Rotaviral Diarrhea in Pigs– Pork Industry Handbook

Michigan State University Extension Service
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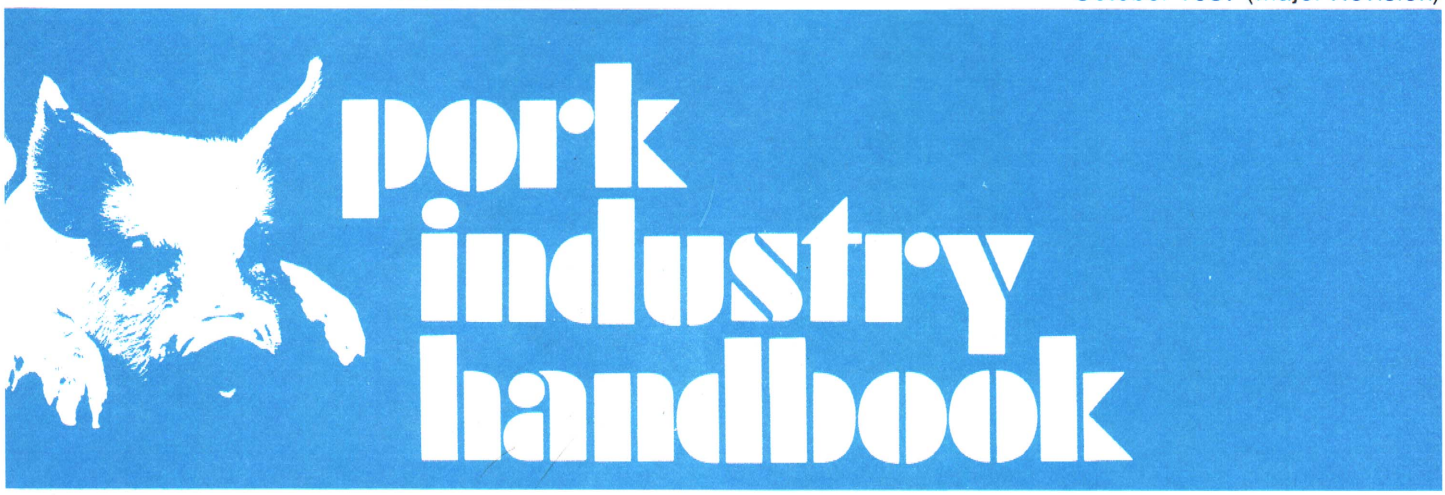
Issued October 1987
6 pages

Extension Bulletin 1337
October 1987 (Major Revision)



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Rotaviral Diarrhea in Pigs

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Rotaviruses were first detected in pigs suffering from diarrhea in 1975. Shortly thereafter rotaviral infections were widely detected in swine, and these infections were commonly associated with diarrhea in suckling and weaned pigs. Early studies also demonstrated that porcine rotaviruses are physically and serologically similar to rotaviruses recovered from other host species including man. These rotaviruses share similar viral determinants or antigens and thus cross-react with one another in various serologic or diagnostic tests. Such conventional rotaviruses are now considered members of group A rotaviruses.

In 1980, viruses that resembled rotaviruses in physical appearance, size, and biochemical composition were detected using electron microscopy on fecal samples from diarrheic pigs. However these rotaviruses were serologically different (did not share similar group A rotavirus determinants) from the previously identified conventional rotaviruses and hence did not react in diagnostic tests commonly used to detect rotavirus. These viruses have been referred to by a number of names including pararotaviruses, rotavirus-like viruses, antigenically distinct rotaviruses, and atypical or novel rotaviruses. They will be referred to collectively as atypical rotaviruses while the conventional or group A rotaviruses will be called only rotaviruses in this fact sheet. At least two distinct groups (groups B and C) of atypical rotaviruses have been identified in swine in the U.S. These atypical rotaviruses infected and caused diarrhea in germ-free pigs, similar to that caused by group A rotaviruses. While atypical rotaviruses have been described in association with diarrhea in other species including man, their incidence and significance as causes of diarrhea in swine herds is unclear. Thus, other than a description of the methods for detection and differentiation of atypical rotaviruses from rotaviruses, the major focus of this report will be on conventional or group A rotaviruses.

