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The detection and prevalence of ungulate tetraparvovirus 3 (porcine parvovirus 2) in European porcine samples.

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Introduction: The *Parvoviridae* is host to non pathogenetic and acutely pathogenic viruses across many host species. The novel parvovirus ungulate tetraparvovirus 3 (porcine parvovirus 2, PPV2) was detected in pig sera in Myanmar 2001. Subsequent studies have detected the virus in other countries. In south-eastern China, researchers isolated PPV2 from pigs with clinical symptoms of “high fever disease”. It still remains unclear if there is a correlation between disease association and the presence of this novel virus. The detection and prevalence of PPV2 was conducted on historical diagnostic clinical pig samples from 1996 to 2012 (n=783).
Materials and Methods: Various sample types (n=783) were collected from across Europe; France (n=340), Northern Ireland (n=253), Republic of Ireland (n=53), Great Britain (n=114) and Belgium (n=23). These were tested for PPV2. Viral nucleic acids were extracted from sera and 10% w/v homogenised tissue and faecal samples. In house primers were applied to detect PPV2 by SYBR Green PCR and positive samples were confirmed on agarose gel before sequencing.

Results: The overall prevalence of PPV2 was 19.3% (151/783) and the virus was detected in all countries. PPV2 was represented in most samples types; abortion fluid 1% (1/98), abortion tissue 0% (0/31), sera 6.1% 12/196, nasal swabs 2% (1/52), lung 50% (21/42), heart 13% (1/8), faeces 7% (2/28), colon and small intestine 45% (9/20), tonsil 28% 5/13, spleen 39% (14/35) and lymph tissue 33% (73/219). The pig sample age was known for n=465 of the samples. No PPV2 was present in abortion tissue samples or pre-weaner pigs (0/78). The weaner age group was positive for PPV2 at 12% (14/122), and the fattener and finisher aged pigs were positive at 51% (51/101) and 68% (19/28) respectively.

Conclusion: PPV2 was found to be widespread in European pigs. Presence of the virus in a wide range of tissues suggests a systemic tropism. The age breakdown of PPV2 virus infection indicates post-weaning exposure. This suggests that the majority of sows have been exposed previously and are providing protection to the piglets through maternal antibodies. There was a very low prevalence of PPV2 virus in abortion related samples perhaps suggesting that no vertical transmission occurs.
A number of novel parvoviruses have recently been detected within the pig population. The role in disease is unclear, they may cause disease outright, they could cause subclinical production losses and they could function as co-factors in poly-microbial conditions. In addition they all have the ability to mutate to more pathogenetic forms. As such continuous surveillance and research is important to determine the true role of these viruses in pig health.

Disclosure of Interest: None Declared

Keywords: porcine parvovirus 2, ungulate tetraparvovirus 3

Viral and Viral Diseases
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Senecavirus A outbreak affecting piglets and sows in Southern Brazil

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Introduction: Senecavirus A (SVA), is an emerging *picornavirus* that has been associated with outbreaks of vesicular disease and neonatal mortality in swine. Vesicular disease with unknown etiology and clinically indistinguishable of other vesicular infections have been reported in U.S, Canada and recently in Brazil. Here, we present an outbreak investigation conducted in swine herds showing an increased neonatal mortality and vesicular disease that have been associated to SVA.
Materials and Methods: Herds A and B are small farrow-to-finish (518 and 843 heads, respectively) research farms that belong to the Brazilian Agricultural Research Corporation (Embrapa Swine and Poultry) in Santa Catarina, Brazil. Herd A is located at 0.2Km from herd B. Both herds are vaccinated for PCV2, atrophic rhinitis, *E. coli*, PPV, leptospirosis, erysipelas and M. hyo. Brazil is free of PRRSV, and the farms are located in a Brazilian state free of CSF, PRV and FMD without vaccination. Clinical samples collected from herd A and B included nasal swabs, vesicular fluids and skin of coronary band. For herd B, three piglets were necropsied and tissues samples were collected for histopathology and for RT-PCR for SVA detection, targeting the VP1-VP3 region.
Results: Clinical signs were first seen in Herd A on October 30th, 2015 and remained until November 8th. On Herd B, clinical signs started on November 3rd. On herd A, five gilts, 28 sows (25 in the gestation and three in the farrowing building) and two boars were affected. On herd B, two sows and piglets with 3 to 5 days-old were affected. Sows from both herds presented vesicles mainly on the snout. Gilts and boars showed lameness and ulcerative lesions on coronary bands. Fever was not observed. Piglets were lethargic and had a watery diarrhea. The mortality rate in piglets increased from 11 to 34%. At necropsy, the piglets’s stomach was empty. It was observed enlargement and edema of inguinal lymph nodes, ascites, severe edema of mesocolon and severe necrosis of coronary band. Microscopic lesions were characterized by necrotic epidermitis and dermatitis of coronary band, mild enteritis with villus degeneration on small intestine and marked mesocolon edema. Senecavirus A was detected by RT-PCR in vesicular fluids of sows, skin of coronary band of gilts; and in intestine, tonsils and coronary band of piglets.
Conclusion: The concomitant ocurrence of vesicles in adult pigs and watery diarrhea with increased mortality in piglets was associated with SVA. Affected adult pigs recovered completely in 10-15 days. As SVA is clinically and economically important due to its resemblance with vesicular diseases, the diagnosis tools are essential.

Disclosure of Interest: None Declared

Keywords: Diarrhea, Senecavirus A, Vesicular disease