

PCV2 infection dynamics under field conditions

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Introduction and Objectives

Porcine Circovirus Type 2 (PCV2) has emerged as a major contributor to disease and mortality in swine. It has been demonstrated that PCV2 viral load is associated with the severity of disease.^{1,2} An objective of this project was to better understand the dynamics of PCV2 viremia in clinical porcine circovirus associated disease (PCVAD) situations in non-vaccinated pigs.

Materials and Methods

Ten farms clinically affected by PCVAD were included in this evaluation, in which a group of non-vaccinated pigs were serum sampled. The diagnostic protocol consisted of serial serum sampling of the growing herd (10 non-vaccinated pigs per age group at approximately 3, 6, 8, 10, 14, 18 and 22 weeks of age). Sample size was based on detecting at least one positive sample when the estimated prevalence of disease was at least 50%. Serum samples were tested using a quantitative PCV2 PCR (qPCR) method to determine the quantity of PCV2 virus. DNA was extracted and PCR was performed per a published method.³ Serial dilutions of a plasmid standard were included in every run to create a standard curve that allowed for determination of the amount of PCV2 virus present in each sample as viral genomic equivalents/ml. For the qPCR test used, “negative” results mean either virus was present below the lower detection limit of the test (10^4 or lower) or was absent altogether. Mortality and cull records were also considered as part of this evaluation.

Results

PCV2 viral loads exceeding the level of detection for the qPCR test ($>10^4$ viral genomic equivalents/ml) were not detected before 10 weeks of age (Table 1). Peak clinical symptoms were also not observed before 10 weeks of age. The age of peak viral load coincided with peak clinical symptoms on all ten farms (shaded cells in Table 1). Peak viremia and peak clinical symptoms occurred at 10 weeks of age on 1 of 10 farms (10%), at 14 weeks of age on 3 of 10

ten farms (30%), and at 18 weeks of age on 6 of 10 farms (60%). No apparent correlation was observed between the age of clinical onset and the severity of disease.

Table1. Distribution of serum PCV2 viral loads in non-vaccinated groups of pigs from 10 different farms.

Sampling (wk)	3	6	8	10	14	18	22	Mort (%)	Culls (%)	Total Loss
Farm 1	*	*	*	*	4.5	6.8	5	9.81	3.04	12.85
Farm 2	*	*	*	*	4.5	6	5.5	4.02	7.7	11.72
Farm 3	*	*	*	*	6.6	7	5.2	10.18	7.24	17.42
Farm 4	*	*	*	4.5	4.8	5.8	5	3.08	4.75	7.83
Farm 5	*	*	*	6.8	5.5	5.3	4.8	7.76	6.31	14.07
Farm 6	*	*	*	6.4	6.6	4.3	n/s	7.07	10.24	17.31
Farm 7	*	*	*	4.2	5.6	5.2	4.8	6.5	7.3	13.8
Farm 8	*	*	*	*	4.6	6.2	5.8	10.3	4.85	15.15
Farm 9	*	*	*	4.3	4.9	4.6	4.2	4.71	10.65	15.36
Farm 10	*	*	*	4.5	4.8	5.8	5	3.08	3.66	6.74

Values represent logs of viral genomic equivalents/ml (ie $4.4=10^{4.4}$). For the quantitative PCV2 PCR test used, “negative” results mean either virus was present below the lower detection limit of the test (10^4 or lower) or was absent altogether. Shaded cells indicate the age of peak viremia and clinical symptoms.

Conclusions

PCV2 viremia was clearly present at high levels in non-vaccinated pigs on these clinically affected farms and coincided with the age of peak clinical symptoms on all farms. Viral load profiles obtained by qPCR illustrated PCV2 infection dynamics in farms clinically affected by PCVAD.

References

1. Segales J. 2002 *Vet Rec* 149:357-361.
2. Segales J. 2007 *Proc. Emerging Diseases Seminar*, Krakow, Poland, p35.
3. Brunborg, I.M. et al 2004. *J Virol Methods* 122:171-179.