

# Enteritis in grower-finisher pigs caused by F18-positive *Escherichia coli*

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## Summary

*Escherichia coli* is a common cause of postweaning diarrhea in swine, but usually does not affect grower-finisher pigs. Eleven-week-old grower pigs presented with an acute and severe watery diarrhea, initially considered to be transmissible gastroenteritis. Diagnostic submissions showed the causative agent to be an F18-positive *E coli* producing shiga-like toxin. No pigs developed neurological signs or other conditions usually associated with edema disease. Affected pigs responded to treatment with neomycin. No significant difference was observed in mortality or growth performance between clinically affected and unaffected groups. This case report describes an unusual presentation of F18-positive *E coli* infection in 11-week-old pigs.

**Keywords:** swine, diarrhea, *Escherichia coli*, F18, shiga-like toxin

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## Resumen - Enteritis en cerdos en crecimiento-finalización causada por *Escherichia coli* F18-positiva

La *Escherichia coli* es una causa común de diarrea post destete en cerdos, sin embargo comúnmente no afecta a los cerdos en crecimiento-finalización. Cerdos de crecimiento de 11 semanas de edad presentaron una diarrea acuosa severa, inicialmente considerada como gastroenteritis transmisible. Los resultados de las muestras enviadas a diagnóstico mostraron que el agente causante era una *E coli* F18-positiva que producía una toxina shiga-semejante. Ningún cerdo desarrolló signos neurológicos u otro problema comúnmente asociados a la enfermedad del edema. Los cerdos afectados respondieron al tratamiento con neomicina. No se observó una diferencia significativa en mortalidad ó desempeño del crecimiento entre grupos clínicamente afectados y no afectados. Este reporte de caso describe una presentación inusual de la *E coli* F18-positiva en cerdos de 11 semanas de edad.

## Résumé - Entérite causée par *Escherichia coli* F18 chez des porcs en période de croissance-finition

*Escherichia coli* est une cause fréquente de diarrhée post-sevrage chez le porc, mais n'affecte pas habituellement les porcs en période de croissance-finition. Des porcs en croissance âgés de 11 semaines ont été présentés avec une diarrhée aqueuse aiguë et sévère, suspectée initialement comme étant de la gastro-entérite transmissible. Les résultats des analyses diagnostiques ont permis de montrer que l'agent causal était *E coli* F18-positif producteur de shiga-toxine apparentée. Aucun porc ne développa de signe neurologique ou d'autres conditions associées habituellement avec la maladie de l'œdème. Les porcs atteints ont bien répondu à un traitement avec de la néomycine. Aucune différence significative n'a été observée entre les groupes affectés et les groupes non-affectés quant à la mortalité ou les performances de croissance. Ce rapport de cas décrit une présentation inhabituelle d'infection par un isolat d'*E coli* F18-positif chez des porcs âgés de 11 semaines.

*Escherichia coli* is a common cause of disease in pigs, other livestock species, and humans. In pigs, the main syndromes associated with *E coli* infection are enteritis, edema disease, and septicemia.<sup>1</sup> Enterotoxigenic *E coli* strains associated with enteritis in swine are commonly characterized by their pilus type (F4 [K88], F5 [K99], F6 [987P], F41, and F18) and the presence or absence of gene combination encoding two heat stable (STa and STb) or one heat labile (LT) enterotoxin. Enteritis caused by *E coli* is seen in pigs of

varying ages, and age of onset is usually related to the pilus type due to the presence or absence of pilus-specific receptors on the enterocytes. *Escherichia coli* strains positive for pilus types F5, F6, and F41 affect pigs at a younger age (< 3 weeks of age), while F4-positive *E coli* can cause enteritis in the preweaning and postweaning periods.<sup>1</sup> For *E coli* strains positive for the F18 pilus to cause disease, age-dependent F18 receptors must be present on the enterocytes, and pigs are affected post weaning, usually at > 3 weeks of age.<sup>2</sup> Both F4-positive and

