

SWINE HEALTH

Title: An Interferon-inducible Porcine Reproductive and Respiratory Syndrome Virus Isolate – NPB #11-106

Investigator: Yan-Jin Zhang

Institution: University of Maryland, College Park, MD

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Scientific Abstract:

Porcine reproductive and respiratory syndrome (PRRS) virus (PRRSV) continues to cause substantial economic losses to the global swine industry. PRRSV appears to inhibit synthesis of type I interferons (IFNs), such as IFN- α and - β , which are critical for the innate immunity and play an important role in the modulation of adaptive immunity. An atypical PRRSV strain, A2MC2, is able to induce type I IFNs *in vitro*. In this study, full length sequence of the A2MC2 genome was determined. Sequence analysis indicated that it is highly homologous to VR-2332, the prototype of North America PRRSV genotype. An infectious clone of A2MC2 was constructed and rescued virus was able to induce interferon synthesis in infected cells. A2MC2 induction of neutralizing antibodies *in vivo* was compared with the Ingelvac PRRS modified live virus (MLV) vaccine strain and VR-2385 (a moderate virulent strain). Three-week-old pigs were exposed to these PRRSV strains via intranasal or intramuscular routes to also account for a possible effect of inoculation routes. The interferon-inducing A2MC2 resulted in earlier onset and significantly higher levels of PRRSV neutralizing antibodies than the MLV in either inoculation routes. In addition, the A2MC2-induced neutralizing antibodies were capable of neutralizing VR-2385, a heterologous strain. The pigs exposed via intranasal route had higher titers of neutralizing antibodies than those injected via intramuscular route. Macroscopic and microscopic lung lesions 14 days post-exposure indicated that A2MC2 had similar virulence *in vivo* as VR-2385. Pulmonary alveolar macrophages (PAMs) collected during the necropsy 14 days post-exposure in the A2MC2 group had higher level expression of IFN- γ than the MLV group. These results indicate that A2MC2 can be further explored for development of an improved vaccine against PRRS.

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 This report has not been peer-reviewed.

For more information contact:

National Pork Board • PO Box 9114 • Des Moines, IA 50306 USA • 800-456-7675 • Fax: 515-223-2646 • pork.org
