CASE REPORT

Evaluation of a transmissible gastroenteritis virus eradication program in a breeding stock supply herd

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Summary

This case report describes an outbreak of transmissible gastroenteritis virus (TGEV) in a large breeding-stock-producing herd that developed an endemic infection. The main clinical and diagnostic features of endemic TGEV are discussed, and an effective protocol to eradicate TGEV is described.

Keywords: *swine, transmissible gastroenteritis virus (TGEV), eradication protocol*

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Endemic transmissible gastroenteritis (TGE) is defined as the persistence of the TGE virus (TGEV) and disease in a herd. Endemic infection is a common sequel to a primary outbreak in herds of more than 300 sows.[1] In these herds, the main clinical findings are diarrhea, primarily in suckling piglets (from 6 days old to about 2 weeks after weaning); diarrhea among recently weaned pigs; and discrete episodes of overt clinical recrudescence in part of the herd. The endemic disease may be mild or inapparent for much of the time. In some herds, the predominant clinical finding is a mild diarrhea in suckling piglets from about 6 days old. In other herds, the disease 'break' occurs primarily during the postweaning period.[2] Pigs in some herds have diarrhea both before and after weaning. Adult pigs usually are not sick. However, occasionally there is diarrhea in gilts or sows housed in the same farrowing room as piglets with diarrhea during periods of recrudescence. During a typical recrudescence, piglet mortality is usually 10% of that during the initial outbreak. The presence of finishing pigs results in a significantly greater incidence of disease relapse in larger herds.[1] The timing of the periods of disease recurrence almost invariably occurs within about 9 months of the end of the primary outbreak, and most within about 4 months. Some herds have several episodes of disease relapses, usually at intervals of 3 or 4 months, while others experience only a single recurrence.

Case history

The herd in this case was a 600-sow farrow-to-partial finish operation located on one farm with additional offsite contract finishing. In addition, a breeding stock holding barn used to house breeding stock for outside sale is located approximately 0.2 km (1/8 of a mile) from the home farm. The facility design consisted of six farrowing rooms with a total of 80 crates. (Figure 1) Three of the farrowing rooms were openly connected. There was a two-stage nursery system. The hot nurseries consisted of seven single-deck rooms opening onto a common hallway and were accessible only by walking through the second-stage starter barn. The starter barn was a continuous-flow barn holding approximately 2.5–3 weeks of production. There was one grower barn and four finishing barns, which were run on a continuous-flow basis. The farrowing rooms and hot nurseries were washed and disinfected between groups of animals. However, there was animal movement between rooms. Pigs were cross-fostered between litters in different farrowing rooms. In addition, the smaller, poorer pigs were collected from two or three hot nurseries and grouped together in a sick pen in one nursery room. The pens in the continuous-flow barns were washed and disinfected between groups. There were two breeding locations; a gilt breeding room and four rooms of pen gestation, plus a separate sow breeding area and a gestation room with stalls and pens. This farm had not instituted a facility-wide washing regime and mice were visibly active throughout the barn.

This herd broke with TGEV on February 10, 1995 (Figure 2). At the time of this outbreak, all sows and boars were exposed to TGEV via a feedback program. After the outbreak on March 10, all sows and boars were vaccinated with an oral TGEV vaccine (Ambico ProSystem I® Ambico, Inc., Dallas Center, Iowa). Since the initial outbreak, herd policy has dictated that sows be vaccinated 2 weeks prefarrowing with an intramuscular TGEV vaccine (Ambico ProSystem I®). The majority of the commercial feeder pigs were finished in contract barns offsite. At the time of the initial outbreak, one contract barn had just been filled. No more pigs were put into that barn from the main herd until May 1995. Shortly after this initial TGEV outbreak, the breeding stock customers were informed and sales of stock ceased.

The group of animals housed in the breeding stock holding barn were vaccinated on February 16 and again on March 10 with the oral TGEV vaccine and, 2 weeks later, the intramuscular TGEV vaccine. All sales of breeding stock ceased at the time of the initial outbreak. These animals were moved back to the home farm sometime after March 24, 1995.

