Effect of PRRS vaccination on average daily gain: A comparison of intranasal and intranasal-intramuscular administration

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Summary

Purpose: To evaluate the effects of two extra-label vaccination (RespPRRS®, NOBL Laboratories, Inc.) procedures against porcine reproductive and respiratory syndrome (PRRS) on average daily gain (ADG) in a commercial offsite nursery.

Methods: Neonatal pigs were assigned to one of three treatment groups at each of 10 offsite sow farms, so that all pigs born in each of three rows of crates in the farrowing room received the same treatment. One group (controls) received no vaccine (n ≈ 5400 pigs); a second group received 1 mL PRRS vaccine intranasally (IN) at 5–7 days of age (n ≈ 5400 pigs); a third group received 1 mL PRRS vaccine IN at 5–7 days of age plus a second 1-mL dose intramuscularly (IM) just prior to weaning at approximately 17 days of age (n ≈ 5400). At weaning, pigs were moved to an offsite nursery, where they were sorted by entry weight (small, medium, and large) and treatment group, and placed in pens of 22 pigs each. Nine pens per room were randomly selected to be monitored in the study; one pen from each treatment and size group. Weight gains were recorded weekly for each pen of pigs and individually for one ear-tagged pig per pen. Serum samples were collected from all tagged pigs on days 0, 28, and 49 and assayed for antibodies (ELISA) and virus. All viruses isolated were characterized by restriction fragment length polymorphism (RFLP). A total of 51 pigs and pens were included in each treatment group. Seventeen rooms were selected for the study for a total of 153 pigs and pens.

Results: No significant differences in weight gains were observed among the treatment groups (*P* =.49), indicating that vaccinated pigs performed no better than nonvaccinated pigs. Nor did route of administration (IN versus IN/IM) have an effect on ADG in this study. Pigs with larger entry weight gained significantly faster than smaller pigs (*P* < .0001). There was no significant interaction between treatment and entry size in this trial (*P* = .24). Nearly all pigs seroconverted to PRRSV, including the nonvaccinated control pigs. Viruses whose RFLP profile was compatible with RespPRRS®/2332 virus were isolated from nonvaccinated control pigs, as well as vaccinated pigs. Viruses whose RFLP profile was not compatible with RespPRRS®/2332 virus were also found in both vaccinates and nonvaccinates. Thus, differentiation of viruses indicated a probable spread of vaccine virus from vaccinated pigs to nonvaccinated pigs with concurrent circulation of field virus in both groups.

Implications: In this commercial production system, vaccination with a PRRSV vaccine was not observed to have an effect on ADG using either of two extra-label protocols. Nonvaccinated pigs housed in proximity to vaccinated pigs became infected with vaccine virus.

Keywords: swine, porcine reproductive and respiratory syndrome, PRRS, average daily gain, vaccine, vaccination, RFLP

