

Effects of Sirrah-Bios PRRSV-RS Vaccine on Mortality Rate and Finisher Pig Performance¹

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Summary

A total of 1,561 pigs (initially 4 d of age) were used to determine the effects of a porcine reproductive and respiratory syndrome virus (PRRSv) subunit vaccine, PRRSV-RS (Sirrah-Bios, Ames, IA), on mortality rate and finisher pig growth performance in a PRRSv-positive commercial herd. Pigs were randomly assigned by litter to either the subunit PRRSv vaccine or non-vaccinated control group. Pigs in the vaccinated group received an intramuscular injection of 1 mL PRRSV-RS vaccine at processing (approximately 4 d after birth) and again at weaning (approximately 24 d of age). Vaccinated and control pigs were comingled in a single nursery during the nursery phase. In the finishing phase, pigs were housed in a standard commercial curtain-sided finisher barn by treatment and gender by pen, with treatments randomly distributed across pens. Mortality was tracked from processing (4 d of age) to market (d 187 to 193). There was no difference between the control and vaccinated pigs for cumulative mortality (21.5% vs. 20.6%, $P = 0.67$) or for mortality during each production phase (processing to weaning: 9.5% vs. 7.1%, $P = 0.08$; nursery: 9.3% vs. 9.2%, $P = 0.95$; finishing: 4.4% vs. 5.9%, $P = 0.20$). Pigs were initially weighed by single-sex pens (control or vaccinated) 2 wk after placement into the finisher (d 0), and at that time, control and vaccinated mean pig weights were not different (58.4 vs. 58.7 lb, $P = 0.90$). Pens of pigs were subsequently weighed every 2 wk, and feed consumption was recorded to calculate ADG, ADFI, and F/G. Overall (d 0 to 112), control and vaccinated pig performance was similar (ADG: 1.96 vs. 1.93 lb, $P = 0.45$; ADFI: 5.35 vs. 5.36 lb, $P = 0.94$; F/G: 2.74 vs. 2.78, $P = 0.15$) throughout the finishing period. This resulted in no difference ($P = 0.79$) in off-test (d 112) weights between control (271.9 lb) and vaccinated (270.4 lb) pigs. These data indicate that this subunit PRRSv vaccine did not affect finisher pig performance or mortality in this commercial herd.

Key words: growth, mortality, PRRSv, vaccine

Introduction

Porcine reproductive and respiratory syndrome is caused by a virus in the family *Arteriviridae*. This virus has become endemic in many herds. Continual evolution of porcine reproductive and respiratory syndrome virus (PRRSv) strains has made development of an effective and reliable vaccine difficult. Modified-live and whole virus inactivated PRRSv vaccine products are available commercially. Inactivated products have not been demonstrated to be efficacious under field conditions. Use of the modified-live vaccines is considered to provide more effective immunity than inactivated products. However, the modified-live PRRSv vaccine is shed and will transmit to unvaccinated

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pigs. Also, there is concern that further transmission of the PRRSv vaccine strain virus will increase the potential for reversion to virulence.

Another class of PRRSv vaccines consists of subunit vaccines. Subunit vaccines are formed by using specific proteins of a virus to which an antibody response is stimulated. Thus, like a whole virus inactivated vaccine product, a subunit vaccine cannot propagate or revert to virulence. Commercially available subunit vaccines have been proven to provide effective immunization against other viruses, such as porcine circovirus type 2. Recently, a new subunit PRRSv vaccine, PRRSV-RS (Sirrah-Bios, Ames, IA), has been made available for use on sows or growing pigs. This vaccine contains an adjuvant and a heterodimer of the PRRSv glycoprotein 5 and matrix protein expressed with an AlphaVax replicon vector. It has been documented in a mouse model that a heterodimer of specific proteins is necessary to promote neutralizing antibodies against equine arteritis virus, also a member of the family *Arteriviridae*. For that reason, it has been suggested that the GP5-M heterodimer may induce cross-protective neutralizing antibodies against PRRSv infection in the pig and potentially allow for differentiating capabilities between vaccinated and infected pigs. However, there is limited data demonstrating subunit PRRSv vaccine efficacy under field conditions. Thus, the objective of this trial was to evaluate the effects of a subunit PRRSv vaccine (PRRSV-RS) vaccine on cumulative mortality rate, growth performance, and feed efficiency of commercial finisher pigs.

