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# Infectious Diarrheas of Young Pigs

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Infectious diarrhea occurs when one or more enteropathogens infect the intestine and produce anatomic or biochemical lesions of sufficient severity to result in the passage of a liquid stool. Enteric disease is a complex problem. Predisposing factors include poor mothering or milking ability of the sow, reduced vigor or birth weight of neonatal piglets, and environmental stressors such as dampness, drafts, high humidity, chilling, lack of a comfortable place to rest, and poor sanitation. Passive immunity to enteric disease agents is largely dependent upon the continuous oral consumption of specific antibodies in colostrum and milk; therefore, it is important that all piglets nurse promptly at birth and regularly thereafter.

Historically, symptomatic therapy based on trial and error was the best available approach for the management of diarrheal diseases. The mass of therapeutic agents marketed for treatment of "scours" is testimony to the prevalent attitude of producers toward the control of diarrhea.

Today, advances in knowledge and technology from the last two decades have made it possible to approach most diarrheal diseases of piglets with specific etiologic diagnosis as the primary goal. Once accomplished, this diagnostic base can be built upon with the traditional medical steps of directed therapy and prevention.

## PATHOGENESIS OF DIARRHEA

Most diarrheas of piglets result from malfunction of the small intestine. The mucosa of the small intestine (duodenum, jejunum, and ileum) has two main anatomic components: crypts and villi. The villi are covered by absorptive cells that participate actively in the digestive process and absorb fluid from the intestinal lumen. The most mature absorptive cells are shed from the villous tips and are continuously replaced by immature cells from the crypts, which mature as they move distally on the villi. The crypts secrete fluid from the blood into the intestinal lumen and also provide cells

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to replace villous cells that are shed normally or as a result of injury from disease. In the large intestine (cecum, colon, and rectum), the mucosa is composed mainly of crypts, with little development of villi. Although there is a large volume of fluid secreted by the crypts in the normal small intestine, the net absorption of fluid by the entire intestine exceeds the volume of fluids secreted by the mucosa, resulting in a stool of normal consistency.

There are three major pathologic mechanisms by which diarrhea is produced: hypersecretion, malabsorption, and increased intestinal permeability.[5]

### Secretory Diarrhea

Hypersecretion is accompanied by little anatomic alteration of the mucosa. Functionally, secretion by the mucosa exceeds the absorptive capacity of the intestine. Colibacillosis is the common type of secretory diarrhea in piglets. Piliated strains of *Escherichia coli* adhere to villous epithelial cells, and enterotoxigenic strains produce toxins that cause hypersecretion of fluid into the lumen. This fluid is isotonic, alkaline, and rich in electrolytes.

### Malabsorption

Decreased absorptive capacity of the intestine in piglets usually is caused by destruction of villous absorptive cells, resulting in villous atrophy. Viruses such as rotavirus and transmissible gastroenteritis (TGE) virus, and *Isospora suis* are the common etiologic agents producing this type of diarrhea in piglets. Villous atrophy results in maldigestion as well as malabsorption, resulting in bacterial fermentation of the incompletely digested ingesta. This osmotically active material causes fluid to flow into the intestine, and fermentation causes the fecal pH to be acid.

### Increased Permeability

Inflammatory or necrotizing lesions of the intestine can result in the passage of fluid exudates into the lumen and may cause a reduction in the absorptive capacity of the intestine. *Clostridium perfringens* type-C enteritis is the most well-known example of this type of mechanism; however, other diseases such as strongyloidosis may also cause diarrhea primarily by this mechanism.

## INFECTIOUS AGENTS ASSOCIATED WITH DIARRHEA IN PIGLETS

Bacteria, viruses, protozoa, and nematodes are known to have a causal relationship to diarrheal disease. The significance and pathogenesis of disease caused by some agents are well characterized, but the role of other agents remains obscure.

### Bacteria

*Escherichia coli* is the most common bacterial enteropathogen in piglets. Pathogenic strains have pili that allow the bacteria to adhere to

villi. The three common antigenic types of pili on piglet enteropathogens are 987P, K88, and K99. Enterotoxigenic strains produce enterotoxins, which cause hypersecretion of fluid from the mucosa, resulting in secretory diarrhea. In some cases, there also is thrombosis and infarction of the distal portion of villi.

