similar

Table 2: Three embryonation experiments on a sow farm comparing three fenbendazole treatment dosages and treatment periods for *Ascaris suum* in naturally infected gestating sows*

		Treatment group								
		CNT		TX1		TX2		TX3		
Experiment	DPT	n	ER (SD)	n	ER (SD)	n	ER (SD)	n	ER (SD)	*P*
1	8	11	95.4[a] (8.6)	18	29.3[b] (24.2)	10	30.5[b] (18.2)	ND	ND	< .001
2	0	13	92.8[a] (13.3)	25	92.5[a] (17.4)	21	85.0[a] (17.9)	ND	ND	NS
	8	11	95.6[a] (8.6)	18	29.4[b] (24.0)	11	38.2[b] (21.2)	ND	ND	< .001
3	0	7	98.6[a] (2.9)	12	97.8[a] (2.9)	9	94.0[a] (5.6)	11	97.1[a] (4.2)	NS
	2	7	99.1[a] (1.1)	10	99.0[a] (1.5)	9	93.1[a] (10.9)	11	95.9[a] (8.7)	NS
	4	7	99.3[a] (0.8)	12	75.4[a] (31.0)	8	70.9[a] (20.1)	8	47.0[b] (34.1)	< .01
	6	7	90.3[a] (21.3)	12	48.6[b] (20.9)	8	28.6[b] (26.1)	11	26.6[b] (28.9)	< .001

[1] In three studies on a commercial sow farm, gestating sows (n = 137; described in Table 1) positive for *A suum* were each randomly allocated to one of four treatment groups (described in Table 1). Fecal samples were collected at various time points by experiment, and *A suum* eggs were isolated and incubated to determine embryonation rates. Treatment groups were compared by mean embryonation rates as a percent of ova reaching full larval development.

[a,b] Values within a row with different superscripts are statistically different (Tukey's studentized range test).

DPT = days post treatment; ER = embryonation rate (percent); SD = standard deviation; ND = not done; NS = not significant (*P* > .05).

to the modified Wisconsin sugar flotation method, but adjusted for a large sample volume as follows. Samples were homogenized by hand within the collection bags, and a 100-gram sub-sample was weighed out and placed in a 1-L plastic container. Feces was then mixed with 200 mL of tap water and homogenized in the container using a kitchen potato masher. The fecal-water homogenate was then strained through a large tea strainer into a second 1-L plastic container to remove large-particle organic material. Up to 200 mL of the strained contents was then poured into 200-mL dilution bottles. The bottles were centrifuged at 145*g* for 10 minutes in a large bucket centrifuge. The pellet was re-suspended in Sheather's solution to a volume of 200 mL, which was then centrifuged at 145*g* for 10 minutes. The suspension was allowed to stand for a minimum of 10 minutes, then the top 10 to 15 mL was poured off into 50-mL conical centrifuge tubes. Tap water was added up to 45 mL and the suspension was homogenized manually by vigorous shaking. The tube was centrifuged at 145*g* for 10 minutes, then the pellet was re-suspended in 30 mL of 0.1 N H_2SO_4 and transferred into a 50-mL filtered top culture flask (item #10062-872; VWR Radnor, Pennsylvania). Egg concentration was evaluated by counting eggs in a 10-µL sub-sample at 40× magnification. Samples with > 25 eggs per µL were diluted with 0.1 N H_2SO_4 to achieve this maximum concentration, as it has been reported that egg density influences development.[82] Culture flasks were held at 4°C until embryonation incubation was initiated for all samples within an experiment.

The embryonation period occurred independently for each experiment, once all samples from all collection days were processed. When all samples within an experiment had been processed, culture flasks were simultaneously incubated at room temperature (approximately 23°C to 25°C) in the dark for 60 days to ensure complete larval development.[79] Flasks were agitated by hand three to four times per week for aeration. At the end of 60 days, flasks were held at 4°C until embryonation rates could be evaluated.

To calculate embryonation rates, culture flasks were shaken and a 10-mL subsample was poured into a 15-mL conical centrifuge tube and centrifuged at 145*g* for 10 minutes. The pellet was re-suspended in 5 mL of Sheather's solution. A volume of Sheather's solution was added in order to create a meniscus at the top of each tube. A 22 × 22 mm glass coverslip was placed on top of each tube for a minimum of 10 minutes. Coverslips were removed and placed on a glass microscope slide then examined under 100× magnification for *A suum* eggs presence and embryonation stage. The first 100 *A suum* eggs observed were evaluated and determined to be either fully embryonated (larvae visualized) or unembryonated, including any stage of development from one-cell to pre-larval stages.[83] If fewer than 100 eggs were visualized on a coverslip, additional 10-mL sub-samples were processed until 100 total eggs were counted. Failure to count 100 total eggs from a culture flask resulted in exclusion of that sample from the experiment. Percent of embryonation was recorded as the number of eggs containing fully embryonated larvae out of 100 eggs.

Statistical analysis

All statistical analyses were performed using Enterprise Guide 5.1 software (SAS Institute, Inc, Cary, North Carolina). In each case, sow was considered the experimental unit. The dependent variable in each shedding experiment was the duration of fecal *A suum* egg shedding, measured as the time to first negative fecal exam for experimental sows. Survival analysis with log-rank test (PROC LIFETEST) was used and the model included treatment group, farm (in the case of experiments), parity, and treatment-farm interaction fixed effects, and the random effect of sow within farm by treatment group. Through backward elimination, effects determined non-significant on the basis of analysis were dropped from further analysis. Comparisons between treatment groups were made using Kaplan-Meier methods as an

estimation for survival function from lifetime data. Mean survival times (± standard error) were estimated for each treatment. Additionally, the percentage of animals that produced a negative fecal exam by the end of the study was evaluated using chi-square and Fisher's exact test methods. A calculated mean environmental burden value for each treatment group was evaluated using analysis of variance (ANOVA) methods to evaluate treatment effects, and treatment means were separated using Tukey's studentized range test. The model implemented included treatment group, farm, parity, and treatment-farm interaction fixed effects. The environmental burden calculation was an attempt to compare observed eggs per gram of feces (EPG) excreted (EPG_{obs}) for the duration of the study period compared to an expected eggs excreted (EPG_{exp}) for each sow and between treatments as a measure of the potential environmental contamination provided by each group. The EPG_{exp} value was that individual's initial (day 0) EPG multiplied by the number of study days. The EPG_{obs} value was calculated as the cumulative sum of EPG on one sampling day (D_i) times the number of days until just prior to the next sampling day (D_{i+x}) through the duration of the study $[\Sigma(EPG_{Di} \times (D_{i+X} - D_i))]$. For example, in an animal with sample day EPG values of 100, 80, and 20 on days 0, 4, and 8 respectively, the EPG_{exp} for this animal is 900 [100 $EPG_{Day0} \times 9$ total days], the EPG_{obs} is 740 [(100 $EPG_{Day0} \times 4$ days)+(80 $EPG_{Day4} \times 4$ days) + (20 $EPG_{Day8} \times 1$ day)], and the environmental burden is 0.822 (740 EPG_{obs} per 900 EPG_{exp}). This environmental burden estimate indicates that the individual shed 82.2% of the eggs over the study duration as was anticipated for that individual.

For embryonation rates, ANOVA methods were used to evaluate treatment effects. When ANOVA effects were significant, treatment means were separated using Tukey's studentized range test. Analyses were conducted separately for each embryonation experiment because of differences in the sampling protocol followed. In each case, sow was considered the experimental unit. The dependent variable in each study was the percent embryonated *A suum* eggs (mean embryonation rate), measured as the number of eggs with visible larvae from the 100 eggs evaluated. Independent variables considered were treatment, sampling day, and a treatment-sampling day interaction.

Results

The number of sows in each experiment and between treatment groups varied considerably due to the method of enrollment described, in which a rapid and efficient survey of a large number of sows (eg, 100 to 250) was conducted.

Egg-shedding experiments

In shedding Experiment 1, there were no recorded samples above 500 eggs per gram. In Experiment 2, two Treatment 1 samples and three Treatment 2 samples were above 500 eggs per gram at day 0. In Experiment 3, a single Control sample at days 8 and 10 and one Treatment 2 sample at day 0 exceeded 500 eggs per gram. In Experiment 4, eleven control samples exceeded 500 eggs per gram, with 10 of the 11 coming between days 8 and 22 of sampling, while 10 Treatment 1 and six Treatment 2 samples at day 0 exceeded 500 eggs per gram. One Treatment 1 day 8 sample exceeded 500 eggs per gram in Experiment 4. In Experiment 5, one Treatment 2 and one Treatment 3 day 0 sample each exceeded 500 eggs per gram, as did one Control sample on days 8, 10, and 21.

In the survival analyses, there were significant farm (experiment) effects ($P < .001$) and evidence of a trend in farm-treatment interaction ($P = .054$) in the full model; therefore, data was analyzed and reported independently by experiment in a reduced model. There was no parity effect, and parity was excluded from the reduced models. In each experiment, there was a high censoring rate in the control groups (range 70.0% to 100.0%), as many subjects remained positive throughout the period of testing. Kaplan-Meier survival analysis revealed a significant difference in time-to-negative egg shedding between controls and each treatment group. In all shedding experiments, Treatment 1, Treatment 2, and Treatment 3 did not differ in time-to-negative shedding. Complete mean time-to-negative, standard error, and range for each experiment are shown in Table 1. A graph of the survival analysis has been included (Figure 1) for the combined data from experiments 1 through 5. Note that due to the different durations of experiments 1 through 5, the control group contains multiple censored data points, while the largest number of sows censored in any treatment group by experiment was one. Therefore, this graph considerably underestimates the impact of fenbendazole treatments and is a conservative assessment.

When all shedding experiments were combined, only nine of 40 control animals (22.5%) were negative for *A suum* eggs throughout the respective study periods. Among the treatment groups, 66 of 67 (98.5%) for Treatment 1, 65 of 67 (97.0%) for Treatment 2, and all of 11 (100.0%) for Treatment 3 were negative for *A suum* eggs at completion of their respective experiments. The percent negative at end of study differences between control and each treatment was significant by Fisher's exact test ($P < .001$). There was no difference in percent negative at end of study among the fenbendazole treatments in any of the five experiments. The results for each experiment are shown in Table 1.

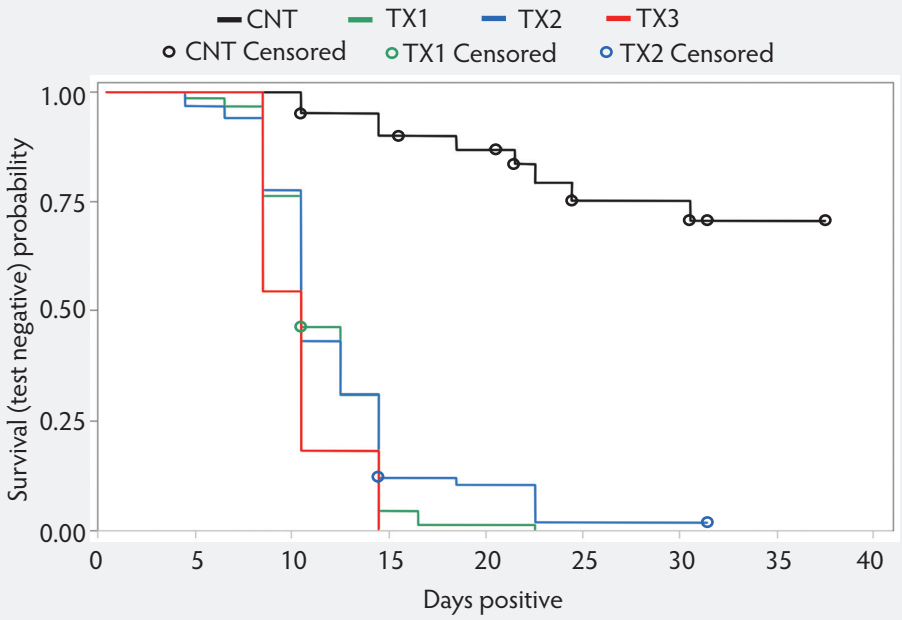
For environmental burden analysis, a significant effect of treatment ($P < .001$) and farm ($P < .001$) and a trend in the treatment-farm interaction ($P = .054$) were observed; therefore, data was analyzed and reported by experiment. The environmental burden analysis demonstrated a significant difference ($P < .05$) between control and all fenbendazole treatments in each experiment (Tukey's studentized range test). There were no differences among treatments in any of the five experiments. The environmental burden estimates for controls in experiments 1, 3, and 5 were greater than 100%, representing increasing eggs per gram counts on subsequent samplings from individual control sows. Average environmental burdens for each experiment are listed in Table 1.

It is valuable to note that during collection, most notably on days 6 and 8 post treatment, many of the treated animals were actively expelling adult ascarids (visually observed or manually extracted during rectal sample collection), presumably due to treatment effects of fenbendazole.

Embryonation experiments

In experiments 2 and 3, the independent variables of treatment, sampling day, and a treatment-sampling day interaction were significant sources of variation; therefore, analyses were conducted for each sampling day within each experiment. In all experiments, embryonation rates of controls ranged from 90.3% to 99.3%. Ovicidal activity within treatments was realized on day 4 post treatment, with significant embryonation rate reduction in Treatment 3 ($P < .01$) and numerical differences in Treatment 1 and Treatment 2 in Experiment 3. After day 4 post treatment (day 6 in Experiment 3 and day 8 in experiments 1 and 2), all treatments

Figure 1: Survival analysis: Kaplan-Meier survival curves for time-to-negative fecal *Ascaris suum* egg shedding from five experiments (data combined) across three commercial sow farms comparing three fenbendazole dosages and treatment periods to treat *A suum* in naturally infected gestating sows. Duration of treatment differed among the five experiments; therefore, in-phase censoring of control sows is over-represented. Study described in Table 1.



were significantly different ($P < .001$) from controls for each experiment. Complete embryonation rates and standard deviations for all embryonation experiments are shown in Table 2.

