Porcine epidemic diarrhoea

Since the nineties, porcine epidemic diarrhoea has manifested itself as an economically important enteric disease in Asia, where typical outbreaks with neonatal mortality are still occurring. In Europe the disease manifests itself mainly as watery diarrhoea at breeding units.

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Porcine epidemic diarrhoea (PED) is a diarrhoeal disease of swine caused by the coronavirus porcine epidemic diarrhoea virus (PEDV)[1,2]. The disease was first described in Europe in the early seventies and the virus was isolated in 1978[3]. In the seventies and eighties, PEDV frequently caused outbreaks of watery diarrhoea in swine of all ages with high mortality in neonatal pigs. PED thus strongly resembled outbreaks of transmissible gastroenteritis (TGE), a viral diarrhoea caused by another coronavirus which was highly prevalent at that time. In the meantime, TGE as an enteric disease has disappeared in Europe due to a spontaneous mutation of the virus towards the respiratory coronavirus that induces cross-protective immunity against enteric TGEV.

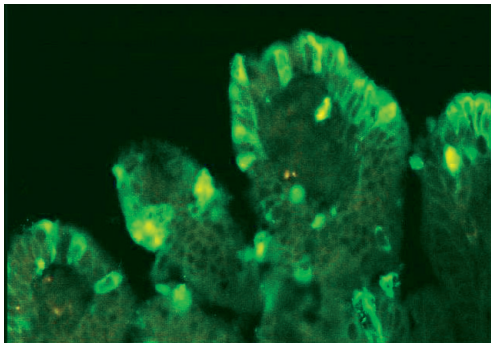
Prevalence in Europe

PED still occurs in Europe but its prevalence is, for unknown reasons, markedly reduced. Serologic surveys in European countries have become scarce. In Spain for example, 55% of 803 breeding farms had animals with antibodies to PEDV 10 years ago. In Belgium, serologic examinations have demonstrated infections with PEDV in 69% of the fattening units sampled in 1986 and in 41% of the units sampled in 1991-92, while no positive animals were found in breeding-fattening farms in 1997, indicating that the virus has circulated less intensively in recent years. However, in some countries such as Hungary, PEDV is still considered to be the most important cause of post-weaning diarrhoea[4]. Also, a case of clinical PED was diagnosed in 1999 in consecutive batches of 8 to 15 week old pigs in a finishing herd in the UK and the disease lasted over 2 months[5].
Recently in the Czech Republic, 27 out of 219 faecal samples from diarrhoeic pigs less than 21 days old were positive for PEDV often in combination with other enteric viruses[6].

Disease descriptions in Europe are now mainly confined to problems of watery diarrhoea occurring persistently and recurrently on breeding farms in subsequent litters of weaned pigs or in feeder pigs entering the fattening stage, particularly when they originate from different sources. Such outbreaks cause growth retardation but mortality is rare. PED is then related to loss of lactogenic immunity or to mingling of infected pigs with uninfected animals in fattening units and this points to an enzootic situation of the virus in some breeding farms where the virus persists in pigs.

Asian outbreaks

Since the nineties, PED has manifested itself as an economically important enteric disease in Asian countries such as Korea and Japan, where typical outbreaks with neonatal mortality have occurred and still are occurring. The virus isolates are no different from those in Europe but the acute and severe clinical appearance probably reflects the entry of the virus into a naive population, as was the case in Europe in the seventies. These Asian outbreaks are characterised by diarrhoea in swine of all ages including sows. Deaths due to watery diarrhoea and dehydration are encountered in neonatal pigs with mortality figures ranging from 50 to 95%. In Korea, for example, 50.4% of 1,258 enteric cases in 5 provinces were diagnosed as PED between August 1997 and July 1999[7].
Several outbreaks were described in Japan in 1993–1994 and in 1996 with 14,000 and 39,000 neonatal pig deaths respectively[8]. The evolution in Asia is thus not caused by a changed virus but presumably due to lack of immunity in the infected swine population. From serologic surveys carried out recently in Korea and India, it appears that PEDV is also becoming enzootic in these



PEDV infected cells on small intestinal villi as shown by immuno-fluorescence.