*Clostridium perfringens* type-C enteritis is prevalent in restricted geographic areas. This organism invades villi of the small intestine and elaborates beta-toxin, a potent necrotizing exotoxin that causes complete necrosis of infected villi. The predominant cause of the exudative diarrhea that follows appears to be increased intestinal permeability.

Species of Salmonella infrequently cause enteritis in nursing pigs. There probably is a secretory component to the diarrhea produced by this organism, but there also are inflammatory and necrotizing lesions that could increase permeability.

*Treponema hyodysenteriae* is the essential infectious agent of swine dysentery. Dysentery is seen occasionally in nursing pigs that are ten days to four weeks old but usually occurs only when the dam has clinical signs of the disease while nursing the litter.

Streptococci are sometimes found to adhere to the ileal villi of diarrheal piglets in the absence of other demonstrable infectious agents, but this organism has not been proved to be an enteropathogen.

*Clostridium perfringens* type-A is commonly present in many piglets with diarrhea and is thought to be enteropathogenic; however, its precise role in the pathogenesis of diarrhea in pigs has not been defined.

### Viruses

The virus responsible for TGE is a coronavirus that causes villous atrophy throughout the small intestine, thereby producing malabsorptive diarrhea. Pigs of all ages are susceptible to infection by this virus.

Rotaviruses produce lesions similar to those of TGE, except that the lesions tend to be segmental, affecting the middle one third of the intestine most severely. Rotaviral enteritis is a very common disease of piglets.

Adenoviral infection of the intestine is uncommon, but the adenovirus can infect and destroy villous epithelium in nursing pigs.[2]

Enteroviruses are demonstrated occasionally in diarrheic piglets, but lesions due to enteroviral infection have not been well characterized.[11]

A coronavirus that is antigenically distinct from TGE virus has been demonstrated to produce signs and lesions similar to TGE.[7]

Reovirus type-1 has been shown to produce diarrhea and focal villous atrophy in colostrum-deprived piglets.[3]

Calici-like viruses are found occasionally in the intestine of diarrheic piglets, but their enteropathogenicity has not been proved.[8]

Pararotavirus is morphologically similar to but antigenically distinct from rotaviruses and produces enteric lesions in gnotobiotic pigs similar to those of rotaviral enteritis.[1]

### Parasites

*Isospora suis* infects and destroys villous epithelium of the ileum and jejunum of piglets and occasionally invades crypt epithelium.[13] Coccidiosis has become prevalent and economically important since 1975.

**Table 1.** *Infectious Enteric Diseases in 1975 Diarrheic Nursing Piglets (South Dakota Animal Disease Research and Diagnostic Laboratory, 1981)*

| ETIOLOGIC AGENT | NUMBER OF ACCESSIONS | | |
|---|---|---|---|
| | *Single Infection** | *Mixed Infection†* | *Total Accessions* |
| Bacteria | | | |
| *Escherichia coli* (enteropathogenic) | 502 | 110 | 612 |
| *Clostridium perfringens* (type C) | 39 | 8 | 47 |
| Salmonella spp. | 3 | 7 | 10 |
| Swine dysentery | 4 | 2 | 6 |
| *Viruses* | | | |
| Adenovirus | 7 | 3 | 10 |
| Calicivirus-like agent | 7 | 4 | 11 |
| Coronavirus, unidentified | 10 | 4 | 14 |
| Enterovirus | 8 | 3 | 11 |
| Rotavirus and rotavirus-like | 183 | 88 | 271 |
| Transmissible gastroenteritis | 273 | 28 | 301 |
| *Protozoa* | | | |
| Cryptosporidium sp. | 5 | 0 | 5 |
| *Isospora suis* | 283 | 53 | 346 |

*Histologic and microbiologic evidence of disease due to a single enteropathogen.

†Histologic and microbiologic evidence of disease due to intercurrent infection by two to four enteropathogens.