The only differences in embryonation rate between fenbendazole dosages were observed on day 4 post treatment in Experiment 3. A numerical difference in embryonation rate was observed in treatments 1 and 2, although it was not statistically significant when compared to controls. By day 6, a difference was no longer observed among treatments and all were different from controls.

It is important to note that during microscopic observation, development was often atypical or arrested in unembryonated eggs in samples from treated sows. Eggs commonly had unequal cleavages, satellite and clustering of blastomeres, smaller, more circular shells, a granular and crystalline appearance to the yolk, lack of any apparent development, and abnormal shapes (Figure 2).

Discussion

Statistical comparison of sow eggs per gram was not conducted due to the inherent high variability of egg counts when assessing *A suum* infection. Fecal egg counts are highly variable over time within the same subject, as well as within the same fecal passing.[44,84] Rather, consistent with the main objectives of the experiments, we assessed the time to cessation of shedding and percent negative sows at the end of respective study periods. In addition, we utilized a calculated environmental burden, which provides a better description of egg shedding, as it uses fecal eggs per gram counts over several time periods to estimate total fecal egg shedding during a period of time. Others have utilized similar calculated estimates of egg contamination. Bernardo et al[6,85] used the average eggs per gram counts to calculate a "lifetime burden" in market pigs and modeled the growth impacts of ascariasis. Mejer and Roepstorff[86] calculated a "relative contamination index" from fecal eggs per gram of *Trichuris suis* and *Oesophagostomum dentatum* in pastured pigs in an attempt to compare contamination rates between experimental paddocks.

The environmental burden calculation was utilized to demonstrate the impact of fenbendazole on reduction of total *A suum* eggs excreted into the environment, which is an important epidemiological aspect of *A suum* control. As can be noted by the higher environmental burden values of controls in shedding experiments 1, 3, and 5, non-treated animals may perpetuate or increase fecal egg shedding, and thus contamination of the environment persists over time.

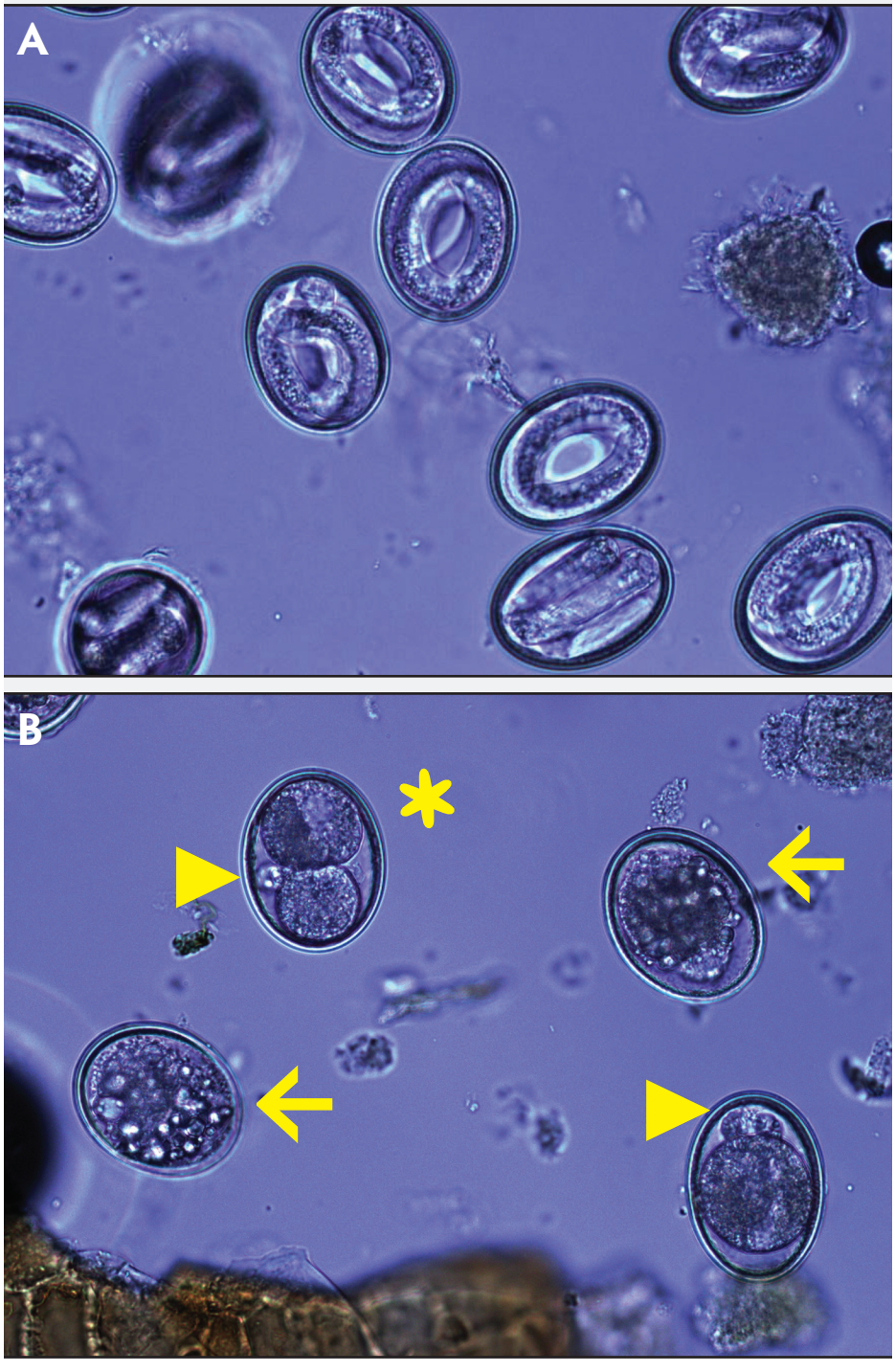
The environmental burden values in shedding Experiment 2 differed statistically between control (79.9%) and treatments 1 (60.9%) and 2 (60.8%), but with less numerical difference between the treatments than in the other shedding experiments. This is likely due to several factors, including the shorter duration of the study (14 days), the number of sampling days (4), and the bias of using a "cutoff" in initial fecal eggs per gram values.

It is important to note that in some fecal samples, eggs per gram was in excess of the 500 eggs per gram cutoff used, and thus introduced bias into the environmental burden calculation. Overall, the use of an eggs per gram cutoff in the shedding experiments resulted in an underestimation of the impact of reduced fecal shedding.

All but one of the above observations (Experiment 4, Treatment 1, day 8 sample) would have resulted in underestimation of the actual environmental burden calculation, since sample cutoff at day 0 would have resulted in a lower expected environmental burden value, and control samples cutoff post treatment would have underestimated the lower environmental burden compared to treatment groups. This could explain the lower numerical differences and reduction in control environmental burden values observed in shedding experiments 2 and 4 compared to experiments 1, 3, and 5.

False-positive results due to low fecal eggs per gram counts or coprophagia,[87] when using a highly sensitive test such as the modified Wisconsin sugar flotation technique, are potential sources of misclassification bias. It should be noted that the flotation method used was selected specifically because of its greater sensitivity (approximately 1 egg per gram) relative to other detection methodologies[78,88,89] and its common use in North American swine parasitology. In contrast, many European studies utilize a modified McMaster's technique with a low limit of detection (20 eggs per gram) and recommend a cutoff (200 eggs per gram) to minimize the false-positive effect.[44,87] For example, if a cutoff of 200 eggs per gram had been used in the present studies, approximately 50.3% of included sows (93 of 185 sows) would have been excluded for false-positive counts at the time of sow enrollment. Since

Figure 2: Micrographs of embryonated and unembryonated *Ascaris suum* eggs after expulsion in three experiments comparing the effects of three fenbendazole treatment regimens on ovicidal activity of *A suum* eggs shed from naturally infected gestating sows. Panel A: Normally developed larvae, after embryonation, from ova isolated from sows on day 0, prior to treatment with fenbendazole (99% embryonation rate); Panel B: Mix of abnormally developed and arrested eggs after embryonation; unequal cell division (asterisk), satellite blastomeres (arrowheads), and crystalline-like yolk (arrows). Samples collected day 8 after treatment with 545.5 mg fenbendazole on a single day (21% embryonation rate). Studies described in Table 1 and Table 2.



necropsy and daily adult worm recovery from enrolled sows was not practical or performed, it is impossible to classify each positive sample result as true-positive or false-positive; however, a few observations support using these data collectively as valid results. First, all sows were housed individually in partially slatted gestation stalls and had limited access to feces. Second, many enrolled females from the treatment groups passed adult ascarids, which is consistent with observations by Boes et al.[34] Unfortunately this was not recorded. Third, the apparent parasite burden (measured as eggs per gram) for sows in published papers from Europe is much greater when compared to observed eggs per gram values from the present studies and experience in other North American sow herds by the authors[36,37] (GHM, personal observation, May 2014). The reason for this discrepancy between reports and geography are not fully understood, but may be related to diagnostic methodology (the McMaster's technique uses a multiplicative calculation and tends to overestimate eggs at the higher concentrations)[88] or inherent management differences (provision of bedding), medication restrictions (anthelmintic use), farm types (multiple ages), and facility designs (group gestation), which result in an overall heavier environmental parasite burden. Another possibility is an inherent geographical variation in egg shedding by adult female parasites, such as has been documented in *A lumbricoides*.[90] Lastly, a decrease in eggs per gram over time was consistently observed throughout each treatment group within each experiment in the present study, while the majority of control animals continued to shed eggs throughout the sampling period, indicating an effect of the treatment on fecal egg shedding.

Future work could minimize the impact of false-negatives by repeated sampling and evaluation of fecal eggs per gram beyond the initial negative test. False-negatives would affect the survival analysis by overestimating the impact of the treatment on treated sows relative to the control sows. Both types of misclassification, false-positive and false-negative, may occur in subjects of both the treatment and control groups.

Some eggs collected for the embryonation experiments might have been associated with coprophagia.[87] While this type of false-positive diagnosis is important in evaluating infection prevalence and response to treatment, it may not be a significant issue in embryonation studies, since eggs resulting

from coprophagia still contribute to environmental contamination and are susceptible to the ovicidal effects of fenbendazole. Benzimidazoles have significant ovicidal activity in vitro, regardless of whether the eggs are extracted from adult worm uteri or collected from feces.[66,67,70,91] Fenbendazole is maintained at a greater in vitro concentration in the gut lumen during the treatment period due to low bioavailability (27.1%);[92] 44% to 50%[60] of the drug remains unchanged and is excreted in the feces, and therefore may have ovicidal activity in eggs in the lumen during treatment periods, although this has not been demonstrated directly. Benzimidazole's ovicidal activity can affect the embryo even after development is initiated (eg, eggs ingested from the environment after a period of development). Eggs that developed for 9 to 10 days and then were exposed to thiabendazole in vitro ceased further development.[67]

In the present study, fenbendazole began to have ovicidal effects as early as day 4 post treatment at the single-day 1636.5-mg dosage (Treatment 3), and across all treatments by day 6 through 8 post treatment. The lack of a statistical difference in embryonation rate observed in treatments 1 and 2 on day 4 may have been a result of inadequate sample size to show a difference, or may represent an early dose effect. Effect on embryonation beyond day 8 was not evaluated. Other investigators have reported rapid ovicidal effects of other helminth eggs after treatment with various benzimidazoles in sheep (8 hours)[91,93] and humans (24 to 72 hours).[70,81,94-97] The more rapid ovicidal activity in these studies, when compared to the present data, may be related to differences in parasite susceptibility, host-parasite interactions, or pharmacokinetics and pharmacodynamics of the anthelmintics for the different host species.

The observations made of atypical egg development, such as unequal cleavages, satellite blastomeres, and crystalline appearance of the yolk, were in agreement with others.[67,97,98] Microtubules are important for cell structure, proper cleavage, chromosome movement during cell division, and thus embryogenesis.[71] Affected eggs often have irregular shapes, atypical blastomeres, and unequal divisions, which result in irreversible arrested development.[34,60,67,98]

It is important to note that *A suum* eggs shed from hosts (ie, breeding sows) are not directly infective to the offspring, an important epidemiological aspect often misunderstood by producers and veterinarians.[45,58,79] Freshly shed *A suum* eggs require a developmental period in the environment outside the host, consisting of two moltings to an infective L3 larvae.[99-101] This development usually takes 1 to 3 months or longer, depending on temperature, humidity, and seasonal climate.[45,79,101-105] It is this external non-infectious developmental period that allows for basic sanitary and husbandry control measures to be effective if applied correctly. In modern swine farms, with early weaning at 3 weeks of age or less, *A suum* transmission directly from dam to offspring is unlikely, due to inadequate time for embryo development to an infectious L3. Multivariable risk factor analysis in 413 Scandinavian herds demonstrated that wean age was a significant factor in growing pigs having ascariasis.[106] Farms that weaned pigs at greater than 6 weeks of age were twice as likely to have finishers positive for *A suum*, when compared to farms that weaned between 3 and 5 weeks of age, suggesting that the additional time exposed to farrowing facilities allowed for development of *A suum* eggs to an infectious stage. In other studies, age-segregated pork production that results in moving pigs to facilities located at a distance from each other reduced the correlation between sow herd *A suum* status and the *A suum* status of grow-finish pigs originating from the same sow herd[107,108] Transmission to offspring is most likely from older on-site animals (finishing, gilts) or contaminated facilities (finisher barns, gilt development units).[109] Indirect transmission is still a concern where piglets may be exposed to infectious eggs remaining in the farrowing environment from previous groups,[109,110] poorly sanitized farrowing facilities that allow maintenance of "hot spots" as described by Nilsson,[111] or by mechanical transmission from other farm areas that are contaminated (eg, breeding, gestation, gilt development, finishers), by the sow (eg, fecal matter on skin, feet), stockpersons (hands, clothing, boots), and fomites.

