Sponsors

University of Minnesota
College of Veterinary Medicine

College of Agricultural, Food and Environmental Sciences

Extension Service

Swine Center

Editors
W. Christopher Scruton
Stephen Claas

Layout
David Brown

Logo Design
Ruth Cronje, and Jan Swanson;
based on the original design by Dr. Robert Dunlop

Cover Design
Sarah Summerbell

The role of *Haemophilus parasuis* in nursery mortality

Simone Oliveira, DVM, MS, PhD candidate, Carlos Pijoan, DVM, PhD, Robert Morrison, DVM, PhD, MBA

Haemophilus parasuis has recently re-emerged as one of the major causes of nursery mortality. The exact factors that have contributed to the increase in the incidence of *H. parasuis* infections in the nursery are not clear. However, some hypotheses have been proposed. Segregated early weaning (SEW)[1] and PRRSV co-infections[2] are two of the factors that have been suggested to be associated with the increase in mortality rates due to *H. parasuis* infections. Control of *H. parasuis* depends on understanding the epidemiology of this agent in affected herds and on defining its role in nursery mortality as a primary or a secondary agent.

Haemophilus parasuis as a primary agent in nursery mortality

In 1997 Dr. Carlos Pijoan proposed a very interesting hypothesis regarding the effects of modern swine practices such as SEW, for example, in the development of disease in the nursery.[1] This hypothesis states that the reduction of the weaning age results in the colonization of only a few pigs with the pathogenic bacterial strains responsible for causing disease in the herd. These colonized pigs are healthy carriers of the pathogenic strains, which later act as the source of infection for naïve pigs in the nursery. The onset of disease is generally observed between four and six weeks in the nursery, when the levels of maternal immunity are no longer protective. This hypothesis has been tested and supported for two major bacterial agents: *Streptococcus suis*[3] and *H. parasuis*.[4] Another factor that has contributed to the increase in nursery mortality is the use of three-site production systems; this has delayed the spread of these organisms in the presence of protective maternal immunity.[1]

The role of *H. parasuis* as a primary agent involved in nursery mortality has been well documented.[4] The severity of clinical signs and lesions associated with *H. parasuis* infections, as well as the age of affected pigs, depends on the health status of the herd and on the virulence of the strains involved in disease. In naïve populations, mortality due to *H. parasuis* affects especially young pigs. In some herds, the onset of disease can be as early as one week after weaning, which indicates a deficiency in maternal immunity. In the majority of the affected herds, however, the peak of infection is generally at four to six weeks after weaning, when the levels of maternal immunity are no longer protective. Disease in susceptible herds is clinically characterized by the development of high fever (107°F), respiratory distress, swollen joints, and central nervous systems signs. Lesions are generally severe, characterized by the presence of fibrinous exudate in the pericardium, pleura, peritoneum, synovia and meninges. Conventional herds tend to experience a delay in *H. parasuis* infections and in many cases only older pigs are affected. Sporadic arthritis and mortality in adult populations involving sows and boars have been associated with *H. parasuis* infections. Another outcome associated with *H. parasuis* infections in stable herds is the development of pneumonia. Pneumonia caused by *H. parasuis* is characterized by the consolidation of the antero-ventral sections of the lung, with the presence of purulent exudate in the airways.

The control of *H. parasuis* as a primary agent causing disease in the nursery should be based on the characterization of the prevalent strains isolated from systemic sites. A significant number of isolates should be characterized and compared to define the best approach for disease control.[5] Serotype information can be used for the selection of commercial vaccines. Homologous protection among similar serovars is generally satisfatory.[6] Another option is to use autogenous vaccines. Genotype information is very useful for the characterization and comparison of *H. parasuis* strains to be included in autogenous vaccines.[5] After genotyping, dendrograms can be constructed and the detection of clusters of prevalent strains can easily be performed. The autogenous vaccine should contain at least one representative of each prevalent cluster of strains.

