Genetic Parameters and Effect of WUR Genotype on Piglet Response to Co-Infection with PRRS and PCV2b, with or without Vaccination for PRRS

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Summary and Implications

Commercial crossbred nursery piglets were either vaccinated or not using a modified live porcine reproductive and respiratory syndrome (PRRS) virus vaccine and all pigs were co-infected with PRRS virus (PRRSV) and porcine circovirus type 2b (PCV2b) 28 days later. Genetic correlations indicate that traits associated with primary exposure to PRRSV infection (PRRS viral load (VL) of vaccinated pigs prior to co-infection and PRRS VL of non-vaccinated pigs post co-infection) are genetically the same trait. The WUR single nucleotide polymorphism on chromosome 4, previously associated with reduced PRRS VL under PRRSV-only infection, was associated with significantly reduced PRRS VL following vaccination and co-infection (for non-vaccinated pigs), but also with reduced PCV2b VL of vaccinated pigs. These results indicate a significant effect of WUR genotype on PRRS VL upon primary PRRS exposure, whether in a PRRSV-only or PRRS and PCV2b co-infected population, but also with PCV2b VL of vaccinated pigs under PRRS and PCV2b co-infection.

Introduction

PRRS is one of the most costly diseases to the US pork industry. A hallmark of PRRSV-infection is suppression of the immune response, resulting in increased susceptibility of co-infection with other diseases. When pigs are not vaccinated against PCV2b, co-infection with PRRSV and PCV2b is commonly observed in the field. Under co-infection, PRRSV increases the replication of PCV2b, enhancing clinical signs, symptoms, and mortality.

In our lab, Boddicker and others identified that the "B" allele for the WUR single nucleotide polymorphism (SNP) on chromosome 4 was associated with increased average daily gain (ADG) and reduced PRRS viral load (VL) under PRRSV-only infection. However, the effect of WUR on VL and ADG upon co-infection with other diseases is not yet known, nor is its effect upon vaccination for PRRS. Therefore, objectives of this research were to estimate genetic parameters and to estimate the effect of WUR genotype on response of commercial nursery pigs to vaccination for PRRS and co-infection with PRRS and PCV2b.

Materials and Methods

Data used included 396 commercial crossbred pigs from two PRRS Host Genetics Consortium trials. All pigs were from the same genetic supplier, where they were pre-selected based on genotype for the WUR SNP; half had the AA genotype and half had the AB genotype, since the "B" allele is completely dominant to "A". At weaning, pigs were sent to Kansas State University and randomly sorted into two rooms. All pigs in one room were vaccinated for PRRS using a modified live vaccine and 28 days later, pigs in both rooms were co-infected with field isolates of PRRSV and PCV2b. Pigs were then followed for 42 days and genotyped using the 80K Porcine BeadChip. PRRS VL after vaccination and post co-infection and PCV2b VL were calculated as area under the curve of serum viremia from -28 to 0, 0 to 21, and 0 to 42 days post co-infection, respectively. All results were generated using ASReml 3.0. Genetic parameters were estimated by fitting multivariate animal models where each trait by vaccination status was considered as a separate trait. Univariate mixed models were used to estimate the effect of WUR genotype as a fixed effect for vaccinated (Vx) and non-vaccinated pigs (Non-Vx) pigs separately for each trait.

Results and Discussion

Heritability estimates following vaccination were 0.31, 0.07, and 0.10 for ADG Non-Vx, ADG Vx, and PRRS VL Vx, respectively. During the co-infection period, heritability estimates were slightly higher at 0.53, 0.57, 0.56, 0.20, 0.18, and 0.15 for ADG Non-Vx, ADG Vx, PRRS VL Non-Vx, PRRS VL Vx, PCV2b VL Non-Vx, and PCV2b VL Vx, respectively. Standard errors ranged from 0.14 to 0.22. A strong, positive genetic correlation (0.95±1.01) was observed for PRRS VL post-vaccination with PRRS VL Non-Vx, suggesting that these traits, both representative of primary PRRSV-exposure, are genetically the same trait. Similarly, the strong positive genetic correlation of PCV2b VL Non-Vx with PCV2b VL Vx (0.92±0.50) indicates that PCV2b VL of Non-Vx pigs is the same trait as PCV2b VL of Vx pigs. For ADG, the strongest genetic correlations

were observed between Vx and Non-Vx groups within an infection period: i.e. Non-Vx and Vx prior to co-infection (0.77±1.08) and Non-Vx and Vx post co-infection (0.74±0.32).

Prior to co-infection, Vx AB pigs had significantly lower PRRS VL (P=0.03) and significantly greater ADG (P=0.001) than AA pigs. Post co-infection, Vx AB pigs also had significantly lower PCV2b VL (P=0.01) and a tendency (P=0.07) towards lower PRRS VL. During the co-infection period, AB Non-Vx pigs had significantly (P<0.001) lower PRRS VL than AA Non-Vx pigs. No evidence of a WUR effect was detected for ADG of Non-Vx (P=0.71) or Vx (P=0.53) pigs post co-infection.

Together, these results suggest that selection for improved performance under co-infection of PRRS and PCV2b is possible. Furthermore, the high genetic correlation between PRRS VL following vaccination and PRRS VL of Non-Vx pigs during co-infection suggests that response to PRRS vaccination may be used to predict response under co-infection with PRRS and PCV2b, given that pigs were not previously vaccinated for PRRS. Results of statistical analyses indicate that WUR genotype is associated with PRRS VL upon primary exposure to PRRSV (whether by vaccination or co-infection) as well as PCV2b VL of vaccinated pigs when co-infected with PRRS and PCV2b.

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