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The "Kansas Cluster" of severe PMWS cases

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Introduction

In Kansas, the first clinical recognition of severe porcine circovirus associated disease (PCVAD) occurred in November 2005, affecting four separate finisher operations located in the same geographic region of the northeastern part of Kansas. All farms had a history of good health with minimal incidence of common pathogens such as swine influenza, proliferative enteritis, and *Mycoplasma*. Two farms were positive for PRRSV. The first indication of a problem was increased morbidity and mortality during finishing with mortality increasing from 3-5% to as much as 15%. Affected pigs exhibited clinical signs typical of porcine multisystemic disease (MSD) and PDNS. In pigs with MSD, the lymphocytes in germinal centers were consistently replaced by large macrophage-like cells and occasional multinucleated giant cells. Immunohistochemical staining consistently revealed abundant PCV2 antigen. In three of the four affected herds, there were affected pigs with rectal prolapse and weakness in the rear legs. In the brain and spinal cord there was non-suppurative inflammation, but without detectable PCV2 antigen staining.

Results

Whole genome PCV2 sequences were obtained by PCR amplification of lung and lymph node homogenates from12 affected pigs from the four farms. All sequences formed a single group, which were approximately 99.5% identical to a representative 1998 French PCV2b isolate, AF055393. A fifth farm located in the same general area was investigated as fitting within the same PCVAD cluster. However, histopathology showed no evidence of PCVAD. Two PCV2 sequences were obtained and identified as PCV2a-like isolates with only about 94% identity to the PCV2b isolates from neighboring PCVAD farms.

We sought to determine if the boar stud, which supplied three of the four farms with semen, could be the source of the PCV2b virus. Based on the differences between the PCV2a and PCV2b viruses, we designed genotype-specific primers that could be used to identify the different genotypes in tissues. Seven boars, culled because of poor semen production, were necropsied. Gross and microscopic analysis confirmed that all boars were negative for PCVAD. PCR amplification of lung and lymph node homogenates identified only the presence of PCV2a. These results indicated that the boar stud was not the source of PCV2b.

Several of the PCVAD pigs had neurological lesions that were not associated with the presence of PCV2 antigen. One possibility was that the neurologic lesions were the result of a porcine enterovirus (PEV) or teschovirus (PTV). PCR amplification of RNA from tissues of PCVAD-affected animals identified the presence of a PTV-like sequence, which was closely related to a group of PTV-6 isolates. Other viruses were also identified in PCVAD pigs, including PRRSV, adenovirus, parvovirus, influenza virus, reovirus and other members of the PTV group. The identification of additional cofactors continues.

Discussion

Severe PCVAD is a disease problem not previously encountered in this region. Results from this study identified PCV2b as the possible source of PCVAD. Because of an absence of historical information on PCV2 sequences in the four Kansas herds, the temporal association between the introduction of PCV2b and the initial PCVAD outbreak is not known. However, we were able to experimentally reproduce clinical disease by infecting CD/CD pigs with a combination of PCV2b and PRRSV, obtained from the same affected herd. The association of PCV2b with increased severity of clinical disease is unclear. One possibility is that PCV2b possesses increased virulence compared to other isolates, or PCV2b may partially escape existing herd immunity offered by PCV2a. Another possibility is a unique association of PCV2b with disease cofactors. Interestingly, both PCV2a and PCV2b were often found in the same PCVAD affected pig, suggesting that pigs can be co-infected with multiple genotypes.

Since this study began, additional farms throughout the region were diagnosed with severe PCVAD, including the identification of PCV2b. The origin of PCV2b and the mechanism of spread between farms remain unclear. Fortunately, vaccination appears to be effective in controlling the disease.

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