THE CAUSE

Rotaviruses are characterized by their wheel-like appearance when viewed using the electron microscope. Rotaviruses are resistant to low pH, lipid solvents, and many commonly used disinfectants, enabling them to survive for long periods under normal environmental conditions. At least two distinct serotypes of group A rotaviruses, referred to as the OSU and Gottfried strains, have been isolated from swine. Pigs infected with one serotype are still susceptible to infection with the second serotype. Hence, outbreaks caused by group A rotaviruses might be more complicated than initially thought, since little is known about the distribution of these two serotypes in swine.

CLINICAL SIGNS AND EPIDEMIOLOGY

Nursing Pigs

Rotaviral diarrhea generally occurs in nursing pigs at 1 to 6 weeks of age, frequently occurring in pigs about 3 weeks old and is a cause of the clinical syndrome referred to as milk scours, white scours, or 3-week scours. The age of peak incidence varies under different management conditions. Probable reasons for the peak occurrence of rotavirus infections at 3 weeks of age include the decline in milk antibody levels coupled with the dilution of this antibody as a result of the pigs ingesting creep feed and water. Rotaviral diarrhea is not as common in pigs less than 1 week of age compared to 3-week-old pigs. This is probably

because of the high levels of passive rotavirus antibodies in the colostrum and milk from the dam.

The diarrhea is characterized by a white or yellow stool which, at the onset, is liquid; but after a few hours or a day in uncomplicated cases, it becomes creamy and then pasty before returning to normal. In pigs sacrificed for post-mortem, undigested milk is often evident in the intestinal contents, and the stomach is often full and distended with milk curd. Diarrhea may persist for only a few hours or for several days. Vomiting may or may not be detected, but it occurs much less frequently than it does in transmissible gastroenteritis (TGE). Under ideal conditions, pigs remain active and usually lose little weight. Present information suggests that rotaviral infections in many pigs results in either no clinical signs of disease or only a mild disease characterized by a short-term diarrhea. However, the severity of the disease, and the death rate may be increased by simultaneous infections with *Echerichia coli* (colibacillosis), TGE virus or other causes (other enteric viruses, clostridia, coccidiosis), by inadequate intake of immune milk, or by stressors such as chilling. The disease is more severe in young pigs. Diarrhea is more profuse and more noticeable in pigs that ingest a large amount of milk.

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. The duration of diarrhea is longer, and dehydration and death losses are greater in enzootic TGE than in rotaviral diarrhea.

In continuous farrowing operations, rotaviral diarrhea may initially be observed in 3- to 4-week-old pigs. As these pigs develop rotaviral diarrhea, the environment becomes heavily contaminated with virus, which leads to exposure of younger pigs to high doses of virus often exceeding the protective capacity of the milk antibodies present in these pigs' intestines (Fig. 1). Subsequently diarrhea may occur routinely in 1- to 2-week-old animals. To break this cycle of infection, an "all in-all out" management system should be practiced in farrowing and nursery units. Housing units should be designed with floors and all surfaces that can be thoroughly cleaned and disinfected between groups.

Weanling pigs

Pigs that have had rotavirus diarrhea during the nursing period may have another episode about 3 to 7 days after weaning. Whether these repeat episodes represent infections with different serotypes of rotavirus is unknown. Other researchers also have documented the importance of rotavirus as a cause of weanling diarrhea. Such infections probably occur at weaning because of the loss of protective antibodies provided in the sow's milk. Two studies have shown that rotavirus infection shortly after weaning leads to intestinal damage which favors the colonization of the gut with enteropathogenic *E. coli*. Results of both studies suggest that pigs infected with two agents develop a more severe diarrhea than that produced by each agent alone.

Diet might also play an important role in weanling diarrhea. A diet high in solids fed only three times daily produced a more severe and prolonged diarrhea than either the same diet fed hourly or a similar diet containing one-third the amount of solids. Malabsorption resulting from rotavirus infection was most severe in pigs fed the diet high in solids, and this diet also favored intestinal colonization by enteropathogenic *E. coli*. Besides diet composition, other management variables that may influence the occurrence and severity of rotavirus *E. coli* weanling diarrhea include: meeting the critical temperature needs of the pigs, avoiding overloading the animal's digestive system with too much food at one time, and isolating the nursery, disinfecting it between batches of pigs, and dividing pigs into smaller groups of similar ages.

The dynamics of virus-host interactions in rotavirus infection is illustrated in Figure 1. Three simple facts are useful in predicting whether rotavirus will be a problem:

1. Rotavirus is widespread in nature. Poor management practices such as continuous use of facilities without a cleanup, fumigation, and resting time between groups of pigs increases the dose of microbes in the environment, including rotavirus.