Connan[103] evaluated development of *A suum* eggs placed in a commercial swine farm (in England) in order to simulate normal environmental conditions and seasonal influences. Unembryonated eggs placed in June and July became infectious in August and early September, while eggs placed in August and September underwent partial development, then experienced arrested development when conditions were unfavorable (ie, winter), and resumed development the following spring, although with reduced percent embryonation.[103] Eggs placed from September through May developed synchronously in the subsequent July. This seasonal development may be recognized as seasonal variations in liver condemnation rates at slaughter plants, with the greatest prevalences seen July through December, when growing pigs exposed to infectious eggs are marketed.[112,113] Seasonal development is seen in pasture-raised pigs, where a "spring rise" and increasing prevalence is observed when pastures are infected the prior fall.[49,86,114] On the other hand, it is expected that environmentally controlled facilities, such as farrowing rooms and stalls with supplemental heat (eg, heat lamps, heat mats, brooder covers), would promote larval development year round.[115]

The US label for Safe-Guard EZ Scoop states "For individual 400 lb sow feeding: Mix 1 level scoop (1.07 ounces) of Safe-Guard EZ Scoop premix into 4 to 6 lb of an individual 400 lb sow's daily ration and feed once daily for 3 consecutive days." However, extra-label treatment of sows with a single-day, single-scoop dose of Safe-Guard EZ Scoop (ie, Treatment 1), equating to a 3 mg per kg dose for a 181.8-kg animal, is commonly used. In addition, farms usually do not weigh individual sows prior to treatment, but rather estimate an average weight for all sows[72] (JSP, personal observation, May 2014). Therefore, treatment may be less than indicated by both dose and frequency. The consequence of imprecise dosing and abbreviated treatment regimens on *A suum* egg shedding has not been thoroughly evaluated. In early studies of fenbendazole in swine, Baeder et al[59] used a single oral dose of 5 mg per kg and eliminated 100% of adult *A suum* from the intestine, and Batte[61] demonstrated the efficacy (96.0% adult *A suum* removed) of 3 mg per kg as a single dose. Extended treatment (3 days or longer) with fenbendazole, even with a lower daily dose, is usually considered more effective than single doses, namely, for treatment of *Trichuris suis*.[61,116] Fenbendazole was used in an off-label manner in this study; however, the authors do not necessarily recommend off-label use under field conditions. It was the authors' intent to mimic the potential application variation of this product as it might be used in the field by pork producers and veterinarians.

The results of the shedding experiments support using fenbendazole in breeding females prior to farrowing, and indicate that treatment is highly effective and should begin 14 days prior to moving sows into clean farrowing facilities in order to minimize *A suum* egg shedding into that environment. Treatment of breeding-herd animals after this time, or upon entry into the farrowing facility, will likely result in increasing facility contamination and increasing transmission risk to suckling piglets due to carryover of embryonated eggs from previous farrowing groups.

The embryonation experiments demonstrated that fenbendazole has ovicidal activity at various treatment levels on *A suum* eggs shed from naturally infected sows, applied in a manner consistent with practical commercial farm methods. Results from these experiments agree with work by others who have evaluated the ovicidal activity of other benzimidazoles on *A suum* and *A lumbricoides* or fenbendazole on other parasites.[33,66,67,70,91] To the authors' knowledge, this is the first description of the ovicidal activity of fenbendazole in *A suum* eggs collected from the feces of naturally infected sows.

In addition to sanitation and anthelmintic treatment to reduce adult worm burden and shedding by hosts, using an effective anthelmintic with ovicidal activity, such as fenbendazole, adds an epidemiological advantage by reducing the effective infectious egg load in the environment. Fenbendazole, with its adulticidal, larvacidal, and ovicidal properties, provides added value when implemented in *A suum* control programs in an infected population.

Implications

- Under the conditions of this study, fenbendazole, as a feed top-dress at 545.5 mg for 1 day, 545.5 mg daily for 3 consecutive days, or 1636.5 mg for 1 day, is effective for *A suum* treatment in naturally infected gestating breeding sows.
- Breeding female swine with naturally occurring *A suum* should be dewormed with fenbendazole at least 14 days prior to entry into clean farrowing facilities to minimize transmission to offspring and reduce facility contamination.
- Under the conditions of this study, fenbendazole, at 545.5 mg for 1 day, 545.5 mg daily for 3 consecutive days, or 1636.5 mg for 1 day, is ovicidal to *A suum* eggs shed from naturally infected gestating sows, starting as soon as 4 days post treatment and lasting through at least 8 days post treatment.
- Fenbendazole as a treatment for *A suum* provides an additional epidemiological advantage through reducing effective environmental contamination resulting from its ovicidal properties.

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Conflict of interest

None reported.

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Case report describing the clinical course of porcine epidemic diarrhea in a commercial boar stud and return of the stud to service after whole-herd inoculation with porcine epidemic diarrhea virus

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Summary

In January 2014, an outbreak of porcine epidemic diarrhea (PED) occurred in a PED-naive commercial boar stud. This report documents the outbreak following whole-herd PED virus (PEDV) inoculation with fecal material, cleaning procedures, sentinel exposure, and semen supply to naive sow farms. Boar saliva samples were diagnostically comparable to rectal swabs and collection dummy Swiffer (Proctor and Gamble, Cincinnati, Ohio) samples for PEDV RNA detection. Viral RNA was not detected in semen samples collected during the outbreak, yet reproductive organs from sacrificed boars 5 days after exposure were positive by polymerase chain reaction. Placed sentinel and replacement animals in the stud remained clinically negative following cleaning procedures, and semen shipments started 13 weeks post inoculation (WPI) to one PED-naive sow farm, with six other naive sow farms resuming shipments after 17 WPI. All sow farms remained naive 10 months later. This report demonstrates that it is possible for a commercial boar stud to experience a PED outbreak without infecting naive sow farms at the onset, retain valuable genetics, and resume semen delivery to PED-naive sow farms after cleaning, disinfection, and testing, without infecting sows upon re-opening.

Keywords: swine, porcine epidemic diarrhea virus, artificial insemination, boar stud

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Resumen - Reporte de un caso que describe el curso clínico de la diarrea epidémica porcina en un centro de sementales comercial y el regreso del centro a servicio después de la inoculación del hato completo con el virus de la diarrea epidémica porcina

En enero 2014, ocurrió un brote de diarrea epidémica porcina (PED por sus siglas en inglés) en una centro de sementales comercial libre al PED. Este reporte documenta el brote seguido de la inoculación del centro completo con el virus del PED (PEDV por sus siglas en inglés) con materia fecal, procedimientos de limpieza, exposición a centinelas, y abastecimiento de semen a granjas de hembras libres de la enfermedad. Las muestras de saliva de los machos fueron comparables diagnósticamente con muestras rectales y los Swiffer (Proctor and Gamble, Cincinnati, Ohio) del banco de recolección en busca de la detección del PEDV RNA. No se detectó RNA viral en muestras de semen recolectadas durante el brote, sin embargo los órganos reproductivos de machos sacrificados 5 días después de la exposición resultaron positivos a la reacción en cadena de polimerasa. Los centinelas colocados y los animales de remplazo en la granja permanecieron negativos después de los procedimientos de limpieza, y el envío de semen inició 13 semanas después de la inoculación (WPI por sus siglas en inglés) a una granja de hembras libre de PED, otras seis granjas de hembras libres reanudaron la recepción después de 17 WPI. Todas las granjas de hembras permanecieron libres10 meses después. Este reporte demuestra que es posible que una centro de machos comercial experimente un brote de PED sin infectar granjas de hembras libres al inicio del brote, retenga genética de valor, y reanude la entrega de semen a granjas de hembras libres a la PED después de limpieza, desinfección, y pruebas, sin infectar hembras al reabrir.

Résumé - Rapport de cas décrivant l'évolution clinique d'une éclosion de diarrhée épidémique porcine dans une verraterie commerciale et reprise des activités après inoculation du troupeau entier avec le virus de diarrhée épidémique porcine

En janvier 2014, une éclosion de diarrhée épidémique porcine (DEP) est survenue dans une verraterie commerciale naive pour la DEP. Le présent rapport documente l'éclosion survenue suivant l'inoculation du troupeau au complet avec le virus de la DEP (VDEP) en utilisant du matériel fécal, les procédures de nettoyage, l'exposition

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d'animaux sentinelles, et la fourniture de semence à des fermes de truies naives. D'un point de vue détection de l'ARN du VDEP, les échantillons de salive de verrat étaient comparables à des écouvillons rectaux et des prélèvements effectués sur les mannequins de collecte à l'aide de Swiffer (Proctor and Gamble, Cincinnati, Ohio). L'ARN viral ne fut pas détecté dans les échantillons de semence prélevés durant l'éclosion malgré que des échantillons provenant des organes reproducteurs de verrats sacrifiés 5 jours suivant l'exposition étaient positifs par réaction d'amplification par la polymérase. Les sentinelles et les animaux de remplacement dans la verraterie sont demeurés cliniquement négatifs suivant les procédures de nettoyage, et les expéditions de semence commencées 13 semaines post inoculation (SPI) a une ferme de truies naives pour la DEP, et six autres fermes de truies naives commençant à recevoir de la semence 17 SPI. Toutes les fermes de truies sont demeurées naives 10 mois plus tard. Ce rapport démontre qu'il est possible pour une verraterie commerciale de subir une éclosion de DEP sans que des fermes de truies naives ne soient infectées au début de l'éclosion, de conserver la valeur génétique du troupeau, et de recommencer la livraison de semence à des fermes de truies naives pour la DEP après nettoyage, désinfection, et tests de détection, sans infecter des truies suite à la remise en opération.

Porcine epidemic diarrhea virus (PEDV) was first identified in the United States in late April 2013.[1] Since then it has spread rapidly across the country and caused significant production and economic losses, with estimates of 7 to 8 million pigs lost from June 2013 to April 2014.[2] While strides have been made in prevention and clinical management in other segments of production, better information is needed to answer questions regarding the course of porcine epidemic diarrhea (PED) in artificial insemination boars, its effects on semen quality and production, and risk to sow farms sourcing from a previously infected stud. Of particular concern is the question of whether PEDV can be shed in semen.

Although confirmation of PEDV infection has been described within numerous sow farms, nursery facilities, and grow-finish barns throughout the United States, infection within a commercial boar stud has not yet been formally documented. Likewise, the veterinary literature lacks reports of mature boar infection. Up to this point, commercial boar studs faced the very real risk of depopulation if infected with PEDV, with the loss of valuable genotypes and inherent slow recovery to previous production levels after restocking with young boars. Given PEDV's predilection for enterocytes,[3] the ability to retain exposed boars, maintain a mature age structure, observe a prudent herd closure time, and re-open without infecting downstream sow farms was theoretically possible, but not yet proven. To the authors' knowledge, this is the first North American PED case report of its kind, specific to artificial insemination boars, that demonstrates the ability to retain previously infected boars and resume service to naive sow farms without transmitting the virus.

Case history

In January 2014, PEDV entered a boar stud in northeast Nebraska that was negative for porcine reproductive and respiratory syndrome virus. This is a facility under veterinary care and certified by Pork Quality Assurance (PQA; National Pork Board).

Rapid detection, intentional whole-herd exposure, and boar retention provided a unique opportunity to capture much-needed data and set the conditions for this clinical case report. At the time of the outbreak, the boar stud held approximately 200 boars in the main barn, with 30 boars present in a connected on-site isolation barn. The site is fully filtered from October 1 to June 1 each year. During times of filtration, the load-out area has a positive pressure system to prevent back-draft of air. The load-out area is used for removal of dead animals, garbage, or other items exiting the site. No known biosecurity breakdowns occurred at this site. The closest known PEDV-positive farm at the time was approximately 11.2 kilometers away.

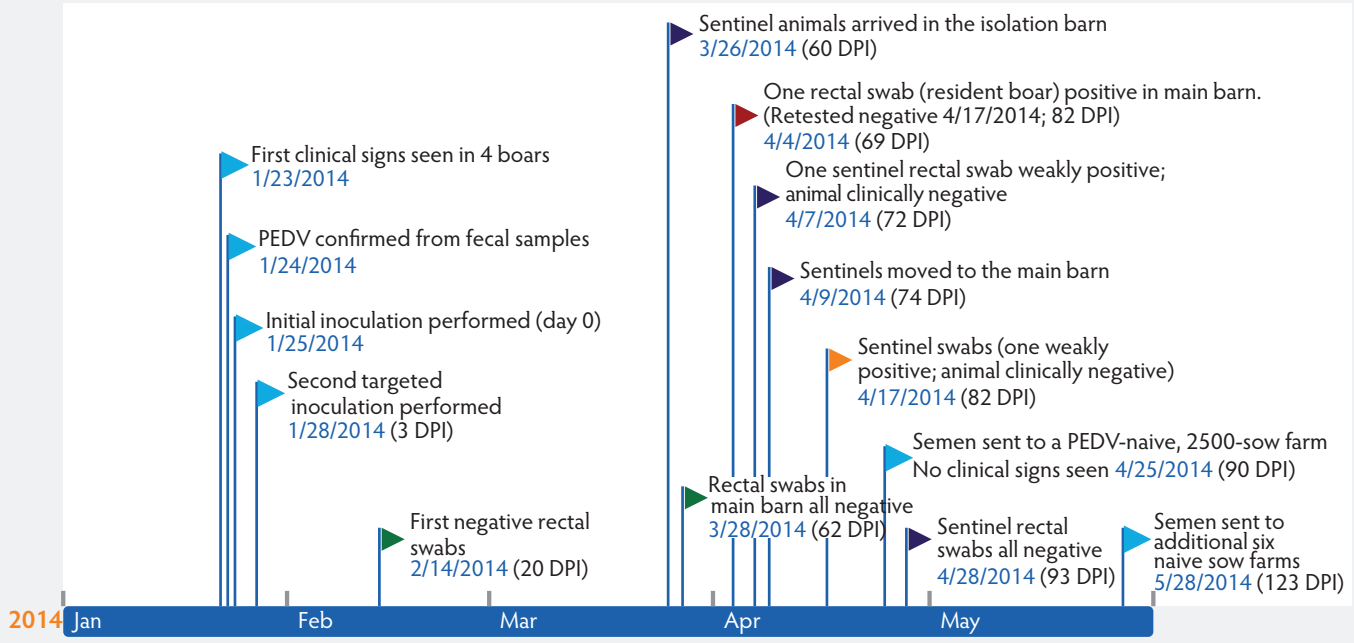
On January 23, in the afternoon, after boar collection had been completed for the day and semen had been shipped to naive farms, diarrhea was observed in four boars in the main barn. All farms were contacted to monitor closely for clinical signs, and semen held at the boar stud for post-production analysis was sent to GeneSeek, Inc (Lincoln, Nebraska) for PEDV testing by polymerase chain reaction (PCR). All samples tested negative. The next morning, 15 boars in the main barn had diarrhea, and semen collection was halted for the day. Fecal samples were collected and transported to GeneSeek, Inc, for PEDV PCR testing. Results were received the same afternoon, with all submitted samples positive for PEDV RNA (Figure 1).

