Effect of vaccination with a modified-live porcine reproductive and respiratory syndrome virus vaccine on dynamics of homologous viral infection in pigs

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Objective—To determine effects of vaccination protocols with modified-live porcine reproductive and respiratory syndrome virus (PRRSV) vaccine on persistence and transmission of virus in pigs infected with a homologous isolate and determine clinical and virologic responses following heterologous viral challenge.

Animals—Four hundred forty 6- to 8-week-old PRRS-naïve pigs.

Procedures—Pigs were allocated into 5 groups. Groups A to D were inoculated with wild-type PRRSV VR2332. Group A (positive control pigs) received PRRSV only. Groups B, C, and D received modified-live PRRSV vaccine (1, 2, or 3 doses). Group E served as a negative control group. To evaluate viral transmission, sentinel pigs were introduced into each group at intervals from 37 to 67, 67 to 97, and 97 to 127 days postinoculation (DPI). To evaluate persistence, pigs were euthanized at 37, 67, 97, or 127 DPI. To assess clinical and virologic response after challenge, selected pigs from each group were inoculated at 98 DPI with a heterologous isolate (PRRSV MN-184).

Results—Mass vaccination significantly reduced the number of persistently infected pigs at 127 DPI. Vaccination did not eliminate wild-type PRRSV; administration of 2 or 3 doses of modified-live virus vaccine reduced viral shedding after 97 DPI. Previous exposure to wild-type and vaccine virus reduced clinical signs and enhanced growth following heterologous challenge but did not prevent infection.

Conclusions and Clinical Relevance—Findings suggest that therapeutic vaccination may help to reduce economic losses of PRRSV caused by infection; further studies to define the role of modified-live virus vaccines in control-eradication programs are needed. (Am J Vet Res 2007;68:565–571)

Porcine reproductive and respiratory syndrome has been estimated to cost the US swine industry 560 million dollars in losses each year. Results of the same study indicate that 88% of the total cost of PRRSV infections in the United States is attributable to increased mortality rates and decreased growth performance in postweaning pigs, whereas the impact of the disease on breeding herds represents only 12% of the total cost. Since the disease was first reported in 1989, anorexia, lethargy, hyperemia of the skin, dyspnea, increase in mortality rates, and reduction in ADG have been the clinical signs consistently described following PRRSV infection in nursery, grower, or finisher pigs. The PRRSV replicates in pulmonary alveolar macrophages, facilitating bacterial coinfections and resulting in cases of streptococcal meningitis, septicemic salmonellosis, Hemophilus parasuis infection, and bacterial bronchopneumonia. Porcine reproductive and respiratory syndrome virus is an arterivirus that initially replicates in macrophages, establishing nonclinical persistent infections. Porcine reproductive and respiratory syndrome virus RNA has been detected in the lymphoid tissues of pigs up to 251 DPI. It continuously replicates at a low level and can be transmitted to susceptible animals following direct contact with pigs inoculated up to 86 days after infection. A known risk factor for transmission is the presence of subpopulations of PRRS-naïve and PRRS-infected swine, coexisting within endemically infected herds, a problem that is frequently exacerbated by introduction of PRRS-naïve replacement gilts. Techniques such as herd closure, acclimation of gilts to their new environment, and mass exposure have been proposed to eliminate such subpopulations and...
reduce the risk of PRRSV shedding; however, results have been inconsistent among farms.

Another method to maximize population immunity to PRRSV is vaccination. The induction of both humoral and cell-mediated immune responses has been described following the application of modified-live PRRSV vaccines in pigs.23-25 Although it has been reported that vaccination with MLV provides incomplete heterologous protection against PRRSV infection,26 multiple experiments23,24,27,28 have revealed substantial reduction in lesions and clinical signs in vaccinated pigs following homologous and heterologous PRRSV challenge. By contrast, research on killed PRRSV vaccines has generated contrasting results regarding stimulation of production of neutralizing antibody and cell-mediated immune responses in pigs.23,26,29 In commercial conditions, the strategic combination of mass vaccination by use of modified-live PRRSV products with herd closure and unidirectional pig flow has been a successful approach to control and, in some instances, to eliminate PRRSV from swine herds.23,24 Although these field reports provide important information regarding the practical application of PRRSV therapeutic vaccination, the need for specific variables to scientifically validate the technique such as the use of positive and negative control groups; exact knowledge of infection time; and control of other factors such as feeding, genetics, health status, and management motivated us to perform a large-scale experiment controlling for those variables and including the use of valuable diagnostic techniques to understand the effect on infection dynamics after mass vaccination.

