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Experimental infection of young pigs with an early European strain of PED virus and a recent US PEDV strain

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Introduction:
Outbreaks of porcine epidemic diarrhoea (PED) were reported across Europe during the 1980’s and 1990’s but only sporadic outbreaks occurred in the following years. PED virus (PEDV) spread for the first time into the USA in 2013 and has caused severe economic losses. Retrospectively it was found that two different strains of PEDV termed “INDEL” and “non-INDEL” that reflect the presence or absence of specific insertions and deletions within the S-gene sequence, were introduced into the US at that time. Since autumn 2014, new outbreaks of PED have been reported again in Europe within a number of countries, including Germany, Italy, France, Spain, Germany and The Netherlands. The viruses circulating in these countries are very similar to the “INDEL” strains found in the USA.

Methods:
In this study, weaned piglets were inoculated with an early European isolate (Br1/87) or faecal/intestinal suspensions derived from pigs infected with a recent European strain of PEDV (from Germany) or a US “non-INDEL” strain of PEDV.

Results (1):
No evidence for infection resulted from inoculation of pigs with the German sample that contained high levels of PEDV RNA (Group 2); there were no clinical signs, excretion of viral RNA or anti-PEDV antibody production. No information on PEDV RNA (Group 2); there were no clinical signs, excretion of PEDV RNA (from Germany) or a US “non-INDEL” strain of PEDV derived from pigs infected with a recent European strain of PEDV.

Results (2):
Mild clinical signs of infection, mainly diarrhoea, occurred in piglets inoculated with the Br1/87 (Group 1) and US PEDV (Group 2) strains. PEDV RNA was detected throughout the intestine from 4 days post-inoculation at high levels and PEDV RNA excretion occurred for at least 2 weeks. The US PEDV RNA was detected at low levels in serum samples on multiple days (Table 1). In addition, low levels of viral RNA were detected in lungs and livers with higher levels in spleens (data not shown). Seroconversion against PEDV occurred in infected animals within 10 days. It is apparent that current diagnostic systems can detect infection by the different virus strains.

Conclusions:
Infection of piglets by the early European isolate of PEDV (Br1/87, a close relative of the CV777 strain) and by a US non-INDEL strain of the virus has been performed and it has been possible to monitor infection by each strain using a range of diagnostic assays for the presence of viral RNA and the induction of anti-PEDV antibodies. The mild clinical outcome observed in this study may be related to the age of piglets used. Newborn piglets appear most severely affected by the infection in the field and hence experimental infection of such piglets can be expected to produce a more severe disease and even mortality. Indeed, the outcome of PEDV infections in pigs appears to be dependent upon a range of factors. Potential differences in the pathogenicity of different strains of the virus require side-by-side comparisons with defined virus isolates under carefully defined conditions.

Table 1. Detection of PEDV RNA in samples from inoculated pigs.

RNA was extracted from faecal swab (fs) and serum (ser) samples collected from the individual pigs on the indicated days post-infection (PID) and assayed by RT-qPCR for the presence of PEDV RNA. Samples giving No Ct value (no PEDV RNA detected) are indicated by a (-). The grey blocks indicate that the animals were euthanised on PID 4. Empty cells indicate no samples were collected on that day. Similar samples were collected and analysed from Group 2 (pigs 6-10) but all were negative throughout the study and so the results are not shown.

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