Cryptosporidium is a coccidian parasite that attaches to the microvillous border of absorptive cells, probably impairing their digestive and absorptive functions. This organism also is seen on the luminal surface of crypt epithelium in the small and large intestines. Cryptosporidiosis in piglets has not been encountered frequently in the past but seems to be increasing in prevalence.

*Strongyloides ransomi* can cause diarrhea in heavily infected pigs up to 10 to 15 days old. Prenatal and transcolostral transmission of larvae has been recognized.[10] Although this parasite has a wide geographic distribution, most clinical cases of piglet enteritis have been reported from the southeastern United States.

The prevalence of these various types of infectious enteritis would be expected to vary among different geographic areas. The infectious agents found in diarrheic pigs examined at a midwestern diagnostic laboratory during 1981 are listed in Table 1.

## DIAGNOSIS

### Case History

An accurate herd history and the findings from clinical examination provide the foundation for establishing an etiologic diagnosis. History essential to diagnosis includes morbidity and mortality data, age at onset of diarrhea, duration of signs, and body condition after cessation of diarrhea.

Age-of-onset patterns generally are similar for each disease when a single agent initiates the problem. *E. coli* and *C. perfringens* type-C often

are solo agents causing diarrhea during the first five days of life. Diarrhea due to coccidial enteritis usually begins in six- to ten-day old pigs and persists for about five days. Rotaviral enteritis often affects 10- to 20-day-old pigs because there usually is sufficient maternal lactogenic immunity to protect younger piglets. Outbreaks of TGE in herds with a low level of immunity to the virus may be expressed as fulminating disease in all pigs more than 18 hours old, including adults. More commonly, TGE is an endemic disease, with onset at 9 to 12 days of age or in the immediate postweaning period.

Course and duration of clinical signs may separate diarrheas of younger pigs into two groups. The first group produces rapid alterations in acid-base balance, vascular integrity, or electrolyte balance, and may kill quickly. Infections in this group include colibacillosis, fulminating TGE, and *C. perfringens* type-C enteritis. The second group causes chronic debility. Infections in this group include rotaviral enteritis, coccidiosis, immune-modified TGE, and cryptosporidiosis. Successful therapy for colibacillosis can result in rapid return to normalcy because there is little physical damage to the intestine. Diseases characterized by villous atrophy (TGE, rotavirus, and coccidiosis) will have a prolonged recovery period of five days or more because convalescence is dependent upon regeneration of sufficient absorptive epithelium for satisfactory intestinal function.

Morbidity and mortality vary with any of the enteric diseases of young pigs. Carefully noted observations over time can suggest patterns in the extent of passive transfer of immunity, physical spread of pathogens, and effective altered management efforts. The dynamics of change often suggest the character of protozoal, viral, or bacterial agents and are helpful in diagnosis.

**Clinical Examinations**

The consistency, color, odor, and volume of the stools are characteristic of certain disease agents. The value of these observations is real, but interpretation is subjective and depends on the observer's past experiences and associations. For this reason, objective evaluation will be stressed in this discussion.

Assessment of diarrheal pH is an initial step in clinical examination. Accuracy depends on the number sampled because variation within a population of scouring pigs can be expected. Sufficient stool to determine pH can usually be obtained by gently squeezing the abdomen of diarrheic piglets. Satisfactory measurements of pH can be made with pH paper with a range of pH 1 to 11. Stools of piglets with colibacillosis are rich in bicarbonate and tend to be alkaline, pH 8 or greater. Malabsorptive diarrheas due to atrophic enteritis commonly are pH 7 or less. Variations within groups of pigs are expected, and mixed infections are common. Therefore, numerous pH measurements are useful to assess the relative influence of *E. coli* in diarrhea of complex etiology. The general trend in pH will help to assess the significance of *E. coli* as a primary or contributing enteropathogen.

Color of the stool usually is not very helpful in differential diagnosis. Observation of blood-stained stools in piglets less than five days old, however, is strong presumptive evidence of *C. perfringens* type-C enteritis.

Body temperature is not a valuable tool for diagnosis or prognosis of enteric disease. Rare individual animals may develop systemic illness in conjunction with bacterial or viral enteritis, but this is the exception rather than the rule.

