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REGULAR ARTICLES

Reproductive parameters following a PRRS outbreak where a whole-herd PRRS MLV vaccination strategy was instituted post-outbreak

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Abstract This study assessed the effect of whole-herd porcine reproductive and respiratory syndrome (PRRS) modified-live virus (MLV) vaccination on herd-level reproductive performance, PRRS virus (PRRSV) viremia, and antibody in a subset of females in a 1,200-sow commercial herd in Thailand. Following a PRRSV outbreak, the entire herd was vaccinated with PRRS MLV twice at 3-week intervals and at 3-month intervals, thereafter. Reproductive performance data over a 3-year period were available for analysis. Serum samples were collected before and after vaccination and tested by PRRSV ELISA and reverse transcription-polymerase chain reaction. Vaccination was statistically associated with a lower abortion rate (1.4 vs. 1.6 %), farrowing rate (83.8 vs. 90.0 %), total born (10.6 vs. 11.4 piglets/litter), liveborn (10.0 vs. 10.3 piglets/litter), stillbirths (4.6 vs. 7.0 %), mummies (0.7 vs. 1.6 %), and a higher return rate (11.3 vs. 5.9 %) when compared with the period before the PRRSV outbreak. Pregnant females vaccinated during early gestation farrowed fewer liveborn and more mummies than the comparison group, whereas females vaccinated during late gestation had a lower farrowing rate. In this herd, PRRS whole-herd vaccination had neutral, positive, and negative effects on reproductive performance. Thus, the decision to implement whole-herd vaccination should be balanced between the benefits derived from reproductive performance improvements, e.g., fewer abortions, stillborn piglets, and mummified fetuses, and the effect of vaccination on pregnant females.

Keywords PRRSV · Modified-live virus vaccine · Whole-herd vaccination · Reproductive performance · Gestation

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Introduction

Porcine reproductive and respiratory syndrome (PRRS) is caused by PRRS virus (PRRSV), a member of family *Arteriviridae*. In general, PRRSV infection in pregnant gilts and sows is characterized by late-term abortions and an increase in mummified fetuses per litter, stillborn piglets per litter, and low viability piglets at birth (Chung et al. 1997). The disease was reported for the first time in the USA in 1987, and the virus was identified for the first time in Lelystad, the Netherlands, in 1990 (Wensvoort et al. 1991). In 1992, PRRSV was divided into two genotypes, i.e., types 1 (European genotype) and 2 (North American genotype) on the basis of genetic, antigenic, and pathogenic differences (Meng 2000).

To date, PRRSV has been found in most major pig-producing areas throughout the world (Zimmerman et al.

2006). A retrospective serological study determined that PRRSV was present in Thailand since 1989 (Damrong watanapokin et al. 1996) and in 1995, it was estimated that 64 % of the commercial swine herds in Thailand were PRRSV-infected (Oraveerakul et al. 1995). Both types 1 and 2 PRRSV genotypes have been isolated in Thailand (Thanawongnuwech et al. 2004).

In the PRRSV-endemic herds, the presence of subpopulations of susceptible pigs may lead to the continual circulation of PRRSV. Herd closure, gilts acclimatization, and whole-herd exposure to wild-type virus or vaccines have been recommended to eliminate these subpopulations (Cano et al. 2007a, b). The types of PRRSV vaccine available in Thailand include both modified-live virus (MLV) and inactivated virus vaccines. The use of vaccination to immunize pigs has been evaluated, in most cases, at the individual pig level and in nursery populations (Martelli et al. 2009). It has been demonstrated that PRRS MLV vaccination can reduce lung lesions in the PRRSV-infected pig and decrease the level and duration of viremia after challenge with homologous virus (Foss et al. 2002; Mengeling et al. 2003). In addition, PRRS MLV vaccination of the entire herd (whole-herd vaccination) was shown to reduce the persistence and duration of the viral shedding, even though wild-type virus was not eliminated (Cano et al. 2007a, b). However, the effect of PRRSV vaccination varies among herds (Alexopoulos et al. 2005; Martelli et al. 2007) and, furthermore, limited information is available on reproductive performance in pregnant gilts and sows following PRRS MLV vaccination. Therefore, the objective of the present study was to monitor the PRRSV status (antibody and viremia) of a subset of gilts and sows and the herd-level reproductive performance over time of a PRRSV-positive breeding herd following whole-herd PRRS MLV vaccination.

