

Associations Among Immunity, Viral Genetics and Reproductive Performance in Breeding Swine Infected with Porcine Reproductive and Respiratory Syndrome Virus

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Summary

Porcine reproductive and respiratory syndrome virus is notoriously variable clinically and genetically. In this study, we demonstrate that the clinical, immunological and genetic variability observed in the virus in the field are epidemiologically intercorrelated. Viruses that induced high cellular immunity caused fewer and less severe reproductive losses in pigs in the field. Viruses nevertheless evolved rapidly under field conditions and showed “quasispecies” type variability.

Introduction

Porcine Reproductive and Respiratory Syndrome Virus (PRRSv; family *Arteriviridae*) is an economically-important pathogen of domestic swine worldwide. PRRSv is noted for its genetic and antigenic variability¹⁻³. This variability is thought to contribute to the inability of vaccination to control PRRS, and to the clinical variability of PRRS in the field. The goal of this study was to quantify genetic diversity and genetic change in PRRSv over time in the field, and to explore associations between viral genetics, host immunity, and reproductive performance in PRRSv-infected swine.

Materials and Methods

Cohorts of 20 PRRSv-naïve pigs were followed from introduction onto a PRRSv-positive farm at 7 weeks of age until the first farrowing as sows. Serial blood samples were collected to monitor the development of humoral immunity (antibodies) and cellular immunity (interferon-gamma-producing, PRRSv-reactive T lymphocytes) over time. Simultaneous tonsil biopsies were collected, and PRRSv genetic material was PCR-amplified and sequenced (at the ORF5 gene) to track the genetic change in the virus. PCR products were also cloned into plasmid vectors, and individual clones were sequenced to measure within-animal genetic diversity over time.

Results

Humoral immunity developed more quickly than did cellular immunity in all swine. Cellular immune levels were highly variable among animals and over time. The virus evolved over time within cohorts, and within individual animals, with degrees of genetic change in ORF5 approaching 5% in some cases over 1 year. All animals harbored multiple PRRSv genetic variants⁴. Cellular immunity, but not humoral immunity, predicted reproductive performance in sows. Animals with strong cellular immune responses had significantly fewer piglets born dead per litter and were significantly less likely to abort than animals that failed to mount strong cellular immune responses.

Discussion

These results indicate that cellular immunity is protective against the clinical manifestations of PRRSv-infection, and that it may influence the genetic evolution and antigenic variability of the virus in the field. Cellular immunity was particularly important in protecting sows against the clinical manifestations of PRRSv. Control strategies aimed at “boosting” cellular immunity should prove profitable. Nevertheless, the high degree of genetic variability observed in animals and over time suggests that biosecurity should be given high priority, to prevent the dissemination of newly evolved viral variants.

References

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