Dehydration assessed by turgor of the skin and retraction of the globe has diagnostic and prognostic significance. Severe dehydration most often accompanies colibacillosis or fulminating TGE. Chronic intestinal disease may lead to moderate dehydration in terminal, cachectic pigs.

**Necropsy and Specimen Selection**

Necropsy is an essential step toward the goal of establishing an etiologic diagnosis. Selection of the proper animals for necropsy is the single most important factor that determines the likelihood of success of the diagnostic effort. If possible, only untreated pigs that have had typical clinical signs for less than one day should be euthanatized for examination and sample selection. (Necropsy of at least one moribund or freshly dead pig sometimes is useful for gross evaluation, but common agonal changes such as intestinal hyperemia or gaseous distention of the intestine usually serve to confuse rather than enlighten the observer.)

Gross visual examination of the viscera is informative but rarely provides sufficient information to establish an etiologic diagnosis. Characteristic gross lesions are seen in several diseases. Piglets with *C. perfringens* type-C enteritis always have mucosal necrosis in the small intestine. In the early stages of the disease there is hemorrhage, sometimes accompanied by emphysema, of the intestinal wall and blood-stained fluid is present in the lumen. In cases of atrophic enteritis due to viral infection, the intestinal wall is thin and flaccid and clots of undigested milk often are seen in the lumen of the caudal portion of the intestine. The small intestine of piglets with uncomplicated colibacillosis usually has good tone and is of normal thickness. In coccidiosis the ileum sometimes appears mildly edematous and inflamed, and about 20 per cent of piglets with *I. suis* enteritis have necrosis of the intestinal mucosa. The rare cases of swine dysentery that occur in nursing pigs are characterized by mucohemorrhagic colitis with a normal small intestine.

Confirmation of an etiologic diagnosis requires the laboratory examination of carefully selected specimens. A useful sampling procedure is as follows:

1. Bacterial isolation—Locate the ileum, cut a small ellipse in the ileal wall about 5 cm cranial to the ileocecal junction, and gently scrape the mucosal surface with a sterile swab.
2. Direct electron microscopic detection of virus particles—Collect 2 to 5 ml of fluid from the cecum (or colon, if necessary) in a leakproof container and add two to three drops of 10 per cent formalin to retard bacterial multiplication. Avoid adding excessive amounts of formalin because this interferes with the accuracy of the procedure.
3. Histopathology—Remove 3 cm segments from five areas of the intestine: ileum, middle small intestine, jejunum, duodenum, and colon. Flush the lumen of the segments with 10 per cent formalin, then immerse them in formalin. The segments should not be ligated. It is imperative that these specimens are immersed in formalin within 10 minutes after euthanasia.

4. Identification of *E. coli* pilus antigen—Prepare four air-dried impression smears of ileal mucosa on glass slides. One slide is for gram-staining, and the other three are for detection of K88, K99, or 987P antigens by immunofluorescence.

5. Viral immunofluorescence—Remove 10-inch-long segments of ileum and middle small intestine, place in plastic bag and refrigerate, but avoid freezing.

In addition to the above listed specimens, it also is useful to examine feces for oocysts and Giemsa-stained scrapings of ileal mucosa for merozoites when coccidiosis is suspected.[9]

Following collection of the specimens, the method by which laboratory procedures are performed varies among veterinary practices. Some practices are prepared to conduct most, if not all, of the necessary procedures and may submit only occasional specimens to a diagnostic laboratory. Other practices choose to submit most specimens to a diagnostic laboratory.

If a complete laboratory examination is to be conducted at a diagnostic laboratory, there are two acceptable methods of specimen delivery. Live, untreated pigs early in the course of the disease can be delivered directly to the laboratory, or the specimens described above can be submitted by common carrier. When specimens are submitted by common carrier, they should be packed with two frozen ice packs in a styrofoam shipping container that has a sturdy outer cardboard covering. Delivery of whole euthanatized pigs is not acceptable, because autolytic changes in the intestine will have advanced sufficiently to render these specimens of little or no value, even if the transit time is as short as one or two hours.

