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The 2009 Allen D. Leman conference proceedings book is made possible by the generous support of **IDEXX**.

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Formatting
Tina Smith

CD-ROM
David Brown

Logo Design
Ruth Cronje, and Jan Swanson;
based on the original design by Dr. Robert Dunlop

Porcine Circovirus Type 2 in swine breeding herds

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Introduction

Porcine Circovirus Type 2 (PCV2) is the causative agent of Porcine Circovirus Disease (PCVD), which included Porcine Multisystemic Wasting Syndrome. In North America, PCVD has caused significant economic losses to producers from increased mortality, culling rates, and poor growth in growing pigs. The introduction of commercial vaccines against PCV2 has been universally successful in minimizing economic losses within growing pigs. Unfortunately, the effects of PCV2 in breeding herds have not been consistently identified, its impact, and the agent's impact on economic performance is not well described within North American herds. This paper will review the impact of PCV2 on breeding herds and present an example of a documented case of PCV2 inducing reproductive losses in a large scale North American herd.

There have been numerous field reports of PCV2 related reproductive failure beginning as early as 1999. In general, these field reports suggest increased rates of mummified and stillborn fetuses. In some cases, decreased rates of conception in farrowing.[1-5] In every case, no impact on economics was reported, but these cases clearly demonstrate that PCV2 can be a cause of reproductive failure. The reports of prevalence of PCV2 causing reproductive failure vary widely ranging from 0-64 percent depending upon the region where the data was collected and the methodologies used.[6-8]

The causes of reproductive failure appear to be due primarily to fetal infection with the virus. Grossly, the pigs infected in utero have cardiac hypertrophy and diffuse myocarditis[4] with isolation of the antigen by both PCR and immunohistochemistry possible in the targeted organs.[4,9,10] In addition, Brunborg reported significant infection of the fetal tissues and it appears that the spleen and heart are adequate samples to collect for the identification of PCV2 in fetal tissues.

When the fetuses are infected it appears to have significant impact on the clinical signs seen. Sows that are infected in the first trimester of pregnancy or immediately prior to breeding as gilts will produce embryonic death and subsequent return to estrus or low number of pigs born alive.[11,12] Infection later in pregnancy tends to produce mummies and stillborns.[9,13-15] It is interesting to note that when litters were infected at 57, 75, or 92 days of pregnancy the virus was only recovered out of the 57 day of gestation pigs and antibodies were only seen in the 75-92 day piglets.[9] In addition, in this experiment, individual pigs were infected in utero and no spread of infection between fetuses was observed.[9]

An infection of breeding herds also appears to have a significant impact on growing pigs. It has been demonstrated that viremic sows have an increased mortality in their piglets as compared to sows that are not viremic in the same herd.[16] In addition, it appears that piglets born to viremic sows have the increased potential for other diseases. Jung in 2006 demonstrated that pigs born of viremic sows had an increased risk of porcine epidemic diarrhea virus infection as compared to that of non-infected sows.[17]

The impact of PCV2 on breeding herds may not be as dramatic as other diseases such as Porcine Reproductive Respiratory Syndrome, but all the evidence would suggest that it can play a role in economic losses within breeding herds.

North American case

A large North American breeding herd, which was free from Porcine Reproductive Respiratory Syndrome, had a significant increase in mummies in the fall of 2008. The mummies increased from approximately 2 percent to over 10 percent for a 10-week period (Figure 1). Diagnostics during this period on stillborn fetuses demonstrated severe myocardial damage due to PCV2 as identified with immunohistochemistry. Following the 10-week spike in mummies, the mummy rate returned to a rate that was twice the rate prior to the outbreak and has remained that way through the spring of 2009.

Additional diagnostics were conducted following the severe outbreak. These diagnostics demonstrated that PCV2 was present in the serum of a high percentage of pre-suckling piglets. Sow colostrum was positive for PCV2 in spite of the fact that dams did not have PCV2 in serum samples.

Mature sows within the same herd did not have a corresponding increase in mummies suggesting that the problem was limited to gilts.

Numerous control measures were attempted during the outbreak to help resolve the mummified fetuses. Beginning in the summer of 2007, gilts destined as replacements were vaccinated with commercial PCV2 vaccine at the time of weaning and for a period of six months (July-December of 2007) gilts were vaccinated prior to entry into the sow herd with a commercial PCV2 vaccine. Once gilts that had been vaccinated at weaning were available for breeding, prebreeding vaccination was discontinued in the winter of 2008.

Upon the outbreak of increased mummies associated with PCV2, vaccination of gilts three weeks prior to entry into the breeding herd was reinitiated and mass vaccination of the entire sow herd was undertaken. It appears, based upon analysis of SPC charts that vaccination was not successful in lowering clinical signs associated with mummies as no SPC signals were generated from either the mass vaccination or the vaccination of gilts prior to breeding.

Discussion

Although PCV2 may not be a common cause of reproductive failure in North American breeding herds, it can have devastating consequences. Routine control measures such as vaccination do not appear to be effective in eliminating clinical signs within the breeding herd. Flow strategies to minimize infection of animals prior to breeding may be necessary to provide better control. We know that none of the available commercial vaccines completely eliminate viremia and it appears that even low rates of PCV2 infection are capable of producing some reproductive losses associated with PCV2.

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