

Featured Speaker

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Commentary

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SARAH:

Hi, this is Dr. Sarah Probst Miller. I want to welcome you to our next edition of *P's in a Pod*. Today, I have with me Dr. Joe Connor and Dr. Jim Lowe and we're going to be talking about *Early Vaccination Strategies for Mycoplasma*.

You know, mycoplasma is one of the simplest organisms that can cause disease. It's really a pretty, elegant, little creature. Jim, at AASV you talked about how it isn't always a pathogen, but it is really an organism that is often present that can cause disease when it exceeds the resistance of the pig. Can you talk a little bit about how as an industry we sometimes misaddress this?

JIM:

Yeah, Sarah, good to be here again and I think the key take home on all of this stuff is we get focused on *Mycoplasma hyopneumoniae* and we sometimes forget that it really causes disease and it's the key player in disease we call Enzootic Pneumonia in growing pigs typically, and that's that chronic, dry, hacking cough sometime after weaning. We historically would have thought about that as a 14-16 week of age pig disease, but it can certainly be younger or older.

And so, we've spent a lot of time the last probably 5-10 years thinking about how do we deal with mycoplasma, instead of maybe how do we deal with disease itself? And so, we've done some crazy things like eradication strategies and maybe some areas were acceptable and maybe lost some ground. And I think a lot of that, Sarah, came back to our ability to do better diagnostics.

We've certainly got "better" diagnostics today. We certainly have more diagnostics today. So we have several new, over the last five years, antibody tests looking for the pig's response. And we have a plethora now of PCR tests to look for the bug. And the challenge with all of these is, is that in the scientific lingo they're highly specific. I.E. If it's positive, it's a true positive, but they're lowly sensitive. So a negative doesn't always mean it's negative. So positives we've got a lot of confidence in. Negatives we don't have a lot of confidence in, and that's really true with the PCR test looking for the antigen, but that's even true with some of the antibody tests and some of the antibody tests aren't very specific either, so we get some false positives there. So really confusing diagnostically for us to understand what's going on in herds. We've gotten ahead; I think we made some assumptions and probably made backwards progress in the last five years instead of forward progress on really controlling mycoplasma.

SARAH:

Jim, I want to come back to that topic in a little bit. The impact of getting a lot of positive results of mycoplasma in a herd over time and trying to analyze that to figure out what is causing disease and what isn't? What does a positive result mean in a herd? But before we go there, I want to talk a little bit about or review a little bit about, what mycoplasma does when it causes disease? I had the unfortunate experience of being diagnosed with Walking Pneumonia shortly after my daughter, Stella,

was born. And it is an interesting disease, because you feel ok, but you just don't feel up to par. You have a low-grade fever, but your lungs clearly aren't functioning at the appropriate level; and so, I can empathize with the pigs a little bit more. You know, I just didn't feel normal. Clearly, our pigs are compromised when they have this disease as well. What happens in the lungs if mycoplasma becomes a disease?

JIM:

Yeah. I want to make sure for everybody that you *did* have mycoplasma. So, that's a great lesson... that mycoplasma species, there's lots of those, in fact a lot of different mycoplasma species of critters. So dogs get mycoplasma, and people get it, and it is the common cause of Walking Pneumonia in people. Cattle deal with Mycoplasma Pneumonia, so it's also a big deal in feed yards . . .and certainly pigs do. Now, each one of those is a separate species, so you don't have to worry about getting *Mycoplasma pneumoniae* from your pig. It does not transmit from pig to human, but when humans get their mycoplasma strain, does create a similar scenario; and so why you felt really kind of yucky, but not terrible, is because that bug, mycoplasma, lives in the airway, Sarah, not in the lung. And so, if we think about other things like *Pastuerella* or Atypical Bacterial Pneumonia, it actually lives in the airway and causes a lot of tissue damage in the lung. Mycoplasma doesn't do that, it kind of lives on the surface, it stops the cilia, the little fingers, that push the other bacteria out of the lung and it causes some swelling, but is not terrible inflammation in the lung. So you feel bad, that's why you've got this dry cough, but it's not productive. There's no, you know, big, old goober to hack out of there.

SARAH:

A gentle way to put it!

