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***Mycoplasma hyopneumoniae* prevalence at weaning: What do we know (and do not know) about it?**

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Introduction

Mycoplasma hyopneumoniae (*M. hyopneumoniae*) is the primary causative agent of Enzootic Pneumonia (Mare and Switzer, 1965; Goodwin et al., 1965), and predisposes infected pigs to colonization with other bacterial and viral pathogens, then becoming a central player in the Porcine Respiratory Disease Complex. *M. hyopneumoniae* is transmitted through pig-to-pig contact. Sow-to-piglet transmission is considered a determinant event in within-herd pathogen perpetuation in segregated production systems. But, very little is known about the colonization process during the pre-weaning phase. The prevalence of *M. hyopneumoniae* at weaning age – evaluated by nasal swab PCR has been suggested as an indicator of sow-to-piglet colonization. Correlation analyses have provided evidence that severity of disease caused by *M. hyopneumoniae* in growing pigs can be predicted by the prevalence at weaning in segregated systems (Fano et al., 2007). Therefore, the purpose of this document is to review the current literature regarding *M. hyopneumoniae* prevalence at weaning and to propose ideas in order to generate information to fill in the knowledge gaps.

***M. hyopneumoniae* prevalence at weaning**

M. hyopneumoniae prevalence at weaning refers to the percentage of piglets positive to the pathogen detected at the end of the lactation period, in a given weaning group.

There are several factors that need to be considered when deciding how to estimate prevalence at weaning. First of all, a sample size representative of the population has to be selected. Sample size calculations should be based on herd size, the expected prevalence and the desired precision level. In most cases the expected prevalence is not known, and a low expected prevalence is assumed as a safer approach. A high precision level combined with a low expected prevalence results in a rather high sample size.

Usually, molecular diagnostic tools, such as species specific PCR assay are used to detect *M. hyopneumoniae*, due to the fact that serodiagnostics (e.g. ELISA test) cannot differentiate between passively acquired maternal antibodies,

antibodies generated after vaccination or antibodies generated after natural exposure.

Then the question comes about the type of sample that needs to be collected. Ideally, bronchial swabs would be the preferred sample, as a higher sensitivity is obtained with this type of sample, but the fact that they can only be collected in *post-mortem* conditions bans the idea of using this sample type in field conditions. Many of the studies referring to *M. hyopneumoniae* prevalence at weaning have been performed by sampling piglets using nasal swabs (Calsamiglia et al., 1999; Ruiz et al., 2003; Sibila et al., 2007; Fano et al., 2007; Villarreal et al., 2010; Nathues et al 2012). However, the argument has been made that other sample types that can be obtained *in vivo*, such as oro-pharyngeal swabs or tracheal swabs can detect positive piglets with a higher sensitivity (Fablet et al., 2010; Nathues et al., 2012). Another important aspect to consider when collecting samples is the subject to be sampled. Most studies show that litters and piglets within the litter are randomly selected, most probably because no information is available regarding the ideal sample subject.

What is known?

Piglets are born free of the *M. hyopneumoniae* and enzootic pneumonia. Studies performed under field conditions have shown that a small proportion of piglets (1.5%) can be colonized with the bacterium as early as one week of age (Sibila et al., 2007), but by the end of the lactation period a significantly varied proportion of animals will be colonized with *M. hyopneumoniae*.

M. hyopneumoniae prevalence at weaning have been measured in farrow to finish as well as farrow-to-wean farms. Some studies have presented repeated estimations within the same herd in consecutive production weeks, while others have sampled pigs in different herds in several different countries. However, the common result for the different investigations is a significant variation in the number of positive pigs at the end of the lactation period. For example, Fano et al., 2007 tested pigs in three different farms, sampling several weekly groups at each farm and found variations from 5.12 to 51.28%, from 0 to 38.46%, and from 2.5 to 5.12%. On the other hand, Villarreal

et al., 2010 tested animals in 9 different European countries (sampling several herds in each country) and found differences that went from 0 to 3.30%, all the way to 0 to 36.7%.

More recently, a few studies have investigated potential risk factors that could have an effect on *M. hyopneumoniae* prevalence at weaning. In one of these studies, herds that vaccinated sows against swine influenza virus (SIV) had a significantly higher risk of a piglet being positive for *M. hyopneumoniae* (OR 3.12; 95% CI 1.43-6.83; Villarreal et al., 2010). While Nathues et al. (2012 a) suggested that herds where batch farrowing is not adopted in a 1 or 3 week interval (OR 2.7), when the number of farrowing pens in one compartment was higher than 15 (OR 3.3), and when the total number of purchased gilts per year was higher than 120 (OR 5.8), then those herds were more often found positive for *M. hyopneumoniae* in suckling pigs.

