



# pork industry handbook

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## Transmissible Gastroenteritis (TGE)

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Among the economically important diarrheal diseases of baby pigs, transmissible gastroenteritis (TGE) is still a major cause of sickness and death. Although accurate statistics are not available, epidemics of TGE are reported every winter. When the disease strikes a seronegative (antibody-free) herd at the time of farrowing, it is not unusual to lose most (often 100%) of the pigs farrowed under 3 weeks of age. A milder enzootic form of TGE, associated with chronic or intermittent episodes of diarrhea usually in 1- to 3-week-old suckling or recently weaned pigs, occurs in partially immune (seropositive) herds that have continuous farrowing or where pigs are regularly added or mixed. A respiratory variant of TGE, referred to as porcine respiratory coronavirus (PRCV), has appeared in the United States and Europe. Porcine respiratory coronavirus infections have complicated the diagnosis of TGE by generating cross-reactive antibodies that cannot be differentiated using conventional serologic tests, even though they are usually associated with only mild respiratory disease or subclinical infections.

### Clinical Signs and Appearance

Transmissible gastroenteritis is a mild disease except in pigs less than 3 weeks of age and in sows infected at or near farrowing. Pigs other than these have a more or less fluid diarrhea for 1 to a few days, vomiting may occur, and the pigs usually go off feed for a short time. Because of reduced food intake pigs may lose weight, but in most cases they regain these losses.

Clinical signs in newborn pigs are much more severe. Diarrhea starts 16 to 30 hours after the pig is exposed to virus. Thus, pigs born in a farrowing house in which a TGE outbreak occurs are born healthy and show the first signs of a TGE infection on the day following birth. Diarrhea observed in baby pigs during their first hours of life is not TGE.

The first sign in baby pigs is almost always vomiting. This may be missed in pens where the sow runs free because she will eat the vomitus. Vomiting is followed very quickly by diarrhea. The first diarrhea, again, can be missed by a casual observer because it is very thin, watery, and will frequently run down the hind legs and drip from the tail. As the disease progresses, the diarrhea becomes more obvious because the skin of the rump is usually wet and soiled, and the odor of the feces becomes especially offensive. As the diarrhea thickens, it becomes yellowish, often containing small curds of undigested milk and is seen in little puddles on the floor.

As the disease progresses, the pigs become dehydrated and have eyes that are sunken and a rough hair coat. They are very thirsty and will drink water or attempt to suckle even when weak. Most pigs that die of TGE do so 2 to 5 days after they become infected, but in some cases death may occur sooner. The younger the pigs are when infected, the shorter the interval from onset of diarrhea to death. Pigs that have TGE are highly sensitive to chilling, and this may be a partial explanation for the rapid deaths sometimes seen. Pigs that live more than 6 to 8 days usually will recover. In most cases recovered

pigs will do well, although in some herds, pigs may remain unthrifty.

Sows infected just before or after farrowing become feverish and depressed, often vomit or refuse feed, and have a greenish diarrhea that persists for 1 to a few days. Feed refusal is the first and most common sign of sows sick with TGE. Their milk quantity diminishes, and they may even quit milking. In some cases, if the litter continues to suckle, they return to normal lactation.

## The Cause

Transmissible gastroenteritis is caused by a virus that belongs to the group called coronaviruses. This virus is characterized on the basis of its distinctive morphology as enveloped, rounded but variably-shaped particles surrounded by a "corona" of club-shaped projections



Figure 1. Electron microscopy (EM) of negatively stained transmissible gastroenteritis virus particles. Arrow points to surface club-shaped projections or spikes that form the "corona" surrounding the particle. Porcine respiratory coronavirus particles have an identical morphology and cannot be distinguished by EM. x195,000.

(Fig. 1). The virus is covered by an envelope that is easily destroyed by detergents and inactivated by warm temperatures, drying, and sunlight. The virus is resistant to acid, allowing it to pass through the stomach and infect the small intestine. It survives for long periods in a cold, dark environment and almost indefinitely when frozen.

