Strain predominance following exposure of vaccinated and naive pregnant gilts to multiple strains of porcine reproductive and respiratory syndrome virus

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Abstract

Two studies were performed in order to test the relative ability of different strains of porcine reproductive and respiratory syndrome virus (PRRSV) to replicate and cross the placental barrier in pregnant gilts. Study 1 comprised 6 nonvaccinated gilts. Study 2 comprised 8 nonvaccinated gilts and 12 gilts that were vaccinated twice before conception. On, or about, gestation day 90 all gilts were simultaneously exposed to 20 field strains of PRRSV (all strains were distinguishable by restriction fragment length polymorphism (RFLP) patterns). Gilts of study 1 were euthanized on day 7 postpartum. Gilts of study 2 were euthanized, or about, gestation day 111. All gilts, pigs, and fetuses were tested for the presence and type of strain of PRRSV. Of 128 samples shown to contain PRRSV, 118 contained a single strain, 4 contained 2 strains, and 2 contained a strain or strains for which the RFLP pattern was undecipherable. Only 8 of the 20 strains were isolated from nonvaccinated gilts and their litters. And only 2 of the 20 strains (notably 2 of the same strains isolated from nonvaccinated gilts and their litters), were isolated from vaccinated gilts and their litters. Moreover, 1 of the 2 strains accounted for most (31 of 37; 84%) of the isolates from the vaccinated group. Collectively these results indicate that strains differ in their ability to replicate in pregnant gilts and cross the placental barrier. And they suggest that maternal immunity, although sometimes insufficient to prevent transplacental infection, can exert additional selective pressure.

Résumé

Deux études ont été réalisées afin de vérifier la capacité relative de différents isolats du virus dysgénique et respiratoire porcin (PRRSV) à se repliquer et traverser la barrière placentaire chez des cochettes gestantes. Dans l’étude 1, 6 cochettes non-vaccinées ont été utilisées, alors que dans l’étude 2 on utilisa 8 cochettes non-vaccinées et 12 cochettes vaccinées à 2 occasions avant la conception. Le, ou aux environs du 90e jour de gestation, toutes les cochettes ont été exposées simultanément à 20 isolats cliniques de PRRSV, tous pouvant être différenciés par le polymorphisme du patron des fragments obtenus à l’aide d’enzymes de restriction (RFLP). Les cochettes de l’étude 1 ont été euthanasiées au jour 7 post-partum. Les cochettes de l’étude 2 ont été euthanasiées le, ou aux environs du jour 111 de gestation. Toutes les cochettes, porcs et fœtus ont été soumis à une analyse pour vérifier la présence et le type de PRRSV isolé. Des 128 échantillons à partir desquels on dêecta la présence de PRRSV, 118 contenaient une seule souche, 4 contenaient 2 souches et 2 contenaient une ou des souches pour lesquelles le patron de RFLP était indéchiffrable. Seulement 8 des 20 souches ont été isolées des cochettes non-vaccinées et de leurs portées. Seulement 2 des 20 souches (notamment 2 des mêmes souches isolées à partir des cochettes non-vaccinées et de leurs portées) ont été isolées des cochettes vaccinées et de leurs portées. De plus, une des 2 souches comptait pour la plupart (31/37; 84 %) des isolats provenant des animaux vaccinés. Dans l’ensemble, ces résultats indiquent que les souches diffèrent quant à leur capacité à se repliquer chez des cochettes en gestation et à traverser la barrière placentaire et ils suggèrent que l’immunité maternelle, bien que parfois insuffisante pour prévenir l’infection trans-placentaire, peut exercer une pression sélective supplémentaire.