Porcine reproductive and respiratory syndrome (PRRS) causes production and financial losses in swine herds worldwide.[1–3] Several researchers have reported that PRRS virus (PRRSV) infection may have an adverse effect on nursery performance.[4,5] Kerkaert, et al.,[6] reported that chronic PRRSV infections in nurseries can reduce total profits up to 70%, with the losses due principally to decreased performance rather than increased mortality. Polson, et al.,[5] observed that the financial losses that resulted from PRRSV infection varied according to mortality rates and duration of the disease episode. Nursery losses in the model ranged from $0.73–$18.21 per head. Mortality and the number of disadvantaged pigs can be increased two- or threefold, and average daily gain (ADG) may be decreased by as much as 25%–50% in the face of chronic PRRSV infections.[1,5,7–9] Dee[9] reported that ADG in PRRSV-infected nurseries was reduced by 0.22–0.33 lb (0.48–0.73 kg).[8,10,11] Chronic PRRSV infections have been reported to reduce ADG of growing pigs by as much as 15%. These losses have prompted researchers and practitioners to search for methods to control the clinical effects of the disease.
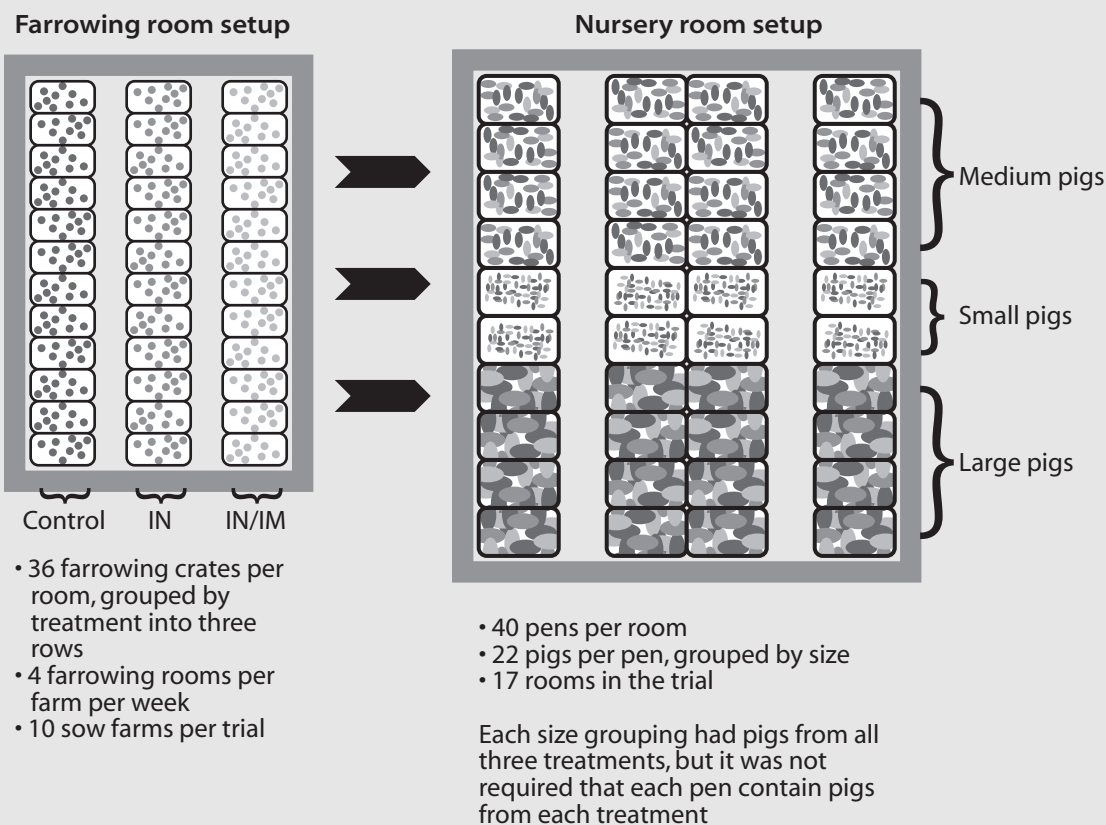
Two general strategies have been used to maintain productivity and minimize financial losses in herds endemically infected with PRRSV:

- eliminating the virus from selected populations within the herd, and
- implementing management techniques designed to reduce the clinical effects of the infection.

Attempts to eliminate PRRSV in off-site nurseries by using three-site production has met with varied success. Dee, et al.,[9] described a

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Farrowing room setup

Nursery room setup

Medium pigs

Small pigs

Large pigs

Control IN IN/IM

- 36 farrowing crates per room, grouped by treatment into three rows
- 4 farrowing rooms per farm per week
- 10 sow farms per trial

- 40 pens per room
- 22 pigs per pen, grouped by size
- 17 rooms in the trial

Each size grouping had pigs from all three treatments, but it was not required that each pen contain pigs from each treatment

Farrowing room and nursery setup

strategy that combined depopulating the nursery and stabilizing virus spread in sow herds. This procedure has been successful in many herds. Depopulating the nursery and thoroughly disinfecting facilities appeared to be cost effective due to improved growth rate, increased number of marketable pigs, and decreased mortality rates, even in herds in which virus was not successfully eliminated.[12]

