https://doi.org/10.22319/rmcp.v12i1.5377

Article

Productive performance and costs of swine farms with different PRRS virus vaccination protocols

Elizabeth Araceli Quezada-Fraide [a]

Claudia Giovanna Peñuelas-Rivas [b]

Frida Saraí Moysén-Albarrán [c]

María Elena Trujillo-Ortega [d]

Francisco Ernesto Martínez-Castañeda [c]*

[a] Universidad Nacional Autónoma de México. Facultad de Estudios Superiores Cuautitlán. Departamento de Ciencias Pecuarias. México.

[b]Kansas Smith Farms. EE.UU.

[c] Universidad Autónoma del Estado de México. Instituto de Ciencias Agropecuarias y Rurales. México.

[d] Universidad Nacional Autónoma de México. Facultad de Medicina Veterinaria y Zootecnia. Ciudad de México, México.

*Corresponding author: femartinezc@uaemex.mx

Abstract:

In order to evaluate the productive performance and production costs per weaned piglet and finished pig in swine farms with different PRRS virus vaccination protocols under field conditions, the indicators total piglets born alive, stillborn piglets, weaned piglets, birth and weaning weights, fattening days and final weight, as well as the costs per weaned piglet and finished pig of two farms under a semi-technification regime were analyzed: a) Protocol 1 (P1), a farm with vaccination of breeding sows and piglets; and b) Protocol 2 (P2), vaccination of breeding sows only. The productivity indicators up to weaning were evaluated with a Time-Repeated Measures Design, and the fattening indicators were evaluated with an analysis of variance with comparison of means. The costs were determined using the general

cost formula. It was possible to observe differences in total number of piglets born and of mummies (P<0.05) in favor of P2, weaned piglets, as well as in birth and weaning weights in favor of P1, but no differences in the rest of the variables. Pigs finished in P1 resulted in 12 more days of fattening and a final weight of 3.13 kg more than P2. The costs per weaned piglet were $389.55 and $424.25 Mexican pesos, and the average cost per day of fattening were $10.01 and $11.43, respectively.

Key words: Productivity, Costs, Pigs, Vaccination, PRRS.

Received: 20/05/2019

Accepted: 08/01/2020

Introduction

Porcine Reproductive and Respiratory Syndrome Virus (PRRSV) in swine has caused significant economic losses to the swine industry worldwide[1]. Losses range from 75,000 euros, in a 1,000-sow farm with a "light" infection, to 698,000 euros[2] and $664 million USD per year in the United States[3]. The disease is caused by an arterovirus that emerged in the late 1980s in the USA[4] and later in Europe, spread rapidly and became enzootic in the pig population worldwide[5]. The disease exhibits a wide variety of signs that reflect the virulence of the strain and are related to the physiological stage of the animals, their immune status and the presence of other diseases[5,6]. The first phase lasts approximately 2 wk and is characterized by acute viremia causing anorexia and lethargy, as well as pyrexia, tachypnea and dyspnea and cutaneous hyperemia with cyanotic extremities. The second phase, which may begin before the first phase is completed and can last up to 4 mo, is characterized by reproductive failure, mainly in sows that were infected during their third of gestation[5], and also it generates a respiratory condition in growing pigs[7].

The disease is caused by a RNA virus of which there are two varieties: classical strains (C-PRRSV) and highly virulent strains (HP-PRRSV)[8]. It is also classified according to its genetic variations and antigenic differences, into two types: PRRSV-1, the European type and PRRSV-2, the North American type[9]. Animal health can be complicated when the virus is associated with other pathogens such as type II porcine circovirus (PCVII), *Pasteurella multocida*, *Haemophilus parasuis*, *Bordetella bronchiseptic*, and Mycoplasmas[10,11,12].

After the outbreak, farm production tends to improve gradually (4 to 6 mo), not reaching preoutbreak production levels. On the other hand, when PRRSV remains circulating, the farm is exposed to disease outbreaks and to the persistence of the virus in the herd[12].

