SHMP Special Report

PED – We've had 3 breaks in previously infected herds over the last 12 weeks. Talking with the veterinarians involved, gilts were purposefully exposed to live PEDv in the GDU at 2 of the farms and the clinical impact has been mild to moderate. At the 3rd farm, gilts had not been previously exposed and were hardest hit. This farm was able to farrow gilts in different rooms from P2+ sows and the veterinarian thought this, in conjunction with strict biosecurity, helped reduce the impact in sows. This experience builds on our recently completed sow challenge study (Leman 2014) where we reported that after challenge, previously infected sows had significantly lower morbidity and mortality in their litters compared to sows with no previous infection.

PRRS – Looking at the EWMA this week, it seems that the epidemic may be starting. We had an unexpected mini-break from 6/11 to 7/23 with a total of 17 breaks. The majority of the peak was highly influenced by weeks 7/9 and 7/23 when we had 9 breaks. These were spread across 6 systems with no known connection among them.

Bob Morrison (BobM@UMN.Edu)

National Pork Board funded research study update

The pathogenesis and characterization of porcine epidemic diarrhea virus (PEDV) and porcine deltacoronavirus (PdCV) in neonatal gnotobiotic swine.

Courtesy of Oglesbee et al. - The Ohio State University
NPB # 14-188

Virulence of porcine deltacoronavirus in gnotobiotic piglets.

We have determined the virulence of PdCV CVM1 in gnotobiotic (Gn) piglets. Five 19-day-old Gn piglets were randomly divided into two groups. Two piglets in group 1 served as uninfected controls. The three piglets in group 2 were infected with PdCV. Prior to oral challenge, pre-inoculation fecal and oronasal swabs and blood for serum were collected for virus detection from the three piglets of group 2. The three piglets in group 2 were orally challenged with 5.0 ml of filter-sterilized intestinal contents (106 genomic RNA copies of PdCV Ohio CVM1). The two piglets in group 1 were challenged with 5 ml of filter-sterilized intestinal contents from normal piglets, negative for above mentioned porcine enteric viruses. After virus challenge, the piglets were observed and evaluated daily for body weight and temperature changes, and clinical signs of PdCV infection. Daily rectal mucosal/fecal swabs were collected from each piglet for virus detection. At day 3 post-challenge, intestinal contents from the duodenum, proximal jejunum, ileum, transverse colon, spiral colon and descending colon were collected from each pig for viral RNA analysis and histological examination. Within 20 h post-infection, a profuse watery diarrhea, vomiting, and dehydration was observed. Clinical signs were associated with epithelial necrosis in small intestine, resulted in severe villous atrophy. High levels of RNA (9- 11 log RNA copies/g) were detected in intestinal tissues/luminal contents and feces of infected piglets. These results demonstrated that PdCV caused severe gastrointestinal diseases in swine.

This research update can be found at http://www.pork.org/wp-content/uploads/2014/05/Oglesbee-14-188-main.pdf

Editor's comment: This study repeats the findings of Dick Hesse et al. that PdCV can infect piglets and cause clinical signs of infection similar to those of PEDv and TGEv. This work is important because researchers at multiple institutions have had trouble consistently causing infection and clinical signs with samples of PdCV assumed to be viable. It is possible that intestinal pathogen co-infection could play a role in this inconsistency.