The study reported here hypothesized that the use of a modified-live PRRSV vaccine would significantly reduce the persistence and transmission of PRRSV in a population of pigs infected with the homologous isolate. The specific aims of the study were to determine whether 3 protocols of vaccination against PRRSV reduced the proportion of persistently infected pigs in the population, to evaluate the effect of 3 protocols of vaccination on PRRSV transmission to susceptible pigs, and to determine the clinical and virologic response of vaccinated pigs following challenge with a highly virulent heterologous PRRSV isolate.

Materials and Methods

Swine and housing—Three hundred thirty-two 6- to 8-week-old pigs (principals) were obtained from a herd known to be free of PRRSV on the basis of 10 years of diagnostic testing. After arrival, pigs were confirmed to be uninfected by use of a commercial ELISA33 and RT-PCR assay.34 Pigs were individually identified with ear tags and randomly assigned to 5 groups (A to E) that were housed in separate rooms at the research farm of the University of Minnesota Swine Disease Eradication Center in west central Minnesota. Groups were designated as follows: group A, infected with wild-type PRRSV only (positive control pigs); group B, infected with PRRSV and administered 1 dose of MLV vaccine; group C, infected with PRRSV and administered 2 doses of MLV vaccine; group D, infected with PRRSV and administered 3 doses of MLV vaccine; and group E, sham inoculated only (negative control pigs; Table 1). Groups A, B, C, and D contained 80 pigs each, and group E contained 12 pigs. All pigs were vaccinated on arrival against H. parasuis, E. rhusiopathiae, and L. intracellularis.35 In conjunction with the 332 principal pigs, 108 PRRSV-naïve age-matched sentinel pigs were introduced at designated periods following the initiation of the study. All protocols and procedures of pig management and care were reviewed and approved by the University of Minnesota Institutional Animal Care and Use Committee. Personnel practiced PRRSV-specific

| Table 1—Experimental design for a study of effect of a modified-live PRRSV vaccine on the dynamics of homologous viral infection in pig populations. |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Group | 0 | 7 | 37 | 67 | 97 | 127 | 37–67 | 67–97 | 97–127 |
| A | Inoculation with PRRSV | NA | Euthanize-sample | Euthanize-sample | Euthanize-sample | Euthanize-sample all remaining pigs | 1st sentinel group* | 2nd sentinel group | 3rd sentinel group |
| B | Inoculation with MLV administration | Euthanize-sample | Euthanize-sample | Euthanize-sample | Euthanize-sample | Euthanize-sample all remaining pigs | 1st sentinel group | 2nd sentinel group | 3rd sentinel group |
| C | Inoculation with MLV administration | Euthanize-sample | Euthanize-sample | Euthanize-sample | Euthanize-sample | Euthanize-sample all remaining pigs | NA | 1st sentinel group | 2nd sentinel group |
| D | Inoculation with MLV administration | Euthanize-sample | Euthanize-sample | Euthanize-sample | Euthanize-sample | Euthanize-sample all remaining pigs | NA | NA | 1st sentinel group |
| E | Sham inoculation | NA | Euthanize-sample | Euthanize-sample | Euthanize-sample | Euthanize-sample all remaining pigs | 1st sentinel group | 2nd sentinel group | 3rd sentinel group |

*Groups of sentinel pigs were in contact with originally inoculated pigs for 30 days and then removed and euthanized, and samples were collected.

Group A = Pigs infected with wild-type virus infection only (positive control pigs; n = 80). Group B = Pigs infected with wild-type virus infection and administered 1 dose of MLV (n = 80). Group C = Pigs infected with wild-type virus infection and administered 2 doses of MLV (n = 80). Group D = Pigs infected with wild-type virus infection and administered 3 doses of MLV (n = 80). Group E = Negative control pigs (n = 12). NA = Not applicable. Euthanize-sample = Euthanize and collect tissues from 10 originally inoculated pigs to assess persistence of infection.
biosecurity protocols across all study groups throughout the experiment.

