CASE REPORT

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Endemic transmissible gastroenteritis: Difficulty in diagnosis and attempted confirmation using a transmission trial

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Summary

A commercial farrow-to-wean herd experienced an outbreak of preweaning diarrhea of several month's duration. Routine diagnostic laboratory tests on affected piglets suggested the involvement of a number of enteric pathogens, including transmissible gastroenteritis virus (TGEV), rotavirus, coccidiosis, enterotoxigenic *Escherichia coli,* and *Clostridium perfringens*. Even though TGEV could not be consistently demonstrated in all affected pigs, it was suspected to be the main cause of the piglet diarrhea. However, it was difficult to confirm that virulent TGEV infection was the cause of the ongoing diarrhea problem using routine diagnostic serologic and antigen detection tests, because affected pigs were not identified early in the disease process and because a modified-live TGEV vaccine was being used concurrently in the herd. Subsequently, a transmission trial was used to confirm the presence of virulent TGE. In this trial, healthy pigs from a specific-pathogen-free (SPF) herd were commingled with diseased pigs from the case herd. The SPF pigs were necropsied 24 hours after being commingled, 6 hours after the beginning of clinical signs of disease. Results conclusively showed that virulent TGEV had been transmitted to neonatal piglets. A transmission trial is an effective tool to confirm a tentative diagnosis of virulent TGEV infection.

Keywords: swine, transmissible gastroenteritis, endemic TGE

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The cause of diarrhea in nursing pigs can be difficult to determine, because the clinical presentation of endemic transmissible gastroenteritis (TGE), rotavirus, coccidia, and colibacillosis infections are similar.[1] Infection with any of these four enteric pathogens can result in diarrhea in nursing piglets and subsequent low weaning weights. Typically, endemic TGE presents as a mild diarrhea in pigs ≥ 6 days of age. The consistency of the feces can range from clear and watery to white and creamy, and in late-stage disease become gray and pasty.[1] Low mortality and concurrent infection are characteristic, although some litters may experience severe clinical signs. Severity of clinical signs is dependent on the age of pig, degree of exposure to the virus, and degree of passive maternal antibody protection.[2]

In this case herd, endemic TGE was suspected as the cause of long-term diarrhea in nursing piglets based on observations of microscopic lesions of villus atrophy in the small intestine. However, this diagnosis was difficult to confirm, because pigs were not identified and autopsied early in the disease process. Routine serologic and antigen-detection tests used in the diagnosis of virulent TGE virus (TGEV) infection cannot differentiate virulent TGEV from the antigenically related porcine respiratory coronavirus (PRCV).[3] The diagnosis was made even more difficult because an oral modified-live virus (MLV) TGEV vaccine was being administered to the affected piglets.

This case report describes a transmission trial that was conducted as an alternate strategy to confirm TGE enteric disease. By deliberately infecting pigs so that challenge times could be known, jejunum could be collected from piglets in the early stages of diarrhea for TGE fluorescent antibody (FA) tests.

Case herd history

In November 1994, the producer expanded a 150-sow farrow-to-finish operation into a 700-sow farrow-to-wean operation, shipping early-weaned pigs offsite at 14–17 days of age. New gilts came from a breeding stock herd that was positive for porcine reproductive and respiratory syndrome virus (PRRSV) and *Mycoplasma hyopneumoniae*, but free of other major swine pathogens. Incoming gilts were vaccinated twice for TGEV, rotavirus, erysipelas, *Escherichia coli*, PRRSV, parvovirus, *Leptospira* spp., *M. hyopneumoniae,* and *Pasteurella multocida* type D.