Introduction

In 1994 the first vaccine for porcine reproductive and respiratory syndrome (PRRS) became commercially available. It comprised a moderately virulent field strain of PRRS virus (PRRSV) that had been attenuated by serial passage in cell culture. Early reports indicated that it, and other attenuated-virus vaccines developed soon thereafter, were effective in preventing the reproductive facet of PRRS (1–3). However, more recent observations, especially those made since the emergence of atypical or acute PRRS in the fall of 1996 (4,5), have emphasized that vaccine-induced immunity is sometimes
The objective of study 1 was to evaluate the consequences of a 20-strain PRRSV challenge in naive pregnant gilts. Six gilts (identified as group A), purchased from a commercial, specific-pathogen-free herd (farm A) were included in this study. During the past several years the source herd had been repeatedly tested and found free of antibody for PRRSV. Each gilt was tested and found free of antibody for PRRSV immediately before and after arrival at our research facility (National Animal Disease Center, NADC). They were bred to 2 boars that had been purchased from the same herd. At, or about, gestation day (GD) 90 they were exposed simultaneously to 20 field strains of PRRSV. Blood was collected from each gilt just prior to exposure, at 7 d after exposure (GD 97), and immediately after farrowing. The gilts were kept in elevated farrowing crates in isolation rooms. Just before farrowing the Likewise, the rear-lateral portions of the farrowing crate. Plastic boxes were positioned below to catch the piglets during the farrowing interval (thus preventing them from sucking before blood samples were obtained). Each pig was identified by ear notches (the ear notcher was sanitized between each pig) and a blood sample was collected. The deck was replaced and a sample of colostrum was collected from the gilt prior to placement of her piglets. Each gilt and her litter were euthanized at, or about, 7 d post-partum by intravenous administration of pentobarbital. Blood and lung lavage fluid were collected from each gilt and pig at necropsy.

The objective of study 2 was to evaluate the effect of vaccination against a 20-strain PRRSV challenge in pregnant gilts. This study included 2 boars and 20 randomly-selected gilts that were raised on farm A until they were moved in 2 groups, at different times, to a separate site (farm B). No swine had been kept on farm B for several years and it was at least 3 km from any other farm where swine were present. The 1st group (group 1) of study 2 that was moved from farm A to farm B comprised 12 gilts. At farm B they were housed in 1 pen of a 4-pen open-front barn. Within the barn, pens were separated by concrete-block walls that were about 1 m high, but all pens shared common air space. Moreover, each pen opened to an outside run that was separated from the adjacent run by only a large-mesh woven wire fence. All gilts of group 1 were vaccinated twice intramuscularly with 2 mL of a commercial attenuated-virus vaccine (RespPRRS/Repro; Boehringer Ingelheim Animal Health, Inc. Saint Joseph, Missouri, USA) on day 0 (day of delivery to farm B) and again on day 28.

The 2nd group (group 2) moved from farm A to farm B comprised the remaining 8 gilts and 2 boars. They were moved on day 56 (28 d after the 2nd vaccination of group 1). At farm B they were placed in the 3 vacant pens of the same barn that housed gilts of group 1. The arrangement of gilts and boars in the barn was as follows: group 1 gilts-boar-boar-group 2 gilts. Group 2 gilts and boars were not vaccinated. Both boars were used for breeding both groups of gilts, and so both boars made frequent direct contact with both vaccinated and nonvaccinated gilts. Moreover, gilts and boars in adjacent pens (runs) also made direct and frequent contact on a daily basis. The first 4 nonvaccinated and 6 vaccinated gilts that came into estrus were bred and identified as groups B and D, respectively. After a delay of 90 d the remaining 4 nonvaccinated and 6 vaccinated gilts were bred and identified as groups C and E, respectively.

All gilts and boars of study 2 were bled at the time of delivery to farm B and at selected times thereafter. At, or about, GD 85, gilts were moved to NADC and acclimated to farrowing crates in isolation facilities. At, or about, GD 90, they were exposed simultaneously to 20 field strains of PRRSV. Exposure of gilts of groups B and D was at, or about, day 160 postvaccination, and exposure of gilts of groups C and E was at, or about, day 250 postvaccination. Gilts were euthanized 21 d after exposure and necropsied (GD 111). Blood and lung lavage fluid were collected from each gilt, blood was collected from all live fetuses, and thoracic fluid or lung tissue was collected from all dead fetuses that were believed to have died after exposure of the gilt to field virus. At the conclusion of breeding at farm B both boars were transported to the NADC and used for additional breeding purposes during an interval of about 6 mo. During that time they were bled periodically.