Materials and methods

Project design

Reproductive data were collected in a commercial breeding herd prior to, during, and after a PRRSV outbreak and mass vaccination of gilts and sows with a PRRSV MLV vaccine (Ingelvac® PRRS MLV, Boehringer-Ingelheim Vetmedica, Inc., St. Joseph, Missouri). The data were analyzed for the effect of mass vaccination on (1) PRRSV ELISA response and viremia, (2) fertility parameters (farrowing rate, return rate, and abortion rate), and (3) litter parameters (total born, live born, stillbirths, and mummified fetuses).

Herd management and vaccination protocols

The study was conducted in a 1,200-sow commercial breeding herd in central Thailand in which in-herd replacement gilts were produced using grandparent stock. Replacement gilts were acclimatized at 22–30 weeks of age, before entering the breeding herd and were assumed to be PRRSV positive. Gilts and sows were housed in a conventional open housing system, i.e., slatted floors and open sides, and the herd health management program was under the supervision of a herd veterinarian. Gilts and sows had never been vaccinated against PRRSV but did receive vaccines against foot-and-mouth disease (2 weeks before farrowing), classical swine fever (2 weeks after farrowing), Aujeszky's disease (mass vaccination every 4 months), and porcine parvovirus (gilts prior to placement in breeding herd, then 2 weeks after farrowing every 3rd parity).

PRRSV monitoring data

Gilts and sows (n=20–30) were tested biannually using a commercial PRRS ELISA assay (HerdChek® PRRSV antibody test kit 2XR®, IDEXX Laboratories, Inc., Westbrook, Maine) for the 3 years prior to the PRRSV outbreak. Based on monitoring results, the herd was considered PRRSV positive, but stable. At the beginning of January 2009, reproductive failure characterized by abortions in gilts and sows mated during October to December 2008, increased return to estrus after mating, and increased mortality in suckling and weaned piglets were noted. In January 2009, a type 2 PRRSV was detected by reverse transcription-polymerase chain reaction (RT-PCR) in serum samples from sows and piglets submitted for testing at the Veterinary Diagnostic Laboratory, Chulalongkorn University (Bangkok, Thailand).

PRRSV vaccination and blood collection

On 15 May 2009, all gilts and sows in the herd were vaccinated with a PRRSV MLV vaccine at 3-week intervals, i.e., weeks 0 and 3. Thereafter, all gilts and sows (both pregnant and nonpregnant) were vaccinated every 3 months. Concurrently with the first PRRS vaccination, six age groups composed of six animals each were selected for PRRSV monitoring: (1) 7- to 8-month-old replacement gilts, (2) 9- to 11-month-old breeding gilts, (3) parity one sows, (4) parity 2 sows, (5) parity 3–4 sows, and (6) parity 5–6 sows. Blood samples were collected from these 36 animals one day before PRRSV vaccination and then 2, 5, 9, 12, and 18 weeks after the first vaccination. Blood samples were allowed to clot at room temperature, after which serum was harvested and either tested immediately for PRRSV antibodies or stored at −20 °C for later testing. Serum samples (n=6) were pooled by age group and tested immediately by PRRSV RT-PCR.

PRRSV antibody and RT-PCR assay

Individual serum was tested for PRRSV antibody using a commercial assay performed according to the manufacturer's

protocol. Pooled serum samples were tested for PRRSV using a commercial RT-PCR assay (AccessQuick™ RT-PCR system, Promega Corporation, Madison, Wisconsin) capable of amplifying open reading frame 7 of either type 1 or 2 PRRSV genotypes. The reaction consisted of upstream and downstream primers (Amonsin et al. 2009), avian myelobalastosis virus reverse transcriptase (Promega Corporation), and RNA template. The reverse transcription and PCR amplification conditions were performed according to kit instructions. The amplified products and standards (GeneRuler™ 100 bp DNA Ladder, Fermentas Inc., Glen Burnie, Maryland) were electrophoresed on 1.0 % agarose gel and stained with ethidium bromide. PRRSV genotypes were differentiated on the basis of the size of the products, i.e., 390 bp for type 1 and 430 bp for type 2 genotypes.