**Infection and vaccination**—On day 0, all 320 pigs in groups A, B, C, and D were intranasally inoculated with 2 mL of cell culture fluid containing 10^7 TCID₅₀/mL of PRRSV VR2332 (cell culture passage 3). At 7 DPI, pigs in groups B, C, and D were vaccinated IM with 2 mL of a homologous modified-live PRRSV vaccine. Groups C and D were then revaccinated 30 days later (37 DPI), and a final dose was administered to group D at 67 DPI. Pigs in group E were sham inoculated intranasally with 2 mL of sterile saline (0.9% NaCl) solution (Table 1). Modified-live PRRSV vaccine and PRRSV VR2332 GenBank accession numbers are AF066183 and FRU87392, respectively. To monitor the protocols of infection and vaccination, 12 pigs from groups A to D and 6 pigs in group E were randomly selected and serum samples were collected in sterile vacuum tubes via jugular venipuncture at 0, 7, 14, 37, 67, 97, and 127 DPI.

**Assessment of transmission**—To determine whether different protocols of vaccination affected virus shedding, groups of 10 individually tagged PRRSV-naïve pigs were introduced 30 days after the last vaccination to each treatment group (Table 1). All sentinel pigs were removed and euthanized, and samples were collected 30 days after introduction. Transmission was defined as either detection of PRRSV nucleic acid in serum or tissues by use of RT-PCR procedures in 1 or more sentinel pigs or detection of anti-PRRSV antibodies as determined by positive results of ELISA that was subsequently confirmed by positive results of indirect fluorescent antibody test in 1 or more sentinel pigs.

**Assessment of persistence**—To evaluate whether different protocols of vaccination affected the proportion of PRRSV persistently infected pigs through time, 10 pigs/group were randomly selected and euthanized, and samples were collected 30 days following the last vaccination (Table 1). The study was terminated, and all remaining pigs were euthanized and sampled at 127 DPI. Serum and tissue samples were tested by use of RT-PCR assay for PRRSV nucleic acid. Tonsil, sternal lymph nodes, and superficial inguinal lymph nodes were collected in the processing plant, stored in separate plastic bags, and transported on ice to the laboratory for testing.

**Clinical and virologic response to heterologous challenge**—At 97 DPI, 10 randomly selected pigs from each group and 2 additional negative control pigs were transported to the isolation facilities of the College of Veterinary Medicine at the University of Minnesota. Each group was housed in 2 rooms (5 pigs/room) at a density of 1.2 m²/pig. Each isolation room had an independent ventilation system and a slurry pit to prevent cross-contamination of pathogens between rooms. All pigs in groups A, B, C, and D and 10 pigs from group E were intranasally inoculated with 2 mL of cell culture fluid containing PRRSV MN-184 isolate (10^8 TCID₅₀/mL). Challenge control PRRSV-naïve pigs were labeled as E+. The group of 2 additional negative control pigs was sham inoculated intranasally with 2 mL of sterile saline solution and labeled as E-. The challenge virus, PRRSV MN-184 (GenBank accession No. AY656992), a highly virulent isolate from a farm in southern Minnesota affected with severe reproductive disease and high sow mortality rate in 2001, was used at cell culture passage 2. The percentage divergence in the ORF 5 nucleotide sequence between MN-184 and VR2332 or the modified-live PRRSV was 15.3%.

Blood was collected from all 52 pigs at 0, 7, 14, and 21 DPI to evaluate viremia and antibody response. At 21 DPI, all pigs were euthanized and samples of tonsil, sternal lymph nodes, and superficial inguinal lymph nodes were collected in separate plastic bags. To evaluate the clinical response following heterologous challenge, rectal temperature, appetite, and mortality rate were measured at 0, 2, 4, 7, 10, 14, 17, and 21 DPI. Pigs were also weighed at 0 and 21 DPI, and ADG (g) per group was calculated. Rectal temperature (°C) was measured by the same person every sampling day between 8 and 9 AM. Every morning, the same operator measured the volume of feed still remaining from the previous day in each room and the percentage of reduction in feed intake was estimated (0% to 100%). Study personnel were unaware of group assignments.

