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pork industry handbook

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Rotaviral Diarrhea in Pigs

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Recently, a group of viruses known as rotaviruses has been found to be a frequent cause of diarrhea in the young of many different animals and man. In 1976, a porcine rotavirus was first reported as a cause of diarrhea in pigs. Since then, only limited information has become available about rotaviral diarrhea in pigs; thus, this report will be incomplete in many respects. However, since infections are very widespread and common in swine, especially infecting young pigs, this fact sheet is included in the Pork Industry Handbook.

The Cause

Rotaviruses have been isolated from different species of animals besides pigs. At least 2 other types, a bovine (calf) rotavirus and a human rotavirus, can also infect and cause diarrhea in pigs; however, there have been no reports of pigs being naturally infected with the bovine or human rotavirus. The name "rota", which is Latin for "wheel," comes from the wheel-like appearance of the virus when seen through an electron microscope. Rotaviruses have been difficult to grow in the laboratory, and this has delayed the recognition of and the research findings on rotaviral diarrhea.

Clinical Signs and Epidemiology

Suckling Pigs. Rotaviral diarrhea has been reported in 1- to 6-week-old suckling pigs, but is probably most common in pigs about 3 weeks of age. It appears that rotavirus is one of the principal causes of a widespread and common type of diarrhea that has been variously referred to as white scours, milk scours or 3-week scours. Some veterinarians and pork producers believe that white scours occurs when pigs ingest more milk than they can digest, which often takes place in litters of high milk-producing sows at about 3 weeks of age. Suckling pigs under 1 week of age seldom have rotaviral diarrhea, apparently because they have obtained sufficient immunity from nursing their mother. Rotaviral diarrhea has not been reported in pigs over 8 weeks of age.

The diarrhea is characterized by a white or yellow stool which, at the onset, is liquid; but after a few hours or a day, it becomes creamy and then pasty before returning to normal. The duration of diarrhea may be for only a few hours to several days. Vomiting occurs much less frequently than it does in transmissible gastroenteritis (TGE). In most cases, pigs remain active and lose little, if any, weight. Present information suggests that rotaviral infection in many pigs results in either no clinical signs of disease or only a mild disease characterized by a short-term diarrhea. However, field observations indicate that the severity of the disease and the death rate are increased by concurrent infections with pathogenic *Escherichia coli* (colibacillosis) or TGE virus, by inadequate intake of immune milk, and by stress such as chilling. The disease is more severe in younger pigs. Diarrhea is more profuse and, thus, more noticeable in pigs that ingest a large amount of milk.

Outbreaks of rotaviral diarrhea will often be first observed in farrowing or nursery units in 3- to 4-week-old pigs. As the disease spreads, younger pigs are increasingly exposed to the virus and may experience diarrhea at 4 to 10 days of age.

In many respects, rotaviral diarrhea is similar to enzootic TGE (persistence of TGE infection in a herd). Sows are usually not sick in either disease. In enzootic TGE, the usual age for diarrhea is at 1 to 6 weeks of age, depending on management conditions, and the death loss may be from 0 to 20%. The duration of diarrhea is longer, and dehydration and death losses are greater in enzootic TGE than in rotaviral diarrhea.

