Investigating the Genetic Basis of Antibody Response to Common Infectious Diseases in Commercial Sows

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Summary and Implications

Disease resistance is one of the most economically important traits affecting pork production. Genetic selection for disease resistance is challenging for the industry since disease traits are not expected to be expressed in the clean genetic nucleus, where selection is performed. Data collected after animals enter commercial farms could be used to estimate breeding values for sires of commercial sows, enabling the selection of robust sires. In this work, we showed that there is genetic variance for antibody response to common infectious diseases in pigs, and major genomic regions were identified for some of these diseases. These results support the possibility of using antibody response to select for robustness in pigs.

Introduction

Infectious disease is one of the main factors causing losses in productivity in the swine industry, motivating the investigation of resistance-related traits for genetic selection. The presence of genetic variation in disease related traits, such as antibody response, indicates that selection for pigs with better immune response is possible. It has been shown that antibody response to PRRS virus infection may be genetically correlated with reproductive performance of PRRS virus-infected sows. Against this background, the goal of this study was to evaluate the genetic basis of antibody response to several common infectious diseases in commercial gilts and sows.

Materials and Methods

The datasets used in this study were provided by a consortium of pig breeding companies that operate in Canada (PigGen Canada). A total of 2,848 Large White x Landrace replacement gilts were sourced from 17 high-health multipliers (7 breeding companies) and introduced to 23 commercial farms with a history of common diseases, following standard acclimation procedures. Blood samples were collected for measurement of antibody response to swine influenza virus (SIV), *Mycoplasma hyopneumoniae* (MH), porcine circovirus type 2 (PCV2), and 8 serotypes of *Actinobacillus pleuropneumoniae* (APP1, 2, 3, 5, 7, 10, 12, and 13) at four time points: when entering the commercial herd (Entry), after acclimation (Post-acclimation), during parity 1 (P1), and parity 2 (P2). Genotype data were available on 42,145 single nucleotide polymorphisms (SNP) on 3,615 animals. Using this genotypic data, heritability was estimated and genomic regions controlling the evaluated traits were identified. We also evaluated the use of markers to predict antibody response.

Results and Discussion

In general, heritability estimates were low to moderate (Table 1), except for sum of APP which was high. Genomic regions found to be associated with antibody response are presented in Table 2. For APP serotypes, genomic regions were only identified at Post-acclimation; on chromosome 14 (chr14; at 2 Mb) for APP3, APP7, APP10, and APP13, explaining 5.6%, 4.7%, 2.8%, and 3.6% of the genetic variance (GV), respectively. A gene within this genomic region is *SYK*, which is involved in the control of immune-receptors. For APP5, a genomic region on chr14 (105 Mb) explained 4.2% of GV, which co-localizes with two genes that are associated with immune-response: *SIKE1* and *NRAS*. For SIV, no genomic regions were identified. A genomic region on chr7 (130-131 Mb) was identified for MH (Parity1, 5.1% of GV) and PCV2 (Entry, 34% of GV; Post-acclimation, 43.4% of GV). When evaluating the use of genetic markers to predict antibody response, PCV2 had the best results, with genomic prediction accuracies of 0.58 and 0.61 at Entry and Post-acclimation, respectively. However, for the other disease and time-points, genomic prediction accuracies were generally low to moderate, ranging from -0.18 (SIV at Post-Acclimation) to 0.39 (SIV at P1). Although most of traits had low heritability, the genomic prediction accuracies for some diseases were moderate to high, especially when genomic regions were identified (e.g. PCV2). These results provide new information on the genetic and genomic basis of response to infectious diseases in sows. Therefore, improvement of immune response in commercial gilts and sows based in antibody response may be feasible.

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Table 1: Heritability estimates (standard errors) of antibody response at various time points to common infectious diseases in pigs

Disease[1]	Entry	Post-acclimation	Parity 1	Parity 2
SIV	0.23 (0.08)	0.04 (0.04)	0.07 (0.04)	0.09 (0.06)
MH	0.26 (0.15)	0.27 (0.10)	0.10 (0.08)	0.27 (0.06)
PCV2	0.14 (0.07)	0.13 (0.04)	0.08 (0.04)	0.01 (0.03)
APP1	0.16 (0.03)	0.10 (0.03)	0.18 (0.04)	0.22 (0.06)
APP2	0.13 (0.03)	0.09 (0.03)	0.03 (0.03)	<0.01 (0.01)
APP3	0.22 (0.03)	0.26 (0.03)	0.16 (0.04)	0.14 (0.05)
APP5	0.14 (0.03)	0.31 (0.03)	0.07 (0.03)	0.10 (0.05)
APP7	0.19 (0.03)	0.19 (0.03)	0.12 (0.03)	0.11 (0.05)
APP10	0.18 (0.03)	0.26 (0.03)	0.18 (0.04)	0.33 (0.06)
APP12	0.24 (0.03)	0.19 (0.03)	0.05 (0.03)	0.15 (0.05)
APP13	0.28 (0.04)	0.29 (0.03)	0.15 (0.04)	0.28 (0.06)
APP SUM[2]	0.40 (0.03)	0.39 (0.03)	0.28 (0.03)	0.39 (0.06)

[1]SIV: swine influenza virus; MH: *Mycoplasma hyopneumoniae*; PCV2: porcine circovirus type 2; APP: *Actinobacillus pleuropneumoniae;*
[2]Sum of antibody response for all APP

Table 2: One Mb genome windows associated with antibody response to common diseases

Trait[1]	Time-point	Chromosome	Position (Mb)	%GV[2]
MH	Parity 1	7	130-131	5.1
PCV2	Entry	7	130-131	33.9
PCV2	Post-accl	7	130-131	43.3
APP3	Entry	8	32	2.5
		14	2	2.1
APP3	Post-accl	14	2	5.6
APP5	Post-accl	4	105	4.1
APP7	Post-accl	6	157	9.8
		14	2	4.6
APP10	Post-accl	14	2	2.8
		15	24	2.4
APP13	Entry	1	58	6.4
		9	131	3.5
APP13	Post-accl	14	2	3.6
APP13	Parity 2	6	81	3.1

[1]MH: *Mycoplasma hyopneumoniae*; PCV2: porcine circovirus type 2; APP: *Actinobacillus pleuropneumoniae;*
[2]%GV: Percentage of genetic variance explained by the window