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The effects of porcine reproductive and respiratory syndrome (PRRS) vaccination on postweaning growth performance

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THE EFFECTS OF PORCINE REPRODUCTIVE AND RESPIRATORY SYNDROME (PRRS) VACCINATION ON POSTWEANING GROWTH PERFORMANCE¹

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Summary

We evaluated the effects of a modified-live virus vaccine for PRRS virus on nursery growth performance. The pigs used in this study were obtained from a herd with sub-standard nursery growth performance attributed to PRRS virus infection. We failed to detect the presence of active circulating field strain virus in either the controls or vaccinated pigs. However, we did detect a strain similar to the vaccine virus strain on d 34 after weaning in the vaccinated pigs. The vaccinated pigs had poorer growth performance from d 7 to 14 after vaccination and were lighter in weight for the remainder of the experiment. Vaccinating uninfected pigs with a modified-live vaccine (RespPRRS™) has a cost in growth performance. Therefore, determining if virus is circulating within the nursery population is necessary before vaccinating.

(Key Words: Porcine Reproductive and Respiratory Syndrome, Nursery Pigs, Vaccination.)

Introduction

Porcine reproductive and respiratory syndrome (PRRS) was first reported in 1987 and has since spread throughout the major pig producing areas of Asia, North America, and Europe. Clinical signs and production

losses are different among herds. This study focuses on the respiratory syndrome associated with PRRS virus infection in nursery populations. The most consistent signs observed in recently weaned pigs include respiratory distress, anemia, lethargy, failure to thrive, and decreased feed intake and growth.

Many different strategies have been proposed to manage active PRRS virus infection in nursery populations. Management strategies for controlling PRRS virus infection include nursery depopulation and MCREBEL (Management Changes to Reduce Exposure to Bacteria to Eliminate Losses). Nursery depopulation is most effective after viral transmission in the breeding and finishing populations has been controlled. The goal of these strategies is to stop active virus circulation within the populations that transmit infection to recently weaned pigs. Another tool for control of PRRS virus is vaccination using a modified-live vaccine (RespPRRS™, Boehringer-Ingelheim, St. Joseph, MO). The vaccine is approved for pigs 3 weeks of age or older, with a labelled intramuscular (IM) dose of 2 ml. Our first goal in this study was to assess the growth performance of pigs in an off-site nursery after weaning from a PRRS virus-infected, commercial herd. Our second goal was to study the effects of PRRS vaccination on growth performance.

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Procedures

Three hundred eighty unvaccinated pigs 17 to 23 days of age were obtained from a commercial herd with ongoing PRRS-associated infection in weaned pigs. Vaccination at weaning with a modified-live virus vaccine (RespPRRS™) was being conducted as a control strategy on the farm. Previous nursery depopulations and the vaccination program had resulted in unacceptable growth performance.

One day before weaning, 25 sows and their litters were selected randomly. Serum was harvested from the sows and frozen for later analysis. Four pigs from each sow were tagged and designated to be followed by PRRS ELISA. Two pigs from each litter were given even-numbered tags for the vaccination group, whereas the other two representing the control group were given odd-numbered tags. The next day (d 0), these 100 pigs were weaned along with 280 other pigs the same age and transported to the segregated early-weaning nurseries at Kansas State University.

Pigs were housed in one of two environmentally controlled, identical nurseries located a minimum of .8 km from other pig-rearing sites. One hundred ninety pigs were placed in one nursery, including the 50 even-numbered pigs, and 190 pigs including the 50 odd-numbered pigs were placed in another nursery. Animal caretakers were not allowed to be in contact with pigs on any other site for the previous 24 hours before entering the nursery. Pigs were checked, fed, or bled in the control barn first. Once in the treatment barn, caretakers could not return into the control barn for 12 hours.

