Porcine Deltacoronavirus (PDCoV) – Fulfilling Koch's Postulates

Synopsis of Leman Conference proceedings – original presentation courtesy of Dick Hesse, KSU
PDCoV Team Members

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PDCoV has been associated with mixed severity of clinical signs in a number of farms across the US. It appears to cause vomiting and occasional diarrhea in sows and gilts as well as mild to moderate diarrhea and vomiting in recently weaned pigs. These are similar clinical signs to PEDV, but there are some anecdotal differences in disease severity in nursing pigs between PEDv and PDCoV:
PEDV – approaching 100% mortality in nursing pigs, and
PDCoV – mortality ranges from 20-60% in nursing pigs.

There has been some debate as to if PDCoV is really the cause of the clinical signs seen in certain affected herds; so this study set out to determine the sites of tissue localization, routes of viral shedding, duration of virus carriage, kinetics of antibody response, and potential of aerosol transmission of PDCoV.

To accomplish this, 10 PEDV/PDCoV naïve dams and their litters were housed in a BSL2 isolation facility at UNL. Both nursing pigs and dams were challenged at 2 days lactation with 5 ml of 22 Ct fecal derived PDCoV tested free of other enteric viruses.

A. 6 litters and dams PDCoV oronasal inoculated
B. 12 nursing pigs as contact controls
C. 2 litters and dams as aerosol controls
D. 2 litters and dams as negative controls

Clinical signs:
Two days post-inoculation (dpi), sows became clinically affected with soft feces that progressed to diarrhea at 3 dpi. Sows were clinically normal after 8 dpi. Piglets developed diarrhea at 2 dpi with 100% morbidity in inoculated piglets and variable mortality. Surviving piglets returned to normal clinical presentation at 12 dpi. Aerosol control liters became clinically ill at 3 and 4 dpi confirming the potential for limited aerosolization of infectious virus and transmission of disease within a confined space.

Fecal Shedding:
All piglets were shedding virus by 2 dpi and all surviving piglets had negative fecal swabs by PDCoV PCR at 28 dpi (Figure 1). Immunohistochemistry to PDCoV showed infection of virtually all enterocytes at 1 dpi similar to what is observed in PEDV infection. An interesting observation of this study is that both small intestine tissue and mesenteric lymph node tissues were still PCR-positive by 42 dpi.

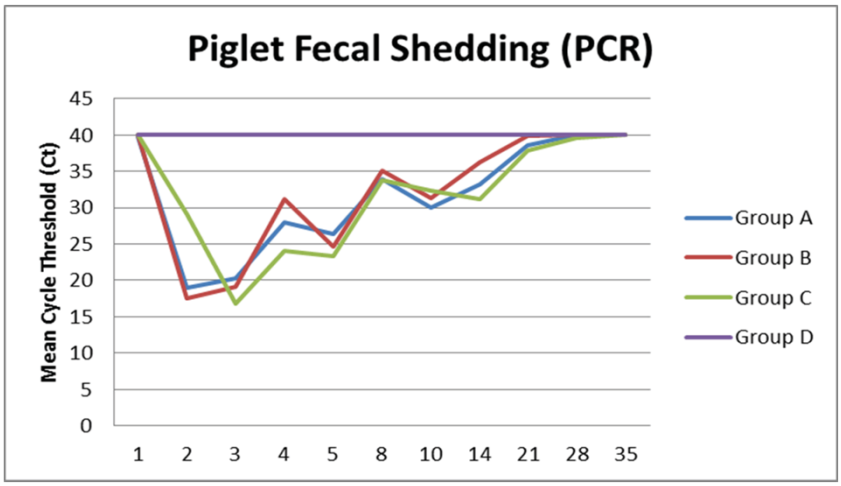

Figure 1: Piglet fecal shedding (measured by Ct-value of fecal swabs) over 35 days post-inoculation

Inoculated sows were all shedding virus in feces by 3 dpi and virus was still detected by 35 dpi in feces, small intestinal tissue, and mesenteric lymph nodes.

Immune Response:
IFA and SN tests showed all animals seroconverting at 14 dpi.

This is an important study to show that PDCoV is a pathogen that can cause clinical disease in both sows and nursing piglets and that the clinical signs are similar to those of PEDV. There maybe an unknown co-factor with PDCoV that has led to the failure of two attempts at experimental infection. Dr. Hesse reported a failure and we at the University of Minnesota also failed to infect pigs prior to a successful challenge with PDCoV field samples in 10-14 day old piglets. This could be indicative of one of the following: specific or limited susceptibilityof different stages of enterocytes, reliance on co-infection with other pathogens, or a difference in virus stability/survivability in the fecal samples used for inoculation. More studies must be completed to evaluate these possibilities. Editor's comments.