

SWINE HEALTH

Title: Determination of the PRRSV minor glycoproteins contribution to antigenicity and protection - **NPB Project # 15-153**

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Scientific Abstract:

Porcine reproductive and respiratory syndrome (PRRS) is one of the most economically important diseases in swine caused by porcine reproductive and respiratory syndrome virus (PRRSV). Sixty-six near complete genome sequences were obtained using metagenomic sequencing of serum samples collected in the U.S. in 2014 to explore contemporary PRRSV genetic diversity. Phylogenetic analysis found three viral lineages with a majority of strains (46) being related to the reference NADC30. Only 16 strains were more closely related to the historical U.S. strain VR2332 and commercial vaccine strains and four strains had unresolved phylogeny. Phylogenetic analysis of the genes encoding the structural glycoproteins (GP) identified four to eight distinct lineages with >87% intraclade identity. To explore the effect of the observed genetic diversity on antigenicity, the genome region encoding either GP2a-GP3-GP4 or GP5-M in strain SD95-21 were replaced with alleles from each of eight distinct PRRSV strains using reverse genetics. The GP2a-GP3-GP4 region from only four of the eight strains yielded viable recombinant virus. When viable, both GP2a-GP3-GP4 and GP5-M variably affected antigenicity. A strain-dependent significant loss in cross reactivity was variably observed by indirect immunofluorescence assays using antisera from pigs vaccinated with commercial modified-live vaccines following replacement of GP2a-GP3-GP4 or GP5-M. Significantly reduced neutralization titers were similarly measured using antisera from naturally PRRSV-exposed pigs. These results illustrate the need to consider genomic regions besides GP5 for PRRSV epidemiology and vaccination.

These research results were submitted in fulfillment of checkoff-funded research projects. This report is published directly as submitted by the project's principal investigator. This report has not been peer-reviewed.

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