Effect of WUR Genotype and PRRS Vaccination on Pigs Co-Infected with PRRS and PCV2b

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Summary and Implications

Average daily gain (ADG) and viral load (VL) were evaluated for pigs co-infected with porcine reproductive and respiratory syndrome (PRRS) virus (PRRSV) and porcine circovirus type 2b (PCV2b). Pigs were pre-selected for WUR genotype (a marker on chromosome 4 associated with weight gain and VL under PRRSV-challenge), half were vaccinated for PRRS, and half were not. Results indicate that vaccination for PRRS resulted in slower growth prior to co-infection and that the AB WUR genotype was associated with faster growth prior to co-infection, lower PRRS VL, and lower PCV2b VL in vaccinated pigs.

Introduction

PRRS is one of the most costly diseases to the US pork industry today. PRRSV weakens the immune system, thereby increasing the likelihood of co-infection with other diseases, such as PCV2. When an animal that is infected with PRRSV also becomes infected with PCV2, PRRSV increases replication of PCV2, enhancing the number and severity of disease symptoms.

The objectives of this research were to determine the effect of genotype for the WUR marker on chromosome 4 and PRRS vaccination on growth rate and viremia in nursery pigs co-infected with PRRSV and PCV2b. A third objective was to evaluate the interaction of vaccination and WUR genotype.

Since the AB genotype for the WUR marker has been associated with increased weight gain and lower PRRS VL in previous studies using only PRRSV-infected pigs, we hypothesized that AB pigs would grow faster and have less PRRS and PCV2b VL once co-infected. We also hypothesized that vaccinated pigs would grow slower following vaccination, but faster with lower PRRS and PCV2b VL following co-infection. We did not expect to observe a significant interaction of WUR genotype by vaccination for growth rate, PRRS VL, or PCV2b VL.

Materials and Methods

Commercial nursery pigs (n=392) from two co-infection trials were used for the analysis of ADG and PRRS and PCV2b VL. Pigs originated from the same breeding company and were pre-selected for WUR genotype (50% AA and 50% AB) before shipment to Kansas State University at weaning age. Upon arrival, pigs were randomly sorted into pens within one of two rooms. Pigs in one room were vaccinated with a modified live PRRSV vaccine.

After 28 days, all pigs were co-infected with field strains of PRRSV and PCV2b. Pigs were weighed weekly from vaccination to 42 days post-infection (dpi) and serum samples were collected at 0, 4, 7, 11, 14, 21, 28, 35, and 42 dpi. ADG was analyzed for the pre co-infection period (-28 to 0 dpi) and the co-infection period in three phases (0 to 21, 21 to 42, and 0 to 42 dpi). PRRS and PCV2b VL were defined as area under the curve of the log of PCR-based serum viremia from 0-21 dpi for PRRSV and 0-42 dpi for PCV2b. Due to availability of data, PCV2b VL was analyzed for one trial only (n=203).

Results and Discussion

Regardless of PRRS vaccination, AB pigs grew faster than AA pigs before co-infection with PRRSV and PCV2b (P=0.01). Vaccinated pigs grew slower (P<0.0001) than non-vaccinated pigs prior to co-infection and no significant interaction of WUR genotype by vaccination was observed (P=0.34). No difference in growth rate was detected between genotypes or vaccination groups and the interaction of WUR by vaccination was not significant for any of the three phases following co-infection. However, vaccination had contrasting effects between trials (P<0.0001) for growth rate from 21 to 42 dpi.

Vaccinated pigs had lower PRRS VL when co-infected (P<0.0001), as did AB pigs (P<0.0001). WUR genotype was not significant for PCV2b VL post co-infection for the non-vaccinated pigs, but of the vaccinated pigs, AB pigs had lower PCV2b VL than AA pigs (P=0.004).

The AB genotype is the favorable WUR genotype under co-infection of PRRSV and PCV2b. AB pigs grew faster prior to co-infection, had overall reduced PRRS VL, and reduced PCV2b VL when vaccinated. Regardless of genotype, vaccination against PRRSV resulted in slower growth prior to co-infection, but no difference in growth rate between vaccination groups was detected after co-infection.

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