Because it is difficult to eliminate the virus from the nursery, many individuals have concentrated on management techniques that reduce the impact of secondary infections. One example is the McREBEL™ (Management Changes to Reduce Exposure to Bacteria to Eliminate Losses from PRRS) procedure.[13] McREBEL is aimed at reducing PRRS-related losses that are due to secondary bacterial infections, not at eliminating the virus from the herd. This type of management practice may be coupled with the use of vaccine. An economic analysis of control strategies performed by Mousing[14] concluded that vaccinating replacement breeding animals in addition to vaccinating the weaned pigs was the most cost effective of the vaccination strategies examined for the control of PRRS in Denmark.

The use of an autogenous inactivated vaccine and a modified-live vaccine (RespPRRS®, NOBL Laboratories, Inc., Sioux Center, Iowa) have been evaluated for their effect on improving performance of nursery pigs. Gillespie[15] used a modified-live vaccine in neonatal pigs to control PRRS in the nursery and observed reduced mortality rates in the

nursery. Trayer[16] used the same vaccine in a labeled and extra-label manner and observed similar results. Sanford[17] reported that ADG and days-to-market were improved in vaccinated pigs compared to nonvaccinates. McCaw and Xu[18] speculated that intranasal (IN) vaccination might be beneficial in obtaining a local as well as systemic immune response in pseudorabies (Aujeszky's disease) vaccination, and we speculated that a similar immune response might be evoked with IN PRRSV vaccination.

The objective of the present study was to evaluate the use of RespPRRS® vaccine in an endemically infected commercial nursery. Our aim was to determine whether the use of RespPRRS® vaccine was improved ADG by using either of two extra-label vaccination protocols (IN administration or IN plus intramuscular [IM] administration) in this particular system. We chose the treatments based on discussions with swine practitioners concerning common use of the vaccine. We also analyzed serological responses and virus isolation patterns to determine their association with weight gains during the 7-week period the pigs were in the nursery.

Materials and methods

Facilities and population

The study was carried out on a commercial hog farm that used three-site production. The production system consisted of sow farms that

had separate breeding, gestation, and farrowing facilities, and off-site commingled nurseries and off-site finishing facilities. All sow herds contributing pigs to the nursery had been shown to be serologically positive to PRRSV prior to vaccination. Reproductive signs of PRRS had not been observed in the system prior to or during the study. PRRS had been diagnosed in a few neonatal pigs that demonstrated clinical signs of substandard weight gain. The nurseries were also infected with PRRSV, with clinical signs of respiratory disease and reduced weight gains. Reductions in weight gain and respiratory signs were diagnosed as PRRSV. *Streptococcus suis* and *Haemophilus parasuis* were also diagnosed in the nurseries. The clinical signs were more pronounced in small entry-weight pigs than in heavier entry-weight pigs.

Approximately 1 year before the study, the farm began a vaccination schedule to reduce the spread of PRRSV to neonatal pigs. The vaccination strategy called for all replacement breeding stock to receive a 2-mL dose of vaccine on arrival at the sow farm.

Experimental design

The sow farms contributing pigs to the nursery had farrowing rooms with three rows of farrowing crates (Figure 1). Each row consisted of 12 farrowing crates, and all pigs born in a row were assigned to one of three treatment groups:

- control pigs, which received no vaccine;
- pigs that received 1 mL PRRS vaccine intranasally at 5–7 days of age; or
- pigs that received 1 mL PRRS vaccine intranasally at 5–7 days of age and a second 1-mL dose intramuscularly at weaning (approximately 17 days of age).

At weaning, all pigs were marked with Sprayola® to identify them by treatment group immediately before they were transported to the nursery.

