Genetic Parameters and Chromosomal Regions Associated with Viral Load and Growth in Pigs Infected with Porcine Reproductive and Respiratory Syndrome Virus

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Summary and Implications

Six hundred commercial crossbred piglets were experimentally infected with the Porcine Reproductive and Respiratory Syndrome (PRRS) virus. Blood samples and body weights were collected at least once per week throughout the 6-week test period. Blood samples were used to measure the degree of infection through viral load. Body weight was measured to look at the impact of the PRRS virus on growth. Serum viral load from day 0 to 21 were summarized by area under the curve. Heritability for viral load and weight gain from 0 to 42 days after infection was 0.28 and 0.26, respectively. All piglets were genotyped for over 60,000 genetic markers comprising single nucleotide polymorphisms (SNPs) distributed across the genome. Regions on chromosomes 3, 4, and X appeared to be associated with area under the curve, while regions on chromosomes 1, 4, 7, and 17 appeared to be associated with weight gain. These results are promising to the swine industry, as it shows that there is genetic variation for resistance to PRRS within a population and that selection for resistance or susceptibility to the virus is plausible.

Introduction

Porcine Reproductive and Respiratory Syndrome is one of the most economically concerning diseases in the swine industry. Many diseases are effectively treated with drug administration or prevented by vaccination. Vaccination has not been effective for PRRS, partially due to the rapid spread and evolution of the virus. The objective of this study was to conduct a genome-wide association study to discover the genetic basis of host response to the PRRS virus using data from the PRRS Host Genetics Consortium NPB and PRRS-CAP project.

Materials and Methods

Three groups of 200 commercial crossbred piglets from disease-free farms were infected with PRRS virus isolate NVSL 97-7985 between 18 and 28 days of age at experimental facilities at Kansas State University. Blood samples and body weights were collected up to 42 days post infection. Whole genome analyses focused on serum viral load up to 21 days post infection and weight gain (WG) from 0 to 42 days post-infection. Serum viral load (VL) was quantified using area under the curve for log-transformed qRT-PCR based serum virus based on 0, 4, 7, 11, 14, and 21 days post-infection. Upon completion of the experiment, tissue samples were collected, DNA prepared, sent to GeneSeek, and genotyped with the Illumina Porcine SNP60 BeadChip, which generated genotypes for approximately 60,000 genetic markers called SNPs.

Heritabilities were estimated with an animal model using ASREML. Associations of SNP genotypes with traits were analyzed by fitting all SNPs simultaneously using the software GenSel.

Results and Discussion

The interaction of experimental group and parity was significant for both traits ($p < 0.05$). Pigs from a first parity litter had lower VL compared to pigs born to later parity sows for the first two groups. Conversely, pigs from a first parity litter in the third group had higher VL compared to pigs from later parities. Similarly for WG, pigs from a first parity litter had the highest WG compared to pigs from later parity litters for the first two groups, while just the opposite was observed for the third group.

After infection with the PRRS virus, all pigs had similar body weights for the first week. However, with the onset of week 2 post-infection, phenotypic variation in body weight began to increase and continued to increase throughout the 6-week test period (Figure 1). Weight gain from day 0 to 42 post-infection ranged from -7.5 to 121 lbs. Heritability for WG was moderate at 0.26. Litter explained 11% of the phenotypic variation in WG, indicating important maternal effects, as expected.

All pigs had a spike in VL on day 4 post-infection (Figure 2) with peak VL by day 11 post-infection. The variability in VL increased with time, in particular beyond 21 days post-infection. The latter can be attributed to the fact that some animals cleared the virus by day 28 post-infection, others later, and some (~33%) apparently

showed reactivation of the virus. Heritability for VL was moderate at 0.28. Litter explained 14% of VL variance, indicating a substantial maternal effect, which is surprising given that sows were PRRS free.

One of the main objectives of this project was to determine regions of the genome that contain genotypes that are associated with VL and WG. Using Porcine sequence build 10, regions on chromosomes 3, 4, and X were found to be associated with VL. Regions on chromosomes 1, 4, 7, and 17 were found to be associated with WG. Furthermore, the region on chromosome 4 was the same for both traits and effects on the two traits in this region had a high negative correlation of -0.98. The latter indicates that pigs that have a genotype in this region that confers low VL are expected to also have greater growth.

Implications

The use of vaccination has been unsuccessful in controlling PRRS virus. Once an animal gets the PRRS virus, the disease spreads quickly to the rest of the population. However, these results indicate a substantial host genetic component in response to infection with the PRRS virus and that selection for resistance or susceptibility is possible. Once specific markers or regions associated with PRRS viral load are identified, producers will be able to genotype their animals for the specific markers at a lower cost, relative to the 60k BeadChip, and use this information in selection. The region on chromosome 4 is a very promising candidate for such selection. Further work is, however, needed to confirm that this same region also confers reduced susceptibility to other strains of the PRRS virus.

Acknowledgements

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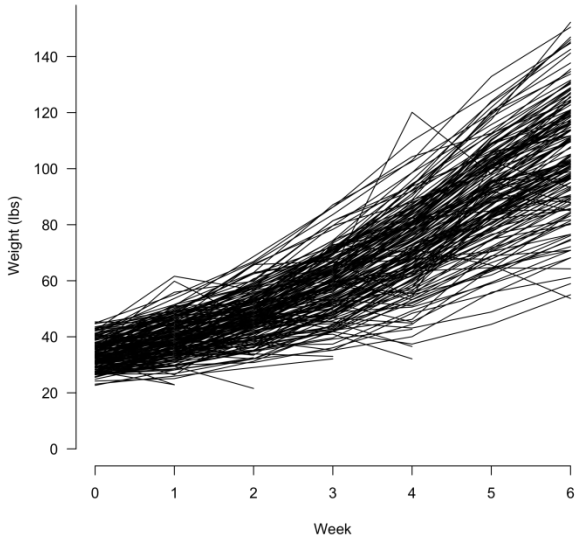

Figure 1. Weekly body weight of pigs from experimental group 2. Body weight graphs for groups 1 and 3 were similar.

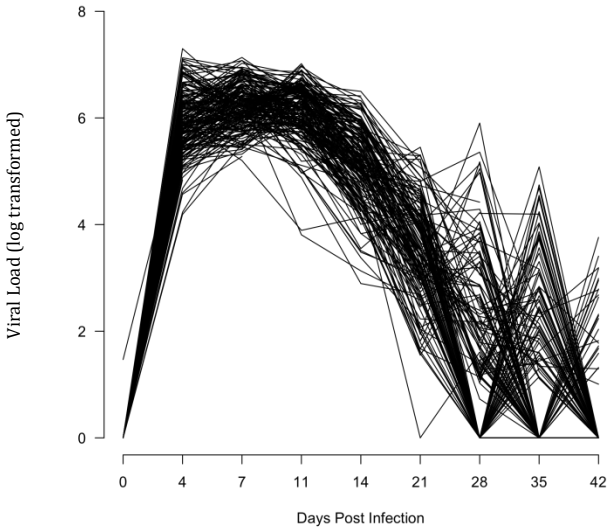

Figure 2. Viral load on pigs from experimental group 2 through 42 days post infection. Viral load graphs for groups 1 and 3 were similar.