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# DIAGNOSIS OF NEONATAL PIG DIARRHEA

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Despite improvements in management and veterinary care, gastroenteric disease remains a primary cause of neonatal mortality. Five common enteric pathogens in neonatal pigs are addressed in this article. All can be identified by appropriate diagnostic testing. Veterinary practitioners should understand how these common agents cause enteritis in neonates, how to recognize agents, and how to use appropriate management techniques to reduce economic losses.

## INTESTINAL PROTECTION MECHANISMS

Pigs have a number of protective mechanisms to prevent or control diarrhea. Nonpathogenic organisms rapidly populate the sterile intestinal environment after birth, competing with potential pathogens for epithelial attachment sites and nutrients.<sup>1</sup> This blocks successful colonization of many pathogens.<sup>1</sup> Immunoglobulins in colostrum and milk help in preventing pathogenic effects of infectious agents. Organisms also stimulate local immune responses by mucosal plasma cells. Once stimulated, plasma cells produce IgA and IgM, providing additional immunologic protection as early as 5 to 10 days after parturition.<sup>1</sup> Intestinal motility results in confinement of most organisms to the lower ileum and colon. It is important to note this mechanism, as this explains the necessity of sampling from these sites for diagnostic specimens.

Inflamed intestines secrete large amounts of fluid, which dilutes pathogenic bacteria or toxins. Damaged epithelial cells are replaced by

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proliferating crypt cells. Unfortunately in neonates, this replacement is slow until 10 to 14 days of age. Exposed surfaces are minimized by contraction of the lamina propria. Local interferon production occurs.<sup>1</sup> There is a remarkable capacity of the colon to absorb excessive fluid. These factors all provide prevention or control mechanisms for intestinal protection.<sup>1</sup>

## PATHOGENIC MECHANISMS OF DIARRHEA/CLINICAL SIGNS

General knowledge of pathogenic mechanisms of diarrhea should help formulate a differential diagnosis list (Table 1), and lead to the initiation of appropriate therapy and proper sampling for diagnostics and management techniques. Enteric pathogens have a limited number of mechanisms that cause diarrhea. Most common and important in the neonatal pig are: (1) toxic effects on epithelial cells causing fluid loss into the gut lumen, (2) direct destruction and loss of intestinal epithelial cells, and (3) local necrotizing effects on epithelium, lamina propria, or underlying tissues, with invasion of organisms into the lamina propria and underlying tissues.<sup>1</sup>

Alterations of intestinal epithelial cells by locally active enterotoxins results in an imbalance of intracellular electrolytes and fluids (secretory diarrhea). Lesions observed include dilated fluid-filled intestines and

**Table 1. NEONATAL PIG DIARRHEA ETIOLOGIES**

<b>Etiology</b>	<b>Age</b>	<b>Clinical Presentation</b>	<b>Fecal pH</b>	<b>Differentials</b>
<i>E. coli</i>	2 h to 4 d	Diarrhea/listlessness/ gaunt dehydration/ gilt litters	Alkaline (chyle in lacteals)	TGE/ <i>Clostridium</i>
<i>Clostridium perfringens</i> Type C and A	12 h to 7 d	Watery yellow scours advancing to bloody pasty scours; rapid death is frequent	Neutral	<i>E. coli</i> /TGE/ coccidiosis
TGE	3 to 7 d	Vomiting/ dehydration/ extreme thirst/ watery, yellow putrid diarrhea/ rapid infection in all litters	Acidic (chyle absent in lacteals)	<i>E. coli</i> /coccidiosis
Coccidiosis— <i>Isospora suis</i>	5 to 15 d (common 7–10)	Pasty to whitish diarrhea advancing to yellow, watery consistency/ gauntiness/rough haircoat	Acidic to neutral (chyle absent in lacteals)	<i>Clostridium</i> /TGE/ rotavirus
Enzootic TGE	10 d to weaning	Similar to acute TGE but typically less severe	Neutral to slightly acidic	Rotavirus/ Coccidiosis/ <i>E. coli</i>
Rotavirus	10 to 14 d (2- to 6-week- old animals more common)	Similar to enzootic TGE	Neutral to slightly acidic	Enzootic TGE/ <i>E. coli</i> / coccidiosis

pronounced mesenteric lacteals containing adequate to abundant chyle. Intestinal villi remain intact. Intestinal contents often have an alkaline pH.

