During the seventies, two forms of diarrhea of unknown etiology where clinically described in England and were also regularly observed in other Western European countries such as Belgium. The first form (called epidemic diarrhea type 1) was reported in 1972 (1) and was clinically characterized by acute outbreaks of diarrhea of swine of all ages except suckling pigs. It was described in England (2) and had also been observed in Belgium. It was characterized by a severe diarrhea in pigs of all ages but not in suckling pigs. In many aspects, these latter outbreaks were similar to ETV outbreaks but the virus was definitely excluded by laboratory studies. This second form was called epidemic diarrhea type 2.

In 1976, a coronavirus-like agent was associated with epidemic diarrhea type 2 outbreaks both in Belgium (3) and in England (4). Further studies with the Belgian isolate CV777, have shown that this agent has several physico-chemical characteristics common to the coronavirus (5). However, antigenic relationship could not be demonstrated with several members of this virus family (5). All attempts to cultivate the agent in many porcine cell cultures have failed (6). Experimental infection of colostomized deprived piglets with CV777 isolate resulted in severe watery diarrhea and death. Pathogenesis studies performed by immunofluorescence have shown that virus replication occurs in epithelial cells of ileum and colon (7). Many similarities, therefore, exist with the pathogenetic behavior of porcine parvovirus, but also of several enteric coronavirus in animals species other than swine. The direct immunofluorescence reaction on cryostat sections of the small intestine, has proven to be a very reliable tool for diagnosis of this viral diarrhea in affected field pigs.

Recently, an elisa test was developed in the authors laboratory for the detection of CV777 antigens in feces and an elisa blocking assay was used for detection of specific antibodies in the serum. Using this elisa test, it was shown that CV777 or a serologically closely related virus is present in several Western European countries and also in other parts of the world (8). In fact, outbreaks of diarrhea in fattening pigs in Germany have already been shown to be caused by the CV777 agent (9,10). Using the elisa in combination with immunofluorescence, it was clearly shown that both earlier mentioned epidemic diarrhea's type 1 and 2 are caused by a virus similar or antigenically closely related to CV777. The latter agent, which was originally isolated from a type 2 outbreak, is therefore also responsible for the earlier described epidemic diarrhea type 1 outbreaks. Such conclusion was based on the following results:

- Pigs samples collected from 43 diarrheic pigs on 20 different farms with outbreaks typical for epidemic diarrhea type 1 (no or very mild diarrhea in suckling pigs) were examined for CV777 antigens by elisa and 26 of the 43 samples were positive.
- In serum samples collected from 11 animals on 2 different farms, affected by diarrhea outbreaks typical for type 1, clear seroconversion against CV777 was shown.
- 2 fattening pigs from 2 different type 1 outbreaks were killed and examined by direct immunofluorescence using a conjugate directed against CV777 and both were positive for CV777 antigens in the small intestinal epithelium.

Water diarrhea was reproduced in 2 experimental fattening pigs inoculated with a bacteriuria free filtrate of fecal samples collected from diarrheic animals on a farm with a typical type 1 outbreak. Another pig of the same age became contact-infected and sick 3 days later. CV777 antigens were detected by elisa in the water stools of all 3 animals whereas pre-inoculation samples were negative. All 3 animals also seroconverted.

From these results, it was definitely concluded that both clinical forms of diarrhea earlier described are caused by the same viral agent and it is suggested that a common name should be used in order to avoid further confusion. The name porcine epidemic diarrhea (PED) caused by porcine epidemic diarrhea virus (PEDV) is suggested. A clinical description of PED will be given. Up to now, it is not clear why such variation exists in the clinical disease observed in the field.

PED is self limiting on breeding farms. Serological追随 were carried out on 3 farms (40, 115 and 100 sows respectively) on which an outbreak of PED had been diagnosed. It was shown that herd by animals born 5, 3 and 4 months after the outbreak had occurred had not built up active immunity at the age of about 9 weeks. These results indicate that PEDV does not persist or does not become endemic on a farm after an outbreak. They may also explain why PED outbreaks have a rather explosive character.

REFERENCES


The financial support of I.M.O.N.D., Brussels, is gratefully acknowledged.