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Published in:
6th International Symposium on Emerging and Re-emerging Pig Diseases

Publication date:
2011

Document Version
Publisher's PDF, also known as Version of record

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P.022
DOES PCV2 REDUCE THE IMPACT OF SUBSEQUENT INFECTION WITH LAWSONIA INTRACELLULARIS?

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Introduction

Lawsonia intracellularis (Li) and Porcine Circovirus type 2 (PCV2) infections have both been associated with diarrhoea in weaned pigs and the two infections are often grossly indistinguishable. The prophylactic (vaccines) and therapeutic (antibiotics) strategies for the two infections are different; therefore it is of utmost importance to employ diagnostic tests to distinguish between them at the herd level. The exclusive detection of PCV2 and Li antibodies and/or DNA in serum or other samples, is not diagnostic for the two infections. Therefore, laborious, time-demanding and expensive histopathological examinations often on several animals are required. Thus, there is a need for more sophisticated discriminating diagnostic tests, which can be performed on samples from live animals. The aims of the study were to investigate the interaction between PCV2 and Li.

Material and methods

Briefly, 24 pigs free from PCV1 and PCV2, PRRSV, swine influenza virus (SIV), porcine respiratory coronavirus (PRCV), and a number of other microbiological agents, including *Mycoplasma* spp. and Li were divided into three groups each with 8 pigs. The treatments were as follows: pigs in group 1 were mock inoculated (PK15 cells) post infection day 0 (PID0) and mock inoculated (SPG buffer) PID14. The pigs in group 2 were mock inoculated (PK15 cells) PID0 and inoculated with Li PID14. Pigs in group 3 were inoculated with PCV2 PID0 and with Li PID14. The pigs were killed and necropsied on PID 50.

The PCV2 challenges were performed using a previously described procedure (1) with a few modifications: A Danish field isolate (PK67782-13 – passage 10 - titer 4.9 log10/Ml) of PCV2 genotype IIb were used as virus and the inocula were given by the nares and orally. The Li challenges were performed using the procedure previously described (2), using a tissue homogenate (titer 9-10 log/ML).

Clinical signs and body temperatures were recorded daily. The pigs were weighed weekly throughout the experimental period. Blood samples and faeces were collected prior to the first inoculation and at least once weekly from all animals. Tissue samples of tonsil, liver, spleen, kidney, lung, thymus, and lymph nodes (mesenteric, bronchial, prescapular and superficial inguinal), duodenum, ileum, caecum and colon were collected at necropsy for histopathological examination and immunohistochemistry.

The level of PCV2 and Li excretion were assessed by testing of all faeces samples by the use of quantitative real time PCRs (3,4). Similarly, the serum samples were tested for PCV2 in real time PCR. The serum samples were tested for antibodies against PCV2 and Li using ELISAs routinely performed at the institute.

Results

The laboratory tests performed on the inocula showed that the Li inoculum were contaminated with PCV2, thus the pigs in group 2 received PCV2 in addition to Li at PID14. No clinical signs were seen in pigs from group 1 (mock infected controls). All pigs in group 2 (challenged with Li at PID14) got severe, watery diarrhoea 4 days after challenge lasting 1-2 days and the pigs were clearly clinically affected and depressed. Interestingly, the pigs in group 3 (infected with PCV2 PID0 and challenged with Li PID14) only got a very light and temporal diarrhoea (not watery) 2 days after Li challenge and these pigs were not clinically affected. Tests of faeces samples from the pigs in groups 2 and 3 showed that the excretion of Li on PID 35 and 42 were one log10 (group means) lower in pigs challenged with PCV2 on PID0 and Li on PID14 compared to the pigs challenged with Li only.

Discussion

The differences in clinical signs and shedding between pigs in groups 2 and 3 may either indicate that previously exposure to PCV2 reduced the subsequent host response to Li or that the PCV2 exposure of the pigs at PID0 had a direct protective effect on the PCV2/Li exposure at PID14.

Acknowledgements

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