Investigative design, sample collection, and laboratory testing

On the morning of January 25, whole-herd PEDV inoculation was performed using fecal material from diarrheic boars that was sprayed into the mouth of each boar. Oral inoculation of boars that had not previously shown clinical signs was repeated 3 days later using fresh fecal material and refrigerated aliquots of the first inoculum (Figure 1). Immediately prior to inoculation, 30 mature boars in the main stud, not showing clinical signs of PEDV infection and not located directly beside a boar that was showing clinical signs, were conveniently selected for prospective diagnostic monitoring. This was done in an effort to time the initial exposure to PEDV inoculation with feces from boars that were either PCR-positive or showing clinical signs, rather than to prior exposure. Rectal swabs were collected from these 30 boars using individual sterile cotton swabs that were placed in 0.5 mL sterile saline in a 5-mL snap-cap tube (Falcon tube; Corning, New York).

Rectal swab samples were subsequently collected from the 30 cohort boars on days post inoculation (DPI) 1-8, 13, 20, 27, 34, 41, 48, 55, 62, and 69 (Table 1) for testing at Iowa State University Veterinary Diagnostic Laboratory (ISU VDL; Ames, Iowa) using a previously described PEDV N-gene-based real-time reverse transcriptase- (RT-) PCR.[4] Because of financial testing constraints, a subset of 10 boars from the original cohort of 30 were conveniently selected for additional collection and testing of semen, serum, and oral-swab samples during the study period (Table 2 and Table 3). Semen was collected utilizing the double gloved-hand method for minimum contamination, with subsamples obtained for further testing in the on-site semen-processing laboratory using aseptic technique.[5] Oral swabs were collected using a sterile cotton swab that was inserted between the lip and gum while boars were mounted on the dummy. Samples were then placed in BD Universal Viral Transport System vials (UVT; Franklin Lakes, New Jersey). Collected semen samples were processed at the ISU VDL as previously reported[6] and individually assayed for PEDV RNA using a described PCR protocol.[4] After semen samples were collected

Figure 1: Timeline of events for a porcine epidemic diarrhea (PED) outbreak in a commercial boar stud, detailing clinical disease, diagnostic testing, sentinel pig exposure, and resuming of semen delivery to PED-naive sow farms after whole-herd oral inoculation with fecal material. On January 23, 2014, an outbreak of diarrhea was observed in a genetic boar stud housing approximately 200 boars in the main building, and 30 boars in a connected on-site isolation barn. Rectal swabs were collected from a total of 30 conveniently selected cohort boars and tested for porcine epidemic diarrhea virus (PEDV) by PCR. Beginning immediately after the first set of samples were collected (day 0), whole-herd PEDV inoculation was performed by spraying into the mouth of each boar fecal material collected from diarrheic boars. Inoculation of boars that did not show clinical signs was repeated 3 days post inoculation (DPI) using fresh fecal material and refrigerated aliquots of the first inoculum. Fecal and oral swabs were collected from cohort boars to evaluate viral shedding, and environmental samples were tested for PEDV genetic material.



from the boars, unscented, dry Swiffer pads (Proctor and Gamble, Cincinnati, Ohio) were soaked in 10 mL of sterile saline, used to wipe the collection dummies, and then placed in sealed plastic bags. Oral swabs and Swiffer pads were assayed using the same PCR protocol with individual results from 0 to 3 DPI and pooled results thereafter. Serum indirect fluorescent antibody (IFA) testing was performed as previously described.[4]

Extended semen samples were submitted to Kuster Research and Consulting Inc (Geneseo, Illinois) for semen quality evaluation, including computer-aided sperm analysis motility (Integrated Visual Optical System; Hamilton Thorne Biosciences, Beverly, Massachusetts), full morphology differential (manual; trained technician), and flow cytometry analysis (Guava EasyCyte Plus; Millipore Corp, Hayward, California). A non-infected boar stud that mirrored the infected site in key aspects (negative control site), including geographic proximity (approximately 14.5 kilometers), shared farm personnel prior to the PED break, genotypes, and production protocols, was chosen for comparison of semen quality. Extended semen samples collected from 10 genotype- and age-matched boars at the negative control site were submitted once a week for 8 weeks to provide comparative semen-quality data. Semen-quality parameters were analyzed by repeated measures analysis of variance (ANOVA), with Tukey's HSD test used to investigate pair-wise comparisons where significant differences ($P < .05$) were noted (Statistix 10.0; Analytical Software, Tallahassee, Florida).

Three non-cohort boars in the main barn were chosen for necropsy to determine if there was evidence of PEDV infection present in reproductive organs that could lead to direct semen shedding. All three boars had shown clinical signs for 1 to 2 days prior to necropsy at 5 DPI. All reproductive organs were harvested with the intent of preventing contamination from the environment or the intestinal contents, with sections of each of the following collected for histopathology, immunohistochemistry (IHC), and PCR testing: testes, epididymides, bulbourethral gland, seminal vesicles, prostate, and penis. Intestinal samples were also collected.

Cleaning and disinfection procedures

Initial cleaning of the main and isolation barns consisted of removing all visible organic material from equipment and floors with a hot-water power washer (87°C) using Biosolve detergent (DuPont, Wilmington, Delaware), then sanitizing with Clorox bleach (Oakland, California). Lemon juice was also used in both barns to remove hard water stains and biofilm, with the additional use of Synergize (Reno, Nevada), a quaternary ammonium-glutaraldehyde disinfectant, in isolation only. The cleaning procedure commenced 14 and 20 DPI in the main and isolation barns, respectively. Boars in isolation at the time of the outbreak were moved into the main barn at 20 DPI.

Unscented Swiffer pads were used to sample for PEDV RNA in cleaned and disinfected areas. Samples were collected from all aspects of isolation, including the shower area, medication room, boar stalls, feed boxes, collection area, and miscellaneous equipment. In addition, laboratory and semen pick-up locations, including insulated

coolers, semen cups, carts, floor, countertops, computer, microscope, cool room, and anteroom floor were tested using the Swiffer pad protocol as described.

Sentinel animals

After cleaning and disinfection, seven commercial, PEDV-naive sentinel grower pigs of mixed gender, weighing approximately 57 kg, were placed in the empty isolation barn 9 weeks post inoculation (WPI). These sentinels were then moved into the main barn 11 WPI (Figure 1). Rectal swabs were obtained from sentinels 10 days after they were placed in isolation, and 8 and 15 days after they were moved to the main barn. In addition, 40 naive replacement boars were placed in the isolation barn 14 WPI and moved to the main barn, with direct contact with previously infected boars, at 17 WPI.

Results and outcomes

The most intense period of clinical disease after inoculation occurred 4 to 6 DPI, with evidence of watery diarrhea, reduced feed intake, lethargy, and occasional vomiting (3.28%). All but seven boars in the entire stud had recorded clinical signs consistent with PED. The last clinical signs were noted in the main barn on February 7, 2014, at 13 DPI. One boar of the 10 initially designated for prospective diagnostic monitoring was removed in the first week due to lameness that prevented semen collection.

At the initial sampling (day 0 immediately before oral inoculation), rectal swabs from 29 of the 30 cohort boars were negative by PCR, with cycle threshold (Ct) cutoff > 35. In the single positive boar, quantity of virus was low (Ct = 34.5). At 3 DPI, all 30 boars were positive by fecal PCR for PEDV, with Ct values ranging from 15.0 to 34.0 (Table 1). With the exception of one boar at 4 DPI, all others remained PCR-positive from 3 to 13 DPI. Inconsistent fecal PEDV shedding was apparent thereafter in the study population (Table 1). At approximately 9 WPI, all 30 cohort boars tested negative by PCR on rectal swabs. One boar tested PCR-positive at 69 DPI after testing negative on the 3 previous weeks. On retest the following week, the rectal swab from this boar once again tested negative.

All semen samples were negative by PEDV PCR on day 0 immediately before oral inoculation with feedback material. Semen samples from all boars at subsequent collection time points were also negative. Serum samples were negative for PEDV antibody by IFA at 1 DPI, and all were positive at 21 DPI. Oral swabs from all sampled boars were PCR-negative on day 0 immediately before oral inoculation. All oral-fluid swabs were positive at 6 DPI (Ct = 25 to 32) and remained positive through 13 DPI (Ct = 32 to 33). Thereafter, all pools were negative for PEDV genomic material (Table 2).

Small intestinal samples from all three necropsied boars showed histopathological changes consistent with PEDV infection, including villus atrophy with variable enterocyte degeneration or attenuation and mild non-suppurative cellular inflammation within the lamina propria. Viral antigen was also detected by IHC in affected sections. In contrast, reproductive organs of all three boars were unremarkable histologically, and PEDV antigen was not detected in testes, epididymis, seminal vesicle, bulbourethral gland, prostate, or penile tissue. However, testicular tissue from two of the boars and penile tissue from the third were PCR-positive (Ct = 29.6 to 34.3).

Semen quality data was not available for five observations (two infected, three controls) due to inability to obtain a sample or non-submission of collected boars. Sperm motility was significantly different between the boars housed at the PEDV-infected site and those in the control site (infected, 73%; control, 81%; $P = .01$), with no interaction between weeks post inoculation and location. Significant differences were not identified for normal morphology comparisons between sites ($P = .09$). Sperm plasma membrane viability and acrosome integrity (VIA) were measured both on fresh semen (tested on arrival) and stored semen (at expiration), with no differences at either time point by location ($P > .05$). While differences in VIA were also not detected for the interaction of location and WPI after storage, this interaction was significant for the fresh VIA analysis overall ($P = .04$). However, Tukey's HSD test revealed no pairwise differences ($P > .05$). Values declined at similar rates of 5% to 8% by location between the fresh and stored readings. Differences in DNA integrity were present between the infected and control sites ($P = .01$) and between WPI ($P < .001$) (Figure 2). Pairwise comparisons revealed that DNA integrity was compromised most at 9 WPI for PEDV-infected boars. Significant differences were noted between ejaculates for individual boars for all parameters monitored, independent of PEDV exposure.

Virus was detected on three of 10 collection-dummy Swiffer pads samples collected

Table 1: Summary results for cohort boars tested for porcine epidemic diarrhea virus (PEDV) by polymerase chain reaction (PCR) on rectal swabs*

	Day post inoculation																	
	0	1	2	3	4	5	6	7	8	13	20	27	34	41	48	55	62	69
n	30	30	30	30	30	30	30	30	30	30	29†	28†	27†	27	27	27	27	27
No. positive	1	9	25	30	29	30	30	30	30	30	24	22	16	24	4	2	0	1‡
Min Ct	34.5	13.0	14.6	15.0	14.7	14.7	12.6	13.9	16.7	19.4	26.2	27.8	28.6	27.2	31.9	32.1	0.0	34.0
Max Ct	34.5	34.7	34.9	34.0	33.0	30.0	33.0	30.3	33.1	34.3	34.4	34.7	34.8	34.5	34.6	33.3	0.0	34.0
Mean Ct	34.5	26.3	26.6	21.9	20.1	19.9	18.7	19.9	23.1	28.9	31.1	31.7	32.7	31.9	33.5	32.7	0.0	34.0

* Study described in Figure 1.

† On each day, one boar was euthanized due to lameness or was unexpectedly found dead.

‡ Retest on this boar the following week was negative.

Ct = cycling threshold; Min = minimum; max = maximum.

prior to inoculation (Ct = 29.6 to 34.6). Individual dummy Swiffer pads were uniformly positive at 2 DPI (n = 5; Ct = 24.3 to 32.9) and 3 DPI (n = 6; Ct = 20.8 to 29.5). Pooled fluids obtained from Swiffer pads were PCR-positive at 6, 20, and 34 DPI with Ct values of 21.7, 31.5, and 32.5, respectively, and were PCR-negative at 48 and 55 DPI (Table 3).

Four rounds of cleaning and disinfection were performed in the isolation barn and two to three rounds of cleaning in the main barn, depending on location. Even after passing visual inspection, five of 15 samples collected from the isolation barn on the third round of cleaning were positive for PEDV by PCR, with Ct = 24.8 to 34.8. Compared to other sampled locations in the main barn, boar stalls had the highest detectable quantities of PEDV RNA. Of the laboratory and semen pick-up locations, three samples were weakly PCR-positive (20 DPI): cart (semen pick-up location), computer, and anteroom, with Ct = 33.5 to 34.4.

Sentinel animals showed no clinical signs of PED when placed in the cleaned and disinfected isolation barn or the main barn holding boars previously exposed to PEDV at 9 and 11 WPI, respectively. However, one rectal swab each from the first and second collections (11 WPI and 15 WPI) was weakly positive by PCR (Ct = 33.7 and 34.2). All other samples were PEDV-negative. Replacement boars did not develop clinical disease after being placed in the isolation and main barns at 14 and 17 WPI, respectively.

At the onset of this PEDV outbreak at the boar stud, the sow base served by this boar stud included a limited number of PEDV-positive farms, and semen shipments to these units (n = 4) resumed within 1 WPI. Semen distribution to PEDV-naive sows resumed at 13 WPI to a 2500-sow farm, and 4 weeks after that to six other PEDV-naive sow farms (17 WPI), until a total of 11 sow sites (including four PEDV-positive or exposed sow farms), with a total inventory of approximately 45,000 sows, were once again being served exclusively by this boar stud. None of the naive farms receiving semen displayed clinical signs of PEDV or produced positive PEDV diagnostic testing after resuming acceptance.