In this operation, one off-site nursery was stocked with pigs from 10 sow farms. Each of the 18 rooms in the nursery was divided into four rows of 10 pens, with each pen holding 22 pigs (Figure 1). Each room was filled within a 24-hour period and the entire nursery was stocked within a 3-day period. Pigs were sorted at the nursery into pens according to entry weight. Each of the three treatment groups was represented in each of three blocks (large, medium, and small entry weights) in each room. One pig was selected at random from each of nine randomly selected pens and was identified by a numbered ear tag, making a total of nine pens and nine individual pigs studied per room. Seventeen rooms were selected for the study, making a total of 153 pigs and pens. Thus, a total of 51 pigs and pens were included in each treatment group.

Each pen of pigs was weighed as a group. Additionally, the individual pig selected from within the pen was weighed on day 0 and weekly thereafter throughout the 7-week period the pigs were in the nursery. Weights were obtained using a platform scale for pen weights. While pigs were small (< 20 lb (kg)) individual pig weights were obtained using a hanging scale in an effort to reduce weighing errors.

Blood was collected from each of the tagged pigs by jugular or anterior *vena cava* venapuncture at days 0, 28, and 49. Serum was harvested from each sample and immediately assayed for PRRSV antibody using a commercial ELISA kit. (IDEXX Laboratories, Westbrook, Maine). Serum was also assayed for the presence of PRRSV by virus isolation.

Virus isolation was attempted as described by Roof[19] using CL2621 cells (Boehringer Ingelheim Animal Health, St. Joseph, Missouri) grown in Eagle's minimum essential media (EMEM) supplemented with 5% fetal bovine serum (JRH Biosciences, Lenexa, Kansas). Cells were standardized to 1×10^6 cells per 1 mL of EMEM and a 500 µL aliquot was distributed into each well of a 24-well plate (Nalgene, Nalge Nunc International, Naperville, Illinois). The cells were incubated at 37°C with 5% CO_2 for 72 hours. A 200-µL serum sample was then placed in each well. Following a 2-hour incubation at 37°C, an additional 500 µL of fresh media was placed in each well. Each plate also included a positive (VR2332-infected) and negative (noninfected) control. Infected 24-well plates were then incubated at 37°C with 5% CO_2 and evaluated for cytopathic effects (CPE) typical of PRRSV for 8 consecutive days. Samples with no noticeable CPE were passed (200 µL) to another plate for confirmation. Samples with CPE were passed (10 µL) to a 96-well plate containing CL2621 cells. Monolayers were fixed after 12 hours and evaluated by indirect IFA using SDOW-17 and SR-10 to confirm the presence of PRRSV.[20]

The RFLP evaluation of PRRSV isolates was conducted as previously described (see pages 8–9 in this issue).

