November and December 2018 • Volume 26, Number 6

Journal of Swine Health & Production

Pneumonia evaluation at slaughter using PCR and histopathology in Argentina
Cappuccio JA, Dibarbora M, Bessone FA, et al

M hyo DNA detection by qPCR in diagnostic cases submitted to the ISU VDL from 2004 to 2016
Rawal G, Arruda P, Rademacher C, et al

Evident similarity of postparturient dysgalactia to subclinical coliform mastitis
Pospischil A, Bertschinger HU

The Journal of the American Association of Swine Veterinarians

Journal of Swine Health and Production

(**ISSN 1537-209X**) Volume 26, Number 6; November and December 2018

The *Journal of Swine Health and Production* is published by the American Association of Swine Veterinarians.

The *Journal of Swine Health and Production* is a refereed publication and is a benefit of membership in the American Association of Swine Veterinarians. Subscriptions ($US) are available to nonmembers at $145.00 per year (six issues) for United States, Canada, and Mexico. The cost is $180.00 for all countries outside North America. For inquiries regarding membership or subscriptions, please contact

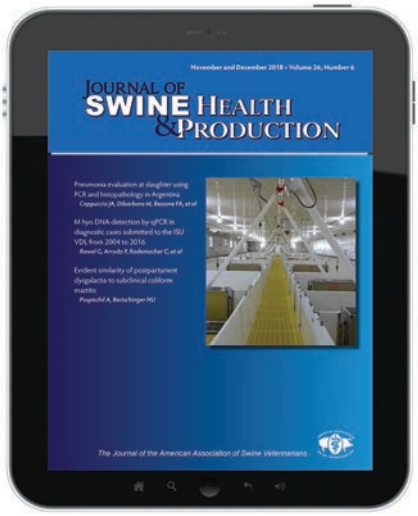
AASV
830 26th Street, Perry, IA 50220-2328
Tel: 515-465-5255; Fax: 515-465-3832
Email: aasv@aasv.org

Editorial questions, comments, and inquiries should be addressed to Karen Richardson, Publications Manager:
Tel: 519-856-2089;
Email: jshap@aasv.org

DISCLAIMER

Scientific manuscripts published in the *Journal of Swine Health and Production* are peer reviewed. However, information on medications, feed, and management techniques may be specific to the research or commercial situation presented in the manuscript. It is the responsibility of the reader to use information responsibly and in accordance with the rules and regulations governing research or the practice of veterinary medicine in their country or region.

Journal of Swine Health and Production is indexed in ISI® *Focus On: Veterinary Science & Medicine,*® and in *CAB Abstracts*, Euroscience VETLINE on CD-ROM



Download this issue to your iPad or Android tablet at
www.aasv.org/shap/issues/v26n6/v26n6jshap.pdf

About the cover…

Ontario nursery ready for new pigs.

Photo courtesy of Terri O'Sullivan

AASV Resources online at www.aasv.org

Author guidelines
www.aasv.org/shap/guidelines.pdf

Journal of Swine Health and Production
www.aasv.org/shap.html

Membership information
www.aasv.org/aasv/membership.html

Subscription information
ecom.aasv.org/journal

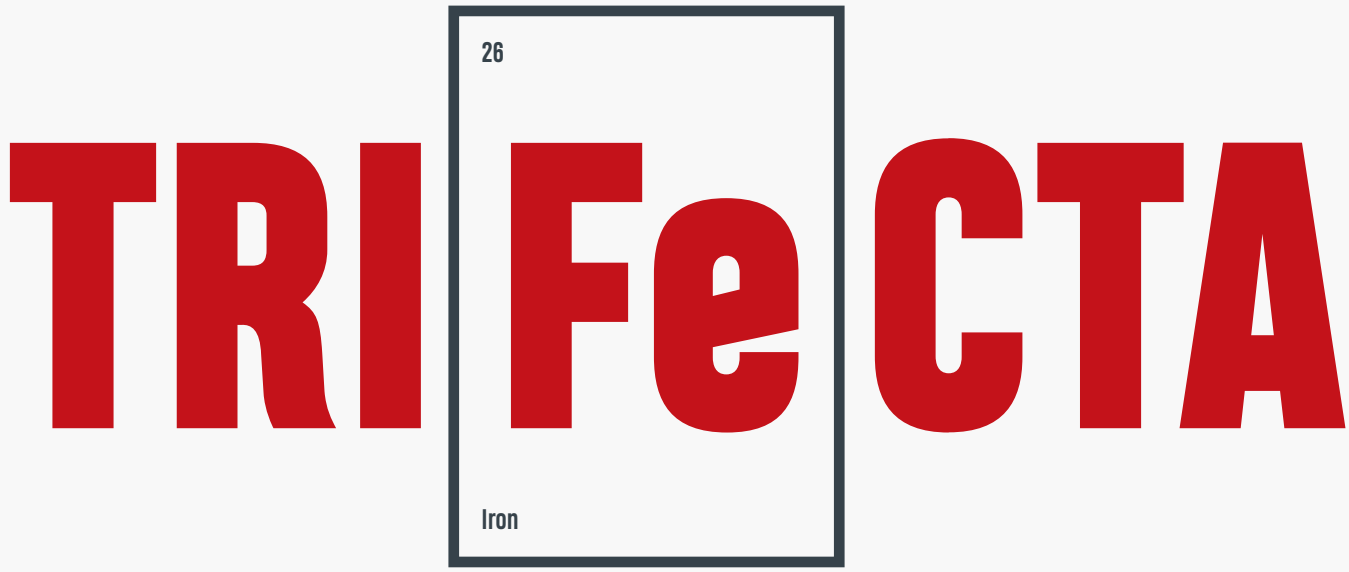
Upcoming meetings
www.aasv.org/meetings

Table of contents

"I would personally like to thank all the peer-reviewers for their hard work and contributions to the journal over the past year. Thank you!"

quoted from the Executive Editor's message, page 301

uniferon® 200



Optimizes Average Daily Weight Gain

Prevents Iron Deficiency Anemia

Meets Both Veterinary and Human Drug Standards

Not only that, but according to a peer-reviewed article recently published in the *Journal of Swine Health and Production*[1], Uniferon® is the only injectable product for use in baby pigs that showed undetectable levels of arsenic and lead.

Get the purest form of iron. Get the trifecta. Get Uniferon®.

[1] Radke, S.L., Olsen, C.W., Ensley, S.M., (2018) Elemental impurities in injectable iron products for swine. The Journal of Swine Health and Production, 26(3).

All iron is not created equal.
Learn more about the study at **purepigiron.com**.

PHARMACOSMOS

TM-046-00

Reframing the problem: African swine fever outbreaks

With our global experience with African swine fever, classical swine fever, porcine epidemic diarrhea virus, and Seneca Valley virus, it seems obvious we need to do everything we can to understand how swine foreign animal diseases are being transmitted around the world. One of the beautiful things about the human mind is that it can be refocused on new problems. It's a stark contrast to facilities and machines that have defined purposes or limits.

Taking time to brainstorm and figure out the unknown links for disease transmission can be a frustrating but ultimately rewarding process. So how can we refocus our minds to address new problems? Thomas Wedell-Wedellsborg has suggested that we often diagnose the problem too quickly in our desire to get to action we think will help.[1] The result of this is that we spend our time implementing changes that will sadly be ineffective. Traditional problem-solving tools like Six Sigma, Root Cause Analysis, and 5 Whys are very helpful but can also lead us in the wrong direction because many problems are multi-causal. Likewise, we rely on experimentation too much. Experimentation is great at validating better outcomes, but it can limit us from identifying what may be the best option. The solution to this is problem reframing. What is problem reframing?

There are seven practices to reframing a problem:

1. Establish legitimacy of the reframing process. Your problem-solving team needs to have a basic understanding of this approach to minimize the frustration of those that want action now.
2. Bring outsiders into the discussion. Outsiders can help you avoid falling in love with a favorite solution, also known as group think. Look for "boundary spanners," those who understand but are not fully part of your world. Seek those who will speak freely. Expect input on the problem, not solutions.
3. Get people's definitions in writing. Pay close attention to wording because slight differences can elucidate a different perspective on a problem.
7. Ask what's missing. Don't jump to debating details of what's already been identified.
5. Consider multiple categories of problems. Are there incentives, expectations, attitudes or usability to consider, not just the hard science?
6. Analyze positive exceptions. Look for situations where there isn't a problem and delve deeply into the reasons why.
7. Question the objective. Clarifying the objectives around the problem can help focus solutions with maximal impact.

This method requires different listening skills. It requires us to hold back from offering solutions right away. A deep understanding of the problem is gained from really listening to how people describe and talk about their problem. Really effective thinking and traditional testing can result in radically more effective change. Try this method on some of the problems you've been frustrated with, or the new one on our minds right now – African swine fever.

"Taking time to brainstorm and figure out the unknown links for disease transmission can be a frustrating but ultimately rewarding process."

C. Scanlon Daniels, DVM
AASV President

Reference

1. Wedell-Wedellsborg, T. Are You Solving the Right Problems? Harvard Business Review. 2017;Jan-Feb:76-83.

Enroflox® 100 (enrofloxacin)

Now Indicated for *E. coli*

Approved for pigs of **all** ages

One-dose Swine Respiratory Disease (SRD) treatment

Same active ingredient and dosing regime found in Baytril® 100

Same SRD pathogens as Baytril® 100

Enroflox® 100
(enrofloxacin)

Approved for the treatment & control of Swine Respiratory Disease (SRD) & Control of Colibacillosis

Associated with *Actinobacillus pleuropneumoniae* (APP), *Pasteurella multocida*, *Haemophilus parasuis* and *Streptococcus suis*

Federal law restricts this drug to use by or on the order of a licensed veterinarian. Federal law prohibits the extra-label use of this drug in food-producing animals. To assure responsible antimicrobial drug use, enrofloxacin should only be used as a second-line drug for colibacillosis in swine following consideration of other therapeutic options. 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.

FOR VETERINARY USE ONLY

www.norbrook.com

0918-495-I05A

ANADA 200-495, Approved by FDA

Enroflox® 100 (enrofloxacin) — **100 mg/mL Antimicrobial Injectable Solution**

For Subcutaneous Use In Beef Cattle And Non-Lactating Dairy Cattle
For Intramuscular Or Subcutaneous Use In Swine
Not For Use In Female Dairy Cattle 20 Months Of Age Or Older Or In Calves To Be Processed For Veal

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.

To assure responsible antimicrobial drug use, enrofloxacin should only be used as a second-line drug for colibacillosis in swine following consideration of other therapeutic options.

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, Streptococcus suis, Bordetella bronchiseptica* and *Mycoplasma hyopneumoniae*. Enroflox 100 is indicated for the control of colibacillosis in groups or pens of weaned pigs where colibacillosis associated with *Escherichia coli* has been diagnosed.

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 Safety Data Sheet (SDS) 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:
Cattle: 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.

Swine: Subcutaneous Safety: 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.

Intramuscular Safety: Transient decreases in feed and water consumption were observed after each treatment. Mild, transient, post-treatment injection site swellings were observed in pigs receiving the 37.5 mg/kg BW dose. Injection site inflammation was found on post-mortem examination in all enrofloxacin-treated groups.

Norbrook Laboratories Limited,
Newry, BT35 6PU, Co. Down, Northern Ireland

I05 October 2017

The coming storm

I am sitting in my office in Perry, Iowa watching the news stories chronicling the advancing hurricane named Florence. If measured by the number of news correspondents giving reports from a North Carolina beach setting, then Florence must be a huge storm. By the time you read this, we will know just how big it was and how much damage actually occurred. It is awe-inspiring to consider this massive force of nature as it bears down on the east coast of the United States.

There is another force of nature approaching the United States and this one is not weather related. It is the disease known as African swine fever (ASF). As I write this, a case of ASF has just been confirmed in Belgium. It is the first case in Belgium since 1985. An alarming fact about this case is that it appears to be a long distance from other known infected countries. At this point in time there is little known about the mode of spread in this case, but long distances between cases can indicate a pandemic.

Other ASF-infected countries include Africa, China, several Eastern European countries, and Russia. I won't bore you with too many of the details of this virus. Suffice it to say, it is a hardy and lethal agent when introduced into pigs. There is no vaccine currently available. In the United States, ASF is considered a foreign animal disease that will immediately halt movements of pigs as well as the export of pork. There is no other way to describe an ASF incursion into the United States than devastating. Besides the potential mass casualties of infected pigs, there will be wholesale depopulation of all pigs within a zone around infected farms.

"Unfortunately, we don't have ASF radar to inform us about the coming storm, so we are left to do the best we can with the resources and resolve we can muster."

The lessons learned in 2013 and 2014 with the spread of porcine epidemic diarrhea virus (PEDV) are still fresh. The initial arrival of PEDV occurred nearly simultaneously in multiple herds over a wide geographic area. Research since then has proven the hypothesis that PEDV can survive the trip in certain feedstuffs from the Far East to the heartland of the United States. I will leave it to you to connect the dots. That disease also demonstrated that the industry we have built to efficiently move pigs and inputs is also quite capable of rapidly disseminating highly infectious viral diseases. Market trucks, lairages, feed mills, and a plethora of fomites that came in contact with the virus were rapidly contaminated with PEDV.

In the news stories about Florence, you could see governmental agencies (eg, Federal Emergency Management Agency), non-governmental organizations (eg, Red Cross), and industries (eg, utilities) start marshaling and staging resources and personnel for the post-hurricane task of rescue and recovery. The scale of this planning and deployment was impressive and time will tell how effective it was. Likewise, an introduction of ASF into the United States will require a massive effort to bring all needed resources together to stop the spread of this disease.

One advantage we have with ASF over a hurricane is that there is, at least as of this writing, an opportunity for us to prevent ASF from entering the United States. Prevention should be on the mind of everyone within the swine industry. It can't be a "wink and a nod" and then go on with business as usual. We all need to confront the reality of the ASF virus and its ability to survive in and on several contaminated fomites including meat products, feedstuffs, vehicles, and equipment.

The role of feed in viral spread can be debated but there is a strong case to be made that contaminated feed is a serious and real risk. The brutal fact is the US pork industry imports a substantial number and quantity of feed additives and ingredients from countries with ASF. This is a wide-open door for incursion unless the US Department of Agriculture, the US Food and Drug Administration, and the feed industry either prevent or mitigate ASF virus-contaminated feed from entering the United States.

Unfortunately, we don't have ASF radar to inform us about the coming storm, so we are left to do the best we can with the resources and resolve we can muster. Pork producer organizations and the AASV are active in assessing the risks posed by ASF and either implementing the actions needed to thwart the entry of the virus or advocating for action by the appropriate governmental agencies. This also includes the funding of research to fill the gaps in knowledge. Swine veterinarians working on farms can play a vital and active role in identifying risks and improving biosecurity aimed at the exclusion of the virus in all possible fomites coming onto a farm.

Prevention of ASF from entering the United States is a daunting and difficult task but that fact can't be allowed to develop into an excuse. George Washington Carver put it this way, "Ninety-nine percent of the failures come from people who have the habit of making excuses." After the storm passes, let's be glad to have done our best to prevent ASF and not be the ones still making excuses.

Tom Burkgren, DVM
Executive Director

Remembering and Giving Thanks

Once again, I really enjoyed reading the manuscript contributions that have formed the scientific component of this issue of the journal. I hope you also enjoy reading this edition of the *Journal of Swine Health and Production* (JSHAP). The papers and messages within this issue are the result of a major team effort that bring applied and diverse topics to our swine information library. As many of you know, the November-December issue of JSHAP publishes a list of all the reviewers who have graciously reviewed manuscripts for the journal over the previous year. I invite you to turn to that list and recognize all those individuals who have volunteered their time and expertise to provide a peer-review. If you see one of these peer-reviewers in your daily travels or at a conference, please pass on a thank-you.

I would personally like to thank all the peer-reviewers for their hard work and contributions to the journal over the past year. Thank you!

In some of my previous messages I have described the peer-review process for JSHAP.[1] I wanted to revisit this topic so that we remember how many people are involved in the process and the time commitment required. The November-December issue is also a timely issue for giving thanks and for remembering, as many of us celebrate Thanksgiving, Remembrance Day, Veterans Day, and many other significant holidays.

The peer-review process of scientific manuscripts can be quite variable from journal to journal. Regardless of the review system in place, the process takes a considerable amount of time and hard work. As a starting point to gaining an appreciation for the time commitment in the length of the process, I ask you to consider the two dates published on the online version of a manuscript: the received date and the accepted date (Figure 1). The received dates published in JSHAP reflects the date the manuscript is submitted to the journal office and the accepted dates represent the date of conditional acceptance for publication. You will see that the time frame between these two dates varies greatly from manuscript to manuscript.

Why do we keep track and publish this information? There are multiple reasons, but one important reason from an administration standpoint is to help the journal monitor the length of time a manuscript takes to go through the review and editing process. We like to monitor this information and see if there are ways or areas where we can streamline the process.

The length of time from submission to acceptance and then publication can depend on many things and it is not unusual for JSHAP to experience delays in the review process. It is a coordinated effort to keep things moving while balancing author and reviewer schedules, eg, changes in personal schedules and reviewer availability during the process. Not to mention the journal's timelines and deadlines, international time zones delaying communications, holidays, etc. I think you get the idea. For all these reasons, JSHAP does not have a guaranteed publication timeline as there are many factors out of our direct control. However, we are sensitive to timely publication and strive to keep timelines reasonable. Karen Richardson, our publications manager, looks after keeping track of the manuscripts, timelines, and people.

> *"Two areas where authors most often fail to follow the guidelines is with providing information regarding animal use and general formatting."*

To elaborate further, the specific review process begins with me, the executive editor. I read the manuscript and decide if it is within the scope of the journal. If the manuscript is out of the journal's scope, it is returned to the author and not accepted for a full peer-review. Additionally, manuscripts are returned to the author for revision if they have not followed the author guidelines, further delaying the review process. Two areas where authors most often fail to follow the guidelines is with providing information regarding animal use and general formatting. These steps and attention to detail are important so that we don't overwhelm our reviewers with requests to review poorly presented manuscripts or manuscripts that do not suit the journal.

Once the manuscript is accepted for review, I request one of the members of the editorial board to act as a lead reviewer. This is a critical component of the review process as the lead reviewer will guide the review process for the individual manuscript from here and help to narrow down the reviewer search for the submission. Typically, 2 or 3 additional reviewers are obtained for each manuscript and the reviewers are given 3 to 4 weeks to return their reviews. The work of the editorial board members brings a wealth of expertise to the review process, the journal, and the body of published scientific literature in general.

I would like to personally thank all the lead reviewers, past and present, for their contributions to the journal. Thank you!

Executive Editor's message continued on page 303

CUSTOM VACCINES PROVIDE MORE OPTIONS TO PREVENT DISEASE

In 2017, Newport Laboratories officially became part of Boehringer Ingelheim, uniting the strengths of both companies to give producers and veterinarians access to a more well-rounded and comprehensive portfolio of animal health solutions.

Located in Worthington, Minnesota, Newport Laboratories has been in business for more than 20 years and is pioneering technological advancements in diagnostics and research for creating autogenous, or custom-made vaccines.

WHY CUSTOM VACCINES?

Custom-made vaccines can help fill the gaps in disease prevention, complementing the protection producers are already getting from commercial vaccines. When a herd has an infection a commercial vaccine can't address, a custom vaccine may be the solution.

Using molecular-biology techniques to quickly diagnose complex diseases and identify the specific pathogen strains causing infections on farms, Newport Laboratories creates custom vaccines that provide targeted protection against those strains.

But how the company goes about selecting the isolates from which it produces a vaccine is one of the things that makes Newport Laboratories unique. Newport Laboratories is the only custom-vaccine manufacturer that conducts its own in-house research for its proprietary database of field-sampled isolates. Newport Laboratories references customer isolates against this data- base to help determine which to use in a vaccine. A field sample may come in containing many different isolates, but not all of them may be good candidates for vaccine creation. Newport Laboratories has the advantage over other custom-vaccine manufacturers of being able to cross-reference field samples against its database and select the specific isolates that have proven themselves to be the most effective at triggering a protective immune response.

ADVANCED DIAGNOSTICS

In addition to custom-vaccine creation, Newport Laboratories operates one of the largest private diagnostic laboratories in the country and is leading the way in both traditional diagnostic services and new molecular diagnostic tools and techniques.

One such tool is metagenomics, which allows Newport Laboratories to provide a complete snapshot of pathogens faster than traditional diagnostics by using fecal or tissue samples instead of cultures. This gives veterinarians the answers they need in less time and with less effort.

Newport Laboratories' diagnostic laboratory uses novel molecular technologies, developed in house, to further characterize isolated pathogens, compare them genetically to genes associated with important virulence factors, and recognize emerging variants. These techniques aid in the selection of those new strains for use in custom vaccines, addressing pathogens for which vaccines were previously unavailable.

Newport Laboratories' diagnostic services also include bacteriological culturing, antibacterial-susceptibility testing, serological profiling to monitor herd-pathogen exposure, and isolation of viral pathogens.

START WITH YOUR VET

Herd health starts by working with your veterinarian. Newport Laboratories is dedicated to working with veterinarians to ensure proper sampling and submission, quick turn- around and accurate results. Talk to your veterinarian about whether custom-made vaccines are right for your operation, or visit www.newportlabs.com to learn more.

NEWPORT LABORATORIES
A Boehringer Ingelheim Company

Executive Editor's message continued from page 301

Figure 1. Received and Accepted dates are published for each manuscript in the *Journal of Swine Health and Production*.

Once the reviews are complete and submitted to the journal office, the lead reviewer will take all the reviews into consideration and make a publication recommendation. At this point, I review the publication recommendation and make the decision to conditionally accept, request revisions, or reject the manuscript. If revisions are requested, the manuscript is returned to the author and they are given 3 weeks to respond. Once the revised manuscript is returned to the journal office, it is sent back to the reviewers for re-consideration. This is the period where a manuscript can accumulate quite a bit of time and skew the distribution of the time to acceptance statistic. Depending on any further revisions required, the manuscript may be conditionally accepted at this time, returned for further revisions, or rejected.

Once the manuscript is conditionally accepted it is forwarded to Sherrie Webb, our associate editor. Any changes usually required at this point are corrections in grammar, punctuation, format, and copy-editing concerns that the associate editor manages. However, some minor revisions or requests for clarification from reviewers may also need to be addressed at this point. Once this phase is completed, the manuscript is converted into an author proof by Tina Smith, our graphic designer, and returned to the author for final proofreading. Once the author accepts the final proof, the review process is finished. Phew!

As you can see, the process is thorough and lengthy and requires the efforts of many critical people and opinions in the process.

It seems that the epidemic of 'busy schedules' continues to escalate with many of us experiencing increased work demands, and it perhaps seems to be approaching a pandemic phase. I recognize it is often difficult to take on additional work and I hope you can now remember that reviewing a paper thoroughly is a big job requiring the time of many people. At the time of writing this message the journal has received 36 manuscript submissions in 2018 and we still have many weeks left in the year. While this is nice to report healthy submission rates for the journal, it also means active recruiting of peer-reviewers remains challenging. Once again, thank you to those who take on extra work during this epidemic of busy schedules. Additionally, in a previous message I put out a call for "JSHAP's Most Wanted."[2] We always need peer-reviewers! If you would like to be on JSHAP's "Most Wanted List" as a willing peer-reviewer, please use the following link to complete the short survey (5 to 10 minutes): **uoguelph.eu.qualtrics.com/jfe/form/SV_3qbWc4gJKeg0GGh**.

