

## SWINE HEALTH

**Title:** The “Kansas Cluster” of severe PMWS cases: characterization of a novel PCV2 as the possible etiological agent. **NPB #06-073**

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### Scientific Abstract

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, formally known as PMWS) 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 revealed abundant PCV2 antigen in affected lymphoid tissues. In three of the four 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. Whole genome PCV2 sequences were obtained by PCR amplification of lung and lymph node homogenates from 12 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 developed steady state and real time PCR assays to distinguish PCV2a from PCV2b. 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 a likely source of PCV2b.

PCR amplification of RNA from tissues of PCVAD-affected animals identified a porcine teschovirus (PTV), which was closely related to a group of PTV-6 isolates. Other viruses were also identified, including PRRSV, adenovirus, parvovirus, influenza virus, reovirus and other members of the PTV group. Disease was reproduced in CD/CD pigs with a combination of PCV2 and PRRSV.

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A randomized controlled clinical trial was conducted to evaluate, under field conditions, the effects of a commercial PCV2 vaccine in a herd with a history of PCVAD. A total of 485 commercial, cross-bred, growing pigs were randomly assigned within litter to receive either PCV2 vaccination or serve as unvaccinated control pigs. PCV2 vaccination reduced finisher mortality, increased finisher pig growth rate, and resulted in fewer lightweight pigs at market. For vaccinated pigs, overall mortality was reduced by 50% and finisher growth rate increased by 9.3%. At market, PCV2 vaccinated pigs were 8.8 kg heavier in the same number of days to market compared to unvaccinated control pigs. In serum from unvaccinated pigs, PCV2 antibody titers increased and PCV2 DNA was detected indicating active PCV2 infection. PCV2 DNA levels were significantly reduced following vaccination. These results indicate that PCV2 vaccination is an effective tool to aid in the control of PCV2 infection.