The animal studies were conducted in accordance with the guidelines of the Guide to the Care and Use of Experimental Animals of the Canadian Council on Animal Care and the NADC Institutional Animal Care and Use Committee. For virus challenge each gilt was restrained with a snare attached to the top of the farrowing crate in a way that her head (snout) was maintained in an elevated position. A 20 mL volume of inoculum, about 10 mL per nostril, was
slowly administered intranasally. The gilt was restrained for an additional 10 s and then released. Abortion was defined as expulsion of fetuses prior to GD 112. Transplacental PRRSV infection defined as detection of virus in one or more fetuses within a litter.

**Virus and antibody identification**

Blood (serum) and other fluids and tissues were tested for virus and antibody, as previously described (9,10).

**Virus**

The inoculum used to expose (challenge) vaccinated and nonvaccinated gilts comprised 20 strains of PRRSV (identified as numbers 1 through 20 for these studies) collected from suspected epidemics of PRRS in North America between 1989 and 1994. Of the 20 strains, 17 were kindly provided to us by Dr. Merwin Frey and Mr. John Landgraf of the National Veterinary Services Laboratory, Ames, Iowa, and 3 were from our repository. Two of these 3 viruses have been tested extensively in our laboratory and strains 1 and 6 are known as NADC-8 (9,11–16) and NADC-9 (7) strains of PRRSV, respectively. Each of the 20 strains was genetically distinct based on nucleotide sequencing of open reading frame 5 (17) and each strain could be differentiated from other strains by RFLP (6). All strains were serially passaged 3 times in MARC-145 cells in our laboratory. The 3rd passage of each strain was titrated for infectivity in MARC-145 cells, range of titers was from 10^5 median cell culture infectious units (CCID50)/mL to 2.5 x 10^8 CCID50/mL. The PRRSV challenge inoculum was produced by pooling about 3 x 10^7 CCID50 of each PRRSV strain and adding minimal essential medium to the virus solution to produce a total volume of 600 mL. This virus solution was aliquoted into 30 challenge doses, each challenge dose contained 20 mL with 1 x 10^8 CCID50 of each virus strain. Each of the virus challenge strains was distinguishable from the attenuated vaccine virus used in study 2 by RFLP.

**Statistics**

The Fischer exact test (18) was used to evaluate the differences between immunized and nonimmunized gilts in the incidence of virus-positive lung lavage fluid and transplacental infection. Results were considered significant at P < 0.05.

**Results**

Gilts in groups A, B, and C were treated similarly; for example, nonvaccinated controls were exposed to PRRSV at, or about, GD 90 to evaluate the effect of a 20 PRRSV strain challenge during late gestation. Results from the nonvaccinated controls were compared to results from groups D and E, the vaccinated gilt group that was similarly challenged with the 20 PRRSV strains at, or about, GD 90. Differences between nonvaccinated and vaccinated gilts were attributed to vaccination.

**Clinical signs of infection**

No clinical signs were observed in the gilts except for abortion. One gilt in each of groups A, C, and D aborted on days 18, 16, and 7, respectively, and 2 gilts aborted in group E on day 18 and 19 after the virus challenge. Other presumed consequences of infection were evident for group A, namely, 2 litters contained weak-born pigs that either grew poorly or died soon after birth (both litters contained congenitally infected pigs), and 1 litter, delivered at term, contained only fetuses that had died in utero.

**Isolation of PRRSV**

As shown in Table I, virus was isolated from: 1) serum (collected at day 7) of 4, 2, and 2 of the nonvaccinated gilts of groups A, B, and C, respectively; 2) lung lavage fluids (collected at necropsy) of all 14 of the nonvaccinated gilts (groups A, B, and C) and 2 (group D) of the 12 vaccinated gilts (groups D and E); 3) colostrum (collected on the day of farrowing) of 1 gilt of group A (data not shown); 4) serum (collected at the time of farrowing) of 1 or more pigs of 5 out of 6 litters of group A; 5) serum (collected at necropsy) of 1 or more fetuses of 4, 5, 3, and 4 litters of groups B, C, D, and E, respectively; and 6) serum (collected 7 d after farrowing) from an additional 6 pigs of group A where the virus was not identified on the day of farrowing. Virus was not isolated from any of the other samples collected during the studies (Table I).