After a period of 3 months, feeder pig movement into the contract finishing facility resumed. In addition, young breeding stock that had been transferred offsite prior to the TGE outbreak were brought back to the home farm. These animals had not been previously vaccinated for TGEV.

No diarrhea was observed in suckling piglets after the cessation of

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acute clinical signs of TGE at the time of the initial outbreak (February 1995). There was some diarrhea in weaner pigs. Pigs from this group were submitted live to the Veterinary Diagnostic Laboratory, Guelph, Ontario. However, no specific diagnosis was made. Clinical signs of mild diarrhea were continuously observed in the finishing barn.

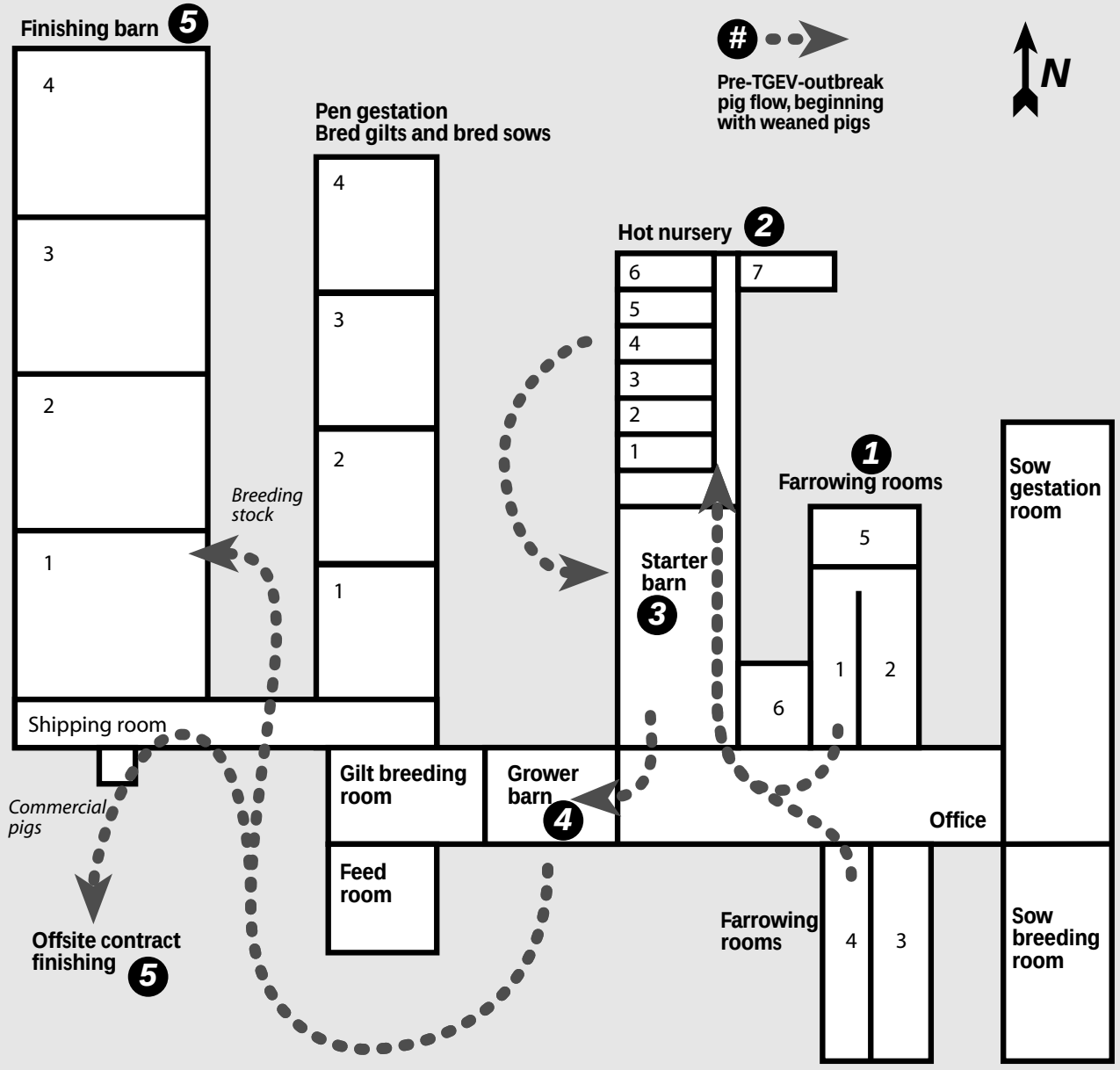
In June 1995, the herd was declared free of active TGEV based on the following evidence:

- Ten seropositive grower pigs from the farm and 10 negative pigs were mixed in adjacent pens in an offsite finishing barn. Manure was spread from pen to pen. The 10 sentinel pigs tested negative 30 days after exposure to the 10 pigs from the TGE source herd;
- A boar introduced into the affected herd remained seronegative 30 days after entry; and
- A group of 50 pigs, some from the TGE herd and others from a negative herd were housed together for 40 days. Ten pigs from the TGEVsource herd were then bled and these tested seropositive. The 10 pigs from the negative herd did not seroconvert to TGEV.

The producer was anxious to begin selling breeding gilts and boars again, and based on the above test results, was told it was safe to do so.

The owner of one of the herds purchasing the breeding stock expressed concern that there had been insufficient testing and that not enough time had elapsed to ascertain that the TGEV was no longer

Figure 1



Site plan showing pig flow prior to TGE outbreak.

active in the herd. The veterinarian, acting on behalf of this commercial herd, contacted an independent veterinarian to provide a second opinion.

Clinical and laboratory findings

A referral herd visit was conducted on September 5, 1995. At that time, the weaner pigs in the hot nurseries had diarrhea and long hair coats, and some of the pigs showed signs of wasting. The diarrhea had first been observed in these pigs 2 weeks previously. Pigs started to experience diarrhea approximately 7–10 days postweaning. Pigs in the starter barn also had diarrhea and a number were gaunt and razor-backed. The recently weaned pigs still looked good. Pigs in the grower-finisher barn did not have signs of diarrhea and appeared to be doing well.

At the time of this herd consultation in September 1995, five of the

Figure 2

Date	Herd event	Clinical and laboratory findings	Treatment applied
February 10, 1995	A 600 sow farrow-to-partial finish operation broke with TGE. All sows and boars were exposed to TGEV via a feedback program		
February 16, 1995			All replacement stock housed in the holding barn were vaccinated with an oral TGEV vaccine
March 10, 1995			All sows and boars were vaccinated with an oral TGEV vaccine. All replacement stock in the holding barn received a second oral TGEV vaccine.
March 24, 1995			All replacement stock in the holding barn received an intramuscular TGEV vaccine
After March 24, 1995	All replacement stock from the holding barn were moved back to the home farm		
May 1995	Resumption of feeder pig movement into a contract finishing facility. Nonvaccinated purebred replacement stock transferred back to the home farm		
June 1995		The herd was declared free of active TGEV based upon the serological tests results from principals (seropositive) and sentinels (remained seronegative) bled 30 to 40 days after mixing	
September 5, 1995	A referral herd visit was conducted by an independent veterinarian. Weaner pigs in the hot nurseries and starter barn had diarrhea, had long hair coats and some of the pigs showed signs of wasting.	Five weaner pigs (5 to 6.5 weeks old) were submitted live to the veterinary diagnostic laboratory. Thirty acute serum samples were taken from pigs in the hot nurseries and starter barn were submitted to the laboratory. These samples were run before the convalescent samples were submitted.	
September 22, 1995		Convalescent blood samples were drawn. Fourteen serum samples were submitted to St. Hyacinthe, Quebec for TGEV/PRCV differentiation and tested positive for TGEV antibodies. The dignosis of the live pigs submitted was chronic, inactive atrophic enteritis with lesions likely to have been virally induced.	
September 29, 1995	150 gilts of various sizes and two boars were purchased from the home farm and placed in a quarantine barn. The objective was to establish breeding stock that were not at risk of shedding TGEV. These animals were intended as replacements for an affiliated commercial herd. Fifty weaner pigs from the affiliated farm were added to the quarantine barn		
October 5, 1995	A protocol for TGE eradication using a partial herd depopulation was reviewed with the owner. The partial depopulation would be delayed until March 1996 but the protocol outlined for the home farm was to be initiated.	Blood was drawn from 29 gilts in the quarantine barn.	
October 15, 1995	The breeding of the gilts commenced in the isolation barn.		
October 17, 1995			All the pigs in the quarantine barn were vaccinated with an oral TGE vaccine.