F18-positive *E coli* cause postweaning diarrhea, also characterized by dehydration, lethargy, and wasting, often resulting in a high mortality rate. In addition, F18-positive *E coli* strains can be classified as shiga toxin-producing *E coli* (STEC) if they contain a gene that encodes for a shiga-like endotoxin (Stx2e). The Stx2e toxin is responsible for vascular damage and permeability, resulting in edema disease in pigs, characterized by generalized edema in several tissues (eg, brain, forehead, eyelids, stomach), ataxia, lateral recumbency, dyspnea, and acute mortality.<sup>1</sup>

Enteritis associated with *E coli* is less common in older pigs (> 10 wk), and when it does occur in grower pigs, it is usually associated with other diseases (eg, rotavirus, porcine reproductive and respiratory syndrome [PRRS], porcine circovirus associated disease [PCVAD]). This case

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describes an uncharacteristic presentation of F18-positive *E coli* enteritis in 11-week-old pigs.

## Case description

### Housing

The case farm had been recently converted from a 675-sow, one-site farrow-to-finish operation to a 5600-head finishing facility. It was composed of six original finishing barns (Barns 1 to 6), one converted breeding and gestation barn (Barn 7), and a combined farrowing and nursery building that was no longer being utilized. Each of the six finishing barns was divided into three rooms, each separated by a full wall and door, but with a common pull-plug pit and attic air space. All rooms were fully slatted and housed a total of 250 pigs in 10 equally sized pens. The converted breeding and gestation barn was partially slatted with 50% solid floors and contained 1400 finishing spaces divided among 44 equally sized pens. The site was completely empty of pigs and cleaned prior to receiving feeder pigs from a different farm.

### Pig flow

At the time of clinical disease, 10-week-old feeder pigs (average weight, 22.7 kg) were being placed from multiple nursery sites, but originating from a single sow farm. Approximately 950 pigs were placed each week for 6 consecutive weeks until the finishing site was filled. Pigs were placed in a barn to capacity and remaining pigs were placed in the next sequential barn. Each barn was emptied, washed, and disinfected prior to placement of new feeder pigs. The site was not completely emptied of older animals (near market weight) before the subsequent groups of feeder pigs were placed. At the time of clinical disease, the farm was receiving its second group of feeder pigs since its conversion from a farrow-to-finish farm.

One trailer load (Load C, approximately 475 pigs) of the placement group (that developed clinical signs) experienced a stress event on the day of placement (Day 0). The trailer hauling the pigs became stuck in mud at the finishing site while backing up to the loading chute prior to unloading. The pigs spent 2 hours in the trailer while it sat idle. Weather conditions during that time were 7°C, 79% average humidity, overcast, and no wind. These pigs, along with another trailer load (Load

D, an additional 475 pigs) were placed in Barns 2 and 3. Approximately 750 pigs in Barn 1 and 200 pigs in Barn 2 had been placed 1 week previous (Loads A and B, Day -7). Barns 4, 5, and 6 were empty and clean, while 140 market-weight pigs remained in Barn 7.

### Clinical description

Six days after feeder pigs from Loads C and D were placed in Barns 2 and 3, acute watery diarrhea was seen in > 10% of the group. The following day (Day 7), this increased to > 50%, and diarrhea was seen in pigs from Loads A and B placed in Barn 2 the previous week (Day -7), but not in pigs from these loads placed in Barn 1. On Day 7, Barns 3 and 4 received a new shipment of feeder pigs (Loads E and F) that were clinically unaffected. A company serviceperson was present at the farm on Day 7 to receive feeder pig Loads E and F. Phone conversations between the serviceperson and the author indicated that watery diarrhea was affecting a large number of pigs, and several pigs were seen vomiting.

