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A controversial view of *Mycoplasma hyopneumoniae* epidemiology

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The epidemiology of Mycoplasma pneumonia has been changing during the last decade. Traditionally, *Mycoplasma hyopneumoniae* resulted in "Enzootic Pneumonia", with early bouts of pneumonia, usually quite apparent at the end of the nursery, characterized by dry cough and labored breathing. Animals at slaughter would usually show pulmonary lesions, but these tended to be small and resolving, making it very difficult to establish meaningful correlations between extent of lesion and individual animal productivity.

With the wide use of segregated weaning, it appeared at first that this system had actually eradicated the disease. While this had worked for nucleus herds, in commercial herds we learned soon enough that we had only delayed the onset of the problem, with signs appearing now at the end of finishing and slaughter lesions being larger and more acute. This form of the disease has been named Porcine Respiratory Disease Complex, or PRDC, a name that implies that other agents are involved in this problem. I think this is a mistake, since "PRDC" can be seen in farms that are free of most common viral agents (including PRRSV). Conversely, Mycoplasma-free herds, even when infected with PRRSV and SIV, do not show the characteristic late cough and lung lesions, suggesting that the central player in this disease is Mycoplasma. In my view, therefore, controlling Mycoplasma is the single (maybe the only) tool towards controlling the problem. In fact, I'm proposing that we should not be using the name of PRDC but instead refer to it at Mycoplasma Pneumonia.

Why then, did we believe at the start of MEW that we had actually eradicated the disease, only to be proved wrong later on?

In part was because this strategy was first driven by breeding stock companies as an effort to generate negative animals from positive sources, using very early weaning together with heavy sow medication.. Therefore, at the start of the MEW revolution, sows had a very robust immunity, due to the fact that they were being constantly re-exposed to the agent coming from the growing pigs. This, together with widespread use of sow antibiotics, probably reduced shedding (in the presence of high levels of maternal immunity) and resulted in very low levels of infected piglets at weaning, which in turn resulted in those groups reaching slaughter without overt signs of the disease.

With time, this robust sow herd immunity started to wane, since the sows were no longer being exposed from the growing pigs. This waning sow herd immunity was further compounded by the use of negative replacement gilts, which further diluted the herd immunity. This decreased immunity, together with discontinuing most sow antibiotic programs, allowed the organism to reemerge by infecting more piglets before weaning.

This theory has now largely been validated by our recent work that has demonstrated that prevalence of infected piglets at weaning is a central risk factor for that group developing the clinical disease. It is now clear that levels of sow immunity and sow shedding are the single most important factors to consider in the epidemiology of Mycoplasma infections in offsite weaning programs.

Persistence of Mycoplasma infections

One of the more frustrating aspects in trying to control the disease is the difficulty found in reducing the levels of infection in the sow herd. One can assume that infection is perpetuated by the entry of negative gilt replacements (or late infected gilts) into the sow herd, even when vaccinated, since the vaccine can decrease, but not eliminate, infection. The question is why does the organism last so long in gestation populations? Part of the answer comes from previous work from our group, which showed that, even after 180 days post infection gilts were still infected having viable organisms in the respiratory surfaces. They could also infect (at least until 150 days pi. the last date measured) other animals. This extremely long infection, unusual in bacteria, explains why populations of sows are continuously recirculating the organism by infecting incoming gilts.

In fact, we do not know how long Mycoplasma infection persists in adult pigs. Very early work, performed in the UK in the 70s suggested that sows of third or more parity had cleared the infection, in turn suggesting that at some point the organism does disappear. Since this was studied in farrow-to-finish farms, with their typical infection in the nursery, one can speculate that clearance of the organism took about 10-12 months to achieve. This, in fact

is the basis for the Swiss Mycoplasma eradication protocol, which culls all animals of less than 10 months of age from the herd and then uses heavy antibiotic treatments in the older animals.

Unfortunately, most of this work was performed before the advent of PCR-based diagnostics, and, in the case of the British work, before adequate serological tests were available. Freedom of Mycoplasma was based on clinical signs and slaughter lesions, which we now know can be absent in infected animals, if the infective dose and herd prevalence are low. In fact, a British program based on establishing herds derived from litters of older sows developed many "unexplained" reinfections which were then assumed to derive from aerosol transmission from neighbor farms, when in fact they probably came from within the population, which had never been truly free of the infection.

The bottom line is that it is near impossible to eliminate infection within gestation units, as long as we continue to bring susceptible replacements into the population. In the absence of an eradication effort, mostly by depop / repop, or maybe by the Swiss method (doubtful in our large, low immunity sow herds), we have to live with infected sow herds which then result in infected suckling piglets. However, we can try to modulate the level of infection in these piglets and thereby achieve a measure of control. Our group has previously shown that both sow vaccination or sow antibiotic treatment, significantly reduced prevalence at weaning, once more reinforcing the view that the sow plays a central role in Mycoplasma epidemiology and that we can modulate this role with appropriate sow intervention strategies.