Timeline of TGE outbreak (continued on next page)

weaner pigs between 5 and 6.5 weeks of age were submitted live to the Veterinary Diagnostic Laboratory. In addition, 30 acute serum samples taken from pigs in the hot nurseries and starter barn were submitted.

Convalescent blood samples were drawn 17 days later on September 22. At this time there were no pigs with diarrhea in either the hot nurseries or starter barn. The overall appearance of the pigs in these areas had dramatically improved.

The diagnosis on the five live pigs submitted to the diagnostic laboratory was chronic, inactive atrophic enteritis with the lesions likely virally induced. Unfortunately, the acute serum samples were run before the convalescent samples were received. However, the pathologist commented that the single titers to TGEV were not consistent with passive postvaccinal titers and were more consistent with ongoing field infections. The TGEV antibodies were detected using the serum neutralization (SN) assay. Fifteen of the titers were > 1024 and two pigs had titers of 4096 (Figure 3). The titers of piglets that have received high levels of colostral antibodies to TGEV from the sow tend to approach 0 at 12 weeks of age. Therefore, these extremely high titers to TGEV, especially in this age of pigs, are more consistent with an ongoing field infection. Fourteen serum samples were sent to St. Hyacinthe, Quebec in order to differentiate whether the antibodies were to TGEV or PRCV. The test was positive for TGEV antibodies.

Endemic TGEV was diagnosed and the owner was advised to discontinue selling breeding stock from his barns. A protocol for TGEV eradication using a partial herd depopulation was devised and reviewed with the owner on October 5, 1995. This protocol called for moving all pigs weighing > 13.6 kg (30 lb) off the farm to contract finishing locations. The protocol for the home farm herd involved:

- Closing the herd: i.e., bringing in all breeding herd replacements necessary for 6 months at staggered weights ranging from 22.67–95.26 kg (50–210 lb).
- Enhancing herd immunity through intensive vaccination of the breeding herd (sows, boars, and replacement stock) and newborn piglets. Sows and gilts were to be vaccinated with two oral doses of Ambico Prosystem I® vaccine (one at 5 weeks and again at 3 weeks before farrowing). All boars and replacement stock were to be vaccinated with two oral doses of Ambico Prosystem I® at a 2-week interval. Piglets were to be orally vaccinated using ⅕ the Ambico

Figure 2

Date	Herd event	Clinical and laboratory findings	Treatment applied
November, 1995		25 pigs were blood tested in the quarantine barn for TGEV. All pigs had seroconverted.	
November 22, 1995	A herd visit to the home farm was conducted. Two recently introduced gilts had diarrhea.		
November 24, 1995		25 pigs were blood tested in the quarantine barn for TGEV. All pigs had seroconverted.	
Early December, 1995			Initiation of oral vaccination of the sows for TGEV prefarrowing and oral vaccination of the piglets on day 1 and day 5.
December 5, 1995		Thirty weaner pigs were blood tested for convalescent samples. Three pigs had a fourfold rise in the level of antibody between the acute and convalescent serum. These results supported the diagnosis of endemic TGE.	
Mid December, 1995	There was a dramatic increase in piglet diarrhea. Some piglets vomited and the feces had a strong, noxious odor. There was relatively little piglet mortality. Pigs in the hot nursery developed diarrhea 10 to 14 days postweaning and went off feed.		
Early January, 1996	The first gilts were moved from the isolation barn to the commercial unit.		
January 18, 1996	A herd visit was conducted. New litters with diarrhea were present. A moderate amount of diarrhea was ongoing throughout the hot nurseries and starter barn. A large number of poor-doing pigs were present in the starter barn.		All sows due to farrow had received two oral and one intramuscular vaccinations for TGEV prefarrowing.
January 23, 1996		Seven live pigs were submitted to the diagnostic laboratory. All pigs had atrophic enteritis. Two pigs had positive fluorescent antibody tests for TGE viral antigen and positive immunoperoxidase tests. A definitive diagnosis of TGE was made.	
Late January, 1996	A second gilt isolation barn was set up for the commercial farm		
February 12, 1996	No signs of TGE were seen on the commercial farm.		