2. 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).

3. The younger the pig, the more vulnerable it is to dehydration and energy and weight losses 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 younger the pigs when weaned in a contaminated environment, the greater the chance that a severe outbreak of rotaviral diarrhea will occur. In addition, the earlier weanings result in higher death losses if pigs develop rotavirus diarrhea. 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. 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 intestine. 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 incompletely digested and poorly absorbed. In suckling pigs, much of the ingested milk will pass through the gut without being digested or absorbed. This can result in diarrhea, loss of water, electrolytes, body weight and sometimes death.

Villous atrophy occurs very rapidly, within 24 to 36 hours, after rotavirus infection of the intestinal cells, and coincides with the onset of diarrhea. However, regeneration of the intestinal villi and recuperation of full normal digestive capacities will take about 7 to 10 days. This is then the most critical time to prevent malabsoption diarrhea and secondary bacterial infections.

DIAGNOSIS

Clinical and laboratory diagnosis of rotaviral diarrhea requires also evaluation for the presence of *E. coli* and/or enzootic TGE or coccidia, other agents that can cause a similar diarrhea syndrome. *E. coli* diarrhea commonly occurs in younger (1 week old or less) pigs, or at 4 to 10 days post-weaning, whereas enzootic TGE, coccidiosis and rotavirus diarrhea often occur in pigs after 1 week of age.