## How the Virus Causes Disease

Transmissible gastroenteritis virus (TGEV) has a special affinity for cells that line the small intestine. These cells cover millions of tiny villi that project into the intestine to provide a huge surface area that functions to digest and absorb nutrients from ingested food (Fig. 2). When TGEV enters these cells, it multiplies and destroys the cell within 4 to 5 hours. Once the cell dies, thousands of virus particles are released to infect other similar cells. After 4 to 5 cycles of virus multiplication, almost all of the digestive cells in the baby pig are killed. As part of a healing response, immature cells in the small intestinal lining are quickly produced to provide a covering for the villi. However, these cells cannot effectively digest or absorb nutrients or water (Fig. 3). The destroyed cells are replaced with normal cells within 1 to 10 days if the pig survives.

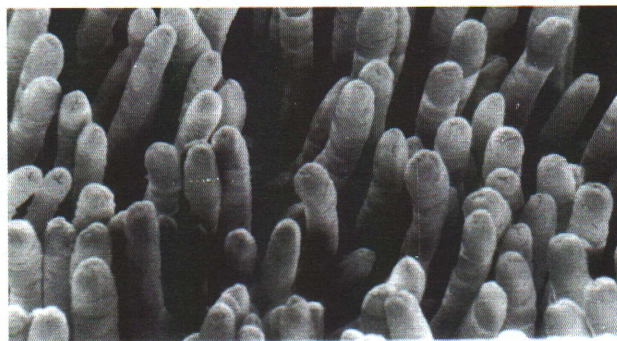


Figure 2. Scanning electron micrograph of normal small intestine, showing villi that line the intestine. These projections increase the surface area and are covered with the cells that digest and absorb nutrients from the feed. x200.



Figure 3. Scanning electron micrograph of the small intestine, as it appears after infection with TGE virus. The surface cells are killed and shed, resulting in shortening of the villi and loss of the ability to digest and absorb nutrients. If the pig survives, villi will regain their original form in 1 week to 10 days. x200.

Milk or other foods that are ingested are not digested in TGEV infected pigs. Undigested nutrients, unabsorbed water, lost body fluids and electrolytes cause diarrhea. Loss of fluids and salts upsets the salt-water balance in the blood which then becomes acidic. The resulting dehydration, and failure to absorb nutrients, causes starvation. The pig dies of a combination of these factors, although complications of stress, chilling, and secondary infections may also be involved.

## Diagnosis

An accurate diagnosis is necessary to establish treatment and management of the infected herd. A strong presumptive diagnosis of TGE may be made on the basis of clinical signs. The pattern of a rapidly spreading diarrheal disease involving breeding stock, feeder pigs, and baby pigs is very typical of TGE, but is not always seen. It may be different in the absence of baby pigs or when part of a herd is immune. Postmortem finding of severe shortening of the villi of the small intestine as shown in Figure 3 is indicative of TGE, but some degree of villous atrophy may occur in other diarrheal diseases of pigs, notably rotaviral infections, so these lesions require careful interpretation.

Traditional serological assays [virus neutralization and fluorescent antibody (FA) staining] are complicated

by the fact that polyclonal antibodies fail to differentiate between TGEV and PRCV. A presumptive diagnosis can be made on clinical signs in the newborn pigs, a histological evaluation to detect villous atrophy, and the FA test to detect TGEV-infected cells in the small intestine. Porcine respiratory coronavirus does not cause vomiting, diarrhea, or villus atrophy in young animals and replicates in the upper respiratory tract.

In some herds, an atypical pattern of TGE occurs in pigs whose mothers are immune as a result of previous exposure to the disease. In such herds, diarrhea occurs mainly in suckling pigs over 8 days of age or in recently weaned pigs. This form of TGE, commonly referred to as enzootic TGE, occurs mainly in herds on a frequent or continuous farrowing schedule. This presents a situation where susceptible pigs perpetuate the infection. An accurate diagnosis of enzootic TGE can be difficult and must be differentiated from diarrhea due to rotavirus since the latter also occurs mainly in pigs of this same age group, and coccidiosis (caused by *Isospora suis*), which mainly causes diarrhea in 7- to 10-day-old pigs.