Reproductive performance dataset

Reproductive performance data were collected for the period from July 2007 to June 2010 from breeding productivity records (PigCHAMP®, version 4.10, Minnesota). The data dictionary was based on conventional definitions of industry terms and formulas. A mating was defined as the insemination of a gilt/sow during a 10-day estrus period and a service included one or more mating events during estrus (Takai and Koketsu 2009). Return-to-estrus, abortion, and farrowing were defined as binomial traits (0, 1). The farrowing rate (FR), the return rate (RR), and the abortion rate (AR) were calculated as the number of females that returned to estrus or aborted or farrowed divided by the number of mated females multiplied by 100. Total born per litter (TB) was defined as the sum of born alive (BA) plus the number of stillborn piglets (SB) plus the number of mummified fetuses (MM). The percentage of SB and percentage of MM were calculated as the number of SB or MM divided by TB multiplied by 100. Pregnant females were classified in terms of PRRSV vaccination status relative to the blanket vaccination that occurred on 15 May 2009: (1) 0 to 30 days of gestation at the time of blanket vaccination; (2) 31 to 60 days of gestation; (3) 61 to 90 days of gestation; and (4) vaccination at >90 days of gestation. The raw data consisted of 8,162 matings and 6,975 farrowing records from 2,543 sows. Records with missing data were removed from the dataset, leaving a total of 7,914 matings and 6,793 farrowings from 2,337 sows for the analysis. Records included sow identity, parity number at service, mating date, number of inseminations, mating result, days until the sow returned to estrus after mating, farrowing date, TB, BA, SB, and MM.

Statistical analyses

Statistical analyses were performed using SAS statistical software (SAS® version 9.0, SAS® Institute, Inc., Cary, North Carolina). Initially, fertility parameters (RR, AR, and FR) and litter parameters (TB, BA, SB, and MM) were analyzed for differences over time, i.e., before PRRSV infection (July 2007 to June 2008), during PRRSV field infection (July 2008 to June 2009), and after vaccination (July 2009 to June 2010), PRRSV vaccination status, parity (0, 1, 2–4, and ≥5), parity by time, and parity by vaccination status using generalized linear-mixed models. Tukey–Kramer adjustments were used for multiple comparisons. $P<0.05$ was considered statistically significant. Quantitative serum ELISA responses (S/P ratios) were evaluated by week of collection (0, 2, 5, 9, 12, and 18) using paired *t* tests. The qualitative ELISA response (positive vs. negative) was analyzed by logistic regression using generalized linear-mixed models that included the week of sample collection (0, 2, 5, 9, 12, and 18) and female classification (replacement gilt, bred gilt, and sow parity numbers 1, 2, 3-4, and 5–6).

Results

Serum testing results

No viremic animals were detected by PRRSV RT-PCR either before or after PRRS vaccination. Among the 36 animals monitored over time, 88.9 % (32/36) were PRRS ELISA antibody positive prior to vaccination (Table 1). After mass vaccination, the percentage of seropositive animals in this group ranged from 85.3 % to a high of 94.4 % for the 18 weeks over which the animals were monitored. Mean ELISA S/P ratios varied from 1.61 prior to vaccination to 1.23 at week 18 post-vaccination.

Reproductive performance

Herd fertility parameters (FR, RR, and AR) and litter parameters (TB, BA, SB, and MM) over time are summarized in Fig. 1a, b and Tables 2 and 3, respectively. Before the PRRSV outbreak, FR, AR, RR, SB, and MM were 90.0, 1.6, 5.9, 7.0, and 1.6 % respectively, while TB and BA were 11.4 and 10.3 piglets per litter, respectively. During the outbreak, especially November 2008 to January 2009, a high AR (16.7 %) and a low FR (71.2 %) were observed. The lowest TB and BA, 9.7 and 8.3 piglets/litter, respectively, and the highest MM (8.4 %) were observed in gilts and sows that farrowed in April 2009 (mated in January 2009). During the PRRSV outbreak, reproductive parameters were significantly affected compared with pre-outbreak levels, i.e., FR (83.9 vs. 90.0 %, $P<0.001$), AR (5.2 vs. 1.6 %, $P<0.001$), RR (8.0 vs. 5.9 %, $P=0.048$), TB (10.9 vs. 11.4 piglets/litter, $P<0.001$), BA (9.9 vs. 10.3 piglets/litter, $P<0.001$), and MM (2.2 vs. 1.6 %, $P=0.004$).