Statistical analysis

Average daily gain of individual pigs and pens of pigs were analyzed using ANOVA with repeated measures. Pigs were blocked by entry weight category (large, medium, small) and by room (n = 17). The three treatment groups were control (n = 51), IN vaccination (n = 51), and IN/IM vaccination (n = 51). The individual pig was the experimental unit used to measure individual ADG and ELISA values. The pen was the experimental unit in the statistical analysis of pen ADG. Linear regression analysis was performed to compare the initial ELISA value of individual pigs with their ADG.
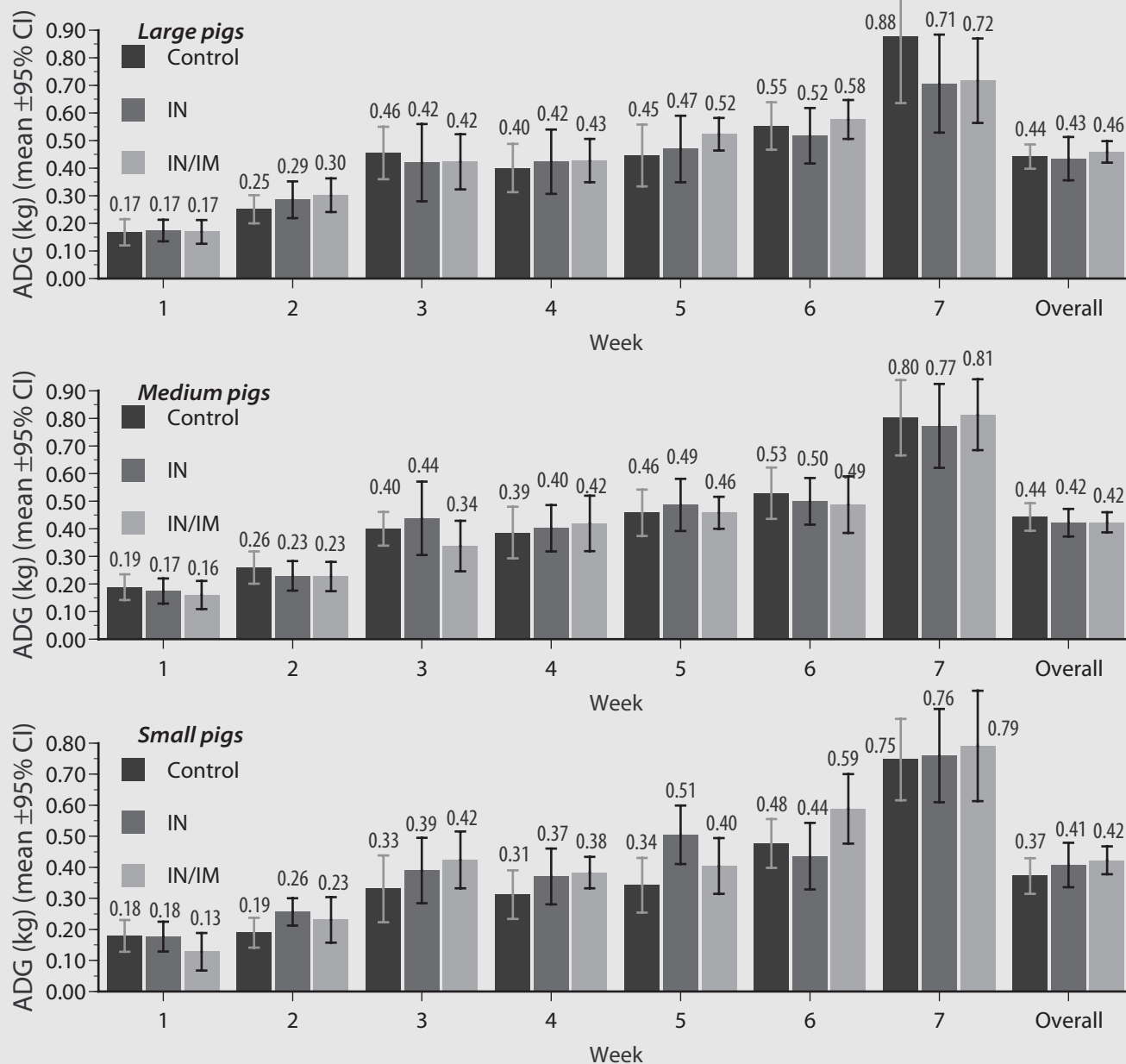
Results

Of the 153 pigs individually tested, three pigs died. One pig was a small nonvaccinated pig, one pig was a small IN-group pig, and one pig was a large IN-group pig. The cause of death of these pigs was not determined. One room of pigs (room 14) experienced a hemolytic *E. coli* infection approximately 2 weeks after the start of the trial.

One week (week 2) of individual and pen weight data was lost from one room. Thus the analysis on individual pigs consisted of 141 observations and weight gain data analysis performed on pens of pigs was based on 144 observations.

Average daily gain

Overall ADG did not differ (*P* < .49) among experimental groups, nor between IN and IN/IM groups (Figure 2). Weight gains were

Figure 2

Average daily gains of vaccinated and nonvaccinated pigs by size
ADG between treatment groups was not different ($P < .69$)
Results did not differ by the interaction of treatment and size ($P < .31$)

significantly better ($P < .01$) for vaccinated pigs than for controls in one of the weekly weighings (week 6) for individual pigs and also approached significance ($P < .08$) for pens of pigs. Vaccination did not have a significant effect on weight gains in any other week for either the individual pigs or pens of pigs (Figure 3).