Timeline of TGE outbreak (continued from previous page)

Prosystem I® sow dose at day 1 and day 5 postfarrowing.

- Creating a strict all-in–all-out (AIAO) movement of pigs, especially in the farrowing rooms and nurseries, with complete cleaning and disinfecting between groups. All cull pigs were to be euthanized to prevent a reservoir of pigs actively shedding TGEV. There was to be no movement of pigs between farrowing or weaner rooms.
- Establishing a rotational washing regimen was to be established in each area of production so that the whole barn was cleaned at least twice within a 5-month period.
- Establishing a stringent rodent control program.

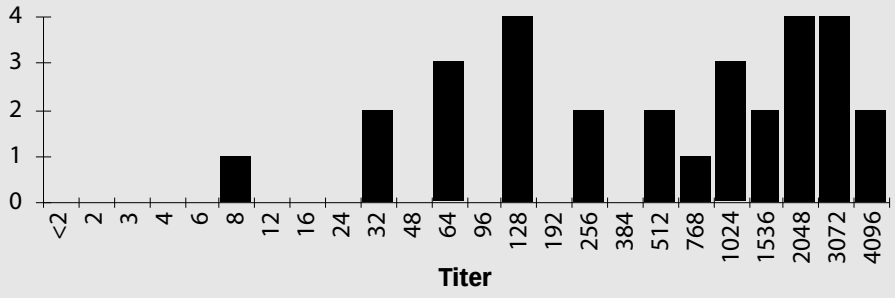
Due to the lack of available contract finishing barns, the partial depopulation was scheduled for March, 1996. In the meantime, the protocol outlined for the home farm was to be initiated. The owner was advised to discontinue selling breeding stock directly from his barns.

In order to supply breeding stock to an affiliated farm, a protocol was developed to establish breeding stock that were not at risk of shedding TGEV. A quarantine barn run on an AIAO basis was established. On September 29, 1995, 150 gilts of various sizes and two boars were purchased from the home farm. Fifty weaner pigs from the affiliated farm were added the first week of October. On October 5, 1995, blood was drawn from 29 of the purchased gilts. The gilts had titers ranging from <2 to 1536 for TGEV (Figure 4). The weaner pigs never showed any clinical signs of diarrhea. On October 17, 1995 all the pigs were vaccinated with an oral TGEV vaccine (Ambico Prosystem I®) following the manufacturer's recommendations. Two weeks later, 25 pigs were blood tested for TGEV. All the pigs appeared to have seroconverted. The original isolation procedures were maintained and breeding of the gilts commenced around October 15. No new animals were added to the herd, but market animals (the original weaners) and cull breeding stock were shipped to market. In early January 1996, the first gilts were moved to the sow unit of the affiliated farm. As of February 12, 1996, there have been no signs of TGEV at that farm. A second gilt isolation barn was set up in late January to repeat the procedure.

Despite the results of the laboratory tests in September 1995, the discussions regarding the TGE status of the farm and the protocol outlined for TGEV eradication, the owner was not convinced that TGE still existed in his herd. As a result, he requested further testing be conducted. Thirty weaner pigs between 2 and 5 weeks of age were blood tested on November 24, 1995 and December 15, 1995. The result of these tests showed that three pigs, which were 2 weeks old at the time of the first test, had a four fold rise in the level of antibody between the acute and convalescent serum (Table 1). These results supported the diagnosis of endemic TGE.