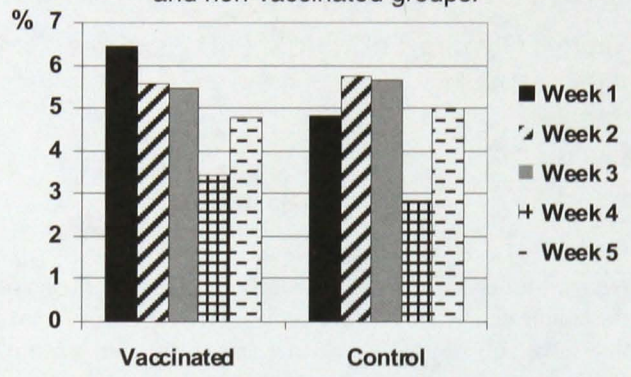
Another important factor to be considered for the successful control of *H. parasuis* infections is the schedule of vaccination. The decision regarding the timing of vaccination depends on the epidemiology of the infections in each specific herd. In herds experiencing an early onset of disease (after weaning), piglets can be vaccinated at processing and at weaning. In herds that are experiencing a delay in the peak of mortality (around four to six weeks in the nursery), the vaccination can be postponed to weaning and two weeks later.

Haemophilus parasuis as a secondary agent in nursery mortality

The role of *H. parasuis* as a secondary agent in nursery mortality has not been completely established. However, field observations suggest an increase in the occurrence of polyserositis in PRRS infected pigs.[2] PRRS virus infection may predispose pigs to secondary infections by impairing the mechanism of non-specific respiratory defenses through the destruction of alveolar macrophages and by the induction of inflammation in the nasal mucosa.[7] Solano et al. (1997) first studied the interaction between PRRS virus and *H. parasuis*.[7] According to the authors, the infection of susceptible pigs with PRRS virus followed by *H. parasuis* intratracheal challenge did not increase bacterial polyserositis as compared to the group inoculated only with *H. parasuis*. Later studies on the effect of PRRS virus infection on the clearance of *H. parasuis* by porcine alveolar macrophages (PAMs) showed that *H. parasuis* uptake was not affected in PAMs from piglets that were exposed to the virus for short periods of time. However, in the later stages of infection (168 and 216 h), PRRSV infected pigs had a marked decrease in the functional ability of their PAMs to kill *H. parasuis* and produce superoxide anion after PAM stimulation.[8] The ultrastructural characterization of PAMs infected in vitro with PRRS virus and *H. parasuis* demonstrated that dually infected cells showed increased numbers of phagosomes and phagolysosomes, however there was no significant effect of PRRS virus infection on *H. parasuis* phagocytosis.[9]

Although the interaction of PRRS virus and *H. parasuis* is not clear in the literature, field experiences have suggested that the mortality associated with *H. parasuis* infection can be increased when PRRS is active in the nursery.[2] Furthermore, it has been observed that control of *H. parasuis* infections is dependent on the stabilization of PRRS virus infections in the herd. We have evaluated the effects of vaccination against *H. parasuis* in a herd that had recently had clinical PRRS in the sow herds. Nursery mortality had increased over the last six months and had reached 10% after the PRRS virus outbreaks. Thirty-two nursery pigs were necropsied and *Haemophilus parasuis* was detected by isolation and/or PCR in 24 of these pigs (75%). Each week, six groups of vaccinated pigs and two groups of non-vaccinated controls were followed and the mortality was recorded. This procedure was repeated over five weeks. Results showed that there was no difference in mortality between vaccinated and control groups (**Figure 1**). There was a reduction in mortality rates that coincided with stabilization of PRRS virus infections in the sow farms and in the nurseries. These results suggested that the high mortality observed in the herd was associated with the interaction between PRRS virus and *H. parasuis*.

Figure 1: Mortality rates observed in vaccinated and non-vaccinated groups.