JIM:

Yes! But it's an inflammation. That's also why our diagnostic tests are tough. It's really an outside pathogen. So if you think the airways and the lungs are kind of like the skin being inverted, right? So it's like we poked the skin in, right? That's the contact with the outside world. The contacts are there all the time. And so that being outside the lung, inside the airway, or outside the tissue of the lung also means that it's really outside the body. We don't get a normal antibody response. It's hard to get positives, because it may not be shed because it's deep in the lung. And so the very nature of the bug, where that bug lives, and really how it causes inflammation is part of the reason why this thing is so complicated; and yet, is so subtle. So in a lot of herds you may have low-grade infection that may or may not compromise pigs much. That's why I've talked a lot about, "Do we have Enzootic Pneumonia or do we have mycoplasma positive?"

SARAH:

Right. When I had Atypical Pneumonia or Walking Pneumonia, they did an x-ray and could see that my lungs were affected. In pigs, it's my understanding the mycoplasma is affecting the cilia of the lungs, the motion of the cilia, causing the lungs to fill up with fluid for the most part.

And let's talk a little bit now about how we discern what is a mycoplasma positive pig look like compared to a group of pigs that have Enzootic Pneumonia?

JIM:

You just asked the million dollar question, right? I mean, that's really the holy grail of where we are at and so if we think about how do we determine if we have economically significant Enzootic Pneumonia versus just a mycoplasma infection, we really have to look at some things.

And so we've talked about really complicated things actually, because we've talked about what we really like to know is the impact of variation . . . if the bell curve of growth is shifted? So, can we look at the distribution of growth rates, because pigs that have chronic Enzootic Pneumonia tend to grow slower and Enzootic Pneumonia tends not to affect the entire population? And so, if we're really sophisticated, if we knew the entry weights of the pigs, and knew all of our slaughter weights, we could go back and create a bell curve or histogram and say, "Ah, is that histogram normal? And how much variation, (which we measure by CV or coefficient of variation), why is that?"

Now practically, we may not have that data in a lot of herds, right? And it's a challenge to get our hands on that. So we are really kind of back to somewhat simple things. We don't take pig x-rays. Although, one might think that is a valuable tool, but we are really back to things like...and even compared to the cattle business where they can go and take a stethoscope and listen to lungs and say, "Ah, that's not right." So we are back to things, like cough scores, so how many pigs are coughing? How much of that dry hack is there? Is the growth rate where it is expected? ...And then, matching some of that back up to necropsies. So not just the antigen test or the test, "Hey, I sent tissues to the lab and I got a positive," but do I have gross lesions? Can I see that typical mycoplasma, that gray, tan lung? Do I have histological lesions to confirm that? I.E. Can my diagnostic technician find it on histo? And thirdly, do I have the agent there?

And then it still gets back to the art versus necessarily the science of practice saying, "How significant is it? Are we getting a lot these pigs spread out? How many pigs have I necropsied? When are those pigs being affected?" Because certainly, it is a chronic disease and so pigs are getting affected early. I.E. 6-10 weeks of age versus 20 weeks of age . . . probably a very, very significant difference in terms of the impact on that pig's economic performance just because of the drag on growth rate and feed conversion.

SARAH:

Joe, let's talk a little bit about the economics of this disease. Historically, what have you seen as the economic impact of this disease?

JOE:

Sarah and Jim, to me the real interesting thing is that we have thought for the last few years we've had quite good control of Enzootic Pneumonia or mycoplasma. But that clearly is changing, and so I think Sarah, the date that we would have used historically, that uncomplicated mycoplasma had very little effect on performance characteristics. We now know, because first off, we hardly ever have uncomplicated *Mycoplasma hyopneumoniae* today. But secondly, either because our population sizes have changed or the transmission rate from sow to pig has changed; we clearly are back to what I would consider the effect that we would of seen 15-20 years ago where it was a primary cause of pneumonia and we're back to the factors that Jim mentioned in terms of how do we evaluate today? We just now are getting to a point where we can look at the distribution of a market group. And what we are going to see and are seeing that the economic effect is much greater than what we would of anticipated prior.

SARAH:

You know, Joe, 20 years ago I was 15 years old. So, there's value in having your experience here on this Podcast!

JIM:

I think she just called us old, Joe! I think we just got called old!

SARAH:

You know, I wasn't as interested in mycoplasma pneumonia back then. Talk to me about how 20 years ago we dealt with mycoplasma and did folks realize return on investment? When did vaccine become a tool for us?