There was a significant difference (P < 0.0001) between nonvaccinated and vaccinated groups (for example, groups B and C versus groups D and E) of study 2 in regard to the prevalence of virus isolated from lung lavage fluids at necropsy, but not in regard to the incidence of transplacental infection.

**Restriction fragment length polymorphism analysis**

Only 8 of the 20 field strains of PRRSV that the gilts were exposed to, were identified by RFLP in the total of 124 virus-positive samples from gilts, pigs, and fetuses. These were strains 1, 5, 6, 9, 10, 12, 15, and 19 (Table II). The vaccine-virus RFLP pattern was not detected in any of the virus isolates for which RFLP patterns could be determined. The most commonly identified strain was strain 12, which was present in more than half of the virus-positive samples. Most samples contained only 1 detectable strain, however, 2 samples contained 2 strains, and 4 samples appeared to contain a mixture of strains that were undecipherable by RFLP. Only a single strain was detected in 12 of the 19 litters shown to be transplacently infected. Two or more strains were detected in the remaining 7 litters with most individual pigs and fetuses infected with only a single strain.

**Serology**

In study 1, all gilts in group A were seronegative prior to exposure to field virus and all developed PRRSV-specific antibodies following exposure (data not shown). In study 2, all vaccinated gilts of groups D and E were seronegative before vaccination and all developed PRRSV-specific antibody following vaccination. All nonvaccinated gilts of groups B and C remained seronegative until after they were moved to the NADC and exposed to the field virus. Antibody levels for groups B, C, D, and E, as determined by ELISA (Herdcheck; IDEXX, Westbrook, Maine, USA), are summarized in Table III. Boars remained seronegative throughout the study and during the additional time they were housed at the NADC (data not shown).

**Discussion**

Our finding that several of the strains of PRRSV tested in this study predominated during replication in pigs was consistent, in
principle, with previous studies where pigs were simultaneously exposed to strains that were either known (7) or presumed (19) to differ in relative virulence. Eight of the 20 PRRSV strains administered to the non-immunized control gilts (groups A, B, and C) were detected, indicating that these strains had the greatest propensity for replicating in vivo. In contrast, the remaining 12 strains were not detected, which could be due to one or more of the following possibilities: the sensitivity of our methods, some virus strains could not establish infection because of attenuation by 3 passages in cell culture, there was competitive inhibition for infection at the virus/receptor level due to the large volume of virus given, or there was competition for in vivo sites of replication.

We believe competition for in vivo sites of replication may play a major role in determining which virus predominates in vivo because: 1) our methods can detect as little as a few virions per mL of serum or lung lavage (20); 2) based on previous reports, several cell culture passages do not dramatically alter pathogenicity (9,11–15,21–23); 3) there must be a substantial number of viral receptors in the oronasal cavity since pigs have become infected with as few as 20 cell culture infectious virions administered intranasally (24); and 4) based on results reported here, it appears 1 strain may be more virulent than others since 37 of the 87 virus isolates recovered from nonvaccinated challenged-gilts were the strain 12. In addition, there appears to be no relationship between a PRRSV strain's ability to replicate in vitro and the chance of isolating that strain post challenge, since 8 of the 20 PRRSV strains recovered in this study reflected the spectrum of in vitro replication (Table II).

Immunized gilts were partially protected; for example, as a group gilts were protected against challenge based on little or no virus replication detected in them; however, they were not protected against reproductive failure since there was no significant difference in the incidence of transplacental infection between vaccinated and nonvaccinated gilts groups. Results from this study are comparable with previous reports where, as a group, swine immunized with a vaccine or field virus were clinically protected from a homologous field virus challenge (1–3,9,13), but following a heterologous field virus challenge, had incomplete or partial clinical protection against transplacental infection (1,3,15). Collectively, the body of work describing protective immunity in pregnant swine suggests this animal model may be very sensitive since it can take only 1 infectious virus to cross the placenta and induce reproductive failure.