## Immunity

One of the most effective and widely used methods of combating viral diseases is immunization, but in spite of considerable effort by federal researchers, universities, and commercial industries, a completely safe and effective TGE vaccine is not available at this writing. Immunity to TGE and other diseases of the intestine is a complicated subject and still incompletely understood.

The problem with immunity to TGE stated briefly and with oversimplification is: the pigs must have immunity during the first days of life. A sow that has been infected naturally provides immunity to its pigs through her colostrum and milk (lactogenic immunity). Her pigs are immune as long as they suckle. If they stop suckling for any reason, they become susceptible within hours because their immunity depends upon the presence of milk antibody in the gut.

Currently there are 8 federally licensed TGE vaccines available for use in pregnant sows to provide some protection for their suckling pigs. The vaccines are either killed virus or modified live virus products and usually are given about 6 weeks and 2 weeks before farrowing. Routes of vaccination include oral, intramuscular, intramammary, or combinations of these routes. These vaccines provide only limited protection against infection and diarrhea, but in some circumstances they may tend to reduce death losses, especially in herds experiencing enzootic TGE. The practical solution is to develop a safe vaccine that will stimulate the same antibodies in sow's milk as a natural infection. Injected vaccines cause high levels of antibody to appear in colostrum (the first milk), but 1 to 3 days after farrowing, and thereafter, only low levels of antibody occur in milk. Thus, the pigs of a sow given an injected vaccine may be transiently protected against TGE while suckling colostrum, but within a few days of birth they become susceptible as the colostrum is replaced by milk.

It is possible to produce substantial immunity by planned infections of sows with the indigenous virulent TGE virus strain. By giving a single dose of homogenized small intestines from young pigs acutely infected with

TGE to a sow at least 3 weeks before farrowing, she will be able to protect most of her suckling pigs against the virulent TGE virus. This is a hazardous method of controlling the disease because the planned infection may not be contained. It may spread to other herds in the area, or start an enzootic TGE infection in the herd. Sows also could be infected with other pathogens from the donor pigs' intestines. Despite the hazards involved, there are certain situations in which the procedure can be justified and beneficial.

## Epidemiology

The most cost-effective means of controlling diseases of livestock are based on sanitation; that is, keeping the animals and pathogens separated. Good sanitation procedures depend upon knowledge of the epidemiology of the disease in question. There are still many unanswered questions as to how TGE virus survives and spreads in nature because such research requires keeping swine in isolation for long periods. It is time-consuming, expensive, and relatively little has been done. Thus, what is stated here as fact may have to be revised as new knowledge is developed.

Transmissible gastroenteritis is a disease exclusively of swine. No reservoir of infection other than swine has been identified. Cats, dogs, and foxes can become infected with TGE virus and may shed the virus in their stools for up to 2 weeks. They could be involved in herd-to-herd spread of virus during an epidemic, but this is not likely in the survival of the virus from year to year. House flies have been proposed as possible vectors for TGE. Transmissible gastroenteritis virus was detected in flies associated with an enzootic herd and experimentally inoculated flies excreted TGE virus for 3 days. They are unlikely to play any role in the survival of the virus from year to year. Massive flocks of starlings can be found in feedlots during the winter when TGE is most prevalent, and circumstantial evidence suggests that they may be involved in herd-to-herd spread of TGE during epidemics. Attempts to infect starlings with TGE virus have failed, although virus can survive in their intestines for at least 36 hours. Thus starlings could eat feed contaminated with virus at one farm and shed the virus in their stools some time later at another farm. It is also possible that starlings carry virus from herd to herd on their feet or feathers. There is nothing, however, to indicate that they function to keep TGE virus alive from one season to another.

The most important animals involved in the maintenance and spread of TGE are swine and humans. Swine are the reservoir of the virus, and people, through their activities, help spread the virus from herd to herd.