In March 1995, the new gilts began farrowing. Approximately 30% of their litters had problems with preweaning diarrhea—an increase of 6% for this herd. The diarrhea began when pigs were approximately 5 days old and spread within the litter. As time progressed, younger piglets were affected. The diarrhea was described as ranging from pasty to creamy to watery. Preweaning mortality remained in the range of 10%–13%, but weaning weights were too low to allow pigs to move to segregated early weaning (SEW) facilities, which had a minimum acceptable weight of 3.5 kg (7.7 lb) for pigs received from farrowing

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Table 1

Summary of laboratory results taken from 14 live pigs submitted from the case herds

Date	Bacteriology ETEC	TGEV FA	TGEV ELISA	TGEV IHC	Rotavirus FA	Rotavirus RLA	Rotavirus IHC	TGEV and Rotavirus isolation	EM	*Coccidia*
Apr 3	1/2	0/2	1/2	0/2	0/2	0/2	ND	0/2	ND	0/2
May 25	0/2	0/2	2/2	ND	0/2	ND	ND	0/2	ND	0/2
Aug 31	2/2	0/2	0/2	0/2	0/2	0/2	0/2	ND	0/2	0/2
Sep 19	2/2	0/2	1/2	ND	0/2	0/2	0/2	ND	ND	1/2
Sep 25	1/3	1/3	2/3	2/3	0/3	0/3	0/3	ND	ND	0/3
Oct 2	2/3	0/3	2/3	1/3	ND	0/3	0/3	ND	ND	0/3

ETEC Enterotoxigenic *E.coli*
FA Fluorescent antibody test
ELISA Coronavirus antigen detection ELISA (Dr. Claude Montpetit, Quebec H7N 4Z3 CANADA)
IHC Immunohistochemistry (TGEV-Dr. Debbie Haines, University of Saskatchewan, Saskatoon, Saskatchewan, CANADA)
RLA Type A rotavirus latex agglutination test (Microgen Bioproducts Ltd., United Kingdom)
EM Direct electron microscopy
ND Not done

units. Of the 14- to 17-day-old pigs, 25%–30% were under this minimum weight. The diarrhea resolved in pigs that were moved to the SEW nursery and their postweaning weight gain was good. The diarrhea was not transmitted to other pigs at the SEW unit.

Between April and August, 14 live pigs (Table 1) and 40 fecal samples, from piglets, (Table 2) were submitted to the diagnostic laboratory for necropsy and diagnostic testing in attempt to identify the cause of the neonatal diarrhea. Single sera were collected from pigs; serum was taken from gilts, sows, and boars. In December, milk and colostrum was taken from sows as part of the follow-up to determine the

Table 2

Number of fecal samples taken from pigs tested positive for various pathogens

Pathogen	April–August 1995	October 1995
TGEV	8/14	2/20
rotavirus	0/20	0/20
Coccidia	0/20	6/20
Giardia	0/20	0/20
Cryptosporidia	0/20	0/20

Table 3

Serum and milk antibody titers for porcine coronaviruses/transmissible gastroenteritis (TGEV) and porcine respiratory coronavirus (PRCV) from the case herd

Date	Age group from which sample was collected	PRCV ELISA (# positive/ # tested)	TGEV ELISA (# positive/ # tested)	TGEV antibody using virus neutralization	Range in TGEV reciprocal of antibody titers using virus neutralization
Aug 1995	3-10 wk old serum	5/18	0/18	Not done (ND)	ND
	Boar serum	0/3	0/3	ND	ND
	Gilt serum	0/7	0/7	ND	ND
Sep 1995	Nursery pigs serum	ND	ND	14/18	2-1024
	Gilt serum	ND	ND	0/12	-
	Sow, boar serum	ND	ND	6/21	2-32
Oct 1995	Nursery pigs serum	ND	ND	3/3	192-1024
	Boar serum	ND	ND	1/2	3
	Paired sera from low weight pigs	ND	ND	8/16	2-48
Dec 1995	Gilt (open) serum	ND	ND	5/5	3-48
	Sow serum	ND	ND	29/41	3-4096
	Sow milk (0-2 days postfarrowing)	ND	ND	2/4	256-384
	Sow milk (3-14 days postfarrowing)	ND	ND	2/6	256-768
Jan 1996	Sow milk (0-2 days postfarrowing)	ND	ND	9/9	64-1536
	Sow milk (3-17 days postfarrowing)	ND	ND	12/13	12-384

effectiveness of the vaccination program. All serum and milk samples were tested for antibodies to porcine respiratory coronaviruses (TGEV/PRCV) and to porcine epidemic diarrhea virus (Table 3).