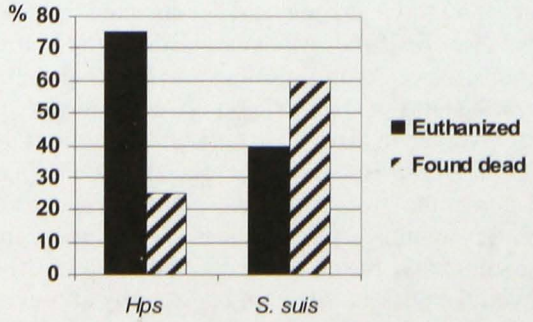
Differential diagnosis of *H. parasuis* infections

The differential diagnosis of *H. parasuis* should include *S. suis*, *Erysipelothrix rhusiopathiae*, and *Mycoplasma hyorhinis*.[10] Misdiagnosis of *H. parasuis* systemic infections are frequent and in several cases. *S. suis* is frequently isolated from cases of polyserositis, especially in dead pigs. In a recent trial, we isolated *H. parasuis* from euthanized, clinically affected pigs (75%) much more frequently than from dead pigs in the same room (25%, **Figure 2**). Conversely, *S. suis* was isolated from 60% of the pigs that were found dead. A mixed infection by *H. parasuis* and *S. suis* was observed in two euthanized pigs which indicates that these organisms can infect nursery pigs at the same time.

Final considerations

The control of *H. parasuis* depends on the definition of the role of this organism in nursery mortality. As discussed in this article, *H. parasuis* can play either a primary or a secondary role in nursery disease. In nurseries primarily affected by *H. parasuis*, control can be attempted by vaccination and/or treatment. However, in nurseries affected by PRRS virus, the control of secondary agents such as

Figure 2: Comparison of *Haemophilus parasuis* and *Streptococcus suis* isolation from pigs euthanized or found dead.



H. parasuis and *S. suis* may be dependent on the stabilization of the circulation of the virus. The isolation of *S. suis* from dead pigs showing polyserositis should be critically evaluated, since this organisms tends to rapidly overgrow *H. parasuis* in the carcass after death.

References

1. Pijoan C., Torremorell M., Solano G. (1997) Colonization patterns by the bacterial flora of young pigs. *Proc Am Assoc Swine Practt* 463-464.
2. Collins, J.E. (1991) Newly recognized respiratory syndrome in North American Swine Herds. *Am. Assoc. Swine Pract.* 3:7-11.
3. Torremorell M, Pijoan C, Dee S. (1999) Experimental exposure of young pigs using a pathogenic strain of Streptococcus suis serotype 2 and evaluation of this method for disease prevention. *Can. J. Vet. Res.* 63:269-275
4. Oliveira S, Batista L, Torremorell M, Pijoan C. (2001) Experimental colonization of piglets and gilts with systemic strains of *Haemophilus parasuis* and Streptococcus suis to prevent disease. *Can. J. Vet. Res.* 65(3):161-7.
5. Oliveira, S and Pijoan, C. (2001) *Haemophilus parasuis*: Improvement of diagnosis by a molecular-based technique. *Proceedings of the 32ND American Association of Swine Veterinarians (AASV) Annual Meeting,* Nashville, p. 479.
6. Oliveira S and Pijoan C. (2002) Use of an uncapsulated *Haemophilus parasuis* vaccine in pigs. *Proceedings of the 17th Congress of the International Pig Veterinary Society,* Ames, v 2, p. 382.
7. Solano G. I., Segales J., Collins J. E., Molitor T. W,, Pijoan C. (1997) Porcine reproductive and respiratory syndrome virus (PRRSv) interaction with *Haemophilus parasuis. Vet. Microbiol.* 55(1-4):247-57.
8. Solano CI, Bautista E, Molitor T, Segales J, Pijoan C. (1998) Effect of Porcine reproductive and respiratory syndrome virus infection on the clearance of *Haemophilus parasuis* by porcine alveolar macrophages. *Can J Vet Res,* 62:251-256.
9. Segales J, Domingo M, Balasch M, Solano G, Pijoan C. (1998) Ultrastructural study of porcine alveolar macrophage infected in vitro with porcine reproductive and respiratory syndrome (PRRS) virus, with and without *Haemophilus parasuis. J Comp Path,* 118-231-243.
10. Barbara E. Straw, Sylvie D Allaire, William L. Mengeling, David J. Taylor (ed) (1999) *Diseases of Swine.* 8ed. Iowa State Press, 1256 pages.