Entry weight had a more significant effect than vaccine on the overall ADG of both the individual pigs and the pens of pigs (Figure 2). Large pigs grew significantly faster than small pigs ($P < .0001$) when ADG was measured on a pen basis. Using the individual pig data, large pigs tended to grow faster than the small pigs ($P < .06$). There were no significant differences between the growth rate of large- and medium-

entryweight pigs, nor between medium- and small-entryweight pigs. In general, the significance of the differences in ADG between entryweight groups decreased over time, an effect more noticeable in individual pigs than in pens of pigs. Weekly ADG were significantly different in weeks 1 and 2 ($P < .05$) only when observing individual pig data. ADG measured by pens of pigs was significantly different ($P < .01$) in weeks 2–6 and tended to be different in week 7 ($P = .07$).

The interaction of vaccination effects and entry weights was not significant when measuring the overall ADG of individual pigs ($P = .37$), nor was it significant when observing overall weight gains of pens of pigs ($P = .22$).

Serology and virus isolation

Concentrations of PRRS antibodies measured on day 0 did not differ significantly between treatment groups ($P = .31$) (Figure 3). The midpoint serological results obtained on day 28 indicated that both groups of vaccinated pigs had significantly ($P < .05$) higher ELISA values than did the nonvaccinated controls. Pigs that were given two doses of vaccine had higher ELISA values at the end of the trial than did the nonvaccinated controls ($P < .05$); however, correlation of the first ELISA titer and ADG was not significant ($r = .065$).

ELISA values at the midpoint of the trial were significantly higher ($P < .01$) in some rooms than others, but this difference did not depend on treatment group ($P = .63$) or size of the pigs ($P = .96$). At the end of the trial, there were no significant differences among ELISA values of pigs in different rooms ($P = .99$).

Of the viruses isolated from four pigs on day 0, three had RFLP profiles compatible with RespPRRS®/2332 (i.e., 252) (Figure 4). The pattern of the fourth virus was not determined because the virus could not be regrown.

Twenty three viruses recovered on day 28 were assayed by RFLP. Eighteen of these had RFLP profiles compatible with RespPRRS®/2332 virus. Five of the 23 viruses were found to have RFLP profiles which were not compatible with RespPRRS®/2332 virus and were assumed to be field isolates.

Four viruses found at day 49 were tested by RFLP. Three of the four viruses were compatible with RespPRRS®/2332 and one had an RFLP profile of 122.

Virus compatible with RespPRRS®/2332 was detected in nonvaccinated control pigs, and field isolates were also detected in vaccinated pigs. The majority of the viruses isolated from the pigs were recovered approximately 4 weeks after arrival at the nursery and approximately 5 weeks post vaccination.