Table 1 Serum testing results by week post-vaccination

Weeks	PRRS ELISA (mean S/P ratio)	ELISA positive	PRRSV RT-PCR
0	1.61±0.19 a, b	32/36 (88.9 %) a	Negative
2	1.88±0.16 a	34/36 (94.4 %) a	Negative
5	1.47±0.16 b	31/36 (86.1 %) a	Negative
9	1.32±0.15 b	32/36 (88.9 %) a	Negative
12	1.46±0.17 b	29/34 (85.3 %) a	Negative
18	1.23±0.07 b	31/33 (93.9 %) a	Negative

Different lowercase letters (a and b) within columns indicate statistically significant differences ($P \leq 0.05$)

Following vaccination against PRRSV, the AR decreased from the outbreak period (1.4 vs. 5.2 %, $P<0.001$) and returned to pre-outbreak levels (1.4 vs. 1.6 %, $P>0.05$), whereas RR remained higher than before the outbreak (11.3 vs. 5.9 %, $P<0.001$) or during outbreak (11.3 vs. 8.0 %, $P<0.001$) (Table 2). The FR did not differ from the outbreak period (83.8 vs. 83.9 %, $P>0.05$), but it remained lower than before the outbreak (83.8 vs. 90.0 %, $P<0.001$) (Table 2). TB and BA were lower than before outbreak (10.6 vs. 11.4 piglets/litter, $P<0.001$ and 10.0 vs. 10.3 piglets/litter, $P=0.012$, respectively) (Table 3). However, while TB was lower than during the outbreak period (10.6 vs. 10.9 piglets/litter, $P=0.015$), BA was higher (10.0 vs. 9.9 piglets/litter, $P=0.012$) (Table 3). SB and MM were both lower than before the outbreak (4.6 vs. 7.0 %, $P<0.001$ and 0.7 vs. 1.6 %, $P<0.001$, respectively) and during outbreak (4.6 vs. 6.1 %, $P<0.001$ and 0.7 vs. 2.2 %, $P<0.001$, respectively) (Table 3). Preweaning mortality before the outbreak, during the outbreak, and following PRRS MLV vaccination was 4.7, 8.5, and 4.4 %, respectively. These estimates are based on pre-outbreak piglet numbers of 24,302 (BA) and 23,254 (weaned), outbreak piglet numbers of 20,999 (BA) and 19,217 (weaned), and post-vaccination numbers of 23,228 (BA) and 22,196 (weaned).

After PRRS vaccination, FR, BA, and MM varied by the state of gestation at the time of vaccination (Tables 4 and 5). Gilts and sows vaccinated at ≥90 days of gestation had a lower FR than those vaccinated at 0–30 (77.3 vs. 88.3 %, $P=0.008$), 31–60 (77.3 vs. 85.1 %, $P=0.055$), and 61–90 days of gestation (77.3 vs. 84.7 %, $P=0.176$) (Table 4). RR and AR were not significantly different among PRRSV vaccination status, although numeric differences were observed. Likewise, FR, RR, and AR varied by parity, but were not statistical significant (Table 4). BA was lowest (9.2 piglets/litter) and MM was highest (5.3 piglets/litter) in females vaccinated at 0–30 days of gestation (Table 5). However, TB and SB did not differ by parity or stage of gestation at the time of vaccination.

