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The Effect of vaccination against PRRSV on quantitative PCV2 PCR results

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Introduction and Objectives

Porcine circovirus type 2 (PCV2) has emerged as a major contributor to disease and mortality with reports of disease (PCVAD) associated with this virus from around the globe. A diagnostic project was initiated in the U.S. in 2006 to better define the role of PCV2 and cofactor dynamics in disease-associated periods of high mortality in commercial swine populations and to evaluate the value of PCV2 quantitative PCR (qPCR) in practice. Studies have shown that clinically sick pigs have significantly elevated duration and magnitude of viremia, usually higher than 1.0 x 10^7 PCV2 genomes per mL of serum. Therefore, the quantification of PCV2 in serum may be a method to accurately identify PCVAD pigs. Literature also suggests that controlling co-factors in PCVAD is essential for the management of the disease (1).

This paper describes the effect of vaccination against PRRSV on quantitative PCV2 PCR results in serum samples.

Material and methods

Fifty-nine herds with confirmed PCVAD were investigated. In each herd, 5 pigs were necropsied at each of three periods: peak mortality; 3-4, and 5-6 weeks pre-peak mortality. In addition, 70 serum samples were collected representing a cross-sectional sampling of sow farm (gilts, sows) and weaned pigs (approximately 4, 10, 14, 18 and 22-24 weeks of age). Diagnostic testing of tissues from necropsied pigs was to identify pathogens associated with PCVAD. The serum samples were analysed to identify the presence of concurrent swine pathogens in the flow. The qPCR PCV2 protocol utilized a TaqMan-based, real-time PCR using a standard curve created from serial dilutions of a plasmid encoding the open reading frame 2 (ORF2) of PCV2 to reveal the viral load in serum samples (2). Each herd completed a survey to elucidate housing, management practices, medications, and vaccinations.

The effects of PRRS vaccination status (yes/no) on qPCR PCV2 were analysed by means of the Wilcoxon-rank-sum test. Results were considered statistically significant if $p \leq 0.05$. The null hypothesis was that vaccination against PRRSV would have no impact on qPCR PCV2 results.

Results

Pigs of 41 projects were not vaccinated against PRRS, pigs of 13 projects were vaccinated with MLV PRRS vaccines and the vaccination status of 5 projects remained unknown. In all vaccinated herds, the PRRS vaccine was applied around weaning.

The analysis of the surveys revealed that peak wasting disease was between 12 and 16 weeks of age in the majority of cases. No peak disease was found before week 8 of age. There was no significant difference in the time point of peak disease between PRRS vaccinated and unvaccinated herds. Based on the Idexx PRRS HerdChek ELISA 2XR more than 76% of the weaned animals were negative at weaning with titres <0.4. The effect of vaccination against PRRS with a modified live vaccine (Ingelvac® PRRS MLV or Ingelvac® PRRS ATP) on qPCR PCV2 results in presented in Table 1.

Table1: Effect of PRRS pig vaccination on PCV2 viral load (qPCR PCV2)

Age in weeks (range)	PRRS Vacc	No animals	Mean rank qPCR	P
4 (3-7)	No	131	79.8	0.2 n.s.
	Yes	30	86.2	
10 (8-12)	No	109	64.0	0.4 n.s.
	Yes	20	70.4	
14 (13-16)	No	111	75.1	**0.02** ***
	Yes	30	56.0	
18 (17-20)	No	110	74.1	**0.04** ***
	Yes	39	57.4	
22- 24	No	110	67.7	**0.03** ***
	Yes	19	49.5	

n.s. = not significant; ***= significant ($p \leq 0.05$)

As evident from table 1 vaccination of pigs against PRRS had no effect on qPCR for PCV2 in 4 – 12 week old pigs ($p>0.05$). However, vaccination against PRRS resulted in significantly lower qPCR results in animals 13 weeks or older compared to non-vaccinated animals. Interestingly, PRRS vaccinates had significantly lower viral loads when peak wasting disease was observed in the herds.

Conclusion

Vaccination against PRRS appears to decrease the magnitude of viremia of PCV2 as measured by qPCR in PRRS positive and PCVAD affected herds. Further research is warranted as this study only included PCVAD case herds and lacks of control herds.

References

1. Allan et all (2000), Vet Rec 170-171
2. Brunborg et al. (2004), J Virol Meth 122, 171-178