Table 2: Results testing for porcine epidemic diarrhea virus (PEDV) by PCR on oral swabs obtained from boars during semen collection*

DPI	No. of samples†	Pooled	PCR results	
			Positive	Negative
0	10	No	0	10
6	9	No	9	0
13	9	Yes	2	0
20	9	Yes	0	2
27	9	Yes	0	2

* Study described in Figure 1. Of the 30 boars described, 10 were chosen for additional, once-weekly collection of oral fluids by swabbing the mouth while the boar was mounted on a dummy. A sterile cotton swab was inserted between the lip and gum and then was placed in virus transport medium. Transport medium was pooled (two pools; one pool of five and one pool of four samples) for testing at 13, 20, and 27 DPI.

† One boar was removed in the first week because of lameness.

PCR = polymerase chain reaction; DPI = days post inoculation.

Table 3: Results of PCR testing of environmental samples obtained from a collection dummy in a boar stud recently exposed (day 0) to porcine epidemic diarrhea virus (PEDV) by whole-herd oral inoculation*

DPI	No. of samples	Pooled	Positive	Negative
0	10	No	3	7
2	5	No	5	0
3	6	No	6	0
6	6	Yes	1	0
20	6	Yes	1	0
27	4	Yes	0	1
34	6	Yes	1	0
48	6	Yes	0	1
55	6	Yes	0	1

* Study described in Figure 1. Unscented, dry Swiffer pads (Proctor and Gamble, Cincinnati, Ohio) soaked in 10 mL of sterile saline were used to wipe down dummies after semen collection. Pads were placed in sealed plastic bags and tested individually (up to 3 DPI) or pooled (one pool of four or six samples) thereafter.

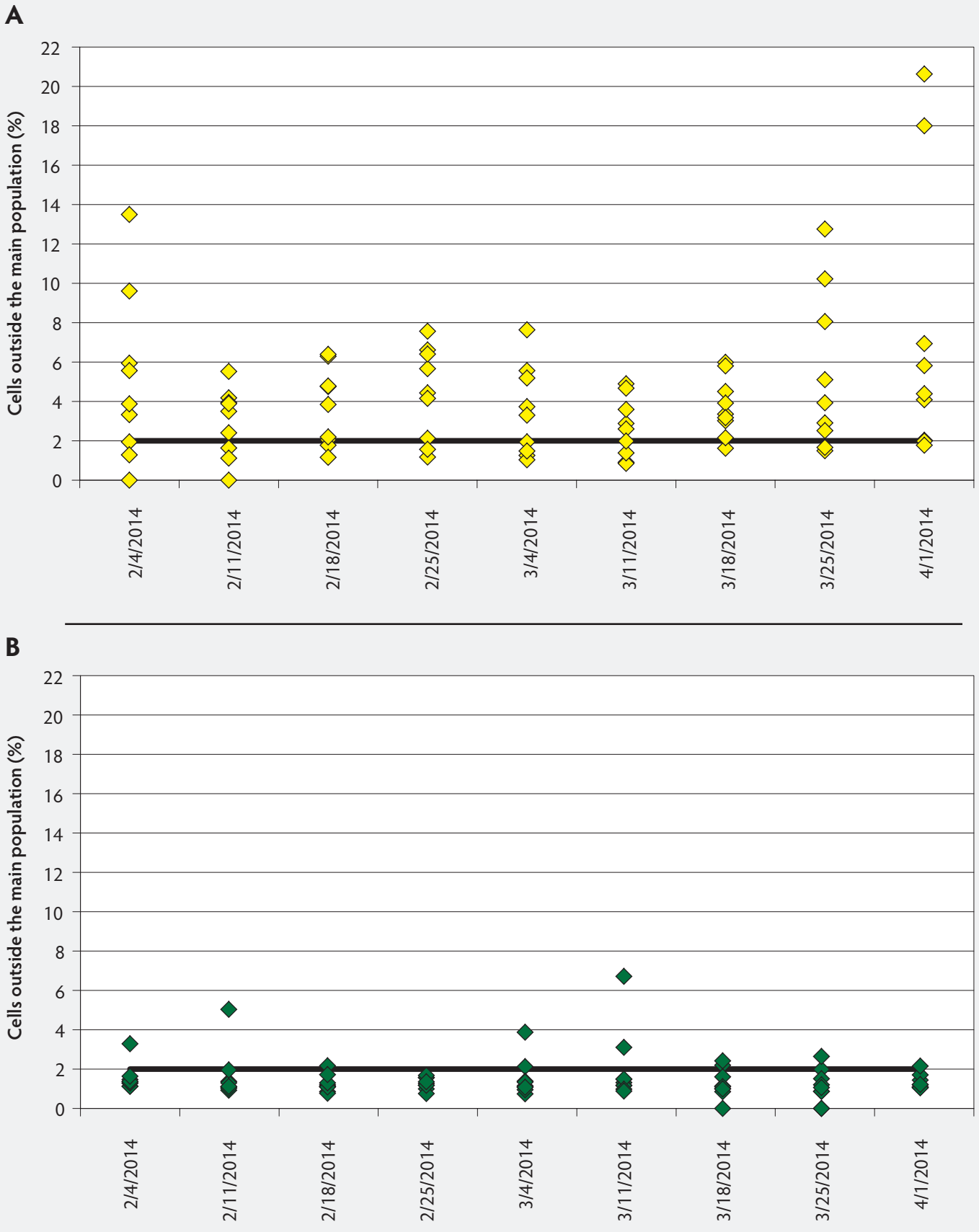
PCR = polymerase chain reaction; DPI = days post inoculation.

Discussion

Clinical signs in this naive farm were an early warning signal to initiate confirmatory testing and closure of the boar stud before potentially infecting sow farms, as demonstrated by no downstream infection. Similar to swine of all ages,[7] individual boars varied in the timing, duration, and severity of disease. However, disease in this case may have been slightly altered by the strategic whole-herd inoculation. Rectal swab PCR testing demonstrated consistent shedding throughout this population of adult boars for at least 2 weeks, with a high proportion remaining in the suspect or positive range for nearly 6 WPI. An abrupt reduction was detected at 7 WPI. Intermittent shedding was demonstrated toward the end of the infection phase.

Environmental sampling from the collection dummy with Swiffer pads was effective at identifying PEDV in the environment throughout the outbreak. Due to the nature of the case report, dummy swab results were not available immediately prior to recognition of clinical signs, but remained positive from the time this sampling method was deployed immediately prior to oral inoculation until 20 DPI, with intermittent results from pooled samples

Figure 2: Comparison of sperm DNA integrity between boars (n = 9) housed at the porcine epidemic diarrhea virus- (PEDV-) infected site (panel A) and genotype and age-matched control boars (n = 10) housed at the negative control site (panel B) located approximately 14.5 km away. Sperm DNA integrity was compromised at the PEDV-infected site, while largely remaining within normal limits at the control location.



thereafter. Use of the dummy Swiffer pads can be considered a tool to detect PEDV in the population, with the boars acting as "bio-swabs" as they become contaminated with virus from themselves, their neighbors, or the environment during normal eating, drinking, lying down, movement to the collection area, and interaction with warm-up or collection pens, then conveniently deposit the virus in a natural bottleneck (collection pen) where it is easily obtained during routine production.

Great care was taken to ensure that collected semen samples were not contaminated with fecal-associated virus. In this report, PEDV was not detected by PCR in raw semen. Both the pellet fraction and the seminal plasma were negative for all tested boars at any day post inoculation. However, testicular tissue from two euthanized boars was PCR-positive with low quantities of detectable PEDV genomic material (Ct = 29.6 to 34.3). Non-testicular reproductive organs were also sporadically PCR-positive in these boars, yet IHC for PEDV was consistently negative for all male reproductive organs and boars. Porcine epidemic diarrhea virus viremia[8] or tissue contamination during the necropsy procedure are potential reasons for the tissue, but not the semen, to contain genomic material.

The semen quality monitoring suggests the possibility of a negative association between PEDV infection and the parameters of sperm motility and morphology routinely assessed at boar studs. Although weeks post inoculation did not influence motility results, a divergent trend was noted in morphology due to increased abnormalities over time at the infected site that may have gone undetected because of the few missing observations. Sperm membrane parameters were apparently unaffected. The most noteworthy finding was the difference in DNA integrity, which was challenged at the infected site, while largely remaining within normal limits for the control location. Cells outside the main population of 2% or more can put boars at risk for suboptimal reproductive performance.[9-11] Of particular concern is the limitation that this assessment is not currently practical at boar studs, and may not be recognized if not evaluated at a veterinary andrology laboratory. This case report indicates the need for prospective work to further investigate the effects of PEDV infection on boar semen quality and potential fertility, especially for boar studs with the opportunity to service previously infected (immune) sow farms soon after a PEDV break.

Successful introduction of PEDV-naive stock indicates both that adequate time had passed and environmental decontamination was successful between the initial outbreak and placement of non-immune boars. Not only had remission of clinical signs occurred and viral shedding abated in inoculated boars, but the barn environment had become safe for naive stock, despite persistently positive environmental swabs. However, it should be noted that rectal swabs from sentinel animals placed in the barn showed weak PCR positivity, despite the absence of clinical signs. It is unknown if this weak positivity was true infectious virus, rogue environmental genomic material, or laboratory contamination, but this finding led to confusion regarding release of semen to naive sow farms and introducing naive boars. This highlights the sensitivity of the PEDV PCR and the reminder that presence of viral genome does not guarantee infection in the clinical setting.

The detection and closure procedure observed by this boar stud at the time of the outbreak was sufficient to prevent PEDV-negative sow farms from becoming infected. Subsequently, the interventions applied allowed semen shipments from previously infected boars to resume to sow farms that had no prior history of PEDV infection, without negative consequences. The timeline details the events of this case from infection to successful return to sow-farm service. Success in this case reinforces the possibility of returning a PEDV-infected boar stud to service and highlights the need to determine how this can be repeated safely after less down time.

Implications

- Boar reproductive organs may contain low quantities of PEDV genomic material in the acute phase of infection; however, under the conditions of this case, virus is not detectable by PCR in semen samples.
- Environmental sampling of the boar collection dummy with Swiffer pads (Proctor and Gamble, Cincinnati, Ohio) can be utilized as a PEDV environmental-monitoring tool.
- In this case, semen from boars previously exposed to PEDV could be shipped to sow farms following strategic suspension and strict collection hygiene protocols.
- Semen quality may be affected during a PEDV outbreak and should be closely monitored when ongoing service to sow farms is considered.

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* Non-referred reference.

Conversion tables

Weights and measures conversions

Common (US)	Metric	To convert	Multiply by
1 oz	28.35 g	oz to g	28.4
1 lb (16 oz)	453.59 g	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)	0.31 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
1 gal (128 fl oz)	3.8 L	gal to L	3.8
0.264 gal	1 L	L to gal	0.26
1 qt (32 fl oz)	946.36 mL	qt to L	0.95
33.815 fl oz	1 L	L to qt	1.1

Temperature equivalents (approx)

°F	°C
32	0
50	10
60	15.5
61	16
65	18.3
70	21.1
75	23.8
80	26.6
82	28
85	29.4
90	32.2
102	38.8
103	39.4
104	40.0
105	40.5
106	41.1
212	100

$^{\circ}F = (^{\circ}C \times 9/5) + 32$
$^{\circ}C = (^{\circ}F - 32) \times 5/9$

Conversion chart, lb to kg (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	135
	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

Checkoff-funded research

Validation of captive bolt as a single-stage euthanasia method

Principle researcher: Dr Suzanne Millman, Iowa State University

Key points:

- The Cash Dispatch captive-bolt device is effective as a single-step euthanasia method for pigs weighing less than 200 kg (441 lb).
- Weight class was significantly associated with the need for a second shot.
- Stock people should be prepared to administer a second shot swiftly when euthanizing mature pigs with a captive-bolt device.

Summary: Limited research has been published on the use of penetrative or non-penetrating captive bolt for swine euthanasia. The Cash Dispatch Kit (Jarvis Industries Canada Ltd, Calgary, Alberta, Canada) is a heavy-duty, cartridge-propelled, captive-bolt device with interchangeable muzzle assemblies. The unit provides a non-penetrating captive-bolt muzzle for piglets and a variety of penetrating bolt assemblies for large nursery-age pigs to mature breeding stock. We conducted two experiments to determine the effectiveness of a single application of the captive-bolt device to euthanize pigs of different ages. Trial #1 explored the effectiveness of the Cash Dispatch captive-bolt device when applied to anesthetized pigs in a laboratory setting. We also evaluated the association between traumatic brain injury and effectiveness of the captive bolt to euthanize pigs at different ages. Forty-two pigs were enrolled in six weight classes: 2 to 3 kg, 7.5 to 10 kg, 15 to 20 kg, 30 to 40 kg, 100 to120 kg, 200 to 250 kg, and >300 kg. All pigs in the five lightest weight classes were effectively euthanized. Four of the 12 pigs in the heaviest weight classes required a secondary method. In Trial #2, we enlisted15 stockpersons from a single farm to perform euthanasia and applied the same seven weight classes to 210 pigs. In all, 97% were effectively euthanized with a single application of the Cash Dispatch Kit. Two sows and five boars in the heaviest weight classes required a second shot, which was sufficient to ensure euthanasia, and can be an alternative to exsanguination (bleeding) or pithing. Restraint of the head through snaring appeared to be important for both efficacy and safety, rather than restraint in a chute or stall.

To learn more, contact Sherrie Webb at SWebb@pork.org or go to www.pork.org and click on the research tab to search.