The results of interventions to reduce the cost of the disease, mainly in the fattening stages, have been positive, but they allow and cause greater losses in breeding. In 2005, losses in the U.S. breeding herd were 12 % of the total cost of PRRS, while in 2011, the cost in the breeding herd amounted to 45 %[3]. During this period, different intervention protocols have been implemented, including vaccination, depopulation, and biosecurity protocols[13], among others.

The negative impact of PRRS on the economic margin per pig produced has stimulated efforts to control and eventually eradicate the disease. PRRS virus control relies on aspects such as early diagnosis and monitoring, biosecurity, herd management and immunization[14]. However, these standard control methods have not been effective as vaccines do not reduce disease prevalence and many producers have to depopulate after an outbreak[15].

PRRS is a host/virus model in which disease is determined by virus pathogenicity, breeding herd susceptibility and phenotype, bacterial co-infection pressure, and environmental conditions[16].

Therefore, the objective of this study was to evaluate the productive performance and production costs per weaned piglet and finished pig in swine farms with two PRRS virus vaccination protocols to determine their effectiveness and which one offers the producer better productive and economic indicators.

Material and methods

The study was carried out in two farms, which, according to the general technological classification of SAGARPA, correspond to semi-technified farms. The farms are located in the Central Highlands of Mexico. One is in the state of Hidalgo and has a dry temperate climate, an average annual temperature of 14 ºC, and an annual rainfall of about 610 mm. The other is in the State of Mexico with a semi-dry temperate climate, an average annual temperature of 16 to 17 ºC, and an annual rainfall of about 600 mm. The number of breeding sows was 480 and 180 respectively. The data analyzed were taken from individual sows' records. The period was from their first birth to the last recorded birth, with cutoff as of September 2017, in a lapse of their productive life, the latest data recorded being the births of the first half of 2017.

The technical indicators analyzed were total piglets born, piglets born alive, piglets born dead, mummies, piglets weaned, piglets' weight at birth, piglets' weight at weaning, age of the pigs at slaughter, weight of the pigs at slaughter, weight of the pigs at slaughter. The farms have been PRRS-positive since 2003 and a vaccination schedule is being implemented on both farms. Vaccination protocol 1 (P1) considers the farm that vaccinates breeding sows

and piglets at approximately 21 days of age. Vaccination protocol 2 (P2) considers the farm that vaccinates only the breeding sows. Mass/blanket vaccination of breeding sows in both farms occurs every four months. Both protocols use a modified live PRRS vaccine with a dose of 2 ml per animal.

Both farms have similar biosecurity, genetics and feeding schemes.
The cost analyses were performed using the modification to the general cost formula of Muñoz and Rouco[17].

TC=F+V, where TC= cost per weaned piglet, F= fixed costs and V=variable costs.
Fixed costs were formed by F=L=L+S+Co+R+A+A+Fi+OC+Oth<, where: L=labor costs, S=supply costs, Co=energy and fuel costs, R= repair and maintenance, A=depreciation of fixed assets, OC=opportunity costs, and Oth=other minor costs.

Variable costs consisted of the following items: V=((ABS+FB+FP+M+T+OC/(TOTS*W))*z; where: ABS= amortization costs of breeding stock; FS= feeding of the sows; FB= feeding the boar; AB= amortization of the boar; FP= feeding of piglets; M= medications; T= transportation; OC= opportunity costs; TOTS= total number of sows on the farm; W= Weighting factor, since all variable costs will refer to the production unit of a commercial piglet, and z=number of weaned piglets.

Depreciation of breeding animals was calculated as follows:
ABS=(PPS-(SCP-(1-MORBS)))/(ANFS/FSY)-BR; where: PPS= purchase price of the sow, SCP= sow cull price, MORBS= mortality of breeders expressed as a percentage, ANFS= average number of farrowing sow, FSY= number of farrowing per sow per year, and BR= breeder replacement.

The average number of births per breeding herd can be calculated at any time during production, regardless of the physiological stage of the sows.

