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Efficacy of Circumvent® PCV preventing PCVAD and maintain growth performance during an outbreak of PRRSv in a Canadian pig herd with a history PCVAD

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Introduction- The efficacy of Circumvent® PCV (Intervet Schering Plough Animal Health) was demonstrated during the severe PCV2 disease outbreaks in Canada (de Grau et al., 2007). Many off these outbreaks involved co-infections with PRRSv, SIV, *Mycoplasma hyopeneumoniae* and other bacteria (Carman et al., 2006). PRRSv plays an important role in enhancing PCV2 replication and PCV2 viremia (Rovira et al., 2002). The objective of this field trial was to compare the efficacy and performance of Circumvent® PCV in comparison to unvaccinated control pigs. A severe PRRSv outbreak occurred during the vaccination trial.

Materials and Methods- The trial was carried out using a randomized, non-blinded and controlled design. In total, 350 pigs were selected and ear tagged at three days of age (D) and allocated to one of two treatment groups, vaccinated (VAC) or control (CON) n = 175 pigs. Vaccinations were performed per label recommendations at weaning (~18 D) with a booster 3 weeks later. All pigs were weighed at 3 D, weaning, end of nursery (56 D), 106 D, 143 D and slaughter (165 D). Individual pig treatments were recorded for assessing morbidity. A post mortem was performed on every dead pig. Blood samples were collected from 12 pigs per group at 3 D, end of nursery, 120 D, and slaughter to evaluate antibody titers by ELISA and viremia by qPCR. Viral presence in the nose at 120 D and slaughter and feces at slaughter was determined in the same pigs. All pigs were observed for local and systemic reactions during the first 72 hours after each vaccination. At slaughter, carcass quality information was retrieved.

Results- Due to the PRRSv outbreak in the nursery, birth to finish mortality was approximately 14 % and did not differ between treatment groups. PRRSv was confirmed by laboratory analysis (PCR, genotyping, histopathology) and caused a mortality peak in the first third of the finisher stage. VAC pigs did not develop PCVAD and only CON pigs exhibited gross and microscopic lesions compatible with PCV2 infection. ADG, ELISA titers and viremia differed between the groups. In the finisher (20 to 120 Kg), ADG was 948g for CON pigs vs. 973g for VAC pigs (25 g difference). However, the difference in ADG between the groups during the highest PCV2 viremic period (106 D to slaughter) was 40 g (1,053g vs. 1, 013g). Grower-finisher ADG was lower in viremic pigs in the sampled group (925g) in comparison to non-viremic sampled pigs (994 g). ELISA titer patterns differed between CON and VAC pigs as CON pigs were seronegative during the disease challenge. PCV2 viremia was first detected in CON pigs at 56 D while no viremia was found in VAC pigs before the second dose or during the remaining of the grower-finisher period. At slaughter, 91% of CON pigs were viremic whereas low levels of PCV2 DNA were detected in 2 VAC pigs (17 %) (10E+04 genome copies) ($p < 0.001$). Nasal and fecal samples were qPCR positive for all pigs sampled. The CON pigs had much higher viral load in the nose.

Discussion- The animals used in this trial originated from a pig flow that had been vaccinated to prevent PCV2 infection for more than 18 months and PCV2 challenge appeared to remain high based on the reduced performance in CON pigs and the presence of PCV2 in feces and nose indicating a constant challenge due to shedding mainly by CON pigs. Vaccination appears to be an important tool to reduce shedding and prevent viremia even in presence of severe PRRS challenge. VAC pigs maintained an adequate ADG. Quantitative ELISA titers and qPCR levels appear to provide a good predictor and indicator, respectively of PCV2 viremia prevention in vaccinated pigs.

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