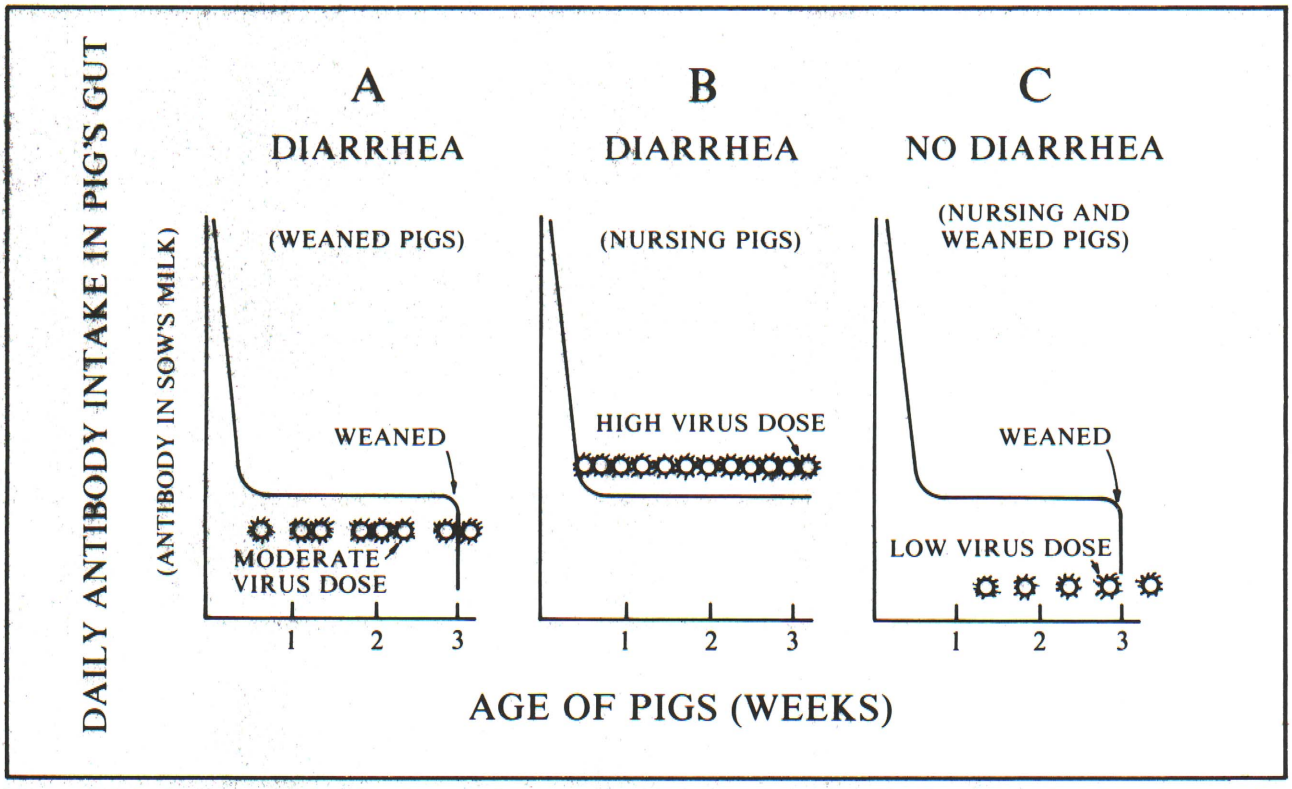

Figure 1. Diarrhea occurs when the dose of virus exceeds the antibody protective capacity in the pig's gut (supplied by sow's milk). A. Diarrhea occurring in pigs weaned abruptly at 3 weeks of age into a moderately contaminated environment. B. Diarrhea occurring in pigs nursing immune sows in a heavily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

However, since disease caused by rotavirus in younger pigs is usually more severe, producers might think the pigs have colibacillosis unless they submit pigs for a complete diagnosis.

Laboratory diagnosis requires the submission of feces or intestinal sections collected early (24 hrs. or less) after the onset of diarrhea. Laboratory methods that are helpful in making a diagnosis (when used in combination), include: histopathology, electron microscopy (EM), fluorescent antibody (FA), and enzyme-linked immunosorbent assay (ELISA).

Feces or intestinal contents can be examined for viral particles (Fig. 2C) using electron microscopy. However an electron microscope may not be available in all diagnostic laboratories, and this technique cannot discriminate between rotaviruses and the morphologically identical atypical rotaviruses. The atypical rotaviruses and other viruses can be differentiated from rotavirus using serologic tests such as immune EM, FA, and ELISA, all of which should employ highly specific antisera for each virus. Currently, commercial FA and ELISA reagents are available only for detection of group A rotaviruses. For FA, one of the most commonly used tests, pigs in the early stages of diarrhea must be sacrificed. Scrapings or sections are made from the lining of the small intestines and stained with antibodies to rotavirus conjugated to a fluorescent dye (FITC). Cells infected with rotavirus react with the FITC antibody and emit a bright apple-green fluorescence when excited by a certain wavelength of light (Fig. 2F). In the ELISA, antibody to rotavirus is stuck to the bottom of a small plastic well or tube. The suspected fecal sample is added to the well. If rotavirus is in the sample, it is captured by the antibody on the plastic. Then another antibody to the rotavirus is added. This antibody has an enzyme conjugated to it. If rotavirus has been captured from the feces, then the enzyme-conjugated antibody will adhere to it. Finally, a substrate that produces a visual color change in the presence of the conjugated enzyme is added. If color is produced, that enzyme-conjugated antibody is assumed stuck to the captured rotavirus; hence, the sample is positive for rotavirus. ELISA can be done on feces or intestinal contents and has the advantages of high sensitivity, requirement for minimal amounts of sample, and rapid results (6 to 24 hrs.). Blood samples aid little in diagnosis, since most swine are positive for rotavirus antibodies.

IMMUNITY

Because most sows are positive for rotavirus antibodies, they will transfer a variable amount of passive immunity to their nursing pigs via colostrum and milk. Studies on immunity to TGE virus have shown that effective protection depends not on blood antibody levels, but on the almost continual presence of milk antibodies in the intestine of the pig, such as occurs following frequent nursing. This type of "lactogenic immunity" is also important in rotavirus infections for protection of susceptible intestinal cells. Various factors which may interfere with this balance between passive immunity and rotavirus clinical infections include: 1) failure of the pig to nurse at frequent intervals shortly after birth or failure of the sow to provide milk may lead to severe rotavirus diarrhea in pigs under a week old; 2) high doses of virus as a result of a heavily contaminated environment may exceed the level of protective antibodies in the milk, leading to rotavirus diarrhea in nursing pigs; 3) ingestion of creep feed and water by 3-week-old nursing pigs may dilute the level of protective