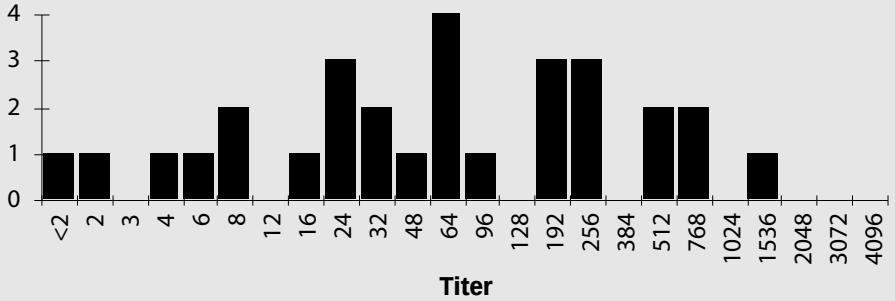
Unfortunately, because of the owner's belief that TGE was no longer a problem in the herd, he had not instituted any of the recommended procedures prior to the first week of December. The owner continued to sell breeding stock during this period despite being strongly advised to stop. His contention was that his customers were aware of the history of TGE in the herd and that there had been no problems to date on the farms that had purchased his breeding stock.

Figure 3



Distribution of TGEV titers in weaner pigs

Figure 4



Distribution of TGEV titers in purchased gilts

At a herd visit on November 22, 1995, two gilts, which had been brought into the herd 7 days previously, had diarrhea. These gilts had been raised in an offsite facility and had been returned to the sow herd as replacement breeding stock.

In approximately mid-December there was a dramatic increase in piglet diarrhea starting from the day of birth to 2 or 3 days postfarrowing. In many cases the piglets vomited, were unresponsive to antibiotic treatment, and the feces had a strong noxious odor. In addition, a parity-three sow had profuse diarrhea at the time of farrowing. Despite the number of litters affected, there was relatively little mortality. There did not appear to be a parity-related predisposition to diarrhea. The pigs in the hot nursery started to experience diarrhea approximately 10–14 days postweaning and went off feed. Oral vaccination of the sows for TGEV prefarrowing and oral vaccination of the piglets for TGEV on day 1 and day 5 started around the beginning of December, 1995. As of January 18, 1996, the sows due to farrow had received two oral and one intramuscular vaccinations for TGEV prefarrowing. A herd visit on January 18, 1996 was conducted and there were litters with diarrhea. A moderate level of diarrhea was ongoing throughout the hot nurseries and the starter barn. There were a large number of poor-doing, small pigs in the starter barn. Affected pigs were navel sucking, unthrifty, razor backed, and many had diarrhea. The overall average weight of the pigs had decreased throughout the farrowing and nursery rooms, including the grower barn. The owner continued to move animals between farrowing units as well as between weaner rooms. Cull pigs were present in the 'sick bay' in the starter barn and in the small room outside the gilt breeding barn. Seven live pigs were submitted on January 23, 1996 to the Veterinary Diagnostic Laboratory. On gross necropsy, three of the four older pigs (4–6 weeks of age) had an effusive diarrhea. These pigs uniformly had atrophic enteritis and the lesions appeared to be post-infectious, with the exception of attenuation of enterocytes covering villi in two of the pigs. These two pigs had positive fluorescent antibody tests for TGEV antigen. One of these pigs had been orally vaccinated at day 1 and at day 5 with TGEV vaccine (Ambico Prosystem I®) while the other pig was unvaccinated. An immunoperoxidase test was done on sections of the small intestines of the two pigs. This test was strongly positive for TGEV antigen in intestinal epithelium in both pigs.

The recent history (mid-December to mid-January) and the clinical signs present in the pigs at the time of the herd visit, both in the farrowing and nursery/starter rooms, was consistent with an acute TGEV outbreak in these areas. The laboratory results confirmed a definitive diagnosis of TGE.

