The virus has been associated with diarrhea in several animal species such as cattle, dogs, sheep, horses, rabbits, chickens, turkeys, pigeons and pigs. In this last species adenoviruses could be isolated from faeces, brains and kidneys. One of the isolated strains was called 8618. With this and other strains several experimental transmissions were performed. A mild diarrhea sometimes was seen clinically, beside lesions such as hepatitis, enterointestinal nephtitis, myocarditis, hepatitis and encephalitis. Also natural cases of enterointestinal enteritis were suspected.

The natural case of adenovirus enteritis and intestinal lesions of experimentally piglets are reported in this paper.

Results

From a naturally infected piglet the lower jejunal and ileal were examined by histology and electron microscopy. Twenty-one heterotypic-derived, colostrum-derived piglets were used. They were infected with the porcine adenovirus strain 8618. Twelve piglets received 4.10^5 plaque-forming units (PFU) of virus, five piglets received 4.10^5 TCID 50 of virus, five piglets received 4.10^5 TCID 50 and one more piglet received 4.10^4 TCID 50 virus on the second or third day of life. The sera were collected on days 12 and 18 h. The animals were killed at 12 and 18 h. The piglet that received 4.10^5 TCID 50 virus on the second day of life was killed at 12 h. The sera were collected on days 12 and 18 h. The piglet that received 4.10^4 TCID 50 virus on the second day of life was killed at 18 h. The sera were collected on days 12 and 18 h. The piglet that received 4.10^5 TCID 50 virus on the third day of life was killed at 18 h. The sera were collected on days 18 h.

As well as the intestinal lesions were the case, presence of intranuclear inclusion bodies in experimentally infected animals has been described, although no clinical signs of diarrhea were observed. The animals however were challenged by the aerosol inoculation in contrast to the present oocidal route of infection. Other experimental infections with the same virus strain revealed an acute enteritis. The mechanism of diarrhea can be explained by the malabsorption syndrome. This is the result of digestive failure which is caused by destruction of villous absorptive cells. Compared to TGE and CV777 coronavirus enteritis the number of enterocytes destroyed in the present adenovirus enteritis is much lower which reflects a not so decreased villus/crypt ratio as in TGE. The number of goblet cells also is decreased which results in a decreased production of mucus. This can imply a decreased protection of the intestinal wall. A third factor in the cause of diarrhea can be the malabsorption of bile salts in the ileum. Bile salts stimulate the secretion in the common epithelium.

Selected references:
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Acknowledgements
The financial support of the INOEL, Brussels is gratefully appreciated.