JOE:

Ah, Sarah, I use the term now, "mature veterinarian," rather than old!

SARAH:

I see, I see!

JOE:

And so it really helps me...well, we've had vaccine for a number of years, but prior to vaccine we would have used therapeutic antibiotics for intervention. But what we didn't understand and actually in those days was the time delay between infection and when clinical signs appeared. And conversely, the necessity to have therapeutic antibiotics or vaccines into pigs at least 30 days or longer prior to the clinical episodes that we were seeing out in the field.

And Sarah, I can remember the first case of mycoplasma negative populations that I serviced that became infected with mycoplasma and I thought those pigs would first of all, all die and secondly, that they would never recover from the ongoing chronic cough. And in actuality, some of those pigs within that population would have been infected for 60-90 days and we essentially just sold those populations out.

SARAH:

Yup, you know my personal experience with mycoplasmal pneumonia is that it took a long time to recover, but eventually the lungs got there with treatment. Jim, share with me a case of mycoplasma that you've experienced recently; and if you don't mind, I'd like you to focus on a case that involves, let's say a naïve group of animals, and the impact of mycoplasma disease-wise and economically for a naïve group.

JIM:

Yeah, so I think Sarah, I think that we have to take one step back to get the full picture of the case, but I'm with Joe, that really we've had much worse control of mycoplasma over the last, oh, two to three years. And what we did is, we've really gone back and looked and I'm going to kind of summarize a bunch of cases as one, because the pattern is the same and it's like this reoccurring theme.

We've worked really hard to clean up our gilt multiplication sources for health and so we don't get a lot of stuff to clean up from PRRS and probably didn't intentionally work very hard at eradicating mycoplasma, but disease strategies work very effectively on PRRS that haven't necessarily eradicated mycoplasma or eliminated mycoplasma from individual herds, but they've suppressed it to a rate that we've got very low rates of infection in our gilts at weaning and then coupled that with really the increase in size. The mobilization, as we've gotten systems bigger and tried to do health upgrades and really try to drive genetic progress, and there's obviously a big benefit to having multiplier farms to drive genetic improvement.

As we've done all that, we've started to batch gilts into gilt grow outs. And so, as we get individualized barns of pigs that we run all-in/all-out, which is a change . . . maybe in eight or ten years ago we ran a continuous flow and they put those gilts back in our multiplier farms and etc., etc. Over time, we pushed those multipliers to the verge of, and I don't want to call them negative, but maybe allegedly negative to quote Dr. Schwartz would be the appropriate view, but they are low, low prevalence farms, subsequently, the pigs and gilts coming out of them are of little or low prevalence. And we've been putting those into our commercial sow farms for two or three years now. You know, practically that's really worked pretty well. We haven't seen much most of the time. But those herds over time tend to go negative.

And because we also tend to batch pigs today in finishing, we've got nice, little neat units of pigs in finishing. Those pigs don't have any older vertical exposure from older or younger pigs and so we've created these really quite large populations of two to six to eight to 10,000 finishing pigs that are mycoplasma naïve and haven't been exposed to the time of weaning. And then, we've had some slight alterations in transmission rate in the sow farms. So, it started kind of slow where we had, what historically now it looks like probably a fairly naïve sow farm, but there were a few affected animals left in there. We dumped in negative, dumped in negative, dumped in negative and so we've kind of gotten over the tipping point. If we want to go back and think about the good, old Susceptible Resisted Infected model.

So we had this huge pile that was no longer resistant and was susceptible, but we didn't eliminate all of the infected. Eventually we had infected susceptibles and we had outbreaks within the sow farms. Now, we continued to vaccinate gilts prior to entry and so the gilts were immunized and so we didn't really see a significant cough in the sow farm. But clearly as we've gone back and modeled that, the transmission rate from sow to pig was very, very high. And so as we think about that, why do we say that? We had infection, clinic infection with mycoplasma pneumonia in pigs at four to six weeks post-weaning, so really, really young pigs with acute mycoplasma. And as Joe said, when these negative herds go positive, it's ugly. And so, in those farms I think in general we tripled the mortality.

SARAH:
Yikes!

