

Respiratory disease

Recently, Dr. Max T. Rodibaugh met with Drs. Randy Jones, Craig Rowles, Steve Dudley, and Pat Halbur to discuss swine respiratory disease.

Have you been seeing a lot more respiratory disease lately?

Pat: We have seen severe respiratory disease outbreaks lately. Most typically at about 3–4 weeks postweaning, pigs are positive for PRRSV, and have interstitial pneumonia and early Mycoplasmal pneumonia lesions. Subsequent infection with swine influenza virus (SIV), porcine respiratory coronavirus (PRCV), or opportunistic bacteria add to the severity of the respiratory complex.

Steve: My understanding was that the *Mycoplasma* lesions typically don't show up that early in the nursery.

Pat: That was my belief in the past, as well. But now we're seeing evidence of Mycoplasmal pneumonia earlier in the production scheme. Perhaps this is due to the sow herd stability or changing production style

Shouldn't we focus on the the sow herd first?

Craig: That's right. That's a big part of the SEW thing that's been left out.

Max: SEW is not going to work as well out here in the field where we're starting up herds with a lot of young sows. All the research has been on old sows — we've just sort of glossed over that.

Steve: I've been involved in some of these discussions about not wanting to vaccinate. I see no disadvantage, other than cost, to vaccinating the sow herd. Is there a disadvantage other than cost to vaccinate the start-up herd, the parity-one herd, for all the respiratory pathogens you can think of?

Max: I can't think of any reason other than cost. That's going to be a factor. Then maybe deciding to change that vaccination schedule once that sow herd matures. But even then, some of those sow herds have pretty high turnover.

Craig: If 40% is our normal rollover rate, you've always got a start-up herd. In the future, we're going to bring in those gilts at 50–60 lb (23–27 kg) and get them associated with that breeding herd somehow, so that we can try to get some natural immunity built much earlier. I think that's particularly important until we have improved PRRS vaccines. We should be happy to have what we've got, but there's a lot of room for improvement. That way you're doubly insured — you've got natural exposure and you've got the vaccination.

What are the most common respiratory diseases you diagnose in your practice?

Craig: In the past 2 years, certainly we've had our fair share of combinations of PRRS and *Mycoplasma*, or PRRS and influenza. Unfortunately in our practice area, PRV is still a viable pathogen. So we're still seeing a variety of viral etiologies, along with *Pasteurella*, some *Actinobacillus pleuropneumoniae*, some *Salmonella*.

Steve: We tend to see the whole gamut. Recently in a herd where we instituted a *Mycoplasma*/PRRS program we had *Actinobacillus suis* that looked like *Salmonella* that was really explosive. But in our practice *Mycoplasma*, PRRS, and influenza are three pathogens that tend to cause the respiratory complex that we routinely see.

Randy: We're seeing *Mycoplasma*, influenza, PRRS, with some bacteria mixed in — *Streptococcus*, *Pasteurella*.

Pat: From a diagnostic perspective, the trend has been an almost six-fold increase in PRRS cases in the last 2 years, about a threefold increase in influenza, and almost a threefold increase in *Mycoplasma*, with the other pathogens staying the same or going down. It's skewed a bit in that veterinarians send in things that they have difficulty diagnosing. They won't send *A. pleuropneumoniae* or *Salmonella* — they're good at culturing those bacteria now. Certainly the three big players from a diagnostic lab perspective are influenza, *Mycoplasma*, and PRRS.

Max: Do you think from a diagnostic lab standpoint that any of that is because of better diagnostic capabilities?

Pat: I'd like to think we're considerably better than we were 2–3 years ago. I think we are improving with newer technologies like immunohistochemistry. We have some better ELISA serology tests for PRRS and *Mycoplasma*, now.

What tools do you use to diagnose respiratory problems? How do you approach a problem in the field?