These preliminary diagnostic tests suggested that the etiology of the diarrhea might be TGEV. The producer began a feedback program in May. Although the sanitation in the farrowing barn was excellent, all farrowing crates were in one room and so only a section of the room could be disinfected each week. The crates had slatted floors, walls made of puck board, and heated floor mats for the creep area.

Serological test results from pigs and breeding animals were confusing. In August, five of 18 nursing piglets were found to be positive for antibody to PRCV using the Quebec TGEV/PRCV- differentiating antibody ELISA (Dr. Y. Elazarhy, University of Montreal, St. Hyacinthe, Quebec). However, seven gilts and three boars were negative for antibody to coronaviruses using the ELISA assay (Table 3). The producer vaccinated the sows with an autogenous *E. coli* vaccine, and improved ventilation in the farrowing room.

Diagnostic tests continued, and in September *Isospora suis* was isolated from one affected piglet. Given the possibility that the herd had coccidiosis, the feedback program—which would have further distributed this agent within the herd and worsened the diarrhea — was discontinued. An oral TGEV vaccine was instead given to 1- and 4-day-old pigs. Fourteen of 18 nursery piglets and six of 21 sows and boars tested were found to be positive for coronavirus antibody using the virus neutralization (VN) assay, while none of 12 gilts had coronavirus antibody using single sera.

In October, feces from 20 additional piglets were tested (Table 2). Feces from six of these pigs were positive for *Isospora suis* and feces from two were positive for coronavirus antigen (Table 2). Feces from 20 sows were negative for parasites using routine fecal floatation. Paired sera for 16 lightweight piglets were tested by VN. Eight piglets found to be negative for coronavirus antibody in both sera samples, seven piglets had low antibody titers in both sera, and only one piglet had seroconverted to coronavirus (<1:2 to 1:48). The attending veterinarian presumed these titers were the result of the use of the MLV TGEV vaccine. Sera from the six piglets tested were found to be negative for antibodies to porcine epidemic diarrhea virus.

Because the diarrhea did not spread to naive pigs in the SEW nursery, and because both the one piglet with the TGEV-positive FA test (jejunum) and the two piglets with the TGE-positive IHC test (jejunum) had been given oral MLV TGE vaccine the day before testing, it was not possible to determine whether TGEV was the primary pathogen. It was at this point that the transmission trial, described below, was conducted. A diagnosis of TGEV was confirmed within 3 days of the start of the trial. Pigs were followed for a full 21 days only to understand the PRCV/TGEV differentiating ELISA test from Quebec.

Post-trial case history

The clinical picture changed dramatically in the case herd during the last week of October. The air inlets in the farrowing barn were inadvertently left open 2 nights in a row, chilling the piglets and sows. Within 3 days, the sows had projectile diarrhea and decreased feed intake. Piglets had watery, projectile diarrhea and some vomited. As the outbreak continued, the age of onset of illness shifted from 7–10 days to < 5 days. Approximately 80% of the litters were affected in this outbreak. The preweaning mortality jumped from 11% to 66%. At this time, the feedback program was reinstituted and cross-fostering was stopped. The oral TGEV vaccine continued to be given to 1- and 4-day old-pigs throughout the 6 weeks of the outbreak.

Serum, colostrum, and milk samples from sows show that TGEV titers increased from September to January (Table 3). In January, the producer discontinued the feedback program and the sow TGE vaccinations. However, although the prevalence of positive serum and milk titers increased, the titers were not uniform. The diarrhea at the farm was resolved by March. After that, weekly preweaning mortality ranged from 8%–14%.

Transmission trial

To verify whether or not endemic TGEV was responsible for the diarrhea in the preweaned piglets and subsequent poor growth rate, we conducted a transmission trial in the third week of October. We conducted the transmission trial at the university facility to provide the convenience of regular access to the pigs, the ability to observe the pigs daily, and to ensure that pigs would be transported to the laboratory for necropsy within 6 hours of showing clinical diarrhea.