ANSF = $\sum$(NS * n)/TOTS; where: NS= the number of sows and n= farrowing number.
FSY=365/(114,5+LAC+INT)*(1-NM+NES/SER)), LAC= duration of lactation, INT= weaning-first fertile service interval, NM= total number of miscarriages, NES= number of empty sows, SER= services performed. In turn, INT is formed by the sum of the weaning-first service intervals (INT1), p. 100 first repetitions*21 (INT2), p. 100 second repetitions*42 (INT3), p. 100 third repetitions*63 (INT4) and p. 100 acyclic repetitions average days of onset.

REP=FSY/ANFS and the weighting factor is w = FSY * PBA * (1 - MOR)*(1-MORT); where: FSY= number of farrowing per sow per year, PBA= piglets born alive per farrowing,

MOR= mortality in lactation, MORT= mortality in transition from weaning to commercial piglet expressed in percentage points.

For the calculation of fattened pig costs, the formulas are as follows:
F= F=L+S+Co+R+A+Fi+CO+Oth, applicable for the fattening process and V=((M+FP+OC)/w)*z, applicable to the fattening process, where z is the number of piglets fattened.

In order to determine differences in sow productivity by farrowing (parity) and by type of vaccination protocol, a time-repeated design was used. The best covariance structure was determined and an adjusted Tukey's test was used to determine significance[18]. As for the fattening production data, the variables days to sale and slaughter weight were determined through an analysis of variance.

The differences in income were determined using as a measure 1 weaned piglet and 1 kilogram of fattened pig. The price for the calculation was $28.00 MXN. In the case of weaned piglets, an average value of $800.00 MXN was used.

Results

The two farms analyzed showed similar levels of technology, animal genetics, feeding, production and sanitary management, with the exception of the PRRS vaccination protocols. Table 1 summarizes the productive performance of breeding sows and their piglets between vaccination protocols. It is noteworthy that although Protocol 2 (P2) resulted in a higher number of piglets born (P<0.05), the number of piglets weaned was higher (*P*<0.05) with Protocol 1 (P1).

Table 1: Comparison of results of the vaccination protocol

	Protocol		
	1 (n=1658)	**2 (n=972)**	
Total piglets born	10.88±0.10	11.43±0.12	*P*<0.0006
Piglets born alive	10.13±0.13	10.08±0.15	*P*>0.05
Stillbirths	0.50±0.04	1.00±0.12	*P*>0.05
Mummies	0.34±0.03	0.50±0.03	*P*<0.0001
Weaned piglets	8.94±0.08	8.31±0.09	*P*<0.0001
Litter weight at birth	14.11±0.13	12.44±0.10	*P*<0.0001
Litter weight at weaning	52.33±0.52	49.53±0.59	*P*<0.0005

In this sense, the number of total piglets born per parity was only different in sows in their first farrowing (P<0.05) with values of 10.14 0.11 and 11.28 0.16 for protocols 1 and 2, respectively.

With respect to parities (Tables 2 and 3), differences were observed in the number of mummies in parity one (0.36 ± 0.03 and 0.66 ± 0.05) and parity five (0.36 ± 0.03 and 0.66 ± 0.05); the number of piglets weaned in parity two (9.19 ± 0.11 and 8.01 ± 0.14) and parity three (9.30 ± 0.13 and 7.89 ± 0.17) in favor of P1. In relation to litter weights at birth, the performance was better for P1 at parities one through five. Litter weight at weaning, however, differed in favor of P1 only, in parities of 1 to 3.