Pigs were blocked by initial weight (heavy, medium, or light) and placed in pens each containing five pigs initially with weight equalized across pens within blocks. Each pen was 4 ft. x 4 ft. with slotted metal flooring, resulting in a population density of 1 pig per 3.2 sq. ft. A self-feeder and nipple waterer were located in each pen to allow

ad libitum consumption of feed and water. Pigs were used simultaneously in a nutrition experiment. On d 15, pigs were reallocated by weight within each barn to one of three blocks (heavy, medium, or light) to be used in a second nutrition experiment. Average pig weight was held constant within barns before and after reallocation. In both nutrition experiments, pigs were fed complete blocks of dietary treatments within each weight block. The pigs were weighed on d 7, 14, 21, 28, 35, and 42, and feed intake was recorded from d 0 to 14 and 21 to 42 to monitor ADG, ADFI, and F/G.

After allocation, prevaccination serum was obtained from the 50 tagged animals in each group and frozen for later analysis. Then the entire population in the control barn was injected with saline and the entire population in the treatment barn was injected with 2 ml IM of a modified-live PRRS virus vaccine (RespPRRS™). Tagged animals were bled on d 14, 35, and 42 after vaccination, and the serum was examined for the presence of antibodies to PRRS virus using an ELISA procedure. Pigs that died were submitted to the KSU Diagnostic Lab for necropsy. Tissues were formalin-fixed and examined for PRRS virus antigen using an immunohistochemical detection procedure.

On d 34, alveolar macrophages were obtained from 29 pigs (14 controls and 15 vaccinates) using alveolar-bronchial lavage. The macrophages were used for virus isolation procedures to detect PRRS virus. Virus isolates from the lavage were examined for similarity to vaccine virus strain using molecular biology techniques.

Data were analyzed as a randomized complete block design with the main effects of vaccination status and dietary treatment. The GLM function of SAS was used to analyze the data. Each pen of pigs was used as an experimental unit. Interactions ($P > .10$) were not detected between dietary treatment and vaccination status. This indicates that the responses to vaccination status and dietary treatment were independent.

Results

Growth Performance. In the first week (d 0 to 7), vaccinated pigs had a slightly higher ADFI compared with control pigs (.38 vs .35, respectively; $P < .05$; Table 1). The vaccinated pigs consumed on average of .03 lb per day more ($P < .05$) than the control group. From d 7 to 14, ADG of the control group was higher ($P < .01$) than that of the vaccinated group (.63 vs .49 lb, respectively). The control group consumed more ($P < .01$) feed than the vaccinated group. Feed efficiency of the control group was better than that of the vaccinated group ($P < .03$; 1.16 vs 1.30, respectively) in this time period. For the d 0 to 14 period, the control group had higher ADG ($P < .01$) and ADFI ($P < .07$) and better F/G ($P < .05$). Average daily gain and F/G did not differ between treatments from d 21 to 42. On d 7, pig weights between treatments were nearly identical. However, on d 14, 21, 28, 35, and 42, pigs in the control group were heavier ($P < .02$) than those in the vaccinated group.

Serology. Sixty percent (15/25) of the sows were seropositive ($> .4$ S/P) for PRRS virus. All pigs in the control group (50/50) were seronegative on d 35. Seventy-five percent (36/49) of the vaccinated group were seropositive for PRRS virus on d 35.

Virus Isolation in Alveolar Macrophages. Porcine reproductive and respiratory syndrome virus was not detected in the lavage samples from the control group on d 34. However, PRRS virus was detected in 20% (3/15) of the vaccinated group lavage samples examined on d 34. Virus samples from these three pigs were differentiated and found to be similar to RespPRRS™/2332 vaccine strain.

Immunohistochemistry. Two pigs that died from the control barn were submitted for necropsy, one each on days 8 and 10, respectively and formalin-fixed tissues were examined for the presence of PRRS virus antigen. Antigen was not detected by immunohistochemical methods.

Discussion

The pigs used in this study were from a commercial herd experiencing substandard growth performance after weaning. Vaccination with RespPRRS™ was being conducted in this herd at weaning. We do not know whether a field strain of PRRS virus was contributing to the decreased growth performance in the nursery population or if the vaccine itself had negative effects on growth performance.