Table 1

TGEV titers. Increasing titers suggesting endemic TGE are highlighted (white background).

Tube	November 24, 1995	December 15, 1995
2-week-old pigs		
1	32	32
2	32	8
3	64	<4
4	128	32
5	Not Done	64
6	64	64
7	Not Done	8
8	64	8
9	32	32
10	128	128
11	64	16
12	128	16
13	64	32
14	32	256
15	16	128
16	16	16
17	64	32
18	8	16
19	32	64
20	8	64
5-week-old pigs		
21	> 512	256
22	256	32
23	64	8
24	64	8
25	64	< 4
26	32	16
27	32	< 4
28	> 512	< 4
29	64	< 4
30	64	< 4

Timeline of TGEV outbreak

Discussion

The observed clinical findings of endemic TGE in this herd are similar to those described by other authors.[1–3] Diarrhea in the nursery pigs typically began 7–10 days postweaning. There were two diagnosed periods of recrudescence in this herd, one in September 1995 involving widespread diarrhea in the weaner facilities and the second occurring in mid-December 1995. The episode in December became apparent as a sudden resurgence of severe diarrhea among the suckling piglets. This was followed by episodes of diarrhea throughout the hot nurseries and starter barn.

In this herd, the reservoir of TGEV is being maintained in the weaner population. Piglets lose the protection of lactogenic immunity when removed from the immune dam.[1] Piglets are clinically affected when viral exposure exceeds the pigs' passive immunity, and the age when this occurs is related to the management system used in the herd and the degree of immunity in the sow.[2] Sera from three piglets tested at 2 weeks of age and at 5 weeks of age showed a fourfold rise in antibody titer between the acute and convalescent serum. These results supported the diagnosis of endemic TGEV, and that active TGEV infection was occurring in the nursery stage of production. In endemic TGEV infections in which the main clinical feature is postweaning diarrhea, the preferred method of laboratory confirmation is the serological examination of paired blood samples usually collected at weaning and 4 weeks later, to detect rising serum neutralisation (SN) titers.[1]

Testing conducted in this study to determine the effectiveness of the initial eradication program involved serologically testing 20 pigs from a TGEV-negative farm 30–40 days after they had been mixed with grower pigs from the breeding stock farm. The serology test results from the 20 sentinel pigs were negative for serum neutralisation titers to TGEV, while sera from 20 of the pigs from the TGE source herd were seropositive. The erroneous conclusion was reached that the TGE virus was no longer active in the source herd. Research has shown that virus has been isolated from the lungs and intestines of TGEV-infected pigs for 104 days post-infection.[2] However studies have reported that while virus can be excreted in the feces for up to 3 weeks after infection, it has not been detected beyond 35 days post infection.[6] In this herd, the active viral circulation was occurring in the weaner stage of production. The grower pigs from the TGEV-infected source barn had recovered and seroconverted to TGEV but were no longer shedding the virus. This would also explain why there had been no reported problems to date from the herds that were receiving replacement breeding stock and why the sentinel weaners placed in the quarantine gilt barn did not have diarrhea.

Testing protocols that have been used to evaluate whether TGEV has been eliminated from a farm involve adding TGEV-negative sentinel animals to the source herd.[2,3,5] Sentinel animals should not be added until after the cleanup phase is finished.[3] These pigs are then monitored daily for clinical signs of TGEV and blood is collected on three occasions, immediately prior to entry into the herd, and at 30 and 60 days post entry, to monitor for seroconversion to TGEV.

In this herd, the testing protocol instituted following the cleanup phase should have been as follows:

- The breeding and growing herd should have been considered as separate herds and tested accordingly.
- TGEV-negative animals should have been added to the breeding herd; housed throughout the barn; and exposed to manure from the herd. These animals should have been monitored daily for clinical signs of TGE and serologically tested at 30 and 60 days after entry into the herd to monitor for seroconversion to TGEV.
- Statistical sampling based on 99% probability of detecting infection in this herd, if at least 10% of swine are seropositive, would mean that the number of sentinel animals to be added would be 43. The testing protocol for the growing herd should have included the addition of 43 sentinel seronegative pigs into the nursery area. The sentinel pigs should have been observed daily for clinical signs of TGE; if diarrhea had occured, the acutely affected pigs should have been submitted live to a diagnostic laboratory for diagnosis. Serum from the sentinel pigs should have been tested monthly, for 3–4 months, to monitor for seroconversion to TGEV.