Thank you to everyone who has contributed, and continues to contribute, considerable amounts of time and effort to this process for JSHAP.

Terri O'Sullivan, DVM, PhD
Executive Editor

References

1. O'Sullivan, T. The peer-review process. *J Swine Health Prod*. 2013;21(6):299.

2. O'Sullivan, T. JSHAP's Most Wanted! *J Swine Health Prod*. 2017;25(6):289.

Brief communication

Peer reviewed

Evaluation of pig pneumonia at slaughter using polymerase chain reaction and histopathology in Argentina

Javier A. Cappuccio, DVM, PhD; Marina Dibarbora, DVM, PhD; Fernando A. Bessone, DVM, MSc; Valeria S. Olivera; Inés Lozada, DVM; Fabrisio E. Alustiza, DVM, PhD; Alejandra Quiroga, DVM, PhD; Estefanía M. Pérez, DVM, PhD; Gustavo C. Zielinski, DVM, PhD; Carlos J. Perfumo, DVM, PhD; Ariel J. Pereda, DVM, PhD; Daniel R. Pérez, PhD

Summary

Histopathology and polymerase chain reaction were conducted on 81 lungs collected at slaughter from 13 swine farms free of porcine reproductive and respiratory syndrome virus and pseudorabies virus infection. *Pasteurella multocida* and *Mycoplasma hyopneumoniae* were the most common pathogens detected. Suppurative and catarrhal bronchopneumonia was present in 59 (72.8%) cases.

Keywords: swine, bronchopneumonia, slaughter, Argentina, porcine respiratory disease complex

Received: January 9, 2018
Accepted: June 9, 2018

Resumen – Evaluación de la neumonía en cerdos en el matadero utilizando la reacción en cadena de la polimerasa e histopatología en Argentina

Se realizó la reacción en cadena de polimerasa e histopatología en 81 pulmones recolectados en el matadero de 13 granjas porcinas libres del síndrome reproductivo y respiratorio porcino, y de la infección por el virus de la pseudorabia. La *Pasteurella multocida* y el *Mycoplasma hyopneumoniae* fueron los patógenos más comúnmente detectados. La bronconeumonía supurativa y catarral estuvieron presentes en 59 (72.8%) casos.

Résumé – Évaluation de la pneumonie porcine à l'abattage en utilisant la réaction d'amplification en chaine par la polymérase et l'histopathologie en Argentine

L'histopathologie et la réaction d'amplification en chaine par la polymérase ont été réalisées sur 81 poumons récoltés à l'abattoir provenant de 13 fermes porcines exemptes du virus du syndrome reproducteur et respiratoire porcin et d'infection par le virus de la pseudorage. *Pasteurella multocida* et *Mycoplasma hyopneumoniae* étaient les agents pathogènes les plus communément détectés. Une bronchopneumonie suppurative et catarrhale était présente dans 59 (72.8%) cas.

In pig production systems, slaughter checks are routinely used to estimate prevalence and severity of respiratory disease as well as for detecting subclinical disease. The term porcine respiratory disease complex (PRDC) is used to describe polymicrobial respiratory infections that affect growing and finishing pigs and are associated with economic losses. The etiology of PRDC varies among countries, regions, and farms.[1-4] The most common pathogens reported worldwide associated with PRDC are *Actinobacillus pleuropneumoniae* (Ap), influenza A virus (IAV), *Mycoplasma hyopneumoniae* (Mh), *Pasteurella multocida* (Pm), porcine circovirus type 2 (PCV2), porcine reproductive and respiratory syndrome virus (PRRSV), and pseudorabies virus (PRV).[1-4] In Asia, Europe, and North America the predominance of PRRSV in cases of PRDC is well documented.[2,4]

Argentina is free of PRRSV, whereas PRV is under an eradication plan with most confinement farms free of infection. Under this scenario, the agents that are most frequently associated with PRDC in Argentina remain unknown. The aim of this study was to investigate the relationship between respiratory pathogens detected by polymerase chain reaction (PCR) and histopathological lung lesions in lungs with pneumonia lesions obtained from PRRSV- and PRV-free pigs at slaughter.

Materials and methods

All samples were collected from three slaughterhouses in Argentina which operate in accordance with slaughtering procedures

JAC, FAB, FEA, GCZ: Estación Experimental Agropecuaria Marcos Juarez, Instituto Nacional de Tecnología Agropecuaria (INTA), Marcos Juarez, Córdoba, Argentina.

MD: Consejo Nacional de Investigaciones Científicas y Técnicas (CONICET), Ciudad Autónoma de Buenos Aires, Argentina.

VSO: Instituto de Virología, Centro de Investigaciones en Ciencias Veterinarias y Agronómicas, Instituto Nacional de Tecnología Agropecuaria (INTA), Hurlingham, Argentina.

IL, AQ, EMP, CJP: Laboratorio de Patología Especial Veterinaria, Facultad de Ciencias Veterinarias, Universidad Nacional de La Plata, La Plata, Argentina.

AJP: Instituto de Patobiología, Centro de Investigaciones en Ciencias Veterinarias y Agronómicas, Instituto Nacional de Tecnología Agropecuaria (INTA), Hurlingham, Argentina.

DRP: Poultry Diagnostic and Research Center, College of Veterinary Medicine, University of Georgia, Athens, Georgia.

Corresponding Author: Dr Javier Cappuccio, EEA Marcos Juarez, INTA, Ruta 12 km 3, 2580, Marcos Juarez, Córdoba, Argentina; Tel: +543472425001; Email: cappuccio.javier@inta.gob.ar.

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

Cappuccio JA, Dibarbora M, Bessone FA, et al. Evaluation of pig pneumonia at slaughter using polymerase chain reaction and histopathology in Argentina. *J Swine Health Prod.* 2018;26(6):304-308.

approved in the country. A non-probability sampling scheme was applied, only lungs with bronchopneumonia lesions that affected more than 20% of the entire lung area were selected. To avoid cross-contamination in sample processing, sterile instruments were used for each sample collected and samples were individually placed in sterile bags and stored at 4°C. A total of 81 samples were collected from pigs originating from 13 farms (6 to 7 samples from each farm), located in the main swine production areas of the country: Buenos Aires, Entre Rios, and Santa Fe provinces. All 13 farms used the same commercial single-dose, single-injection Mh and PCV2 combined vaccine at the time of weaning (FLEXcombo, Boehringer Ingelheim, St. Joseph, Missouri). No other vaccines against respiratory pathogens were used.

For histopathology analysis, samples were collected and assigned an identification number by a practitioner at the abattoir and then routinely processed according to Laboratorio de Patología Especial Veterinaria procedure manual by a laboratory technician. The prepared blocks were then analyzed in a blinded manner by a single pathologist. Lung lesions were diagnosed into one of the following categories: suppurative bronchopneumonia (SBN) defined by the presence of neutrophils, macrophages, and mucus in bronchioles and alveoli; catarrhal bronchopneumonia (CBN) defined by bronchiole and alveoli filled with mucus exudate, macrophages, and scarce or no neutrophils present; bronchointerstitial pneumonia (BIN) defined by macrophages and lymphocytes infiltrating the alveolar and peribronchiolar septa, bronchiolar necrosis, or hyperplasia of pneumocytes type II; bronchitis and bronchiolitis defined by inflammation or necrosis restricted to airway walls and presence of neutrophils and cellular debris in airway lumen; fibrinous bronchopneumonia (FB) defined by alveoli and interlobular connective tissue filled with serofibrinous exudate, presence of oat cells, and thrombosis of capillaries and lymphatic vessels; or no lesions.

For PCR assays, lung homogenates were processed according to Cappuccio et al.[5] Extraction of DNA and RNA was made using Roche High Pure PCR Template Preparation Kit and High Pure RNA Isolation Kit (Roche Diagnostics, Mannheim, Germany). Polymerase chain reaction assays were performed on Veriti Thermal Cycler or StepOnePlus Real-Time PCR System (Applied Biosystems, Foster City, California). The presence of IAV, PCV2, Mh, Pm, and Ap were determined as previously described (Table 1).

The statistical relationship between frequency of detection of each pathogen and the histopathological category was evaluated by Fisher`s exact test using manual calculation. Statistical significance was set to $P < .05$.

Results

The most common pathogens detected in 81 samples tested by PCR were Pm in 64 cases (79%) and Mh in 59 cases (72.8%). Our study revealed no PCR positive samples to Ap. Viral pathogens were detected in a lower percentage of samples: PCV2 in 17 cases (21%) and IAV in 6 cases (7.4%) (Figure 1). Coinfections of two or more pathogens were detected in 52 cases (64.2%), with the most common coinfection being Pm and Mh coinfection in 34 of 52 samples (65.4%). In relation to viral pathogens, PCV2 was detected as a coinfection in 15 cases and the 6 IAV positive cases were coinfections.

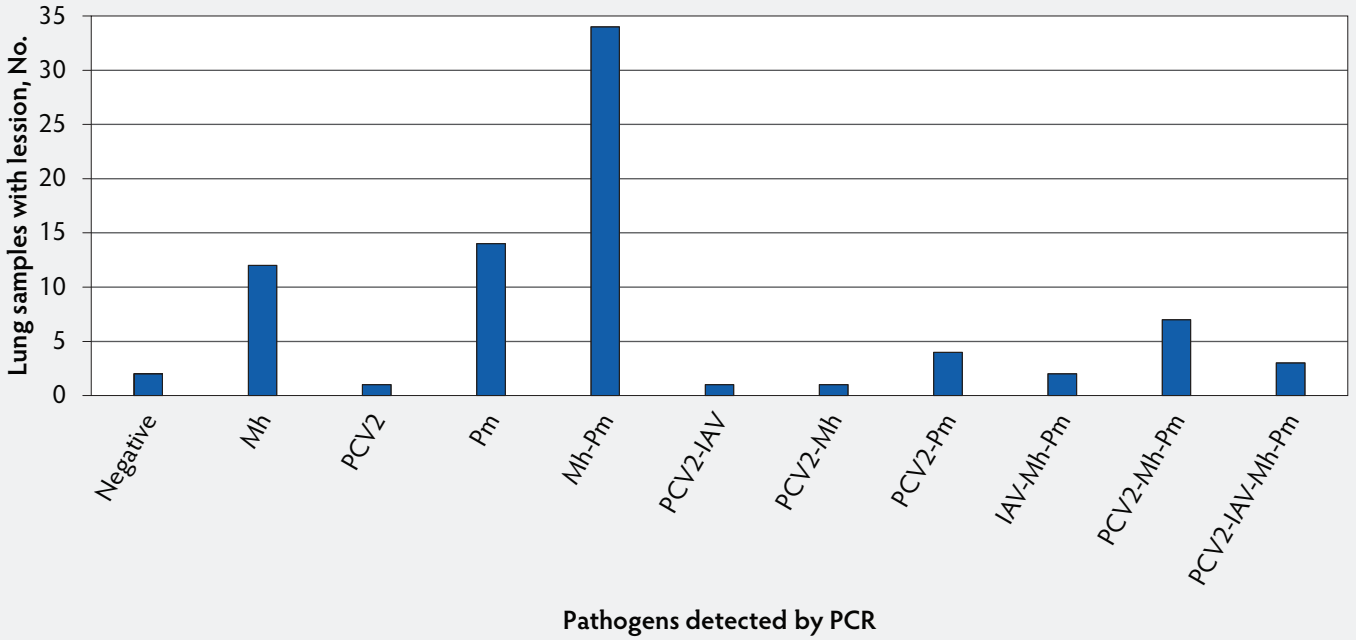
In relation to histopathology analysis, SBN and CBN were present in 59 (72.8%) of the 81 cases (Figure 2) and were most commonly found with either single Mh (7 of 59 cases, 11.9%) or Pm (10 of 59 cases, 16.9%) infections or a combination of both pathogens in 27 of 59 cases (45.8%). Detection of PCV2 occurred in 9 of 34 cases (26.5%) categorized as SBN, of which 3 were also positive for Mh

Table 1: Polymerase chain reaction assays used to measure respiratory pathogens present in lung tissue samples

Pathogen	Primer sequence	Amplicon size (bp)	Specificity	Type of PCR	Threshold of detection
IAV*	5´-GACCRATCCTGTCACCTCTGAC-3´ 5´-AGGGCATTYTGGACAAAKCGTCTA-3´	60	Matrix	RT-PCR	4.17×10^5 $TCID_{50}$/reaction
PCV2†	5´- GGGAGGAGTAGTTTACATA-3´ 5´- CGCACTTCTTTCGTTTTC-3´	460	ORF2	PCR	4.42×10^5 copies/µL
Ap‡	5´-GGGGACGTAACTCGGTGATT-3´ 5´-GCTCACCAACGTTTGCTCAT-3´	377	ApxIV	qPCR	5 CFU/reaction
Pm§	5´-ATCCGCTATTTACCCAGTGG-3´ 5´-GCTGTAAACGAACTCGCCAC-3´	460	KMT1	PCR	4.09×10^3 organisms
Mh¶	5´-GAGCCTTCAAGCTTCACCAAGA-3´ 5´-TGTGTTAGTGACTTTTGCCACC-3´ 5´-ACTAGATAGGAAATGCTCTAGT-3´ 5´-GTGGACTACCAGGGTATCT-3´	649 352	16S ribosomal	N-PCR	80 organisms

* The presence of IAV was determined as previously described.[5]
† The presence of PCV2 was determined as previously described.[6]
‡ The presence of Ap was determined as previously described.[7]
§ The presence of Pm was determined as previously described.[8]
¶ The presence of Mh was determined as previously described.[9]
bp= base pair; PCR = polymerase chain reaction; IAV = influenza A virus; RT-PCR = reverse transcription PCR; $TCID_{50}$ = 50% tissue culture infective dose; PCV2 = porcine circovirus type 2; ORF2 = open reading frame 2; Ap = *Actinobacillus pleuropneumoniae*; ApxIV = *Actinobacillus pleuropneumoniae* toxin IV; qPCR = quantitative PCR; CFU = colony-forming units; Pm = *Pasturella multocida*; KMT1 = *Pasteurella multocida* species identification gene; Mh = *Mycoplasma hyopneumoniae*; N-PCR = nested PCR.

Figure 1: Frequency of pathogen detection using PCR in 81 lung samples collected at slaughter in Argentina. Single and coinfections are presented. Mh = *Mycoplasma hyopneumoniae*; PCV2 = porcine circovirus type 2; Pm = *Pasturella multocida*; IAV = influenza A virus; PCR = polymerase chain reaction.



and Pm, 3 were also positive for Mh, Pm, and IAV, 1 was also positive for Pm, 1 was also positive for IAV, and 1 showed no sign of coinfection. Only 3 cases categorized as CBN were positive for PCV2 and were also positive for Mh and Pm (Figure 2). Ten cases were categorized as BIN and tested positive for either Mh, Pm, or both but only 2 cases were PCV2 positive. Only 2 cases were categorized as FB and were negative to Ap and positive to Pm. No statistical association ($P > .05$) was detected between histopathological classification and pathogen detection by PCR.

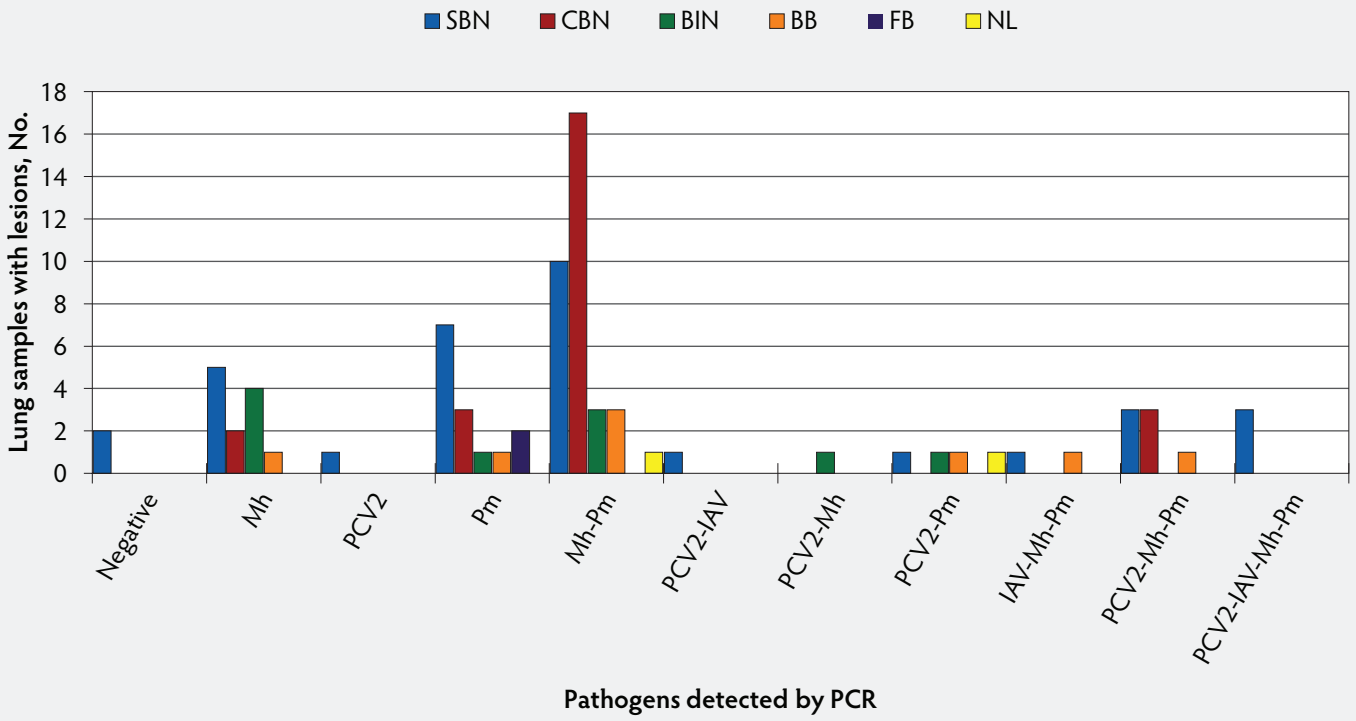
Regardless of histopathological classification, necrotizing bronchiolitis and bronchiolesclerosis were detected in 33 of 81 cases (40.8%), which were most frequently categorized as SBN (23 of 33 cases, 69.7%) and were positive for Mh and Pm (30 of 33 cases, 90.9%). Only 7 cases with necrotizing bronchiolitis and bronchiolesclerosis were positive for PCV2 (5 of 33 cases, 15.2%) or IAV (2 of 33 cases, 6.1%).

Discussion

Few studies have been done to investigate the relationship among pathogen detection and histopathological lesions in PRDC affected pigs.[2-4,10] To the best of the authors' knowledge, this is the first study carried out in Argentina that investigated lung lesions collected from pigs at the time of slaughter. However, it must be taken into consideration, upon interpretation of results, that only a small number of non-randomly selected herds were included in this study and hence, this represents a biased sample of the Argentinian swine population. Regardless of this bias, Pm and Mh were the pathogens most frequently detected. The detection rate of these pathogens is consistent with studies carried out in Asia, Europe, and North America.[2-4] The detection of Mh should not be considered a lack of vaccine effectiveness as vaccination reduces clinical signs and lung lesions, but does not prevent the colonization of the organism.[10,11] The incidence of viral infections was lower than in previously published studies.[2-4] Coinfections of two or more pathogens were detected in a high number of cases (52 of 81; 64%), with the most common being Pm and Mh coinfection. Most of the PCV2 positive cases were coinfections (15 of 17; 88%) further supporting the possible role of PCV2 in coinfections.[3,4,10] Influenza A virus was detected in a low number of cases and always in coinfections. The authors hypothesize that the predominance of bacterial detection over viral detections in this study is related to the absence of PRRSV infection and the implementation of vaccination against PCV2 in the farms evaluated but must also reiterate that this was not a randomly generated sample of lung lesions. In the case of IAV, the low detection rate could be affected by a combination of factors including the age of pigs at which the sample was collected, the acute nature of the infection, and the short persistence of the virus in the lungs.[2,5]

Similar to previous studies,[3,5] SBN and CBN were the most common histopathological diagnosis and could be explained by the higher number of samples positive to Pm, Mh, and their coinfection. When CBN or SBN occurs with a bacterial infection without evidence of viral infection, the bronchiolar epithelium is generally normal.[12] It is commonly accepted that virus replication leads to inflammation and necrosis of the bronchioles with concomitant obstruction of the lumen that ultimately affects clearance of bacteria and exudates from the alveoli leading to more severe lesions.[2,3,12] In this study, necrotizing bronchiolitis or bronchiolesclerosis was detected in 41% of the cases, most frequently associated with SBN and Pm and Mh detection. Bronchointerstitial pneumonia occurs particularly in viral infections and is the more frequent lung lesion associated with PCV2 infections.[12] In this study, however, all BIN cases tested positive for either Mh, Pm, or both. Previous studies describe interstitial and peribronchial

Figure 2: Frequency of histopathological categories detected for pathogens present in 81 lung samples collected at slaughter in Argentina. Histopathological categories were: suppurative bronchopneumonia (SBN; 34 samples) defined by the presence of neutrophils, macrophages, and mucus in bronchioles and alveoli; catarrhal bronchopneumonia (CBN; 25 samples) defined by bronchiole and alveoli filled with mucus exudate, macrophages, and scarce or no neutrophils present; bronchointerstitial pneumonia (BIN; 10 samples) defined by macrophages and lymphocytes infiltrating the alveolar and peribronchiolar septa, bronchiolar necrosis, or hyperplasia of pneumocytes type II; bronchitis and bronchiolitis (BB; 8 samples) defined by inflammation or necrosis restricted to airway walls and presence of neutrophils and cellular debris in airway lumen; fibrinous bronchopneumonia (FB; 2 samples) defined by alveoli and interlobular connective tissue filled with serofibrinous exudate, presence of oat cells, and thrombosis of capillaries and lymphatic vessels; and no lesions (NL; 2 samples). Mh = *Mycoplasma hyopneumoniae*; PCV2 = porcine circovirus type 2; Pm = *Pasturella multocida*; IAV = influenza A virus; PCR = polymerase chain reaction.



infiltration lesions to be associated with Mh and Pm infections.[12] The 2 cases of FB were positive for Pm and negative for Ap. The relationship between Pm and FB has been previously reported to be associated with toxin production.[3] It is highlighted that only one pathologist scored the lung lesions and pathological analysis was performed in a different laboratory than the PCR assays.

The lack of statistical association between histopathological diagnosis and PCR detection of each pathogen could be related to the small number of samples evaluated. Further studies using a more comprehensive epidemiological approach are needed to verify this lack of relationship.

This brief communication reports on the presence of the polymicrobial nature of PRDC in slaughter-aged pigs presumed free of PRRSV and PRV infections and that were vaccinated for Mh and PCV2 at weaning. The presence of bacterial and viral coinfections with SBN lesions in this study supports the continued need to control respiratory infection on swine farms in Argentina. Slaughterhouse inspection of carcasses is used to protect public health by ensuring food safety.[13] However, these data collected also have value for other purposes such as passive surveillance activities. For example, the data has been used to estimate prevalence of a particular disease or pathological condition, determine risk factors associated to pleurisy or pneumonia, and to determine the economic effect of pneumonia.[14-16] More recently, a well-established slaughterhouse national surveillance system has been considered valuable as an early warning system for an emerging disease or as an initial database to design specific studies (eg, pleurisy or tail lesions).[14-17] In this context, slaughter surveys can be applied to generate significant information about the etiology, severity, and interactions of PRDC.

