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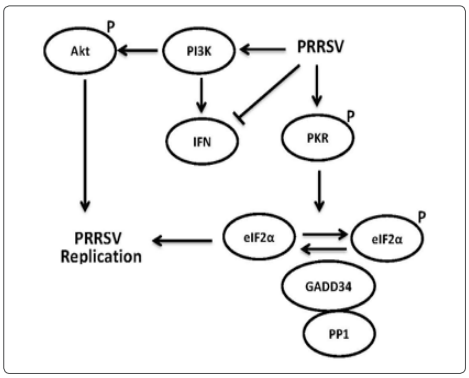
Strategies used by porcine reproductive and respiratory syndrome virus to evade host's innate immunity

Statement of the Problem: Porcine Reproductive and Respiratory Syndrome Virus (PRRSV) is a single-stranded, positive-sense RNA virus with a genome size of approximately 15 kb. PRRSV belongs to the Arteriviridae in the order of Nidovirale. PRRSV causes acute respiratory disease in neonatal and young piglets and reproductive failure in pregnant sows. The role of PRRSV nonstructural and structural proteins in modulating the transcriptional activation of type I interferon in MARC-145 cells and human cell culture system including HeLa cells and 293T cells has been described previously.



Aim: The purpose of this study is to investigate the interaction between PRRSV and host's innate factors including type I interferon, interferon-induced genes such as Protein Kinase R (PKR) and stress granules.

Methodology & Theoretical Orientation: Porcine monocyte-derived dendritic cells and MARC-145 cells were used to study the virus-host interaction. Real-time RT-PCR, RNA silencing, western blotting, ELISA, and TCID50 were used to understand the strategies used by PRRSV to evade host's innate defense mechanisms.

Findings: PRRSV activates the transcription of type I interferon in porcine monocyte-derived dendritic cells. However, PRRSV interferes with the translation of type I interferon in these cells partly through cytopathogenicity since UV and heat-inactivated viruses lose their ability to interfere with the induction of type I interferon by porcine Poly I:C. PRRSV transiently activates PKR during early infection. PKR is not a major contributor to the phosphorylation of eIF-2**α**, but it plays a pro-viral role in PRRSV replication. PRRSV fails to induce stress granules in infected MARC-145 cell. PRRSV interferes with the formation of stress granules formed by treatment with arsenite sodium.

Conclusion & Significance: PRRSV has evolved multiple strategies to evade host's innate immunity. A better understanding of the virus-host interaction may provide novel insights on the design of innovative strategies to control and prevent the devastating disease in pigs.

Biography

Xiuqing Wang has received her PhD degree in Virology/Pathobiology at the University of Connecticut, USA. She has completed her Postdoctoral degree in Dr. Stephen Dewhurst's Laboratory in the Department of Microbiology and Immunology at the University of Rochester Medical Center.

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