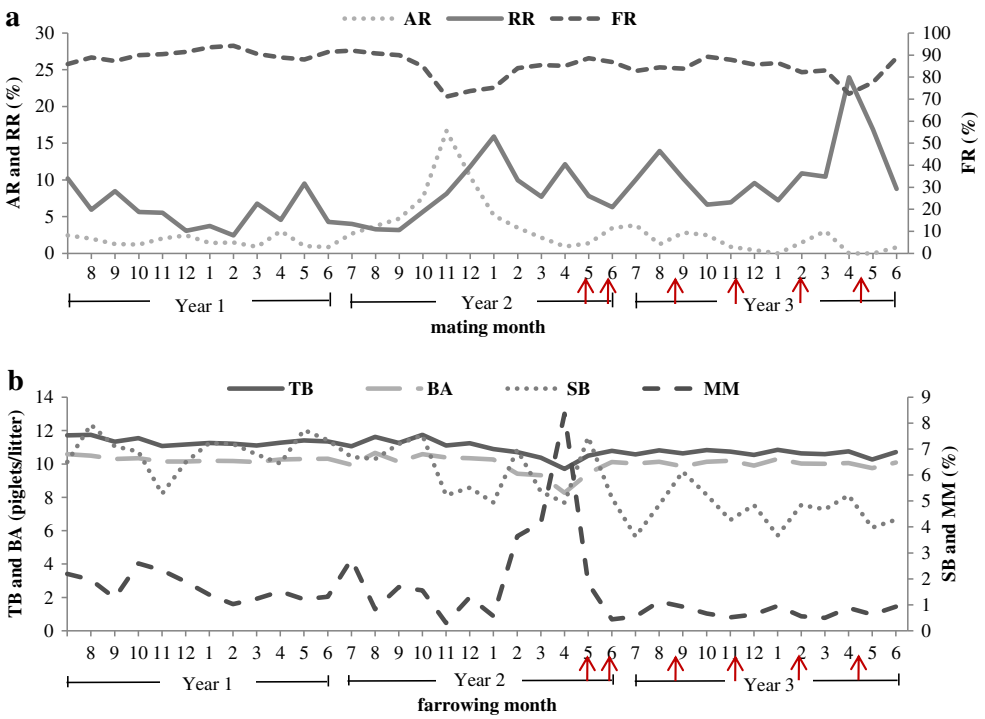

Fig. 1 **a** Farrowing rate (*FR*), abortion rate (*AR*), and return rate (*RR*); **b** the number of total piglets born per litter (*TB*), the number of piglets born alive per litter (*BA*), the percentage of stillborn piglets per litter (*SB*), and the percentage of mummified fetuses per litter (*MM*). *Arrow* indicates dates of PRRS MLV vaccination

Table 2 Comparison of fertility parameters by parity over time

Fertility parameters	Year 1 (July 2007-June 2008 (before outbreak))	Year 2 (July 2008–June 2009 (during outbreak))	Year 3 (July 2009–June 2010 (post-vaccination))
Number of sows	1,332	1,253	1,452
Number of mating	2,582	2,540	2,792
Farrowing rate (%)	90.0 a	83.9 b	83.8 b
Parity 0	86.6 a	87.0 a	87.2 a
Parity 1	91.2 a	84.4 a	85.8 a
Parity 2–4	91.5 a	82.1 b	84.3 b
Parity ≥5	88.0 a	84.6 a, b	78.3 b
Return rate (%)	5.9 a	8.0 a	11.3 b
Parity 0	7.5 a	8.4 a	10.1 a
Parity 1	5.4 a	8.6 a	10.9 a
Parity 2–4	5.4 a	8.9 b	10.0 b
Parity ≥5	6.0 a	5.5 a	14.8 b
Abortion rate (%)	1.6 a	5.2 b	1.4 a
Parity 0	1.8 a	2.8 a	1.0 a
Parity 1	1.3 a	4.4 a	1.0 a
Parity 2–4	1.4 a	5.9 b	1.7 a
Parity ≥5	2.9 a, b	6.5 b	1.6 a

Clinical signs suggestive of PRRS in late 2008, with virus detected in serum by RT-PCR in January 2009. PRRS MLV vaccination begun 15 May 2009. Different lowercase letters (a and b) across rows indicate statistically significant differences ($P \leq 0.05$)

Discussion

In general, the reproductive performance of this herd was good relative to its peers in Thailand (Olanratmanee et al. 2010; Tummaruk et al. 2010). However, a decline in reproductive performance, i.e., an increase in abortions and mummified fetuses, was noted for several months before the use of the PRRSV vaccine. The decline in reproductive parameters was attributed to PRRSV based on the clinical experience of the herd veterinarians and the results of diagnostic testing, e.g., positive PRRSV RT-PCR testing. These data justified the decision to vaccinate the entire sow herd with PRRSV MLV vaccine, regardless of individual animals' stage in the reproductive cycle. In hindsight, taking this course of action 6 months earlier (at the peak of abortions) might have foreshortened overall reproductive losses (Fig. 1a).