Craig: Today the use of diagnostic labs has never been more important. Because of all these different viral etiologies that we're dealing with, you can't just separate that out with a gross pathologic exam and culture sensitivities. We need the increased capabilities that diagnostic labs bring to the table. So not only are we doing more tissue submissions, we're also doing a lot more acute/convalescent sera. It takes that whole battery of information before you really know what's going on.

Steve: I certainly agree with that. We're using some serology, some histology, and some traditional culture and sensitivity. With most of those respiratory cases, I think what Pat has been saying is that we need to send in more samples, we need to send in more animals, we

need to have a statistical sampling procedure, and so we certainly want to increase the sample size of how many pigs we kill and send. I don't think we have changed much on serology. We send 10–12 samples for a 60- to 100-head barn, which maybe is a little on the light end. What we tend to see is that it's either there or not — *Mycoplasma*, influenza, PRRS. We also send in four to five live animals and that helps us.

Randy: The more acutely affected animal you can find, I think, the better. You're going to find influenza early, and then it's going to be gone. Some of those others will hang around. I think the selection of animal that you get samples from is very important. You really have to decide what pathogens are most important. Which one are you going to try to control?

Pat: That's right. That's why I like to do these cross-sectional necropsies of pigs at all different stages, because then you can see what's been there from the beginning and where to implement each treatment. I like looking at healthy pigs in the same age group to see what pathogens may not be as important.

Max: Pat, would you describe how you suggest doing a cross-sectional exam — say on a 500-sow herd?

Pat: In a herd of that size, we would usually start on pigs that have been weaned a week or so. Submit three or four sick pigs and one healthy pig. Then at the end of the nursery phase, submit the same numbers. Then a couple weeks into finishing and maybe two more times during finishing, submit three or four pigs and one healthy pig. We usually end up spending \$30–\$50 per pig for a complete workup. That's not a lot of money when you think about the decisions you guys have to make in those herds, and whether to vaccinate a 500-sow herd for *Mycoplasma*. How much would that cost?

Max: If you vaccinate every pig you're going to be looking at \$10,000.

Pat: So you bring in 30 pigs and spend \$800–\$900 on diagnostics. It seems to be cost effective. At least the clients keep coming back for those kinds of workups.

Max: Do you prefer that the pigs themselves be submitted, or the tissues, or either?

Pat: Definitely live pigs if that's possible. The necropsy skills of veterinarians are getting very good, so the vet can call and get a protocol for what tissues that lab wants.

Max: It seems to me with that kind of program, if you take tissue samples in the field you need a really good recording system to keep all that straight. How many people are you seeing submit samples like that?

Pat: We'll get a couple submissions a week that way. Instead of spending it in small pieces, they get aggressive and do it quarterly — more as a herd-health monitoring strategy than anything else.

Max: So you are saying in some respects this might replace the slaughter check as a more accurate tool?

Pat: I don't think there is any comparison to the useful information

you get out of this compared to the slaughter check. I think slaughter checks are still valuable for atrophic rhinitis and maybe, although less so, for *Mycoplasma*. To reflect what's going on in the nursery pigs or early grow/finish phase, I'm not sure the slaughter check is of much value.

Max: So in many cases when you initially do a workup, people are following those up even if they're not having respiratory problems in the herd, just as a monitoring situation.

Pat: They're definitely following up with serology and in some cases continue to do periodic necropsies.

Do you think serology is good enough? What kind of procedure would you set up to monitor a herd serologically?

Randy: We started doing a lot of serial bleedings on tagged pigs. I think a lot of those are easier to interpret but take longer. So we go in and tag a group of 3-week-old pigs and do some 9-, 14-, and 20-week-old pigs at the same time and get a cross sectional with it. Then we've got the group of tagged pigs that follow. At any point when you bleed those tagged pigs, do another cross sectional if you want to and see what's on either side of them. PRRS-ELISA, *Mycoplasma*-ELISA, SIV-HI is what we're running right now.