Table 2: Reproductive performance over time (parity of 1 to 4)

	Parity number							
	1		**2**		**3**		**4**	
T	1 (n=493)	2 (n=261)	1 (n=401)	2 (n=204)	1 (n=284)	2 (n=160)	1 (n=208)	2 (n=125)
TP	10.14±0.11 *P*<0.0001	11.28±0.16	10.84±0.16 *P*>0.05	10.86±0.18	11.38±0.15 *P*>0.05	11.54±0.20	11.56±0.17 *P*>0.05	11.77±0.23
PBA	9.39±0.17 *P*>0.05	9.65±0.23	10.44±0.19 *P*>0.05	9.59±0.26	10.57±0.22 *P*>0.05	10.53±0.29	10.56±0.26 *P*>0.05	10.34±0.33
SB	0.38±0.03 *P*>0.05	0.93±0.07	0.31±0.03 *P*>0.05	0.81±0.12	0.46±0.06 *P*>0.05	1.12±0.19	0.46±0.05 *P*>0.05	0.95±0.09
MM	0.36±0.03 *P*<0.0001	0.66±0.05	0.32±0.04 *P*>0.05	0.43±0.05	0.38±0.04 *P*>0.05	0.53±0.06	0.51±0.05 *P*>0.05	0.55±0.07
WP	8.70±0.10 *P*>0.05	8.27±0.14	9.19±0.11 *P*<0.0001	8.01±0.14	9.30±0.13 *P*<0.0001	7.89±0.17	9.23±0.15 *P*>0.05	8.46±0.20
LWB	13.55±0.14 *P*<0.0001	12.49±0.20	14.70±0.16 *P*<0.0001	12.40±0.22	14.77±0.19 *P*<0.0001	12.79±0.25	14.69±0.21 *P*<0.0001	12.82±0.28
LWW	51.80±0.59 *P*<0.0001	46.21±0.81	55.18±0.66 *P*<0.0001	46.34±0.92	55.47±0.78 *P*<0.0001	46.94±1.03	54.36±0.90 *P*>0.05	50.39±1.16

T= treatments; TP= total piglets born; PBA= piglets born alive; SB= stillbirths; MM= mummies; WP= weaned piglets; LWB= litter weight at birth; LWW= litter weight at weaning.

Table 3: Reproductive performance over time (parity of 5 to 7)

	Parity number					
	5		**6**		**7**	
T	1 (n=144)	2 (n=98)	1 (n=93)	2 (n=76)	1 (n=34)	2 (n=48)
TPB	11.23±0.21	11.71±0.26	10.90±0.26	11.70±0.29	10.10±0.42	11.29±0.36
	P>0.05		*P*>0.05		*P*>0.05	
PBA	10.24±0.31	10.40±0.38	10.24±0.39	10.05±0.43	9.20±0.62	9.99±0.53
	P>0.05		*P*>0.05		*P*>0.05	
SB	0.47±0.06	1.08±0.12	0.53±0.08	1.13±0.15	0.89±0.23	0.81±0.13
	P>0.05		*P*>0.05		*P*>0.05	
MM	0.36±0.03	0.66±0.05	0.32±0.04	0.36±0.09	0.18±0.12	0.44±0.11
	P<0.006		*P*>0.05		*P*>0.05	
WP	9.05±0.18	8.67±0.22	8.46±0.23	8.59±0.25	8.67±0.37	8.29±0.31
	P>0.05		*P*>0.05		*P*>0.05	
LWB	14.55±0.26	12.33±0.31	13.62±0.32	12.73±0.36	12.89±0.53	12.48±0.44
	P<0.0001		*P*>0.05		*P*>0.05	
LWW	52.14±1.08	52.89±1.31	48.07±1.33	51.41±1.48	49.30±2.18	52.55±1.85
	P>0.05		*P*>0.05		P>0.05	

T= treatments; TPB= total piglets born; PBA= piglets born alive; SB= stillbirths; MM= mummies; WP= weaned piglets; LWB= litter weight at birth; LWW= litter weight at weaning.