Loss of intestinal epithelium with subsequent contraction of the lamina propria is recognized at necropsy by a thin-walled intestine and blunting of intestinal villi. Loss of absorptive surface and apical cells results in decreased absorption of ingesta (malabsorptive diarrhea). Undigested content is converted from lactose to lactate in the lumen by bacterial flora, resulting in an acid pH of content of the distal ileum and colon. Lactate, being an osmotically active molecule, draws fluid into the intestinal lumen, resulting in further potentiation of diarrhea. Loss of absorptive apical enterocytes results in poor absorption of other substances such as fat. Consequently, mesenteric lacteals are devoid of chyle or contain less than normal quantities.

Localized necrosis of the epithelium and underlying lamina propria initiates watery feces in early stages. Rapid progression of disease results in intestinal contents, which may contain fibrin, necrotic debris, and blood. Intestinal loops frequently are red to red-black and may be characterized by submucosal as well as mural emphysematous pocket formation.

## COLIBACILLOSIS

### Cause

Colibacillosis is the most common cause of enteric disease in neonatal swine.<sup>26</sup> Colibacillosis is caused by enterotoxigenic *Escherichia coli* (ETEC) strains. ETECs adhere to mucosal epithelium by pili. Pili associated with ETEC strains producing colibacillosis in pigs include F4 (K88), F5 (K99), F6 (987P), F14, and F18.<sup>3</sup> The combined presence of pili and enterotoxins Sta, STb, or LT enables pathogenic *E. coli* to adhere and multiply on intestinal mucosal surfaces.<sup>3, 12, 23, 27</sup> Secreted enterotoxins cause severe cellular biochemical alterations leading to clinical diarrhea, dehydration, and high mortality rates. Commonly isolated ETECs belong to O149, O8, O147, or O157, and are F4 positive.<sup>3</sup>

### Clinical Signs

Colibacillosis can involve three age groups of pigs: shortly after birth to 4-day-old, 3-week-old, and weaned pigs approximately 7 to 10 days after weaning. There is a high incidence of infection and mortality in 12-hour to 4-day-old pigs. Onset of signs is rapid, often occurring less than 12 hours postparturition, and may occur 2 hours after birth.<sup>26</sup> Listlessness and diarrhea are followed by dehydration, gauntness, and development of a rough haircoat. Perineal skin becomes wet and pasted with yellow fecal material. Marked perineal reddening may be noted. Death often occurs 12 to 24 hours after onset of diarrhea. Mortality can

reach 70% in affected litters.<sup>3</sup> Usually not all litters in a farrowing group are affected.

## Differential Diagnosis

A definitive differential diagnosis of colibacillosis requires laboratory confirmation of ETEC strains in feces or intestines of diarrheic pigs. Clinical presentation of colibacillosis and transmissible gastroenteritis (TGE) is similar in many respects. Some disease patterns can help in the clinical differentiation of these two diseases. Colibacillosis has a shorter age of onset (2–24 hours) than TGE (approximately 72 hours). Colibacillosis spreads slowly and typically does not affect all litters. Clinically, TGE spreads rapidly, affecting most litters in a short time. In colibacillosis litters from gilts are more frequently affected than litters from sows. Mortality in colibacillosis varies from 5% to 70%, whereas TGE mortality approaches 100% in pigs less than 7 days old.<sup>3, 21</sup> Sows or older pigs are not affected in colibacillosis but they are in acute outbreaks of TGE. By moistening a strip of pH test paper with feces at the anus, a presumptive diagnosis can be made on the basis of pH of diarrheic feces. A pH of 8.0 or higher is highly suggestive of colibacillosis. Fecal pH in viral or protozoal diarrheas is 7.0 or lower.

## Treatment

Numerous treatment regimens are available. Typically regimens are limited to injectable or occasionally oral antibiotics or sulfonamides, fluid replacement, oral dosage of cultures of *Lactobacillus acidophilus* milk, and increasing the farrowing house temperature.

## Control and Prevention

Control of colibacillosis is best accomplished by management practices designed to reduce the problem. The following practices are suggested.

