Genetic Improvement of Sow and Gilt Reproductive Performance via PRRS Immunity

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1. Introduction

Porcine reproductive and respiratory syndrome (PRRS) is a major viral disease impacting pork production worldwide (Rowland et al., 2012). Sows infected with the PRRS virus (PRRSV) have increased rates of abortions, stillbirth, and mummies (Lunney et al., 2011). It has been estimated that losses due to PRRSV cost over $ 660 million every year in the US, with an average loss of $115 per breeding female and $3.08 per pig marketed. Current strategies used to reduce the impact of PRRSV include biosecurity, herd management (e.g. depopulation, gilt introduction), and vaccination. However, these strategies have shown limited success in the field. For instance, many factors contribute to the

success of biosecurity, such as proximity of pig sites and movement in the farm. The location of replacement gilts during quarantine in the farm can affect the success in controlling the virus spread (Holtkamp et al., 2010). As far as vaccination, the purpose of vaccinating animals against PRRS is to reduce clinical losses rather than preventing infection with PRRSV (Charerntantankul, 2012). Therefore, complementary or alternative control strategies are needed to decrease the impact of PRRS.

Exploitation of the host (animal) genetics appears as an alternative and complementary strategy. Recent studies show that selection for improved performance may be feasible during PRRSV infection. Lewis et al. (2009) were the first to report that farrowing traits in sows infected with PRRSV are heritable. These authors reported that genetics account for 12, 17, and 15% of the differences in the observed number of stillborn, mummified, and born alive piglets, respectively, in sows infected with PRRSV. In nursery pigs infected with PRRSV, Boddicker et al. (2012) reported that 30% of the differences in weight gain and amount of PRRSV in the blood are due to genetics. In addition, these authors identified a major genomic region on chromosome (Chr) 4 that explained part (11-16%) of the genetic variation of host response to PRRS in these animals. This region was further explored and the specific mutation responsible for this effect was discovered (Koltes et al. 2015).

In this work, we will present results from an ongoing project entitled “Gilt Acclimation Project”. The goals of this project are to evaluate the general

immune capacity of gilts and sows, and to perform genetic (heritabilities and correlations) and genomic (mapping and prediction) analyses for PRRS response.

2. Background

2.1. Description of the data

The dataset used in this project were provided by a consortium of the main pig breeding companies (genetic suppliers) that operate in Canada (PigGen Canada, http://www.piggencanada.org/). The dataset included data on (1) purebred multiplier gilts and sows (called Outbreak dataset) and (2) crossbred replacement gilts (called GA dataset). The Outbreak dataset included data on 607 purebred Landrace gilts and sows from a commercial multiplier herd in Canada that experienced a PRRS outbreak that was estimated to have occurred on November 20th, 2011 (Serão et al., 2014). The GA dataset included data on 2,852 crossbred replacement gilts. Animals in the GA dataset were sourced from seventeen multiplier herds (2 to 3 multipliers per genetic supplier), across 7 genetic suppliers. Gilts were placed in 23 commercial herds (i.e. farms) across Canada with historical occurrence of natural outbreaks.

2.2. Phenotypes and Genotypes

Reproductive performance in the Outbreak dataset (402 sows) included traits such as number of piglets born alive (NBA), alive at 24h (NA24), of stillborn piglets (NSB), and of mummified piglets (MUM) during the PRRS phase.

Although reproductive performance was also available in the GA dataset (~800 animals), preliminary analyses showed that there was no evidence of PRRS during the farrowing period (i.e. there was no negative impact of PRRS on reproductive performance). Thus, these data were not used for analyses.

Antibody response to PRRSV in the blood, measured as sample-to-positive (S/P) ratio, was assessed in animals from both datasets by ELISA (IDEXX PRRS X3, IDEXX Laboratories Inc., Westbrook, ME, USA). In the Outbreak dataset, S/P was measured at approximately 45 days post the estimated PRRS outbreak date. In the GA dataset, S/P ratio was measured in replacement gilts at different times between entry into the farm and acclimation (29 to 88 days) across the 23 farms (one time point per farm), with an overall average of 40.8 ± 16.3 days. In the Outbreak data, 100% of the animals were PRRS-positive according to the ELISA test (S/P ratio > 0.40), while 81% of the gilts in the GA dataset were positive for the ELISA test. In addition, while S/P ratio in the Outbreak dataset was due to a confirmed PRRSV infection, we were not able to obtain concrete information about the source of antibody response in the GA dataset. Therefore, S/P ratio in the GA could be due to vaccination to PRRS, as many of the farms included in the study used vaccination as a standard operating procedure, or due to exposure to PRRS during the acclimation period. All animals were genotyped by Illumina PorcineSNP (60K or 80K; Illumina Inc., San Diego, CA, USA).

2.3. Analyses

Genetic parameters were estimated for both datasets. In the Outbreak dataset, a pedigree including 2,995 animals was used for estimation of heritabilities for NBA, NSB, MUM, and S/P ratio, and of genetic and phenotypic correlations of S/P ratio with NBA, NSB, and MUM.

