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所在系：预防兽医系 职称：教授 导师类别：博士生导师

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Education

2003-2008 Ph.D in Animal Virology, University of Minnesota-Twin Cities

2000-2003 M.S in Veterinary Microbiology, China Agricultural University

1996-2000 B.A in Veterinary Medicine, China Agricultural University

Positions and Employment

2009-2014 Postdoctoral Fellow with John W. Wills, Ph.D., in the Department of Microbiology and Immunology, Penn State University

2014-now Professor, Department of Preventive Veterinary Medicine, China Agricultural University

Teaching

2014- now “Science of Virology” to Graduate Students

2015-now “Preventive Veterinary Medicine Seminar” to Ph.D students

2015-now “Advances in Veterinary Science” to Ph.D students

Honors

2014 Outstanding postdoctoral scholar, Penn State College of Medicine

2014 NIH K99 Award

Commercialized patent

“PRRS viruses, infectious clones, mutants thereof and methods of use”. Kay S. Faaberg, **Jun Han**, Gongping Liu and Yue Wang.

Research interest

Molecular mechanisms of herpesvirus cell-cell transmission. Alpha-herpesviruses cause a variety of important human and animal diseases but there is no effective vaccine currently available in particular for herpes simplex virus (HSV) and porcine pseudorabies virus (PRV). A major means by which this class of viruses disseminate in vivo iscell-to-cell spread (CCS), a strategy that is highly related to viral pathogenesis and is commonly used by many DNA and RNA viruses for transmission within their hosts. My long-term goal is to elucidate how these viruses conduct cell-cell transmission so that better strategies and control measures can eventually be found

to stop the spread of infections. We use glycoproteins gE/gI as a start point and focus on addressing four essential questions: (1) How is the gE/gI function regulated by tegument proteins? (2) how does the gE/gI heterodimer crosstalk to the viral core fusion machinery? (3) What are molecular determinants in gE and gI required for CCS and syncytia formation? (4) What are the cellular factors required for the function of gE/gI?

Molecular biology and pathogenesis of HP-PRRSV. Porcine reproductive and respiratory syndrome virus (PRRSV), a positive-stranded RNA virus in the family Arteriviridae, currently remains a major threat to the worldwide pork production. Rapid evolution of this virus in the field has led to emergence of many highly virulent strains, including the most recent Chinese highly pathogenic PRRSV(HP-PRRSV) that has been plaquing the Chinese swine industry for the past 10 years. This property of PRRSV renders the classic vaccines inefficient against challenge by heterologous strains. PRRSV also has evolved strategies for strongly inhibiting host innate immune responses and resulting in delayed, low level induction of neutralizing antibodies and CTL responses. Down-regulation of host immunity is thought to prevent clearance of the virus in an efficient and timely manner, leading to persistent infections in swineherds. Current studies of viral proteins have just begun to reveal some of the evasion mechanisms of PRRSV, but most details await to be discovered. In addition, PRRSV uses a complex mechanism for the entry process. In stead of one or two proteins that are commonly required for other RNA viruses, PRRSV entryrequiresat least three proteins (GP2, GP3 and GP4). The exact molecular mechanism remains poorly understood, such as how the virus-cell fusion process is regulated and coordinated. The complexity of this process is reminiscent of herpesviruses, which also use multiple glycoproteins for virus entry. Moreover, the Chinese HP-PRRSV appears to have expanded tropism as it was found to infect multiple types of cells and tissues, indicating PRRSV may use more than one cellular receptor in vivo.

Extramural funding

“Molecular mechanisms of nonstructural protein 2 (nsp2) in regulation

of PRRSV sgmRNA synthesis”, NSF, 31472189, 2015-2018, Han (PI).

“Screening for and molecular mechanisms of PRRSV proteins that induce immunosuppression and antagonize host immune signaling transduction”, National “973” program subproject, 2014CB542702, 2014-2018, Han (PI).