Procedures

Procedures used in this trial were approved by the Kansas State University Institutional Animal Care and Use Committee.

A total of 1,561 pigs from 140 litters within a single week of farrowings across 5 sow farms were assigned to either a non-vaccinated control or subunit PRRSv vaccine treatment group. Treatment groups were formed by randomly assigning the first litter processed at each sow farm to one of the treatments and then alternating vaccine treatment assignments on subsequently processed litters. This resulted in 70 litters represented within the 781 control pigs and 70 litters represented within the 780 vaccinated pigs. Pigs in the vaccinated group received 1 mL of PRRSV-RS vaccine intramuscularly at processing (4 d of age) and again at weaning (approximately 24 d of age). All pigs were weaned as a group into a single nursery.

Pigs were identified by ear tags, and mortality was tracked by collecting ear tags of pigs that died or were humanely euthanized. Mortality was tracked from processing to weaning, weaning to the end of the nursery period, and throughout the finishing period until the majority of the pigs were marketed. Cumulative mortality was determined by identifying the number of pigs in each treatment group that died or were euthanized from processing to marketing day divided by the initial number of pigs in each treatment.

Throughout the nursery period, control and vaccinated pigs were comingled within single-sex pens, and all test pigs were contained within a common room. All pigs were vaccinated with a 2-dose porcine circovirus type 2 vaccine and a *Mycoplasma hyopneumoniae* vaccine during the nursery period according to routine nursery procedures. Similar diets were fed to all pigs throughout the nursery period.

Pigs were moved to a single finisher barn and separated by vaccine treatment (vaccinated or control) and gender (barrow or gilt). There were 12 pens of each treatment × gender combination, with the exception of vaccinated barrows, for which there were 13 pens. Pens (10 × 18 ft) for each treatment were randomly distributed throughout the barn. Each pen was equipped with a double swinging waterer and a 3-hole dry self-feeder, allowing for ad libitum access to water and feed. An automated feeding system (FeedPro; Feedlogic Corp., Willmar, MN) was used in the barn to deliver and measure feed added to individual pen feeders. Pigs were weighed and feed intake was recorded beginning 2 wk after arrival in the finisher (d 0) and again on d 14, 28, 41, 56, 70, 90, and 112. From these data, ADG, ADFI, and F/G were calculated. On d 90, there were 0, 2, or 4 heavy pigs removed per pen in a balanced manner across treatment and gender, resulting in 84 “top” pigs marketed per vaccine treatment. At the end of the trial, pigs were marketed over 2 consecutive days in a balanced fashion, with the last pigs being weighed off test on d 112.

Finisher growth and feed performance data were analyzed as a completely randomized design using the GLIMMIX procedure of SAS (SAS Institute Inc., Cary, NC) and pen as the experimental unit. Vaccine treatment was managed as the main fixed effect of interest; however, gender was added in the model to control for expected differences in growth rate between barrows and gilts. Differences between treatments were determined by using least squares means ($P < 0.05$).

Mortality data were analyzed using the FREQ procedure in SAS. Mortality differences between treatments were determined using the chi-square test ($P < 0.05$). Analysis was performed on mortality data both within production phase (processing to weaning, nursery, and entry to finisher to off test) and cumulatively.

Results and Discussion

There were no gender × vaccine treatment interactions for the response criteria in the finishing trial. Although barrows were 1 lb lighter (58.0 vs. 59.0 lb, $P = 0.90$) than gilts initially, growth performance across genders was as expected. Barrows had greater overall ADG (2.01 vs. 1.87 lb, $P < 0.001$) and ADFI (5.65 vs. 5.07 lb, $P < 0.001$) and poorer F/G (2.81 vs. 2.70, $P < 0.001$) than gilts.

