

Abstract for the 9th International Veterinary Immunology Symposium (9th IVIS), Tokyo, Japan Aug. 16-20, 2010. <http://www.frc.a-u-tokyo.ac.jp/>

Immune pathways regulating swine responses to porcine reproductive and respiratory syndrome virus infections.

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Our goal is to identify pathways and genes regulating early responses to porcine reproductive and respiratory syndrome virus (PRRSV) infections by comparing pig responses to pathologic acute PRRSV infections, protective vaccinations and ineffective cross-protection against heterologous virus. PRRS, the most economically important pig disease worldwide, causes pneumonia and reproductive losses, and the highly pathogenic porcine high fever disease in China and Vietnam. For these experiments mucosal tissues [tracheobronchial lymph nodes (TBLN), cranial and caudal lung lobes, and tonsils] were harvested from pigs infected with PRRSV (MNW2B, NC Powell or VR2332), vaccinated (modified-live PRRSV Ingelvac ATP[®]) or controls. Tissues were collected between 3-6 days post infection, vaccination, or secondary challenge with homologous versus heterologous virus. Amplified RNA was labeled with Alexa Fluor[®] 555 and 647 dyes (Invitrogen) and hybridized to Swine Protein-Annotated Oligonucleotide Microarray www.pigoligoarray.org using a loop design to test for changes in gene expression between all groups. Comparisons of differentially expressed genes revealed pathways involving interferons, other cytokines and chemokines as critical for differentiating infected from vaccinated pigs. Subsequent experiments using RT-PCR of candidate genes affirmed that NC Powell infected or secondary challenged pigs had the greatest viral levels and greatest differential alterations in candidate gene expression. Final statistical and pathway analyzes are underway to identify the biological functions, candidate genes and regulatory pathways that are most significant for pathologic versus protective responses and to determine which mechanisms are subverted when cross-protection is ineffective. Supported by USDA ARS project #1265-32000-088 and NIFA PRRS CAP grant #2008-55620-19132 funds.