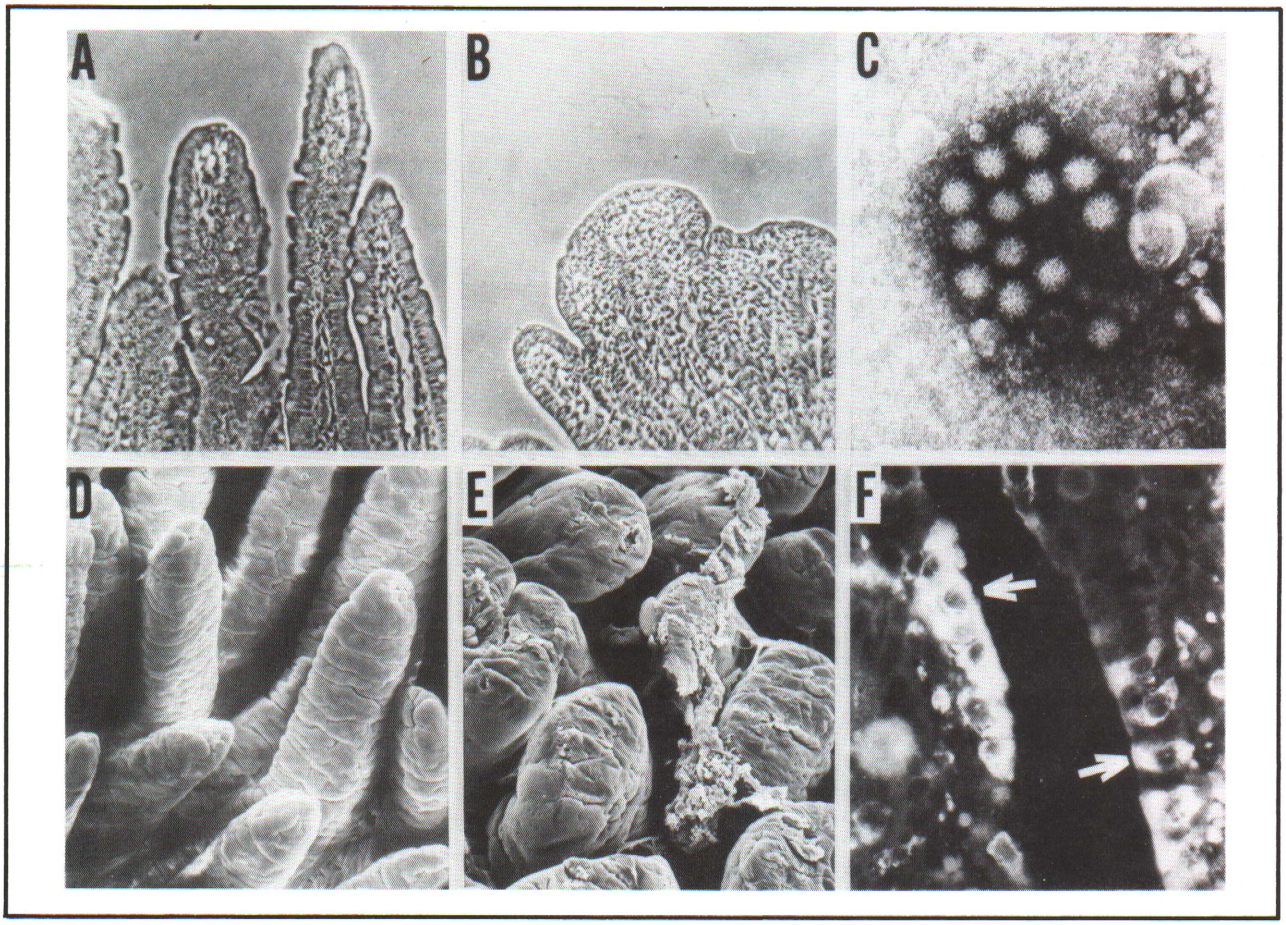

Figure 2. A. Normal villi, (Phase contrast, X75). B. Shortened, blunted villi from an infected pig (Phase contrast, X75). C. A group of rotaviruses (Electronmicrograph, X100,000). D. Normal, elongated villi (Scanning electromicrograph, X413). E. Shortened, blunted villi from an infected pig (Scanning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

antibodies leading to rotavirus diarrhea; and 4) weaning, which results in complete loss of protective milk antibodies, may cause severe diarrhea and death losses to younger pigs which are weaned in a contaminated environment.

Parenteral (intramuscular or subcutaneous) rotavirus immunization of rotavirus antibody positive sows shortly before or after farrowing can increase rotavirus antibody levels in colostrum and milk. The practical application of these immunization methods might be to enhance passive lactogenic immunity, thereby delaying the onset of rotavirus diarrhea in herds with a history of severe rotavirus diarrhea and high mortality in pigs under 2 weeks of age.

Protection of weanling pigs against rotavirus diarrhea requires active immunization, probably via the oral route, prior to weaning. Multiple serotypes of porcine rotavirus and interference by maternal antibodies make this type of potential vaccine a less feasible prospect.