Non-vaccinated control pigs performed similarly to vaccinated pigs during the finishing period (Table 1). When pigs were first weighed, 2 wk after entry to the finisher, there was no difference ($P = 0.90$) in weight between controls (58.4 lb) and vaccinates (58.7 lb). From this point forward, there was no difference ($P > 0.06$) in ADG, ADFI, or F/G between the 2 treatment groups. This lack of difference in performance during the finishing period resulted in similar ($P = 0.79$) off-test (d 112) weights between controls (271.9 lb) and vaccinates (270.4 lb).

Mortality, either cumulative or within production phase, was not different ($P > 0.08$) between treatment groups (Table 2). Historically, during the nursery period, pigs in this production system undergo natural exposure to PRRSv and influenza. During the nursery period, pigs used in this trial exhibited clinical signs indicating similar exposure to PRRSv and influenza virus. The lack of difference in growth performance detected in this trial between controls and vaccinates indicates that the vaccine did not have a

negative or positive impact on growth or mortality. This is important because it appears that the majority of the cost associated with the vaccine would be due to administration materials, labor, and the vaccine product itself.

Although this subunit PRRSv vaccine is made from viral strains similar to historical strains, which are considered to provide some cross-protective immunity, it is unknown whether the vaccine-induced level of protection varies with viral strain challenge. In this herd, which has historical PRRSv-associated challenge, this subunit PRRSv vaccine failed to influence overall mortality or growth performance during the finishing phase.

Table 1. Effect of PRRSV-RS vaccine on growth performance of finisher pigs¹

Item	Treatment ²		Probability, <i>P</i> <
	Control	Vaccinated	
Initial wt, lb	58.4 ± 1.7	58.7 ± 1.7	0.90
d 0 to 112			
ADG, lb	1.96 ± 0.03	1.93 ± 0.03	0.45
ADFI, lb	5.35 ± 0.08	5.36 ± 0.08	0.94
F/G	2.74 ± 0.02	2.78 ± 0.02	0.15
Final wt, lb	271.9 ± 3.9	270.4 ± 3.8	0.79

¹ A total of 1,561 pigs (barrows or gilts) from 140 litters across 5 sow farms were assigned to 1 of 2 treatments at processing (4 d of age) by randomly assigning entire litters to either the vaccinated or non-vaccinated control groups. Control and vaccinated pigs were comingled in the nursery and then separated by vaccine treatment and gender in the finisher barn. Treatment pens were randomly distributed throughout the barn. There were 24 pens of control pigs and 25 pens of vaccinated pigs. All pens of pigs (1,292 pigs total) were initially weighed 2 wk after placement in the finisher (d 0) and then on d 14, 28, 41, 56, 70, 90, and 112.

² Treatments were: Control = no vaccine administered and Vaccinated = 1 mL PRRSV-RS administered intramuscularly at processing and weaning (approximately 24 d of age). Results are reported as least squares mean ± standard error of the mean.

Table 2. Effect of PRRSV-RS vaccine on within-period and cumulative mortality¹

Item	Treatment ²		Probability, <i>P</i> <
	Control	Vaccinate	
Inventory			
Processing ³	781	780	---
Weaning ⁴	707	725	---
Entry to finisher ⁵	641	658	---
Off test ^{6,7}	529	535	---
Within-period mortality			
Processing to weaning, %	9.5	7.1	0.08
Nursery, %	9.3	9.2	0.95
Finisher, %	4.4	5.9	0.20
Cumulative mortality			
Processing to weaning, %	9.5	7.1	0.08
Processing to end of nursery, %	17.9	15.6	0.23
Processing to off test, % ⁶	21.5	20.6	0.67

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² Treatments were: Control = no vaccine administered and Vaccinated = 1 mL PRRSV-RS administered intramuscularly at processing and weaning.

³ 4 d of age.

⁴ Weaning age range was 20 to 26 d of age.

⁵ Entry-to-finisher age range was 60 to 66 d of age.

⁶ Off-test age range was 187 to 193 d of age.

⁷ Inventory at off test (d 112) excludes pigs marketed (84 controls and 84 vaccinates) on d 90 of the trial.