Oedema disease in pigs: prevention is the solution

Escherichia coli belongs to the Enterobacteriaceae family of facultative anaerobic Gram-negative bacteria.

Within this species there are E. coli strains that belong to the individual's commensal bacteria and other pathogenic strains that may potentially cause gastrointestinal diseases.

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The pathogenic capacity of this bacteria depends on the presence of so-called virulence factors – bacterial antigen structures that are part of E. coli's natural composition – as well as on its capacity to produce toxins, which are substances with various pathogenic effects that act on the individual.

Included amongst the most studied virulence factors are fimbriae (F), flagella (H), somatic antigens (O), and capsules and microcapsules (K). There are several classifications into which we can divide the more than 25 E. coli strains in the gastrointestinal tract of a pig and the more than 120 described species.

If we focus on the combination of the fimbrial antigens, the types of toxins produced, and the pathology that develops in swine affected by E. coli, Table 1 sets out the most prevalent diseases associated with this bacteria in swine.

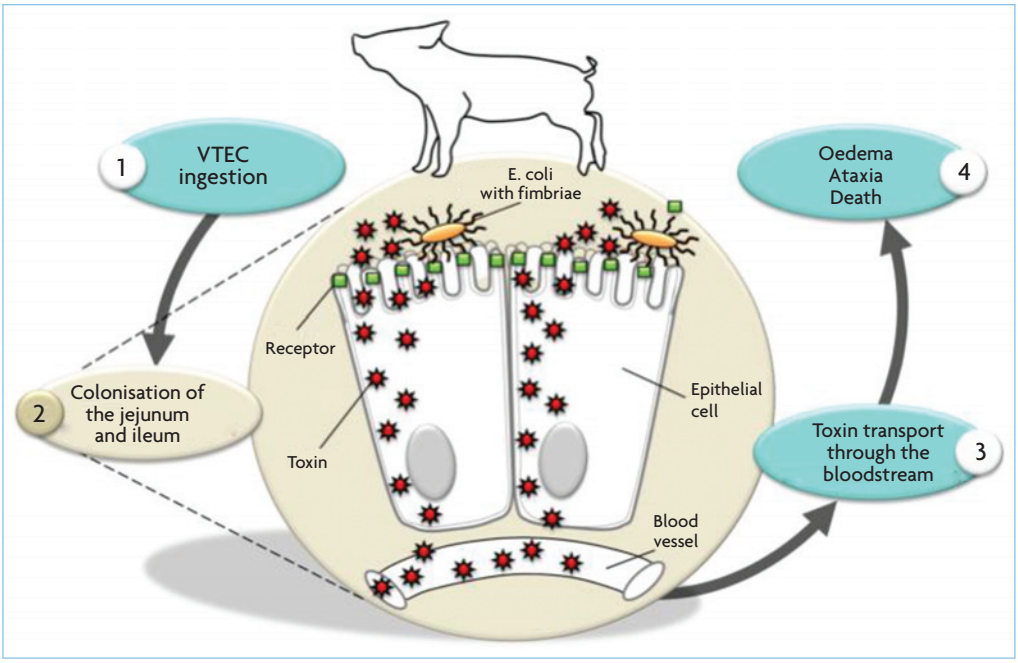

Fig. 1. The epidemiology of oedema disease.

On an epidemiological level, there are many interesting things to observe about the way this bacteria lives in the pig's gastrointestinal tract.

For example, the dominant strains of E. coli may vary from one day to another, with the greatest proliferation and strain variation taking place in the small intestine, while the number of bacteria in the ileum and the rectum remains constant.

During illnesses, these strains are often present in several diseased animals and often continue in successive batches, as mixed infections caused by more than one strain are more common than infections caused by a single strain.

Finally, it should be noted that cleaning and disinfection routines often fail to break the E. coli infection cycle, therefore, infections caused by this agent often recur on farms that are exposed to it.

Table 1. Diseases associated with E. coli in pig farming.

Disease	Fimbrial adhesin	Toxin	Name
Neonatal diarrhoea	F5 - F6 - F41 - F4	STa - STb - LT	Enterotoxigenic E. coli, ETEC
Post-weaning diarrhoea	F4 F18 (F18ac variant)	STa - STb - LT - VT	Enteropathogenic E. coli, EPEC
Oedema diseases	F18 (F18ab, F18 ac variant)	VT (Vt2e)	Verocytotoxigenic E. coli, VTEC
Colisepticaemia, polyserositis, urogenital infection			Extraintestinal pathogenic E. coli. ExPEC

(ST; heat-stable toxin. STa and STb LT; Heat-labile toxin, VT; verotoxin; Vt2e).

The forgotten issue

As shown in Table 1, oedema disease is caused by VTEC, as the Verotoxin 2e (Vt2e) producing or Shiga-like toxin 2e (STx2e) strains of oedema disease-producing Escherichia coli (EDEC) are the only strains able to produce this disease. This dual nomenclature – Vt2e and Stx2e – has a dual origin.

On the one hand, it is due to the lethal effect Vt2e has on Vero cells, culture cells that are commonly used in laboratories, and on the other, the toxin's structure is similar to that produced by Shigella dysenteriae, which led the toxin to have a similar name to that of Shigella (Stx2e).

Normally, E. coli that mostly have F18ab adhesion fimbriae but also F18ac are the strains that produce Vt2e.

This determines the time of onset of the disease, as the F18 E. coli adhesion receptors are not fully present in piglets under 20 days old, which means that the disease appears around 5-14 days after weaning, that is, between 21 and 28 days old.