Implications

- Under the conditions of this study, Pm and Mh were the most frequently detected pathogens from grossly affected lungs collected from pigs at slaughter in Argentina.
- This study supports the necessity for the development of a national based slaughterhouse monitoring or surveillance system to continue to document and understand lesions and pathogens present in the Argentinian swine population.

Acknowledgements

This work was supported by the National Institute of Allergy and Infectious Diseases, Center for Research on Influenza Pathogenesis through the University of Georgia, and Instituto Nacional de Tecnología Agropecuaria on NIAID contract No. HHSN272201400008C. This work was

also funded by the Instituto Nacional de Tecnología Agropecuaria Programa Nacional de Sanidad Animal Proyecto Epecífico 1115057; Secretaría de Ciencia y Técnica, Universidad Nacional de La Plata, subsidio V218 and Proyecto de Investigación Científica y Tecnológica 2015-1232, Agencia Nacional de Promoción Científica.

Conflict of interest

None reported.

Disclaimer

Scientific manuscripts published in the *Journal of Swine Health and Production* are peer reviewed. However, information on medications, feed, and management techniques may be specific to the research or commercial situation presented in the manuscript. It is the responsibility of the reader to use information responsibly and in accordance with the rules and regulations governing research or the practice of veterinary medicine in their country or region.

References

1. Opriessnig T, Giménez-Lirola LG, Halbur PG. Polymicrobial respiratory disease in pigs. *Anim Health Res Rev*. 2011;12:133-148.

2. Choi YK, Goyal SM, Joo HS. Retrospective analysis of etiologic agents associated with respiratory diseases in pigs. *Can Vet J*. 2003;44:735-737.

3. Hansen MS, Pors SE, Jensen HE, Bille-Hansen V, Bisgaard M, Flachs EM, Nielsen OL. An investigation of the pathology and pathogens associated with porcine respiratory disease complex in Denmark. *J Comp Pathol*. 2010;143:120-131.

4. Kim J, Chung HK, Chae C. Association of porcine circovirus 2 with porcine respiratory disease complex. *Vet J*. 2003;166:251-256.

5. Cappuccio J, Dibarbora M, Lozada I, Quiroga A, Olivera V, Dángelo M, Pérez E, Barrales H, Perfumo C, Pereda A, Pérez DR. Two years of surveillance of influenza a virus infection in a swine herd. Results of virological, serological and pathological studies. *Comp Immunol Microbiol Infect Dis*. 2017;50:110-115.

6. Pereda A, Piñeyro P, Bratanich A, Quiroga MA, Bucafusco D, Craig MI, Cappuccio J, Machuca M, Rimondi A, Dibárbora M, Sanguinetti HR, Perfumo CJ. Genetic characterization of porcine circovirus type 2 from pigs with porcine circovirus associated diseases in Argentina. *ISRN Vet Sci*. 2011;2011:560905. doi:10.5402/2011/560905

7. Tobias TJ, Bouma A, Klinkenberg D, Daemen AJ, Stegeman JA, Wagenaar JA, Duim B. Detection of *Actinobacillus pleuropneumoniae* in pigs by real-time quantitative PCR for the apxIVA gene. *Vet J*. 2012;193:557-560.

8. Townsend KM, Boyce JD, Chung JY, Frost AJ, Adler B. Genetic organization of *Pasteurella multocida* cap loci and development of a multiplex capsular PCR typing system. *J Clin Microbiol*. 2001;39:924-929.

9. Calsamiglia M, Pijoan C, Trigo A. Application of a nested polymerase chain reaction assay to detect *Mycoplasma hyopneumoniae* from nasal swabs. *J Vet Diagn Invest*. 1999;11:246-251.

10. Fablet C, Marois-Créhan C, Simon G, Grasland B, Jestin A, Kobisch M, Madec F, Rose N. Infectious agents associated with respiratory diseases in 125 farrow-to-finish pig herds: A cross sectional study. *Vet Microbiol*. 2012;157:152-163.

11. Maes D, Sibila M, Kuhnert P, Segalés J, Haesebrouck F, Pieters M. Update on *Mycoplasma hyopneumoniae* infections in pigs: Knowledge gaps for improved disease control. *Transbound Emerg Dis*. 2017;65(Suppl 1):110-124. doi:10.1111/tbed.12677

12. Caswell JL, Williams KJ. Respiratory System. In: Maxie MG, ed. *Jubb, Kennedy, and Palmer´s Pathology of Domestic Animals*. Vol 2. 6th ed. St Louis, Mo: Elsevier Inc; 2016:465-593.

13. Nielsen SS, Nielsen GB, Denwood MJ, Haugegaard J, Houe H. Comparison of recording of pericarditis and lung disorders at routine meat inspection with findings at systematic health monitoring in Danish finisher pigs. *Acta Vet Scand*. 2015;57:18. doi:10.1186/s13028-015-0109-z

14. Brewster VR, Maiti HC, Tucker AW, Nevel A. Associations between EP-like lesions and pleuritis and post trimming carcass weights of finishing pigs in England. *Livest Sci*. 2017;201:1-4.

15. Jäger HC, McKinley TJ, Wood JLN, Pearce GP, Williamson S, Strugnell B, Done S, Habernoll H, Palzer A, Tucker AW. Factors associated with pleurisy in pigs: A case-control analysis of slaughter pig data for England and Wales. *Plos One* 2012;7:e29655. doi:10.1371/journal.pone.0029655

16. van Staaveren N, Vale AP, Manzanilla EG, Teixeira DL, Leonard FC, Hanlon A, Boyle LA. Relationship between tail lesions and lung health in slaughter pigs. *Prev Vet Med*. 2016;127:21-26.

17. Holt HR, Alarcon P, Velasova M, Pfeiffer DU, Wieland B. BPEX Pig Health Scheme: a useful monitoring system for respiratory disease control in pig farms? *BMC Vet Res*. 2011;7:82. doi:10.1186/1746-6148-7-82

General overview of the detection of *Mycoplasma hyopneumoniae* DNA by quantitative polymerase chain reaction in diagnostic cases submitted to the Iowa State University Veterinary Diagnostic Laboratory from 2004 to 2016

Gaurav Rawal, DVM; Paulo Arruda, DVM, PhD; Christopher Rademacher, DVM; Ran Bi, MS; Daniel C. L. Linhares, DVM, MBA, PhD

Summary

Mycoplasma hyopneumoniae (Mhp) is the etiologic agent of enzootic pneumonia and a major causative agent of the porcine respiratory disease complex. This study summarizes and describes the general diagnostic trends on Mhp detection by quantitative polymerase chain reaction (qPCR) in cases submitted to the Iowa State University Veterinary Diagnostic Laboratory from 2004 to 2016. The following variables were included in the analysis: animal age, geographic location, sample type, season, and submission year. The overall frequency of detection found was 27.04% and ranged from 17.9% to 40.7%. Lung homogenate and bronchial swabs had a greater Mhp qPCR detection rate than other sample types ($P < .001$) followed by bronchoalveolar lavage ($P < .001$), while oral fluids had the lowest Mhp detection rate ($P < .001$). The fall season had a greater percentage of positive Mhp qPCR results when compared to other seasons ($P < .001$), while spring had the lowest percentage. Finishing-age pigs had a greater percentage of Mhp qPCR detection when compared to other age groups ($P < .001$), while suckling pigs had the lowest percentage ($P < .001$).

Keywords: swine, *Mycoplasma hyopneumoniae*, polymerase chain reaction

Received: November 14, 2017
Accepted: April 25, 2018

Resumen – Resumen general de la detección del DNA del *Mycoplasma hyopneumoniae* por medio de la reacción en cadena de la polimerasa cuantitativa en casos de diagnóstico enviados al Laboratorio de Diagnóstico Veterinario de la Universidad Estatal de Iowa del 2004 al 2016

El *Mycoplasma hyopneumoniae* (Mhp por sus siglas en inglés) es el agente etiológico de la neumonía enzoótica, y un agente causante mayor del complejo respiratorio porcino. Este estudio resume y describe las tendencias de diagnóstico generales para la detección del Mhp por medio de la reacción en cadena de la polimerasa cuantitativa (qPCR por sus siglas en inglés) en casos enviados al Laboratorio de Diagnóstico Veterinario de la Universidad Estatal de Iowa del 2004 al 2016. El análisis incluyó las siguientes variables: edad del animal, localización geográfica, tipo de muestra, estación, y año de presentación. La frecuencia general de detección fue de 27.04% con un rango de 17.9% a 40.7%. Los homogenizados de pulmón y los hisopos bronquiales tuvieron un mayor índice de detección a la qPCR Mhp que otros tipos de muestra ($P < .001$) seguidos por el lavado bronquioalveolar ($P < .001$), mientras que los fluidos orales tuvieron el índice más bajo de detección de Mhp ($P < .001$). El otoño tuvo el mayor porcentaje de resultados positivos a la Mhp qPCR comparado con otras estaciones ($P < .001$), mientras que la primavera tuvo el porcentaje más bajo. Los cerdos en edad de finalización tuvieron un porcentaje mayor de detección a la Mhp qPCR al compararlos con grupos de otra edad ($P < .001$), mientras que los cerdos en lactancia tuvieron el porcentaje más bajo ($P < .001$).

GR, PA, CR, DCLL: Veterinary Diagnostic and Production Animal Medicine Department, College of Veterinary Medicine, Iowa State University, Ames, Iowa.

RB: Department of Statistics, College of Liberal Arts and Sciences, Iowa State University, Ames, Iowa.

Corresponding author: Dr Daniel C. L. Linhares, 2221 Lloyd Veterinary Medical Center, 1600 S 16th Street, Ames, IA 50011; Tel: 515-294-9358; Fax: 515-294-1072; Email: linhares@iastate.edu.

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

Rawal G, Arruda P, Rademacher C, et al. General overview of the detection of *Mycoplasma hyopneumoniae* DNA by quantitative polymerase chain reaction in diagnostic cases submitted to the Iowa State University Veterinary Diagnostic Laboratory from 2004 to 2016. *J Swine Health Prod.* 2018;26(6):309-315.

Résumé – Aperçu général de la détection d'ADN de *Mycoplasma hyopneumoniae* par réaction d'amplification en chaine par la polymérase quantitative dans des cas diagnostiques soumis au Iowa State University Veterinary Diagnostic Laboratory entre 2004 et 2016

Mycoplasma hyopneumoniae (Mhp) est l'agent étiologique de la pneumonie enzootique et un agent causal majeur du complexe des maladies respiratoires porcines. La présente étude résume et décrit les tendances diagnostiques générales de la détection de Mhp par réaction d'amplification en chaine par la polymérase quantitative (qPCR) dans les cas soumis au Iowa State University Veterinary Diagnostic Laboratory entre 2004 et 2016. Les variables suivantes étaient incluses dans l'analyse: âge de l'animal, localisation géographique, type d'échantillon, saison, et année de soumission. La fréquence globale de détection trouvée était de 27.04% et variait entre 17.9% et 40.7%. Un homogénat de poumon et des écouvillons bronchiaux avaient un taux de détection de Mhp par qPCR plus grand que les autres types d'échantillons ($P < .001$) suivi par le lavage broncho-alvéolaire ($P < .001$), alors que les fluides oraux avaient le taux de détection de Mhp le plus bas ($P < .001$). Le plus grand

pourcentage de résultats qPCR positifs pour Mhp a été obtenu à l'automne comparativement aux autres saisons ($P < .001$), et le pourcentage le plus bas a été trouvé au printemps. Les porcs en période de finition avaient un pourcentage de détection de Mhp par qPCR plus grand comparativement aux autres groupes d'âge ($P < .001$), alors que les porcelets à la mamelle avaient le pourcentage le plus faible ($P < .001$).

Mycoplasma hyopneumoniae (Mhp) is the causative agent of enzootic pig pneumonia,[1,2] a respiratory disease affecting pigs worldwide, characterized by significant economic losses due to slower growth, poor feed conversion, and death.[3] The losses associated with Mhp were estimated in the range of $375 to $400 million annually in the United States.[4] *Mycoplasma hyopneumoniae* increased the cost of production by $2.50 per pig in the grow-finish phase with an additional increase in the cost of therapeutics by $0.75 to $0.90 per pig at weaning in the United States.[5] The isolation of Mhp is challenging due to the fastidious culture requirements and the relatively slow Mhp growth, often resulting in overgrowth by other mycoplasmas including *M flocculare* and *M hyorhinis*.[6] Therefore, detection of Mhp for diagnostic purposes is typically performed by quantitative polymerase chain reaction (qPCR), which offers rapid turnaround at a high sensitivity and specificity.[7-9] Multiple specimens including bronchial swabs, bronchoalveolar lavages, laryngeal swabs, lung homogenates, lungs, nasal swabs, oral fluids, and tracheal swabs have been used to detect Mhp in pigs.

The Iowa State University Veterinary Diagnostic Laboratory (ISU VDL) is one of the largest in the United States receiving over 80,000 cases annually, of which over 75% are swine related. The objective of this study was to summarize and describe the patterns of Mhp DNA detection by qPCR over time from cases received at the ISU VDL from 2004 to 2016. *Mycoplasma hyopneumoniae* detection rate was reported by year, age group, specimen, season, and US geographic location.

Materials and methods

This study was based on data derived from diagnostic laboratory submissions, so an Institutional Animal Care and Use Committee approval was not needed.

Eligibility and exclusion criteria

All porcine cases where Mhp qPCR was performed were gathered. For this study, a case was defined as one accession identification number with at least one sample. A case was considered positive when there was at least one sample testing positive for Mhp by qPCR. The data were screened and cases with non-conventional sample types for Mhp diagnostics, including cell cultures, conjunctival swab, environmental, extract, inoculum, liver, semen, and vaccine, were excluded from the analysis. Infrequent sample types (those comprising less than 1% of submissions) such as fibrin, fibrin swab, fluid, fresh tissue, lymph node, tonsil, and tonsil scrapings were also excluded from the analysis, as were cases with inconclusive or suspect results. Submissions were classified by season (December through February as winter, March through May as spring, June through August as summer, and September through November as fall), age group (pigs less than 3 weeks of age were defined as suckling, 3 to 6 weeks as nursery, 7 to 11 weeks as growing, 12 weeks to 200 days as finishing, and greater than 200 days as adult), and sample type, which included bronchial swab, bronchoalveolar lavage, laryngeal swab, lung, lung homogenate, nasal swab, oral fluid, swab, tracheal swab, and other. All specimens are described using the same terminology reported in the VDL submission form. Samples labeled as saliva were included under the oral fluid category and samples labeled as lung swab, multiple, oropharyngeal swab, pharyngeal swab, pleural swab, serum, and tonsil swab were identified as other.

Microsoft Excel (Microsoft, Redmond, Washington) was used to organize the data and Tableau (Tableau, Seattle, Washington) was used to produce plots.

Statistical analysis

Statistical analysis was performed with SAS 9.4 software (SAS Institute Inc, Cary, North Carolina). For statistical analysis, the frequency of positive results was compared across groups (age, season, and sample type) with Pearson's Chi-square test. The significance level was determined at $\alpha = .05$.

The K-means clustering method was implemented to categorize the Mhp detection rate as low, medium, or high for specimen types. The aim of the K-means algorithm is to divide the set of observations into K groups, minimizing the within-cluster sum of squares.[10] The K-means procedure searched for 3 partitions with locally-optimal, within-cluster sum of squares by moving points from one cluster to another.

Results

A total of 94,986 qPCR results from 37,574 cases were used in the analysis. In total, 1128 qPCR results were excluded from the data analysis due to non-conventional or infrequent sample types and 178 qPCR samples were excluded due to inconclusive status of results. Analysis were conducted with 93,680 samples, from which 25,339 (27%) tested positive for Mhp qPCR, ranging from 17.9% to 40.7% (Table 1).

The most common sample types submitted for Mhp qPCR at the ISU VDL were lung, followed by oral fluids, nasal swab, and bronchoalveolar lavage (Figure 1). The Mhp qPCR detection rate was 64% in bronchial swab (2503 of 3909), 49% in bronchoalveolar lavage (3370 of 6872), 18% in laryngeal swab (313 of 1777), 33% in lung (13517 of 40633), 66% in lung homogenate (962 of 1455), 10% in nasal swab (808 of 7892), 8% in oral fluids (1850 of 23804), 18% in swab (341 of 1922), 34% in tracheal swab (606 of 1777), and 27% in other (965 of 3574). Lung homogenate and bronchial swab had a greater Mhp qPCR detection rate than other sample types ($P < .001$), followed by bronchoalveolar lavage ($P < .001$), while oral fluids had the lowest Mhp detection rate ($P < .001$). Using the K-mean clustering method, bronchial swab, bronchoalveolar lavage, and lung homogenate had a high detection rate; lung, tracheal swab, and other had a medium detection rate; and laryngeal swab, nasal swab, oral fluid, and swab had a relatively low detection rate. The fall season had a greater percentage of positive Mhp qPCR results when compared to other seasons ($P < .001$), while spring had the lowest percentage ($P < .001$) (Figure 2). Finishing-age pigs had a greater percentage of Mhp qPCR detection when compared to other age groups ($P < .001$), while suckling pigs had the lowest percentage ($P < .001$) (Figure 3). The majority (30%) of cases submitted to the ISU VDL for Mhp testing by qPCR were from Iowa, but there were cases from 33 other states (Figure 4). Bronchoalveolar lavage had a greater detection rate of Mhp by qPCR, as compared to other specimens, for all age groups except growing pigs (Figure 5). In growing pigs, lung homogenate had the highest detection rate among the specimens. Nasal swab had the lowest Mhp detection rate for the suckling- and finishing-age groups. In growing pigs, oral fluid had a relatively low Mhp detection rate.

Table 1: *Mycoplasma hyopneumoniae* DNA detection by qPCR at the ISU VDL from 2004 to 2016

Year	Total porcine case submissions, No.	Porcine case submissions for Mhp qPCR testing, No. (%)	Cases positive for Mhp, No.
2004	17,744	1175 (6.6)	263[a]
2005	20,916	2402 (11.5)	680[b]
2006	28,112	4391 (15.6)	1235[b]
2007	27,846	4241 (15.2)	1195[b]
2008	25,254	4456 (17.6)	1499[c]
2009	22,992	6291 (27.4)	1187[d]
2010	25,216	5014 (19.9)	1646[c]
2011	28,938	8948 (30.9)	2789[e]
2012	33,892	8667 (25.6)	3527[f]
2013	38,656	9022 (23.3)	2133[a]
2014	51,146	9163 (17.9)	2513[b]
2015	53,382	13,609 (25.5)	3756[b]
2016	55,275	16,301 (29.5)	2916[d]

[abcdef] Different letters indicate significant differences ($P < .005$; Chi-square analysis for percent of Mhp positive cases over time).
qPCR = quantitative polymerase chain reaction; ISU VDL = Iowa State University Veterinary Diagnostic Laboratory; Mhp = *Mycoplasma hyopneumoniae*.

Figure 1: *Mycoplasma hyopneumoniae* DNA detection by specimen type using qPCR on cases submitted to the ISU VDL from 2004 to 2016. For each bar, blue indicates cases that tested negative and red represents cases with at least 1 positive sample. The number at the top of each bar indicates the percentage of positive cases within each specimen. Different letters indicate significant differences ($P < .05$; Chi-square analysis). qPCR = quantitative polymerase chain reaction; ISU VDL = Iowa State University Veterinary Diagnostic Laboratory; Mhp = *Mycoplasma hyopneumoniae*.

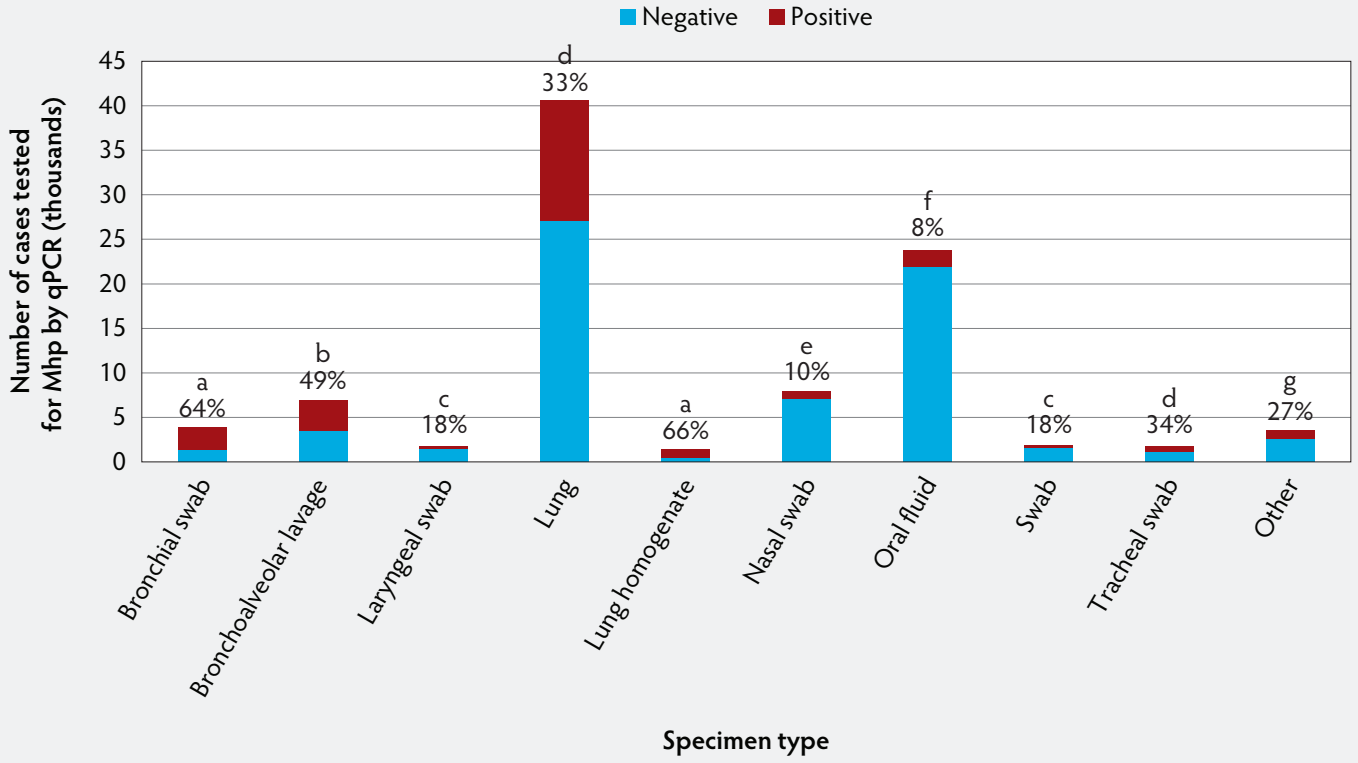
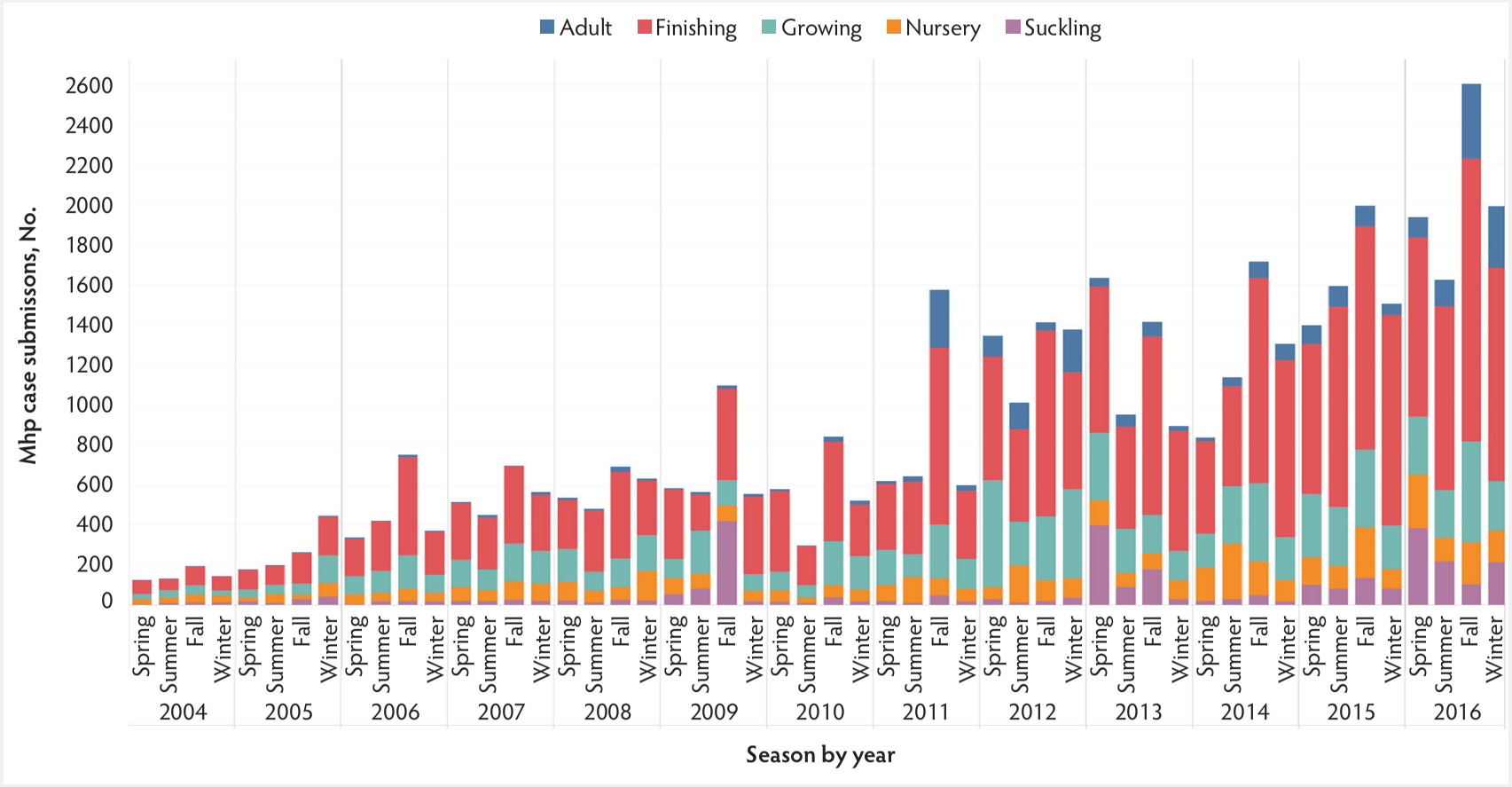