Methods

Experimental design

Eight unvaccinated pigs from the case farm, ranging in age from 5–9 days, were selected as carriers of infectious agents. They came from four litters: one pig from each litter had clinical diarrhea, and the other was clinically normal. We divided the pigs into two groups of four (each remaining with its littermate) and placed them in clean, disinfected isolation rooms at the University of Guelph. The rooms had been empty for several months. The rooms were heated to 23.5°C (75°F), with heat lamps over the pen and a solid mat under the lamp. Milk replacer was provided by a MOM™ (Shurgain Division, Canada Packers Inc., Ontario, Canada) artificial rearing unit.

On day 0, four 3-day-old colostrum-fed, specific-pathogen-free (SPF) sentinel pigs were added to each group of case herd pigs. The SPF unit was known to be free of antibody to porcine coronaviruses in virus neutralization assays (VN) using sera. Two other pigs from the SPF unit were placed in a third isolation room to act as unexposed control pigs. The TGEV-seronegative sentinel pigs allowed us to expose fully susceptible pigs that were free from the complicating effects of existing TGEV or PRCV antibodies. Also, these pigs were euthanized at defined times after the onset of diarrhea so that more reliable acute samples could be collected for the diagnostic tests.

Before the final postmortem results were available, some of the sentinel pigs had died. There was concern that the cause was enterotoxigenic *E. coli* because these pigs were the smallest, weakest pigs from the donor SPF litter. Therefore, we added five more healthy, 9-

day-old crossbred SPF sentinel pigs to the trial on day 2, two to three to a pen for a total of 13 sentinel pigs. On arrival, we injected the five added pigs with gentamicin IM at 2 mg per kilogram bodyweight.

People traffic between the two nonexposed pigs and the exposed pigs was at first carefully restricted. We fed the nonexposed pigs, and then changed coveralls and boots between the isolation rooms. After 1 week, however, we became less strict with regard to changing coveralls.

The pigs were observed for 0.75–1 hour and given fresh milk at 8:00, 14:00, and 21:00 by the first author and were observed for 10 minutes at 2:00 by a university technician. The pens were cleaned twice a day. At each visit, all pigs were roused and observed.

Postmortem examinations

Ten pigs (three from the case herd and seven from the SPF unit) were euthanized and presented for postmortem at 48 hours post exposure. Postmortem evaluations were conducted on 15 pigs (four from the case herd, eight from the initial group of SPF pigs, and three from the second group of SPF pigs). Pigs that died suddenly, the 'unexposed' pigs, and the pigs that lived for 21 days post exposure were not examined. Bacterial and viral diagnostic tests were conducted on eight of the 15 pigs that were examined at postmortem.

Serology

Blood samples were taken from 18 pigs on arrival at the university; five from the case herd and 13 from the SPF herd. Convalescent samples were taken from the two "unexposed" and the six pigs that lived for 21 days post exposure. All samples were analyzed for antibodies to TGEV and PRCV using the Quebec differential ELISA (University of Montreal).

Results

Clinical signs

Within 18 hours, all eight exposed pigs from the SPF unit were vomiting and had watery, projectile diarrhea. At 24 hours post exposure, one pig from the case herd and four exposed pigs were dehydrated and were euthanized. Gross examination showed there was feed in the stomachs and the intestinal contents were yellow and watery, and contained flocculent material. The tentative diagnosis was atrophic enteritis.

One week into the trial, shortly after we stopped changing coveralls, the two unexposed control pigs came down with diarrhea and were euthanized. Gross postmortem examinations of the nonexposed pigs were similar to the exposed pigs and revealed yellow fluid in the intestine.

Of the five additional SPF pigs added to the trial on day 2, two of these new pigs were vomiting with in 18 hours. After 24 hours they had watery, projectile diarrhea. By 48 hours after introduction, all five pigs in the second group from the SPF herd were vomiting and all of the pigs in the trial had diarrhea.

