Asociación Mexicana de Médicos Veterinarios Especialistas en Cerdos
XXXVI Congreso Nacional Querétaro 2001
Julio 25 – 29 de 2001

A SUMMARY OF PRRSV RESEARCH FROM OUR GROUP

SA Dee, S Otake, M Bierk, KD Rossow, J Deen, C Pijoan and TW Molitor
University of Minnesota College of Veterinary Medicine
Center for Swine Disease Eradication

Porcine reproductive and respiratory syndrome virus (PRRSV) is an economically significant pathogen of the global swine industry. Published data have documented losses averaging $229 US per inventoried sow on an annual basis secondary to elevated mortality rates, reduced growth, and excessive vaccination and medication costs [1]. While a number of control strategies have been developed, they have produced inconsistent results and therefore, cannot be applied to all farms.

The ability of the PRRSV to produce persistent infection, and spread vertically and horizontally from infected dams to both in utero and to offspring prior to weaning results in the formation of infected litters that carry virus into the nursery [2,3]. Recent work from our group at the University of Minnesota College of Veterinary Medicine has centered on understanding how PRRSV is maintained within infected breeding herds and how the virus can be transmitted from pig to pig. The following is a summary of conclusions we have drawn following analysis of the results of the 6 studies:

1.The prevalence of breeding animals that harbor PRRRSV in endemically infected field populations is low [4].

A diagnostic investigation of 60 adult swine (45 sows and 15 boars) from a 1500 sow commercial swine farm indicated that only 1 of 60 actually harbored infectious virus. PRRSV was isolated from the lymph nodes of a sow having an ELISA s/p ratio of 1.20. In an effort to eradicate PRRS, the breeding herd had been closed to the introduction of replacement gilts for six months at the initiation of the study and nine months at the time the infected sow was identified. This virus proved to be both infectious and virulent, as administration of the virus to PRRSV-naïve sows at 95-days of gestation resulted in the formation of clinically affected as well as clinically normal, infected litters (5).

2. Persistently infected sows can shed PRRSV over extended periods [5].

Experimental infection of PRRSV-naïve sows resulted in the formation of sows that harbored virus for up to three months following infection. A subset of these animals (3/12) was also able to shed PRRSV to naïve contact controls from approximately 45 to 86 days post-infection. Infected and naïve contact sows were separated by a fence, allowing for nose-to-nose contact. All index sows (n =12) possessed numerous tissue sites that were positive for PRRSV-nucleic

acid by PCR at necropsy; however, virus was not detected consistently in any specific site. Microscopic lesions of PRRSV-infection (germinal centers with the presence of blast-lymphocytes) were also detected in multiple sites.

3.Tonsil biopsy is an ineffective ante-mortem test for the detection of PRRSV-carriers [6].

While PRRSV has been reported to persist in tonsil, the actual collection of tonsil biopsies did not result in successful identification of all infected animals. Reasons for this conclusion included the inability to consistently collect tonsil tissue and the ability of the virus to persist in anatomical sites other than tonsil. Finally, the procedure was both labor-intensive and harmful to the animal. While oropharyngeal scrapings may be a better test for the collection of tonsil tissue, our studies demonstrated that not all sows harbored virus in this site.

4.Genetically diverse strains of PRRSV can co-exist in endemically infected farms [7].

Based on the sequencing of the open reading frame 5 region of the PRRSV, we detected three different strains of the virus within one 1500-sow herd. These viruses were determined to be independent entities, and not the result of mutation or a recombination event. These viruses were all detected throughout the various stages of production. Whether commercial vaccines will be able to cross-protect against all three strains, or if gilt acclimation projects can insure consistent exposure and immunity across all strains is unknown at this time.

5.Mechanical transmission of PRRSV can occur by contaminated needles, coveralls and boots [8,9].

Within infected farms, it is important to minimize the spread of PRRSV from pig to pig. Work from our group has demonstrated that it appears to be possible to infect PRRSV-naïve pigs following exposure to virus-contaminated clothing and footwear. Virus can also be spread through the act of injection, if the needle and syringe has recently been in contact with a viremic pig. These data support the frequent changing of needles between animals within PRRSV-infected farms, particularly within production stages such as the nursery, where PRRSV viremia is known to be especially active. To compromise and balance cost, labor and time, producers in the US are changing needles every 10 sows, and between every litter of piglets, and every pen of nursery or finishing pigs. Frequent changing of clothing and footwear also appears to be important, as our studies identified infectious PRRSV on contaminated coveralls and boots. Finally, infectious virus was also recovered from the hands of personnel following exposure to experimentally infected pigs, but not immediately following the washing of the hands with commercial soap and warm water.