**Diagnostic testing**—The PRRSV antibody response was evaluated by use of a commercial ELISA test. The presence of PRRSV nucleic acid in serum and tissues was determined by use of RT-PCR assay. Tissue samples collected at euthanasia were pooled by individual animal and 1 g was placed in 13 mL of lysis buffer in a sterile plastic tube. After homogenization with the appropriate equipment, samples were centrifuged (700 × g for 15 minutes) to remove disrupted cells and debris. Total RNA was extracted and purified with a commercial kit according to the manufacturer’s protocol from 200 µL of serum or 50 µL of the middle layer of homogenized tissue supernatant. The RNA was eluted in 50 µL of water, dried in a vacuum centrifuge, and rehydrated in 5 µL of water. Every sample was assayed in duplicate by use of 2 µL of the rehydrated sample in a 20-µL RT-PCR assay with primers and probe directed to the ORF 7 region of the North American PRRSV. All reactions were conducted in a real-time PCR instrument.

Extracted RNA from selected tissue samples with positive PCR assay results was submitted to the University of Missouri, Columbia Veterinary Diagnostic Laboratory to sequence the PRRSV ORF 5 region by use of a procedure modified from a published protocol. Amino acid sequence comparisons were used to differentiate wild-type from vaccine viruses.

Tissue RNA from pigs in the heterologous challenge phase of the study was tested by use of an isolate-specific PCR assay targeting a nucleotide sequence on the ORF 5 region of PRRSV MN-184. Total RNA was extracted as described. The PCR included the RT-PCR kit, forward primer (5′-TCACTTAAACGATTGATGGTCGAC-3′), reverse primer (5′-AAACATTGTACGAGCCGACC-3′), and probe (5′FAM-CTGGCTGAAACACATTITGTTGCGGACGACTTTCATC-LCTAMRA). A standard curve was developed for the quantitative RT-PCR procedure by preparing 10-fold dilutions of PRRSV MN-184 stock starting at 10^9 TCID₅₀/
mL. Results were reported in number of RNA copies (ie, RNAc) per gram of tissue.

Statistical analysis—The proportions of PRRSV persistently infected pigs were compared among groups within the same DPI by use of the Fisher exact test. The number of PRRSV RNA copies per gram of tissue was log transformed to stabilize the variance prior to analysis. Rectal temperature (°C), ELISA S:P ratio mean, ADG (g) and log 10 RNAc per gram of tissue were compared among groups by use of 1-way ANOVA. All analyses were performed with standard software. A value of P < 0.05 was considered significant.

Results

Viremia and antibody response in monitor pigs—Porcine reprodutive and respiratory syndrome virus nucleic acid was detected in serum at 7 DPI in 85% of monitor pigs from all inoculated groups (A to D). The proportion of viremic pigs decreased over time up to 67 DPI when all serum samples from monitor pigs in all groups yielded negative results of PCR assays. At 14 DPI, 90% of monitor pigs in all inoculated groups had positive results of ELISA (S:P ratio > 0.4). No significant differences in S:P ratio means were observed among inoculated groups during the first 97 DPI independently of the vaccination protocol; however, at 127 DPI, pigs in groups A and B had significantly (P = 0.008) higher S:P ratio mean values than pigs in groups C and D (Table 2). Negative control pigs (group E) had negative results of ELISA and PCR assay throughout the study.

Transmission to sentinel pigs—Transmission of PRRSV was detected at 37 to 67 DPI and 67 to 97 DPI in 85% of monitor pigs from all inoculated groups (Table 3). Between 97 and 127 DPI, sentinel pigs introduced into groups A (positive control pigs) and B became infected as determined by results of PCR assay or ELISA, whereas sentinel pigs in groups C and D remained uninfected.

Proportion of persistently infected pigs—The modified-live PRRSV vaccine did not reduce the proportion of persistently infected pigs at 37, 67, or 97 DPI (Table 3). However, at 127 DPI, the proportion of infected pigs was significantly reduced in vaccinated groups.

Twenty tissue samples that yielded positive results of PCR assay at 127 DPI from groups B, C, and D were submitted for nucleic acid sequencing of the ORF5 region. Seven samples yielded sufficient product for sequencing. In every sample, the sequence corresponded more closely to that of VR2332 wild-type virus than to that of MLV vaccine.