In this herd, the eradication program initiated after the acute TGE outbreak in February 1995 did not eliminate TGEV from the herd and endemic infection developed. A feedback program was initiated at the time of the outbreak to expose all sows and boars in the herd to TGEV. The objective of this procedure was to deliberately infect the sow herd with virulent virus to eliminate immunologically naive hosts, which perpetuate the infection. However, our clinical observations and serological findings indicate that it is not possible to achieve 100% infection via feedback, especially in large herds.[7] Serum neutralization titers of naive animals throughout a TGEV outbreak range from 320–640 and occasionally are >1000. One month postepidemic, titers will follow a normal distribution curve and range from 320–640 and occasionally ≥1000, as well as 10%–15% of titers ≤ 2, a value most laboratories consider negative. Typically the titers of naive animals (e.g., baby pigs and gilts) that have been orally vaccinated only for TGEV are >100. It is difficult to get titers >100, because only the IgA antibody is present, not the IgG. However, with two oral as well as an intramuscular vaccination, the IgG antibodies are triggered and titers range between 128–640, with 320 being a common value. The titers of animals that have been vaccinated and then exposed to wild-type TGEV can be as high as 3000 and, in the occasional animal, >6000. In endemic herds that have not been vaccinated for TGEV, a cross-sectional blood test would show titers of ≤ 2, 128, 640, and occasionally 1000.[8]

The Ambico ProSystem 1® TGEV vaccine has been used in addition to other management control procedures to successfully eliminate endemic TGEV from a large herd.[7] The use of a vaccine program following a natural TGEV outbreak is intended to booster the herd immunity; even after a massive epidemic, 10%–15% of the animals do not seroconvert. Studies of herds with endemic TGEV have shown that intramuscular vaccination is not as effective in boostering immunity as a combined oral and intramuscular administration.[8] The vaccination protocol instituted after the initial TGE outbreak in this case herd was not effective in preventing endemic TGE from developing in this herd. The protocol did not follow the manufacturer's recommendations with respect to the use of the oral vaccinations in either the sows (gilts) prefarrowing, the incoming breeding stock, or the baby pigs. In addition, vaccinating the gilts prefarrowing and not at entry allows susceptible animals to be exposed for an extended period of time to any viral shedding in the herd.

The farm did not institute a strict AIAO movement in the farrowing and nursery room. Pigs continued to be moved between rooms both in the farrowing and hot nursery rooms. The starter barn was run on a continuous-flow basis and a sick pen was maintained in this area. Strict AIAO pig flow is required in the farrowing rooms and nurseries to prevent the enzootic form of the disease from developing.[5] Pigs weaned from immune sows, at which time they lose their passive milk immunity to TGEV, will be susceptible to infection.

Completely cleaning and disinfecting the facility should begin approximately 2 weeks after the clinical signs have subsided, because the virus might survive in the environment if conditions were favorable. Rodents and birds should be controlled because mice and starlings can act as short-term reservoir hosts.[1]

Implications

- The TGEV was not eliminated from this breeding-stock-producing farm after the acute outbreak in February 1995. The endemic form of TGEV occurred, resulting in two acute episodes of disease and

nursery diarrhea.

- The failure of the initial eradication program was due to the absence of strict AIAO movement of pigs (especially in the nursery rooms), failure to follow the vaccine manufacturer's guidelines, and lack of complete washing procedures necessary to eliminate the virus from the environment. Susceptible weaned piglets became infected and a reservoir of TGEV was established among the weaner population.
- Initial testing to evaluate the TGEV status of the farm was insufficient with respect to the number of animals tested, the testing frequency, and the fact that it did not represent an accurate herd profile.

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