**Mycoplasma hyopneumoniae**: Gilts, are they the problem?

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**Lack of information, lack of consensus**

Porcine Respiratory Disease Complex (PRDC) continues to account for a majority of the economic losses due to disease in the late finishing period. *M. hyopneumoniae* (Mhp) is a key contributor to PRDC in the late finishing period. While there are many things that we think we know about Mhp under field conditions, there is a paucity of peer-reviewed published information to support most of the “conventional wisdom” of veterinarians in managing Mhp’s role in PRDC. This has resulted in many divergent and often directly conflicting approaches to controlling Mhp in modern production systems.

**High costs, high risk**

With high market values and input costs, mortality late in the growing period from PRDC is costly. Solutions that are not evidence based will result in failure, economic losses for our producers and further marginalization of veterinarians in science-driven production systems. Evidence exists to better guide our decisions on the management of Mhp in production systems.

**Fix the gilts, fix Mycoplasma**

Control of PRDC associated with Mhp in late finishing will not be achieved until the Mhp infection status of gilts at the time of entry is managed.

- The number of pigs infected at weaning determines the disease load from Mhp in growing pigs.
- The number of pigs infected at weaning is directly related to the number of sows shedding Mhp at the time of farrowing.
- Females that are infected in the 200 days prior to farrowing are likely to shed Mhp to their piglets.
- Gilts are the most likely animals to be infected in the 200 days prior to farrowing.

**Piglet infection is root cause**

The first step in understanding the potential impact of an infectious disease in a population is to understand the root mechanisms of source and transmission. In the case of Mhp, a slow-growing organism with long infection to clinical impact period, infection early in life is important. Without a sufficient period for the organism to grow in the host prior to harvest, then clinically important disease does not occur.

This means that for disease to occur in the population, increased transmission early in the growing period is important. There are multiple ways to promote transmission but the simplest is to have more infected pigs in the population when the group starts, meaning that less transmission is needed to produce clinically important disease.

This hypothesis was tested by Fano et al. where he clearly demonstrated that increasing the number of infected piglets at weaning produced earlier and more severe respiratory disease associated with Mhp. If more piglets infected at weaning are the cause of the disease in late finishing, how do piglets get infected?

**Shedding sows mean infected pigs**

While it is possible that piglets could get infected from lateral sources during transport or immediately after weaning, this appears to be an unlikely source of infection. The dam is the most logical source of infection in pigs at weaning for most diseases, and Mhp is no exception.

There have been numerous investigations of infection status of piglets at weaning and the impact of sow infection and antibody status on piglet infections. In a review of all the available evidence, Sibilia et al. conclude that sow to pig transmission is a likely driver of clinical disease in infected herds. It is logical to assume from the available evidence that controlling this transmission could be key to improving clinical outcomes in late finishing pigs.

**When not if is important**

The available evidence would suggest that some but not all sows are shedding Mhp to their piglets as the reported infection rates at weaning are all low. This raises the question of why are some sows more likely to shed than others.
James F. Lowe

There is clear evidence in the literature that animals that are within 200 days of a recent Mhp infection are capable of infecting other pig.\(^6\) This means that any female infected with in the last 200 days is capable and likely to infect her offspring but those that are infected more than 200 days ago are not likely to infect their offspring.

**Gilts not sows are the problem**

There is evidence to suggest that once an animal is infected with a single strain of Mhp they are unlikely to be reinfected or shed the organism again\(^7\). This suggests that if the herd does not have new introductions for at least 200 days Mhp shedding would stop. There is evidence to suggest that this does occur. In the case of Mhp elimination programs herd closure for extended periods of time will stop sow to piglet transmission\(^8\).

In typical breeding herds with continuous or intermittent introduction and removals the new animals are the most likely to be infected within 200 days of farrowing. There are two potential routes for this to happen. First naïve gilts could be introduced into an infected herd and become infected during the gestation period. The second scenario is gilts that have been recently infected prior to arrival (late in the development period) and have not cleared the organism prior to farrowing.

Clinically both of these scenarios have the same outcome and from the view of late finishing PRDC are not distinguishable.

**It is not the vaccine**

The other potential hypotheses for PRDC associated with Mhp are enhanced transmission post weaning and enhanced sow to pig transmission. In both cases there is clear evidence that these are not the likely problem. It has been demonstrated that PRRS infection does not enhance Mhp transmission in pigs\(^9\) suggesting that co-factors would be likely to change the transmission rates of Mhp in pig populations.

In addition there is clear evidence that conventional killed bacterins do not alter the transmission rates of Mhp in populations\(^6,10\). This means that while Mhp bacterin may improve clinical sigs from Mhp in pigs\(^11,19\) but it is does not alter the number of pigs that are infected. While bacterins will remain a valuable tool and serve as proven insurance for losses they will not solve the root cause of losses or minimize the impact of Mhp on profit.

**Understanding is the first step**

For long term success in Mhp management understanding infection dynamics within herds is critical. Matching infection status of replacements with the herd (+/+,-/-) is the first step. In infected herds, understanding transmission patterns of Mhp during gilt development is necessary to understand any potential solutions to the problem.

As an industry we know very little about what drives the changes in infection timing within herds and gilt developers. A concerted effort to understand infection dynamics will be needed to have meaningful progress on solutions. We know what the problem is, but do not have enough knowledge to have evidence based solutions to our problems.

**References**

7. Pieters, M., Duration of immunity following infection with *M. hyopneumoniae* J.F. Lowe, Editor. 2012: St Paul, MN.
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**Figure 1:** The status of gilts at entry to the sow farm determine the number of infected pigs at weaning which in turn determines if there is clinically important disease associated with Mhp during the growing period. The there is no other interpretation of the logic path because the alternative paths shown with a solid line are either biologically implausible or there is sound evidence to suggest that they cannot be meaningfully changed with known interventions.