Shedding of PRRS Virus in Milk and Colostrum

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Summary and Implications

Porcine reproductive and respiratory virus (PRRSV) is endemic in many swine herds. The means by which PRRSV continues to circulate in herds is not well understood. Under experimental conditions, we found that that PRRSV is shed in the milk and colostrum of infected dams. Virus-contaminated mammary secretions could play an important role in virus transmission in endemically infected herds.

Introduction

Sow herd stabilization has been a management strategy to control clinical PRRS(1). However, even after sow herd stabilization some herds continue to experience problems associated with PRRS in nurseries, with no apparent new source of virus(2).

The host cell of PRRSV infection is the macrophage(3). Swine mammary secretions are rich in macrophages(4) thus making milk a possible source of virus. This experiment was designed to determine whether PRRSV could be shed in mammary secretions of infected sows.

Materials and Methods

Experiment 1: Six PRRS-virus negative pregnant sows were divided into three groups. Group I received a 2–ml intramuscular (IM) dose of a commercially produced modified live PRRSV vaccine between days 85–97 of gestation. Group II received an intranasal challenge of a PRRSV field isolate between days 85–90 gestation. Group III served as a negative control.

Mammary secretions were collected for 18 days post farrowing on all sows. These secretions were separated into lipid, whey, and cell portions via centrifugation. The lipid portion was discarded. Whey was drawn off and frozen until assayed. The cell portion was washed three times and resuspended in a buffered salt solution prior to freezing. Virus isolation (VI) was attempted on all whey and cell samples by using porcine alveolar macrophages (PAM) and MARC 145 cells. The presence of virus was confirmed by an immunoflourescent stain with PRRSV-specific monoclonal antibody.

Swine bioassays were conducted to confirm the negative VI results. For each sow, negative cell samples from across her lactation were pooled, and injected intramuscularly into 4 week-old PRRSV-negative pigs. The whey samples were handled in an identical fashion. The pigs were then bled weekly for up to 28 days to detect seroconversion.

Experiment 2: Group I (vaccinated sows) from Experiment I were bred by artificial insemination with semen from a PRRSV-negative boar. These sows were then vaccinated with a 2 ml IM dose of a different commercially produced modified live PRRS vaccine. Samples of mammary secretions were collected for 18 days, and handled as outlined above.

Results and Discussion

In Experiment 1, virus was recovered from cell fractions collected from one vaccinated sow on days 2 and 4 post farrowing and one field isolate-challenged sow on days 8 and 9 post farrowing. The whey fraction from the same field isolate-challenged sow was also VI positive on day five post farrowing. On bioassays, 3 of 4 pigs receiving VI-negative pooled whey fractions became infected, whereas none of 4 pigs administered VI-negative cell fractions became infected. Thus, PRRS virus was detected in the mammary secretions of 2 of 2 vaccinated sows, and one of 2 field isolate-challenged sows. Detection of virus by bioassay from VI-negative samples suggests that virus was present at levels below the detectable level of cell culture.

In Experiment 2, no virus was isolated from either cell or whey fractions in cell culture. Swine bioassays of pooled VI-negative samples failed to demonstrate the presence of virus in these samples. Thus, previously vaccinated sows did not shed virus when re-vaccinated with a different commercial PRRS vaccine.

References

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