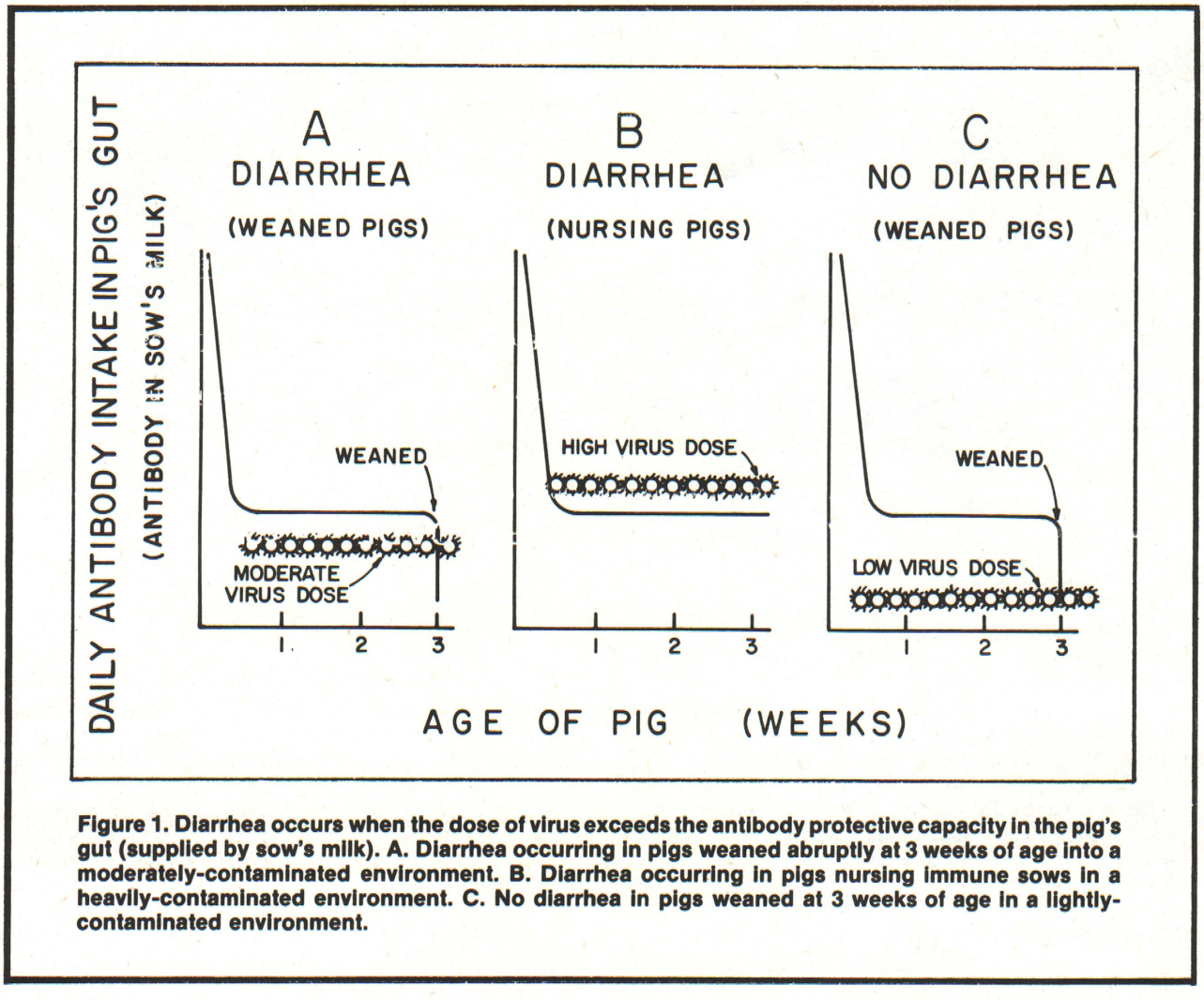

Figure 1. Diarrhea occurs when the dose of virus exceeds the antibody protective capacity in the pig's gut (supplied by sow's milk). A. Diarrhea occurring in pigs weaned abruptly at 3 weeks of age into a moderately-contaminated environment. B. Diarrhea occurring in pigs nursing immune sows in a heavily-contaminated environment. C. No diarrhea in pigs weaned at 3 weeks of age in a lightly-contaminated environment.

Both rotaviral and enzootic TGE are more apt to occur and to be a problem in herds which are (1) on a frequent or continuous farrowing schedule, and (2) which do not practice the "all in and all out" management system in the farrowing or nursery units. As a result, infections may persist in these housing units because each group of incoming pigs becomes exposed and infected by the previous older groups.

Weanling Pigs. Three simple facts must be kept in mind when predicting whether or not rotavirus will be a problem in weanling pigs:

- Rotavirus is widespread in nature. Poor management practices such as continuous use of facilities without benefit of a clean-up, fumigation and a resting time between groups of pigs increases the dose of microbes in the environment, including rotavirus.
- Most sows have protective antibodies in their milk and colostrum (gilts may have less). Rotavirus (like TGE virus) grows in and destroys the cells of the gut. Therefore, to protect the pig's gut cells from rotavirus, antibody must be present in the gut (antibody in the pig's blood is NOT protective).
- The younger the pig, the more vulnerable he is to the diarrhea caused by rotavirus.

Keeping these facts in mind and referring to Figure 1, it is easy to see that pigs will have problems with rotavirus every time the dose of the virus exceeds the protective antibody level in the pig's gut, this antibody being supplied by the sow's milk. Therefore, the earlier a pig is weaned in a contaminated environment, the more likely a severe outbreak of rotaviral diarrhea will occur. Figure 1A illustrates the situation for pigs weaned at 3 weeks of age. Even though pigs are somewhat resistant to rotaviral diarrhea at this time, the abrupt removal of the pigs from the protective antibody in the sow's milk leaves them vulnerable to the moderate dose of rotavirus that is in their environment. Of course, the earlier the weaning, the higher the death losses. It is also true that if the dose of virus is high enough, pigs nursing immune sows will also experience rotaviral diarrhea in the farrowing house (Fig. 1B). The ideal management situation is illustrated in Figure 1C. In this case, the virus dose is too low to make the pigs sick.