Prevalence at weaning: A risk indicator

We have seen in the previous presentation that prevalence at weaning is the main risk factor for a population subsequently developing the disease. This of course applies to segregated production systems, where the most likely source of infection comes from within the group. Depending on how the nursery site is set up, some horizontal transmission may also occur, which changes the predictability of prevalence at weaning. Nurseries using a "motel" design are susceptible to cross infections between groups, particularly if personnel movement is unregulated and they don't wash their hands or change clothes between groups. Another source of unpredictability comes from multi-source nurseries, in which prevalence at weaning may vary considerably between the different sow herds. With these provisos, one can see that there are three potential prevalence at weaning scenarios: low (less than 5% of animals infected), medium (5-20%) and high (>20%):

High prevalence

It is relatively uncommon to find herds with prevalence of more than 20% in offsite situations. High prevalence at weaning usually means a young start-up sow herd or, more likely, an uncontrolled herd where gilt acclimatization and vaccination is not done well, or where there is considerable sow movement. More commonly, high prevalence tends to be a post-weaning event, especially in continuous flow farms, where lateral infection from older pigs is common. As we said before, this increased post-wean prevalence may also be the result of poor nursery design and uncontrolled personnel movement between nursery groups.

Whatever the cause, high prevalence levels usually result in rapid dissemination of the agent within the group, resulting in early infection of the "Enzootic Pneumonia" type, which is uncommon today.

Medium prevalence

As a result of continuous Mycoplasma-negative gilt entry, together with lowered herd immunity, medium levels of prevalence at weaning are the most commonly seen in commercial, offsite herds. These levels result is a slow spread of the infection, taking time to reach most animals within the group. The end result is delayed finishing infection, with more severe signs and more production impact than in early infections (this being the result of the fact that adult animals are more sensitive to Mycoplasma infection and develop a more severe disease). This is the normal "PRDC" form that we usually see today.

Low prevalence

Again not commonly seen, but probably the norm at the start of the offsite system in the early 90's. In this case, even though the group is infected, there are so few shedders (and probably shedding at a low level) within the group, that infection is much delayed and may not be apparent at slaughter. These groups behave clinically and production wise identical to groups from herds that are truly Mycoplasma free. Obviously, this is the scenario which we should be trying to achieve.

Achieving low prevalence at weaning

There has been a change in Mycoplasma vaccination programs, which may have an effect on control of this disease. When vaccines became available, the recommended program was of early piglet vaccination, during the suckling period. This made sense, since at that time most farms were continuous flow and early infection was the expected event, therefore requiring that piglets obtain immunity as early as possible. This strategy is still commonly used in Europe, where Mycoplasma vaccination is widely seen as very successful, being able to control clinical disease in most farms. Conversely, in the US this program was

rapidly modified to a delayed (6 and 8 weeks of age, with conventional two-shot vaccine) program. This was done in an effort to control the delayed form of the disease, but this program was never properly studied or validated. The end result is that Mycoplasma vaccination here is less successful, requiring in many instances the additional use of antibiotics in the growing pigs. An additional problem of this delayed vaccination is that it is given at a time when PRRSV is commonly active, thereby spreading the virus and probably worsening the impact of this disease.

Since prevalence at weaning is such a central event, and early vaccination of growing pigs has also been shown to decrease prevalence, we should seriously contemplate returning to this program and not using the delayed vaccination approach any more.

Provided that the nursery site is well managed, low prevalence is achieved mainly by sow intervention. We have previously shown that both sow vaccination and/ or sow antibiotic treatment can significantly lower the prevalence level. A combination of these two interventions would presumably give the best effects, but it is probably too costly in commercial settings. Therefore a decision of whether to vaccinate or to treat the sow needs to be taken and this decision also involves what interventions we are going to give to the growing pigs. Regarding this issue, the likely intervention programs would be the following:

Sow vaccination with no growing pig vaccination

A rather ideal scenario, using only sow vaccination can reduce prevalence levels significantly, in many cases below the threshold for manifesting clinical disease. However, this works well only in if lateral spread is controlled, such as in multi-site situations (with the problems associated from multiple source nurseries), or in systems where a tight control of animal and personnel movement within the nurseries can be achieved.

Sow and piglet (early) vaccination

This program can achieve considerable reductions in prewean prevalence, since it attacks shedding from both sows and infected piglets. However, this may be too costly and labor intensive for commercial farms. There are some concerns on interfering with piglet vaccination success by boosting sow immunity, a concern that has been partly studied but does not still have a satisfactory answer, although it would appear that interference from sow immunity may be less than what previously thought.

Early piglet vaccination without sow intervention

The classical "European" approach may be successful in many farms, but fail in others, depending on prevalence levels. In high pre-wean prevalence farms, this approach may move disease up from the late nursery to finishing, an event that probably happened at the start of offsite programs, giving the impression that early vaccination was not working, resulting in the late vaccination programs that are common today. In medium prevalence farms, this strategy would work well, but will probably vary considerably between groups, depending on prevalence levels.

Piglet vaccination with sow treatment

This combination seems ideal for commercial farms, since it reduces sow shedding through antibiotic treatment, while also reducing piglet shedding through vaccination. Its main drawback may be cost and a careful evaluation of the disease vs. intervention costs would be necessary.

In summary, Mycoplasma epidemiology in offsite herds is very much centered on the sow. Sows are the main source of infection for suckling pigs, which then drag the organism into the nursery. Depending on the prevalence of infected pigs at weaning, together with the production system, this prevalence results in a number of disease scenarios, ranging from early signs within the nursery to the absence of signs and lesions all the way to slaughter. Modulation of pre wean prevalence is therefore crucial and a number of intervention scenarios to achieve this are discussed.

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