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Hemagglutinating Encephalomyelitis Virus (HEV) infection in young pigs

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HEV is an RNA virus. It is a group 2 Coronavirus (TGE and PRCV are group 1 Coronaviruses).

HEV was first described in the early 1960's in Canada and later in Europe. NVSL has prior experience with experimental infection/disease reproduction. Initial observations about HEV infection were based on descriptions in smaller herds in comparison to today's larger herds. In addition, HEV virus infection was monitored by virus isolation and frozen tissue section FA testing, not PCR so there is most likely an increased sensitivity of detection now. So, there appears to be a change in the clinical presentation of HEV.

Historically, HEV in a "non-clinical" form is described as a "uniform infection/distribution in sow herds" (most likely by direct contact). Sows pass maternal antibodies to pigs. These maternal antibodies are "protective" against expression of clinical disease. As these antibodies wear off in older pigs, infection occurs from direct contact and is supposed to be an "asymptomatic" disease in older pigs. Farrowing pigs and nursery pigs shouldn't be circulating virus according to accounts.

What we've observed is:

1. There is a marked difference in the number HEV positive pigs in different pig flows.
2. If you test enough pigs, it isn't unusual to find some tonsils of post-weaning pigs positive for HEV in groups without the clinically associated disease.
3. When we have found HEV in post-weaning pigs in tissues besides tonsil — stomach, lungs, salivary glands and nasal turbinates, it has correlated to an expression of clinical disease. That is, there is a relationship between the number of HEV positive pigs and HEV positive tissues within a pig and clinical presentation.
4. When we find post-weaning clinical disease and go back to the pre-weaning pigs, we also find positives in multiple tissues from pigs with clinical disease issues ranging from inappetence to scours.
5. Items 3 and 4 suggest a disease pattern similar to what we see for flu and PRRSV — apparent non-uniform infection in the breeding herd leading to increased virus circulation and clinical disease in young pigs instead of low circulation with most transmission occurring in older pigs.
6. The clinical expression of this disease is different compared to PRRSV and flu in that it seems to remain at lower morbidity and mortality levels even with "unstable breeding herds."
7. Clinical expression of HEV associated disease is increased by co-infections with PRRSV. I'm also suspicious of a similar interaction with flu but it has not been as obvious as PRRSV.
8. There also appears to be a different clinical expression of the disease not related to a "fall-back" pig syndrome. We can find the virus in lungs of post-weaning pigs — negative for flu, PRRSV and PCV2 — exhibiting a "increased/unexpected" susceptibility to systemic and respiratory bacterial infections, esp. *Streptococcus suis.* This is a clinical/empirical observation that has not been experimentally reproduced to my knowledge.
9. We have been able to grow HEV in HRT-18 cells from some cases but overall it has been difficult to grow. We've been most successful growing it from lung, oral fluids and nasal turbinates.
10. For now, it is an explanation for disease in some situations and it is another reason to try and minimize other disease issues.
11. Serology, even with homologous virus, hasn't been that rewarding in trying to understand the disease. It should be "ok" as a plus/minus evaluation — like parvovirus. It may need a different type of test such as a whole antigen Elisa to better understand the disease using serology.

For the diagnosis of recurring, frustrating post-weaning disease syndromes, I like to have a full set of tissues and serum submitted to rule-out common infectious causes of post-weaning diseases as well as HEV. In addition, whole heads can be submitted (properly chilled) to enable us to sample tonsil, nasal turbinates, salivary glands, peripheral nerves and brain. Five pigs in the early stages of the syndrome as well as five pigs in the end-stage form are useful in order to demonstrate the number of HEV positive pigs

and HEV tissues per pig as well as the range of histological lesions.

Post-weaning disease syndromes are a constant challenge in that there are numerous factors involved that are constantly changing. Minimizing the disease challenge to the post-weaning pig starts in the breeding herd. For some agents, picking when you want pigs to get infected - not preventing infection - is the goal.