On the other hand, dam vaccination has been suggested as a potential way to decrease *M. hyopneumoniae* prevalence at weaning, as Ruiz et al. (2003) showed a trend for lower prevalence in the offspring of dam vaccinated pre-farrowing. Also, Sibila et al. (2008) showed a lower proportion (only numerical) of colonized piglets born to vaccinated sows that were compared to piglets born to unvaccinated sows.

What is not known?

In general, there is a big knowledge gap in understanding how the clean newborn piglet gets colonized during the lactation period. In reality, to date no studies have been published about within litter transmission dynamics. In other words, no information is available about the way in which piglets get infected, and many questions can be asked about how this process happens.

Where does the initial *M. hyopneumoniae* come from? The first plausible explanation is that piglets get colonized from their mothers during the lactation period, but it is also known that not all piglets within a litter get colonized during the lactation period. Thus, what is it that makes a certain piglet more prone to colonization from the mother?

Do early colonized piglets with *M. hyopneumoniae* pose a colonization risk to littermates? It is not known whether piglets that are colonized during the first days of life can shed a bacterial load that is high enough to colonize littermates. However, the high contact rate among piglets within a litter could point at an ideal environment for pathogen transmission.

What is the role of the environment and fomites in *M. hyopneumoniae* transmission? Very few studies have been published about *M. hyopneumoniae* in the environment and the role of fomites in transmission during the lactation

period. The infectious pressure of a farrowing group as a whole, and the potential risk of individuals should also be questioned here. A recent study has shown that almost 15% of the pig farmers tested that were in contact with suckling piglets, harbored *M. hyopneumoniae* in their nasal mucosa (Nathues et al., 2012 b). However, the cause or effect aspect was not elucidated in that study.

How fast (or slow) can *M. hyopneumoniae* “move” within the litter during the lactation period? Experimental data have been published about the reproduction ratio for *M. hyopneumoniae* during the nursery period (Meyns et al., 2004; R_0 ; or number of pigs that get infected from one infected animal in a certain period of time), but such information have not been generated for the lactation period.

For the few risk factors for *M. hyopneumoniae* prevalence at weaning that have been identified, how well do they explain the variability of infected pigs within the same farm from week to week? Sow vaccination against SIV or the number of farrowing pens in one compartment seem to be factors that would affect the herd in the same way as a whole. Therefore, they do not appear as factors that would be responsible for the wide variation in prevalence at weaning from group to group. However, the total number of purchased gilts per year could be seen in a different way, as the proportion of newly introduced gilts in each farrowing group could vary from week to week and could help explain the variation, but the parity effect has not been conclusive when evaluating *M. hyopneumoniae* prevalence at weaning. Although it has been documented that higher proportions of young mothers are positive to *M. hyopneumoniae* when compared to old mothers within the same system.

How do management practices, vaccination and medication protocols affect *M. hyopneumoniae* prevalence at weaning? Several management factors applied at the sow or the piglet level could have an effect on *M. hyopneumoniae* transmission. For example, the length of the lactation period can vary from farm to farm and within farm, depending on the weekly weaning schedule. Maybe longer periods of time would result in higher prevalence at weaning, as there would be more time to establish effective contacts between infected and uninfected animals, but this hypothesis has not been identified as a risk factor in previous studies. In the case of cross-fostering, it has been demonstrated how profoundly it can affect maternal transfer of *M. hyopneumoniae* specific immunity. Perhaps cross-fostering could contribute to the transfer of colonized piglets into naïve litters, or naïve animals into colonized litters, but this effect has not been investigated.

Vaccination and/or medication protocols are usually applied at the sow or the piglet level. Sow vaccination has shown a trend for reduction in the proportion of colonized

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pigs at the end of the lactation period. On the other hand, vaccination of naïve animals does not protect from colonization (Haesebrouck et al., 2004; Meyns et al., 2006; Pieters et al., 2010; Villarreal et al., 2011), however these studies have been performed in older animals and such investigations have not been conducted in lactating piglets.

Medication with antimycoplasmal drugs is often practiced in swine farms, and although it can be directed to treatment of other pathogen(s), it would also have an effect of *M. hyopneumoniae* bacterial load. The effect of antimycoplasmal medication at the sow and/or piglet level and the resulting *M. hyopneumoniae* prevalence at weaning should be more closely investigated.

Conclusions

M. hyopneumoniae prevalence at weaning can be important indicator of disease severity in growing pigs. Thus, control measures directed at lowering *M. hyopneumoniae* prevalence at weaning would have a significant impact in disease presentation in grow-finishing pigs. However, it results difficult to design strategies or protocols to lower the proportion of animals that get colonized during lactation, when the risk factors have not been fully identified. Therefore, more investigation is needed to fill in the knowledge gaps regarding the dynamics of *M. hyopneumoniae* transmission during the lactation period.