Post-processing chemical mitigation strategies to control PEDV in feed and ingredients

Principle researcher: Dr Cassandra Jones, Kansas State University

Key points:

- Medium-chain fatty acids, essential oils, and formaldehyde effectively mitigate post-processing feed-ingredient contamination with porcine epidemic diarrhea virus (PEDV).
- A chemical's success is dependent on the feed matrix, as is PEDV stability over time.
- The PEDV is more stable in meat and bone meal and spray-dried animal plasma than in blood meal or a complete diet.

Summary: Post-processing contamination of PEDV in feed and feed ingredients is a significant concern to the swine industry. Irradiation and thermal processing have been hypothesized as possible mitigation options, but both are point-in-time solutions. They do not provide residual benefits to prevent potential recontamination or cross-contamination within feed or feed-ingredient manufacturing, transport, or storage. This study aimed to find a possible mitigation strategy to help minimize the threat of PEDV recontamination in feed and feed ingredients. The results suggested that feed or feed ingredients or both can be treated with different chemical treatments as a means to mitigate PEDV contamination. Importantly, the success of various chemical mitigants was dependent upon the feed matrix, and PEDV stability over time also was matrix-dependent. The PEDV was more stable in meat and bone meal and spray-dried animal plasma than in blood meal or a complete swine diet. Ultimately, this research helps provide potential solutions to mitigate PEDV infectivity when transmitted by feed, and thereby ultimately lessens PEDV-associated losses to the swine industry.

To learn more, contact Lisa Becton at LBecton@pork.org or 515-223-2791.

Impact of temperature and time in pelleted diets on PEDV survivability in complete diets

Principle researcher: Dr Jason Woodworth, Kansas State University

Key points:

- Feed can be a vehicle for porcine epidemic diarrhea virus (PEDV) transmission.
- The minimum infectious dose of PEDV is equivalent to 1 gram of infected pig feces diluted in approximately 500 tons of feed.
- The pelleting process used in many commercial mills can act as a point-in-time mitigation step in PEDV-associated risk prevention plans.

Summary: Since late January 2014, suspicion grew that porcine epidemic diarrhea outbreaks may have been associated with consumption of PEDV-positive feed or feed ingredients. However, there was a lack of information to confirm feed as a PEDV vector. Also, there was no available data describing the minimum infectious dose of PEDV in a feed matrix. Additionally, it was believed that the normal temperature and retention times used by commercial pellet mills would adequately mitigate PEDV infectivity; however, no research had tested this hypothesis. Therefore, our goals were to determine the minimum infectious dose of PEDV in a feed matrix and to determine whether the retention time and temperatures used in commercial pellet mills influence PEDV infectivity. Our results confirmed that feed can be a vehicle for PEDV transmission and that the minimum infectious dose of PEDV in a feed matrix is quite low. A PEDV dose corresponding to a polymerase chain reaction (PCR) cycle-threshold value of 37 was low enough to lead to infectivity. The pelleting process

used in many commercial mills can act as a point-in-time mitigation step in PEDV-associated risk prevention plans. None of the virus-inoculated, processed feeds lead to infectivity in the pig bioassay model, even though the PCR analysis showed that PEDV RNA was present in the processed feed. In contrast, the non-processed feed led to PEDV infectivity.

To learn more, contact Lisa Becton at LBecton@pork.org or 515-223-2791.

Checkoff creates Antibiotic Resource Center

In light of the final rule for the Veterinary Feed Directive as outlined in the Food and Drug Administration's (FDA's) Guidance 213, the Pork Checkoff has created a one-stop Antibiotic Resource Center on its main Web site at www.pork.org/antibiotics. It provides easy access to recent news on the subject, fact sheets, links to Pork Quality Assurance Plus, FDA links, and more.

For more information, go to the Pork Checkoff Antibiotic Resource Center located at www.pork.org/antibiotics.

National Pork Board creates innovation team focused on food-chain outreach

Consistent with the National Pork Board's 5-year strategic plan to build consumer trust and grow consumer demand, the organization is updating its food-chain outreach structure. These changes will allow the National Pork Board to be even more effective in collaborating with channel partners in a focused effort to stimulate pork demand. "Our strategic plan defines a blueprint for industry success by addressing the changing world facing US pork producers," said Derrick Sleezer, president of the National Pork Board and a producer from Cherokee, Iowa. "Our marketing effort taps into the emotional connections consumers have with their food and will fuel a fresh dialogue about modern pork production and continuous improvement for the benefit of people, pigs, and the planet." Toward that end, the National Pork Board implemented the following staff changes:

- Jarrod Sutton is named vice president of channel marketing, innovation and social responsibility.
- Patrick Fleming is named director of market intelligence and innovation.
- Rob Kirchofer is named director of retail marketing and innovation.
- Stephen Gerike is named director of foodservice marketing and innovation.
- Ceci Snyder will continue to lead domestic marketing, advertising and public relations programs as vice president of consumer marketing.
- Stacie Schafer is named director of state marketing and consumer insight.

The National Pork Board plans to expand efforts in product innovation this year to grow consumer demand. This effort will be supported by consumer research, market data analysis and channel insights, product design, market testing, channel marketing, and channel communications. "We're very excited about this new direction in leadership within our organization," said Sleezer. "These changes will allow us to have greater focus and efficiency as we work with our foodservice and retail partners to increase pork demand."

For more information, contact Jill Criss at JCriss@pork.org or 515-223-2636.

National Pork Board adopts three-pronged antibiotic stewardship plan

Building on its years of supporting responsible antibiotic use, the National Pork Board has adopted a three-pronged, new antibiotic stewardship plan that is **proactive, collaborative, and aggressive** in its strategy and scope. Using education, research, and communication tactics, the plan will ultimately work for the betterment of people, pigs, and the planet.

The National Pork Board will lead the industry in adjusting to the phase-out of growth-promotion uses of medically important antibiotics and embracing increased veterinary oversight of antibiotic use on the farm. Although the federal government's initiatives on antibiotics poses new challenges for the industry, **US pig farmers pledge to go above and beyond compliance,** because they are committed to continuous improvement to ensure responsible antibiotic use on the farm.

To help producers achieve this goal, the National Pork Board will continue to implement a comprehensive plan that helps to **guide and support the responsible use of antibiotics**. As always, the longstanding Pork Quality Assurance Plus (PQA Plus) program will serve as the centerpiece of this effort. In fact, the certification program is currently being revised and expanded to reflect the latest federal guidance on antibiotics that will include veterinary oversight and maintenance of current medical records on the farm.

Additional efforts will include the **ongoing collaboration with allied-industry partners, suppliers, and regulatory agencies** to help assure that antibiotics that are needed for animal health remain in place and are used under veterinary oversight as called for in the industry guidance.

As always, the National Pork Board's overarching goal is to **serve and protect pig health and promote food safety**. Healthy animals make for safer food. The execution of this plan will help America's pork producers keep that promise.

NPB news continued on page 275

SRD PROTECTION
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For use by or on the order of a licensed veterinarian. Federal law prohibits the extra-label use of this drug in food-producing animals. Swine intended for human consumption must not be slaughtered within 5 days of receiving a single-injection dose. Use with caution in animals with known or suspected CNS disorders. Observe label directions and withdrawal times. See product labeling for full product information.

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Brief Summary: Before using Enroflox® 100, consult the product insert, a summary of which follows.

CAUTION: Federal (U.S.A.) law restricts this drug to use by or on the order of a licensed veterinarian. Federal (U.S.A.) law prohibits the extra-label use of this drug in food-producing animals.

PRODUCT DESCRIPTION: Each mL of Enroflox 100 contains 100 mg of enrofloxacin. Excipients are L-arginine base 200 mg, n-butyl alcohol 30 mg, benzyl alcohol (as a preservative) 20 mg and water for injection q.s.

INDICATIONS:
Cattle - Single-Dose Therapy: Enroflox 100 is indicated for the treatment of bovine respiratory disease (BRD) associated with *Mannheimia haemolytica, Pasteurella multocida, Histophilus somni* and *Mycoplasma bovis* in beef and non-lactating dairy cattle; and for the control of BRD in beef and non-lactating dairy cattle at high risk of developing BRD associated with *M. haemolytica, P. multocida, H. somni* and *M. bovis.*

Cattle - Multiple-Day Therapy: Enroflox 100 is indicated for the treatment of bovine respiratory disease (BRD) associated with *Mannheimia haemolytica, Pasteurella multocida* and *Histophilus somni* in beef and non-lactating dairy cattle.

Swine: Enroflox 100 is indicated for the treatment and control of swine respiratory disease (SRD) associated with *Actinobacillus pleuropneumoniae, Pasteurella multocida, Haemophilus parasuis* and *Streptococcus suis.*

RESIDUE WARNINGS:

Cattle: Animals intended for human consumption must not be slaughtered within 28 days from the last treatment. This product is not approved for female dairy cattle 20 months of age or older, including dry dairy cows. Use in these cattle may cause drug residues in milk and/or in calves born to these cows. A withdrawal period has not been established for this product in pre-ruminating calves. Do not use in calves to be processed for veal.

Swine: Animals intended for human consumption must not be slaughtered within 5 days of receiving a single-injection dose.

HUMAN WARNINGS: For use in animals only. Keep out of the reach of children. Avoid contact with eyes. In case of contact, immediately flush eyes with copious amounts of water for 15 minutes. In case of dermal contact, wash skin with soap and water. Consult a physician if irritation persists following ocular or dermal exposures. Individuals with a history of hypersensitivity to quinolones should avoid this product. In humans, there is a risk of user photosensitization within a few hours after excessive exposure to quinolones. If excessive accidental exposure occurs, avoid direct sunlight. For customer service, to obtain a copy of the Material Safety Data Sheet (MSDS) or to report adverse reactions, call Norbrook at 1-866-591-5777.

PRECAUTIONS:
The effects of enrofloxacin on cattle or swine reproductive performance, pregnancy and lactation have not been adequately determined.
The long-term effects on articular joint cartilage have not been determined in pigs above market weight.
Subcutaneous injection can cause a transient local tissue reaction that may result in trim loss of edible tissue at slaughter.
Enroflox 100 contains different excipients than other enrofloxacin products. The safety and efficacy of this formulation in species other than cattle and swine have not been determined.
Quinolone-class drugs should be used with caution in animals with known or suspected Central Nervous System (CNS) disorders. In such animals, quinolones have, in rare instances, been associated with CNS stimulation which may lead to convulsive seizures. Quinolone-class drugs have been shown to produce erosions of cartilage of weight-bearing joints and other signs of arthropathy in immature animals of various species. See Animal Safety section for additional information.

ADVERSE REACTIONS: No adverse reactions were observed during clinical trials.

ANIMAL SAFETY:
In cattle safety studies, clinical signs of depression, incoordination and muscle fasciculation were observed in calves when doses of 15 or 25 mg/kg were administered for 10 to 15 days. Clinical signs of depression, inappetance and incoordination were observed when a dose of 50 mg/kg was administered for 3 days. An injection site study conducted in feeder calves demonstrated that the formulation may induce a transient reaction in the subcutaneous tissue and underlying muscle. In swine safety studies, incidental lameness of short duration was observed in all groups, including the saline-treated controls. Musculoskeletal stiffness was observed following the 15 and 25 mg/kg treatments with clinical signs appearing during the second week of treatment. Clinical signs of lameness improved after treatment ceased and most animals were clinically normal at necropsy. An injection site study conducted in pigs demonstrated that the formulation may induce a transient reaction in the subcutaneous tissue.

Norbrook Laboratories Limited,
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I04 March 2015

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NPB news continued from page 273

National Pork Board's three-pronged antibiotic stewardship plan

1. **Education**

- The pork industry will work closely with allied partners, including swine veterinarians, feed organizations, breed associations and show-pig groups, animal-health companies, and associated commodity groups to collaborate and **develop educational materials** for our more than 60,000 pork producers and the academic and swine veterinarian community about the new FDA regulations and antibiotic stewardship.
- We will revise and give added emphasis to antibiotic stewardship in the industry's **Pork Quality Assurance Plus (PQA Plus) program** (PQA Plus is the pork industry's certification program for best practices on swine health and welfare, public health and worker health, and environmental sustainability.) This action will ensure America's pork producers understand the importance of the veterinarian-client-patient relationship and are prepared for implementation of the new changes to antibiotic use.
- Throughout 2016, the National Pork Board will use a mix of **paid and earned media opportunities** to help educate farmers about FDA's new rules and the steps required for compliance.
- **Collaboration and investment with like-minded organizations** will help increase the reach and frequency of educational messages on antibiotics.

