Summary of: Studies on Heterologous Protection Between Japanese Type 1 and Type 2 Porcine Reproductive and Respiratory Syndrome Virus Isolates

Hiroshi Iseki, Kenji Kawashima, Michihiro Takagi, Tomoyuki Shibahara, Masaji Mase

Key Points:

- This study evaluated the cross-protective immunity between type 1 and type 2 porcine reproductive and respiratory syndrome virus (PRRSV) isolates in growing pigs
- Immunity induced by the type 1 infection may play a role in reducing viremia caused by the type 2 PRRSV.
- The immunity induced by the type 2 may not contribute to 40 cross-protection against the type 1.

PRRSV strains within a same genotype can potentially present substantial genetic and antigenic differences. While most Japanese pig farms are endemic for type 2 PRRSV and the virus being present in the country since 1994, the first type 1 PRRSV was isolated in 2009. Cross-protection conferred by one type against the other is an important issue yet to be completely elucidated. This study aimed to assess the antigenic relationship between Japanese type 1 and type 2 PRRSV isolates in-vivo.

Five-week-old pigs from a herd negative for PRRS, pseudorabies, porcine epidemic diarrhea, transmissible gastroenteritis, atrophic rhinitis, Mycoplasma pneumonia, swine dysentery, salmonellosis, toxoplasmosis, and actinobacillosis were used. These pigs also tested ELISA negative for PRRSV antibodies. Experiments were carried out with type 1 (Jpn EU 4-37) and type 2 PRRSV, EDRD1. Experiment 1 consisted of 14 pigs randomly allocated to three groups: Group 1 - type2/type1 (n=5, inoculated via nasal spray with EDRD1 on day zero of the study and with Jpn EU 4-37 on week 5 afterwards), Group 2 - type1 (n=5, inoculated via nasal spray with Jpn EU 4-37 on week 5) and Group 3 - control (n=4, uninfected).

In experiment 2, 14 pigs randomly allocated in three groups: Group 1 - type1/type2 (n=5, inoculated via nasal spray with Jpn EU 4-37 on day zero and with EDRD1 on week 5), Group 2 - type2 (n=5, inoculated via nasal spray with EDRD1 on week 5) and Group 3- control (n=4, uninfected). Antibodies against PRRSV were detected by ELISA in serum from -35 up to 21 dpi. Lungs, tonsils, tracheobronchial lymph nodes, liver, kidneys and spleen were collected at necropsy and tested by RT-PCR.

None of the animals showed clinical signs. However, rectal temperature was higher than the control group for type1 at 10 dpi, for type1/type2 at 4 to 8 dpi, and for type2 at 2-4 dpi and 8-13 dpi. While the serum viral load in the type2 group remained at approximately 1.9×10^3 copies/ml at 22 dpi, the viral load of the type1/type2 group rapidly declined to 76 copies/ml 10 at 22 dpi. Antibodies against PRRSV were not observed in the control group, but were in type1 and type2 groups from 10 dpi, and in type2/type1 and type1/type2 groups from 7 days post exposure with steeper increases in sample-to-positive ratio at 10 and 4 days post challenge. Type1 and Type2 PRRSV RNA was detected in all the tested organs of the type2/type1 and type1 groups. The quantity of type 2 PRRSV RNA in the tonsils in the type1/type2 group was significantly lower than in that in the type2 group.

The main conclusion is that pigs in the acute phase are partially protected against heterologous type 2 PRRSV challenge by the Japanese type 1 PRRSV isolate; however, immunity conferred by the type 2 PRRSV infection did not decrease the persistence of type 1 PRRSV in the blood of the infected pigs. Still, authors acknowledge the limitations of the study in that only a single replicate of the experiments was performed and power might be limited.

Full text is available here: https://www.jstage.jst.go.jp/article/jvms/advpub/0/advpub_20-0122/_pdf