How the Virus Causes Disease

Rotavirus, like TGE virus, has a special affinity for cells which line the small intestines. These cells cover the millions of long finger-like projections—called villi—which make up the inside lining of the small intestines (Fig. 2, A and D). When these cells are infected and destroyed by rotavirus, the villi become short and blunt (Fig. 2, B and E), and nutrients are improperly digested and absorbed. In suckling pigs, much of the ingested milk will pass through the gut without being digested or absorbed and will appear as whole milk or curds. This can result in diarrhea, loss in body weight, and sometimes death.

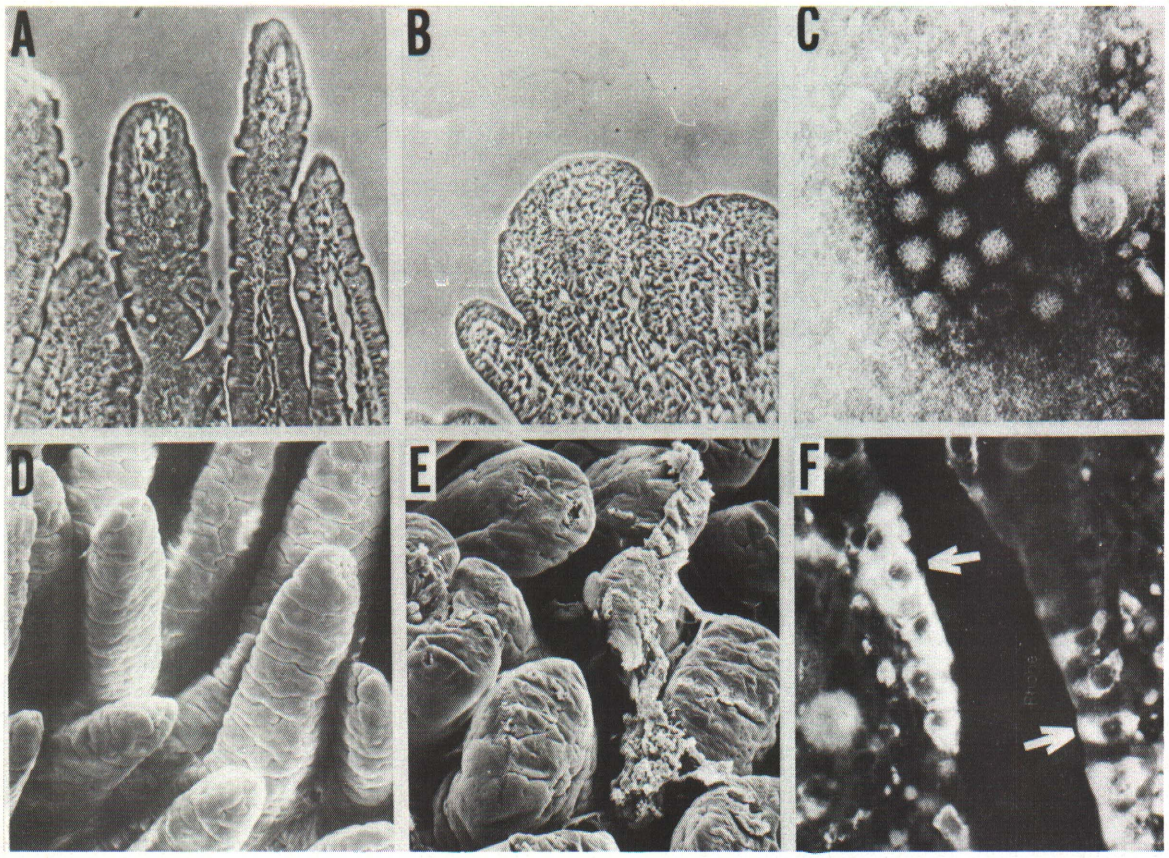

Figure 2. A. Normal villi, (Phase contrast, X75). B. Shortened, blunted villi from an infected pig (Phase contrast, X75). C. A group of rotaviruses (Electronmicrograph, X100,000). D. Normal, elongated villi (Scanning electromicrograph, X413). E. Shortened, blunted villi from an infected pig (Scanning electronmicrograph, X413). F. Fluorescent antibody stain of a section of the villi of an infected pig; arrows point to rotaviral infected cells, X200.

Diagnosis

Rotavirus should be considered as a possible cause when diarrhea occurs (1) in suckling pigs with a yellow or white stool, especially in pigs over 1 week of age, and (2) in early weaned pigs. Transmissible gastroenteritis virus or pathogenic *E. coli* should also be considered, either occurring alone or in combination with rotavirus. Rotaviral diarrhea can closely resemble enzootic TGE in many respects, and their clinical and epidemiological similarities have been discussed in a preceding section.

