

ANNUAL REPORT PROJECT NC-229

PERIOD COVERED: June 2008 to November 2009

INSTITUTION OR STATION:

A. NC-229 REPRESENTATIVE:

Roman Pogranichniy, Purdue University, rmp@purdue.edu

Other PRINCIPLE LEADERS associated with the projects

B. PROGRESS OF WORK AND PRINCIPAL ACCOMPLISHMENTS:

*Per station for ALL Accomplishment = Maximum 3,000 characters including spaces;
Full NC229 report for ALL Accomplishment = Maximum 30,000 characters):*

This section focuses on intended activities, outputs, and short-term outcomes. The report should also reflect on the items that stakeholders want to know, or want to see. The accomplishments should cover only the current year of the project.

Objective 1. Elucidate the mechanisms of host-pathogen(s) interactions.

Indicate progress in the following areas.

1. Research related to pathogenesis/persistence. Cells response to virus infection. PRRSV virus inhibition by SiRNA.
2. I this project stealth RNAi antisense designed from specific PRRSV cellular receptor CD163 and co-receptor Siglec-1 (sialoadhesin, Sn, CD169) and 5'UTR region of viral genome we have demonstrated significant inhibition of PRRSV infection and spread in MARC-145 cell culture line. PRRSV infection in MARC-145 cultured cell in the absence of endogenous Sn expression resulted in enhanced cellular expression of PTEN which is indicative of negative regulation of the Akt pathway leading to cell arrest and apoptosis. However, in the presence of endogenous Sn, the activation of the Akt pathway was demonstrated by the up regulation of tissue metalloproteinase -9 (MMP-9) mRNA leading to cell growth and survival. We have shown that PRRSV infection and replication in MARC-145 cultured cells modulated the Akt signaling pathway important for both cell survival and arrest. Moreover we have demonstrated that in M145 cultured cells, the presence of PRRSV virion in the supernatant of infected cells was the determining factor in viral accumulation and concentration in the cell supernatant.

C. IMPACT AND VALUE OF RESEARCH TO STAKEHOLDERS:

Impact statements (500 characters per statement)

We proposed to do more work in vivo and in collaboration with Dr. Joan Lunney from USDA BARC obtain grant from PRRSV CAP to study PRRSV and pig response to virus infection on molecular level. More work to be reported next year.

D. PRRS PUBLICATIONS ISSUED OR “IN PRESS”

1) Refereed publications

Give full citation

2) Abstracts or Proceedings

N/A

3) Book chapters or monographs

Give full citation

E. FUNDING SOURCES FOR PRRSV RESEARCH

1) Current PRRSV – CAP

Title of proposal: Characterization of host factors that contribute to PRRS disease resistance and susceptibility

F. WORK PLANNED FOR NEXT YEAR

We are planning to characterize molecules that are up-regulated and down-regulated due to PRRSV infection in a control pigs challenge study. Tissue material (lung, tonsil, lymph node) and PMBC from experimentally PRRSV-infected animals will be tested for expression of panel mRNA. Host factors important for PRRSV infection will be identified during this future experiment.