The pigs housed in the off-site nurseries at KSU from weaning until d 42 proved to be free of field strain PRRS virus. All control pigs were PRRS ELISA negative. Nearly three-fourths of the vaccinated population were PRRS ELISA positive. Antibodies can be detected first by the ELISA at 9 to 13 days after vaccination (> 0.4 S/P ratio), peak 4 to 6 weeks later (2.0 to 4.0 S/P ratio), and are estimated to revert below detectable levels by 4 to 5 months after infection.

Alveolar macrophages collected by lavage of the lungs of infected pigs can be useful for virus isolation. Results with this dependable tool for PRRS diagnosis supported the serology results and allowed the isolated strain to be differentiated. The only strain isolated was one having a genetic profile similar to that of the RespPRRS™/2332 strain, indicating that the antibody response mounted by the vaccinated pigs was to the vaccine and not to a field strain virus. PRRS virus antigen was not found by immunohistochemistry on the formalin-fixed tissues of the necropsied control pigs, providing further supporting evidence that virus was not actively circulating.

Our results reflect a decrease in post-weaning growth performance after vaccination with RespPRRS™. The largest differences in growth performance occurred 7 to 14 days after weaning and vaccination. Average daily gain and ADFI were lower and F/G poorer for vaccinated compared to control pigs during this time period. Pig weights significantly differed by d 14. We found that ADG and F/G did not differ between treat-

ments from days 21 to 42 after infection. However, because of the decrease in performance from d 7 to 14, vaccinated pigs weighed less on d 21, 28, 35, and 42. This indicates that the weight difference was not getting larger and the vaccinated pigs did not compensate with increased growth performance.

These findings stress the importance of determining the presence of active PRRS virus within nursery populations before vaccination are initiated. Sixty percent of the sows from which the pigs came in this study were PRRS ELISA positive. However, this does not provide evidence of the viral status in the nursery population. Serology coupled

with virus isolation in nursery pigs should give practitioners and producers a good idea of what is happening in that population. Our results show that vaccination has a significant influence on growth performance and is not recommended if the nursery is free of circulating field-strain virus.

In conclusion, vaccinating uninfected pigs with a modified-live vaccine (RespPRRS™) has a cost in growth performance. Producers must know if a field-strain virus is circulating within the nursery population before vaccinating. Live vaccine virus was detected on day 34 after vaccination, and the ELISA titer of the dams at weaning was not predictive of PRRS virus infection in the nursery.

Table 1. Influence of a Modified-Live PRRS Vaccine on Growth Performance, Immune Response, and Viral Shedding^a

Item	Control	Vaccinates	P<	CV
<u>d 0 to 7</u>				
ADG, lb	.28	.30	.39	26.8
ADFI, lb	.35	.38	.05	17.0
F/G	1.24	1.28	.54	21.2
<u>d 7 to 14</u>				
ADG, lb	.63	.49	.01	15.0
ADFI, lb	.73	.64	.01	16.4
F/G	1.16	1.30	.03	20.3
<u>d 0 to 14</u>				
ADG, lb	.46	.39	.01	12.0
ADFI, lb	.54	.51	.07	13.9
F/G	1.19	1.29	.05	11.0
<u>d 21 to 42</u>				
ADG, lb	1.11	1.08	.23	8.7
ADFI, lb	2.14	2.05	.04	7.9
F/G	1.95	1.91	.34	8.1
<u>Pig weight, lb</u>				
d 7	14.2	14.3	.40	3.6
d 14	18.6	17.9	.01	4.0
d 21	26.0	25.1	.01	3.1
d 28	33.3	31.5	.01	3.8
d 35	40.4	39.4	.02	4.0
d 42	49.6	47.7	.01	4.9
<u>ELISA results, % > .4</u>				
d 35	0 (0/50)	73 (36/49)	--	--
<u>Lavage results, % Pos VI</u>				
d 34	0 (0/14)	20 (3/16)	--	--

^aEach number is the mean of 36 pens (five pigs/per pen) from d 0 to 14 after weaning and 30 pens from d 21 to 42 after weaning. Pigs initially averaged 12.2 lb. Vaccine was administered to pigs in the vaccinated group on the day of weaning (d 0). All VI isolates on d 34 were similar to the vaccine strain (RespPRRS™).