Postmortem examinations

At necropsy, the stomachs of piglets were filled with coagulated milk and with watery yellow and sometimes flocculent contents in the thin-walled small intestine. One pig had foamy yellow small intestinal contents and gastric mucosal hemorrhages. Another pig had bronchopneumonia with fibrinous pleuritis. Histological examinations of the intestines of the first five pigs (euthanized within 24 hours of exposure) showed marked villus atrophy and villus fusion in the small intestine. The crypt to villus ratio was 1:1. No coccidial organisms were seen.

Isolates from three of the four SPF pigs were positive for enterotoxigenic *E. coli* (Table 4). The coronavirus antigen-detection ELISA test was negative for the case herd pig, but positive for all four pigs from the SPF herd. Fluorescent antibody (FA) tests for TGE were performed on three sections of jejunum for each pig. All five pigs had positive FA tests for TGE. Tests for rotavirus and cultures for *Salmonella* were all negative.

Histological examinations of the second group of 10 pigs euthanized 48 hours post exposure showed atrophy and fusion of villi, gastritis, and mild colitis. All but two of the second group of SPF pigs were

Table 4

Diagnosis of infectious agents causing diarrhea in transmission trial pigs

Infectious agent	*E.Coli* (#positive/#tested)	Coronavirus ELISA (#positive/#tested)	TGE FA (#positive/#tested)
24 hours post exposure			
Case herd	1/1	0/1	1/1
SPF herd	3/4	4/4	4/4
48 hours post exposure			
Case herd	Not done (ND)	ND	3/3
SPF herd	1/3	ND	5/7

Table 5

Serological test results from acute and convalescent sera taken from transmission trial pigs

Source	Acute TGE (#positive/#tested)	Convalescent TGE (#positive/#tested)	Acute PRCV (#positive/#tested)	Convalescent PRCV (#positive/#tested)
Case herd	5/5	2/2	0/5	0/2
Exposed	0/11	4/4	0/11	0/4
"Unexposed"	0/2	0/2	0/2	2/2

demonstrated to be positive for TGE using FA tests. All diagnostic tests were negative for the other previously named enteric pathogens.

Serology

The acute samples from the SPF pigs tested negative for antibodies to both coronaviruses, TGEV, and PRCV (Table 5). The acute samples from the case herd pigs were positive for TGEV antibodies. The convalescent samples from the pigs that lived for 21 days post exposure were positive for antibody to TGE. The two "unexposed" pigs that became infected by fomite contamination tested positive for antibody to PRCV.

Discussion

We suggest that future transmission trials be conducted at the farm site. Pigs that are not receiving milk antibodies should be used for the trial because they are more likely to show classical signs of TGE. The attending veterinarian could early wean healthy 3-day-old piglets, preferably from a gilt, placing piglets on milk replacer in a farrowing crate with affected piglets in the affected farrowing room. Alternatively, 3-day-old pigs from a SPF unit could be purchased and put in an empty farrowing crate with affected piglets. These new pigs can then be monitored closely for the first signs of vomiting and diarrhea and submitted alive for postmortem within 6 hours of the onset of clinical diarrhea.

Although this transmission trial was performed on a different site under different housing and management, we believe that it was beneficial in arriving at a primary diagnosis of endemic TGE infection as the cause of the preweaning diarrhea. The presence of enterotoxigenic *E. coli* and coccidia in some animals probably contributed to the diarrhea problem in individual piglets with subsequent slow weight gain. Transmission trials on or off the farm can provide an important addition to a diagnostic workup, where animals in the earliest stages of disease can more readily be identified to provide timely and high quality specimens for diagnostic testing.

Case discussion

This case is remarkable for the difficulty we had in confirming that TGEV was responsible for the preweaning diarrhea and decreased growth rate in piglets. The presence of villus atrophy in pigs of this age, with a clinical history of diarrhea, was highly suggestive of endemic TGEV infection. However, because TGEV vaccine had been used in the herd, test results over several months couldn't be considered conclusive. The feces of some pigs tested positive for TGEV by ELISA, but this test cannot differentiate between field and vaccine viruses. Similarly, the one positive fluorescent antibody test for TGE and the two positive IHC tests for TGE were from piglets that had been given oral MLV TGEV vaccine for 3 days before, making these results inconclusive.