## Diagnostic testing and results

### Preliminary diagnosis

Immediately, a tentative and precautionary diagnosis of transmissible gastroenteritis (TGE) was made. Swabs (BBL Culture-Swabs with Stuart's medium; Becton, Dickinson and Company, Sparks, Maryland) were used by the serviceperson to collect pooled samples from three to five fresh diarrhea puddles throughout Barns 2 and 3. A total of 10 swabs were collected. Swabs were placed on ice and submitted overnight to the Iowa State University Veterinary Diagnostic Laboratory (ISU-VDL) for testing by polymerase chain reaction (PCR) for TGE and porcine respiratory coronavirus (PRCV). Communications were made to key personnel in the production system to limit the exposure of this site to other farms, pigs, trucks, and equipment. Unnecessary visits to the farm were prohibited until a diagnosis could be determined.

### Preliminary diagnostic testing

Each of the 10 fecal swabs was PCR-negative for TGE-PRCV. It was then determined that a herd visit by the veterinarian would be immediately conducted to inspect the clinical presentation of the disease, collect further information about the course of disease, and collect additional fecal and tissue samples. On the basis of

the clinical signs, TGE was still suspected as the cause, and this raised concerns about the quality, handling, or testing of samples.

## Herd visit

The farm was visited by the veterinarian 3 days after the onset of diarrhea (Day 9). Pigs in Barns 2 and 3 still had severe watery diarrhea. By this time, > 50% of the pigs from the first placement (Loads A and B, Day -7) of Barn 2 were affected. There was still no evidence of diarrhea in pigs in Barn 1 or in pigs placed in Barns 3 or 4 (Loads E and F, Day 7). The 140 remaining market animals in Barn 7 had been sold that morning prior to the veterinarian's arrival; however, no clinical signs were noted in these pigs by the farm manager or serviceperson. There were no other animals on-site.

Clinical signs were characterized by severe, watery, uncontrolled, and projectile diarrhea. Diarrheic feces were present throughout each pen, along the walls, on the side curtains, and in the aisles. Diarrhea from some pigs was seen to project as far as 1 meter and across the aisles into adjacent pens. Individual diarrhea varied in color from dark green-brown, tan, and yellow, to watery-clear appearance (Figure 1). Non-digested feed particles were present in the stools. Diarrhea was also apparent on many pigs in the perineal region and between the hind legs. During the visit, there was evidence of at least five animals vomiting, either observed directly or by finding fresh vomitus in separate pens. There was no evidence of blood, mucous, or parasites in the stools. Approximately 30% to 40% of the pigs appeared dehydrated and chilled, although room temperatures were 18°C to 21°C. Only about 5% of the animals had a noticeable cough, rectal temperatures were within normal range, and no other major clinical signs were observed. There was no evidence of moldy feed in the feeders or feed bins. Feed in all barns was from the same delivery. On the basis of clinical presentation, TGE was still the primary differential, while other considerations were salmonellosis, rotavirus diarrhea, spirochetal colitis (*Brachyspira pilisicoli*), and swine dysentery (*Brachyspira hyodysenteriae*).

## Further diagnostic procedures

### Sample collection

A swab set, comprising one each of three types of swabs, was used to sample composite diarrhea puddles: a Universal Viral Transport Sterile Swab Applicator (Becton,

**Figure 1:** Eleven-week-old grower-finisher pig with severe watery diarrhea caused by a strain of *Escherichia coli* positive for the gene responsible for the F18ac pilus and for genes coding for heat-labile enterotoxin (LT), heat-stable enterotoxins STb and EAST-1, and shiga-like endotoxin (Stx2e).



Dickinson and Company, Franklin Lakes, New Jersey) placed in a sterile 7.0-mL serum Vacutainer (Becton, Dickinson and Company) containing 4.0 mL of sterile 1× phosphate buffered saline (1× PBS) (Fisher Scientific, Pittsburgh, Pennsylvania); a BBL CultureSwab with Stuart's media; and a Viral Culturette collection and transport system (Becton, Dickinson and Company). Each swab in a set was used to sample the same five diarrhea puddles. This process was repeated three more times in different areas of the barn, for a total of 20 diarrhea puddles sampled and four of each type of swab (12 total fecal swabs). In addition, one swab set was collected from a pool of three fresh vomitus samples.