Table 3 Fertility parameters by stage of gestation subsequent to blanket vaccination

Fertility parameter	Stage of gestation			
	0–30 days	31–60 days	61–90 days	>90 days
Number of animals	213	222	228	216
Farrowing rate (%)	88.3 a	85.1 a, b	84.7 a, b	77.3 b
Parity 0	93.6 a	86.5 a	92.3 a	82.5 a
Parity 1	94.9 a	95.1 a	81.8 a	78.4 a
Parity 2–4	81.8 a	77.5 a	82.6 a	77.2 a
Parity ≥5	90.9 a	89.1 a	86.1 a	72.3 a
Return rate (%)	8.5 a	11.3 a	8.8 a	13.4 a
Parity 0	3.2 a	13.5 a	5.1 a	10.0 a
Parity 1	5.1 a	4.9 a	9.1 a	13.5 a
Parity 2–4	11.4 a	16.8 a	11.9 a	16.3 a
Parity ≥5	9.1 a	5.4 a	2.8 a	10.6 a
Abortion rate (%)	0.9 a	0.9 a	2.6 a	5.6 a
Parity 0	0.0 a	0.0 a	0.0 a	7.5 a
Parity 1	0.0 a	0.0 a	2.3 a	5.4 a
Parity 2–4	2.3 a	1.1 a	1.8 a	5.4 a
Parity ≥5	0.0 a	1.8 a	8.3 a	4.3 a

PRRS MLV vaccination on 15 May 2009. Different lowercase letters (a–c) across rows indicate statistically significant differences ($P \leq 0.05$)

Table 4 Litter parameters (means±SEM) by parity over time

Litter parameters	Year 1 (Jul 2007–Jun 2008 (before outbreak))	Year 2 (Jul 2008–Jun 2009 (during outbreak))	Year 3 (Jul 2009–Jun 2010 (post-vaccination))
Number of sows	1,233	1,120	1,365
Number of farrowing	2,362	2,116	2,315
Total born	11.4±0.1 a	10.9±0.1 b	10.6±0.1 c
Parity 1	10.3±0.1 a	10.2±0.1 a	10.2±0.1 a
Parity 2-4	11.6±0.1 a	11.0±0.1 b	10.8±0.1 b
Parity ≥5	11.6±0.1 a	11.2±0.1 a	10.7±0.1 b
Born alive	10.3±0.1 a	9.9±0.1 b	10.0±0.1 c
Parity 1	9.3±0.1 a	9.0±0.1 a	9.4±0.1 a
Parity 2-4	10.6±0.1 a	10.1±0.1 b	10.3±0.1 a, b
Parity ≥5	10.4±0.1 a	10.1±0.1 a	10.1±0.1 a
Stillbirths (%)	7.0±0.2 a	6.1±0.2 b	4.6±0.2 c
Parity 1	7.2±0.5 a	6.9±0.5 a	5.8±0.4 a
Parity 2-4	6.3±0.2 a	5.3±0.3 a	4.1±0.2 b
Parity ≥5	8.2±0.4 a	6.9±0.4 a	4.6±0.3 b
Mummified fetuses (%)	1.6±0.1 a	2.2±0.2 b	0.7±0.1 c
Parity 1	1.8±0.3 a	3.7±0.6 b	1.4±0.3 a
Parity 2-4	1.6±0.2 a	2.1±0.3 a	0.6±0.1 b
Parity ≥5	1.6±0.2 a, b	1.8±0.3 a	0.5±0.1 b

Clinical signs suggestive of PRRS in late 2008, with virus detected in serum by RT-PCR in January 2009. PRRS MLV vaccination begun 15 May 2009. Different lowercase letters (a–c) across rows indicate statistically significant differences ($P \leq 0.05$)

In agreement with previous reports, vaccination produced a measureable response both in terms of an increased proportion of seropositive animals and an increase in mean PRRSV ELISA S/P values (Murtaugh et al. 2002; Scortti et al. 2006b). Although the antibody ELISA does not measure neutralizing antibodies (Yoon et al. 1995; Foss et al. 2002), none of the monitored animals were viremic during the 2 to 18 week observation period post-vaccination.