JIM:
So if it was a 5% wean-to-market mortality, we took it to 15%. If it was a 7% wean-to-market mortality source, we took it to 21%. And that happened for about 20 weeks. And that's about the kind of consistent number that we saw and so what do we see in those herds, we see the finishing pigs break very early and within that flow, the finishing pigs would break very early then they would get slightly older, slightly older, slightly older and that's really just a function on about how many pigs are infected at weaning. See if you think about it, you've got that outbreak in the sow farm. There's a lot of shedding sow to pig in the sow farm. So those pigs are breaking early because there are a lot of infected pigs. So there is a lot of material to cause infection.

Over time, the number of shedding sows is reduced so there are fewer infected pigs at weaning. And so those pigs break later and later. So it really required pretty extensive intervention in those herds to come back . . . and you know we're using Lincomix® 44, pretty typically in the feed to try to control that. We've had to medicate the sow farms to try to control infection there. We've mass vaccinated sow farms and I'm not sure where we've affected. We may have been late to the call on that. And then

certainly, we had to alter vaccination schedules in those pigs to try to make sure we got vaccination in, as Joe said, “30 days ahead of infection.”

So if you think about it with pigs breaking four weeks post-weaning, you know we would have historically vaccinated at weaning at three weeks later. I mean, that’s kind of our regimen. I’ve kind of been on and off the one-dose thing over time, but we’ve kind of said ok, we philosophically believe in two-doses or I did philosophically believe in two-doses probably not based on fact, but you know the three most dangerous words in medicine “*in my experience.*”

And so, we had done that and really did not have good control in spite of the fact that we vaccinated those pigs. And we can call it vaccine failure, but I think vaccine failure is an inappropriate description. It was infection just flat overwhelmed with vaccination.

And so vaccine is an insurance policy, right? We’re not buying elimination; we’re buying insurance. And we just probably can’t expect a vaccine to perform that way. So, we had “vaccine failure” in those flows, but it wasn’t because it was a bad vaccine or we really had the timing that wrong. We screwed up the sow farm. You know, we’ve got too much sow to pig transmission. So, it went coughing downstream. We’ve got all of those stabilized, but we’ve done a lot of work and a lot of money. You know, with the triple mortality maturity growth rates are off ect. that’s a little bit harder in my world to interpret because we get a lot of variation in growth rate depending upon where those pigs are at and whether their infections they would normally get, but significant, significant cost.

SARAH:

Yup. Joe, I have a herd that I think it is a pretty typical case of wean-to-market mycoplasma exposure. These pigs cough late. They cough probably, you know, somewhere between 20-26 weeks of age. And if we look at their serological profile, they’re really about 60-80% positive at eight weeks of age and then at 12 weeks of age they go down to 20%, 16 weeks of age. They’re down close to 0%, but then they start to come back up serologically to the point where at that 26-week period, they are 80% positive serologically.

We typically have vaccinated at weaning and two weeks later. And Joe, I think I have you to blame for this. They recently read your *National Hog Farmer* article and so they’re asking me right now whether or not we should move to early vaccination? I really feel pretty good about the control that we’ve achieved so far with vaccination and occasional late medication prior to that cough. Talk to me, Joe. What should we do? Should we consider this? The reason they want to do it is for labor reasons.

JOE:

Well, Sarah, first I’m glad they read the article, so that’s reassuring!

SARAH:

Right!

JOE:

And I also think in your example that says there is some late term exposure occurring with less than stellar control. and The reason we did this study was to try to determine if we could get the same response from early piglet vaccination compared to what we would of considered our goal standard, which was three weeks of vaccination and a second dose subsequent to that. In addition to the labor as being one component, probably more importantly than that as Jim noted, it’s becoming increasingly

clear to me that we have to have pigs vaccinated prior to the time of transmission from pig to pig. And today, we lack the techniques to fully understand the sow to pig prevalence of transmission, and so we are relying on the clinical responses. So when we set this study up, key drivers for that was to see if we could get equal and adequate performance when we've vaccinated pigs from early piglet vaccination compared to our three week and post-weaning vaccine. At the same time, we were understanding that when we look at a number of systems, compliance is easier to achieve in the farrowing house than it is in the wean-to-finish barn strictly because of the number of sites that we have to monitor or audit and the consistency of labor and really the understanding of the importance of doing procedures correctly.

