

# Transmissible gastro-enteritis

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## Nature of the disease

Recently a respiratory variant of the TGE virus (TGEV), known as the porcine respiratory coronavirus (PRCV) emerged in Europe, causing interference in the diagnosis of TGE.

## Classification

OIE List B disease

## Susceptible species

Clinical disease only occurs in pigs. Cats, dogs and foxes have been described as potential carrier but without clinical signs of the disease.

## Distribution

TGE is present in much of Europe, the Americas (North, South and Central), Asia (including China, Japan and Korea), South East Asia, and parts of West Africa. TGE is suspected in some countries of the region but few surveys were conducted (absent from French Polynesia, New Caledonia, Solomon Islands, Wallis and Futuna).

As a result of the development of the blocking ELISA test (which differentiates between TGEV and PRCV) the prevalence of TGE has dramatically decreased in many Western European countries and the prevalence of PRCV has proportionally increased.

## Clinical signs

When introduced into a susceptible herd, TGE spreads rapidly. Pigs less than two weeks are the most severely affected.

Clinical symptoms include:

In old pigs a mild disease occurs with the following signs:

# Post-mortem findings

Gross lesions are confined to the gastro-intestinal tract, except for the dehydration:

## Differential diagnosis

The following should be considered:

## Specimens required for diagnosis

Virus identification can be done by isolation from tissue culture, fluorescent antibody test, ELISA and PCR. Samples include faeces from carcass, particularly loops of affected small intestine. The loops should be tied-off to retain the contents and kept chilled at 4°C. For immunological methods the best samples are sacrificed piglets (less than 1 week) at the beginning of the disease.

Blood samples (20 ml) should be collected from pigs in the acute and convalescent stages of the disease for serology. Techniques include virus neutralisation, indirect ELISA and competitive ELISA. These tests can be used to demonstrate a free status for trade purpose.

In addition the blocking ELISA test allows differentiation between TGEV and PRCV.

## Transmission

Outbreaks of TGE usually start following the introduction of infected pigs. Sub-clinically infected animals can be a source of infection and recovered pigs often become carriers and can shed the virus for 2-3 weeks in their faeces. Large amounts of virus are excreted in the faeces of infected animals.

The exact mechanisms of transmission of the disease are still unclear; the proposed means are:

In Northern countries the disease is clearly related to winter outbreaks, probably because of the sensibility of the virus to warm temperatures. Therefore it is likely that under tropical conditions, the role of fomites would be less important.

## Risk of introduction

The greatest risk of introducing TGE is through the importation of pigs from endemically infected countries.

Fresh or frozen pigmeat imports from infected countries could represent a potential source as the virus is stable in cold temperatures. The OIE has recommendations on the trade of semen and embryo although there is no formal evidence that these materials are at risk.

## Control / vaccines

Vaccination of pregnant sows has been under investigation but commercial vaccines are not yet available. Commercial oral and injectable attenuated virus vaccines appear to be better at boosting immunity, rather than inducing a primary immune response.

Treatment of TGE should consist in alleviating dehydration and starvation.

To reduce risk of spread, only introduce animals from serologically negative herds and sterilize swills.

If an outbreak occurs, good hygiene practices can help reducing the impact of the disease. It appears to be difficult to eradicate the disease. One approach consists of preventing contamination of newborn piglets by isolation. The other approach aims to minimize the duration of the disease by exposing all the pregnant sows to the disease to develop transplacental immunity of farrows.

## References

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