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# ABSTRACT

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immunity by RNA interference (RNAi). Until recently, however, it was unclear whether the mammalian RNAi pathway has a natural antiviral function. In a paper published in October 2013, my lab has presented two main lines of evidence to support a natural antiviral function of RNAi in mammals (Li et al., *Science* 342:231). First, we detected abundant production of canonical viral siRNAs during mouse infection by Nodamura virus (NoV), a mosquito-transmissible positive-strand RNA virus. Second, we found that without viral suppression of RNAi by the B2 protein of NoV, suckling mice are able to launch an antiviral RNAi response sufficiently potent to terminate lethal viral infection. Using the suckling mouse model for NoV infection, we have recently characterized the function and mechanism of antiviral RNAi in mammals and the in vivo mechanism of RNAi suppression by the viral B2 protein. Results from these recent studies and other related studies will be presented.

**S012 Exploring genetic control of swine responses to viral diseases**. Joan Lunney (ARS USDA), Bob Rowland and Benjamin Trible (Kansas State University), Igseo Choi and Samuel Abrams (ARS, USDA), Carlos Souza (Embrapa Pesca e Aquicultura), James Reecy, Eric Fritz-Waters, James Koltes, Chris Eisley, Christopher Tuggle, Andrew Hess, Jenelle Dunkelberger and Jack Dekkers (Iowa State University), Nicholas Boddicker (Genesus, Inc.), Juan Steibel and Catherine Ernst (Michigan State University), Le Luo Guan, Hua Bao, Arun Kommadath, Paul Stothard and Graham Plastow (University of Alberta) and Andrea Ladinig and John CS Harding (University of Saskatchewan)

Our goal is to understand genomic control of viral disease responses focusing on the economically most important disease of pigs, porcine reproductive and respiratory syndrome (PRRS) (annual losses of $664M). The PRRS Host Genetics Consortium (PHGC) was established to combine efforts of scientists from university, government and commercial pig genetics and animal health companies to assess the role of genetics in determining pig resistance/ susceptibility to PRRS virus (PRRSV) infection, pathology and growth effects. We utilized a nursery pig PRRSV infection model with deep sampling for phenotypic analyses, extensive genotyping (60K SNPchip) and a shared database http://www.animalgenome.org/lunney/. We have completed 15 trials using ~200 PRRSV-infected pigs each and identified a genomic region on SSC4 which has a significant impact on variation in viral load and growth response following challenge with each of 2 different PRRSV isolates. More recent trials involve complex challenges (PRRSV and porcine circovirus) combined with PRRS vaccines, as well as field trials; each comparing pigs with different SSC4 haplotypes. To address disease resistance mechanisms we probed serum protein expression (antibody and cytokine) and the blood transcriptome (using microarrays and RNAseq) of PHGC pigs. We have verified proteins and genes that are differentially expressed in PRRS resistant versus susceptible pigs and are probing this data for alternate control and regulatory networks. This data will help us identify new resistance pathways that may be used for new vaccines and biotherapeutics. An alternate gilt PRRSV infection model has been established to determine the effects of third trimester infection on fetal development and viability. Using deep phenotypic analyses and genotyping of gilts and fetuses, new predictors of PRRS severity in gilts and fetuses are being identified. Support: US National Pork Board, USDA ARS and NIFA, Genome Canada, Genome Alberta, Genome Prairie, pig breeding companies.

**S013 Editing miRNAs target sequence with SNPs for animal breeding**. Zhiying Zhang (Northwest A&F University)

miRNAs are small non-coding RNAs and emerge as key regulators for many developmental processes. The mechanism that miRNAs regulate target gene expression is either affecting target gene mRNA stability or inhibiting mRNA translation upon binding mRNA 3'UTR target site. Previous studies demonstrated that SNPs identified within either miRNAs or their target sequences jeopardized