For laboratory diagnosis, best results can be obtained if examinations are conducted on specimens collected from live or sacrificed pigs within 24 hours after onset of diarrhea. Laboratory methods which are helpful in making a diagnosis include the following: (1) The lining of the small intestines can be examined for evidence of villous atrophy (Fig. 2, B and E), but this also occurs in TGE. (2) A stool sample or intestinal contents can be examined for the presence of viral particles (Fig. 2C). However, this can be done only by the use of an electron microscope, and few diagnostic laboratories are so equipped for this type of test. (3) The fluorescent antibody test is the one most reliable and applicable for most laboratories at the present time. For this test, scrapings or sections are made from the lining of the small intestines and are stained by a special procedure. This test will demonstrate those cells which are infected with rotavirus (Fig. 2F). Other laboratory tests are being developed but are not yet proven or are not readily available. Blood testing for the presence of rotaviral antibodies will probably be of little diagnostic help because most swine are positive.

Immunity

Present evidence indicates that most sows have been infected with rotavirus. As a result, they can provide their suckling pigs with a variable degree of immunity. This type of immunity appears similar to that seen in TGE. It has been called lactogenic immunity because it is dependent on the presence of immune milk in the gut, which occurs only if pigs frequently nurse an immune sow. Pigs do not receive an adequate level of lactogenic immunity in three circumstances: (1) when weaned, (2) when milk is ingested infrequently or is of low immune value, or (3) when immune milk in the gut is diluted by ingestion of creep feed. Under these conditions, pigs become more susceptible to intestinal infections. In most herds, sows provide adequate immunity to their suckling pigs for the first week of life, but thereafter,

immunity may be overwhelmed if pigs are severely exposed to rotavirus.

Prevention and Treatment

Most swine herds are probably infected with rotavirus, and there appears to be no practical method for preventing infections in conventional herds. It is even doubtful if the techniques usually employed for maintaining specific pathogen-free herds are adequate for keeping out this widespread and resistant rotavirus, but this remains to be seen. Thus, the problem is how best to live with this infection so as to minimize losses. To answer this need, we require more information on the factors which contribute to the onset of infection and to the severity of the diarrheal disease. It appears that the severity of the disease can be reduced or minimized by providing good sanitation, by keeping pigs comfortable (warm and dry), and by ensuring that pigs get adequate colostrum or milk at an early age.

There is no licensed vaccine for preventing rotaviral diarrhea in pigs. Some veterinarians have orally administered to newborn pigs a commercially-available calf rotavirus (reovirus-like) vaccine for preventing scours in 2- to 4-week-old pigs. However, this vaccine has not been approved for use in pigs and, as such, cannot be recommended. Also, there have not been any reported, controlled experiments indicating benefit of this vaccine. It has been administered to germ-free pigs and colostrum-free newborn pigs by the authors, but no detectable protection was afforded when challenged 19 to 21 days later with porcine rotavirus. However, it is possible that planned infection of suckling pigs at a suitable time or age with a porcine rotavirus could produce only a mild or sub-clinical disease because of the lactogenic type of immunity obtained from the sow, but still be capable of stimulating an adequate level of a durable type of immunity. Undoubtedly, "natural immunization" of this sort spontaneously occurs in those herds which are experiencing little difficulty with this disease.

The prospect of vaccinating pregnant swine with a rotavirus vaccine to provide additional immunity to suckling pigs appears to be of questionable value. The reason is that most pregnant swine have been infected with rotavirus and as a result may not respond favorably to vaccination or even to additional rotavirus exposure.

Cow's colostrum contains antibodies to bovine rotavirus. Feeding cow's colostrum has protected lightly-infected piglets, weaned at 1 day of age. In a very preliminary trial, heavily infected piglets weaned at 3 weeks of age were not protected by cow's colostrum.

Antibiotics or other drugs are not effective against rotaviral infections and would be of no value in treatment, unless there is a concurrent bacterial infection, such as with pathogenic *E. coli.*

Summary

Seven points can be summarized from this fact sheet:

- Porcine rotavirus was first reported as a cause of diarrhea in pigs in 1976.
- Infection of swine with this virus is considered very common and widespread. Probably most swine herds are infected.
- This virus is frequently associated with a diarrheal syndrome commonly referred to as white scours, milk scours, or 3-week scours. Diarrhea is most frequently observed in 1- to 4-week-old suckling pigs or in pigs weaned around 3 weeks of age or earlier.
- The infection and diarrhea resemble that seen in enzootic transmissible gastroenteritis but is less serious.
- Laboratory diagnosis can be made by fluorescent antibody staining of mucosal scrapings from the small intestine.
- Death loss in suckling pigs is usually very low unless there are complications owing to concurrent infections or stress, such as chilling.
- Present control measures must rely on good management such as ensuring that pigs get adequate colostrum and milk at an early age, providing good sanitation, and keeping pigs comfortable, especially warm. Protection against pathogenic *E. coli* by immunization or other means should help reduce the severity of rotaviral diarrhea in those herds having combined infections.

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