Discussion

This study used both the individual animal and groups of animals to estimate treatment effects. Bahnson[21] has advocated the use of
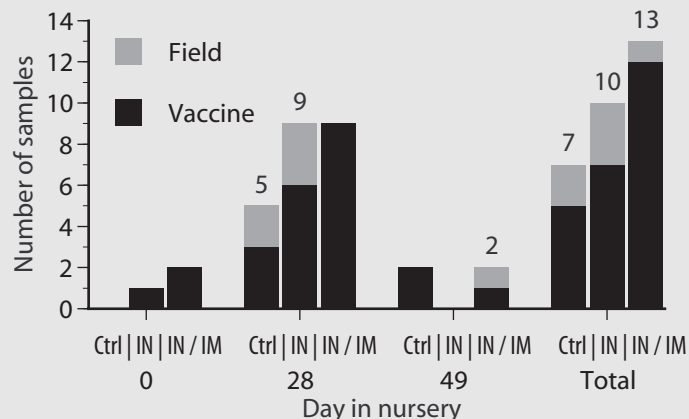
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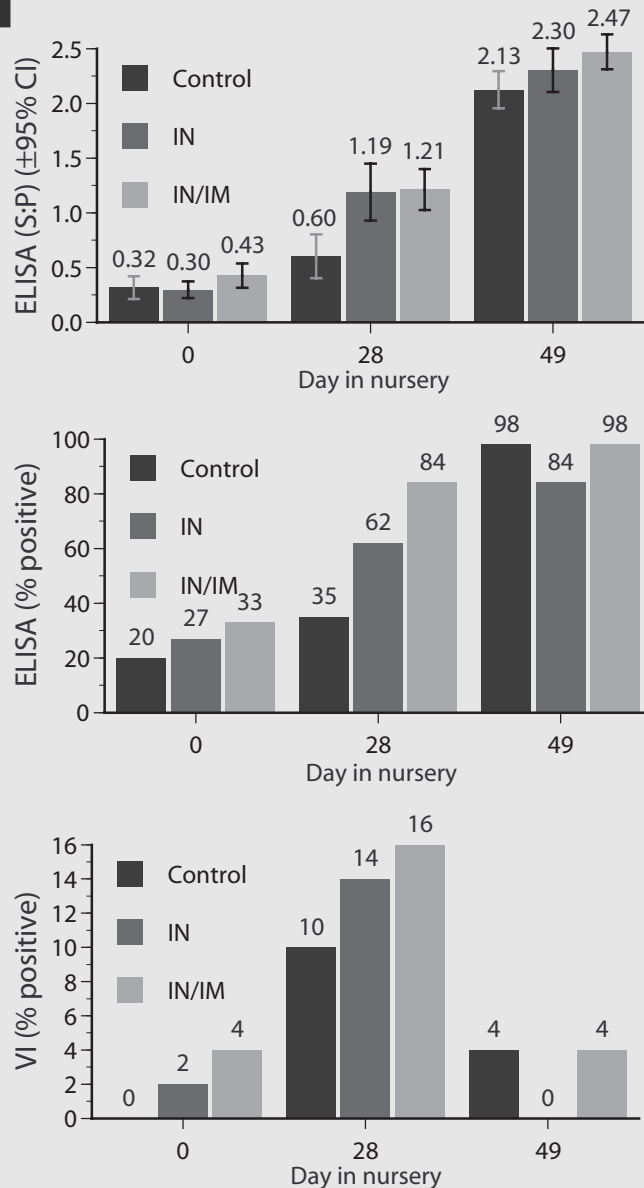
Summary of mean enzyme-linked immunosorbent assay (ELISA) values from serum and virus isolation (VI) results

S:P   samples : positive ratio

*   value is significantly different from others in same day ($P < .05$)

Figure 4

Restriction fragment length polymorphism (RFLP) results from PRRSV isolates

both individual pigs and groups of pigs in assessing disease effects on production. The individual pig is useful in assessing immunological responses to challenge and the impact on growth rates. The group performance is important in large-scale production and may be more important than the individual pig in assessing the effect of disease conditions on growth rates. The large number of pigs in the study was intended to detect even small differences of ADG between treatment groups. However, even with a relatively large number of experimental units, no differences were observed among treatment groups.

Recent studies by Morrison, et al.,[22] and Gorcyca, et al.,[23] suggest that passive immunity may play a significant role in reducing the effects of PRRSV challenge in 5-week-old pigs. Morrison, et al.,[22] showed that higher doses of virus were required to infect pigs with passive immunity than pigs with no maternal protection. The number of viremic pigs was reduced in populations with passive immunity, regardless of the challenge dose of virus. However, Park, et al.,[24] observed that viremia was present and virus spread occurred despite the ingestion of colostrum from infected dams, indicating that maternal immunity may not always protect pigs from PRRSV infection. In our study, PRRSV antibody concentrations declined in 12 pigs, seven of which had not been vaccinated, between day 0 and day 28 (approximately 17 to 45 days of age), indicating that their antibody titers were probably maternal in origin. However, weight gains were poorly correlated with the ELISA titer on entry (r = .06), indicating maternal antibody level was not a good predictor of ADG.