The number of total piglets born on the farm with P1, recorded an increase in parities 2, 3, 4 and 5 with respect to farrowing 1 ($P<0.05$). The values were 10.14 ± 0.11, 10.84 ± 0.16, 11.38 ± 0.15, 11.56 ± 0.17 and 11.23 ± 0.21, respectively. In the farm where P2 was applied, there was no difference in the indicator. The number of mummies in P1 exhibited no differences, while the number of mummies in P2 differed ($P<0.05$) in farrowings 1, 6 and 7. The values were 0.66 ± 0.05 (birth one), 0.43 ± 0.05, 0.36 ± 0.09 and 0.44 ± 0.11. The piglet weaning performance with P1 was different in farrowings 2 and 3 with respect to farrowing 1, while no differences in this indicator were recorded for pigs in P2. Birth weights with P1 were lower in farrowings 2, 3, 4 and 5 ($P<0.05$) and exhibited no differences with P2. Finally, litter weaning weights behaved in a regular manner, with differences in piglets weaned under P1 in farrowing 2 and 3, in relation to farrowing 1; in P2, the difference was observed in parity five.

In the case of fattening, the days to sale recorded were 181.08 5.01 and 168.81 4.81 for P1 and P2, respectively ($P<0.05$); the final weight was 95.46 ± 3.27 and 92.28 ± 3.93 ($P<0.05$), respectively.

Production costs per weaned piglet in P1 were $389.55 pesos, and $424.25 in P2. Of the total costs, 93.62 % were variable costs in P1 while, in P2, variable costs were 96.27 %.

The fattening cost was $1,812.81 in P1 and $1,930.07 in P2. With these production indicators, the average cost per day of fattening was $10.01/day in P1, and $11.43/day in P2.

The income per weaned piglet was $410.45 M/N for P1, and $375.75 in P2. Likewise, for a finished pig, the income was $860.07 and 653.77 M/N, respectively.

Discussion

When breaking down the data by parameter, by farm and by parity and comparing it to the PIC[19] production cluster for Mexico, 2.75 and 2.2 fewer total piglets were born in farms with P1 and P2, respectively. As for the number of piglets born alive, the difference was -2.18 and 2.23, and for piglets weaned, -1.95 and -2.58, respectively. These PIC data correspond to the top 10% in terms of production. However, the farms with P1 and P2 are the values are lower by 1.21 and 1.26, respectively, compared to the 10% with the worst productive performance[20]. Similarly, weaning weights were lower in the analyzed farms: -470 and -360 g.

In general, the differences in these results suggest that PRRS affecting breeders can cross the placenta approximately at day 70 of gestation, causing premature deliveries, on the one hand, and a higher number of stillborn piglets and mummies, on the other. Likewise, it is possible to register an increase in pre-weaning mortality, meaning a lower number of weaned piglets[14,21]. Although the literature mentions that an affected farm returns to "relative normality" within six months[14], there are some farms where this disease is chronically found[21]. The PRRS virus creates synergy with other viruses or bacteria that may be the cause of increased mortality during lactation[22]. In an open cycle, the PRRS virus in unstable farms can be detected in all groups of pigs, including piglets[16], which implies that adverse outcomes caused by the virus can occur, even including vaccination protocols.

In studies involving PRRS virus vaccination of multiparous sows and gilts, it has been shown that passive immune protection is conferred on piglets up to 84 days of age, regardless of whether or not the piglets are vaccinated before this period[23]. Although studies on protection at older ages are scarce, these data suggest susceptibility in fattening animals, as long as they are not immunized.

In a study carried out in stable farms with modified live PRRS virus vaccination, it was found that there are some losses and negative changes in the sows' productive indicators[24]. There was a slight increase in the preweaning mortality rate, and there were no significant changes

in the rate of miscarriages, neonatal losses, pigs weaned per litter and wean-to-first-service interval.

Mass vaccination of the entire breeding herd has been reported as a favorable strategy for protection against the PRRS virus, increasing by 1 weaned piglet per sow per year[25]. However, other studies suggest that the change is minimal[24] with no significant differences[26] and even adverse indicators have been reported[27].

Regarding fattening performance, taking as a reference the average PIC results for Mexico, the difference in age was 18 days more for P1 and 6 days more for P2 and -23.63 kg for P1 and -26.81 kg for P2.