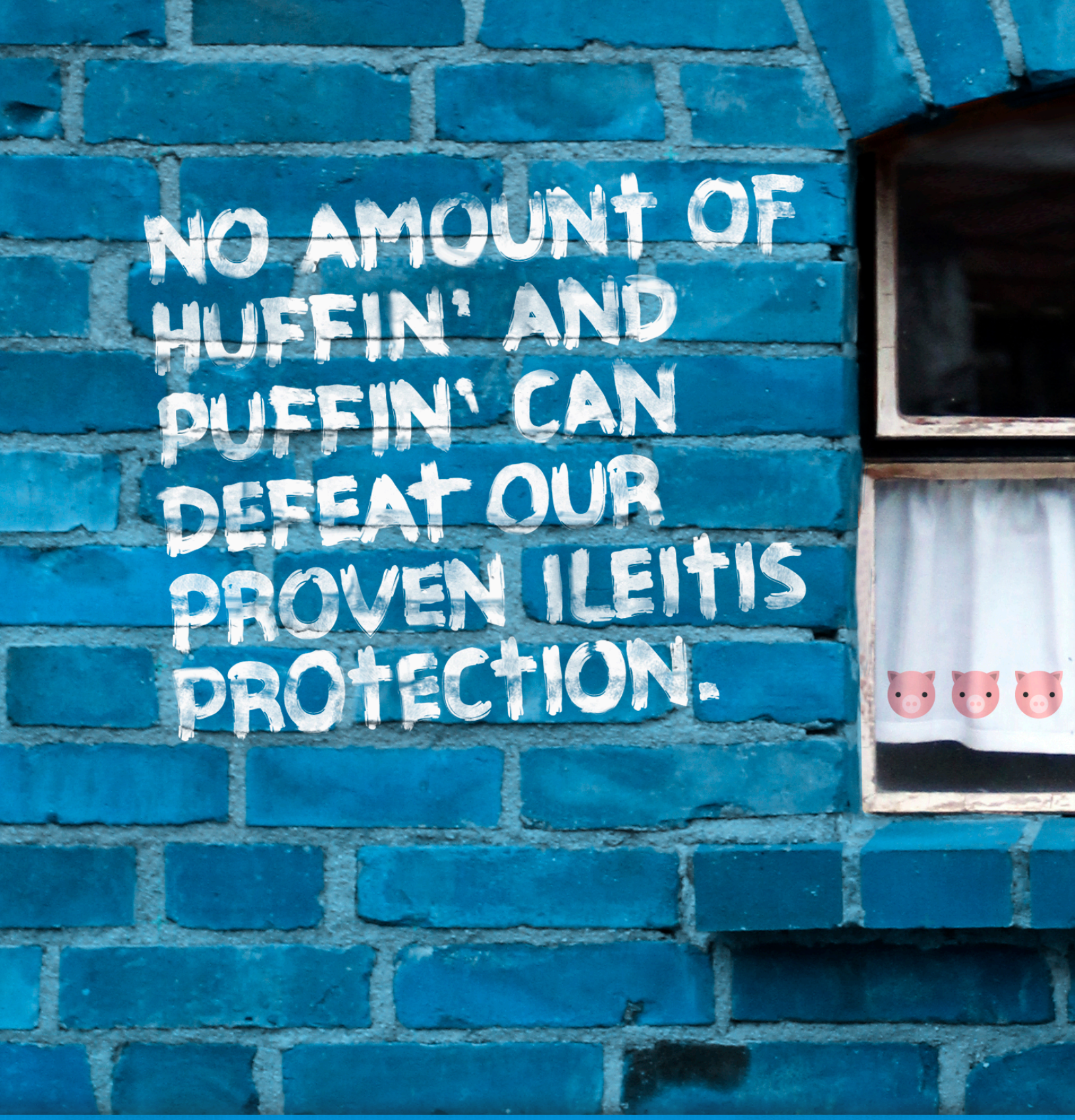
2. **Research**

- The National Pork Board will make antimicrobial use and resistance a **top research priority** in our 2016 budget. Since 2000, the Pork Checkoff has invested $5.3 million in research on the epidemiology of antimicrobial resistance, as well as efforts to define alternatives to antimicrobial use. We will invest close to a million dollars of new money in additional research in 2016.
- We will **identify specific risk assessments** to better understand the relationship between antimicrobial use in pork production and bacterial resistance. This research will augment past studies of interventions and alternatives with the goal of reducing the need for antibiotics.
- In addition to our existing producer committee of experts, we will convene a **blue-ribbon task force** of nationally recognized experts specifically focused on antibiotic use and resistance. This task force's goal will be to objectively review and provide recommendations to Pork Checkoff policies and programs.
- We will continue to work closely with federal agencies and other commodity-group partners to research and identify **models and metrics** that will provide value to the pork industry for continual improvement of antibiotic use practices.

3. **Communication outreach**

- Communication regarding antibiotics will continue to be a main emphasis for the National Pork Board to all parts of the pork chain with **special emphasis on pig farmers and the upcoming new FDA rules**.
- Pork Checkoff publications include a quarterly magazine, monthly newsletters, research e-newsletters, Web-based articles, fact sheets, **pork.org** (Antibiotics Resource Center), radio broadcasts, online videos, social media, and more.
- The National Pork Board will continue to proactively work with all national and international media that are interested in US pork production to serve as a resource about how **US pig farmers use antibiotics responsibly**.
- The National Pork Board will continue to **share the progress of our industry** with retailers and food-service companies who are interested in antibiotic use in pork production and respond to provide credible responses to their inquiries.
- Ongoing **outreach will continue with all state pork associations** that will help amplify all antibiotic news and information to farmers and state-level allied industries.
- **Collaboration will continue** with the American Association of Swine Veterinarians, the National Pork Producers Council, the American Feed Industry Association, land-grant universities, and others. All will serve to coordinate and multiply the National Pork Board's communications efforts.

For more information, contact John Johnson at **jjohnson@pork.org** or 515-223-2765.



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AASV NEWS

Nominate exceptional colleagues for AASV awards

Do you know an AASV member whose dedication to the association and the swine industry is worthy of recognition? The AASV Awards Committee requests nominations for the following five awards to be presented at the upcoming AASV annual meeting in New Orleans.

Howard Dunne Memorial Award – Given annually to an AASV member who has made a significant contribution and rendered outstanding service to the AASV and the swine industry.

Meritorious Service Award – Given annually to an individual who has consistently given time and effort to the association in the area of service to the AASV members, AASV officers, and the AASV staff.

Swine Practitioner of the Year – Given annually to the swine practitioner (AASV member) who has demonstrated an unusual degree of proficiency in the delivery of veterinary service to his or her clients.

Technical Services/Allied Industry Veterinarian of the Year – Given annually to the technical services or allied industry veterinarian who has demonstrated an unusual degree of proficiency and effectiveness in the delivery of veterinary service to his or her company and its clients, as well as given tirelessly in service to the AASV and the swine industry.

Young Swine Veterinarian of the Year – Given annually to a swine veterinarian who is an AASV member, 5 years or less post-graduation, who has demonstrated the ideals of exemplary service and proficiency early in his or her career.

Nominations are due December 15. The nomination letter should specify the award and cite the qualifications of the candidate for the award. Submit to AASV, 830 26th Street, Perry, IA 50220-2328: Fax: 515-465-3832, E-mail: **aasv@aasv.org**.

Call for abstracts – AASV 2016 Student Seminar

Veterinary Student Scholarships

The American Association of Swine Veterinarians announces an opportunity for veterinary students to make a scientific presentation during the Student Seminar at the AASV Annual Meeting in New Orleans, Louisiana, on Sunday, February 28, 2016. 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 (2015-2016) student member of the AASV at the time of submission, and must not have graduated from veterinary school prior to February 28, 2016. Submissions are limited to one (1) abstract per student.

Abstracts and supplementary materials must be ***received*** by Dr Alex Ramirez (**alex@aasv.org**) by **11:59 PM Central Daylight Time** on **Monday, September 21, 2015** (firm deadline). All material must be submitted electronically. Late abstracts will not be considered. Students should receive an e-mail confirming the receipt of their submission. If they do not receive this confirmation e-mail, they must contact Dr Alex Ramirez (**alex@aasv.org**) by Wednesday September 23, 2015, with supporting evidence that the submission was made in time, otherwise the submission will not be considered for judging. The abstracts will be reviewed by an unbiased professional panel consisting of a private practitioner, an academician, and an industry veterinarian. Fifteen abstracts will be selected for oral presentation in the Student Seminar at the AASV Annual Meeting. Students will be notified by October 15, 2015, and those selected to participate will be expected to provide the complete paper or abstract, reformatted for publication, by November 16, 2015.

As sponsor of the Student Seminar, Zoetis provides a total of $20,000 in support to fund travel stipends and the top student presenter scholarship. The student presenter of each paper selected for oral presentation receives a $750 stipend to help defray the costs of attending the AASV meeting.

Each veterinary student whose paper is selected for oral presentation competes for one of several veterinary student scholarships awarded through the AASV Foundation. The oral presentations will be judged to determine the amount of the scholarship awarded. Zoetis funds the $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 $2500 each for 2nd through 5th place, $1500 each for 6th through 10th place, and $500 each for 11th through 15th place.

Abstracts that are not selected for oral presentation in the Student Seminar will be considered for participation in a poster session at the annual meeting. Zoetis and the AASV fund a stipend of $250 for each student who is selected and participates in the poster presentation. In addition, the presenters of the top 15 poster abstracts compete for awards ranging from $200 to $500 in the Veterinary Student Poster Competition sponsored by Newport Laboratories.

Complete information for preparing and submitting abstracts is available on the AASV Web site at **www.aasv.org/annmtg/2016/studentseminar.htm**. Please note: the rules for submission should be followed carefully. For more information, contact the AASV office (Tel: 515-465-5255; Fax: 515-465-3832; E-mail: **aasv@aasv.org**).

Call for submissions – Industrial Partners

The American Association of Swine Veterinarians (AASV) invites submissions for the Industrial Partners portion of the 47th AASV Annual Meeting, to be held February 27-March 1, 2016, in New Orleans, Louisiana. This is an opportunity for commercial companies to make brief presentations of a technical, educational nature to members of the AASV.

As in the past, the oral sessions will consist of a series of 15-minute presentations scheduled from 1:00 to 5:00 PM on Sunday afternoon, February 28. A poster session will take place on the same day. Poster authors will be required to be stationed with their poster from 12:00 noon until 1:00 PM, and the posters will remain on display throughout the afternoon and the following day for viewing by meeting attendees.

Restricted program space necessitates a limit on the number of presentations per company. Companies that are members of the *Journal of Swine Health and Production* Industry Support Council (listed on the back cover of each issue of the journal) may submit two topics for oral presentation. Sponsors of the AASV e-Letter may submit an additional topic for oral presentation. All other companies may submit one topic for oral presentation. In addition, every company may submit one topic for poster presentation (poster topics may not duplicate oral presentations). All topics must represent information not previously presented at the AASV Annual Meeting or published in the meeting proceedings.

Topic title, a brief description of the presentation content, and presenter information (name, address, telephone and fax numbers, e-mail address) must be received in the AASV office by October 1, 2015. Please identify whether the submission is intended for oral or poster presentation. Send submissions via mail, fax, or e-mail to Commercial Sessions, AASV, 830 26th Street, Perry, IA 50220-2328; Fax: 515-465-3832; E-mail: **aasv@aasv.org**.

Authors will be notified of their acceptance by October 15, 2015, and must submit the paper for publication in the meeting proceedings by November 16, 2015. All presentations – oral and poster – will be published in the proceedings of the meeting. Papers for poster presentations are limited to one page of text plus one table or figure. Papers for oral presentations may be up to five pages in length (including tables and figures), when formatted according to the guidelines provided to authors upon acceptance of their presentation. Companies failing to submit papers in a timely manner may not be eligible for future participation in these sessions.

FDA issues Veterinary Feed Directive final rule

On June 2, the US Food and Drug Administration (FDA) issued the Veterinary Feed Directive (VFD) final rule, an important piece of the agency's overall strategy to promote the judicious use of antimicrobials in food-producing animals. This strategy will bring the use of these drugs under veterinary supervision so that they are used only when necessary for assuring animal health. The VFD final rule outlines the process for authorizing use of VFD drugs (animal drugs intended for use in or on animal feed that require the supervision of a licensed veterinarian) and provides veterinarians in all states with a framework for authorizing the use of medically important antimicrobials in feed when needed for specific animal-health purposes. The final rule becomes effective October 1, 2015. For specific details regarding the veterinarian's role in the VFD process, see the "Advocacy in action" article in this issue of the *Journal of Swine Heath and Production*.

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1. Thacker, B. and Creel, J. PCV2 vaccination update: Field experiences. 2015.
2. Thacker, B., Blomme, R., Holtkamp, D. and Creel, J. Field comparison of PCV2 vaccines: A retrospective production data analysis. Proc. 45th Annual Meeting of the American Association of Swine Veterinarians, Dallas, Texas. 2014. pp. 139-143.

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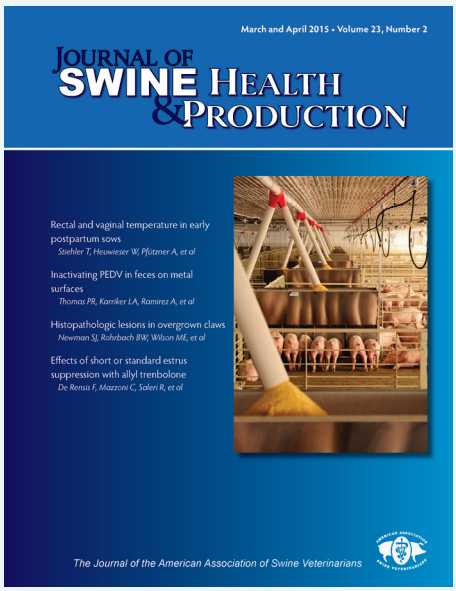