Figure 2: Detection of *Mycoplasma hyopneumoniae* DNA by season using qPCR on cases submitted to the ISU VDL from 2004 to 2016. Seasons are defined as March through May as spring, June through August as summer, September through November as fall, and December through February as winter. A, the number of Mhp cases submitted in each season per year. B, the percentage of positive cases in each season. qPCR = quantitative polymerase chain reaction; ISU VDL = Iowa State University Veterinary Diagnostic Laboratory; Mhp = *Mycoplasma hyopneumoniae*.

Figure 3: Detection of *Mycoplasma hyopneumoniae* DNA by age group and season using qPCR on cases submitted to the ISU VDL from 2004 to 2016. Seasons were defined as March through May as spring, June through August as summer, September through November as fall, and December through February as winter. Pigs less than 3 weeks of age were defined as suckling, 3 to 6 weeks as nursery, 7 to 11 weeks as growing, 12 weeks to 200 days as finishing, and greater than 200 days as adult. qPCR = quantitative polymerase chain reaction; ISU VDL = Iowa State University Veterinary Diagnostic Laboratory; Mhp = *Mycoplasma hyopneumoniae*.



Discussion

Mycoplasma hyopneumoniae is one of the most economically important respiratory pathogens in the US swine industry. This study provides valuable information regarding the distribution of Mhp qPCR results over age group, geographic location, season, specimen, and time. The relatively low positivity rate of Mhp detection by qPCR in 2016 (17.9%) was associated with increased testing from adult- and suckling-age groups and from oral fluids. There was a relative and consistent increase in the total Mhp qPCR performed from 2004 to 2016 at ISU VDL but the percentage of the positive results remained relatively similar, which may be due to the endemicity of Mhp. Finishing-age pigs had a greater number of total cases and greater prevalence of Mhp detection compared to other age groups, which was expected as Mhp is a pathogen known to cause clinical disease in that age group of pigs.[11] The trend of increasing submissions from adult and suckling pigs, as compared to growing pigs, may reflect the implementation of monitoring programs within breeding herds as part of disease control or elimination efforts.[12] Due to a relatively long incubation period and low transmission rate,[13] enzootic pneumonia is usually not reported in pigs younger than 6 weeks of age.[14] Therefore, in this study, Mhp detection was relatively low in suckling and nursery pigs as compared to finishing pigs. Bronchoalveolar lavage fluid and tracheobronchial samples have been reported as specimen types with greater sensitivity when tested with nested PCR to detect Mhp infection, as compared to other specimen types such as nasal, tonsil, and tracheobronchial swabs and lung tissues.[7] In this study, Mhp qPCR detection rate was 49% in bronchoalveolar lavage fluid, 64% in bronchial swab, and 34% in tracheal swab. Likewise, nasal swabs and lung tissues had a relatively lower Mhp detection rate in experimentally infected pigs.[7] In our study, Mhp qPCR detection rate was 10% in nasal swab, and 33% in lung.

The number of cases submitted and the percentage testing positive for Mhp by qPCR were highest during the fall season (September, October, and November). Although not entirely comparable, similar results have been reported in *Mycoplasma pneumoniae* infection in humans.[15] The increased sample submissions in the fall season may be partly attributed to the seasonality of other respiratory pathogens including porcine reproductive and respiratory syndrome virus[16] and influenza A virus.[17] The majority of cases were from the Midwest region of the United States with the greatest number of cases from Iowa, which is not surprising due to the high density of swine in Iowa and the location of the ISU VDL.

Limitations of this study include the fact that testing positive for Mhp by qPCR does not necessarily imply clinical disease. Despite most cases being associated with field investigations, cases involving monitoring and surveillance projects were also included which could have affected conclusions. Also, this analysis was limited to cases submitted to the ISU VDL.

Implications

- Despite the relative and consistent increase of Mhp qPCR performed from 2004 to 2016, the percentage of the positive results remained relatively similar. The overall increase of Mhp qPCR performed is likely a reflection of the overall increase of diagnostic cases submitted to the ISU VDL during this period.
- While the results from samples submitted to the ISU VDL for Mhp qPCR testing had variable detection rates, it is important to keep in mind that these results are based on passive surveillance data.
- The recent increase of Mhp qPCR from samples of suckling and adult pigs may reflect more recent efforts to monitor the progress towards Mhp control and elimination projects rather than investigations of clinical disease in those age groups.

Figure 4: State of origin for cases submitted to the ISU VDL for *Mycoplasma hyopneumoniae* DNA testing by qPCR from 2004 to 2016. The intensity of the blue color represents the number of cases submitted for Mhp testing to the ISU VDL. ISU VDL = Iowa State University Veterinary Diagnostic Laboratory; qPCR = quantitative polymerase chain reaction; Mhp = *Mycoplasma hyopneumoniae*.

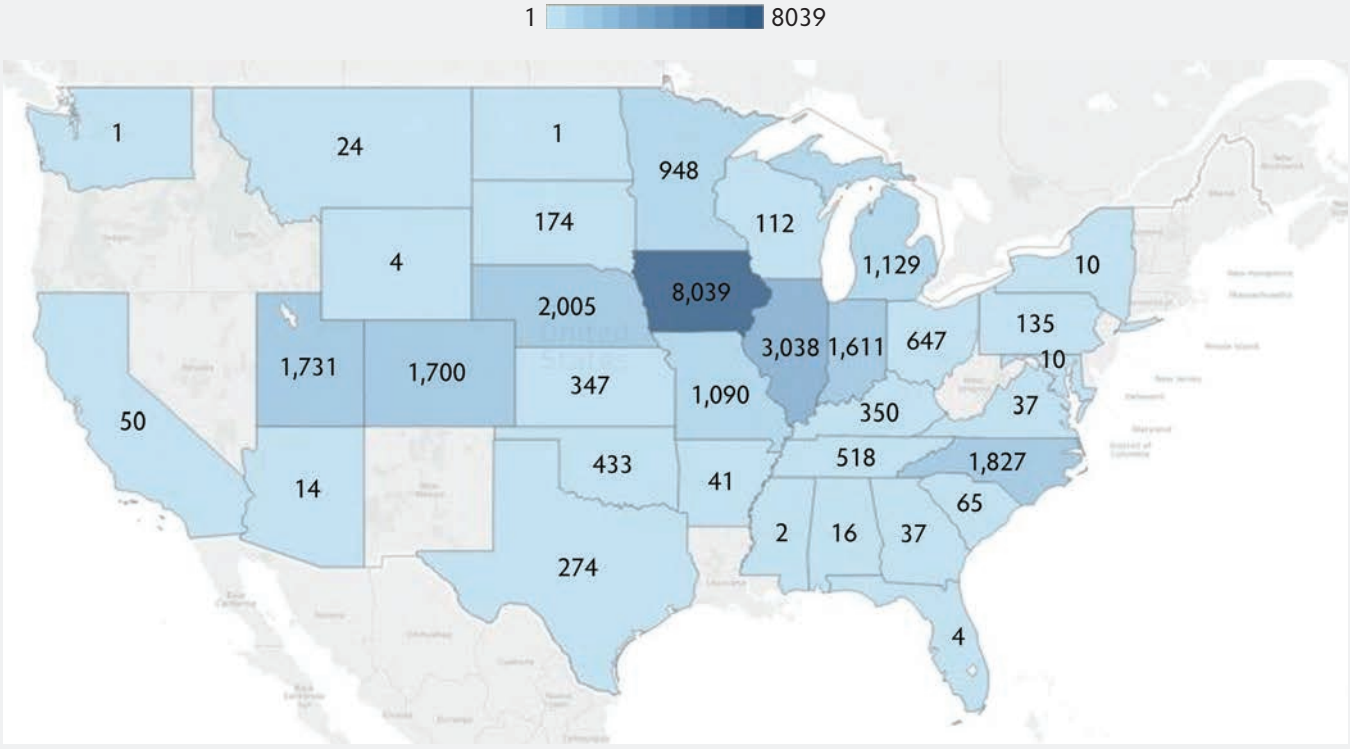
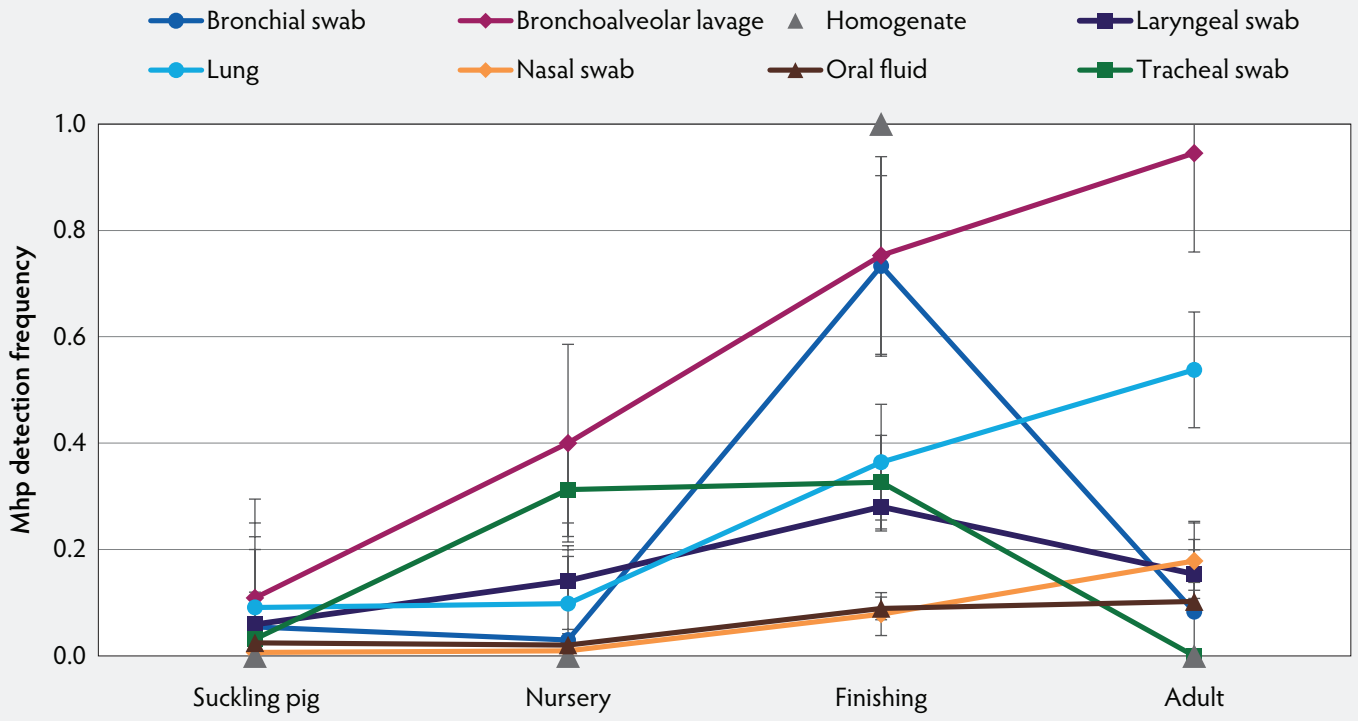


Figure 5: LS Means (with standard deviation) of Mhp DNA detection frequency by age group and specimen type using qPCR on cases submitted to the ISU VDL from 2004 to 2016. Pigs less than 3 weeks of age were defined as suckling, 3 to 6 weeks as nursery, 7 to 11 weeks as growing, 12 weeks to 200 days as finishing, and greater than 200 days as adult. Mhp = *Mycoplasma hyopneumoniae*; qPCR = quantitative polymerase chain reaction; ISU VDL = Iowa State University Veterinary Diagnostic Laboratory.



Acknowledgements

The authors appreciate the cooperation of the ISU VDL in making the database available for assessment.

Conflict of interest

None reported.

Disclaimer

Scientific manuscripts published in the *Journal of Swine Health and Production* are peer reviewed. However, information on medications, feed, and management techniques may be specific to the research or commercial situation presented in the manuscript. It is the responsibility of the reader to use information responsibly and in accordance with the rules and regulations governing research or the practice of veterinary medicine in their country or region.

References

1. Goodwin RF, Hurrell JM, Whittlestone P. Production of enzootic pneumonia in pigs with a *Mycoplasma suipneumoniae* grown in embryonated hens' eggs. *Br J Exp Pathol*.1968;49:431-435.

2. Mare CJ, Switzer WP. New species: *Mycoplasma hyopneumoniae*; A causative agent of virus pig pneumonia. *Vet Med Small Anim Clin*. 1965;60:841-846.

3. Kobisch M, Friis NF. Swine mycoplasmoses. *Rev Sci Tech*. 1996;15:1569-1605.

*4. Holtkamp D. Economic impact of *Mycoplasma hyopneumoniae* on pig farms. Pig333 Web site. **https://www.pig333.com/articles/economic-impact-of-mycoplasma-hyopneumoniae-on-pig-farms_8936/**. Published 2014. Accessed September 19, 2014.

*5. Schwartz M. The cost of *Mycoplasma hyopneumoniae* in growing pigs. *Proc Allen D. Leman Swine Conf*. St. Paul, Minnesota. 2015.

6. Friis NF. Some recommendations concerning primary isolation of *Mycoplasma suipneumoniae* and *Mycoplasma flocculare* a survey. *Nord Vet Med*. 1975;27:337-339.

7. Kurth KT, Hsu T, Snook ER, Thacker EL, Thacker BJ, Minion FC. Use of a *Mycoplasma hyopneumoniae* nested polymerase chain reaction test to determine the optimal sampling sites in swine. *J Vet Diagn Invest*. 2002;14:463-469.

8. Sorensen V, Ahrens P, Barfod K, Feenstra AA, Feld NC, Friis NF, Bille-Hansen V, Jensen NE, Pedersen MW. *Mycoplasma hyopneumoniae* infection in pigs: duration of the disease and evaluation of four diagnostic assays. *Vet Microbiol*. 1997;54:23-34.

9. Strait EL, Madsen ML, Minion FC, Christopher-Hennings J, Dammen M, Jones KR, Thacker EL. Real-time PCR assays to address genetic diversity among strains of *Mycoplasma hyopneumoniae*. *J Clin Microbiol*. 2008;46:2491-2498.

10. Hartigan JA, Wong MA. Algorithm AS 136: A K-means clustering algorithm. *J R Stat Soc Ser C Appl Stat*. 1979;28:100-108.

11. Done SH. Enzootic pneumonia (mycoplasmosis) revisited. *Pig J*. 1996;38:40-61.

12. Holst S, Yeske P, Pieters M. Elimination of *Mycoplasma hyopneumoniae* from breed-to-wean farms: A review of current protocols with emphasis on herd closure and medication. *J Swine Health Prod*. 2015;23(6):321-330.

13. Meyns T, Maes D, Dewulf J, Vicca J, Haesebrouck F, Kruif AD. Quantification of the spread of *Mycoplasma hyopneumoniae* in nursery pigs using transmission experiments. *Prev Vet Med*. 2004;66:265-275.

14. Ross RF. Mycoplasmal diseases. In: Straw BE, D' Allaire S, Mengeling WL, Taylor DJ, eds. *Diseases of Swine*. 8th ed. Ames, Iowa: Iowa State University Press; 1999:495-505.

15. Kim EK, Youn YS, Rhim JW, Shin MS, Kang JH, Lee KY. Epidemiological comparison of three *Mycoplasma pneumoniae* pneumonia epidemics in a single hospital over 10 years. *Korean J Pediatr*. 2015;58(5):172-177.

16. Tummaruk P, Surapat P, Sriariyakun S, Seemakram O, Olanratmanee EO, Tantilertcharoen R, Thanawongnuwech R. Porcine reproductive and respiratory syndrome virus detection in Thailand during 2005-2010 in relation to clinical problems, pig types, regions, and seasons. *Trop Anim Health Prod*. 2013;45:771-779.

17. Chamba Pardo FO, Alba-Casals A, Nerem J, Morrison RB, Puig P, Torremorell M. Influenza herd-level prevalence and seasonality in breed-to-wean pig farms in the midwestern United states. *Front Vet Sci*. 2017;4:167. doi:10.3389/fvets.2017.00167

* Non-refereed references.

COMMENTARY PEER REVIEWED

Evident similarity of porcine postparturient dysgalactia to subclinical porcine coliform mastitis

Andreas Pospischil, DVM, Prof emeritus; Hans U. Bertschinger, DVM, Prof emeritus

Summary

The present commentary aims to motivate future research and initiate new investigation on porcine periparturient disorders. After a short characterization of the clinical presentation of coliform mastitis, this commentary concentrates on the subclinical variant. The subclinical form of the disease resembles in most aspects what is referred to as postparturient dysgalactia syndrome of sows, and is considered highly prevalent in the field. Since the recent introduction of the ill-defined postparturient dysgalactia syndrome, experimental work has declined. Except for review articles, there is a shortage of recent publications in this area. Previously published experimental data led to a promising approach to prevent coliform mastitis by reducing the level of teat contamination by coliform bacteria. With the ongoing need to reduce antimicrobial use in food-producing animals, there is a continued need to investigate preventive strategies.

Keywords: swine, review, mastitis, dysgalactia, prevention

Received: December 9, 2017
Accepted: June 9, 2018

Resumen – Semejanza evidente entre la disgalactia porcina postparto y la mastitis coliforme subclínica

Este comentario pretende motivar estudios futuros e iniciar nuevas investigaciones sobre los trastornos periparto porcinos. Después de una corta caracterización de la presentación clínica de la mastitis coliforme, este comentario se concentra en la variante subclínica. La forma subclínica de esta enfermedad se parece en casi todos los aspectos, a lo que se llama síndrome disgalactico postparto de la cerda y se considera altamente prevalente en el campo. Desde la reciente introducción del mal llamado síndrome disgalactico postparto, el trabajo experimental ha declinado. A excepción de los artículos de análisis, existe una escasez de publicaciones recientes en esta área. La información experimental publicada anteriormente llevó a una estrategia prometedora para prevenir la mastitis coliforme al reducir el nivel de contaminación de la teta con bacterias coliformes. Con la necesidad actual de reducir el uso de los antimicrobianos en animales para consumo, existe una necesidad constante de investigar estrategias de prevención.

Résumé – Similarité évidente de la dysgalactie post-partum porcine et de la mammite subclinique à coliforme porcine

Le présent commentaire vise à motiver des recherches futures et à initier de nouvelles études sur les désordres péri-partum porcins. Après une brève caractérisation de la présentation clinique de la mammite à coliforme, ce commentaire se concentrera sur la variante subclinique. La forme subclinique de la maladie ressemble en plusieurs points à ce qui est appelé le syndrome de dysgalactie postpartum des truies et est considéré comme très prévalent sur le terrain. Depuis la récente introduction du syndrome mal défini de dysgalactie postpartum, le travail expérimental a diminué. À l'exception de quelques article de revue, il y a une pénurie de publications récentes sur le sujet. Des données expérimentales déjà publiées avaient mené à une approche prometteuse pour prévenir la mammite à coliforme en réduisant le niveau de contamination du trayon par les bactéries coliformes. Avec le besoin en cours de réduire l'utilisation d'antimicrobiens chez les animaux de rente, il y a un besoin continu à étudier des stratégies de prévention.

AP: Institute of Veterinary Pathology, Vetsuisse Faculty, University of Zürich, Switzerland.

HUB: Institute of Veterinary Bacteriology, Vetsuisse Faculty, University of Zürich, Switzerland.

Corresponding author: Dr Andreas Pospischil, Zimikerriet 32, CH-8603 Schwerzenbach, Switzerland; Email: apos@vetpath.uzh.ch.

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

Pospischil A, Bertschinger HU. Evident similarity of porcine postparturient dysgalactia to subclinical porcine coliform mastitis. *J Swine Health Prod.* 2018;26(6):316-322.