Steve: We're doing the same thing — serially bleeding pigs. We feel pretty confident that the PRRS-ELISA is pretty accurate at monitoring where we're at. I've been a lot more confident recently now that the *Mycoplasma* test has become more accurate. We're still not happy with the serological test for influenza. We'll get a histopathology/necropsy diagnosis with influenza and still have negative animals.

Randy: Dr. Erickson at Mount Rollins Diagnostic Laboratory says that the younger the pigs are, the less accurate HI is. He says that there is some interference of the maternal antibodies with the HI test. He prefers the ELISA on young pigs, but on animals without maternal antibodies the HI has done pretty well.

Max: I've heard references to 1.5 as a cutoff on the ELISA for herds. Others will say if it's over 1.5 that's an absolute indication of active infection, and under that is perhaps just vaccine-induced. What's your interpretation of that? I worry that we're going back into the PRV titer interpretation game.

Pat: In pigs that we have experimentally vaccinated by intramuscular means we rarely get titers over 2. Then you subsequently challenge them and they're pretty impressive — ≥ 2.5 . It doesn't make any sense to me that it would be any different than a natural infection in terms of the antibody response, but it does seem to generally hold to that rule. But again the sequential serology is necessary because you don't know whether the antibody level has leveled off or is still on its way up or down.

Randy's point on influenza serology is very well taken. We commonly will isolate influenza out of pigs and detect it by immunohistochemistry and then go back to bleed penmates and titers have gone down or they will never have developed HI titers. There are likely more antigenic

variants of SIV out there than we think. This makes relying on serology for influenza questionable. Especially if you're going to make a decision to vaccinate for influenza — don't base it on serology alone.

Max: Do you feel that you should send in the serological samples individually or wait the whole 4- to 5-month period and then send them all in?

Pat: It's always best to run them all same day. It's less an issue if you're using the ELISA, but for the IFA and tests with subjective endpoints I would want to run those the same day. I feel confident in PRRS-ELISA and *Mycoplasma*-ELISA. Every time I send a sample through that lab it comes back about the same titer or S : P ratio.

Max: What about porcine respiratory coronavirus (PRCV)?

Pat: I've had the opportunity to work with a lot of PRCV isolates in pigs — maybe half a dozen or so — that we've purified and put back in pigs. With most of those, clinically you can't tell you infected the pigs. There are a small percent of those that will induce transient respiratory disease, like a mild influenza, but won't damage much of the lung (maybe 10%–15%). In combination with other pathogens, PRCV may be a significant part of the complex. It's one of those things that's fun to diagnose, but I don't know how to control it. It can help you explain some things that are there, and if you see a pattern of seroconversion at a certain age then maybe you could direct your medication for bacteria at that point in time. If you don't have a serious PRRS or *Mycoplasma* problem they'll move through PRCV without any trouble. Unfortunately the serology test is very expensive and turnaround time is not good.

Max: Pat, do you think cross-sectional serology is a pretty good reading of the herd?

Pat: In your smaller herds, one- or two-site herds, I think it is. When you deal with the kind of herds Randy does — with multiple finishing sites and multi-source nurseries going to different sites — it's more of a stretch to apply that to a whole system.

Randy: We do see differences in the same groups. Whether this is from lateral infection from neighboring sites or just a change in the dynamics of that population is hard to evaluate.

Max: It's an interesting observation. We talk about subpopulations and breeding herds, and I think now people are talking about patterns in subpopulations and finishers. It almost sounds as if you've got populations and not really subpopulations of differences in disease exposure.

Randy: A lot goes back to how they handle those pigs in farrowing. We get a lot of people who want to hold pigs back. It's like pulling teeth to get them either to euthanize or send those pigs on through the system.

Porcine respiratory disease complex (PRDC) — what do you think has changed that's really warranted the industry to come up with that acronym?

Craig: In some ways we do a disservice. Obviously, we've stolen it from the bovine respiratory disease complex. I think it's important for all of us to recognize that, yes, there are a variety of etiologic agents, but let's not just call it PRDC and walk away like there's nothing we can do about it. You need to do the type of analyses that we've been describing here, so that you can get a handle on exactly what you're dealing with. I think it's important we don't fall into that trap.