References

1. Calsamiglia M, Pijoan C, Bosch GJ. 1999. Profiling *Mycoplasma hyopneumoniae* in farms using serology and a nested PCR technique. *Swine Health Prod.* 7(6):263–268.
2. Calsamiglia, M., Pijoan, C. 2000. Colonisation state and colostral immunity to *Mycoplasma hyopneumoniae* of different parity sows. *Vet Rec.* 146:530–532.
3. Fablet C, Marois C, Kobisch M, Madec F, Rose N. 2010. Estimation of the sensitivity of four sampling methods for *Mycoplasma hyopneumoniae* detection in live pigs using a Bayesian approach. *Vet Microbiol.* Jul 14;143(2–4):238–45.
4. Fano E, Pijoan C, Dee S, Deen J. 2007. Effect of *Mycoplasma hyopneumoniae* colonization at weaning on disease severity in growing pigs. *Can J Vet Res.* Jul;71(3):195–200.
5. Goodwin, R.F.W., Pomeroy, A.P., Whittlestone, P. 1965. Production of enzootic pneumoniae in pigs with a mycoplasma. *Vet. Rec.* 77:1247–1249.
6. Haesebrouck, F., Pasmans, F., Chiers, K., Maes, D., Ducatelle, R., Decostere, A., 2004. Efficacy of vaccines against bacterial diseases in swine: what can we expect? *Vet. Microbiol.* 100: 255–268.

7. Mare, C.J., Switzer, W.P. 1965. New species: *Mycoplasma hyopneumoniae*, a causative agent of virus pig pneumonia. *Vet. Med.* 60:841–846.
8. Meyns, T., Dewulf, J., de Kruif, A., Calus, D., Haesebrouck, F., Maes, D. 2006. Comparison of transmission of *Mycoplasma hyopneumoniae* in vaccinated and non-vaccinated populations. *Vaccine* 24: 7081–7086.
9. Meyns, T., Maes, D., Dewulf, J., Vicca, J., Haesebrouck, F., de Kruif, A. 2004. Quantification of the spread of *Mycoplasma hyopneumoniae* in nursery pigs using transmission experiments. *Prev. Vet. Med.* 66:265–275.
10. Nathues H., Woeste H., Doehring S., Fahrion AS., Doherr MG., Beilage EG. 2012 b. Detection of *Mycoplasma hyopneumoniae* in nasal swabs sampled from pig farmers. *Vet Rec.* Jun 16;170(24):623. Epub 2012 May 29.
11. Nathues H., Woeste H., Doehring S., Fahrion AS., Doherr MG., Beilage EG. 2012 a. Field study investigating the herd specific risk factors for *Mycoplasma hyopneumoniae* infections in suckling pigs in the region of Northern Germany. *IPVS 2012.* Jeju – South Korea. P 176.
12. Pieters, M., Fano, E., Pijoan, C., Dee, S. 2010. An experimental model to evaluate *Mycoplasma hyopneumoniae* transmission from asymptomatic carriers to unvaccinated and vaccinated sentinel pigs. *Can J. of Vet Res.* April 74(2):157–160.
13. Ruiz AR, Utrera V, Pijoan C. 2003. Effect of *Mycoplasma hyopneumoniae* sow vaccination on piglet colonization at weaning. *J Swine Health Prod.* 11(3):131–135.
14. Sibila M, Bernal R, Torrents D, Riera P, Llopart D, Calsamiglia M, Segalés J. 2008. Effect of sow vaccination against *Mycoplasma hyopneumoniae* on sow and piglet colonization and seroconversion and pig ling lesions at slaughter. *Vet Microbiol.* Feb 5;127(1–2):165–70.
15. Sibila, M., Nofrarias, M., López-Soria, S., Segalés, J., Riera, P., Llopart, D., Calsamiglia, M. 2007. Exploratory field study on *Mycoplasma hyopneumoniae* infection in suckling pigs. *Vet. Microbiol.* 121: 352–356.
16. Stärk KD, Miserez R, Siegmann S, Ochs H, Infanger P, Schmidt J. 2007. A successful national control programme for enzootic respiratory diseases in pigs in Switzerland. *Rev Sci Tech.* Dec;26(3):595–606. Review.
17. Villarreal I, Meyns T, Dewulf J, Vranckx K, Calus D, Pasmans F, Haesebrouck F, Maes D. 2011. The effect of vaccination on the transmission of *Mycoplasma hyopneumoniae* in pigs under field conditions. *Vet J.* Apr;188(1):48–52.
18. Villarreal, I., Vranckx, K., Duchateau, L., Pasmans, F., Haesebrouck, F., Jensen, J.C., Nanjiani, I.A., Maes, D. 2010. Early *Mycoplasma hyopneumoniae* infections in European suckling pigs in herds with respiratory problems: detection rate and risk factors. *Veterinari Medicina*, 55, 2010 (7): 318–324.