SARAH:

I think this would definitely be a system that would benefit from having this task at the farrowing site and it's nothing against the wean-to-finish labor team, but it's just sometimes harder to catch all of those pigs in those large pen wean-to-finish situations.

JOE:

The other thing, Sarah, too is when you look at how we fill wean-to-finish barns today, unless it's a large system, there is frequently a one to two week fill and so when we look at transmission rates and trying to set the time from vaccine to transmission, that may be a culprit back to our good old fill time as to why there is differences in population efficacy between one flow and the other flow.

SARAH:

Yeah. The other thing that is going on in my mind, this is a mycoplasma positive sow herd and they are bringing in negative gilts and so we probably do have some increased gilt to pig transmission that could be spreading out when our pigs are getting exposed. These are large sites too. There are 10,000 pigs at one site. So these pigs are going to get exposed to mycoplasma probably fairly early considering the aerosol transmission factor. So I can justify in my mind why we should consider giving it at farrowing, but what do I tell my farrowing crew? I mean, they're going to balk a little bit. Why is this important enough that they need to add more to their plate?

JIM:

Yeah. I think the key message is Sarah for one, as I look at wean-to-finish guys; we tend to do things only every six months.

SARAH:

Right.

JIM:

Because the way the barn works . . . and so if we got one barn off to the side, particularly the contract partner that would be raising those pigs, it's just hard and even if you know what you are doing and you care a lot, if you only ever do it every six months, it's hard to get good at it.

SARAH:

Right.

JIM:

And so, we do it every day in the farrowing house so that it's an advantage. But I think as we talk to our farrowing house guys, the real message is that "A. You're picking the pig up anyway, right? We're not

going to add an extra deal. B, you're the best at this and we need you to get it done. And C, it's what's best for the pig. I think those are kind of the three key messages as I carry these things forward.

SARAH:

Absolutely.

JIM:

And so, I mean, as we've gone back and looked at it, you know, tenths of a second per pig to get that extra vaccine at processing and we're already picking up those pigs and as I look at early vaccination, we aren't going to add a shot, we aren't going to add a pickup, we'll just move that vaccination around to when we're picking that pig up anyway. So whether that's a day, three or four or seven or whatever that is, we'll move that vaccine around to avoid the picking up.

SARAH:

This farm picks up their pigs on Day 3, that's just when we process on that farm. So, it may not change how mycoplasma affects pigs in the finishers, but we should get, at least according to this research, the same benefits that we are seeing by currently vaccinating at weaning. Am I saying that right?

JIM:

Well, you know, we might get improved control on a couple of standpoints. So the reason you do a good controlled study is that, A, we just struggled taking field data and extrapolating it, a lot of times because we get implementation errors of getting the vaccine done.

So as Joe talked about, how we only do it every six months and we miss some pigs because we are doing them on the floor. So, we're getting 70% of them vaccinated instead of 100% of them vaccinated effectively. And so, if we increase that in farrowing, in accuracy, we may get some improved performance, but it's not because the vaccine works better, it's because we've got IP vaccine instead of OP vaccine. So in the pig (IP) is always better than on the pig (OP).

SARAH:

Right.

JIM:

And if we look at how we do a lot of vaccinations on the floor in three-week old pigs, I mean, there's a lot of OP vaccination that goes on. Just because of the speed, been there, done it myself. So in farrowing we need to increase that accuracy.

JOE:

The other reason to do this type of study, Sarah and Jim, is to still accept that we have a lot of concomitant viral infections that are occurring and we still have not been able to have consistently control PRRS episodes and the thing that we know from mycoplasma is that if we have viremic pigs at the time of vaccination,...

JIM:

...with PRRS or flu or Rota virus or TGE or whatever, right?

JOE:

Absolutely. And we also need to add PCV2 in there.

JIM:

That's right.

JOE:

And we know that we have a percentage of pigs that are viremic at birth, viremic at weaning. So to try to avoid vaccination when we have viral circulation is also a key component of this type of study, which then allows us to change our strategies and would give us a strategy of vaccination when we had the lowest amount of viremic virus moving or viremic pigs.

SARAH:

So, I heard a story referring to this early vaccination strategy and tell me if it was a hocus pocus story or not. That there is an opportunity of time in the pig's immune system that you can "hyper-stimulate" their immune system so that they have a better response when this vaccine is given in that one to five day window. What has the research told us?