1. Transfer the neonatal pig from the birth canal to the sow's teat for an early feeding of colostrum while reducing its exposure to disease-producing *E. coli*. Good sanitation, a clean sow, and a clean and disinfected farrowing house is mandatory.
2. Keep pigs warm, clean, dry, and out of drafts.
3. Vaccines containing pili from ETEC strains are available to immunize pregnant gilts and sows.<sup>18</sup> Preferably, gilts and sows should be vaccinated at midgestation and again approximately 3 weeks before farrowing. Some situations may indicate the use of Kohler milk vaccine, in which a live unit-specific strain of ETEC grown in milk is top-dressed on gestating sow's feed.<sup>7</sup> This stimulates a

good secretory antibody (IgA) response in the sow. Consequently, antibodies should be present in both colostrum and milk.<sup>7</sup> There is an inherent danger in using Kohler milk vaccine as neonates are exposed to live virulent ETEC. Sows must be dosed at least 3 weeks before farrowing to allow time for fecal shedding to diminish.<sup>7</sup> Sows should be dosed in a separate facility from farrowing areas and washed before being moved into farrowing crates.<sup>7</sup> As with all vaccines, efficacy is dependent on adequate milk production by the gilt or sow, as well as the ability of pigs to nurse and ingest sufficient quantities of immunoglobulin.<sup>7</sup>

## **TRANSMISSIBLE GASTROENTERITIS**

### **Cause**

Transmissible gastroenteritis is caused by a coronavirus. The disease was first described in 1946.<sup>21</sup> Since then, TGE has been reported in most of the major swine-producing countries. TGE is highly contagious.<sup>21</sup> Producers are apprehensive about TGE because (1) mortality in the neonate is high, (2) there is no effective practical treatment, (3) entrance of the virus into a herd is difficult to prevent because of the role of birds, especially starlings, and (4) commercial vaccines available are of limited effectiveness.<sup>4, 16, 17, 21</sup>

### **Clinical Signs: Acute or Epizootic TGE**

Acute TGE is generally first observed in growing and finishing pigs on the farm. Pigs in these age groups exhibit generally mild clinical signs of vomiting and diarrhea. Following infection of older pigs, TGE often appears explosively in the farrowing house, with most neonatal pigs becoming ill within a 24- to 48-hour period. Initial signs in neonates are roughening of haircoat, shivering, dehydration, vomiting, diarrhea, refusal to nurse, and extreme thirst. Neonatal pigs often attempt to drink water from any source and squeal weakly when handled. This is followed closely by a severe, watery diarrhea that has an acrid odor characteristic of sour milk. Pigs become dehydrated, weak, and die within 2 to 5 days. The mortality rate of pigs under 7 days of age during acute TGE breaks approaches 100%. Mortality is lower in older nursing pigs but can be influenced by stress such as chilling, dampness, and secondary bacterial infection. Lactating sows are susceptible during acute TGE. Sows may vomit, develop severe diarrhea, become anorexic, and cease lactation.

TGE infection rate approaches 100% in weaned pigs. Pigs show all the clinical signs of TGE that are evident in nursing pigs. The mortality rate, however, is greatly reduced as pigs are more mature and have more stored energy. Stress factors again may influence the morbidity and mortality. Older growing and finishing pigs, gilts, gestating sows,

and boars in the breeding herd can become infected and frequently show inappetence or only a few clinical signs.

Even in today's management environment TGE occurs more frequently during winter to early spring months (December to April). In continuous farrowing operations, however, TGE can occur in any season and can affect each subsequent farrowing for several months. TGE virus has been found in feces of pigs 8 weeks after apparent recovery from infection. TGE virus has been found in feces of starlings up to 32 hours after they were fed the virus.<sup>20, 21</sup> Birds carrying the virus have been responsible for contamination of facilities and feed resulting in acute outbreaks in negative units.

### **Differential Diagnosis**

Diagnosis of TGE is by history, clinical signs, and postmortem examination. Postmortem, the stomach usually contains curdled milk. Small intestinal walls are thin and fluid-filled. Mesenteric lacteals often are devoid of chyle (malabsorptive diarrhea). Villi in the lower two thirds of the intestine are shortened. Intestinal content is watery, yellow, has flecks of curdled milk, and is malodorous.

TGE must be differentiated from colibacillosis as described. Diagnostic laboratories can determine the presence of TGE virus in the intestine of pigs in acute stages of diarrhea. Samples taken after 24 hours of diarrhea may yield negative results due to loss of virus by dilution of intestinal contents.