During the sow's peripartum period, several disorders are frequently observed and of great significance to economics, animal welfare, and pressures to reduce antimicrobial use for prevention and therapy in food-producing animals. Reviews and textbook chapters published during the last four decades illustrate the diverse nomenclature used for these disorders and the absence of a generally accepted theoretical model of pathogenesis.[1-5] In the authors' view, the most recent nomenclature used for these disorders, postparturient dysgalactia syndrome (PPDS), does not account for knowledge achieved in earlier studies about the syndrome and we propose the term PPDS be revisited. The focus of the present commentary is on the gaps in the literature on previous experimental work and is meant to challenge and motivate researchers to revisit the syndrome and address these gaps with ongoing research.

Coliform mastitis

Brief characterization of clinical coliform mastitis

Coliform mastitis (CM) is a febrile peripartum disease, formerly called milk fever, most often observed during the first 24 h after parturition but can also be observed on the day before and up to 2 days post parturition.[6,7] In addition to pyrexia, clinical signs reported include reluctance to allow nursing, anorexia, constipation, thickened white vaginal discharge, increased respiratory rate, reluctance

to rise, signs of insufficient caloric intake in piglets, and lower weight gain in piglets.[2,6,8] These signs are nonspecific and not pathognomonic.[9] To a producer monitoring their animals closely, these clinical signs are more obvious than lesions in the udder.[10] Mastitis is caused by coliform bacteria, ie, members of the family Enterobacteriaceae, that invade through the teat duct.[11] The mode of the bacterial invasion and their spread within the mammary gland determine the irregular expansion of the inflammation.[12,13]

Diagnosis of CM

Classical signs of inflammation, such as swelling, firmness, increased local skin temperature, soreness, and reddening of the skin, may or may not be present. In one field study, only half of the mastitic glands were diagnosed by clinical examination where isolation of *Escherichia coli* was possible and where cytologic smears showed inflammatory cells.[14] In most cases, the udder lesions are limited to single complexes, subcomplexes, or foci of a few centimeters or less in diameter.[15] According to a comprehensive histological study based on 25 tissue blocks per inoculated gland, the most numerous foci and the most severely affected areas are situated dorsally in the udder, ie, close to the abdominal wall and therefore not easily accessible by manual palpation.[13] The glandular tissue is infiltrated by edema, fat, and covered by relatively thick skin.[2] During the critical time postpartum, a variable proportion of glands remain partially or totally unsuckled and therefore can remain congested with colostrum.[16]

These aspects of mastitis presentation in the sow support why it is often difficult to diagnose clinical mastitis even by careful external examination of the udder. This difficulty in diagnosis likely plays a role in the underdiagnosis of mastitis by many clinicians and producers with relation to post-parturient disorders.

A valid determination of mastitis and intramammary infection is only possible if milk samples are collected from each subcomplex of the gland.[2,8] If milked as usual, a sample from a teat is a composite sample of the secretions originating from the two subcomplexes. According to the experience of the authors, a mixed sample will primarily be composed of the secretion of the healthy or healthier subcomplex due to the higher viscosity of the secretion from the affected tissue. The low secretory activity of mastitis-affected foci will also decrease the ability to diagnose mastitis in samples from focally affected subcomplexes. The involution of complexes not suckled by a piglet renders the interpretation of cytological results difficult due to the increased total somatic cell counts (SCC) from such glands which may be significantly higher than counts from mastitic glands.[15,16] Markedly elevated counts of polymorphous neutrophils (PMN) are present as well in secretions from glands undergoing involution. Wegmann and Bertschinger[16] proposed the use of a threshold value composed of a combination of SCC and PMN to discriminate between involution and mastitis. Thus, a SCC of 5×10^6 cells/mL is indicative for mastitis if the proportion of PMN exceeds 70%.[16] The authors experience shows this threshold value is not applicable to samples obtained later than 2 days after parturition. In a more recent paper, a threshold for SCC of 2×10^6 cells/mL was used. However, Kotsarev et al[17] did not explain how they distinguished mastitis from involution without PMN determination.

To the authors' knowledge, a reliable and rapid test for on-farm diagnosis of mastitis is currently not available. Tests developed for use in cattle are not recommended due to the generally higher cell content of sow milk.[4] Additionally, the measurement of milk pH is of limited diagnostic value.[6,14]

A study comparing postmortem lesions in affected sows with control sows demonstrated a significant association between agalactia and mastitis.[6] However, necropsy results in mild cases were not necessarily reliable since inflammatory lesions may be too small to be sampled and affected areas cannot easily be differentiated from unaffected glandular areas macroscopically.[12] In view of the independent processes started by different organisms and at different times, this variability of the mastitic foci in a given sow is not unexpected.[2]

The sensitivity of histologic examination of affected glandular tissue is highly dependent on the way the udder is sectioned and on the number of tissue blocks examined. The most prevalent histologic findings in affected and control sows are edema and congestion.[6] In addition, affected areas show an acute catarrhal-purulent mastitis with conserved acinar structure of the gland. The lactic ducts are filled with epithelial and inflammatory cells. In more severe cases, extensive necrotic foci are sometimes surrounded by neutrophil demarcation.[12,13] Histological findings in other organ tissues have not been significantly correlated with the disorder.[9,12,15]

The results of bacteriological examination of tissue samples taken at the time of necropsy are more reliable than those based on milk samples, since the contamination with environmental or teat skin flora during milk collection is difficult to avoid. Samples from the teat skin and samples of milk exhibit a similar flora when analyzed following enrichment in a fluid medium.[18] This sampling contamination has led to variability and the confusing conclusion that secretions from glands with and without mastitis contain a similar bacterial flora.[5,19,20] Investigators who inoculated solid culture media directly with material from superficially sterilized affected mammary tissue could reliably identify Enterobacteriaceae.[21,22] Since inflammatory lesions persist for longer time periods than culturable bacteria, the latter cannot be consistently isolated from sites with microscopic lesions.[13,14,23] Other non-coliform bacteria, such as *Streptococci* and *Staphylococci*, are only rarely associated with mastitis of mild degrees.[22]

To improve on the effort to obtain a tentative diagnosis based on cytology and bacteriology of the secretion, there have been attempts to utilize clinical pathology criteria for the diagnosis of CM. A marked transitory leukopenia has been observed in experimental acute mastitis.[11,13] However, in field cases this finding is less marked, probably due to the variable time between the onset of an infection in individual glands and the sampling period.[15] Other investigators have observed and reported an increased erythrocyte sedimentation rate and a decreased ratio of plasma protein to fibrinogen.[8] These parameters were confirmed in sows with subclinical mastitis as well.[15] Following experimental inoculations, tumor necrosis factor-α and IL-6 were found to be promising markers for the severity of mastitis[24] while other acute phase proteins proved to be less specific.[24,25]

In summary, the diagnosis of CM in the field is still a challenge. Even with efforts to examine secretions from each gland, some degree of uncertainty in reaching a diagnosis remains.

Subclinical CM

Sows affected by CM may exhibit a wide range of clinical signs ranging from subclinical and

mild hypogalactia to severe mastitis with severe systemic signs.[26] The authors believe that subclinical mastitis, ie, mastitis without visible clinical signs, has not been thoroughly investigated and gaps in our knowledge remain. In postmortem studies comparing mammary glands from mastitis-affected sows to healthy control sows, a variable proportion of the latter showed inflammatory lesions in the glands as well. However, as a rule, foci were less numerous and showed a lower degree of inflammation.[9,12,15] It appears that spontaneous mastitis of all degrees of severity occurs more frequently than the severe cases would indicate. In a field study report, samples of colostrum from 59 sows from 15 herds with a mastitis problem were examined cytologically and bacteriologically. Eighty-three percent (49 of 59) of the sows were affected by mastitis and coliform bacteria were isolated from 71.2% (42 of 59) of the sows. However, no more than 39% (23 of 59) of these sows were febrile at the time of examination.[27] In a research swine herd with an extremely low incidence of clinical CM, subclinical mastitis diagnosed cytologically was detected in 61% (97 of 159) of the farrowings.[28] In a Swedish study based on a population of clinically healthy sows, mastitis with pure cultures of *E coli* and significantly increased SCC was observed in 15.6% (15 of 96) of farrowings on the first day of lactation.[14] Persson et al[14] reported that the bacteria were eliminated between days 3 and 8 of lactation. The importance of subclinical mastitis was also emphasized in a recent study by Kotsarev et al.[17] However, since the exclusion of glands undergoing involution was not mentioned and only SCC were reported, the results cannot be directly compared to other studies.

Additionally, the economic significance of subclinical CM has received little attention to date. The increased erythrocyte sedimentation rates and the decreased ratio of plasma protein to fibrinogen in sows with subclinical mastitis indicate a negative health effect of the disease in affected sows.[15]

Effect of subclinical CM on suckling piglets

Data on milk yield of sows with subclinical coliform mastitis are rare. When average daily gain of piglets from spontaneously agalactic sows with clinical CM was compared to piglets from unaffected sows, piglets from agalactic sows lost weight on the first 2 days postpartum and gained significantly less weight on the third day.[8,29] Sows experimentally inoculated with *E coli* experienced high piglet mortality due to piglet starvation. The surviving piglets grew significantly slower on days 1 through 3 of age but were not significantly lighter at 14 days of age than piglets from resistant and non-inoculated sows. The experimentally inoculated sows that did not develop mastitis had no piglet mortality compared to litters of susceptible sows and piglet average daily gain through 14 days of age was identical to control litters suckling non-inoculated sows.[30] Piglet weights and health of the mammary glands suckled by the piglets were sequentially recorded in a study focusing on the hygiene of the farrowing environment.[23] Subclinical CM developed in 16 of 24 sows (66.7%). Piglets suckled 64.9% (185 of 285) of the healthy glands and 43.5% (27 of 62) of the glands with positive cytology. During the first 4 days of lactation, piglets suckling healthy glands had an average daily gain of 125 g as compared to 105 g in piglets which had suckled glands with mastitis. Average daily gain from day 5 through day 21 was identical regardless of the gland suckled.[23]

In another project studying the influence of four farrowing systems on CM, 159 farrowings were observed. Clinical CM developed in 4 farrowings and subclinical CM was diagnosed in 97 farrowings. The percentage of piglets dying of starvation increased linearly to the incidence of mastitis.[28] Increased piglet mortality is most often caused by starvation. Piglet mortality in a litter is negatively correlated with the weight gain in the first 3 days of life of the surviving litter mates suggesting that low milk production by the sow is associated with piglet starvation.[31] Thompson and Fraser[32] saw marked variation in weight gain within litters in the first 3 days of life. Average daily gains of piglets were negatively correlated with the rectal temperature of the sow. Litters with low initial gains showed more variable gains as well. Such litters were not associated with obvious mastitis suggesting that subclinical disease of the sow might lead to inadequate milk production.[32] In a Swedish study based on 369 farrowings, piglet mortality in the first week and the within-litter standard deviations for weights at 3 weeks of age were correlated to the rectal temperature of the sow in the first 48 h post parturition. Many of these correlations were significant even though the sows affected by clinical mastitis were omitted from the analysis. This indicates that subclinical mastitis negatively affects production performance.[33]

Factors affecting CM severity

The virulence of the pathogen can normally be considered an important factor for the severity of the subsequent disease. However, in the case of CM, such an influence is not well documented. When sows are inoculated intramammarily, the course of the experimental disease induced with identical bacterial cultures may vary greatly.[13,26,30,34] The number of bacteria inoculated does not explain the variable outcome.[1,11,30] Outbreaks of the severe form of the disease have been reported in which almost all sows farrowing over a period of several weeks may be affected and then suddenly no further cases develop for no evident reason.[2] This observation serves as an argument against an increased susceptibility of certain sows. In accordance, sequential observation of 39 sows over six consecutive farrowings resulted in no evidence for individual disposition to CM.[7]

Sows from a specific-pathogen-free herd were resistant to a standardized experimental infection, whereas sows from a conventional herd were highly susceptible.[30] One of several explanations to support these divergent outcomes could be an inapparent viral or bacterial infection in the conventional herd leading to some sort of immunosuppression. Further, a functional difference was detected in the PMN of susceptible sows possibly indicating impaired PMN function.[35] This latter result differs from the findings from a study involving experimental inoculation of 12 sows shortly before parturition. Four sows developed clinical mastitis but this did not appear to impact the functional traits of the circulating granulocytes such as chemotaxis, phagocytosis, or CD18 expression. Österlundh et al[34] concluded that factors other than granulocyte function determine whether a sow will develop clinical mastitis following infection with *E coli*.

Bacteria in the secretion within the mammary gland are immediately exposed to a new microenvironment. Their proliferation is an important factor for the host-microbe balance. The severity of experimental CM depends on the proliferation of the inoculated bacteria. Numerical estimates of the bacteria sequentially recovered from the secretion indicate that glands of susceptible sows harbor substantially more organisms than resistant sows.[30] However, further information to support this finding is scarce. Two strains of *E coli* isolated from CM grew significantly faster in lactoserum taken on

the day of farrowing compared to lactoserum sampled later. These *E coli* strains also grew faster in lactoserum from sows affected with mastitis compared to lactoserum from healthy sows.[36] When a greater number of isolates were examined in untreated colostrum and milk, the situation appeared to be more complex. Not all isolates from porcine CM behave in a similar manner. There are isolates with much slower growth.[37] In addition, there is variation in the bacterial growth rate in secretions from individual sows.[37] In secretions from a healthy, suckled gland, a strain of *E coli* exhibited continual growth throughout lactation, whereas the viable count of the same strain remained either constant or was even reduced in the secretions from healthy, non-suckled glands or from glands with mastitis.[37] The latter finding corresponds with the spontaneous elimination of the organisms from infected glands within about one week.[37]

During gestation, the mammary secretion strongly reduces the growth of coliform bacteria. Following experimental intramammary inoculation two days prior to parturition or external contamination of the teat orifices at the same time, signs of mastitis develop only after the start of parturition.[11,26] The mechanism of this inhibition is not known.

Mammary gland exposure to coliform bacteria

Coliform mastitis is an example of a non-contagious infectious disease. Under the conditions of outdoor pig production, it is rare.[2,6] In 9 Danish herds with outdoor farrowing systems, only 1.1% (13 of 1206) of sows developed CM as compared to 17.1% (286 of 1674) of sows from 9 other herds managed in a traditional indoor confinement system.[38] These observations may indicate a fecal contamination of the teats as a potential source of the infection.

To further test the role of fecal contamination of the teats, 12 sows farrowed in an experimental pen designed to allow the sow to choose where to lay and 12 sows farrowed in a conventional farrowing crate. Viable Enterobacteriaceae counts were performed from the floor in the laying area and from the surface of every teat apex from 3 days before until 1 day after parturition. Colostrum was collected from every teat beginning immediately after parturition and repeated every 12 h. Bacterial counts on the floor and the teats differed between the two systems by a factor of 10 to 1000. Furthermore, *E coli* was isolated from 3 mammary glands in the experimental pen as compared to 27 glands in the conventional crate and about half of the infections were detected in the first sample collected after parturition. In glands with positive cytology but no viable bacteria, mastitis must have been of shorter duration.[23]

Farrowing systems were compared under field-like conditions in another study.[28] Forty sows were assigned to one of four farrowing systems. Viable counts of coliforms on the teat ends were done on gestation day 112 and colostrum was aseptically collected once within 36 h after parturition. Clinical CM was very rare, but subclinical mastitis developed in 61% (97 of 159) of the farrowings. The incidence of mastitis was significantly dependent on the design of the farrowing system which differed with regard to the separation of the areas for laying and for defecation. The incidence of mastitis correlated to enterobacterial counts on the teat ends.[28]

Prevention of CM

Research on CM has identified ways to prevent the disease by protecting the teats from contamination with coliform bacteria during late gestation and the first 3 days of lactation. Coliform bacteria are natural inhabitants of the digestive and urinary tract of the sow. The farrowing system should be designed in a way to prevent the sow from lying in her own excreta. Visual absence of any fecal traces on the ventral skin of the sow is a simple criterion, but not always easy to accomplish with indoor climatic conditions changing throughout the year. If coliform bacteria can successfully be kept away from the lactiferous system of the gland, other prophylactic measures, eg, antibiotic treatment, become less necessary.[23,28]

The role of the sow's own microbiome as a reservoir of coliform bacteria sheds light on the reasons surrounding failure of sanitation measures for the prevention of CM.[2,6] For example, details such as the type of bedding may be important. In a survey of 3000 farrowings in units where wood shavings were used as bedding, 180 (6%) of the farrowings needed treatment for mastitis. Following a change to straw bedding, the incidence over the next 1800 farrowings dropped to 2.5% (45 farrowings).[39] In cattle, where coliform mastitis causes significant loss, maintenance of low levels of coliform bacteria in the bedding is the only effective method of control.[40] Coliform bacteria have the capacity to pass through the bovine streak canal between milking times. The frequency of this event depends on the number of organisms applied.[40] Highest coliform counts are found in sawdust bedding. Incubation of contaminated sawdust at temperatures above 22°C has been reported to allow 1 or 2 $\log_{10}$ proliferation of the organisms.[40]

Postparturient dysgalactia syndrome

Terminology

Many clinically healthy sows nurse litters with increased mortality and poor and uneven growth rates. The various physiological processes occurring around parturition make it difficult to differentiate health problems in these apparently non-diseased sows. Therefore, the early lactation problems should be described as PPDS. That term is preferred over the more traditionally used mastitis-metritis-agalactia (MMA) syndrome.[41] Variations in criteria, assessments, and reporting explain the difficulty to precisely define PPDS. A differentiation of PPDS from MMA is not clearly possible.[5] Careful investigations revealed that metritis is rather uncommon in sows with problem litters[10] and complete agalactia is a very rare exception. Thus, the authors believe that MMA has become a widely used misnomer. Reiner et al[42] preferred the term PPDS over MMA because lactational failure can be a consequence of different pathological processes and lactational failure is the cardinal sign of the economically important average daily gain of piglets. Coliform mastitis is considered a subtype of PPDS[43] or the emerging tip of the iceberg represented by PPDS.[20]

Diagnosis of PPDS

Reports on clinical trials in PPDS are quite rare. Epidemiological research often relies on data collected by animal caretakers. Not all signs must be expressed at the same time in the same sow so a generally accepted clinical description for PPDS based on objective parameters does not exist.[44] Increased rectal temperature was nearly always mentioned as a clinical sign.[19,25,44-47] However, reference values for body temperature are inconsistent with recent findings in healthy sows.[48,49] Other clinical signs recorded include external signs of mastitis,[19,25,44-47] anorexia,[19,25,46] appetite measured indirectly as

change in backfat thickness through lactation[45,50] or body weight,[50] constipation, or vaginal discharge.[25,45,46,50]

Piglet vitality has been assessed by recording mortality during the first weeks of lactation,[45,50] piglet daily body weight gain[45,50] or observation of changes in piglet behavior.[19,44,47] A weakness of the literature is that some investigations into piglet nutrition and development were performed without a detailed look at the health of the sow.[51-53] Colostrum yield through 24 h postpartum was calculated from the weight change of the litters.[51] The wide variability of colostrum yield was attributed to hormonal, environmental, and nutritional factors. Surprisingly, litter size is known to have a strong influence on milk production but does not affect colostrum yield.[51] Sows producing a low amount of colostrum were characterized by leaky mammary epithelium and reduced synthesis of lactose, which may be related to hormonal changes prior to parturition.[52] The within-litter variation of piglet performance until 3 weeks of age appears to be significantly correlated with birth weight and milk intake, whereas birth order and location of the preferred teat do not have a significant influence.[53] A recent review emphasizes the need for more research to improve the yield and composition of colostrum, yet makes no reference to sow health.[54] In summary the authors feel that veterinarians, epidemiologists, and animal science researchers have investigated similar shortcomings of piglet performance without drawing on the expertise of supporting professions.

Etiology and pathogenesis of PPDS

A primary difficulty is the establishment of a functional definition for PPDS. Microbiologists see PPDS primarily as an infectious disease, endocrinologists see it as a hormonal disturbance, and others consider it a nutritional disease.[55] Several studies attribute a central role to endotoxins.[42,43,55] It is well established that inoculation of endotoxin into a pig's circulation induces a systemic disorder which mimics the general signs of CM, whereas oral administration of endotoxin does not induce obvious clinical symptoms. The pig is the least sensitive of mammalian species to parenterally applied endotoxin.[56] Furthermore, local mammary lesions have never been reproduced by endotoxin application except when given intramammarily and endotoxin has been detected in the blood of affected sows only in a minority of CM cases.[57] In a manner similar to that seen in ruminants, De Ruijter et al[58] showed that coliform mastitis-causing bacteria in sows induce acute-phase mediators, which are locally released into the mammary gland, enter the circulation, and act on other tissues including the thermoregulatory central nervous center. Endotoxins, however, essentially remain in the affected gland. Extremely high doses of endotoxin must be inoculated into the mammary gland to be detectable in the blood.

A team of experts in PPDS have presented a new explanation for the syndrome calling it a change in homeorhesis, ie, a fault in the orchestrated changes in metabolism of body tissues necessary to support a physiological state like gestation or lactation.[20] They propose the dys-homeorhesis occurs during the shift from gestation to lactation inciting the development of PPDS and that the physiopathology of PPDS includes feed and feeding in gestation, endotoxemia, and stress. The concept of dys-homeorhesis is broadly discussed at length but the intended benefit of the new theory remains unclear.[20]

Reports addressing results of laboratory examinations of mammary secretions or of necropsy results of sows affected with PPDS were not identified by the authors. Nor has detailed literature regarding the performance of litters from affected sows been detected. The implied multifactorial nature of PPDS[20,42,43] may limit investigators from analyzing the syndrome in more detail. It must be kept in mind that any factor affecting the lactation performance of the sow is defined to take part in the syndrome. In consequence, the search for disease causing or disease accelerating mechanisms may be unrewarding. The authors believe the introduction of PPDS as a new concept coupled with the perceived decrease of active research in CM is very likely due to the idea that the new concept would offer alternative solutions to the problem.

The search for risk factors for PPDS is the only research field where some publications have appeared. The risk factors are typically linked with multifactorial diseases. Many of the factors were found to have minor effects if present independently but found to cause disease if more than one were present.[5] From field observations, the associations of risk factors are not additive but synergistic.[55] Risk factors are often identified based on questionnaires completed by herd owners who diagnose sows affected by PPDS. Examples of statistically significant risk factors are feed and feeding regime, housing, management practices,[43] time of moving sows to the farrowing unit, farrowing induction, feeding sows ad libitum during lactation, frequent farrowing supervision,[59] integration of gilts into the herd after the first farrowing, firm fecal consistency in gestating sows, soiled troughs in lactating sows, low water flow rate in drinking nipples, and high prevalence of lameness.[60]

Similarities between PPDS and subclinical CM

The authors propose that PPDS presents with evident similarities to subclinical CM. The characteristic period of occurrence immediately before and after parturition, the clinical signs in the sow such as fever, anorexia, reluctance to nurse and move, increased vaginal discharge, reduced milk production in the absence of gross mammary lesions, and insufficient milk supply for piglets do not allow distinction of the two affections (Table 1). Clinical CM is considered the proverbial tip of the iceberg of subclinical CM as well as for PPDS.[20] Both PPDS and subclinical CM occur at a high incidence and are assumed to be the cause of uneven development of piglets and litters. Differences are restricted to limited laboratory results for mammary secretions and necropsies in the case of PPDS as well as speculative explanations about the etiology and pathogenesis of PPDS.[55]

Implications

- In view of the wide distribution of subclinical CM and PPDS, investigations into the potential relationship between these two conditions remain an important research area.
- Should further research support that the syndromes are indistinguishable from each other, the development of economically sustainable farrowing systems that aim to reduce the incidence of the syndromes is necessary.
- So far, PPDS is only described in the porcine species and so it is the opinion of the authors that PPDS is indistinguishable from subclinical CM and that a return to the use of subclinical CM terminology will allow future investigators to take advantage of the many parallels to CM in the bovine species and thus inspire new research approaches.