Clinical and virologic response to MN-184 challenge—Porcine reproductive and respiratory syndrome virus RNA was not detected in serum of previously infected pigs (groups A, B, C, and D) at 7, 14, or 21 days after MN-184 inoculation. Only the challenge controls (group E+) had a detectable viremia: 10 of 10 pigs at 7 DPI, 7 of 10 at 14 DPI, and 1 of 10 at 21 DPI. The mean S:P ratios among previously infected groups (A to D) before MN-184 inoculation were not significantly (P = 0.321) different. Seven days after MN-184 administration, all challenge controls (E+) yielded negative ELISA results. In contrast, previously exposed pigs had increased ELISA S:P ratios by day 7, without significant (P = 0.934) difference across these groups. One week later, every pig in group E+ yielded ELISA positive results and the S:P ratio means were not significantly (P = 0.864) different among all inoculated groups (A to E+). Similar results were obtained 21 days after MN-184 in-
occlusion (P = 0.965). Negative control pigs (E⁻) had negative results of ELISA and PCR assays during this phase of the study.

Isolate MN-184 was detected in tissue samples of 70% to 100% of the pigs from all challenged groups (A to E+) at 21 DPI. The proportion of infected pigs per group was not significantly affected by previous PRRSV exposure status. No significant (P = 0.366) difference among groups was detected in the mean number of PRRSV RNA copies per gram of tissue (Table 4).

Overall, pigs from groups that had been previously exposed to PRRSV had less severe clinical signs than newly infected pigs, 1 of which died 14 days after MN-184 challenge. It had fever (41.3°C), dyspnea, cyanosis of extremities, lethargy, and anorexia before death. Noncollapsed lungs and enlarged lymph nodes were the only remarkable lesions observed during necropsy. Interstitial pneumonia was detected via histologic examination, and PRRSV RNA was detected in lymph nodes and serum by use of RT-PCR assay. Although appetite was not affected in negative control pigs (E⁻) or groups A to D, a 5% to 15% mean reduction in feed intake was recorded in group E+ from 4 to 16 days after MN-184 inoculation. Pigs in group E+ also had significantly (P = 0.002) lower ADG than pigs in groups A, B, C, and D. Mean rectal temperatures in all groups were not significantly different before MN-184 inoculation (P = 0.071). Inoculated groups (A to E+) had significantly (P = 0.036) higher rectal temperatures than negative control pigs at 2 DPI; however, between 4 and 14 DPI, pigs in group E+ had significantly higher rectal temperature than previously infected (A to D) or negative control pigs (Table 4).

Discussion

The purpose of the study reported here was to understand the dynamics of endemic PRRSV infection in pig populations following the application of a modified-live PRRSV vaccine as an initial means to determine whether vaccination could be a potential tool in control or eradication programs or both. After inoculation of pigs with PRRSV VR2332, the infection and seroconversion pattern was consistent with previous observations. Various protocols of repeated vaccination with MLV did not affect the ELISA S:P ratio dynamics from 14 to 97 DPI; however, at 127 DPI, the 2 groups that received multiple vaccinations (C and D) had significantly lower S:P ratios than the positive control (A) and 1-dose group (B). These results are in accordance with previous reports that the commercial ELISA test detected an antibody response to initial infection but not to single or repeated homologous inoculations. In the present study, repeated vaccination reduced the variation in ELISA results, as reflected in the SE of the S:P ratio mean.

Another observation was that repeated vaccination (2 or 3 doses) reduced the duration of PRRSV transmission to sentinel pigs, perhaps because vaccinated groups had a significantly lower proportion of persistently infected pigs at 127 DPI. Despite the significant effect of the vaccination on viral persistence, the originally inoculated wild-type virus was not eliminated from the pigs in 127 days.

Although the reduction in persistence and duration of transmission by vaccination of infected populations may offer relevant information to improve PRRSV-control strategies such as herd closure and acclimation of gilts, results of the present study need to be interpreted with caution for a number of reasons. A limitation of the study was that the wild-type virus in the population was homologous to the commercially available MLV vaccine, which is not common. Because this experiment was part of a long-term study, the first logical step was to analyze the use of mass vaccination under homologous conditions to determine whether vaccination provided any benefit under optimum conditions. Detection of actively shedding pigs at 127 DPI was unexpected, and therefore, a longer trial may have provided more information. However, given the duration of shedding reported in the literature and the standard acclimation period used by the industry, it was expected that any evidence of vaccination effect on transmission or persistence would have been observed within 127 days. It is possible that performing mass vaccination on a population with low infection prevalence would reveal a larger benefit of mass vaccination than in the present experiment. Most of the prior reports of success of vaccination in controlling PRRSV resulted from a study that combined this strategy with herd closure. Constant introduction of sentinel pigs, which was necessary to assess transmission over time, may have allowed for re-infection of principal pigs. However, re-infection of pigs with a homologous PRRSV has not been reported.