PRRS affects fattening pigs by producing respiratory type diseases with pulmonary lesions, allowing other viral or bacterial diseases such as Influenza, *Streptococcus suis*, *Mycoplasma hyopneumoniae*, *Salmonella cholerasuis*, *Haemophilus parasuis*, *Pasteurella multocida*, Porcine circovirus, Porcine coronavirus, and *Actinobacillus pleuroneumoniae*[14,22] to be associated with it. These diseases reduce pig growth, decrease daily gain, and increase days to slaughter.

It is important to note that the farms used in this study are PRRS-stable farms according to the American Association of Swine Veterinarians, and these results are not applicable to PRRS-free farms or PRRS-positive unstable farms.

Despite differences between scientific reports, after 20 years, vaccination against the PRRS virus with modified live virus vaccines continues to provide protection, and the results have been confirmed in 35 million pigs that have been vaccinated[28].

Conclusions and implications

As mentioned, these data provide information to feed economic models that will assist swine production specialists and producers in making field-evidenced decisions regarding the use of PRRS vaccination as a preventive strategy. Although no significance was observed on farms vaccinating breeders, the cost per weaned piglet was lower with P1, at $389.55, than with P2 ($424.25). For the fattening or finishing stage, vaccinating both the breeding sows and the piglets (P1) had the best productive and economic results.

Acknowledgments and conflict of interest

The authors are grateful to the producers for their support and willingness, and to CONACyT for the scholarship that financed the studies of Dr. Elizabeth Araceli Quezada Fraide, veterinary physician.

The authors declare that they have no conflict of interests.

Literature cited:

1. Lunney JK, Benfield D, Rowland RRR. Porcine reproductive and respiratory syndrome virus: an update on an emerging and re-emerging viral disease of swine. Virus Res 2010;154:1–6.

2. Nauthes H, Alarcon P, Rushton J, Jolie R, Fiebig K, Jimenez M, Geurts V. Cost of porcine reproductive and respiratory syndrome virus at individual farm level – An economic disease model. Prev Vet Med 2017;142:16-29.

3. Holtkamp DJ, Kliebenstein JB, Neumann EJ, Zimmerman JJ, Rotto HF, Yoder TK, *et al*. Assessment of the economic impact of porcine reproductive and respiratory syndrome virus on United States pork producers. J Swine Health Prod 2013;21(2):72-84.

4. Keffaber KK. Reproductive failure of unknown etiology. Amer Assoc Swine Pract 1989;1:1-10.

5. Zimmerman JJ, Karriker LA, Ramirez A, Schwartz KJ, Stevenson GW. Diseases of swine. 10th ed. USA: Wiley-Blackwell; 2012.

6. Wang G, Yu Y, He X, Wang M, Cai X. Zimmerman JJ. Porcine reproductive and respiratory syndrome virus infection of bone marrow: Lesions and pathogenesis. Vir Res 2019;265:20-29.

7. Oh T, Kim H, Park KH, Jeong J, Yang S, Kang I, Chae C. Comparison of four commercial PRRSV MLV vaccines inherds with co-circulation of PRRSV-1 and PRRSV-2. Comp Immunol Microbiol Infect Dis 2019;63:66-73.

8. Wang G, Yu Y, Zhang C, Tu Y, Tong J, Liu Yonggang, *et al*. Immune responses to modified live virus vaccines developed from classical or highly pathogenic PRRSV following challenge with highly pathogenic PRRSV strain. Dev Comp Immunol 2016;62:1-7.

9. Xie J, Christiaens I, Yang B, Van Breedam W, Cui T, Nauwynck H. Molecular cloning of porcine Siglec-3, Siglec-5 and Siglec-10, and identification of Siglec-10 as an alternative receptor for porcine reproductive and respiratory syndrome virus (PRRSV). J Gen Virol 2017;98:2030-2042.

10. Yin SH, Xiao CT, Gerber PF, Beach NM, Meng XJ, Halbur PG, Opriessnig T. Current porcine circovirus type 2a (PCV2a) or PCV2b infection increases the rate of amino acid mutations of porcine reproductive and respiratory syndrome virus (PRRSV) during serial passages in pigs. Vir Res 2013;178:445-451 .