Table 1: Studies reporting clinical signs observed in sows diagnosed with subclinical coliform mastitis or postparturient dysgalactia syndrome

Clinical sign	Published reference	
	Subclinical coliform mastitis	Postparturient dysgalactia syndrome
Fever	Blood et al[2] Martin et al[6] Persson et al[7] Hermansson et al[10] Ross et al[15]	Pendl et al[25] Preissler et al[44] Claeyé et al[45] Tummaruk and Sang-Gasanee[46] Guillou et al[50]
Anorexia	Blood et al[2] Persson et al[7] Hermansson et al[10]	Pendl et al[25] Tummaruk and Sang-Gasanee[46]
Reluctance to rise	Blood et al[2] Martin et al[6] Hermansson et al[10]	Pendl et al[25]
Constipation	Blood et al[2] Martin et al[6] Hermansson et al[10]	Pendl et al[25] Tummaruk and Sang-Gasanee[46] Guillou et al[50]
Vaginal discharge	Blood et al[2] Martin et al[6] Persson et al[7] Hermansson et al[10]	Pendl et al[25] Tummaruk and Sang-Gasanee[46]
External signs of mastitis*	Blood et al[2] Martin et al[6] Hermansson et al[10] Ross et al[15]	Pendl et al[25] Preissler et al[44] Tummaruk and Sang-Gasanee[46]
Hungry piglets	Blood et al[2] Ross et al[15]	Pendl et al[25] Preissler et al[44] Claeyé et al[45] Guillou et al[50]

* Clinical signs absent in the subclinical variant.

Acknowledgements

The support of the reviewers and the editorial staff is highly appreciated.

Conflict of interest

None reported.

Disclaimer

Scientific manuscripts published in the *Journal of Swine Health and Production* are peer reviewed. However, information on medications, feed, and management techniques may be specific to the research or commercial situation presented in the manuscript. It is the responsibility of the reader to use information responsibly and in accordance with the rules and regulations governing research or the practice of veterinary medicine in their country or region.

References

1. Jones JET. Acute coliform mastitis in the sow. *Vet Ann*. 1979;19:97-101.

2. Blood DC, Henderson JA, Radostits OM. *Veterinary Medicine*. 5th ed. London: Tindall Baillère;1979.

3. Gerjets I, Kemper N. Coliform mastitis in sows: A review. *J Swine Health Prod*. 2009;17:97-105.

4. Martineau GP, Farmer C, Peltoniemi O. Mammary system. In: Zimmerman JJ, Karriker LA, Ramirez A, Schwartz KJ, Stevenson GW, eds. *Diseases of Swine*. 10th ed. Ames, Iowa: Blackwell Publishing; 2012:270-293.

5. Fairbrother JM, Gyles CL. Colibacillosis. In: Zimmerman JJ, Karriker LA, Ramirez A, Schwartz KJ, Stevenson GW, eds. *Diseases of Swine* 10th ed. Ames, Iowa, Blackwell Publishing; 2012:723-749.

6. Martin CE, Hooper BE, Armstrong CH, Amstutz HE. A clinical and pathologic study of the mastitis-metritis-agalactia syndrome of sows. *JAVMA*. 1967;151:1629-1634.

7. Persson A, Pedersen AE, Göransson L, Kuhl W. A long term study on the health status and performance of sows on different feed allowances during late pregnancy. I. Clinical observations, with special reference to agalactia post partum. *Acta Vet Scand*. 1989;30:9-17.

8. Ross RF, Zimmermann BJ, Wagner WC, Cox DF. A field study of coliform mastitis in sows. *JAVMA*. 1975;167:231-235.

9. Nachreiner RF, Ginther OJ, Ribelin WE, Carlson IH. Pathologic and endocrinologic changes associated with porcine agalactia. *Am J Vet Res*. 1971;32:1065-1075.

10. Hermansson I, Einarsson S, Larsson K, Bäckström L. On the agalactia post partum in the sow. A clinical study. *Nord Vet Med*. 1978;30:465-473.

11. Bertschinger HU, Pohlenz J, Middleton-Williams DM. Untersuchungen über das Mastitis-Metritis-Agalaktie-Syndrom (Milchfieber) der Sau [Studies on the mastitis-metritis-agalactia syndrome (milk fever) in sows. III. Galactogenic induction of Klebsiella-induced mastitis.] *Schweizer Archiv für Tierheilkunde*. 1977;119:265-275.

12. Middleton-Williams DM, Pohlenz J, Lott-Stolz G, Bertschinger HU. Untersuchungen über das Mastitis-Metritis-Agalaktie-Syndrom (Milchfieber) der Sau. I. Pathologische Befunde bei Spontanfällen.[Studies on the mastitis-metritis-agalactia syndrome (milk fever) in sows. I. Pathological findings in spontaneous cases]. *Schweizer Archiv für Tierheilkunde*. 1977;119:213-222.

*13. Loepfe PJ. Experimentelle Mastitis bei der Sau. Korrelation der pathologisch-anatomischen und histologischen Befunde 4-30 Tage nach der Ansteckung mit *E. coli* und *Klebsiella pneumoniae* [Experimental mastitis in the sow. Correlation of the pathological and histological findings 4-30 days after inoculation with *E. coli* and *Klebsiella pneumoniae*]. [Thesis]. Switzerland: University of Zürich; 1993.

14. Persson A, Pedersen Mörner A, Kuhl W. A long-term study on the health status and performance of sows on different feed allowances during late pregnancy. III. *Escherichia coli* and other bacteria, total cell content, polymorphonuclear leucocytes and pH in colostrum and milk during the first 3 weeks of lactation. *Acta Vet Scand*. 1996;37:293-313.

15. Ross RF, Orning AP, Woods RD, Zimmermann BJ, Cox DF, Harris DL. Bacteriologic study of sow agalactia. *Am J Vet Res*. 1981;42:949-955.

*16. Wegmann P, Bertschinger HU. Sequential cytological and bacteriological examination of the secretions from sucked and unsucked mammary glands with and without mastitis. *Proc IPVS*. Ghent, Belgium, 1984;287.

*17. Kotsarev VN, Skrylmikov OH, Sotnikov AB. Subclinical mastitis in sows. *Svinovodstvo* (Moskva) 2010;6:33-34.

*18. Preissler R, Kemper N. Bacteriological analysis of the skin flora on sow's mammary glands. *Proc 14th Int Cong Int Soc Anim Hyg*. Vechta, Germany, 2009:259-262.

19. Kemper N, Gerjets I. Bacteria in milk from anterior and posterior mammary glands in sows affected and unaffected by postpartum dysgalactia syndrome (PPDS). *Acta Vet Scand*. 2009;51:26-32.

20. Martineau GP, Le Treut Y, Guillou D, Waret-Szkuta A. Postpartum dysgalactia syndrome: A simple change in homeorhesis? *J Swine Health Prod*. 2013;21(2):85-93.

21. Armstrong CH, Hooper BE, Martin CE. Microflora associated with agalactia syndrome of sows. *Am J Vet Res*. 1968;29:1401-1407.

22. Bertschinger HU, Pohlenz J, Hemlep I. Untersuchungen über das Mastitis-Metritis-Agalaktie-Syndrom (Milchfieber) der Sau. II. Bakteriologische Befunde bei Spontanfällen. [Studies on the mastitis-metritis-agalactia syndrome (milk fever) in sows. II. Bacteriological findings in spontaneous cases]. *Schweizer Archiv für Tierheilkunde*. 1977;119:223-233.

23. Bertschinger HU, Bürgi E, Eng V, Wegmann P. Senkung der Inzidenz von puerperaler Mastitis bei der Sau durch Schutz des Gesäuges vor Verschmutzung [Reduction of the incidence of puerperal mastitis in the sow by protection of the mammary gland against faecal contamination]. *Schweizer Archiv für Tierheilkunde*. 1990;132:557-566.

24. Zhu Y, Österlundh I, Hultén F, Magnusson U. Tumor necrosis factor-α, interleukin-6, serum amyloid, haptoglobin, and cortisol concentrations in sows following intramammary inoculation of *Escherichia coli*. *Am J Vet Res*. 2004;65:1434-1439.

25. Pendl W, Jenny B, Torgerson P, Spring P, Kümmerlen D, Sidler X. Auswirkungen einer Bestandesbetreuung auf das Vorkommen des postpartalen Dysgalaktie Syndroms (PPDS) und die Tierbehandlungsinzidenz [Effect of herd health management on the prevalence of postpartum dysgalactia syndrome (PPDS) and the treatment incidence]. *Schweizer Archiv für Tierheilkunde*. 2017;159:109-116.

26. Magnusson U, Pedersen Mörner A, Persson A, Karlstam E, Sternberg S, Kindahl H. Sows intramammarily inoculated with *Escherichia coli*: Influence of time of infection, hormone concentrations and leukocyte numbers on development of disease. *J Vet Med B Infect Dis Vet Public Health*. 2001;48:501-512.

*27. Wegmann P, Bertschinger HU, Jecklin H. A field study on the prevalence of coliform mastitis (MMA) in Switzerland and the antimicrobial susceptibility of the coliform bacteria isolated from the milk. *Proc IPVS*. Barcelona, Spain. 1986:92.

*28. Bertschinger HU, Drossaart van Dusseldorp P, Troxler J. Incidence of intramammary infection and mastitis in sows kept in four farrowing systems. *Proc Int Soc Anim Hyg*. St. Paul, Minnesota. 1994;93-95.

29. Nachreiner RF, Ginther OJ. Porcine agalactia: Hematologic, serum chemical, and clinical changes during the preceding gestation. *Am J Vet Res*. 1972;33:799-809.

30. Ross RF, Harmon RL, Zimmermann BJ, Young TF. Susceptibility of sows to experimentally induced *Escherichia coli* mastitis. *Am J Vet Res*. 1983;44:949-954.

31. De Passillé AMB, Rushen J. Using early suckling behavior and weight gain to identify piglets at risk. *Can J Anim Sci*. 1989;69:535-544.

32. Thompson BK, Fraser D. Variation in piglet weights: Weight gains in the first days after birth and their relationship with later performance. *Can J Anim Sci*. 1988;68:581-590.

33. Göransson L. The effect of feed allowance in late pregnancy on the occurrence of agalactia post partum in the sow. *J Vet Med A*. 1989;36:505-513.

34. Österlundh I, Hultén F, Johannisson A, Magnusson U. Sows inoculated intramammarily with *Escherichia coli* at parturition: I. Functional capacity of granulocytes in sows affected or non-affected by clinical mastitis. *Vet Immunol Immunopathol*. 2002;90:35-44.

35. Löfstedt J, Roth JA, Ross RF, Wagner WC. Depression of polymophonuclear leucocyte function associated with experimentally induced *Escherichia coli* mastitis in sows. *Am J Vet Res*. 1983;44:1224-1228.

36. Raekallio M. Enhanced growth of *E. coli* in whey from sows with agalactia syndrome. *Acta Vet Scand*. 1987;28:177-180.

*37. Wegmann P. Zur Pathogenese der Colimastitis beim Mutterschwein. [Contribution to the pathogenesis of coliform mastitis in the sow]. [Thesis]. Zürich, Switzerland: University of Zürich; 1985.

*38. Mortensen B, Ruby V, Pedersen BK, Smidth J, Aarestrup Larsen V. Outdoor pig production in Denmark. *Pig News and Inform*. 1994;15(4):117N-120N.

*39. Muirhead MR. Veterinary problems of intensive pig husbandry. *Vet Rec*. 1976;99:288-292.

*40. Bramley AJ, Neave FK. Studies on the control of coliform mastitis in dairy cows. *Brit Vet J*. 1975;131:160-169.

*41. Klopfenstein C, Farmer C, Martineau GP. Diseases of the mammary glands and lactation problems. In: Straw BE, D'Allaire S, Mengeling WL, Taylor DJ, eds. *Diseases of Swine*. 8th ed. Ames, IA: Iowa State University Press; 1999:833-860.

*42. Reiner G, Hertrampf B, Richard HR. Postpartales Dysglaktiesyndrom der Sau – eine Übersicht mit besonderer Berücksichtigung der Pathogenese. [Postparturient dysgalactia syndrome in the sow - Review with a special focus on the pathogenesis]. *Tierärztliche Praxis Grosstiere* 2009;305-318.

43. Maes D, Papadopoulos G, Cools A, Janssens GPJ. Postpartum dysgalactia in sows: pathophysiology and risk factors. *Tierärztl Prax*. 2010;38:S15-S20.

*44. Preissler R, Gerjets I, Reiners K, Looft H, Kemper N. Prevalence of postpartum dysgalactia syndrome in sows. *Proc Int Soc Anim Hyg*. Vienna, Austria. 2011;63-65.

*45. Claeyé E, Beck J, Meyns T, Maes D. Effect of ketoprofen treatment in the prevention of postpartum dysgalactia syndrome in sows. *Vlaams Diergeneesk Tijdschr*. 2015;84:127-132.

*46. Tummaruk P, Sang-Gasanee K. Duration of parturition is associated with incidence of postparturient disorders in sows. *Thai J Vet Med*. 2011;41Suppl:150-151.

47. Preissler R, Tetens J, Reiners K, Looft H, Kemper N. A genome-wide association study to detect genetic variation for postpartum dysgalactia syndrome in five commercial pig breeding lines. *Anim Genet*. 2013;44:502-508.

48. Sipos W, Wiener S, Entenfellner F, Sipos S. Physiological changes of rectal temperature, pulse rate and respiratory rate of pigs at different ages including the critical peripartal period. *Wiener Tierärztliche Monatsschrift*. 2013;100:93-98.

49. Stiehler T, Heuwieser W, Pfützner A, Burfeind O. The course of rectal and vaginal temperature in early postpartum sows. *J Swine Health Prod*. 2015;23:72-83.

50. Guillou G, Benthem de Grave X, van der Aar PJ, Molist R. Influence of fiber type and content and amino acid levels in the sow lactation diet on farrowing process, sow health, and piglet vitality. *J Anim Sci*. 2016;94:146-149.

51. Devillers N, Farmer C, Le Dividich J, Prunier A. Variability of colostrum yield and colostrum intake in pigs. *Animal*. 2007;1:1033-1041.

52. Foisnet A, Farmer C, David C, Quesnel H. Relationship of colostrum production by primiparous sows and sow physiology around parturition. *J Anim Sci*. 2010;88:1672-1683.

*53. Hemsworth PH, Winfield CG, Mullaney PD. Within-litter variation of piglets to three weeks of age. *Anim Prod.* 1976;22:351-357.

54. Farmer C, Quesnel H. Nutritional, hormonal, and environmental effects on colostrum in sows. *J Anim Sci*. 2009:87Suppl.1:56-65.

55. Martineau GP, Smith BB, Doizé B. Pathogenesis, prevention, and treatment of lactational insufficiency in sows. *Vet Clin North Am Food Anim Pract*. 1992;8:661-684.

56. Holst H, Edqvist LE, Kindahl H, Rylander R. Effects of oral and intravenous administration of endotoxin in prepubertal gilts. *J Vet Med A*. 1993;40:33-44.

57. Pejsak Z, Tarasiuk K. The occurrence of endotoxin in sows with coliform mastitis. *Theriogenology*. 1989;32:335-341.

58. De Ruijter K, Verheijden JHM, Pijpers A, Berends J. The role of endotoxin in the pathogenesis of coliform mastitis in sows. *Vet Q*. 1988;10:186-190.

59. Papadopoulos GA, Vanderhaeghe C, Janssens GP, Dewulf J, Maes DG. Risk factors associated with postpartum dysgalactia syndrome in sows. *Vet J*. 2010;184:167-171.

60. Jenny B, Vidondo B, Pendl W, Kümmerlen D, Sidler X. Erhebung von Risikofaktoren für Mastitis-Metritis-Agalaktie in Schweinebetrieben in der Schweiz [Evaluation of risk factors for mastitis-metritis-agalactia in pig farms in Switzerland]. *Schweizer Archiv Tierheilkunde*. 2015;157:689-696.

* Non-refereed references.

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

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

Conversion chart, kg to lb (approx)

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

Rethink the norm:

Are today's large litters getting adequate nutrition?

by Mark Eisenhart, DVM, Director of Technical Services, Tonisity

Genetic companies have done an amazing job creating sow lines that produce more live pigs. But this gain comes with a challenge: having enough nutrition to support piglet viability and larger litter growth.

Sometimes we forget that farrowing is not the end of development. In the first 10 days of life, the absorptive surface area of a pig's intestinal tract doubles. During this critical developmental period, nutritional supplementation of the intestinal tract can help pigs achieve maximum productivity and reach their full potential. This is why we recommend feeding Tonisity Px™ to pigs from 2 to 8 days of age.

"The challenge is having enough nutrition to support piglet viability and larger litter growth."

- Mark Eisenhart, DVM

Boost intestinal development

Producers have a real opportunity to improve production with Tonisity Px, a one-of-a-kind intestinal development solution. Unlike anything else on the market, it nourishes the enterocytes of the piglet intestine and has the same composition as its body's cells, making it easily absorbed. Equally important is its taste profile. It is designed just for pigs with a taste that baby pigs love and will eagerly consume in the first days of life. When you supplement pigs with Tonisity Px Days 2-8, it keeps them eating, drinking – and growing.

Research shows Tonisity Px improves intestinal integrity by increasing surface area, giving every pig a chance to maximize productivity. This results in more pigs weaned, less size variability and faster weight gain. These benefits provide pigs a solid foundation for continued performance and economic success.

Learn more about Tonisity Px at www.tonisity.com.

Why intestinal development matters

Keeping pigs eating and drinking is crucial, says Nicholas Gabler, Ph.D., Iowa State University swine nutrition specialist. "Keeping the intestine hydrated and providing it with nutrients improves its development and ability to recover," he says.

Dr. Gabler says products like Tonisity Px support the structural and functional aspects of the intestine. "Good intestinal function allows the pig to optimize nutrient, water and energy absorption to support growth," he explains.

"If we can provide nutritional and energetic support to the intestine – the largest immune organ in the body – we can help the pig facilitate efficient and effective nutrient digestion and absorption. Doing so allows for optimal uptake of nutrients to support lean tissue growth."

"Keeping the intestine hydrated and providing it with nutrients improves its development and ability to recover.

- Nicholas Gabler, Ph.D., Iowa State University swine nutrition specialist

Pork groups working together with US Department of Agriculture to protect the United States from African swine fever

The National Pork Board, along with the American Association of Swine Veterinarians, the National Pork Producers Council, the Swine Health Information Center, and the US Department of Agriculture (USDA), continues to work on key strategies and tactics to keep US pig farms free of African swine fever (ASF).

The USDA Animal Plant and Health Inspection Service has a disease response strategy for African swine fever called *FAD PReP*. States and the pork industry use this information to do state-specific planning. They have also committed to work with the industry to develop and host an ASF-specific exercise in 2019 to test key response functions necessary for successful ASF management and containment. Exercise participants will include pork producers, swine veterinarians, packer and processors, and allied industry. Invitations to participate will be sent to Canada and Mexico.

For more information, contact Dave Pyburn at DPyburn@pork.org or 515-223-2634.

Illinois farmer becomes new America's Pig Farmer of the Year

This year's finalists vying for the National Pork Board's America's Pig Farmer of the Year program were Patrick Bane of Arrowsmith, Illinois; Bill Luckey of Columbus, Nebraska; Brad Lundell of Kiron, Iowa; and Kevin Rasmussen of Goldfield, Iowa. After an in-depth interview with a third-party national judging panel, Bane took the top honor and will be traveling over the next year to represent the nation's pig farmers.

"The finalists in this competition demonstrate how pig farmers embrace the We Care ethical principles as their daily standard of care," said National Pork Board President Steve Rommereim, a pig farmer from Alcester, South Dakota. "We congratulate Pat Bane. We know he will do a great job over the next year."

For more information about the program or to nominate someone for the 2019 program, contact Mike King at MKing@pork.org or 515-223-3532.

Patrick Bane - 2018 America's Pig Farmer of the Year

Pork industry focuses on feed ingredients to combat African swine fever threat

With the ongoing outbreak of African swine fever (ASF) in China, the National Pork Board, along with the American Association of Swine Veterinarians, the National Pork Producers Council, the Swine Health Information Center (SHIC), and the US Department of Agriculture, are working closely to help keep the United States free of ASF and other foreign animal diseases (FAD). This includes focusing on the importation of feed ingredients, a key area of potential high risk of disease transport.

Thanks to Checkoff-funded research conducted after the porcine epidemic diarrhea virus outbreak, swine industry experts now have some peer-reviewed science to rely on when looking at ways to mitigate the current risk posed by ASF in China and other countries. This includes work done on imported feed ingredients.

Paul Sundberg, DVM, director of the Swine Health Information Center, cites SHIC-funded research that shows viruses do have the potential to travel long distances via feed ingredients, which proves the theoretical ability of a FAD pathogen to reach US shores. To help prevent this potential risk from becoming a reality, swine industry experts have compiled these seven critical points for pig farmers to raise with their feed and feed-ingredient suppliers with the objective of starting a dialog about feed ingredient safety. They are:

1. Describe the facility's biosecurity program to minimize the spread of pathogens from people, vehicles, and ingredients.
2. Describe the facility's employee training on feed safety.
3. Describe the facility's pest control program.
4. Describe the facility's traceability program.
5. Describe the facility's supplier approval program.
6. Is the facility certified by a third-party certification body for food safety?
7. Does the facility utilize ingredients that were manufactured or packaged outside of the United States?

For more information, go to www.securepork.org and www.pork.org/FAD.

Circumvent® G2 PCV-M

START EARLIER. LAST LONGER.[1]

FOR PROTECTION THAT BEGINS AS YOUNG AS 3 DAYS, DOI of up to 5 months and over a decade of success with more than 100 million pigs vaccinated, veterinarians and pig producers choose Circumvent® G2.

Producers who use Circumvent® G2 see reduced mortality, fewer culls, improvement in ADG and improved feed conversion rates, all of which help to elevate herd health – and protect your bottom line.[2]

DRIVEN BY PREVENTION™

DrivenByPrevention.com

START ON DAY 3 LAST THROUGH 5 MONTHS

1 Based on label claims of product including 3 days of age and demonstrated five-month duration of immunity
2 Versus non-vaccinated pigs

9/16 SW-55269-2

MERCK Animal Health

AASV NEWS

Who will make history at the 50th AASV Annual Meeting?

Twenty-five years ago, at the 1994 AASP Annual Meeting, Dr Beth Lautner became the first woman – one of only two ever – to receive the prestigious Howard Dunne Memorial Award. At the same meeting, Dr Alan Davis, a practitioner from Flanagan, Illinois, was named the Swine Practitioner of the Year, and AASP charter member Dr Wally Brandt received the Meritorious Service Award. Dr Brandt was instrumental in recording the history of the first 25 years of the association, raising the question: Who will make history at the 50th AASV Annual Meeting?

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

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 service to the AASV members, AASV officers, and the AASV staff.