Steve: Obviously the biggest challenge veterinarians have is making priorities in disease-control strategies for these operations. That's why with the diagnostic information you can't just call it a complex and throw it out. The best strategy for farm, season, and situation are going to differ and so the treatment strategies tend to differ.

Randy: The risk is that they lump the complex into one disease then you tend to lump your treatments into one. Whether there's PRRS involved or not, there will be treatment for PRRS. Whether there's influenza involved or not, there will be vaccination for influenza. Treating it as a complex is not going to be cost effective.

Max: Do you think the PRRS is the major change in having to come up with this label for respiratory complex, or are there other factors you would throw into the mix?

Craig: We've got totally different systems than we used to have. All of a sudden now we're using different technologies. We have larger numbers of groups. We have multisite nurseries. It puts a whole different perspective on herd health, the population, and the epidemiology of what's going on. That's a huge factor in what we're doing.

Randy: You've changed exposure levels and colonization levels of these pigs by weaning them earlier.

Steve: We have the subpopulations, we have potentially more naive animals, and the whole potential line variation or change in the pigs as we go to the high-lean pigs.

Pat: Certainly there have been changes in production style. I'm a big believer in different genetic susceptibilities to diseases — particularly in PRRS. What's new in infectious diseases that wasn't here 5 years ago? Three things really: antigen variance of influenza, PRRS, and PRCV. I have a feeling that something has changed with *Mycoplasma* too, because it is much more severe. Maybe it's just the viruses that have made it that way, but I think there needs to be some serious research there to see why *Mycoplasma* vaccines aren't working as well as they had in the past. For *Mycoplasma* we're getting some serious vaccine failures.

How do you approach treatment of the various complexes or the individual diseases that make up the complex — for example, what about PRRS?

Randy: It really goes back now to checking the breeding herd and trying to stabilize the breeding herd. Herds at this time a year ago were undergoing major seroconversion in the nurseries; now we've stabilized their sow herd and the nurseries are negative. We've dumped a lot of vaccine into those herds. We've tried to emphasize acclimatization of gilts and that kind of thing too; the management things you do to keep a herd stabilized. We basically depopulate nursery sites every time we fill them. That depopulation technology is there for a lot of those producers with large sow farms and nursery sites. What we haven't really done is clean up our finisher sites. You're going to end up with some buildings there with older pigs when you put in younger pigs. So we still get some seroconversion on the finisher. In those instances we're vaccinating pigs as feeder pigs at 40–50 lb (18–23 kg).

Craig: Our experience has been the same. We felt that we had to stabilize the breeding herd. In many cases once we got the breeding herd stabilized, and did some of our cross-sectional studies in finishing, we've essentially dropped a lot of pig vaccine. We've experienced a number of the antigenic variants Pat described in regards to SIV as well and that is an instance where we have had good response to vaccine. One herd in particular was experiencing it all the way from nursery through finishing and in that particular instance we blanketed the sow herd and used vaccine on that herd for a short period of time — it wasn't permanent. By going in and blanketing the sow herd and using a vaccine for a period of about 4–5 months we were able finally to get it shut off. It was an influenza that would last nearly 30 days.

Max: Do you think that PRRS impairs the lungs' ability to fight the influenza, or do you think it's just a different strain of it?

Pat: I think more likely some of those pigs are still protected with passive antibodies and not yet susceptible. I think that's a big part of it at least to 11–12 weeks of age. I also think that if they have a lot of serious *Mycoplasma* plus PRRS damage to the lung, it's going to take them longer to clear influenza from herds. My impression is that influenza vaccines are being more widely used. Practitioners that I talk to on the phone each day seem to be pretty happy with that product.

Max: Vaccines are definitely being used more. I think part of it is the frustration with the respiratory complex and not getting a response with PRRS. You find PRRS, try a vaccination, and if it doesn't work you jump to the next thing. Some of the increase in use in influenza would be because of that.