The identification of genomic regions controlling reproductive performance during PRRSV outbreak and antibody response was performed by associating SNPs and phenotypes using Bayes-B, a method in which all markers are fitted simultaneously in the model. Analyses were performed for S/P ratio in the GA dataset, and for S/P, NBA, NSB, and MUM in the Outbreak dataset.

We evaluated the use of SNP genotypes to predict S/P ratio and reproductive performance in the Outbreak data using S/P ratio from the GA dataset as the training data. In other words, the GA dataset (training) was used to estimate SNP effects and the resulting estimates were used to predict S/P ratio in the Outbreak dataset (validation). These same estimates were also used to predict reproductive performance in the Outbreak dataset (validation). Accuracy of prediction was estimated as the correlation between the predictions and phenotype, divided by the square root of heritability of the trait.

All genomic predictions were performed using 5 sets of SNPs, which were selected based on the GWAS results reported by Serão et al. (2014), where

two regions on Chr 7 were associated with S/P ratio: (1) ALL: all SNPs across the genome; (2) MHC: SNPs between 23-33 Mb on Chr 7; (3) Mb130: SNPs located between 128 and 132 Mb on Chr 7; (4) SSC7: the combined sets from (2) and (3); and (5) Rest: All SNPs except those used for SSC7.

3. Results and Discussion

Estimates of heritabilities for reproductive performance during PRRS (Outbreak dataset) were 9, 6, and 8%, for NBA, NSB, and MUM, respectively. Although these heritabilities were low, a major part of differences in S/P ratio were due to genetics, with genetics explaining 45 and 47% of the variation in S/P ratio in the Outbreak and GA datasets, respectively. These high values indicate that selection for S/P ratio is possible and can be accurate. We also evaluated the relationship of S/P ratio with reproductive performance during the PRRS outbreak. Phenotypic correlations were virtually zero, ranging from -0.07 for NSB to 0.06 for NBA. However, the genetic correlations of S/P ratio with reproductive traits were high: 0.73, -0.72, and -0.66, for NBA, NSB, and MUM. On this basis, sows with a greater genetic value for S/P ratio are expected to genetically show favorable reproductive performance during a PRRS outbreak. Therefore, S/P ratio has the potential to be used as an indicator trait to select for improved reproductive performance during PRRSV infection.

No associations between SNPs and phenotypes were found for MUM and NBA, but strong associations were found for NSB and S/P ratio in the

Outbreak dataset (Serão et al., 2014). A region on Chr 1 (32-35 Mb) explained 11% of the genetic variation for NSB. For S/P ratio, the same regions on Chr 7 were found to be important in the Outbreak and GA datasets. The MHC region explained 25% and 20% of the genetic variation of S/P ratio in the Outbreak and GA datasets, respectively. The Mb130 region explained 15% and 7% of the genetic variation of S/P ratio. There were no other strong associations with S/P ratio in the genome.

As expected, for S/P ratio, SSC7 showed the greatest accuracy of prediction (0.48), followed by ALL (0.45), MHC (0.26), Mb130 (0.40), and Rest (0.10). Therefore, S/P ratio might be predicted with moderate accuracy using genotypes of SNPs in the MHC and Mb130 regions, whereas the rest of the genome has very little predictive ability. In addition, because part of the training dataset (GA) consisted of vaccinated animals, S/P derived from response to vaccination can be used to predict S/P ratio resulting from PRRSV infection. However, results for genomic predictions of reproductive performance using S/P ratio showed an opposite trend. For instance, correlations of predictions with NBA, NSB, and MUM were greater when using the SNPs outside the MHC and Mb130 regions (Rest) for prediction of S/P ratio, with 0.39, -0.07, and -0.50, respectively. In addition, the direction of the predictions was opposite to the genetic correlations when only the SNPs in the MHC and Mb130 regions were used (i.e. negative for NBA and positive for NSB and MUM). These results indicate that, although the MHC and Mb130 regions control S/P ratio, the rest of

the genome should be used to predict reproductive performance during a PRRS outbreak.

4. Summary

The use of field data from different genetic suppliers has allowed us to assess the genetic variation of response to PRRS in commercial gilts and sows. We observed that reproductive performance during PRRSV infection has low heritability (<10%). However, antibody response to PRRSV (measured as S/P ratio) had high heritability (~45%) in two different datasets. Furthermore, the high genetic correlations of S/P ratio with farrowing traits during a PRRS outbreak suggests that S/P can be used as an indicator trait to improve reproductive performance during PRRSV infection.

The use of genomic data indicated that specific regions of the genome control NSB during PRRSV infection and S/P ratio. We observed that S/P ratio can be predicted with moderate accuracy when two regions on Chr 7 are used. However, we observed that accuracy of prediction of reproductive performance during PRRSV infection was higher when the other genomic regions were used.

These analyses was performed using data that might be derived from vaccination for PRRS, suggesting that farms that perform vaccination for PRRS could be targeted to collect PRRSV antibody data. Altogether, these results

indicate that response to PRRS in gilts and sows is heritable and that immune and reproductive performance might be improved using genomics.

New data is still being generated in this project, including immune measures for other diseases (e.g. PCV2, swine influenza, etc) and reproductive performance across parities. The use of more data on reproductive performance and new data on different diseases will increase our understanding of the genetics of immunity and reproductive performance in replacement gilts sows.

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