Swine Practitioner of the Year – Given annually to the swine practitioner who is an AASV member and 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, and 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, Iowa 50220, Email: **aasv@aasv.org**.

AASV members receive discount on the 11th edition of *Diseases of Swine* - now with color photos

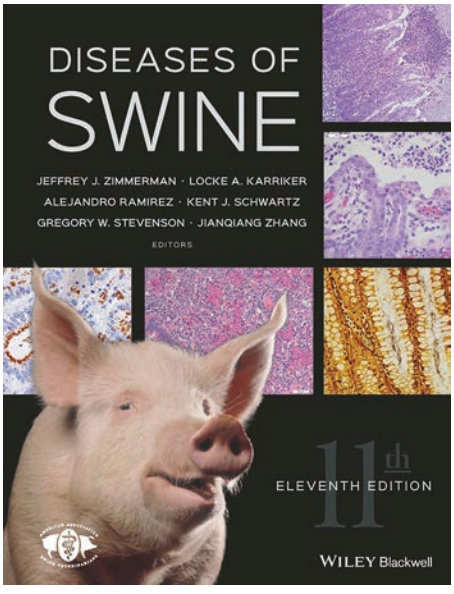
The classic veterinary reference *Diseases of Swine* has been completely revised, and preorders for the 11th edition may be placed now at **www.wiley.com**. The publisher anticipates a mid-April 2019 release date. AASV members receive a 20% discount when purchasing the book using the order code available at **www.aasv.org/members**.

Diseases of Swine has been the definitive reference on swine health and disease for over 60 years. This new edition has been completely revised to include the latest information, developments, and research in the field. Now with full color images throughout, this comprehensive and authoritative resource has been redesigned for improved consistency and readability, with a reorganized format for more intuitive access to information.

The book's editors, Drs Jeffrey Zimmerman, Locke Karriker, Alejandro Ramirez, Kent Schwartz, Gregory Stevenson, and Jianqiang Zhang, are all AASV members and faculty at Iowa State University.

Diseases of Swine covers a wide range of essential topics on swine production, health, and management, with contributions from more than 100 of the foremost international experts in the field. This revised edition makes the information easy to find and includes expanded information on welfare and behavior.

Written for veterinarians, academicians, students, and individuals and agencies responsible for swine health and public health, *Diseases of Swine* is considered by many to be an essential guide to swine health.



Boehringer Ingelheim

AWARDS FOR ADVANCING RESEARCH IN RESPIRATORY DISEASE

$105,000

- Three awards of $35,000 each will be presented
- Projects can be in the areas of PCV2, Mhp, IAV-S and PRRS
- Open to veterinarians, researchers and academia
- Established in 2002 by Boehringer Ingelheim to help advance solutions to the PRRS challenge

Proposal Deadline: January 15, 2019

Visit **www.SwineResearchAwards.com**
for complete information and application instructions.
1-800-325-9167

FOUNDATION NEWS

Just DO it!

The time is NOW to join the efforts to achieve $2 million in AASV Foundation restricted funds before the 2019 AASV Annual Meeting. All AASV members can play a part in achieving this goal and in celebrating 50 years of educational opportunities with our organization.

Our AASV Foundation endowment provides financial help in many ways to our swine veterinary profession. The Foundation funds scholarships for exceptional veterinary students as well as for member swine veterinarians seeking advanced degrees and certifications, including 4 individuals pursuing board certification in animal welfare. It supports research into the diseases you fight every day and emerging disease, too. The Foundation provides dollars to veterinary students seeking swine practice experience, and funds travel stipends for students attending our annual meeting. In addition, it supports funding for annual meeting keynote lectures that inspire and motivate our membership every year.

Because we all have benefitted from one or more of these foundation-funded programs, it is time for each of us to rise to the challenge! Please help continue the legacy of support that has enabled the foundation to accomplish so much - donate to the AASV Foundation endowment. Endowed contributions are invested to produce income to ensure the availability of funding well into the future. Your donation will have a lasting impact on the profession.

AASV Foundation endowed giving programs

Leman

If you're not already a Leman Fellow, you should be! Named for the late industry leader and former AASV president, Dr Allen D. Leman, this giving program confers the title of Leman Fellow upon those who contribute $1,000 or more to the foundation endowment.

Heritage

The Heritage Fellow program represents the next level of support for the foundation, recognizing contributions of $5,000 or more.

Legacy

The Legacy Fund provides an opportunity to recognize a principal donor or an honoree through a significant contribution to the endowment. A donor, multiple donors, or a veterinary practice may establish and name a Legacy Fund with a monetary gift of $50,000 or more. The fund may be named after the donor, another individual, or group.

In addition to monetary donations, Heritage and Legacy Fellows may select from additional contribution options, including the assignment of in-force life insurance policies and stock transfers.

For more information about the AASVF endowment giving programs or to make a contribution, see **aasv.org/foundation**, or contact the AASV Foundation by phone, 515-465-5255, or email, **aasv@aasv.org**.

AMVC golfers earn first place at foundation fundraiser

With a team score of 59, the foursome from AMVC took top honors at this year's AASV Foundation golf fundraiser. The event took place on August 23 at Landsmeer Golf Club in Orange City, Iowa. Golfers Mike Bauer, Josh Ellingson, Nick Weihs, and Gavin Yaeger combined their efforts in the best-ball tournament to secure the win over ten other teams on the course.

Dave Bomgaars, Austin DeZeuw, Dave Iverson, and Doug Sullivan made up the second-place team, hosted by Elanco Animal Health. They trailed the leaders by only 2 strokes, achieving a score of 61. Likewise, 2 strokes separated them from the third-place team hosted by the National Pork Producers Council. Jack Bair, Derek Kindwall, Jeff Kindwall, and Greg Thornton worked together to come in 8 under par with a score of 63.

Regardless of their placings, the 43 golfers enjoyed an afternoon filled with contests, drawings, and giveaways as they made their way around the course thanks to exceptionally strong support from sponsors. Fifteen companies chipped in to provide financial support for the outing in the form of beverage, lunch, dinner, and golf hole sponsorships. Besides adding to the enjoyment of the participants, their support increased the event's profitability for the foundation.

The proceeds from the annual golf outing support foundation programs including scholarships, research grants, travel stipends for veterinary students to attend the annual meeting, tuition support for the Swine Medicine Education Center, swine externship grants for veterinary students, and more.

The event concluded with the awards dinner sponsored by Boehringer Ingelheim Animal Health. The golf outing coordinator, Josh Ellingson, announced the team and individual contest winners as follows:

Championship flight

First place team hosted by AMVC (score of 59): Mike Bauer, Josh Ellingson, Nick Weihs, and Gavin Yaeger

Second place team hosted by Elanco Animal Health (score of 61):
Dave Bomgaars, Austin DeZeuw, Dave Iverson, and Doug Sullivan

Third place team hosted by NPPC (score of 63): Jack Bair, Derek Kindwall, Jeff Kindwall, and Greg Thornton

First flight

First place team hosted by Boehringer Ingelheim Animal Health (score of 65): Jeff Blythe, Brent Carmichael, Tom Grady, and Justin Rustvold

Second place team hosted by Ceva Animal Health (score of 65): Keith Bretey, Jeff OKones, Julie Schwalbe, and Jon Thompson

Third place team (score of 66): Daryl Hammer, Michael Lash, and Dan Rosener

Second flight

First place team hosted by Fast Genetics (score of 67): Marcus Kehrli, Kent Schwartz, Steve Sprague, and Jeff Zimmerman

Second place team hosted by Pharmgate Animal Health (score of 71): Adam Gutierrez, Jeff Hall, Alex Hintz, and Ralph Wilson

Third place team hosted by Topigs Norsvin (score of 71): Mitch Christensen, Jordan Graham, Sam Holst, and Mike Terrill

Individual contests

Hole #3, **Closest to 100-yard marker:** Jack Creel

Hole #4, **Closest to the pin:** Jeff OKones

Hole #8, **Closest 2nd shot:** Nick Weihs

Hole #9, **Longest putt:** Jeff Zimmerman

Hole #10, **Longest drive:** Nick Weihs

Hole #13, **Closest to the pin:** Tom Grady

Hole #17, **Longest putt:** Daryl Hammer

Hole #18, **Drawing for cooler:** Steve Sprague

The team hosted by AMVC took top honors at this years' AASV Foundation Golf Outing. Left to right: Josh Ellingson, Gavin Yaeger, Mike Bauer, and Nick Weihs.

The second place team was hosted by Elanco Animal Health.
Left to right: Austin DeZeuw, Dave Bomgaars, Doug Sullivan, and Dave Iverson.

The National Pork Producers Council hosted this year's third place team. Left to right: Jeff Kindwall, Greg Thornton, Jack Bair, and Derek Kindwall.

Photos by Erin Kinman, courtesy of Andrew Kleis at Insight Wealth Group.

Foundation extends call for research proposals, due January 18

As part of its mission to fund research with direct application to the profession, the American Association of Swine Veterinarians Foundation is accepting research proposals to be considered for funding in 2019. Proposals are **due January 18, 2019** and may request a maximum of $30,000 per project. A maximum of $60,000 will be awarded across two or more projects. The announcement of projects selected for funding will take place at the AASV Foundation Luncheon in Orlando, Florida on Sunday, March 10, 2019. Awardees will be notified in advance.

Proposed research should fit one of the five action areas stated in the AASV Foundation mission statement (see grey sidebar).

The instructions for submitting proposals are available on the AASV Foundation Web site at **www.aasv.org/foundation/2019/research.php**. Proposals may be submitted by mail or email (preferred).

A panel of AASV members will evaluate and select proposals for funding based on the following scoring system:

- Potential benefit to swine veterinarians/swine industry (40 points)
- Probability of success within timeline (35 points)
- Scientific/investigative quality (15 points)
- Budget justification (5 points)
- Originality (5 points)

For more information, or to submit a proposal:

AASV Foundation
830 26th Street
Perry, IA 50220-2328
Tel: 515-465-5255
Fax: 515-465-3832
Email: **aasv@aasv.org**

Thank you!

The AASV Foundation appreciates the support of the following companies who "chipped in" to sponsor the AASV Foundation Golf Outing. Their financial support, in addition to the contests, drawings, and giveaways they provided for the golfers, helped make the event profitable for the foundation as well as fun for the participants.

DINNER SPONSOR
Boehringer Ingelheim Animal Health

LUNCH SPONSOR
APC

BEVERAGE SPONSOR
Zoetis

GOLF HOLE SPONSORS
Aurora Pharmaceutical
Cambridge Technologies
Ceva Animal Health
Elanco Animal Health
GlobalVetLINK
Hog Slat
Huvepharma
Insight Wealth Group
Merck Animal Health
National Pork Producers Council
Pharmgate Animal Health
Topigs Norsvin

AASV Foundation Mission Statement

The mission of the AASV Foundation is to empower swine veterinarians to achieve a higher level of personal and professional effectiveness by:

- enhancing the image of the swine veterinary profession
- supporting the development and scholarship of students and veterinarians interested in the swine industry
- addressing long-range issues of the profession
- supporting faculty and promoting excellence in the teaching of swine health and production
- funding research with direct application to the profession

AASV semicentennial anniversary celebration: Our golden jubilee

Orlando, what a great place to be visiting and relaxing with friends, colleagues, and family at the end of a hard winter. Undoubtably this is the family vacation center of the world. As you know, this meeting marks a great milestone in the life history of AASV (AASP). We achieved 50 years and it is time to celebrate, but also a time for reflection and commitment. Please give back to AASV at our foundation auction, thus assuring a legacy for those yet to come.

The Foundation Committee is calling this our semicentennial for a great reason. Fifty years of AASV stardom will soon be past and it is nearly certain another 50 years will proceed. In the United Kingdom and other Commonwealth countries, a golden jubilee celebration is held in the 50th year of a monarch's reign. While there have been no monarchs in our realm, our dedicated leaders and membership have built a profession to last. There has been much progress over the past 50 years through dedicated leadership producing a world class organization based on cumulative experience, shared scientific progress, and new technological breakthroughs. We are here to stay and remain relevant to veterinary medicine and the swine industry.

From another perspective, a golden wedding anniversary is a celebration of 50 years of life spent together and great things accomplished with love and commitment. Typically, a 50th anniversary is considered a great honor for that couple's endurance, love, and devotion. Golden wedding anniversaries should be marked with memorable gifts and celebrations. Likewise, we have given our hearts and commitments to AASV and our clients, thus it is time to celebrate our 50th year and contribute memorable gifts supporting the lasting existence of AASV through the Foundation.

So, "Let's Go for the Gold!"

If you have questions or just want to discuss possibilities, please contact any of the committee members. Download the donation form at **www.aasv.org/foundation/2019/Donationform.pdf** and submit a description and image of your item(s) by December 1. Your contribution will be recognized in the printed auction catalog as well as on the auction website, and your name will appear in the JSHAP full-page spread recognizing all our auction item donors. If that's not enough, there's a good chance Dr Harry Snelson will say something witty about your donation in the AASV e-Letter, too!

The AASV Foundation is committed to ensuring the future of the swine veterinary profession. Proceeds from the auction enable funding for AASV Foundation programs, including:

- Administering endowments for the Howard Dunne and Alex Hogg Memorial Lectures
- Administering the Hogg Scholarship for a swine veterinarian pursuing an MS or PhD
- Administering funding for veterinary student scholarships
- Scholarships for veterinarians pursuing board certification in the American College of Animal Welfare
- Co-sponsoring travel stipends for veterinary students attending the AASV Annual Meeting
- Providing swine externship grants to veterinary students
- Funding swine research with direct application to the profession
- Providing support for Heritage Videos
- Providing tuition support for out-of-state veterinary students to attend the Swine Medicine Education Center

2019 Auction Committee

Butch Baker (Chair)
Natalie Baker
Laura Bruner
Dyneah Classen
Joe Connor
Jack Creel
Chris Deegan
Jer Geiger
Bill Hollis
Derald Holtkamp
Daryl Olsen
Sarah Probst Miller
Nathan Schaefer
Cameron Schmitt
Chase Stahl
Jon Van Blarcom
John Waddell

Swine veterinarians invited to apply for Hogg Scholarship

The American Association of Swine Veterinarians Foundation is pleased to offer the Hogg Scholarship, established to honor the memory of longtime AASV member and swine industry leader Dr Alex Hogg. Applications for the $10,000 scholarship will be accepted until February 1, 2019, and the scholarship recipient will be announced on Sunday, March 10 during the Foundation Luncheon at the AASV 2019 Annual Meeting in Orlando.

The intent of the scholarship is to assist a swine veterinarian in his or her efforts to return to school for graduate education resulting in a master's degree or higher in an academic field of study related to swine health and production.

Dr Alex Hogg's career serves as the ideal model for successful applicants. After twenty years in mixed animal practice, Dr Hogg pursued a master's degree in veterinary pathology. He subsequently became Nebraska's swine extension veterinarian and professor at the University of Nebraska. Upon "retirement," Dr Hogg capped off his career with his work for MVP Laboratories. Always an enthusiastic learner, at age 75 he graduated from the Executive Veterinary Program offered at the University of Illinois.

The scholarship application requirements are outlined below, and on the AASV website at **www.aasv.org/foundation/hoggscholarship.htm**.

Hogg Scholarship Application Requirements

An applicant for the Hogg Scholarship shall have:

1. Three or more years of experience as a swine veterinarian, either in a private practice or in an integrated production setting
2. Five or more years of continuous membership in the American Association of Swine Veterinarians

Applicants are required to submit the following for consideration as a Hogg Scholar:

1. Current curriculum vitae
2. Letter of intent detailing his or her plans for graduate education and future plans for participation and employment within the swine industry
3. Two letters of reference from AASV members attesting to the applicant's qualifications to be a Hogg Scholar

Applications and requests for information may be addressed to:

AASV Foundation
830 26th Street
Perry, IA 50220
Tel: 515-465-5255
Email: **aasv@aasv.org**

Veterinary students: Apply for $5000 scholarships by December 31

The AASV Foundation and Merck Animal Health are pleased to announce the continuation of the AASVF-Merck Animal Health Veterinary Student Scholarship Program. Ten $5000 scholarships will be awarded to sophomore and junior veterinary students in 2019. Now in its fourth year, the program seeks to identify future swine veterinarians and assist with their educational expenses. Applications are due December 31, 2018 for scholarships that will be announced at the 2019 AASV Annual Meeting.

Second- and third-year veterinary students enrolled in AVMA-accredited or -recognized colleges of veterinary medicine in Canada, the Caribbean Islands, Mexico, South America, or the United States are eligible to apply. All applicants must be current (2018-2019) student members of AASV. Students who have previously been awarded one of the scholarships are not eligible to reapply.

To apply, students submit a resume and the name of a faculty member or AASV member to serve as a reference, along with written answers to four essay questions. The application and instructions are available at **www.aasv.org/foundation/2019/AASVF-MerckScholarships.php**.

A committee of four conducts the selection process. Two AASV Foundation board members and two AASV members-at-large rank the applicants by scoring their past and current activities, level of interest in swine veterinary medicine, future career plans, and financial need. The scholarship recipients will be announced during the 2019 AASV Annual Meeting in Orlando, and the scholarship funds will be disbursed after the conference.

The AASVF-Merck Animal Health Veterinary Student Scholarship Program is part of how Merck Animal Health and the AASV Foundation fulfill a shared mission of supporting the development and scholarship of students and veterinarians. For more information on scholarships and other AASV Foundation programs, see **www.aasv.org/foundation**.

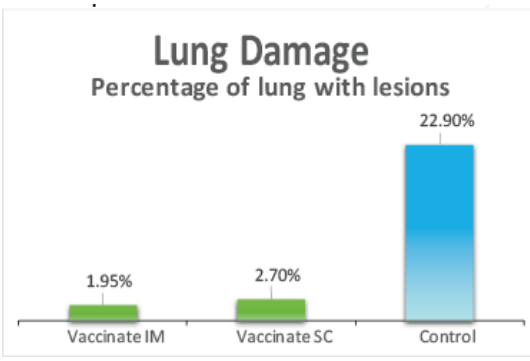
EMULSIBAC®-APP

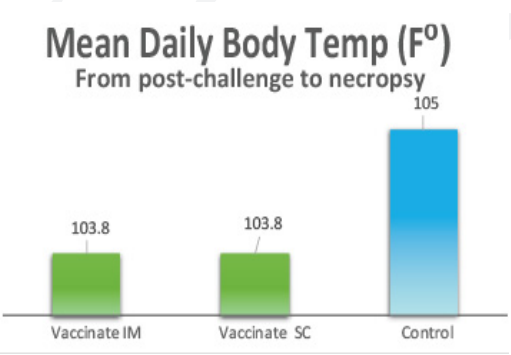
Full Immunogenic Expression To *Actinobacillus pleuropneumoniae*

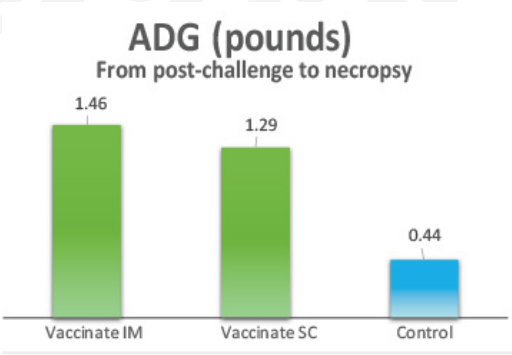
- Phibro Animal Health's ***EMULSIBAC***-APP is for the vaccination of healthy swine over four weeks of age against pneumonia caused by *Actinobacillus pleuropneumoniae*.
- ***EMULSIBAC***-APP has been shown to protect susceptible swine against intranasal challenge with virulent serotypes 1, 5, and 7 of *Actinobacillus pleuropneumoniae.*
- ***EMULSIBAC***-APP contains ***Emulsigen***®
  - Unique emulsified oil-in-water adjuvant
  - An oil-based adjuvant approved by USDA for either intramuscular or subcutaneous injection of swine
  - Reduces the risk of site abscesses

EMULSIBAC-APP Protects Against the Profit-Robbing Effects of Pneumonia Caused by APP

Post-challenge test results show that ***EMULSIBAC***-APP effectively protects pigs against a severe challenge dose, while non-vaccinated pigs developed significant pleuropneumonia, elevated body temperature, and reduced weight







To learn more, visit ***www.PAHC.com***

Tailor-Made® Vaccines / AB20® / Mecadox®

Phibro ANIMAL HEALTH CORPORATION™

4805 G Street
Omaha, NE 68117-1414
Email: pvolabs@pahc.com
Website: pahc.com
Telephone: 1.800.856.4648
Fax: 402.331.8776

HEALTHY ANIMALS. HEALTHY FOOD. HEALTHY WORLD.®

Thank you, reviewers

Working together and creating a journal to be proud of!

The editorial staff of the Journal of Swine Health and Production *would like to acknowledge the invaluable assistance of the following individuals for their service as referees for the manuscripts that were reviewed between September 23, 2017, and September 22, 2018.*

Glen Almond
David Baum
Lisa Becton
Renée Bergeron
Pat Blackall
Susan Brockmeier
Mike Brumm
Eric Bush
Robert Charette
Chris Chase
Jane Christopher-Hennings
Nigel Cook
Cesar Corzo
Osmar Dalla Costa
Peter Davies
Scott Dee
Pierpaolo Di Giminiani
Steve Dritz
Mike Eisenmenger
Gene Erickson
Mark Estienne
Robert Friendship
Phil Gauger
Connie Gebhart
Jerome Geiger
Laura Greiner
Carlton Gyles
John Harding
Ben Hause
Peggy Anne Hawkins
Trish Holyoake
Marlin Hoogland
David Hurley
Anna Johnson
Rob Knox
Jim Kober
Yihang Li
Daniel Linhares
Drew Magstadt
Annelies Michiels
Adam Moeser
Joaquim Morales
Carissa Odland
Fernando Osorio
Tom Parsons
Tim Pasma
Andres Perez
Maria Pieters
Roman Pogranichniy
Zvonimir Poljak
Dale Polson
Chris Rademacher
Mrijendra Rajput
Alex Ramirez
Luienne Rocha
Max Rodibaugh
Leigh Rosengren
Marcus Rostagno
Joe Rubin
Cameron Schmitt
Frederick Schmoll
Kent Schwartz
Yolande Seddon
Erin Strait
Bob Thaler
Mike Tokach
Montse Torremorell
Megan Verdon
Per Wallgren
Charlotte Winder
Kyoung-Jin Yoon
Beth Young
Jianqiang Zhang
Jeff Zimmerman

We apologize if we have inadvertently left a reviewer's name off the list.