JIM:

Maybe two sides...I can tell you where that story came from a little bit, Sarah.

SARAH:

Ok.

JIM:

So there is a theory out there that that's true. And there's been some work done I think in guinea pigs or another lab type species and maybe some work in mice as well. I don't remember exactly, but I know that there's been some guinea pig work done and it would suggest that really early immunization may stimulate more immune cell or differentiation when those immune cells are differentiating shortly after birth, particularly T cells. In the thymus, you may get more shifting to a preferred status. I.E. mycoplasma specific-- if they get exposed to those agents early the work has really been done around, because they are trying to figure out asthma and all this other stuff in kids and so it is . . . because we don't expose kids to enough bugs when they're early, because their immune system does not get them, themselves the recognition, blah, blah, blah...ok. So there's an offshoot...we've never tested that in pigs and we've never really tested it in any food species. So we don't know if it is actually really true or not. I think it's a great theory, but I don't know if it's true.

SARAH:

Ok.

JIM:

Joe, can talk through this set of data, but my interpretation of this data is that that's not true. At least based upon this data and the challenge model we used that there was no difference in performance.

SARAH:

So Joe, talk to me about this trial. You vaccinated pigs at an early age and then again at weaning and I believe there were other subsets. Can you talk to me about that trial.

JOE:

We also vaccinated another subset of pigs at weaning and 21 days post-weaning. We then, through the guidance of Dr. Erin Straight, inoculated those pigs with her M. hyo challenge so we knew we had a determined challenge that would give us a very, very good exposure. The summary of that data was that the vaccination schedules and the vaccines all performed equally well. And that's important, because that was our hypothesis.

SARAH:

So what you were trying to do was show that vaccination of pigs at an early age would give us a similar performance and similar control as to what we would consider our gold standard vaccination program.

So when I consider application of this in the field, one thing for me that is reassuring is that the U.S. is not the first to implement this strategy. This is a timing strategy that has been used very successfully around the world. Joe, can you comment on that?

JOE:

I certainly can. Vaccination of suckling pigs has been done for quite a number of years in Europe successfully. And I think it is quite interesting, Jim, that we would of argued maternal antibody interference as a reason why in the U.S. we did not do that or at least specifically myself, personally, would have said that I had some concerns. This trial again, reemphasized to me that those concerns are not correct and that we can effectively vaccinate suckling pigs.

JIM:

Yeah, I think this whole maternal antibody thing has maybe gotten a little blown out of proportion in terms of...that if you look at the studies, and there's numerous people trying to look at this. That it's like every time you turn around, one says, "X," and one says, "Y," in terms of whether it protects or not. And I think we got confused because we confuse the fact of shedding sows versus high maternal antibody. And so, if you look at the antibody titers at work, particularly again ELISA antibodies . . . and there's various formats of the ELISA whether it's between the Tween 20, the IDEX or the DAKO and there's differences in sensitivities, but they all test circulating antibody.

Two or three things to remember that is important. One, if an animal's antibody positive it doesn't predict whether to protect her or not. So, we don't know just because they have antibodies if they're actually immune or those antibodies mean squat in terms of protection. We know they are not correlated. So just because a piglet's got circulating antibody doesn't necessarily mean that it's going to have anything to do with protection nor its dam. Two, probably 200 days after that animal clears the infection, which might be 30-60 days after initial infection. They are going to go antibody negative and so if we've got high levels of antibody in a dam being passed through a pig, it probably suggests it's been infected fairly recently by even the last 100 days. And so, we don't measure shedding very well and it's also likely that they may be shedding even though we have or have not measured. It is as Joe said earlier, we really cannot quantify sow to pig transmission right today. The technology is just not there. So, I think we've confused that. I think the maternal antibody thing is a little bit muddy, but I don't think it's muddy to the point of saying it doesn't influence anything. I think it's got a lot of us confused, but probably not clinically important with respect to control.

SARAH:

So not a factor when considering early vaccination programs?

JIM:

That's right, yeah. And I think the other question that has come up is duration of immunity stuff. How long does immunity last? I think I'm pretty comfortable if you kind of look at all the vaccine studies, data in aggregate, for the practical purposes of a slaughter pig; it's lifelong. And so where we've had duration of immunities, I think really if you go back and look at what came out of Thacker's lab several years ago at Iowa State, Ilene's lab, at Iowa State. There's this whole argument that Joe was talking about with con-commitment viral infections and really any virus infection really whacks the immunity, you induce, you get some immunity, but it's not as good. More virus equals worse immunity. And so, we were getting poor, late protection because we were probably vaccinating those pigs right in the face of, you know, one, two, three, four viral infections and so, vaccine didn't work.