### **Chronic or Enzootic TGE**

Enzootic (endemic) TGE is a persistent form of the disease in which carrier pigs continuously shed virus and infect susceptible pigs. Enzootic TGE is found in those herds with multiple or continuous farrowings so that susceptible pigs are present at all times. TGE has been reported to occur in these herds in pigs as young as 10 days of age, although 10 days to weaning appears to be the most common age of infection.

Enzootic TGE has many of the clinical signs seen in acute TGE, but they are less severe in older pigs. Consequently, it is much more difficult to differentiate between enzootic TGE, rotaviral, and *E. coli* infections. Mild villous atrophy can be found in both rotaviral and enzootic TGE infections. Proper laboratory samples and diagnostic tests can distinguish between the two diseases. Dual or multiple pathogen infections are common.

### **Treatment**

When lactating sows have TGE, their milk production is greatly reduced. Their young pigs also become infected and resulting dehydra-

tion is compounded by lack of fluid intake caused by the sow's reduced milk production. Fluid replacement therapy and use of antibiotics to reduce effects of secondary bacterial pathogens are necessary.

## Control

Avoiding exposure of pigs to TGE virus by isolation of swine units has proved to be highly effective in preventing infection; however, this has left units open to wide-spread severe acute TGE breaks, particularly in early weaned nurseries. Rigid isolation of units from dogs, foxes, starlings, and cats is imperative. These animals have been shown to be mechanical vectors of TGE virus for periods up to 14 days.<sup>9, 11, 20, 28</sup> They are not believed to be natural reservoirs involved in maintenance of the virus through seasonal periods.<sup>9, 11, 20, 28</sup> Feeder pigs that may be carriers should not be brought onto smaller units and farms just before or during the farrowing season.

All swine producers should practice some of the rules for specific pathogen-free (SPF) programs. These are (1) visitors are not allowed into the swine facility without shower-in-shower-out or at the minimum clean boots and outer garments; (2) trucks and truckers are not allowed near the main breeding herd; (3) new additions to swine herds are isolated a minimum of 60 days before introduction and evaluated serologically for incidence of TGE, porcine respiratory coronavirus and other common swine pathogens; (4) movement of dogs, wild animals, and birds are controlled. Modified live virus TGE vaccines are available commercially, although their efficacy is suspect. Maintenance of negative units is far more efficient.

Eradication of TGE virus from a herd without depopulation is possible. Eradication is based on the assumption that infected pigs usually do not shed virus longer than 2 weeks.<sup>7</sup> Initially, the herd must be closed with any needed replacements introduced before initiating eradication and should remain closed for at least 6 months.<sup>7</sup> Herd immunity is then brought into equilibrium by dosing all animals deliberately with virulent virus by infecting neonates and feeding back intestinal homogenates to adults. Strict sanitation and all-in, all-out movement is required to eliminate virus in the environment.<sup>7</sup> Approximately 3 months after whole herd dosing, seronegative sentinel animals are introduced.<sup>7</sup> Providing these animals remain seronegative after 30- and 60-day tests, successful eradication likely has been accomplished.<sup>7, 10</sup>

## ROTAVIRUS DIARRHEA

### Cause

Rotaviruses are an important cause of diarrhea in swine. Swine rotavirus is ubiquitous and herd infection rates approach 100%.<sup>7</sup>



## Clinical Signs

Rotaviral infections in pigs have an incubation between 2 and 4 days, depending on virulence of the virus strain, age of the pig, immune status of the sow, and most importantly environmental and management conditions. Field outbreaks of diarrhea can be observed in neonatal pigs but are found more commonly in 2- to 6-week-old animals, as most sows provide degrees of colostrum protection. Rotaviral infections limited to neonatal pigs are discussed briefly in this section as a potential differential and discussed further in the article on nursery to grow-finish age animals.

Infected pigs become anorexic and depressed a few hours before onset of diarrhea. Vomiting has been observed but is not as common a clinical sign as with TGE. Adult animals generally do not exhibit clinical signs.

Diarrhea can be quite severe starting with watery to creamy feces and changing rapidly to profuse yellow to green liquid feces. Diarrhea can last for up to 10 days. Return to normal feces is gradual and may take 1 to 2 additional weeks after recovery from a severe infection. Dehydration is more evident in younger pigs and in those with prolonged diarrheic periods. Morbidity generally is higher than 80%.<sup>19</sup> Mortality can reach 20% in infected animals, although it is often higher in younger pigs.<sup>19</sup> Rotavirus is likely synergistic with other enteric pathogens including *E. coli*, *C. perfringens*, *Isospora suis*, and TGE virus.