Four untreated pigs showing signs of active profuse watery diarrhea were selected and euthanized (two pigs from each affected barn). Postmortem examinations of all four animals revealed large volumes of watery ingesta and fecal material throughout the small intestine, cecum, and colon. All animals had full stomachs. Spleen, liver, and mesenteric lymph nodes were of normal size and without gross pathology. There were no gross lesions suggestive of ulcerative colitis. Small areas of patchy consolidated pneumonia in one pig affected < 5% of the lung. All other pigs and organs were unremarkable.

Tissue samples, focused on intestinal tract organs, were collected from each pig and pooled by barn. Multiple sections of stomach, duodenum, jejunum, ileum, cecum, colon, and lymph nodes (mesenteric and colonic) were collected fresh and fixed in 10% formalin. Tissues and swabs were placed on ice and shipped overnight to the ISU-VDL for testing.

### Diagnostic testing

Tissue samples were submitted for histopathology and for immunohistochemistry (IHC) testing for TGE virus, rotavirus, PRRS virus (PRRSV) and porcine circovirus type 2 (PCV2). Fecal contents were submitted for bacterial culture and sensitivity. The four fecal swabs and one vomitus swab submitted in 1× PBS were pooled at the laboratory and tested for TGE-PRCV by PCR and for type A rotavirus by antigen-capture enzyme-linked immunosorbent assay (ELISA). All other swabs were held in case additional testing was warranted.

### Diagnostic results

Histopathological examination of the small intestines of all pigs revealed long slender villi with mats of short, rod-shaped bacteria adhered to the surface of the enterocytes

(Figure 2). Small intestinal mucosa was diffusely infiltrated by lymphocytes and lesser numbers of plasma cells. Colonic lesions were described as having multifocally eroded and flattened enterocytes with subjacent neutrophils. Glands were infrequently dilated and contained necrotic cellular debris. The mucosa was expanded by lymphocytes, plasma cells, and lesser numbers of neutrophils. No lesions suggestive of viral enteritis (ie, villous atrophy) were seen. Histological diagnoses of acute diffuse enteritis and acute multifocal erosive colitis were made. Immunohistochemistry testing on intestinal sections was negative for TGE virus, Type A rotavirus, PRRSV, and PCV2. The fecal-vomitus swab pool was negative by PCR for TGE-PRCV and by antigen-capture ELISA for type A rotavirus. Culture of fecal contents revealed high numbers of hemolytic *E coli*. Further characterization of the *E coli* isolate was carried out. *Salmonella* enrichment and *Brachyspira* species cultures on intestinal contents were negative.

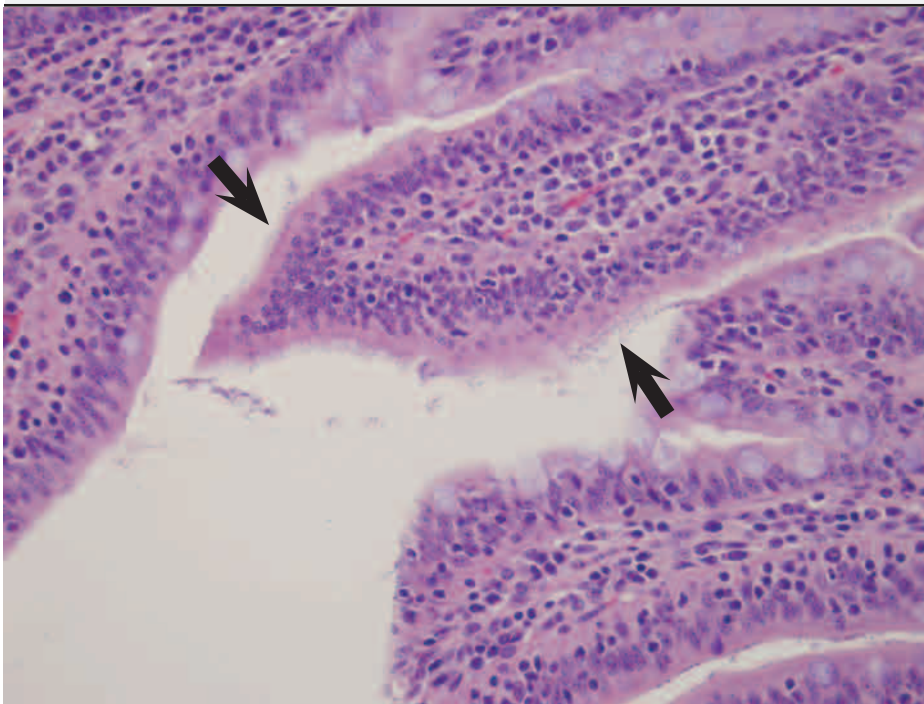
### *Escherichia coli* isolate characterization