VACCINES

Currently, only one manufacturer produces a federally-licensed vaccine, available in different combinations, for porcine rotavirus. The most recent vaccine incorporates the two serotypes of porcine rotavirus and is to be administered orally plus intramuscularly to pregnant swine or orally to nursing pigs. In theory, administration of a rotavirus vaccine to pregnant swine should boost colostrum and milk antibodies providing increased lactogenic immunity to nursing pigs. However, there are no reported controlled studies on the efficiency of this vaccine for boosting rotavirus antibodies in colostrum and milk or for preventing rotavirus-associated diarrhea in nursing pigs.

On the other hand, the prior commercial vaccine (containing only one serotype of rotavirus) was tested independently for protection of weanling pigs. It did not prevent rotavirus shedding or diarrhea or aid post-weaning weight gains. Possible reasons for vaccine failure include multiple rotavirus serotypes other than the vaccine serotype (OSU) or interference by maternal antibodies with active immunization of pigs. The latter was considered the more likely possibility since the serotype of rotavirus recovered from the infected pigs was the same as the OSU serotype.

PREVENTION AND CONTROL

Although most studies suggest that rotavirus infections cannot be prevented, their severity can probably be moderated by optimal management conditions. These include "all in-all out" systems in farrowing and nursery units. Careful and thorough cleaning and disinfection of the premises should be done routinely since high viral doses may lead to earlier onset of and possibly more severe infections in nursing pigs. Disinfectants which are effective to various degrees against rotavirus include: 3.7% formaldehyde, chloramine T (Multichlor®), 5% lysol, hexachlorophene (Septisol®); and triclosan (Triclosan® hand soap). It is likely that fecal material may further reduce the

effectiveness of many of these rotavirus disinfectants, necessitating complete cleanliness to achieve maximal disinfection.

Attention should be given to providing adequate heat to suckling and weaned pigs since this affects their clinical response to rotaviruses and other enteric infections. Pigs with diarrhea caused by rotaviruses or other infections that damage the villi do not absorb nutrients well and are more susceptible to chilling. Although villous repair should occur within a few days, chilling and other stresses may delay this, and the pig may develop multiple nutritional deficiencies and become stunted or a chronic "poor-doer." It is essential to ensure that neonatal pigs receive adequate colostrum and milk.

Control of weanling diarrhea may depend on factors such as: 1) feeding newly weaned pigs small quantities of feed at frequent intervals for the first few days post-weaning; 2) dividing pigs into small groups of similar ages since mixing pigs of various ages at weaning may lead to stress and favor transmission of infection from older to younger pigs; 3) emptying and disinfecting the premises between groups; 4) weaning age—younger weaned pigs usually are more severely affected than older pigs; 5) meeting critical temperature needs of pigs; and 6) ventilating for minimal levels of noxious gases (ammonia).

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

Ten points can be summarized from this fact sheet:

- Porcine rotavirus was first detected from diarrheic pigs in 1975.
- In 1980, atypical rotaviruses were detected in swine and found identical in appearance but serologically distinct from conventional rotaviruses. The latter are now classified as Group A rotaviruses and the atypical rotaviruses are classified as Groups B and C rotaviruses.
- Infection of swine with rotavirus is very common and widespread. Probably all swine herds are infected.
- Rotavirus is frequently associated with a diarrhea 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 to 5 weeks of age or earlier.
- Little is known about the prevalence or severity of infections with atypical rotaviruses.
- The infection and diarrhea caused by rotaviruses resembles that seen in coccidiosis and enzootic transmissible gastroenteritis but is less serious than the latter infection.
- Laboratory diagnosis of rotavirus can be made by fluorescent antibody staining of mucosal scrapings from the small intestine or immune EM or ELISA tests done on feces.
- Diagnosis of rotavirus solely by electron microscopy (EM) may not be accurate because of atypical rotaviruses; diagnosis and differentiation of these viruses from rotaviruses requires use of specific antisera in immune EM or fluorescent antibody staining.
- Death loss in suckling pigs is usually very low unless there are complications owing to concurrent infections or stress such as chilling.

Issued in furtherance of Cooperative Extension work in agriculture and home economics, acts of May 8, and June 30, 1914, in cooperation with the U.S. Department of Agriculture. W.J. Moline, Director, Cooperative Extension Service, Michigan State University, E. Lansing, MI 48824.

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