All of the laboratory procedures listed previously are essential to the establishment of an accurate etiologic diagnosis, but histopathology is the most important single procedure. Histology provides confirmatory evidence of colibacillosis (villous colonization), *C. perfringens* type-C enteritis (villous necrosis with bacterial adherence), adenoviral enteritis (intranuclear inclusion bodies), coccidiosis (villous atrophy and developmental stages of the organism), cryptosporidiosis (organisms on the surface of epithelial cells), and strongyloidosis (characteristic inflammation plus demonstration of the parasite). Histology also enables assessment of the severity of villous atrophy produced by TGE, rotavirus, and other agents, even though these lesions are not pathognomonic for a specific agent. In addition, histology provides an estimation of the relative significance of each pathogen when two or more agents concurrently affect a piglet or litter.

An important function of diagnostic laboratories is to collect, summarize, and analyze data from a large number of cases of various diseases. This information is useful for designing sound preventive and control measures. Diagnostic laboratory data is of little value if it is incomplete. Therefore, producers and practitioners benefit when detailed case information and good quality specimens that allow comprehensive examination are submitted on each case.

## THERAPY AND PREVENTION

Medications for treatment of diarrhea generally fit one of three rationales. First, an antimicrobial compound may be selected to attack a known

pathogen such as *E. coli*. Second, a medication may be used for its prophylactic effects against a known pathogen, such as amprolium to prevent coccidial enteritis. The third purpose is nonspecific and includes many diverse preparations. Vitamins, protectorants, and antispasmodics are intended to enhance the well-being of the pig without regard to the specific disease entity present.

## Colibacillosis

Antibiotic administration of proper therapeutic doses is very effective against colibacillosis if the drug is given early in the course of the disease and if the organism is sensitive to the selected drug. Resistance to most commercially available antibiotics is common, and some antibiotics are nearly ineffective. Antibiotic sensitivity testing is a most useful clinical tool in drug selection and should be accomplished whenever possible.

Aminoglycoside antibiotics are extensively used in the treatment of colibacillosis. Streptomycin, neomycin, kanamycin, and gentamicin are the most often used, in order of increasing effectiveness. Parenteral or oral routes may be chosen for administration. Macrolide antibiotics, including tylosin and erythromycin, are useful when dealing with sensitive strains of the organism. Synthetic penicillins (ampicillin and amoxicillin) and potentiated sulfonamides such as sulfadiazine/trimethoprim are fairly effective against many strains of *E. coli*. Chloramphenicol is a highly effective compound, although resistance may be encountered in some herds.

Drugs formerly effective against *E. coli* include tetracycline, nitrofurans, sulfonamides, streptomycin, and neomycin. Cases will still be encountered in which these agents are effective, but in most cases they are of little value. Antibiograms for the nitrofurans often suggest bacterial sensitivity in vitro that cannot be demonstrated in vivo and should be considered when expected response to treatment does not occur. Bacterial sensitivity testing is quite accurate as a predictor of therapeutic drug value when the bacterial pathogen is isolated in pure culture and the etiology is uncomplicated. Failure to control colibacillosis with a drug to which the organism is sensitive should suggest that additional etiologic agents are present or that the pathogenic strain of *E. coli* was not isolated.

Antibiotics have little or no prophylactic value toward colibacillosis in suckling pigs.

Hygiene curtails the potential reservoir of most enteric disease agents and is a major part of prevention for any enteric disease. The sow is a less easily managed source of pathogens and may be a passive carrier of *E. coli*. Medication to limit shedding of *E. coli* by the sow is a popular but unproven effort in control of colibacillosis.

Immunization of the sow against specific pathogenic strains of *E. coli* enable passive transfer of immunity to piglets through colostrum and milk. Oral inoculation with large numbers of live pathogenic *E. coli* in late gestation has proved to be effective for the control of colibacillosis occurring in pigs less than five days old.[4] In later infections, protection from secretory immunity is not complete. In addition, these later infections commonly are complicated by concurrent infection with other enteropathogens.

The recent availability of pilus vaccines that are rich in K88, K99, and

987P antigens offers much hope for specific prevention of neonatal colibacillosis.[6]

### Viral Enteritis

Direct antiviral therapy is not attempted in piglets. In most instances, therapy for TGE and rotavirus infection is supportive. Fluid and electrolyte solutions are provided orally, and an antibiotic is administered if intercurrent bacterial infection is demonstrated.