## Differential Diagnosis

Diagnosis of rotaviral infection in swine presents some specific problems. Clinical signs of rotavirus and TGE virus infections in pigs are similar especially in cases of enzootic TGE. Generally, villous atrophy produced by TGE is quite severe, but in enzootic TGE, the mild degree of villous atrophy closely resembles that of rotavirus infections making it difficult to separate the two conditions on that basis alone. A number of tools are available in diagnostic laboratories to differentiate the two disease entities, including immunohistochemistry, ELISA tests, fluorescent antibody tests, direct electron microscopic examination, and *in situ* hybridization as well as dot and slot hybridization.<sup>22</sup>

## Treatment

Fluid therapy; antibiotics; a warm, draft-free environment; and vitamins A, C, and E as indicated for TGE are recommended.

## Control

Good sanitation practices can control the risk of infection with rotavirus as with other viruses or bacteria. Immunity to rotavirus in

swine follows the same pattern as that of TGE virus infections. Vaccines are available commercially for the immunization of pregnant gilts, sows, and nursing pigs.

## **CLOSTRIDIUM PERFRINGENS ENTERITIS**

### **Cause**

The bacteria *C. perfringens* types C and A cause clostridial enteritis. Type A infections are now recognized with increasing frequency in neonatal pigs.<sup>24</sup> Pathogenesis of *C. perfringens* infections is mediated by one or more of nearly 20 potent exotoxins.<sup>24</sup>

### **Clinical Signs**

Clostridial enteritis affects young pigs and is reported worldwide. Other names for clostridial enteritis are enterotoxemia, hemorrhagic enteritis, and bloody scours. The disease usually affects pigs during the first week of life. Nursing pigs up to 1 month of age can be affected.

Diarrhea typically begins as a watery yellow scours that may contain traces of blood. After a few hours feces become bloody, and pigs may die within a few hours to 2 days.

### **Differential Diagnosis**

Diagnosis is determined by history, clinical signs, postmortem examination, and laboratory assistance in which causative bacteria are identified. Acute disease is characterized by localized epithelial and underlying lamina propria necrosis inducing watery feces, which rapidly progress to content containing fibrin, necrotic debris, and blood. Intestinal loops are frequently red to red-black and may be characterized by emphysematous pocket formation in the wall. Chronic cases exhibit necrotic enteritis that is difficult to distinguish from other chronic enteropathies, particularly coccidiosis.<sup>7</sup>

### **Treatment**

Treatment is not effective after clinical signs of clostridial enteritis appear. Administration of type C antitoxin or autogenous type A antitoxin may help in acute and subacute cases.<sup>7, 24</sup>

## Prevention

Injection of type C antitoxin in newborn pigs as soon after birth as possible aids in prevention. This disease can be prevented or the severity reduced in future farrowings by giving sows type C toxoid 10 weeks before farrowing and repeating the injection 3 weeks before farrowing. Presumably, autogenous type A toxoid injection provides similar prevention.<sup>24</sup> Most important is adherence to good sanitation, particularly in regard to cleaning of the sow prior to placement in the crate. Use of prophylactic antibiotics including bacitracin in sow feed beginning 2 weeks prior to farrowing as well as a prophylactic dose of penicillin or amoxicillin given to pigs at birth in units previously diagnosed can be considered.<sup>7</sup>

## COCCIDIOSIS

### Cause

*I. suis* is the only coccidian associated with clinical disease in neonatal pigs. Coccidiosis is a major problem in systems with continuous farrowing and poor sanitation. Infection is spread by scouring pigs. Sporulated oocysts are subsequently ingested by other pigs, thus perpetuating the cycle. Organisms infect and destroy apical enterocytes resulting in villus atrophy (malabsorptive diarrhea).