Given that this TGE outbreak occurred post vaccination, the efficacy of the vaccine protocol can be questioned. Previous studies indicate that commercially available TGEV vaccines provide incomplete immunity. Piglets develop an immune response within 3–5 days post innoculation but will not respond to the vaccine if they have passive immunity.[4] The vaccination of sows results in decreased mortality but not decreased morbidity in their offspring.[4]

The pigs submitted directly to the laboratory by the producer were those that were too small to go into the SEW unit. These pigs were likely chronically affected, and would no longer have had virus-infected cells present in the intestine. Laboratory diagnoses for TGEV infection are based primarily on FA or IHC evaluation of sections of jejunum or ileum. For these tests to be useful, pieces of intestine must be frozen or fixed in formalin within minutes of euthanizing a piglet in the very early stages of disease, within 24 hours of infection.[1] After this time, the virus will have lysed infected villus epithelial cells, and viral antigen will have been lost into the lumen of the gut, unable to be detected by FA or IHC tests. Thus, the window of opportunity to demonstrate viral antigen in infected cells in the small intestine of TGE infected pigs is very narrow: less than 24 hours after the onset of diarrhea. Tests for TGEV have best diagnostic sensitivity if live pigs are submitted for postmortem within 6 hours of when they first show clinical diarrhea, to ensure that large numbers of virus–infected cells are still present in the intestinal epithelium.[3]

Routine virus neutralizing serological tests to measure coronavirus antibodies are often used to confirm a diagnosis of TGE,[3] and seroconversion or significant fourfold changes in antibody in paired sera are a useful way of demonstrating endemic TGE in large herds.[1] It is beneficial to collect paired serum samples from pigs at weaning and again 4 weeks later in an attempt to confirm diagnosis.[1] Exposed sows have rising VN titers in serum and replacement gilts may show seroconversion. Often gilts will seroconvert after farrowing or during recrudescence of the disease within the herd.[1]

The difficulty of differentiating TGEV/PRCV antibody using virus neutralization and in the early stages of infection is a significant limitation of the TGEV/PRCV-differentiating ELISA.[4–6] As was seen in this case, acutely affected pigs were determined to be positive for antibody to PRCV using the differentiating ELISA. Pigs in the later stages of infection were determined to have antibody to TGEV using the same assay. It has been reported that this TGEV/PRCV-differential ELISA cannot accurately determine the antibody status of TGEV-infected pigs prior to 28 days post-exposure.[6] The ELISA serology test should be used as a herd-based test, rather than for evaluation of TGE status of individual pigs.

If piglets are euthanized within 24 hours of becoming ill and tissues are frozen or fixed rapidly, there will be many TGEV-infected epithelial cells readily observed by either FA or IHC. However endemic TGE is much more difficult to diagnose at necropsy, because piglets suckling milk with TGE antibody from partially immune herds experience a milder clinical syndrome than is found in epidemic herds.[1,4,7] Among endemic TGE cases, only 25% of pigs display the typical histologic lesions of TGE.[1] It is more difficult to demonstrate TGEV-infected cells with FA or IHC tests in herds with endemic TGE than when an epidemic outbreak occurs, because fewer virus-infected enterocytes are present in the intestine due to the partial protection afforded by milk antibody.[1] With milder clinical signs it is more difficult to identify affected pigs at a time when antigen detection tests are useful. Often the postmortem findings are variable and more suggestive of enterotoxigenic *E. coli* than TGE.[1] In the study by Pritchard,[1] 33% of the pigs had a concurrent enterotoxigenic *E. coli* infection that also complicated diagnosis.

These concurrent infections with multiple organisms make the diagnosis particularly difficult.

The pH of feces will be acidic in cases of diarrhea such as TGE, which is due to malabsorption. However, with secretory diarrhea caused by *E. coli*, the pH of the feces is alkaline. This test was not conducted in the case herd.