Manipulations within a farrowing group may be possible and beneficial in some cases. Endemic TGE often affects some litters in a farrowing group while other sows continue to suckle normal litters. On the assumption that the normally suckling sows have a high level of TGE antibody in their milk, producers switch acutely affected scouring pigs to these sows and transfer their pigs elsewhere or wean them. This shuffling procedure appears to enhance survival.

Since antibody to TGE virus is secreted continuously in milk, any boost in the flow of milk may be beneficial therapy as a source of antibody and fluids. Increasing the amount and energy density of the lactation ration might help to increase milk flow and lessen the severity of the disease.

Immunity and prophylactic procedures for rotaviral diarrhea are less clearly understood than for TGE. The similarity in the intestinal lesions suggests that the same basic principles would apply to both diseases. Keeping the farrowing pens warm and dry to minimize heat loss from the piglets is helpful in reducing starvation due to malabsorption.

Federally licensed vaccines are available for TGE prophylaxis. Given to sows late in gestation, these live virus vaccines increase the serum and secretory antibody levels against TGE virus in the sow. These vaccines are not as efficacious in all cases as might be wished, yet they are used to economic benefit in many herds. The complexity of TGE pathogenesis and the efficacy record of the vaccines suggest that selective use is economically prudent. The producer and veterinarian should discuss and understand the limitations and costs associated with TGE vaccination.

Because of antigenic variations among the rotavirus and rotavirus-like group of agents, development of vaccines for these agents has been slowed by lack of sound data on the relative prevalence of different antigenic variants.

### *Clostridium perfringens* Type-C Enteritis

There is little benefit from treatment of pigs with signs of this disease because necrotizing enteritis usually is advanced when signs first appear.

Successful prophylaxis during an outbreak requires administration of type-C antitoxin as soon after birth as possible. Six- to eight-hour old piglets may have irreversible intestinal lesions, even though the pigs appear clinically normal at that time. Ideally, antitoxins should be administered within 30 minutes of birth. Dosage may be given orally, parenterally, or both. A common approach is to administer 2 ml orally and 2 ml subcutaneously to each newborn pig.

Immunization of the sow with *C. perfringens* type-C toxoid in late gestation is effective as a herd approach to prevention. Two doses at three-

week intervals are administered in primary vaccinations, with a single booster dose given prior to subsequent farrowings. Effectiveness of vaccination of sows depends upon prompt nursing because adequate protection requires the ingestion of colostral antibody shortly after birth.

**Coccidiosis**

Diarrhea appears several days after infection by *I. suis*. Therapy directed against coccidiosis may abbreviate the course of the disease and diminish severity but will not result in a rapid or complete response because villous damage is well advanced at the onset of diarrhea. Daily treatment for at least three days is required; five to seven days is a preferable treatment period.

Very little information is available on the relative therapeutic value of compounds now in use. Extrapolation of information on coccidiosis in other animal species has suggested experimental use of several compounds. The nitrofurans, sulfonamides, amprolium, and decoquinate are used most frequently. Secondary bacterial infections are more common in coccidiosis than in other types of atrophic enteritis; therefore, antibacterial therapy usually is helpful to prevent the development of necrotic enteritis from secondary bacterial infections.

Sources of infective oocysts include contaminated farrowing environment and sow feces. Anticoccidial agents are added to the terminal gestation diet and are continued through lactation in an effort to limit sow shedding of oocysts. Optimum dosage is unknown. Some regimens that have been tried experimentally include the feeding of decoquinate at a rate of 250 mg per sow per day, amprolium at a level of 10 mg per kg body weight, and nitrofurans at a level of 400 gm per ton in gestation diets and 200 gm per ton in lactation diets. Pending further investigation into the prophylaxis of coccidiosis, the experience of the clinician must guide drug selection.