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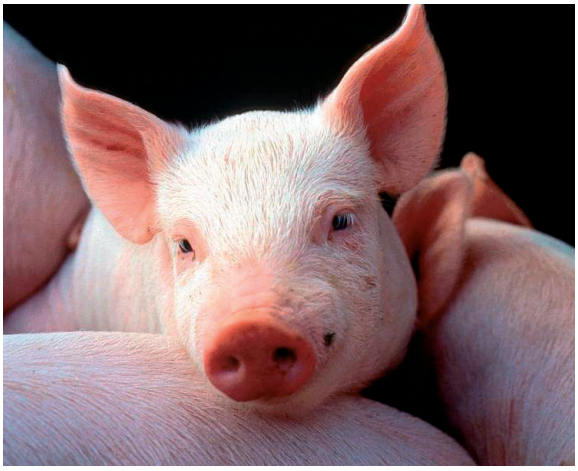

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countries, as an overall average of 21.2 % of 2 to 6-month old pigs were positive in India and 45 % were positive in Korea. To date there have been no reports of PEDV occurrence in North America.

Pathogenesis of PEDV

From a pathogenesis point of view, PEDV is taken up orally and thus the faecal-oral route of spread is most common. Virus introduction mainly occurs through infected pigs but may also take place by virus-carrying fomites such as trucks and boots. Often, in an enzootic situation, the virus is directly transmitted from infected litters to other newly introduced or recently weaned litters.

After oral uptake, the virus replicates in the small intestines and causes villous shortening that is particularly severe in neonatal pigs, where it rapidly leads to watery diarrhoea, dehydratation and death. Older animals easily recover. Virus replication also occurs in the colon but it is not clear how far this contributes to disease. In Europe, outbreaks in the seventies and eighties were sometimes accompanied by acute deaths in older fattening swine and sows (2 to 3%), and particularly in stressed animals, but the pathogenetic mechanism of such acute deaths has never been unravelled and no mention of this has been made in Asian outbreaks.

Differentiation with rotavirus diarrhoea is rather easy since outbreaks with the latter virus rarely involve animals older than 2 to 3 weeks. Differentiation on a clinical basis in typical PED cases where neonatal pigs and older animals are involved cannot be made with TGE but differential laboratory techniques are available. Compared to TGE, mortality figures in neonatal pigs are lower in PED outbreaks as PEDV spreads more slowly.

Diagnosis and prevention

Diagnostic means are fully available in the laboratory to confirm a clinical presumption and they can be applied directly either to faecal material (PCR; ELISA) or to the small intestines of diseased pigs upon euthanasia (immunofluorescence). Serologic examination for detection of antibodies can also be performed on a routine basis using ELISA's on paired serum samples. Routine cultivation of the virus for diagnostic purposes is difficult since adaptation to cell cultures often requires serial passages. Vero cells are susceptible for PEDV.

Weaned pigs suffering from PED diarrhoea must have free access to water. To interrupt the persistence of PED weaning diarrhoea on a farm, the cycle of consecutive weaning must be broken for at least 3 weeks so that susceptible pigs are temporarily absent and the virus dies out.

In outbreaks in which neonatal pigs are involved like in Asia, oral administration of chicken egg yolk or cow's colostrum containing anti-PEDV immunoglobulins to unaffected newborn or to early diseased pigs, is claimed to prevent or reduce losses. Attenuated vaccine strains have also been developed in Asia. In Korea, this was done with the PPEDV-9 strain[9] and in Japan it was realised with the P-5V strain[10]. These vaccines are designed for use in sows to protect newborn pigs. However, their efficacy is not unequivocally established and needs to be elucidated more clearly. **PP**

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