At the time of challenge, some of the vaccinated gilts in group D and E would be considered seronegative; for example, the gilts had an ELISA S/P ratio < 0.4, a value that has been set by the ELISA kit's manufacturer as the threshold between negative and positive. However, in the case of the vaccinated gilts, the S/P ratios less than 0.4 should be considered specific or positive since all gilts were known to be seronegative prior to vaccination (S/P values of 0.0), all seroconverted after vaccination (S/P values > 0.4), and the low S/P ratios in groups D and E on GD 90 (Table III) indicate a decay in antibody titers postvaccination. The increase in S/P ratios on GD 97 for the vaccinated gilts in comparison to the nonvaccinated gilts indicates a priming of their immune system by vaccination that is reflected through GD 104 and 111. Only 2 (#12 and #15) of the 8 strains shown to have a propensity to replicate in the non-vaccinated group were detected in the vaccinated group and, based on the incidence of RFLP patterns, it appears that there is a higher incidence of the #12 strain in the vaccinated group (31 out of 37 isolates; 84%) compared to the nonvaccinated group (37 out of 87; 43%). This apparent difference may be due to a lack of cross-protection between the vaccine and the #12 strain, since 6 of the strains that were detected in the nonvaccinated group were not detected in the vaccinated group. There are at least 2 possibilities for lack of cross protection that merit discussion. First, the exposure (challenge) dose was especially high in terms of infectious units and may have simply overwhelmed

### Table I. Virus isolation results following challenge exposure of gilts in studies 1 and 2

<table>
<thead>
<tr>
<th>Group</th>
<th>n</th>
<th>DPV</th>
<th>Serum Necropsy</th>
<th>Lung lavagea</th>
<th>Parturition/21 d post challenge</th>
<th>Necropsy</th>
</tr>
</thead>
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<tr>
<td>Study 1b</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>A</td>
<td>6</td>
<td>d</td>
<td>6 (4)c</td>
<td>6 (0) 6 (6)</td>
<td>(5) 26 (14) 7 (4) 34 (3)</td>
<td>20 (12)</td>
</tr>
<tr>
<td>B</td>
<td>4</td>
<td></td>
<td>4 (2)</td>
<td>4 (0) 4 (4)</td>
<td>(4) 42 (21) 0 0</td>
<td>0</td>
</tr>
<tr>
<td>C</td>
<td>4</td>
<td></td>
<td>4 (2)</td>
<td>4 (0) 4 (4)</td>
<td>(3) 27 (17) 14 (6)</td>
<td></td>
</tr>
<tr>
<td>D</td>
<td>6</td>
<td>130</td>
<td>6 (0) 6 (2)</td>
<td>6 (0) 6 (2)</td>
<td>(3) 53 (7) 4 (0)</td>
<td></td>
</tr>
<tr>
<td>E</td>
<td>6</td>
<td>245</td>
<td>6 (0) 6 (0)</td>
<td>6 (0) 6 (0)</td>
<td>(4) 67 (22) 13 (6)</td>
<td></td>
</tr>
</tbody>
</table>

DPV — Days postvaccination when gilt was challenged with an inoculum of 20 porcine reproductive and respiratory syndrome virus (PRRSV) strains on gestation day (GD) 90. Serum collected from gilt on GD 97 and at the time of gilt necropsy (7 d postpartum in group A and GD 111 in groups B-E)

TP — transplacental infection: number of litters tested for fetal PRRSInfection (number of litters with transplacental infection)
a Lung lavage collected at gilt necropsy
b In study 1, condition of pigs at time of birth reported as live-born, stillborn, or died in utero. Live pigs euthanized and necropsied 7 d after birth
c In study 2, condition of fetuses at time of gilt necropsy reported as live or dead
d Not applicable
e Number of animals tested for virus (number positive)
a preexisting immunity that would have been protective under other less demanding conditions (16). It certainly far exceeded what might be expected from natural exposure. Second, simultaneous exposure to 20 somewhat genetically different strains may appreciably increase the likelihood of vaccine failure. For example, the vaccine might have provided complete protection against any one strain by itself and perhaps against many strains if they were antigenically similar. If so, a multi-strain exposure with strains currently present in a particular geographic area might be a better predictor of overall vaccine efficacy. It might also explain the different experiences and opinions on the value of vaccination for PRRS.