SARAH:

So, if we go back to that same case, that's where I might have improved immunity is I was vaccinating in the face of something else going on which I am thinking in my head, there may be some other viral challenges there and so I may get better protection by moving that vaccine early in that case.

JIM:

Absolutely. So the data says that a controlled study and again the value of this study is that when we do them in the field we never really know when they get exposed. Here we drew a line in the sand and knew it was 30 days after the second vaccination, so seven weeks post-wean. We would need to infect those seeder pigs and we got significant clinical disease in that part. So we knew when they got exposed so that we could sort out what happened. Controlled data says, "Ah, they're all equivalent." In the field, we know they're not equivalent. So we know it's not a vaccine performance issue, it's an application issue of am I giving it in the face of high sow challenge? "Am I giving the face of a viral infection? Am I not giving it to the pig? I.e. It's on the pig instead of in the pig." And so, as I think about myco vaccine timing, if you look at where we're at today there's flexibility literally to go to any time post-weaning. It just allows us as practitioners to insert the vaccine at the point where we get the fewest other things that ruin the efficacy of the vaccine.

SARAH:

Let's bring it home. Joe, as a swine consultant, when you've looked over everything that we've talked about today and the research that your group has done, as a swine consultant, as someone going to farms, what's your take home points for your clients?

JOE:

Sarah, this study was designed to make us comfortable that we could effectively vaccinate suckling pigs. Even in the face of maternal animal antibodies. The design of the study was such that we knew, as Jim mentioned, the timing of that exposure. The outcome of the study was that there was no difference between any of those treatments. So what it's done for me is that now I can look at that specific herd, that specific flow of pigs, pull in all the other information I have of con-commitment, viral infections, changes in the transmission rate of either viruses or mycoplasma in the sow herd and feel comfortable that I can design this vaccination program for mycoplasma that will have the highest chance of success.

SARAH:

Ok. Great, Jim? What are your action points?

JIM:

Very similar wrap-up, but I've probably spend more time in the last year really trying to focus on, how do we understand the gilt coming into the herd better? How do I understand what is going on in the

sow farm? How do I get acclimation? I think it just continues to bop me over the head that as I think about helping sow farms improve health, which is really how do I help them improve growth and finish health, because that's a starting point obviously. Is how do I work on acclimation of those gilts and mycoplasma becomes part of that. If we are going to batch the gilts and bring them under the system, how do I get them into this sow farm early, maybe a 150 lbs. or less? And if I'm going to bring them in as weaners, which is pretty common today, how do I make sure I get those gilts acclimated, because sometimes we forget to think about the gilt when she is getting acclimated on the sow farm her entire life. And so we raise them, but they're in a separate barn and kind of batched by themselves. So how do I think about the acclimation process of those gilts starting very young to avoid those hiccups on farm?

SARAH:

Right. And for me, my take home message that I really want to emphasize as an opportunity to my clients is I see this early vaccination as a *big* opportunity to have our best people giving a vaccine that has a clear return on investment that if we know that our pigs are going to experience mycoplasma in the wean-to-market period that they need to have this vaccine on board to avoid the variation that mycoplasma causes when it becomes Enzootic Pneumonia.

With that, we are going to wrap things up and send you on your way. Stay on the road. Thanks for listening to our latest edition of *P's in a Pod*. Tune in next month when we talk about the Individual Pig Care Program that we've been working on with Pfizer Animal Health.

This podcast was sponsored by Pfizer Animal Health. A reminder for those of you who are Walking the Pens and doing Individual Pig Care each day.... Coughing pigs affected with mycoplasma and potential secondaries need injectable therapy. Draxxin is labeled to fight all 5 major respiratory pathogens that threaten growing and finishing hogs . . . including *M. hyo*. The withdrawal on Draxxin is only 5 days which is great when you need to treat those late finisher hogs. Remember if we lose that 200lb hog to pneumonia, it will cost us between \$80 to \$100 in feed alone and sometimes more. Treat them. It's the right thing to do.