The natural route of infection of pigs with TGE virus is by ingestion, or in close quarters, by airborne virus as pigs may be infected by inhalation. Virus multiplies in the gut and when passed in the stools, other pigs become infected. The quantity of virus in the diarrheic stools is very high (up to a million infectious doses per gram) during the first days of the disease. It decreases as the pigs recover and the stool becomes solid. With one questionable exception, virus has not been found in the feces of pigs for more than 2 weeks after infection. Virus may persist in the gut and possibly the respiratory tract for

as long as 2 or 3 months. Whether this virus ever starts new infections is an unresolved question.

TGE virus can persist for long periods under conditions in which new pigs are continuously added to a group. In large continuously farrowing herds, most of the sows may be immune and their pigs will be totally or partially protected while they are suckling the sows; however, the pigs will have no protection after weaning. As discussed under "Diagnosis," this pattern of enzootic TGE in a herd can be difficult to diagnose.

Weaned pigs with TGE usually do not die as a result of the infection but may provide a reservoir of the virus for other pigs in the same herd. It seems logical, therefore, that situations in which weaned pigs from different herds are commingled, may also serve to maintain TGE virus; in fact TGE has been documented in pigs involved in medicated early weaning studies.

The major means of spread of TGE virus among herds is stool (feces) from recently infected pigs. TGE virus can be carried among herds most effectively by movement of pigs, but also, by anything that is likely to be contaminated with manure and moving among herds. This includes equipment, feed, trucks, fomites, and people. People may carry virus on their hands and clothing as well as on their shoes or boots.

New TGE outbreaks usually occur in the winter. This can be explained at least partially by the fact that the climatic conditions of winter, that is, low solar radiation due to long nights and cloudiness, and low temperatures, favor the survival of the virus as it is carried among herds in pig stools. The conditions of intense solar radiation and high temperatures occurring in the summer reduce the chance for virus to be carried. Also of possible importance is the tendency for large numbers of starlings to feed close to swine in the winter months, especially after a snowfall.

## Treatment

As is the case with almost all viral diseases, no drugs are effective against TGE virus in swine. Lacking specific antiviral drugs, treatment must be directed at the effects of the virus rather than at the virus itself. As was indicated earlier, the cause of death in TGE is starvation, dehydration, and acidosis. It would be possible, theoretically, to replace all of the water, nutrients, and electrolytes that are lost through diarrhea by injecting them intravenously or subcutaneously. To some extent this has been done in the laboratory, but the process is expensive, time consuming, and impractical on the farm. Although affected pigs have a reduced ability to absorb water and nutrients, the provision of water and supplemental heat will help to offset dehydration and decrease the demand for energy by keeping pigs warm.

Doing two things for TGE-infected pigs, keeping fresh water before them at all times and providing a draft-free place with a temperature of 90+°F, can significantly reduce losses especially in pigs that are 3 to 4 days or more of age at the time they are infected. Pigs infected during the first day of life do not cope as well and the disease is usually fatal.

In some cases, secondary bacterial infections appear to be involved, and antibiotic treatment may be advisable

even though it will not affect the course of the viral infection in the pig.

## Management of TGE-Infected Herds

Management is the most effective and least expensive means of reducing losses in a TGE-infected herd. It should be directed not only at saving pigs already infected, but also at preventing infection of pigs to be farrowed, and very importantly, preventing spread to other herds in the area.

It is very difficult to make blanket recommendations because each herd differs in its facilities, farrowing schedule, marketing goals, availability of labor, and other factors that influence management decisions. A veterinarian who understands in detail the pathogenesis, epidemiology, and immunity to TGE (which were briefly outlined previously) can be most useful. Some procedures to be considered follow.

Pigs born to non-immune sows in a room in which other pigs have TGE are almost certain to break with TGE on the second day of life. Therefore, sows farrowing in the 10 days or 2 weeks after an outbreak starts should be moved to other quarters. This reduces the probability that their pigs will become infected, and shortens the time that the farrowing room will be contaminated. Individual houses with attached pens, separated from each other by a few feet, are ideal but not usually available on modern farms. It is usually possible, however, to find some accommodation for sows outside of the infected farrowing house, even though it otherwise would be considered inadequate. It is often surprising how effective this procedure can be. Even sows that farrow as early as the first night after removal from the contaminated farrowing house may have litters that remain healthy if care is taken to maintain their isolation.