We were able to characterize the predominate population of virus in 118 of the virus isolations as a single virus strain, in 2 isolations as a mixed infection with 2 strains each, and in 4 isolations there was an indeterminable RFLP pattern. The indeterminable patterns may represent 3 or more virus strains, which would make RFLP analysis difficult, or they may represent an event in ORF 5 that affects our ability to read the RFLP pattern, such as, recombination, a series of nucleotide mutations affecting the restriction enzyme digestion sites, or both. Additional studies are warranted to fully evaluate this possibility and decipher the indeterminable RFLP patterns. Moreover, the fact that strain 12 was isolated more often than all other strains combined with an apparent lack of cross-protection makes its further characterization of particular interest.

Although study 2 was not specifically designed to test horizontal PRRSV transmission, the housing of vaccinated gilts, naive

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**Table II. Incidence of restriction fragment length polymorphism (RFLP) patterns by gilt group**

<table>
<thead>
<tr>
<th>RFLP pattern</th>
<th>Virus titer&lt;sup&gt;b&lt;/sup&gt;</th>
<th>Controls</th>
<th>Vaccinates</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>A</td>
<td>B and C</td>
<td>D and E</td>
</tr>
<tr>
<td>1</td>
<td>$1.6 \times 10^6$</td>
<td>9&lt;sup&gt;c&lt;/sup&gt;</td>
<td>13</td>
<td>—</td>
</tr>
<tr>
<td>5</td>
<td>$1.0 \times 10^7$</td>
<td>2</td>
<td>7</td>
<td>—</td>
</tr>
<tr>
<td>6</td>
<td>$3.2 \times 10^8$</td>
<td>—</td>
<td>3</td>
<td>—</td>
</tr>
<tr>
<td>9</td>
<td>$7.9 \times 10^8$</td>
<td>—</td>
<td>2</td>
<td>—</td>
</tr>
<tr>
<td>10</td>
<td>$1.3 \times 10^7$</td>
<td>—</td>
<td>1</td>
<td>—</td>
</tr>
<tr>
<td>12</td>
<td>$5.0 \times 10^5$</td>
<td>14</td>
<td>23</td>
<td>31</td>
</tr>
<tr>
<td>15</td>
<td>$3.2 \times 10^6$</td>
<td>1</td>
<td>2</td>
<td>5</td>
</tr>
<tr>
<td>19</td>
<td>$2.5 \times 10^8$</td>
<td>1</td>
<td>4</td>
<td>—</td>
</tr>
<tr>
<td>Mixed</td>
<td>—</td>
<td>1</td>
<td>1</td>
<td>—</td>
</tr>
<tr>
<td>?</td>
<td>—</td>
<td>1</td>
<td>2</td>
<td>1</td>
</tr>
</tbody>
</table>

<sup>a</sup> Virus isolations recorded in Table I characterized by RFLP technique that differentiates the 20 challenge procine respiratory and reproductive syndrome virus (PRRSV) strains; RFLP pattern corresponds to challenge strain identification number. Gilt group determined by PRRSV exposure history (see text for details)

<sup>b</sup> Titer of in vitro propagated virus used to produce pooled challenge virus inoculum, reported in log<sub>10</sub> values

<sup>c</sup> Number of isolates that had a specific strain RFLP pattern

Mixed — apparent mixed infection with 2 different strains

? — RFLP indeterminable

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**Table III: Serology results for gilts in study 2 following virus challenge at gestation day (GD) 90**

