A Comparison of the Genetic Factors Influencing Host Response to Infection with One of Two Isolates of Porcine Reproductive and Respiratory Syndrome Virus

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Summary and Implications

Host genetic differences in viral load (VL) and weight gain (WG) during porcine reproductive and respiratory syndrome virus (PRRSV) challenge were assessed for thirteen trials of ~200 commercial crossbred piglets each, from several different commercial suppliers. Piglets were experimentally infected with PRRSV isolates NVSL-97-7895 (NVSL) or KS-2006-72109 (KS06). VL and WG were moderately heritable and were antagonistically related for both virus isolates. The genetic correlation of host response to NVSL with host response to KS06 was high for both VL and WG. Consistent with previous findings, animals that were heterozygous (AB) for the WUR10000125 (WUR) marker on Chromosome 4 (SSC4) had significantly lower VL than their AA counterparts when infected with either virus isolate; however, a significant increase in WG was only observed when piglets were infected with the NVSL isolate. These results suggest that selecting for increased resistance or reduced susceptibility to PRRSV may be effective across virus isolates. Selecting for the AB genotype for WUR is expected to reduce VL across PRRSV isolates but its effect on WG during infection may differ between virus isolates.

Introduction

Porcine reproductive and respiratory syndrome (PRRS) is the most costly disease to the North American pork industry; vaccines, biosecurity measures, and proposed methods for eradication have had limited success. The aim of the PRRS Host Genetics Consortium (PHGC) is to identify genomic markers and pathways associated with host response to PRRSV, which could be used for genetic selection of pigs for increased resistance or reduced susceptibility to PRRSV infection.

Boddicker et al. (2012) identified a SNP on SSC4, WUR, for which the favorable allele (B) was associated with reduced viral load (VL) and increased weight gain (WG) under infection with the NVSL-97-7895 (NVSL) PRRSV isolate. It was hypothesized that selection for increased resistance or reduced susceptibility to one PRRSV isolate will result in increased resistance or reduced susceptibility to other PRRSV isolates. To test this hypothesis, the PRRSV isolate KS-2006-72109 (KS06), which is genetically distinct from NVSL, was used to experimentally infect piglets using the same challenge model. The objectives of this study were to 1) estimate genetic parameters and the effects of WUR on VL and WG of piglets experimentally infected with either the NVSL or KS06 PRRSV isolates, and 2) estimate the genetic correlations of pig responses (VL and WG) between the KS06 and NVSL PRRSV infections, in order to assess the feasibility of genetic selection of pigs with increased resistance or reduced susceptibility to PRRSV that is not dependent on virus isolate and the usefulness of WUR for marker assisted selection.

Materials and Methods

This study used data from nine PRRSV infection trials with NVSL and four trials with KS06, which encompassed multiple different genetic backgrounds. Approximately 200 commercial crossbred piglets per trial were experimentally infected intramuscularly and intra-nasally with 10^5 tissue culture infectious $dose_{50}$ of PRRSV isolate at 28-35 days of age. Blood samples were collected at 0, 4, 7, 11, 14, 21, 28, 35, and 42 days post infection (dpi). Body weight was collected weekly from infection to 42 dpi. Viremia was measured using a qPCR assay for PRRSV RNA, and VL was defined as the area under the curve of Log viremia from 0-21 dpi. WG was defined as weight gain from infection to the end of the trial. Pigs were genotyped using the 60K SNP chip, which includes the WUR SNP.

Heritabilities and the genetic correlation between VL and WG were estimated using pedigree information in ASREML 3.0, using the following model: parity nested within trial as a fixed effect, age and weight at infection as covariates, and animal, litter, and pen nested within trial as random effects. WUR genotype was additionally fitted as a fixed effect when estimating its effect on VL and WG. Genetic correlations for VL and WG between isolates were estimated with the same model but using marker-based relationships since no pedigree was available to connect pigs in the NVSL and KS06 trials.

Results and Discussion

Heritability of VL was 0.31±0.12 for NVSL and 0.40±0.09 for KS06; heritability of WG was 0.30±0.09 for NVSL and 0.24±0.11 for KS06. VL and WG had negative phenotypic (-0.25±0.03 for NVSL; -0.17±0.04 for KS06) and genetic (-0.32±0.23 for NVSL; -0.47±0.20 for KS06)

correlations. Including WUR genotype as a fixed effect showed that AB pigs had lower VL than AA pigs for both isolates (-4.6±0.4 units for NVSL, $p<0.0001$; -3.6±0.7 units for KS06, $p<0.0001$), and higher WG, although the latter was significant only for pigs infected with NVSL (+2.0±0.2 kg, $p<0.0001$ for NVSL; +0.6±0.4 kg for KS06, $p=0.11$).

The genetic correlation of response to NVSL with response to KS06 was high for both VL (0.95±0.20) and WG (0.78±0.28). Consistent with the WUR effect estimates, adding WUR as a fixed effect reduced the estimate of the genetic correlation for VL (0.8±0.25) but hardly affected the estimate of the genetic correlation for WG (0.8±0.30).

These results suggest that selecting for increased resistance or reduced susceptibility to one PRRSV isolate will increase resistance or reduce susceptibility to another PRRSV isolate. Selecting for the AB genotype for WUR is expected to reduce VL across PRRSV isolates but its effect on WG during infection may differ between virus isolates.

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