The target animal in coccidiosis, the neonatal piglet, is difficult to medicate effectively on a daily basis. Experience with coccidiosis in other animal species would suggest that coccidiostats should be administered prior to and during the expected at-risk period. Including coccidiostats in oral iron preparations fed daily to pigs, adding coccidiostats to water supplies, and manual dosing of individual piglets has been accomplished with evident success in some herds.

Producers in one of our practice areas (S.C.H.) believe that most benefit has been gained by medicating pigs from birth through weaning with decoquinate in a peat moss–based iron product. Eight ounces of 6 per cent decoquinate are added to 25 lb of oral iron product. One large handful, approximately 1 oz, is fed daily to each litter from birth through three weeks of age. The effectiveness of this procedure has not been firmly documented, but an apparent decrease in the incidence of diarrhea has been observed.

Large numbers of infective oocysts can remain on surfaces of the farrowing area and equipment. Physical cleaning to remove as much organic matter as possible is followed by thorough rinsing. Application of household ammonia (8 oz per gal), Lysol (4 oz per gal), or sodium hypochlorite (5 per cent) reduces sporulation of *I. suis* oocysts.[12] Clean, dry farrowing areas

may be flamed lightly with a propane torch. Each of these procedures is intended to lower the potential infective dose of oocysts. The current lack of basic information on reservoirs and spread of the disease limits the development of more effective preventive programs.

## REFERENCES

1. Bohl, E. H., Saif, L. J., Theil, K. W., et al.: Porcine pararotavirus: Detection, differentiation from rotavirus, and pathogenesis in gnotobiotic pigs. J. Clin. Microbiol., *15*:312–319, 1982.
2. Coussement, W., Ducatelle, R., Charlier, G., et al.: Adenovirus enteritis in pigs. Am. J. Vet. Res., *42*:1905–1911, 1981.
3. Elazhary, M. A. S. Y., Morin, M., Derbyshire, J. B., et al.: The experimental infection of piglets with a porcine reovirus. Res. Vet. Sci., *25*:16–20, 1978.
4. Kohler, E. M., Cross, R. F., and Bohl, E. H.: Protection against neonatal enteric colibacillosis in pigs suckling orally vaccinated sows. Am. J. Vet. Res., *36*:757–764, 1975.
5. Moon, H. W.: Mechanisms in the pathogenesis of diarrhea: A review. J. Am. Vet. Med. Assoc., *172*:443–448, 1978.
6. Nagy, B., Moon, H. W., Isaacson, R. E., et al.: Immunization of suckling pigs against enteric enterotoxigenic *E. coli* infection by vaccinating dams with purified pili. Infect. Immun., *21*:269–274, 1978.
7. Pensaert, M. B., and deBouch, P.: A new coronavirus-like particle associated with diarrhea in swine. Arch. Virol., *58*:243–247, 1978.
8. Saif, L. J., Bohl, E. H., and Theil, K. W.: Rotavirus-like, calicivirus-like, and 23-nm virus-like particles associated with diarrhea in young pigs. J. Clin. Microbiol., *12*:105–111, 1980.
9. Stevenson, G. W., and Andrews, J. J.: Mucosal impression smears for diagnosis of piglet coccidiosis. Vet. Med. Small Anim. Clin., *77*:111–115, 1982.
10. Stewart, T. B., Stone, W. M., and Marti, O. G.: *Strongyloides ransomi:* Prenatal and transmammary infection of pigs of sequential litters from dams experimentally exposed as weanlings. Am. J. Vet. Res., *37*:541–544, 1976.
11. Stewart, W. C., Carbrey, E. A., Kresse, J. I., et al.: Infections of swine with pseudorabies virus and enteroviruses: Laboratory confirmation, clinical and epizootiologic features. J. Am. Vet. Med. Assoc., *165*:440–442, 1974.
12. Stuart, B. P., Bedell, D. M., and Lindsay, D. S.: Coccidiosis in swine: Effects of disinfectants on in vitro sporulation of *Isospora suis* oocysts. Vet. Med. Small Anim. Clin., *76*:1185–1186, 1981.
13. Stuart, B. P., Lindsay, D. S., Ernst, J. V., et al.: *Isospora suis* enteritis in piglets. Vet. Pathol., *17*:84–93, 1980.

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