<table>
<thead>
<tr>
<th>Group&lt;sup&gt;a&lt;/sup&gt;</th>
<th>GD</th>
<th>90</th>
<th>97</th>
<th>104</th>
<th>111</th>
</tr>
</thead>
<tbody>
<tr>
<td>B</td>
<td></td>
<td>0.01&lt;sup&gt;b&lt;/sup&gt;</td>
<td>0.11</td>
<td>0.94</td>
<td>0.94</td>
</tr>
<tr>
<td></td>
<td>(0.00 – 0.04)</td>
<td>(0.01 – 0.18)</td>
<td>(0.53 – 1.27)</td>
<td>(0.64 – 1.37)</td>
<td></td>
</tr>
<tr>
<td>D</td>
<td></td>
<td>0.45</td>
<td>1.99</td>
<td>2.32</td>
<td>2.14</td>
</tr>
<tr>
<td></td>
<td>(0.22 – 1.13)</td>
<td>(0.91 – 2.29)</td>
<td>(1.51 – 2.82)</td>
<td>(1.39 – 2.75)</td>
<td></td>
</tr>
<tr>
<td>C</td>
<td></td>
<td>0.00</td>
<td>0.07</td>
<td>1.13</td>
<td>0.91</td>
</tr>
<tr>
<td></td>
<td>(0.00 – 0.00)</td>
<td>(0.00 – 0.18)</td>
<td>(0.74 – 1.51)</td>
<td>(0.74 – 1.19)</td>
<td></td>
</tr>
<tr>
<td>E</td>
<td></td>
<td>0.37</td>
<td>2.22</td>
<td>2.62</td>
<td>2.36</td>
</tr>
<tr>
<td></td>
<td>(0.18 – 0.55)</td>
<td>(1.03 – 2.67)</td>
<td>(2.25 – 3.02)</td>
<td>(2.08 – 2.91)</td>
<td></td>
</tr>
</tbody>
</table>

GD — gestation day

<sup>a</sup> Groups B and C were nonvaccinated controls and groups D and E were vaccinated about 160 and 250 d before challenge on GD 90, respectively

<sup>b</sup> ELISA S/P — Average value for group (range of S/P ratio for group)
gilts, and boars at the same site (farm B) in the same open barn over a period of several months, and with varying degrees of direct and indirect contact between the vaccinated and nonvaccinated groups, reflects on this issue. The absence of seroconversion of any of the nonvaccinated gilts or boars suggests that age and time may be especially critical factors relative to the spread of vaccine virus. In addition to the lack of seroconversion in the nonvaccinated gilts and boars, is the lack of any vaccine-like virus isolate recovered from these studies. This supports the observation of no horizontal virus transmission, as well as no apparent vertical transmission of vaccine virus months after vaccination of the dam. The 2 points that we believe require special emphasis are: 1) naive gilts and boars were not brought to farm B until 1 mo after gilts at that site were vaccinated a 2nd time; and 2) all of the gilts and both boars were at least 7 mo of age at the time they first inhabited the same barn. We emphasize that these results were not unique in that they were consistent with several of our previous observations (unpublished) and the observation of others regarding the transmission of PRRSV among adult swine (25). Moreover, the same barn has been used for cohabitation of vaccinated and naive gilts and boars since the completion of the studies reported here, again without any evidence of virus transmission. In contrast to our experiences, reports of vaccine virus readily spreading, even from farm to farm (26), points out the gap that still exists in our knowledge of the epidemiology of PRRS.

In conclusion, gilts are capable of replicating at least several PRRSV strains following a single inoculation with multiple PRRSV strains. This process may serve as an in vivo selection method for the more virulent strains contained within the inoculum. Gilts immunized with attenuated PRRSV can develop cross-protection that will protect them against genetically distinct virus strains; however, this cross-protection is not perfect and is probably dependent on many factors, one of which may be the virulence of the challenge virus. The in vivo selection for more virulent strains may help explain epizootics of PRRS that occur in seropositive herds; for example, a PRRSV strain enters the herd that is sufficiently virulent to override the immunity of either vaccinated or naturally infected animals. This scenario stresses the importance of preventing inter-herd PRRSV transmission.

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Reference