The *E coli* isolate was sensitive to ampicillin, ceftiofur, enrofloxacin, neomycin, and trimethoprim-sulphamethoxazole and was resistant to all other antimicrobials tested. Molecular genotyping of the *E coli* isolate was positive for genes encoding the F18 pilus and toxins STb, LT, and Stx2e.

The *E coli* isolate was forwarded to the University of Minnesota Veterinary Diagnostic Laboratory, St Paul, Minnesota, for *E coli* virulence-factor PCR (genotyping), specifically to evaluate the presence of genes encoding for adhesion involved in diffuse adherence (AIDA-1), attaching and effacing factor (eae), and porcine attaching and effacing-associated factor. The isolate was confirmed to have STb, LT, and Stx2e toxin genes, and also a gene encoding for heat-stable enterotoxin 1 (EAST-1). The isolate was negative for genes encoding AIDA-1, eae, and porcine attaching and effacing-associated factor.

The isolate was also forwarded to the *E coli* Reference Center at the Pennsylvania State University, University Park, Pennsylvania, for further characterization. Testing confirmed the above characteristics and further classified the *E coli* isolate as F18ac-positive.

**Figure 2:** Mats of small, rod-shaped bacteria (arrows) adhered to enterocytes of an 11-week-old pig with severe acute watery diarrhea (H&E stain; magnification  $\times 200$ ). Culture of fecal contents revealed high numbers of hemolytic *Escherichia coli*, and molecular genotyping identified *E coli* positive for genes coding for the F18ac pilus and for heat-labile enterotoxin (LT), heat-stable enterotoxins STb and EAST-1, and shiga-like endotoxin (Stx2e). *Salmonella* enrichment and *Brachyspira* species cultures of intestinal contents were negative.



## Diagnosis

Histological lesions, culture, and isolate genotyping were consistent with enteric colibacillosis caused by *E coli* positive for F18ac, LT, STb, EAST-1, and Stx2e.

## Treatment

At the time of sample collection (Day 9), treatment of the affected animals was initiated with neomycin sulfate (NeoMed 325; Bimeda, Inc, Le Sueur, Minnesota) in the drinking water at a rate of 22 mg per kg of body weight for 5 days, followed by 5 days of electrolytes (Ag ProVision electrolyte pack; Animal Science Products, Inc, Nacogdoches, Texas) also metered through the water. The course of treatment was completed and no other antibiotics were given when sensitivity testing and clinical response to treatment confirmed a sensitivity of the *E coli* isolate to neomycin. Treatment of disease was considered successful by the cessation of clinical diarrhea near the end of the neomycin treatment period, as witnessed by the producer and company serviceperson.

