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PCV2 disease in vaccinated growing pigs in Southern Brazil

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Introduction

Postweaning multisystemic wasting syndrome, the most common clinical manifestation of porcine circovirus type 2 (PCV2) disease (PCVD), was first described in 1996 in Canada (6). In Brazil, PCV2 was first detected in 2000 in Santa Catarina state where the disease was characterized by wasting and severe lesions in lymphoid tissues (1). PCVD has caused economic losses with high mortality rate in pig farms in several Brazilian states until the introduction of PCV2 vaccine in commercial pig farms in 2008. During December of 2012, a suspect PCVD case was reported in 57-67 days-old pigs in a vaccinated wean-to-finish farm in Southern Brazil. The affected pigs showed coughing, dyspnea, enlargement of inguinal lymph nodes, wasting and diarrhea around 35 to 42 days after housing. A mortality rate up to 5% was registered. The objective of this study was to diagnose and characterize the PCV2 infection in a PCV2 vaccinated pig herd.

Materials and Methods

Lymph node, kidney and lung samples were collected and submitted to laboratory analysis. Tissue samples were processed according to conventional methods for histopathology (HE) and subjected to qPCR (4) and immunohistochemistry (IHC) test (1) to PCV2 detection. DNA sequencing was performed by the Sanger method. The obtained sequences were analyzed and assembled with the Phred/Phrap/Consed software (2). Phylogenetic analyses of the whole genome and the ORF2 gene were performed using the Neighbor-Joining method in the MEGA 5.2 software (7) based on nucleotide and amino acid sequences. Using homology molecular modelling (MODELLER) (5), a structural model of the capsid protein was obtained. This model was validated and the mutated residues were identified.

Results

The diagnostic of PCVD was confirmed by histopathological lesions characterized by multifocal granulomatous lymphadenitis, multifocal lymphohistiocytic interstitial nephritis and multifocal lymphohistiocytic interstitial pneumonia. According to the IHC test results, PCV2 antigen was associated with the lesions. Lymph node samples were positive to PCV2 by qPCR ($5.67x10^{11}$ DNA copies/uL). The complete genome sequence (1.7Kb) of one sample (303/12) was performed. Based on the alignment with other PCV2 strains, the PCV2-303/12 analyzed in this study clustered within the PCV2-b genotype. The comparison among the ORF2 amino acid sequence of the PCV2 described here and other Brazilian PCV2 revealed three amino acids substitutions, in domains 57 (F to I), 178 (N to S) and 190 (A to T). The structural model shows that the N178S mutation probably disrupts the secondary structure of the epitope, which is important to the recognition by antibodies (3).

Conclusions and Discussion

This is the first description of PCVD caused by a PCV2b variant in pigs in a vaccinated herd in Brazil. PCV2 infection was demonstrated by HE, IHC, qPCR and DNA sequencing. In Brazil, both PCV2a and PCV2b genotype have been detected in pigs (1). Nevertheless, after the wide use of PCV2 vaccines, PCV2b became the most prevalent genotype worldwide (6). Moreover, it was possible to build a structural model of the ORF2 gene. The results of the structural modeling indicate a possible disruption in the protein structure around the 178 residue which is an important site for antibodies recognition (3). However, other *in vivo* and *in vitro* studies are needed to confirm this hypothesis.

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References

1. Ciacci-Zanella et al.:2006, Ciência Rural 36:1480-85.
2. http://www.phrap.org
3. Khayat et al.: 2011, J Virol 85:11542.
4. Opriessnig et al.: 2003, Vet Pathol 40:521–529.
5. Sanchez, R. and Sali, A.: 2000, PSP 143:97-129.
6. Segalés et al.: 2013, Vet Microbiol 165:13-20.
7. Tamura et al.: 2007, Mol Biol Evol 28:2731-2739.