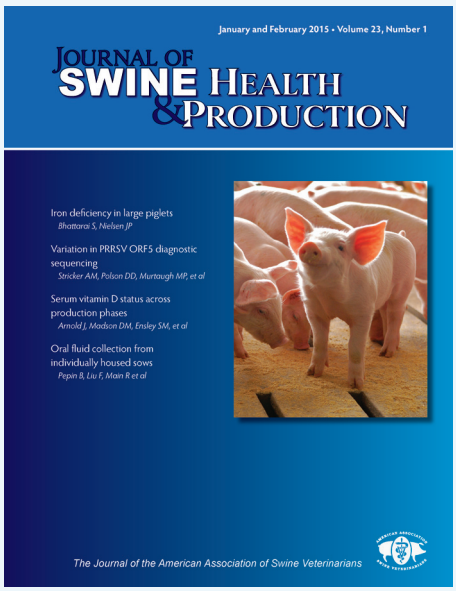

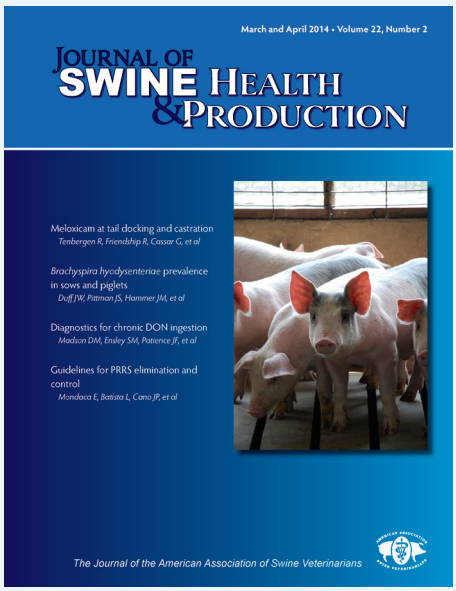

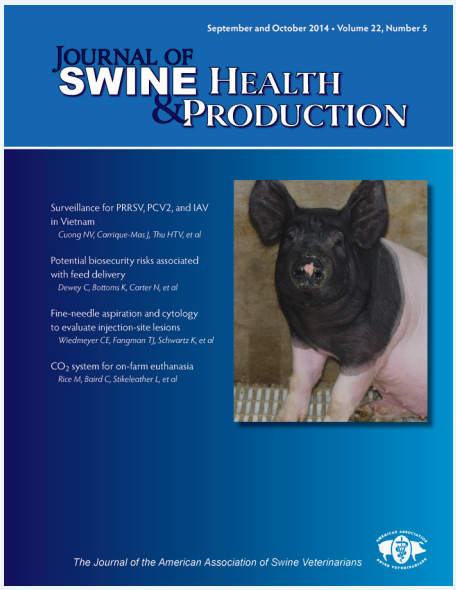

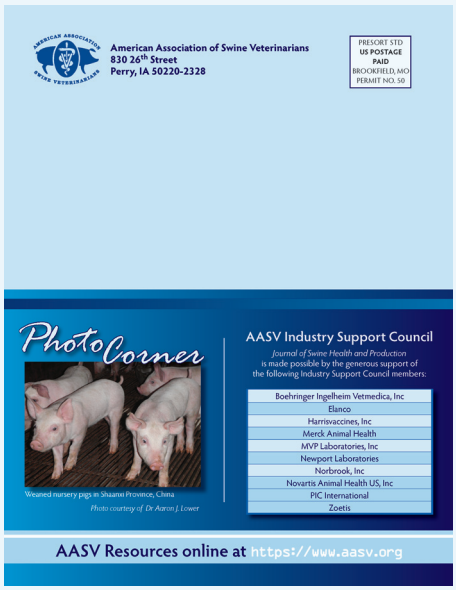

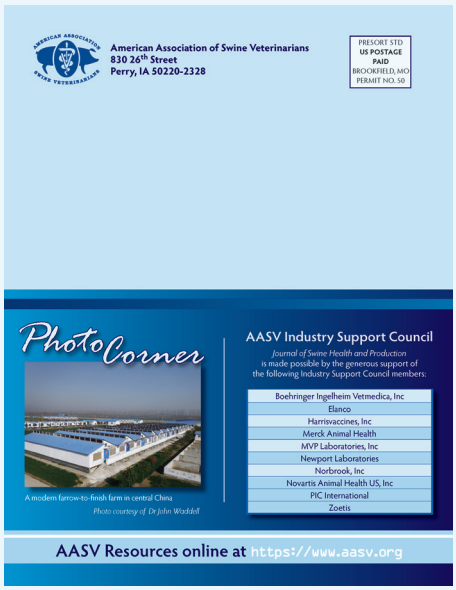

The *Journal of Swine Health and Production* would like to publish digital photographs submitted by our readers. Images used either on the front cover or in the photo corner on the back cover are to represent healthy pigs and modern production facilities. Please ensure that the photos do not include people. Select the largest image size available on your camera, of the quality or compression that allows you to store the fewest images on a given memory card. Do not resize, crop, rotate, or color-correct the image prior to submission to the journal. Please send the images by e-mail attachment to **tina@aasv.org**. Tina will also need to know your name, affiliation, and the approximate location of the subject, or other details that you would like to submit that describe the image.

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BOOTH #55

Veterinary Feed Directive – The veterinarian's role

As everyone hopefully knows by now, the United States Food and Drug Administration (FDA) has issued a final rule revising the regulations governing the Veterinary Feed Directive (VFD).[1] These changes to the VFD are a key part of FDA's strategy to ensure these drugs are used judiciously and only when appropriate for specific animal-health purposes.

These regulatory changes, in conjunction with the implementation of Guidance for Industry (GFI) #209[2] and GFI #213,[2] are designed to eliminate the growth-promotion uses of medically important antimicrobials and enhance veterinary oversight of antimicrobial use in livestock. Pharmaceutical manufacturers have agreed to voluntarily remove the feed efficiency indications from affected product labels by December 2016. The new VFD rules take effect on October 1, 2015. This means that after that date, the use of any feed-grade antimicrobials with a VFD label will be subject to the new rules. This includes tilmicosin, florfenicol, and avilamycin, which are all currently VFD drugs labeled for use in swine.

A list of "medically important" antimicrobials can be found in FDA's Guidance #152 Appendix A.[2] Basically, all swine antibiotics will be affected except bacitracin, carbadox, bambermycin, ionophores, and tiamulin.

These antibiotics will remain available for growth promotion or over-the-counter (OTC) distribution or both.

Veterinary responsibilities

In order to comply with the new VFD rules, the veterinarian must

- Be licensed and operating in the course of normal practice in compliance with all state and federal regulations;
- Write VFD orders in the context of a veterinary-client-patient relationship (VCPR) as discussed below;
- Only issue a VFD that is in compliance with approved use;
- Prepare a written (nonverbal) VFD including the veterinarian's signature;
- Ensure the VFD includes all required information (shown in Box 1). There is no FDA-approved standardized VFD form;
- Include certain drug-specific information for each VFD drug when authorizing drug combinations that include more than one VFD drug;
- When issuing a VFD combining VFD and OTC drugs, include on the VFD order an affirmation of intent either to restrict authorized use only to the VFD drug cited on the VFD form or to allow the use of the cited VFD drug in an approved combination with one or more OTC drug(s);
- Provide the distributor and client with a copy of the VFD order either in hardcopy or electronic form or by fax;
  - Retain the original VFD for 2 years (the client and distributor must likewise retain their copies for 2 years); and
    - Provide the VFD orders for inspection and copying by FDA upon request.

In addition, it should be emphasized that extra-label use of feed-grade antimicrobials remains ILLEGAL for both veterinarians and producers.

Veterinary-client-patient relationship

A valid VCPR must exist between the veterinarian, the client, and the animals to be treated in order to issue a VFD. However, there are numerous versions of the VCPR requirements, including versions associated with federal regulations governing extra-label drug use, the American Veterinary Medical Association's model practice act, and state veterinary practice acts. For the purposes of issuing a VFD, FDA defaults to the VCPR requirements defined in the state veterinary practice act provided those requirements meet the following minimum standards:

1. The veterinarian has engaged with the client to assume responsibility for making clinical judgments about patient health,
2. The veterinarian has sufficient knowledge of the patient by virtue of patient examination, visits to the facility where the patient is managed, or both, and
3. The veterinarian is available to provide for any necessary follow-up evaluation or care.

If the state practice act either does not include a VCPR requirement or does not meet those minimum standards, the VCPR requirement to issue a VFD defaults to the VCPR as defined in association with the Animal Medicinal Drug Use Clarification Act (21 CFR § 530.3[i]).[3] The FDA will compile a list of states that require a VCPR that includes the key elements of the federally defined VCPR in order for a veterinarian to issue a VFD. This list will be provided online.

Additional changes of interest

1. The veterinarian must assign an expiration date to the VFD. This date refers to the length of time during which the VFD is valid and the producer can feed the VFD feed, not the date on which the drug expires. The expiration date must comply with the VFD expiration date indicated on the VFD drug label if

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Advocacy in action continued from page 283

the product specifies an expiration date (the veterinarian cannot deviate from this date). If the product label does not indicate a specific date, the veterinarian must assign a date not to exceed 6 months from the date of issue.

2. There has been much discussion regarding refills. The veterinarian must specify the number of refills if refills are allowed according to the VFD drug label. Currently, there are no approved medications for which refills are allowed on the label. Thus, refills are illegal unless a future product approval allows refills.
3. The veterinarian issuing the VFD must comply with the veterinary practice act regulations in effect in the state in which the animals reside that are to receive the VFD feed.
4. In contrast to the current VFD requirements, the new rule requires that the veterinarian estimate the number of animals that will receive the VFD feed, rather than the volume of feed that needs to be produced.
5. In another change, the VFD may now be transmitted to the feed manufacturer or distributor and to the client electronically (eg, by fax or through a compliant third-party electronic database, but not by telephone) instead of only by hard copy. The veterinarian retains the original copy in whatever format it was generated. The distributor and client copies may be kept either as electronic copies or hard copy. All copies of the VFD must be retained for a minimum of 2 years by the veterinarian, client, and distributor.
6. If any drug in an approved combination drug product is a VFD drug, the use of that combination must comply with the VFD rule.
7. The veterinarian may write a VFD that covers animals in multiple locations (animal-production facilities) to be fed the VFD feed by the expiration date on the VFD, provided he or she can do so in compliance with professional licensing and practice standards and provided the VFD feed is supplied to such multiple locations by a single feed manufacturer (distributor).

Information required on VFD

1. The veterinarian's name, address, and telephone number;
2. The client's name, business or home address, and telephone number;
3. The premises at which the animals specified in the VFD are located;
4. The date of VFD issuance;
5. The expiration date of the VFD;
6. The name of the VFD drug(s);
7. The species and production class of animals to be fed the VFD feed;
8. The approximate number of animals to be fed the VFD feed by the expiration date of the VFD;
9. The indication for which the VFD is issued;
10. The concentration of VFD drug in the feed and duration of use;
11. The withdrawal time, special instructions, and cautionary statements necessary for use of the drug in conformance with the approval;
12. The number of reorders (refills) authorized, if permitted by the drug approval, conditional approval, or index listing;
13. The statement "Use of feed containing this Veterinary Feed Directive (VFD) drug in a manner other than as directed on the labeling (extra-label use), is not permitted;"
14. An affirmation of intent for combination VFD drugs as described in 21 CFR 558.6(b)(6); and
15. The veterinarian's electronic or written signature.

8. Electronic VFD orders issued by veterinarians must be compliant with 21 CFR part 11,[4] and electronic VFD orders received *and* electronically stored by distributors and clients must also be compliant with 21 CFR part 11,[4] which does not apply to paper records that are, or have been, transmitted by electronic means (such as facsimile, e-mail attachments, etc).
9. There are additional requirements to meet if a veterinarian also distributes VFD feed.

In summary, effective October 1, 2015, all VFD-labeled products must comply with the new VFD rules. I have attempted to highlight the key responsibilities of the veterinarian, but I urge you to familiarize yourselves with the regulation. The FDA has compiled a fact sheet describing the background and reasons for the changes to the VFD.[5] The agency has also published an additional draft guidance document, GFI #120,[6] which answers many of the most frequently asked questions. All these documents can be found online as referenced.

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6. US Food and Drug Administration. Guidance #120 VFD Frequently Asked Questions. Available at: http://www.fda.gov/downloads/AnimalVeterinary/GuidanceComplianceEnforcement/GuidanceforIndustry/UCM052660.pdf. Accessed 14 July 2015.

Harry Snelson, DVM
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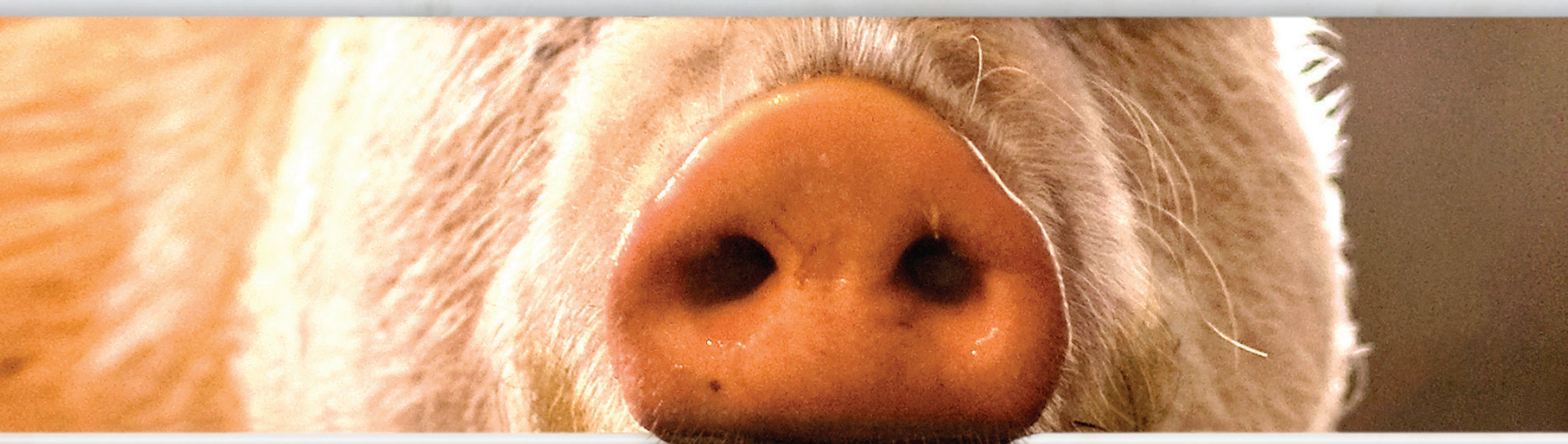

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Web: http://www.passionforpigs.com

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Michigan State University
Tel: 517-355-8398
E-mail: rozeboom@msu.edu
Web: http://animalmortmgmt.org

The 4th Leman China Swine Conference
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Program Director: Frank Liu
Veterinary Diagnostic Laboratory
1333 Gortner Avenue, St Paul, MN 55108
Tel: 612-625-2267; Fax: 612-624-8707
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Web: http://www.cvm.umn.edu/lemanchina/

International Conference on Feed Efficiency in Swine
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E-mail: niaa@animalagriculture.org
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Web: http://www.extension.iastate.edu/registration/events/conferences/swine/

2015 North American PRRS Symposium
December 5-6, 2015 (Sat-Sun)
Intercontinental Hotel
505 N Michigan Avenue, Chicago, Illinois

For more information:
Web: http://ksvma.site-ym.com/?NAPRRS

American Association of Swine Veterinarians 47th Annual Meeting
February 27-March 1, 2016 (Sat-Tue)
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For more information:
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