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Experimental porcine circovirus type II infection in young pigs

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Porcine circovirus (PCV) type II has been associated with "Postweaning Multisystemic Wasting Syndrome" (PMWS), although Koch's postulates have not yet been fulfilled with the virus. We conducted an animal trial to assess the clinical, pathological, and immunological responses of young pigs to PCV type II (PCV-II) infection.

Five 8-week-old caesarian-derived, colostrum-deprived pigs free of antibody to both PCV type I and II were inoculated with an isolate of PCV-II recovered from a pig diagnosed with PMWS. Animals received a dose of 10^4 $TCID_{50}$/ml by intranasal and intramuscular routes. Pigs were monitored for clinical signs, viremia, and antibody response for 5-weeks post inoculation (PI). Serum samples were collected on day zero and every 7 days thereafter until the termination of the study on day 35 PI.

No clinical signs were observed in any animals during the study period. In all pigs, however, virus was detected by a PCR assay in serum collected on days 7, 14, and 21 PI. Virus-specific antibodies were detected by an indirect fluorescent antibody (IFA) assay on day 14 PI. IFA antibody titers rose to 1:2560 by the end of the study. In contrast, neutralizing antibodies were not detected until day 28 PI. As neutralizing antibodies developed, cross-reactivity with PCV type I (PCV-I) was also observed on the IFA test. Western immunoblot analysis of serum samples revealed that PCV-II consisted of at least 3 polypeptides with molecular masses of 28, 28.5, and 35kD. The 35kD protein was also demonstrated in PCV-I, suggesting that this protein was responsible for cross-reactivity between PCV-I and -II. Antibody to the 28kD protein was detected in day 14 PI samples and all samples thereafter, indicating that this protein was the most immunogenic. Because of its immunogenicity and specificity to PCV-II, the 28kD protein might provide the antigenic basis for the development of diagnostic tests for detection of PCV-II antibody. Antibodies to the two other proteins were detected on day 21 PI and persisted until the termination of the study, suggesting that one or both of these proteins were associated with virus neutralization. No gross lesions were observed at necropsy (35 days PI). Microscopically, all inoculated pigs had mild, multifocal, lymphoplasmacytic hepatitis. Very mild depletion of lymphoid follicles in tonsil, spleen, and ileum was observed in some pigs. In these tissues, the viral genome and proteins were detected by an *in situ* hybridization technique and immunohistochemistry, respectively.

Collectively, naive young swine were shown to be susceptible to PCV-II. The absence of overt clinical signs and pathological changes compatible with those described for PMWS suggested that factors or conditions not present in this study must be involved in the etiology of PMWS.