6.Aerosol spread is an unlikely route of PRRSV transmission into naïve farms [10].

A recently completed study indicated the inability to demonstrate aerosol transmission of PRRSV under controlled field conditions. Using an abandoned commercial swine facility, 200 PRRSV-naïve gilts were introduced to the facility. On a specific day, 140 were infected intranasally with a field strain of PRRSV, while the 60 non-infected animals were dispersed evenly between all pens to serve as contact controls. In the last pen within the facility, a positive control group of 10 non-infected pigs were separated from animal contact by a pen space of approximately 2.3 meters. The commercial facility was a mechanically ventilated finishing facility with three exhaust fans on the side of each wall. On days five, six, and 7 post-infection of the 140 gilts, two livestock trailers, each containing 10 15 kg piglets were placed along side the opposite sides of the building. Trailer A was parked 1 meter from one set of fans, while the other trailer was parked 30 meters from the other set of fans. Pigs were housed in trailers for 72 consecutive hours, then removed to separate facilities on the same site and tested for the presence of PRRSV and PRRSV-antibodies at seven and 14 days following the three-day exposure to the exhaust fans. During the exposure period, it was confirmed that air from the exhaust fans was entering the animal airspace in trailer A. Evidence of PRRSV infection was detected in the index animals, the contact controls, and the positive control groups, with all animals becoming PCR and ELISA positive over the 14-day testing period. All 20 sentinel pigs were negative for all tests at both testing dates.

Based on these studies, it appears that the biggest risk to successful control or eradication of PRRS is the persistently infected sow. While we have clarified a number of issues, many questions still exist, such as:

1.How can we best eliminate subpopulations of PRRSV-naïve sows in endemically infected breeding herds?
2.What is the proper length of time to close an infected breeding herd to outside replacement animals, in order to minimize the risk of developing persistently infected breeding stock?
3.Can we identify non-porcine routes of PRRSV entry into naïve farms to better protect herds that choose to eradicate the disease?

At the University of Minnesota Center for Swine Disease Eradication, we are in the process of initiating studies to answer these questions. We are collaborating with leaders in the swine industry and developing teams of experts in order to attack this problem using all available resources.

References

1. Dee SA, Joo HS, Polson DD and Marsh WE. Evaluation of the effects of nursery depopulation on the profitability of 34 pig farms. *Vet Rec*. 1997. 140:498-500.
2. Christianson WT, Choi CS, Collins JE, et. al. 1993. Pathogenesis of PRRS virus infection in mid-gestation sows and fetuses. *Can J Vet Res.* 57:262-268.
3.Dee SA.and Philips RE. Use of polymerase chain reaction to detect vertical transmission of PRRS virus in piglets from gilt litters. *Swine Health and Production*.1999. 7:237-239.
4. Bierk MD**,** Dee SA**,** Rossow KD, Collins JE, Pijoan C, Guedes MI, and Molitor TW. A diagnostic investigation of chronic PRRS virus infection in a swine breeding herd. *Vet Rec* (accepted for publication).
5. Bierk MD, Dee SA, Rossow KD, Collins JE, Guedes MI, and Molitor TW. Transmission of PRRS virus from persistently infected sows to contact controls. *Can J Vet Res* (submitted for publication).
6.Bierk MD, Dee SA, Rossow KD, Guedes MI, and Molitor TW. Experiences with tonsil biopsy as an ante-mortem diagnostic test for detecting PRRS virus infection in breeding swine. *Journal of Swine Health and Production.* 2000. 8(6):279-282.
7.Dee SA, Torremorell M, Rossow K, Otake S, and Faaberg K. Identification of genetically diverse sequences of PRRSV in a swine herd. *Can J Vet Res* (accepted for publication).
8.Otake S, Dee SA, Rossow KD, Deen J, Joo HS, and Molitor TW. Transmission of PRRSV by needles. *Vet Rec* (submitted for publication).
9.Otake S, Dee SA, Rossow KD, Deen J, Joo HS, and Molitor TW. Transmission of PRRSV by contaminated fomites and personnel and the impact of sanitation protocols. *Journal of Swine Health and Production* (submitted for publication).
10.Otake S, Dee SA, and Pijoan C. Assessment of aerosol transmission of PRRSV under controlled field conditions *Vet Rec* (submitted for publication).