11. Li J, Wang S, Li C, Wang C, Liu Y, Wang G, *et al*. Secondary *Haemophilus parasuis* infection enhances highly pathogenic porcine reproductive and respiratory syndrome virus (HP-PRRSV) infection-mediated inflammatory responses. Vet Microbiol 2017;204:35-42.

12. Willis RW, Zimmerman JJ, Yoon KJ, Swenson SL, McGinley MJ, *et al*. Porcine reproductive and respiratory syndrome virus: a persistent infection. Vet Microbiol 1997;55,231-240.

13. Silva GS, Corbellini LG, Linharesa DLC, Bakera KL, Holtkamp DJ. Development and validation of a scoring system to assess the relative T vulnerability of swine breeding herds to the introduction of PRRS virus. Prev Vet Med 2018;160:116-122.

14. Pileri E, Mateu E. Review on the transmission porcine reproductive and respiratory syndrome virus between pigs and farms and impact on vaccination. Vet Res 2016;47:108-121.

15. Prather RS, Whitworth KM, Schommer SK, Wells KD. Genetic engineering alveolar macrophages for host resistance to PRRSV. Vet Micriobiol 2017;209:124-129.

16. Drigo M, Giacomini E, Lazzaro M, Pasotto D, Bilato D, Rueggeri J, Boniotti MB, Alborali GA, Amadori M. Comparative evaluation of immune responses if swine in PRRS-stable unstable herds. Vet Immunol Immunopatol 2018;200:32-39.

17. Muñoz LA, Rouco YA. Análisis de costos de producción de lechón comercial en explotaciones tipo de la Región de Murcia. Archiv Zoot 1995;44:391-402.

18. Kramer CY. Extension of multiple range tests to group means with unequal numbers of replications. Biometrics 1956;12:307–310.

19. PIC. Análisis de la industria porcina en Latinoamérica. PIC 2017;15:1-20.

20. PigCHAMP Magazine. Benchmark. 2016. USA.

21. Morilla A. Manual para el control de las enfermedades infecciosas de los cerdos. 2a ed. ed. México: Manual Moderno; 2005.

22. Schwartz KJ. Swine disease manual. Third ed, Perry, USA: American Association of Swine Practitioners 2005.

23. Kittiwan N, Yamsakul P, Tadee P, Tadee P, Nuangmek A, Chuammitri P, Patchanee P. Immunological response to porcine reproductive and respiratory syndrome virus in young pigs obtained from a PRRSV-positive exposure status herd in a PRRSV endemic area. Vet Immunol Immunopat 2019;218: 09935.

24. Moura CAA, Johnson C, Baker SR, Holtkamp DJ, Wang C, Linhares DCL. Assessment of immediate production impact following attenuated PRRS type 2 virus vaccination in swine breeding herds. Porc Health Manag 2019;5:13.

25. Linhares DCL, Johnson C, Morrison RB. Economic analysis of immunization strategies for PRRS control. PLoS One. 2015;10(12): e0144265.

26. Eclercy J, Renson P, Lebret A, Hirchaud E, Normand V, Andraud M, *et al*. A field recombinant strain derived from two type 1 porcine reproductive and respiratory syndrome virus (PRRSV-1) modified live vaccines shows increased viremia and transmission in SPF pigs. Viruses 2019;11(3).

27. Dewey CE, Wilson S, Buck P, Leyenaar JK. The reproductive performance of sows after PRRS vaccination depends on stage of gestation. Prev Vet Med 1999;40(3-4):233–241.

28. Jeong J, Choi K, Kang I, Park C, Chae C. Evaluation of a 20 year old porcine reproductive and respiratory syndrome (PRRS) modified live vaccine (Ingelvac1 PRRS MLV) against two recent type 2 PRRS virus isolates in South Korea. Vet Microb 2016;192:102-109.