## Serological testing

Twenty-one days after clinical signs had first been noticed, blood samples were

collected from 10 pigs in each of three barns (Barn 1, unaffected; Barn 2, severely affected; and Barn 3, moderately affected) to attempt to better understand the disease process. Samples were submitted overnight to the ISU-VDL for testing for antibodies to TGE and PRCV, PRRSV, *Salmonella*, and *Lawsonia intracellularis*. All samples were seronegative for TGE virus and seropositive for PRCV (Svanovir TGEV-PRCV-Antibody ELISA; Svanova Veterinary Diagnostics, Uppsala, Sweden; positive test, > 60% inhibition). All pigs tested in Barns 1, 2, and 3 were seropositive for PRRSV (HerdChek PRRS 2XR ELISA; Idexx Laboratories, Inc, Westbrook, Maine; positive test, sample: positive [S:P] ratio  $\geq 0.40$ ), with average S:P ratios of 2.44 (SD, 0.92), 2.29 (SD, 0.81), and 2.33 (SD, 0.73), respectively. Ten samples of 10, 8 of 10, and 7 of 10 were seropositive for *Salmonella* in Barns 1, 2, and 3, respectively (HerdChek Swine *Salmonella* Antibody ELISA; Idexx Laboratories, Inc; positive test, S:P  $\geq 0.25$ ). Two of 10 samples from each of the three barns were seropositive for *Lawsonia intracellularis* (bioScreen Ileitis Antibody ELISA; Synbiotics Europe SAS, Lyon, France; positive test, S:P  $\geq 0.40$ ).

## Finishing performance

Finishing performance was evaluated for affected lots and compared to an equal number of lots not clinically affected. Only group summaries were available for comparison and not individual pig performance; therefore, weights, average daily gain, and feed conversion could not be compared. Chi-square analysis of percent mortality, percent culls, and both combined demonstrated no statistical difference between clinically affected and unaffected groups (Table 1). The impact of subclinical disease or other diseases on the clinically unaffected barns is not known.

## Discussion

The mechanism by which *E coli* causes diarrhea is primarily hypersecretory. Production of diarrhea is mediated first by attachment of the *E coli* via fimbria (pili) to receptors on enterocyte microvilli, then initiation of production of one or more enterotoxins released into the intestinal lumen. These enterotoxins enhance chloride ion secretion (LT), inhibit sodium ion absorption (LT, STa, and EAST-1), cause fluid secretion (STa and EAST-1), and possibly activate enteric nerves (STb), leading to severe fluid loss into the intestinal lumen, thus resulting in diarrhea and dehydration.<sup>3</sup> It has been shown that *E coli* strains with the capability to produce multiple enterotoxins produce more severe diarrhea, likely through additive effects of the enterotoxins.<sup>4,5</sup> Shiga-like toxin, also known as a verotoxin, is considered an endotoxin.<sup>6</sup> The Stx2e toxin enters the bloodstream via the intestinal wall and causes destruction of vascular endothelium, resulting in inflammation, permeability, and edema. In addition to being an endotoxin, the Stx2e toxin experimentally causes diarrhea, sometimes hemorrhagic, due to the toxin's ability to cause ischemic necrosis and hemorrhage of the intestinal epithelium.<sup>6,7</sup> Diarrhea in cases of edema disease is usually related to the STEC's ability to co-produce endotoxins and enterotoxins. Multiple combinations of these toxins can be found in various *E coli* isolates and pilus types; however, the genes associated with Stx2e production are found most commonly in F18-positive *E coli* isolates that produce one or more enterotoxins.<sup>8-10</sup> Two fimbrial variants are currently classified within F18-positive *E coli*: F18ab and F18ac.<sup>11</sup> Most F18-positive isolates from cases of edema disease

**Table 1:** Comparison of finishing performance between groups of 11-week-old pigs clinically affected with severe watery diarrhea (Barns 2 and 3) and unaffected (Barns 1 and 4)\*

	Barn			
	1	2	3	4
Clinically affected	No	Yes	Yes	No
Number started	751	754	742	682
Average start weight (lb)	47.55	51.48	48.54	49.32
Mortality (%)	2.4	4.2	3.9	4.0
Culls (%)	4.6	4.6	2.8	5.2
Average days on feed	121	118	123	126
Average daily gain (lb/day)	1.73	1.68	1.68	1.57
Feed conversion (lb feed/lb gain)	2.39	2.62	2.35	2.85
Average weight sold – markets (lb)	257.95	251.79	257.27	250.37
Average weight sold – culls (lb)	116.82	120.21	127.35	123.88