The final phase of the study attempted to recreate the common situation of external PRRSV reintroduction to endemically infected swine herds to gain knowledge about the clinical and virologic responses against a highly

Table 4—Virologic and clinical variables in pigs after challenge inoculation with PRRSV MN-184.

<table>
<thead>
<tr>
<th>Group</th>
<th>Viremia</th>
<th>Seroconversion*</th>
<th>Mean ± SE log₁₀ RNAc/g</th>
<th>Deaths</th>
<th>Reduction in appetite</th>
<th>Mean ± SE ADG (g)</th>
<th>Hyperthermia†</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>—</td>
<td>7 DPI</td>
<td>3.26 ± 0.3*</td>
<td>—</td>
<td>—</td>
<td>510 ± 68*</td>
<td>2 DPI</td>
</tr>
<tr>
<td>B</td>
<td>—</td>
<td>7 DPI</td>
<td>3.81 ± 0.1*</td>
<td>—</td>
<td>—</td>
<td>603 ± 69*</td>
<td>2 DPI</td>
</tr>
<tr>
<td>C</td>
<td>—</td>
<td>7 DPI</td>
<td>3.67 ± 0.2*</td>
<td>—</td>
<td>—</td>
<td>717 ± 85*</td>
<td>2 DPI</td>
</tr>
<tr>
<td>D</td>
<td>—</td>
<td>7 DPI</td>
<td>3.62 ± 0.2*</td>
<td>—</td>
<td>—</td>
<td>574 ± 110*</td>
<td>2 DPI</td>
</tr>
<tr>
<td>E⁺</td>
<td>+</td>
<td>14 DPI</td>
<td>3.8 ± 0.3*</td>
<td>1 pig died 14 DPI</td>
<td>(+4–16 DPI)</td>
<td>208 ± 89*</td>
<td>2, 4, 7, 10, and 14 DPI</td>
</tr>
<tr>
<td>E⁻</td>
<td>—</td>
<td>—</td>
<td>0*</td>
<td>—</td>
<td>—</td>
<td>810 ± 54*</td>
<td>—</td>
</tr>
</tbody>
</table>

*Significant (P < 0.05) increase in group mean ELISA S:P ratio. †Group mean rectal temperature (°C) was significantly (P < 0.05) different from that of the negative control group on that DPI. RNAc = RNA copies. + = Detected. − = Not detected.

†Within a column, values with different superscripts are significantly (P < 0.05) different. See Table 1 for remainder of key.
virulent and heterologous challenge. After MN-184 challenge, only the challenge controls had viremia, whereas previously exposed pigs generated a rapid serologic response without evidence of viremia. The MN-184 virus was found in the tissues of nearly all pigs from all groups regardless of the previous PRRSV infection status. A low level of transient viremia may have occurred before the first sampling at 7 DPI because an antibody response and tissue infection were observed. Consistent with partial protection against infection, fewer clinical signs and enhanced growth performance were observed in all groups that were previously exposed to PRRSV.

Under the conditions of this study, the use of a modified-live PRRSV vaccine reduced the proportion of persistently infected pigs and the duration of shedding of wild-type virus in a population of pigs infected with a homologous PRRSV isolate. Previous exposure to PRRSV significantly improved the clinical response against a highly virulent heterologous challenge, but vaccination did not eliminate the wild-type virus from lymphoid tissues or prevent heterologous infection. These results offer a detailed and controlled evaluation of the benefits and risks of the use of PRRSV mass vaccination under controlled field conditions. Although some field experiences have been reported, to the authors’ knowledge, large-scale controlled experiments evaluating the effect of therapeutic vaccination on persistence, transmission, and clinical response against a heterologous PRRSV challenge have not been published. Although previous reports\textsuperscript{2,24} provide valuable information about the potential applications of therapeutic vaccination by interpreting field results, the present experiment included positive and negative control groups; intense diagnosis monitoring via modern diagnosis tools; and strict control of a number of factors such as infection time, feeding, genetics, health status, and management to analyze the results with scientific criteria. Further studies are needed to assess the effect of mass vaccination on PRRSV-infected populations with heterologous isolates under field conditions to further define the role of MLV vaccines in control-eradication programs.

References

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