BUILT TO LAST

Celebrating 50 Years of Progress

AASV Annual Meeting

March 9-12, 2019

Lake Buena Vista, Florida

REGISTRATION OPENS IN LATE DECEMBER

Reserve lodging:

Hilton Orlando Buena Vista Palace

1900 E Buena Vista Drive • Lake Buena Vista, Florida • 407-827-2727

www.aasv.org/annmtg

2019 ANNUAL MEETING PROGRAM

AASV's 50th Annual Meeting

March 9-12, 2019
Orlando, Florida

BUILT TO LAST: Celebrating 50 Years of Progress

SATURDAY, MARCH 9

8:00 AM
Entrance examination: American Board of Veterinary Practitioners, Swine Health Management

Pre-conference seminars

1:00 PM – 5:00 PM (except Seminar #5, 12:00 PM - 5:15 PM)

Seminar #1	AASV's Got Talent *Jeff Harker, chair*
Seminar #2	Swine Welfare and Behavior *James Kober, chair*
Seminar #3	Emerging Technologies for the Swine Industry *Chris Rademacher and Dale Polson, co-chairs*
Seminar #4	Conducting Effective Outbreak Investigations: Learning from Our Mistakes *Derald Holtkamp, chair*
Seminar #5	Operation Main Street Training *Al Eidson, chair*
Seminar #6	Leading People: Leadership Styles Training for Developing More Effective and Productive Working Relationships *Emily Byers, chair*

SUNDAY, MARCH 10

Canadian Swine Veterinarians

8:00 AM – 12:00 PM

Pre-conference seminars

8:00 AM – 12:00 PM

Seminar #7	Boar Stud Topics *Darwin Reicks, chair*
Seminar #8	Swine Nutrition: Setting the Foundation *Dwain Guggenbiller, chair*
Seminar #9	Diagnostics *Fabio Vannucci, chair*
Seminar #10	Swine Medicine for Students *Jeremy Pittman and Angela Supple, co-chairs*

Research Topics

8:00 AM – 12:00 PM
Session chair: Chris Rademacher

8:00 AM	Comparison of different cell lines for improving porcine reproductive and respiratory syndrome virus isolation from clinical samples *Wannarat Yim-Im*
8:15 AM	Investigating biosecurity aspects related to porcine reproductive and respiratory syndrome virus outbreaks *Gustavo de Sousa e Silva*
8:30 AM	Comparison of production impact following porcine reproductive and respiratory syndrome clinical outbreaks in breeding herds adopting killed or attenuated porcine reproductive and respiratory syndrome virus vaccination protocols *Gaurav Rawal*
8:45 AM	Economic and production benefit of 2 porcine reproductive and respiratory syndrome modified live virus doses compared to a single dose vaccination program on nursery pigs *Cesar Moura*
9:00 am	Modeling the dilution effect of porcine reproductive and respiratory syndrome virus RNA in processing fluid field samples on the probability of virus detection by quantitative polymerase chain reaction *Will Lopez*

9:15 AM	Put a CLAMP on it! Polymerase chain reaction-based strategy to selectively sequence wild-type porcine reproductive and respiratory syndrome virus in vaccinated herds *Karen Harmon*
9:30 AM	Use of oropharyngeal swabs and udder wipes to monitor porcine reproductive and respiratory syndrome virus in breeding herds *Jorge Garrido Mantilla*
9:45 AM	Experimental transmission of influenza A virus and porcine reproductive and respiratory syndrome virus from nurse sows to adopted pigs during lactation *Jorge Garrido Mantilla*
10:00 AM	REFRESHMENT BREAK
10:15 AM	Shedding of a newly commercialized live attenuated influenza vaccine under field conditions *Gustavo Lopez*
10:30 AM	Identification of metabolite markers for enzootic pneumonia in pigs *Maria Pieters*
10:45 AM	*Mycoplasma hyosynoviae* diagnostics: What experimental data can tell us about field strains *Nubia Macedo*
11:00 AM	Oral infectious dose of African swine fever virus when consumed naturally in feed and liquid *Megan Niederwerder*
11:15 AM	Efficacy of an inactivated Seneca Valley virus vaccine in nursery-aged pigs *Alexandra Buckley*
11:30 AM	Transmammary delivery of firocoxib from medicated sows to nursing piglets reduces stress and improves average daily gain after castration, tail docking, and teeth clipping *Hans Coetzee*
11:45 AM	Potential risk factors for pelvic organ prolapses: Survey of 104 US commercial breeding herds *Chris Rademacher*
12:00 PM	Session concludes

Poster session: Veterinary Students, Research Topics, and Industrial Partners

12:00 PM – 5:00 PM

Poster authors present from 12:00 PM to 1:00 PM
Poster display continues on Monday, 9:00 AM to 5:00 PM

Concurrent sessions

1:00 PM– 5:15 PM

Session #1	**Student Seminar** *Andrew Bowman and Maria Pieters, co-chairs*
Session #2	**Industrial Partners** *George Charbonneau and Jessica Seate, co-chairs*
Session #3	**Industrial Partners** *Peggy Anne Hawkins and Brent Pepin, co-chairs*
Session #4	**Industrial Partners** *Melissa Hensch and Amy Maschhoff, co-chairs*

MONDAY, MARCH 11

General Session
Built to Last: Celebrating 50 Years of Progress

8:00 AM – 12:15 PM
Program and Session Chair: Nathan Winkelman

8:00 AM	**Howard Dunne Memorial Lecture** Built to last: 50 years of AASV *John Waddell*
9:00 AM	**Alex Hogg Memorial Lecture** Today's swine veterinarian: Challenges and opportunities for the future *Deborah Murray*
10:00 AM	REFRESHMENT BREAK
10:30 AM	AASV Golden Anniversary Video
10:45 AM	US pork production: Your Pork Checkoff dollars at work *Bill Even*
11:15 AM	International pork markets and the influence of global megatrends *Brett Stuart*
12:15 PM	LUNCHEON

Concurrent Session #1: Disease Control and Elimination

2:00 PM– 5:30 PM
Session chair: Bill Hollis

Part 1: Porcine Reproductive and Respiratory Syndrome

2:00 PM	Elimination failures and live virus inoculation experiences *Jay Miller*
2:15 PM	Economic models of porcine reproductive and respiratory syndrome control *Dale Polson*
2:30 PM	Herd closure time analysis *Daniel Linhares*
2:45 PM	Porcine reproductive and respiratory syndrome monitoring methods *Will Lopez*
3:00 PM	Porcine reproductive and respiratory syndrome control and elimination: Question and answer roundtable *Miller, Polson, Linhares, and Lopez*
3:15 PM	REFRESHMENT BREAK

Part 2: *Mycoplasma*

3:45 PM	Diagnostic methods for effective *Mycoplasma hyopneumoniae* control and elimination *Maria Pieters*
4:00 PM	*Mycoplasma* isolation and elimination experiences *Bob Thompson and Maria Jose Clavijo*
4:15 PM	What if a naïve herd breaks? *Dyneah Classen*
4:30 PM	*Mycoplasma* control and elimination: Question and answer roundtable *Pieters, Thompson, Clavijo, and Classen*

Part 3: Porcine Epidemic Diarrhea Virus

4:45 PM	Porcine epidemic diarrhea virus: People during breaks *Mary Battrell*
5:00 PM	Porcine epidemic diarrhea virus elimination and persistence *Laura Batista*
5:15 PM	Porcine epidemic diarrhea virus control and elimination: Question and answer roundtable *Battrell and Batista*
5:30 PM	Session concludes

Concurrent Session #2: Practical Vaccinology and Immunology for the Swine Veterinarian

2:00 PM– 5:30 PM
Session co-chairs: Marie Culhane and Emily Byers

Part 1: General Immunology, Gastrointestinal Immunology, and Vaccinology

2:00 PM	General immunology: Concepts and key points for swine veterinarians *Brian Aldridge*
2:25 PM	Gut immunology *Adam Moeser*
2:50 PM	Impact of vaccination on transmission of *Lawsonia intracellularis* *Fabio Vannucci*
3:10 PM	Farm application of wean-to-finish vaccines for enteric diseases *Nate Winkelman*
3:25 PM	REFRESHMENT BREAK

Part 2: Respiratory Immunology and Vaccinology with a Flu Focus

3:55 PM	Respiratory immunology *Amy Vincent*
4:20 PM	Universal influenza vaccines *Daniela Rajao*
4:45 PM	Herd immunity and transmission *Montse Torremorell*
5:00 PM	Influenza vaccinations: Live versus killed *Chong Li*
5:15 PM	Vaccinations in antibiotic-free farms *Marlin Hoogland*
5:30 PM	Session concludes

Concurrent Session #3: Production Innovations

2:00 PM– 5:30 PM
Chair: Deborah Murray

2:00 PM	Sow mortality: The Danish perspective *Michael Agerley*
2:15 PM	Low sow mortality: What's the secret? *Ron Ketchem*
2:30 PM	Production drivers that drive mortality *Pedro Mosqueira*
2:55 PM	Training, motivation, and a culture of safety *Tia Landry*
3:10 PM	Production Innovations: Question and answer roundtable *Agerley, Ketchem, Mosqueira, and Landry*
3:30 PM	REFRESHMENT BREAK
4:00 PM	Comparative lameness: What are we learning in other species? *Jan Shearer*
4:35 PM	Structural and pathological changes in growing pig lameness compared to sow lameness *Stephanie Rossow*
4:55 PM	Antibiotic use measurement: Where are we at? *Peter Davies*
5:10 PM	The politics of antibiotic use in food-producing animals *Liz Wagstrom*
5:30 PM	Session concludes

TUESDAY, MARCH 12

General Session: Transboundary Disease Threats and Outbreak Preparedness

8:00 AM – 12:00 PM
Session chair: Nathan Winkelman

8:00 AM	African swine fever: A global threat *Klaus Depner*
8:45 AM	A China perspective: Diseases, diagnostics, and biosecurity *Keith Erlandson*
9:10 AM	The foreign animal disease risk of feed *Scott Dee*
9:40 AM	Industry and AASV response to foreign animal disease risk *Paul Sundberg*
10:00 AM	REFRESHMENT BREAK
10:30 AM	Fifty-plus years of US swine disease eradication *Joe Connor*
11:00 AM	What's next for disease elimination: *Mycoplasma hyopneumoniae*, porcine reproductive and respiratory syndrome, porcine epidemic diarrhea, or ileitis? *Paul Yeske*
11:30 AM	When "IT" hits the fan: Will we be prepared? *Patrick Webb*
12:00 PM	Session and meeting conclude

African swine fever: What is the government's role?

I wanted to provide an update on AASV activities regarding African swine fever (ASF). New outbreaks of ASF continue to be reported in China. In addition, Bulgaria and Belgium have reported their first outbreaks of the disease. The United States swine industry continues to monitor the situation closely and is focusing on efforts to prevent the introduction of the virus into the US herd.

Dr Tom Burkgren and I, along with representatives from the National Pork Board, the National Pork Producers Council, and the Swine Health Information Center, met with United States Department of Agriculture (USDA) and Food and Drug Administration (FDA) officials on September 5, 2018 to discuss ASF prevention concerns. Although the meeting focused mainly on prevention, discussion topics also included diagnostic testing, surveillance, feed and feed ingredient issues, garbage feeding controls, and monitoring the importation of pork casings. In summary, the prevention issues discussed were:

Communication

- The USDA is scheduling biweekly calls for updates and discussion of ASF-related issues.
- The USDA has set up a website dedicated to ASF at **www.aphis.usda.gov/animalhealth/animal-disease-information/swine-health** and will update the website as the situation evolves.

Waste feeding and the Swine Health Protection Act

- The USDA's Animal and Plant Health Inspection Service (APHIS) has had controls in place for decades on international garbage, including food waste from ships, airlines and international conveyances. These controls require all international garbage to be disposed of appropriately and under APHIS supervision, for example, garbage transported under seal to approved incineration facilities.
- The authority to ban the feeding of plate waste containing meat is under the regulatory oversight of individual states, not with USDA.
- The USDA will assess what needs to be done, if anything, to improve inspection of licensed waste-feeding facilities and enforcement on unlicensed facilities.

Importing meat products

- Import restrictions imposed by APHIS prohibit the entry of untreated animal products, including meat and meat products, from countries or regions considered affected with certain diseases. Fresh and frozen pork is prohibited from regions affected with ASF, classical swine fever, foot-and-mouth disease, or swine vesicular disease, while meat that has been cooked is allowed under APHIS regulations.
- The European Union is transparent in their review and zoning of the current ASF situation.
- Members of APHIS are in regular contact with the European Union about current zoning status.
- Safe trade in meat and meat products around the world is built on the understanding that government veterinary authorities in the product country of origin inspect and certify those products in accordance with the requirements of the country of destination. Imposing additional requirements, such as testing products for viruses after arrival in the country of destination, destroys the credibility of the certification system and leads to consequences of reciprocal testing of US exports.

"You should ensure that tonsil is included with all laboratory submissions."

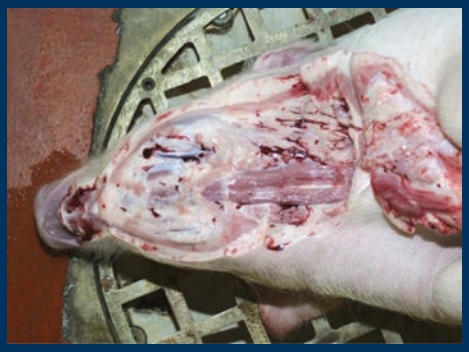
Swine casings

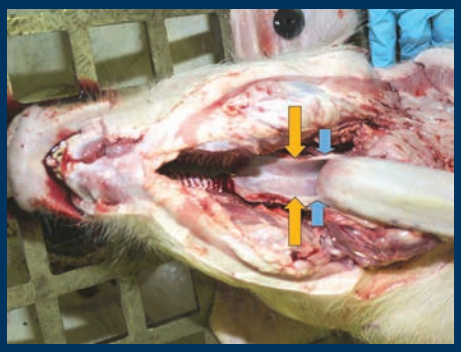
- Swine casings that originate from ASF-positive countries or regions are prohibited entry into the United States under APHIS regulations.
- Entry of Chinese-origin swine casings was denied by APHIS when ASF was found in China.
- Current APHIS regulations allow US-origin swine casings to be processed in ASF-affected countries or regions under certain conditions. With strong support of the casings industry, APHIS is working to review the processing of US-origin swine casings in Chinese facilities.
- Casings are shipped with a 6-week transit time to and from facilities in saturated brine solutions that will inactivate foreign animal disease (FAD) viruses as referenced in the World Organization for Animal Health's guidelines.

Testing imported feed and feed ingredients

- Both USDA and FDA identified many concerns and potential consequences that could arise from testing imported feed and feed ingredients.
- Both USDA and FDA believe there are currently many unknowns and data gaps that should be identified to help define or validate feed risk. In the absence of information regarding the predictive ability of unvalidated test results to accurately determine the potential risk associated with feed, the design and implementation of a testing strategy is not feasible.

Advocacy in action continued on page 343





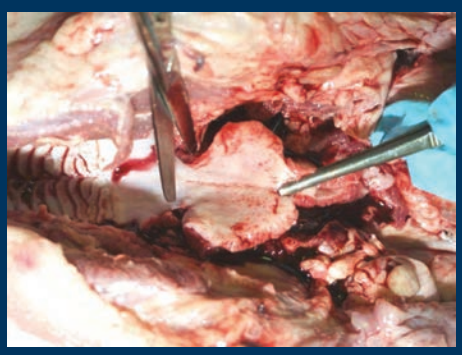
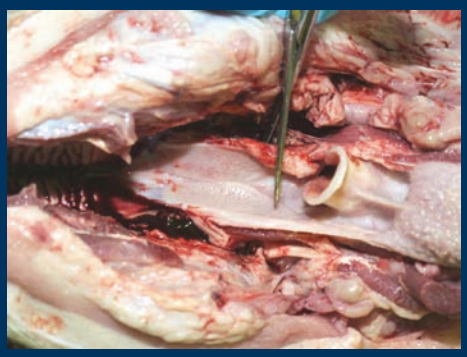
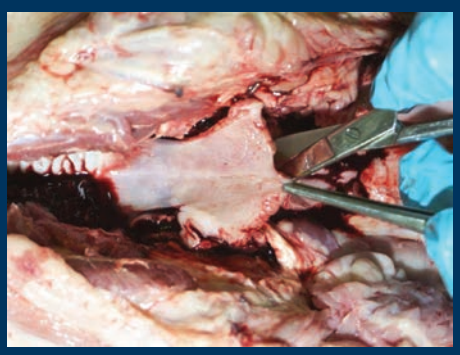
GOT TONSIL?

Include tonsil with all laboratory submissions!

Tonsil tissue is an important tool in diagnosing a number of endemic swine diseases as well as classical swine fever and African swine fever.

www.aasv.org/aasv/documents/GotTonsil.pdf

Download the brochure for complete details on proper technique for sample collection and submission.

Advocacy in action continued from page 341

- There are significant logistics issues, including whether a validated test and a validated sampling method exist, who does the sampling, who pays for the testing, who pays for storage or demurrage charges while product is held, that would present challenges.
- Testing could bring potential consequences that must be considered such as false interpretation of results, the different industries that could be impacted by testing as some ingredients are shared between the human and animal food streams, and the impact on US exports if additional testing requirements are imposed.
- Without further information and because of recognized data gaps, a government testing program is not feasible currently.
- Both USDA and FDA continue to work with industry representatives to assess the potential risk of non-animal origin feed ingredients to US agriculture and the feed supply.
- The National Animal Health Laboratory Network (NAHLN) will issue a guidance to the veterinary diagnostic labs advising them to not do unofficial FAD testing.

Other key discussion points

- The pork industry will need to work with the feed industry and other affiliated industries to develop programs to address feed safety. Both APHIS and FDA are willing to help with these discussions and processes.
- The FDA is willing to facilitate expedited regulatory review with any sponsor submitting a possible mitigation product that can be added to feed that may help address the animal health concerns associated with FAD transmission.
- The USDA has asked US Customs and Border Protection to target inspections of passengers and cargo coming from ASF-positive regions.
- The USDA is on heightened alert for illegal pork products in non-traditional markets.

In addition, we also discussed concerns with diagnostic capabilities including sample validation and lab capacity issues. Currently, the only sample type approved for use by the NAHLN labs is whole blood. Whole blood was chosen due to the virus' predilection for macrophages, but it is not a sample routinely collected by swine veterinarians. The USDA indicated that tonsil would be approved for the NAHLN labs by the end of September and oral fluids sometime in 2019. Following approval, tonsil will be validated for both classical swine fever and African swine fever at the diagnostic labs. **You should ensure that tonsil is included with all laboratory submissions.** The AASV developed a brochure on tonsil collection and submission entitled "Got Tonsil". The brochure can be ordered from the AASV office (**aasv@aasv.org**) or downloaded from the AASV website at **www.aasv.org/aasv/documents/GotTonsil.pdf**. We are also encouraging USDA to evaluate and validate lungs and spleen, as these are common diagnostic tissues submitted by swine veterinarians**.**

Diagnostic and private labs are considering requests to test feed and feed ingredients for ASF. Given there are no validated tests or sampling methodologies for feed and considering the severe consequences of a false-positive finding, USDA will not allow non-official ASF testing at the NAHLN laboratories.

To address potential sources of infection, the four industry organizations are encouraging producers and veterinarians to interact with their feed and feed-ingredient suppliers on issues associated with the importation of products which may pose a heightened risk of disease exposure. To facilitate this interaction, the group has published a list of questions for producers and veterinarians to pose to their suppliers. A link to this list can be found on the AASV website at **www.pork.org/news/pork-industry-focuses-feed-ingredients-combat-african-swine-fever-threat/**.

The group also compiled a list of frequently asked questions regarding the role of USDA and FDA in ASF prevention. That list is posted on the AASV website at **www.aasv.org/documents/ASFFAQ91018.pdf**. We will continue to keep our members informed as these interactions proceed. Please feel free contact me with any questions.

Harry Snelson, DVM
Director of Communications

Cumulative index

The *Journal of Swine Health and Production* cumulative index is updated online throughout the year as issues go to press. Articles can be accessed via the "Search" function and from the Abstracts page, **www.aasv.org/shap/abstracts/**.

Index by title 2018

Index by author 2018

Porcilis™ ILEITIS

A SOLUTION THAT FITS.

Ileitis prevention doesn't have to restrict your other treatment needs. With the flexibility of Porcilis™ Ileitis, one dose gives you an industry-leading 20 weeks of protection* without impacting other treatment efforts on your operation.

DRIVEN BY PREVENTION™
DrivenByPrevention.com

20 WEEKS DOI PORCILIS™ ILEITIS

7 WEEKS DOI COMPETITOR'S VACCINE*

*Frozen formulation; non-frozen formulation label does not include DOI.

1/17 SW-PI-55755-2

MERCK Animal Health

Upcoming meetings

2018 ISU James D. McKean Swine Disease Conference

November 1-2, 2018 (Thu-Fri)
Scheman Building, Iowa State University, Ames, Iowa

For more information:
Registration Services
Iowa State University
1601 Golden Aspen Drive #110, Ames, Iowa 50010
Tel: 515-294-6222; Fax: 515-294-6223
Email: registrations@iastate.edu
Web: register.extension.iastate.edu/swinedisease

For questions about program content:
Dr Chris Rademacher, Conference Chair
Iowa State University
Email: cjrdvm@iastate.edu

Humane Endings Symposium

November 2-4, 2018 (Fri-Sun)
Westin O'Hare, Rosemont, Illinois
Hosted by American Veterinary Medical Association

For more information:
Email: humaneendings@avma.org

2018 North American PRRS Symposium

December 1-2, 2018 (Sat-Sun)
Chicago Marriott, Downtown Magnificent Mile

For more information:
Dr Bob Rowland, Executive Director
Email: naprrs@vet.k-state.edu
Web: www.vet.k-state.edu/na-prrs/

Passion for Pigs Seminar and Trade Show

December 4, 2018 (Tue)
Columbia, Missouri

For more information:
Julie A. Lolli
Tel: 660-651-0570
Email: julie@passionforpigs.com
Web: www.passionforpigs.com

2019 Pig-Group Ski Seminar

February 13-15, 2019 (Wed-Fri)
Copper Mountain, Colorado

For more information:
Pig Group, Lori Yeske
39109 375th Ave, Saint Peter, MN 56082
Tel: 507-381-1647
Email: pyeske@swinevetcenter.com
Web: www.pigski.com

American Association of Swine Veterinarians 50th Annual Meeting

March 9-12, 2019 (Sat-Tue)
Hilton Orlando Buena Vista Palace
Lake Buena Vista, Florida

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

Asian Pig Veterinary Society Congress 2019

August 26-28, 2019 (Mon-Wed)
BEXCO, Busan 55, APEC-ro, Haeundae-gu, Busan
Republic of Korea
Tel: +82 51-740-7300

For more information:
Amy Chang (Secretariat of APVS 2019):
802, InnoN, 66, Seongsui-ro, Seongdong-gu, Seoul
Republic of Korea
Tel: +82 2-2190-7331
Email: moon@innon.co.kr

Sue Jo (Secretariat of APVS 2019):
Tel: +82 2-2190-7327
Email: sue@innon.co.kr
Web: www.apvs2019.com

Pig Welfare Symposium

November 13-15, 2019 (Wed-Fri)
Hosted by the National Pork Board

For more information:
Web: www.pork.org/pws

26th International Pig Veterinary Society Congress

June 2-5, 2020 (Tue-Fri)
Florianopolis, Brazil

For more information:
Tel: +55 31 3360 3663
Email: ipvs2020@ipvs2020.com
Web: www.ipvs2020.com

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

AASV Industry Support Council

The JSHAP is made possible by the generous support of the following Industry Support Council members:

Boehringer Ingelheim | Elanco | MERCK Animal Health

Newport Laboratories | Norbrook | PHARMACOSMOS Committed to Quality

Pharmgate Animal Health | Phibro Animal Health Corporation | provimi

Tonisity | Virox Animal Health | zoetis

Photo Corner

Thirsty pigs at University of Missouri Swine Teaching Center.

Photo courtesy of Barbara Molnár Smith