Do all PRRSV variants have the same level of infectivity?

C.M. Melini[1*], M. Kikuti[1], M. Torremorell[1], K. VanderWaal[1], S. Rossow[2], J. Torrison[2], C. A. Corzo[1]
[1]Department of Veterinary Population Medicine, University of Minnesota, [2]Veterinary Diagnostic Laboratory, University of Minnesota

Key Findings:

- The L1C 144 variant requires fewer infectious particles to infect pigs when compared to L1A 174 or L9 142.
- Tissues were severely more affected in L1C 144 infected pigs when compared to the other two variants.
- Nasal and fecal shedding even though it started on day 1 and 4 post-infection it was intermittent.

Data on the degree of PRRSV variant infectiousness is scarce. We hypothesize that some variants are more infectious than others, which consequently results in higher transmission rates between pigs and farms. A study was designed to compare the degree of infectivity among three variants including the newly emerged PRRS L1C 1-4-4 variant, alongside the 2014 L1A 1-7-4 and the 2000 L9 1-4-2 also known as MN 30-100. For each variant, thirty-six PRRSV ELISA and RT-PCR negative three-week-old barrows were randomly allocated to five challenge groups and one control group. Pigs were individually housed in separate rooms according to treatment groups. Pigs in the same room had no nose-to-nose contact but did share the same air space. All three viruses were grown in MARC-145 cells to a 10^5 $TCID_{50}$/ml concentration and through 10-fold dilutions five different inocula concentrations were obtained ranging from 10^4 to 10^0 $TCID_{50}$/ml. Pigs were intranasally challenged (1 ml per nostril) according to the group's selected virus concentration. Blood, nasal and rectal swabs were collected at day post-infection (DPI) 0, 1, 2, 4, 7, 11, 16, 21, 26, and 30 to assess infection and viral shedding through PRRSV RT-PCR. Researchers changed their gloves, masks, and gowns between sampling each pig. On DPI 11, two pigs from each group were euthanized, and tissues (e.g., brain, lung, lymph nodes) were collected for histopathological assessment. For this study, DPI 4 was chosen as the day to determine whether pigs had become infected.

Using a probit model, the estimated median infectious dose 50 (ID_{50}) (infective dose needed to infect 50% of exposed individuals) was $10^{1.3}$ $TCID_{50}$/ml (95% CI $10^{0.4}$–$10^{2.2}$) for the L1C 1-4-4 variant, $10^{2.3}$ $TCID_{50}$/ml (95% CI $10^{1.6}$–$10^{3.0}$) for the L1A 1-7-4 variant, and $10^{2.6}$ $TCID_{50}$/ml (95% CI $10^{2.0}$–$10^{3.2}$) for the L9 1-4-2 variant. When adjusting for the inoculum dose in $TCID_{50}$/ml, the pigs challenged with L1C 1-4-4 were 13 (95% CI 3–16) times more likely to be RT-PCR positive on DPI 4 compared to pigs challenged with L9 1-4-2. Virus detection in nasal swabs began at 1 DPI and cycle threshold values decreased until DPI 7 (Figure 1). Fecal shedding began at DPI 4 and peaked at DPI 7, intermittent nasal and rectal shedding patterns were similar for all viruses with slight differences between inoculum groups during the study period. Lungs had similar microscopic lesions regardless of dose, but those in pigs infected with the L1C 1-4-4 were consistently more severe. Brain lesions were found in at least 1 pig within each concentration group in the L1C 1-4-4 challenged group, whereas this was limited to only 1 pig per variant in either the L1A 1-7-4 or L9 1-4-2 challenged groups. Heart lesions were mostly present in the L1C 1-4-4 variant group. Study results indicate that compared to the L9 1-4-2 and L1A 1-7-4 variants, the L1C 1-4-4 virus requires fewer viral particles to successfully infect half of the pigs by DPI 4. In addition, our data showed that the L1C-1-4-4 variant leads to more frequent and more severe tissue lesions in the lung, heart, and brain as compared to the other evaluated PRRSV variants.

Figure 1. Nasal shedding results