Case herd dynamics

Endemic TGE is a common sequelae of an epidemic of TGE, especially in large herds.[1] In these herds, a significant number of susceptible pigs are able to escape infection during the primary TGE outbreak and are the nidus for maintaining the infection in the herd.[4] Furthermore, the source and number of purchased pigs brought into an endemic TGE herd are important for facilitating the persistence of the disease.[5]

A continuous farrowing system also provides a constant supply of susceptible piglets, therefore ensuring that the virus remains active in the herd. Clinical signs occur in piglets suckling seronegative dams, sows with a low degree of immunity, and those with a poor milk supply.[4]

The case herd had a history of a TGE epidemic 5 years prior to the problem described in this case study. The expansion of the herd, with new gilts added to the old sow population, established both the source for TGE and naive animals in which the virus could persist.

It is unusual for endemic TGE to persist in a herd after a feedback program has been initiated. It is possible that the feedback program was not implemented effectively, for example:

- The producer harvested intestines after the pigs had died naturally, so there would not have been optimal amounts of infectious virus in the material.
- The harvested material was kept at room temperature, and was not used immediately.
- Sows were group-housed, making it difficult to dose individual animals. Less aggressive sows may not have received the feedback material.
- Perhaps some sows were less susceptible to the feedback material because of previous immunity (due to vaccination or natural exposure in the farrowing barn). If this was so, then fewer sows would be getting sick and shedding virus. As a result, the spread of the natural infection in the gestation barn would have decreased.

Implications

Feedback

The goal of a TGE feedback program is to ensure that all sows are exposed to TGEV. In this way, the sows produce protective antibodies in milk and decrease the spread of disease in a farrowing barn. In an effective feedback program, you should:[4]

- purchase 6 months of replacement gilts and then close the herd;
- use strict all-in–all-out management in the farrowing room. This should stop the transmission of the virus from affected piglets to susceptible piglets;
- euthanize the piglets within 6 hours of the first clinical signs to ensure high doses of virus in the material used for feedback. Harvest the intestine and feed the material to the sows immediately;
- feed virus immediately. If it is not convenient to feed right away, refrigerate it as the virus is susceptible to heat. If virus will not be used the same day, freeze it in a non-self defrosting freezer;
- dose each sow individually with the live virus meal for best results. Material from two pigs can be mixed in a 5-gallon (20 L)bucket. Each sow should receive 1 to 2 cups (0.5 L) of this material. Mark each sow as dose is received; and
- separate groups of litters in the farrowing room by empty crates to decrease spread between litters.

References

1. Pritchard GC. Transmissible gastroenteritis in endemically infected breeding herds of pigs in East Anglia, 1981-1985. *Vet Rec*. 1987;120:226–230.

2. Bohl EH, Gupta RKP, McCloskey LW, Saif L. Immunology of transmissible gastroenteritis. *JAVMA*. 1972:543–549.

3. Garwes DJ, Stewart F, Cartwright SF, Brown I. Differentiation of porcine coronavirus from transmissible gastroenteritis virus. *Vet Rec*. 1988; 86–87.

4. Reeves D, Baker RB, Hill H, Kohler EM, Templeton C, Wiseman B. Roundtable : Transmissible gastroenteritis, *Agri-Practice*. Part 1: 1992;13,7:30–33. Part 2: 1992;13,8:23–29. Part 3: 1992;13,9:23–28.

5. Yanga ST, Garnder IA, Hurd, HS, Eernisse KA, Willeberg P. Management and demographic factors associated with sero positivity to transmissible gastroenteritis virus in US swine herds, 1989-1990. *Prev Vet Med.* 1995;24:213–228.

6. Collins JE, Lang T. Interpreting PRCV/TGE Serological Test Results. *Proceedings of the 29th Annual Meeting of the American Association of Swine Practitioners*. 1998;363–364.

7. Halbur P, Paul P, Frey M, Morozov I, Sirinarumitr T. TGE-like disease outbreak: A foreign disease or a variant? *Proc A D Leman Swine Conf*. 1995;1–2.