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Evidence for a major QTL associated with host response to porcine reproductive and respiratory syndrome virus challenge.

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Abstract

Porcine reproductive and respiratory syndrome (PRRS) causes decreased reproductive performance in breeding animals and increased respiratory problems in growing animals, which result in significant economic losses in the swine industry. Vaccination has generally not been effective in the prevention of PRRS, partially because of the rapid mutation rate and evolution of the virus. The objective of the current study was to discover the genetic basis of host resistance or susceptibility to the PRRS virus through a genome-wide association study using data from the PRRS Host Genetics Consortium PRRS-CAP project. Three groups of approximately 190 commercial crossbred pigs from 1 breeding company were infected with PRRS virus between 18 and 28 d of age. Blood samples and BW were collected up to 42 d post infection (DPI). Pigs were genotyped with the Illumina Porcine 60k Beadchip. Whole-genome analysis focused on viremia at each day blood was collected and BW gains from 0 to 21 DPI (WG21) or 42 DPI (WG42). Viral load (VL) was quantified as area under the curve from 0 to 21 DPI. Heritabilities for WG42 and VL were moderate at 0.30 and litter accounted for an additional 14% of phenotypic variation. Genomic regions associated with VL were found on chromosomes 4 and X and on 1, 4, 7, and 17 for WG42. The 1-Mb region identified on chromosome 4 influenced both WG and VL, exhibited strong linkage disequilibrium, and explained 15.7% of the genetic variance for VL and 11.2% for WG42. Despite a genetic correlation of -0.46 between VL and WG42, genomic EBV for this region were favorably and nearly perfectly correlated. The favorable allele for the most significant SNP in this region had a frequency of 0.16 and estimated allele substitution effects were significant ($P < 0.01$) for each group when the SNP was fitted as a fixed covariate in a model that included random polygenic effects with overall estimates of -4.1 units for VL (phenotypic SD = 6.9) and 2.0 kg (phenotypic SD = 3 kg) for WG42. Candidate genes in this region on SSC4 include the interferon induced guanylate-binding protein gene family. In conclusion, host response to experimental PRRS virus challenge has a strong genetic component, and a QTL on chromosome 4 explains a substantial proportion of the genetic variance in the studied population. These results could have a major impact in the swine industry by enabling marker-assisted selection to reduce the impact of PRRS but need to be validated in additional populations.

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