Steve: Overall we haven't felt that the PRRS vaccine has been terribly effective. So we're only using it in farms where there's severe outbreaks. We're trying, obviously, to get the sow herd quiet and that's the key. We are trying to isolate some of those viruses and we have done some killed vaccines in the sow herds and in certain herds that has helped. I think the homologous versus heterogeneous challenge model

is really important. But because it has been less predictable, it has been harder for us to justify to producers to routinely do that.

Any further comments on influenza treatments or preventive use of vaccine? How are you using it in the practice?

Craig: We pretty much have stayed with the double dose in the face of an acute challenge.

Max: Is there the potential that a single dose for influenza or *Mycoplasma* will do some good?

Pat: I think you're counting on there being some exposure there already and it being a booster. I would have serious concerns, particularly with the influenza vaccine. One dose will probably not protect a naive pig against challenge.

Max: Do you think it could be the reverse? You dose a naive pig and rely on low environmental exposure to give the secondary response. But if the exposure is heavy, you're out of luck.

Pat: That's right. The exposure dose is all important with all these viral diseases. So if you can have proper pig density and ventilation and pig flow your dose is going to be lower. You may get by with one dose of vaccine in some facilities.

Randy: We've compared two-dose versus one-dose influenza on some sites where we've got four or five buildings. We'll vaccinate some with one dose, some with two doses, and some won't get any vaccine. The biggest thing we see where we know there's influenza is not a lot of death loss — just culls. Morbidity is really the problem we see with influenza where there are no other major complicating factors; e.g., ulcers. Most of those pigs die from ulcers. So we've seen no difference between one and two doses as far as the reduction in cull rate, but you will get a reduction in cull rate in our hands with the influenza vaccine, even with one dose. Clinically, the pigs are coughing, but they're still eating and growing. We're giving that single dose right when they come out of the nursery or a day or two after they get to the finisher — about 8–9 weeks of age. Like you say, where there are really some acute problems, we'll give a second dose and then we back down to one. We've had to go back — in some cases in late nursery — and vaccinate for PRRS to get better control. We are also looking at *Mycoplasma* vaccines as well. The question of which pathogen is the most important can be difficult to answer.

What about *Mycoplasma* vaccination, treatments, and control?

Steve: Where we decide to make a decision to use *Mycoplasma* we're always using two doses. We tend to recommend that we use it in an early-weaned pig where they're weaning between 14 and 18 days. We like to wait 2 weeks after they're into the nursery, give them the first dose, and 2–3 weeks later hit them with the second dose. We're also using some of the Lincocin® in the feed at various ages — some high levels for a couple weeks, and then lowering that dosage. This combination of protocols has been helpful in the control of *Mycoplasma*.

Randy: Where you don't really ever expose those younger pigs back to older pigs I think Lincocin[®] has really helped. Some producers just don't want to give a lot of shots — it's a labor problem. When you move that shot from the farrowing house to the nursery, there's a big difference. That's when you really have to start convincing them they need to do it.

Craig: I think one of the things that we really need to focus on is to be able to go in and retrospectively financially analyze what we're doing for these herds. I think it really behooves us to get involved financially in the records so that we can show them in black and white what the economic benefits or disadvantages of doing something are. I think we all need to work harder not just on biological or medical control, but also financial control of what we're doing in herds.

Steve: Craig's right. It's difficult, though, for people who work with

smaller operations to evaluate this, because there are not a lot of sub-populations to compare. So we will be looking at people in larger systems to help with that. We're involved in a very large system where they're trying to evaluate just feed additives (Lincocin[®]) without any vaccination versus vaccinations of one-dose and two-doses. We're keeping records on all of the 18,000–20,000 sows in that database — hopefully in a year that study will be revealing.

Craig: The problem is that a lot of this information is going to be generated internally and privately and we're in an industry now where there's not a lot of publicly funded research being done on financial analysis, so somehow we need to figure out a way to distribute information amongst ourselves.