\* Percent mortality, culls, and mortality and culls combined were not statistically different between groups as tested by chi-square at  $P < .05$ .

have been characterized as F18ab-positive; however, this is not exclusive and some have been classified as F18ac-positive, as in this case.<sup>11-13</sup> The characteristics, classifications, and pathogenesis of *E coli* have been reviewed in further detail elsewhere.<sup>14,15</sup>

In the case described above, it is clear that the severe diarrhea was caused by the enterotoxins (LT, STb, and EAST-1) produced by the F18-positive *E coli*. Clinical disease was limited to two barns where one shipment of feeder pigs were placed, indicating a difference in the epidemiology of the disease in this subpopulation. The fact that one shipment of pigs had a stress event prior to unloading likely predisposed this group of pigs to develop disease. Colibacillosis in grower-finisher pigs is uncommon and is usually attributed to a stressful event, immunocompromised hosts, or concurrent disease (eg, PRRS, PCVAD).<sup>1,16-19</sup> Stressful events, such as weaning, cold stress, and mixing, can enhance *E coli* infections, disrupt the intestinal mucosa, and increase fecal shedding of *E coli* in swine.<sup>20-22</sup> In addition to the stressful event, these pigs had been exposed to PRRS, *Salmonella* serovars, and *L intracellularis*, as demonstrated by ELISA serological testing; however, clinically unaffected pigs in other barns had similar levels of exposure to these diseases. No concurrent infections were diagnosed in the samples submitted to the ISU-VDL. It is unclear whether secondary disease predisposed these pigs to develop such severe diarrhea.

Interestingly, there was a lack of typical neurological signs usually associated with edema disease and no evidence of edema, which would be expected with an F18-positive, Stx2e-positive *E coli* infection. There are several possibilities as to why edema disease did not manifest in this case.

The *E coli* was positive for the *Stx2e* gene by PCR; however, this suggests only that the isolate has the ability to express the gene and does not relate to expression in vivo. Clinical development of edema disease is dose-dependent on the number of STEC colony-forming units, likely relating to the total number of STEC organisms present in the gut, and also possibly delayed until adequate levels of toxin are present.<sup>23,24</sup> It is possible that either numbers of STEC or amount of Stx2e were not at high enough concentrations to cause edema disease in these relatively older, larger pigs.

Incidence of edema disease in pigs is age-dependent due to increased expression of F18 receptors on enterocytes as the pig increases in age.<sup>2</sup> Receptors are expressed starting at approximately 3 weeks of age and persist until at least 23 weeks of age,<sup>25</sup> which can result in edema disease in older pigs.<sup>19</sup> Grange et al<sup>26</sup> showed age-related reduction in expression of the LT receptor GM1. It may be speculated that similar age-related reduced expression of the Stx2e receptor, Gb4, may explain the lower

incidence of edema disease in older pigs. In this case, the severity of the diarrhea suggests that the dose of STEC and amount of toxin production were sufficient to cause edema disease. Therefore, it is more likely that either a higher tolerance to the Stx2e endotoxin or fewer Gb4 receptors in these older pigs was responsible for the lack of edema disease.

This case demonstrates an unusual presentation of F18-positive, Stx2e-positive *E coli* infection in 11-week-old grower-finisher pigs resulting in severe watery diarrhea but without neurological signs of edema disease.

## Implications

- Strains of *E coli* positive for the F18 pilus, enterotoxins, and Stx2e endotoxin should be added to the list of differential diseases that cause severe diarrhea and vomiting in grower-finisher pigs.
- Episodes of stress or concurrent disease may predispose grower-finisher pigs to clinically express diseases not commonly observed in those age groups.

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