Vaccination against PRRSV in nonpregnant pigs has been shown to produce no negative reproductive consequences and improve some measures of reproductive performance, e.g., FR, BA, SB, and MM (Dewey et al. 2004; Alexopoulos

Table 5 Litter parameters (means±SEM) by stage of gestation subsequent to blanket vaccination*

Litter parameter	Stage of gestation			
	0–30 days	31–60 days	61–90 days	>90 days
Number of farrowing	188	189	193	167
Total born	10.5±0.2 a	10.6±0.2 a	11.0±0.2 a	11.1±0.2 a
Parity 1	9.8±0.6 a	9.6±0.6 a	10.0±0.4 a	10.4±0.5 a
Parity 2-4	10.1±0.3 a	10.8±0.3 a	11.3±0.3 a	11.1±0.3 a
Parity ≥5	11.2±0.3 a	10.9±0.4 a	11.0±0.3 a	11.6±0.4 a
Born alive	9.2±0.2 a	9.4±0.2 a	10.3±0.2 b	10.3±0.2 b
Parity 1	8.1±0.6 a	8.4±0.5 a	9.2±0.4 a	9.5±0.4 a
Parity 2-4	8.6±0.3 a	9.9±0.3 a, b	10.6±0.2 b	10.4±0.3 b
Parity ≥5	10.2±0.3 a	9.1±0.4 a	10.5±0.3 a	10.4±0.3 a
Stillborn (%)	6.0±0.6 a	6.5±0.9 a	4.8±0.6 a	5.6±0.7 a
Parity 1	6.1±1.7 a	4.8±1.7 a	6.4±1.4 a	7.1±1.8 a
Parity 2-4	5.7±1.0 a	5.2±0.9 a	4.9±1.0 a	3.9±0.8 a
Parity ≥5	6.2±0.9 a	9.0±2.1 a	3.5±0.9 a	7.4±1.3 a
Mummified fetuses (%)	5.3±1.3 a	4.2±1.1 a, c	0.7±0.3 b, c	1.6±0.5 c
Parity 1	9.9±4.1 a	5.3±2.9 a	0.7±0.5 a	1.4±1.0 a
Parity 2-4	6.9±2.2 a	2.7±1.5 a	0.7±0.4 b	1.5±0.8 a, b
Parity ≥5	1.5±1.1 a	5.6±2.0 a	0.7±0.7 a	1.8±0.9 a

PRRSV MLV vaccination on 15 May 2009. Different lowercase letters (a–c) across rows indicate statistically significant differences ($P \leq 0.05$)

et al. 2005). Furthermore, vaccination against PRRSV has been shown to provide protection against reproductive losses. Scortti et al. (2006b) reported that inoculation of unvaccinated, seronegative gilts with PRRSV at 90 days of pregnancy resulted in 43.4 % stillborn piglets, 20 % weak-born piglets, and 76.7 % pre-weaning mortality. In contrast, vaccinated gilts challenged with PRRSV at 90 days of pregnancy farrowed 5.2 % stillborn and reproductive performance otherwise indistinguishable from the negative control group (Scortti et al. 2006b). Overall, Scortti et al. (2006a) concluded that PRRS MLV vaccination did not cause clinical signs or affect reproductive performance in pregnant gilts. However, PRRS vaccination in pregnant pigs, especially during late gestation, has also been shown to have negative consequences in terms of the number of BA, SB, MM, pigs weaned per litter, and an increase of the mortality rate in nursery pigs (Nielsen et al. 2002; Dewey et al. 2004).

Based on the data analyzed in this study, PRRS whole-herd vaccination had neutral, positive, and negative effects on reproductive performance. In particular, the stage of gestation at the time of vaccination affected the reproductive outcome. A lower FR was noted in gilts and sows vaccinated at >90 days of gestation; whereas, a lower BA and a higher proportion of MM was observed in animals vaccinated at 0–30 days of gestation. At the herd level, whole-herd vaccination reduced AR and SB and MM, but did not improve the FR over that observed during the outbreak period and was associated with an increased return rate and a lower TB and BA.

A review of the literature showed that these data are compatible with previous reports that PRRS vaccination in PRRSV-infected herds reduced the duration of PRRSV shedding (Cano et al. 2007a, b) and improved some reproductive performance parameters, e.g., FR, BA, SB, and MM (Alexopoulos et al. 2005). Thus, it may be concluded that the decision to implement whole-herd vaccination using a PRRSV MLV vaccine should be balanced between the benefits derived from reproductive performance improvements, e.g., fewer abortions, stillborn piglets, and mummified fetuses and the effect of vaccination on pregnant females.

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