In herds with enzootic TGE, deliberate infection of pregnant sows with the virus already on the farm may be useful. This is best done by chopping or grinding the virus-containing intestines of newly infected pigs in water and adding the slurry to the sows' feed. There is no evidence to suggest that live TGE virus administered to pregnant sows causes abortions or *in utero* infections. The infected sows develop some immunity in about 10 days and it becomes stronger during the next weeks. Such sows may then be moved into the farrowing house. The effectiveness of this procedure is increased if all sick pigs are removed and the room is thoroughly cleaned and allowed to stand at about 70° to 80°F for 2 or more days.

Under some circumstances, it may be useful to deliberately expose growing swine as well as sows. This may shorten the total time the disease is active on the farm over what it would be if the infection were allowed to progress naturally through the herd.

Decisions concerning planned infections should be made with professional consultation to assure proper handling of the virus and infected animals. It is possible to spread agents other than TGE virus in this way. Improper management could result in swine being infected at the wrong time, or spread to swine other than those intended.

Preventing the spread of TGE to other farms is an

important responsibility. Dead pigs should be incinerated or kept in a container so they are not available to dogs or vermin that may travel between farms. Workers should not go to other farms or to places where other pork producers gather without a complete change of clothing.

The usual question raised by pork producers is how long one should wait before infected or exposed swine can be safely moved into other herds. A common recommendation is that swine should not be moved to other herds for at least 1 month after the last signs are seen in the herd. This period is somewhat arbitrary since it has been shown that virus may actually persist in pigs for longer periods, but no cases of spread from swine sold after such a time have been confirmed. The actual time should be based upon such factors as the need for the sale, the possible consequences of transmitting infection, the possibility of quarantine of the pigs in the herd receiving them, the time of year, and an understanding with the new owner.

### **Porcine Respiratory Coronavirus**

A respiratory variant of TGEV, PRCV, has been isolated from pigs of all ages in the United States and Europe. The virus has an altered cell tropism because it grows very well in the epithelial cells lining the upper respiratory tract, but not very well, if at all, in the epithelial cells lining the intestinal tract. The virus can be isolated in cell cultures from tonsil and nasal swabs, but rarely from rectal swabs. After the initial infection, PRCV will usually spread to all other pigs on the farm. In Europe, PRCV has been shown to infect pigs on farms several miles away, presumably by aerosol spread. In young pigs, PRCV produces a subclinical or mild respiratory infection characterized histologically as a diffuse intersti-

tial pneumonia. No diarrhea is observed in PRCV-infected pigs. The infection may cause slight weight loss, which is rapidly recovered. However, recent information suggests that combined infections of PRCV and other respiratory pathogens may lead to more severe respiratory disease than infection with each agent alone.

In some European countries, the percentage of pigs with TGEV antibody may approach 100%. This high percentage is due to PRCV and not TGEV. Because of this cross-neutralization, pigs tested for TGEV antibodies may give a false positive response. Most regulations governing the import and export of pigs require them to be free of TGEV antibodies. Pigs testing positive cannot be given a permit. The regulations now allow pigs with antibodies to PRCV, but not to TGEV, to be granted an export permit. Therefore, it is necessary to differentiate between pigs exposed to TGEV and PRCV. A commercial diagnostic test from Canada is available at the National Veterinary Services Laboratories (USDA, APHIS) that differentiates between serum from pigs exposed to TGEV or PRCV. Similarly, researchers in the United States also have developed differential reagents and diagnostic serological tests based on U.S. strains of PRCV and TGEV. Limited serological surveys indicate that PRCV infections are increasing in prevalence in the United States. Preliminary cross-protection studies between PRCV and TGEV have produced varying results. Most experimental studies indicate that PRCV infections induce limited protection in pigs against challenge exposure with TGEV. However, in Europe the widespread prevalence of PRCV infections in swine herds has been accompanied by a concomitant decrease in classical TGE outbreaks. These data suggest that prior exposure to PRCV moderates the severity of TGEV infections in the field at least under the swine management systems used in Europe.

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