Only four pigs were detected to be viremic upon entering the nursery. All four pigs had been vaccinated and all four viruses isolated were compatible with RespPRRS®/2332. Maternal antibody may have played a role in limiting the number of viremic pigs. Four weeks after weaning, 23 pigs were viremic. It is reasonable to speculate that the decline of maternal antibody was associated with the increase in the number of viremic pigs. There were four viremic pigs at the end of the trial.

The recovery of both vaccine virus and field virus from the serum of pigs in all three treatment groups (Figure 4), suggests that vaccine virus spread to nonvaccinates and field virus spread to vaccinates. This conclusion is corroborated by the work of Torrison,[25] who observed that vaccine virus can spread from vaccinated pigs to nonvaccinated pigs. The method of spread of virus in this nursery is unknown; documenting the means of spread was beyond the scope of this study. Vaccinated pigs and nonvaccinated pigs were housed in the same room (i.e., within the same air space). In some cases, vaccinated pigs were in nose-to-nose contact with nonvaccinates. Thus, it is possible that virus was spread by airborne means, by nose-to-nose contact, or by caretakers and investigators as they moved between groups of pigs. The isolation of vaccine virus from nonvaccinated pigs suggested that seroconversion to PRRSV by nonvaccinated pigs could have been due either to vaccine virus shed from vaccinated pigs or from exposure to field virus present in the herd. The isolation of field virus from vaccinated and nonvaccinated pigs suggested that vaccination did not prevent infection with PRRSV field strains.

The results of this study disagree with earlier studies by Sanford,[17] Gillespie,[15] Trayer,[16] and Gorcyca,[26] who reported enhanced growth performance in association with vaccination against PRRS. There are several possible reasons for the differences observed in our study with previous work. For instance, there were relatively few secondary infections associated with this group of pigs. Several investigators have pointed to the relative lack of clinical signs in pigs infected with PRRSV in the absence of secondary infections such as *H. parasuis* or *S. suis*[27] and that PRRSV infection is more severe in the presence of other swine diseases such as *Salmonella choleraesuis*.[28] Alternatively, a dampening effect (herd immunity) could have resulted from the vaccination of approximately two-thirds of the pigs in a room. Because field isolates are more virulent that vaccine virus, the relatively high percentage of vaccinated pigs could have reduced the level of field virus circulating in the population with a resultant reduction in clinical signs.

The apparent spread of vaccine virus from vaccinated pigs to nonvaccinated controls may have resulted in the nonvaccinated controls being "vaccinated," thereby reducing potential differences in performance between treatment groups. One drawback in the design of field studies is the difficulty in maintaining true negative controls. The study was designed to compare the effects of vaccine within the same airspace, in pigs exposed to the same pathogens and management procedures. At the time of the design of this study, evidence for the spread of vaccine virus was not widely known.

The extra-label administration of the vaccine could be another factor influencing the results of this study. Intranasal administration was chosen in an attempt to obtain local and systemic immunity in a manner similar to that previously demonstrated with pseudorabies vaccine.[17] Although the amount of vaccine administered in the IM injection in the IN/IM group pigs was a reduced dosage (1 mL IM), this dosage was deemed to be adequate for protection after discussions with several swine practitioners and researchers, an assumption corroborated by the significantly higher ELISA titers observed in the vaccinates compared to the nonvaccinates at the midpoint of the trial.

Implications

- We observed no significant advantage in growth performance resulting from vaccinating pigs against PRRSV using intranasal and/or intramuscular methods prior to entry into a commingled nursery.
- Virus compatible with RespPRRS®/2332 virus was isolated from nonvaccinated control pigs and field isolates were isolated from vaccinated pigs.
- Entry weight had a significant impact on the ADG in this nursery. Large pigs gained significantly faster than small pigs.
- It may be necessary to assess the impact of PRRS vaccine in each system, with different methods of administration, to determine its cost effectiveness.

Acknowledgements

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