

Sponsors

University of Minnesota

College of Veterinary Medicine

College of Agricultural, Food and Environmental Sciences

Extension Service

Swine Center

Virial Recombination Between Strains of Porcine Reproductive and Respiratory Syndrome Virus

Kay Faaberg¹, Shishan Yuan¹, Chris Nelsen¹, Beverly Schmitt² and Michael Murtaugh¹

¹University of Minnesota and ²National Veterinary Services Laboratories

*Study was funded by Minnesota Pork Producers Association and PIC, USA

Introduction. Porcine reproductive and respiratory syndrome virus (PRRSV) remains a significant pathogen of swine herds worldwide in spite of the availability of efficacious vaccines. In addition, some recent field isolates appear to have developed altered disease phenotypes, such as increased rates of abortion and sow mortality (1) and neurovirulence (2). The clinical evidence for the emergence of new PRRSV strains is supported by molecular analysis of 10 U.S. strains isolated in 1990-1992. (3). Significant genetic variation was observed; furthermore, the data strongly suggested that viral recombination may play a role in evolution of PRRSV. This study was implemented to provide direct experimental evidence if viral recombination may be contributing to the continued evolution of PRRSV strains.

Results. Additional molecular genetic analysis of 50 PRRSV samples, isolated within a 7 year time period from the start of PRRS, showed that an isolate from North Carolina in 1993 was the product of recombination between two strains on farms in adjacent counties. The recombinant virus had a new major envelope glycoprotein, which is thought to be responsible for attachment of the virus to macrophages.

Controlled studies on viral recombination were implemented using a continuous cell line and two cell-adapted strains of PRRSV. RT-PCR analysis on progeny virions using differential primers specific to each strain revealed that viral recombination had occurred between the two strains. Furthermore, the recombination was evident through 5 passages in culture, suggesting that some of the recombinants are able to replicate and are stable (4). Efforts are underway to ascertain if this *in vitro* evidence

can be repeated in swine macrophages, the host cell of PRRSV.

Conclusion. Viral recombination between different strains of PRRSV may play a role in the continuing evolution of this virus. Attempts to develop multivalent live virus vaccines should be analyzed for potential recombination events that may create undesirable viral progeny. Furthermore, deliberate mixing of different PRRSV strains as by simultaneous vaccination with multiple products is not advisable since it increases the likelihood of recombination and could artificially accelerate the rate of genetic change in PRRSV.

References.

1. Lager, K. M., Mengeling, W. L., Wesley, R. D., Halber, P. G., & Sorden, S. D., 1998. Acute PRRS. Proc. AASP Meet., p. 449.
2. Rossow, K. D., Shivers, J. L., Yeske, P. E., Polson, D. D., Rowland, R. R., R. Lawson, S. R. Murtaugh, M. P. Nelson, E. A., and J. E. Collins. 1998. Porcine reproductive and respiratory syndrome virus infection in neonatal pigs characterized by marked neurovirulence. Vet. Record, submitted.
3. Kapur, V., Elam, M. R., Pawlovich, T. M., & Murtaugh, M. P. 1996. Genetic variation in porcine reproductive and respiratory syndrome virus isolates in the midwestern United States. J. Gen. Virol. 77:1271-1276.
4. Recombination between North American Strains of Porcine Reproductive and Respiratory Syndrome Virus. 1998. Shishan Yuan, Chris J. Nelsen, Michael P. Murtaugh, Beverly J. Schmitt and Kay S. Faaberg, J. Gen. Virol. submitted.