It is important to consider that oedema disease may also appear during the fattening period, as the introduction of pigs into the feedlot is a critical point in its development.

Once these receptors have been activated, EDEC colonises the small intestine, attaches to it using its fimbriae, and begins to secrete Vt2e.

Once the toxin reaches the bloodstream, it spreads to the target tissue where it acts by destroying the walls of the blood vessels, which leads to the appearance of the oedema that characterises the disease.

	Clinical signs (%)	P-value
Vepured (Hipra)	0.6	‹0.001
Placebo	6.5	

ANOVA test performed with R software was used to calculate statistical differences between groups. Results are statistically significant if p‹0.05.

Table 2. Summary of percentage of animals showing clinical signs of oedema disease.

	Clinical signs (%)	P-value
Vepured (Hipra)	0.3	‹0.001
Placebo	4	

ANOVA test performed with R software was used to calculate statistical differences between groups. Results are statistically significant if p‹0.05.

Table 3. Summary of percentage of animals showing mortality attributed to oedema disease.

When EDEC is ingested in sufficient quantities, it spread very rapidly, reaching massive quantities of around 10^9 CFU/g.

This degree of colonisation and therefore of Vt2e production will determine the development of the infection and with it the presentation of the disease.

The clinical presentation of the disease can differ depending on the degree of colonisation and the Vt2e production capacity.

Different presentations

Clinical or acute form:
This is the typical form of the disease and often presents in animals after they are weaned, but it may also present in animals that have started the fattening period.

This presentation is characterised by its sporadic nature and it may affect the entire group, although only some suddenly die without presenting any other associated clinical sign.

The most common clinical signs are the following:

- Loss of appetite.
- Swelling of the eyelids and the front of the head.
- Typical squealing and snoring sounds.
- Lack of motor coordination.
- Difficulty breathing.
- No fever or diarrhoea.
- In terminal stages, a small number of animals may have watery diarrhoea with fresh blood clots.

Some animals that develop these clinical signs will become exhausted and die.

Mortality rates may vary from low levels of around 1-3% that may even go unnoticed, to extreme levels of between 50-90% of animals in the group.

Chronic form:
The animals that are able to survive the acute form of the disease will remain in the group, presenting growth retardation and possibly unilateral neurological disorders such as 'pedalling', twisting of the head or muscular atrophy in the extremities, with progressive weakness.

Subclinical form:
The piglets are clinically healthy, but develop vascular trauma that may cause growth retardation.

Typical lesions

The following signs are the most common and typical lesions associated with clinical and chronic oedema disease:

- Subcutaneous oedema in the head and eyelids.
- The stomach often appears to contain apparently recent and fresh foodstuff. Oedemas are found in gastric submucosa and occasionally in the gastric fundus.
- Oedematous mesocolon.
- Oedema in the mesentery of the small intestine, as well as the gallbladder.
- Mesenteric and colon nodules often appear oedematous, congestive and swollen.
- Pericardial, pleural and peritoneal cavities may have a slight increase in serous fluid with the possible presence of fibrin strands.
- Pulmonary oedema often occurs, although involvement can vary from slight oedema to sublobular patchy congestion.

Risk factors

Although oedema disease has a single causal agent – Vt2e – it could be considered a multifactorial disease, given the multitude of factors that determine its development. Included among the main risk factors are the following:

- Early weaning.
- Stress during the weaning period and start of the fattening period.
- Absence or reduced presence of maternal antibodies to Vt2e.
- Genetic factors causing rapid growth and high food consumption.
- Mixing of animals of different origins and sanitary statuses.
- High raw protein levels in the first weaning and fattening nutrients.
- Dietary changes, as well as diets with poorly-digestible protein.
- High concentrations of soya beans.
- Low-quality untreated water.
- The presence of concomitant diseases, in particular all those associated with E. coli, such as post-weaning diarrhoea.

Prevention of the disease

Vaccination with a vaccine that comprises a recombinant VT2e antigen is an effective preventive tool against oedema disease.

Hereby we present a study conducted on farms with a history of oedema disease and positive diagnoses for VT2e.

For this, 764 piglets were used for the group of vaccinated animals and 643 piglets were used in the control group.

The animals were followed up from the day of birth until they were sent to the slaughterhouse.

Thanks to this follow-up, a statistically significant improvement was observed in the weight of the vaccinated animals compared to that of the non-vaccinated animals at the time of slaughter (Vaccinated: 109.64 ± 14.35, Control: 105.54 ± 14.81. P-value ‹0.001).

With regard to daily weight gain, the improvement was also statistically significant, with the vaccinated group presenting the best results (Vaccinated: 598.63 ± 82.99 Control: 575.13 ± 85.11 p-value ‹0.001).

Conclusions

While oedema disease is an old pathology, it is still often directly associated with post-weaning diarrhoea. This, added to the fact that oedema disease is not often present in differential diagnoses of piglets with neurological clinical signs and mortality during weaning or at the start of the fattening period, suggests that oedema disease is incorrectly diagnosed and is more prevalent than we may think.

Therefore, we may identify E. coli as the cause of clinical signs other than early age or post-weaning diarrhoea.

When neurological symptoms are observed, which are generally attributed to agents such as Streptococcus suis or Haemophilus parasuis, it is important, for all the reasons given above, to include verotoxin 2e-producing E. coli strains in the differential diagnosis in order to correctly approach the disease and arrive at a correct final diagnosis.

In terms of prevention, vaccination against VT2e on commercial farms has been shown to reduce clinical signs and prevent mortality. ■

Signs associated with clinical and chronic oedema disease.