### Clinical Signs

Coccidiosis affects pigs from 5 to 15 days of age, with the most common range being 7 to 10 days of age. Pigs are often gaunt and dehydrated. Diarrheic feces range in appearance from yellow to gray-green and watery to pasty without blood. Fecal pH is usually acidic (malabsorptive diarrhea). The condition is unresponsive to antibiotics. Morbidity and mortality are variable and often influenced by the presence of secondary bacterial pathogens.<sup>6</sup>

### Differential Diagnosis

Diagnosis of coccidiosis is based on history, clinical signs, and postmortem examination. Cases are often randomly distributed within the farrowing unit. Typically, there is no distinctive difference between gilt and sow litters affected. Fecal flotation for coccidia is often unrewarding as oocysts are not detected until 5 to 9 days after infection. Walls of the ileum and distal jejunum are thickened. Mesenteric lacteals are often segmentally devoid of chyle. A mild to severe fibrinonecrotic enteritis usually limited to the ileum and jejunum may be evident.

Impression smears of mucosal scrapings and microscopic examination should be used to confirm clinical impressions. Laboratory diagnostics are needed in many cases of coccidiosis to identify associated secondary pathogens as well as to differentiate the disease from chronic *C. perfringens*, enzootic TGE, and rotaviral infections.<sup>6</sup>

## **Treatment**

No coccidostats are approved for use in swine. Extralabel uses should be considered with caution and with adherence to appropriate guidelines.

## **Prevention**

Control of coccidiosis is best accomplished by management practices to reduce exposure. Measures should include all-in, all-out with close adherence to thorough sanitation of farrowing crates. Particular attention should be paid to removal of organic matter. Disinfection with 5% chlorine bleach or quaternary ammonia compounds have increased efficacy. Use of raised woven wire or expanded metal crates tend to reduce the incidence but do not replace sanitation practices.

## **GENERAL THERAPY**

### **Antimicrobial Therapy**

Antibiotic therapy continues to be the most widely used approach for diarrhea in neonatal pigs. Obviously the basic theory is to kill or inhibit offending organisms. Unfortunately, there are many situations in which this approach does not work. These include (1) viral or protozoal etiologies, (2) when rapid reinfection of offending bacterial agents occurs due to environmental contamination, (3) when intestinal damage is severe before antibiotic therapy (e.g., *C. perfringens* type C infections), (4) when killed organisms release endotoxins as they die, which subsequently harms or kills the pig and, (5) when offending organisms are resistant to antibiotics used.<sup>1</sup> Appropriate antibiotic therapy requires that bacteria be isolated and antimicrobial sensitivities performed routinely.

### **Competitive Colonization**

Attempts at populating the neonatal pig with high numbers of nonpathogenic organisms that compete with colonization of pathogenic bacteria have met with variable success.<sup>1</sup> Oral dosing of a high number of organisms such as *Lactobacillus spp.*, enterococci, and nonpathogenic

*E. coli* has been used. Using antibiotics in combination with this type of program is counterproductive.<sup>1</sup> Labor intensity often limits its application in large units if supplemental fluid feeding lines are not in place.

### **Physiologic Therapy**

Therapeutic compensation for diarrhea includes rehydration of animals with electrolytes and decreasing ingestion of osmotically active agents. Subcutaneous and intraperitoneal fluid replacement are the only practical routes available in neonatal pigs. While weaning pigs early should in theory reduce the effect of osmotically induced diarrhea, the unfortunate consequence is hypoglycemia and death in pigs less than 2 to 3 weeks of age because of starvation. Concurrently, intestinal repair mechanisms may be impaired so epithelial continuity is not adequately restored before death. Intensity of this therapy option, particularly in today's management systems, typically yields disappointing results.<sup>1</sup>

### **Immunologic Prevention/Therapy**

Immunologic prevention of neonatal pig diarrhea includes endeavors to impede (1) adherence of bacteria or viruses to intestinal epithelium, (2) effects of secretory toxins, (3) penetration of epithelium by bacteria or viruses, and (4) invasion of the lamina propria and subsequently the entire system by organisms.<sup>1</sup> These approaches are open to certain problems including improper selection of organisms for use as an antigen, choosing the proper antigen to ensure preventive effects, route of administration to obtain desired effects, and antibody loss through normal decay and degradation by intestinal proteolytic enzymes.<sup>1</sup>

### **Protocol for Swine Diarrhea Evaluation**

Diagnosis of preweaning diarrhea in swine continues to be a part of day-to-day swine practice regardless of the management system or antibiotic availability. Often a broader approach to diagnosis beyond simple bacterial culture and a few snips of intestine for histopathology is required. The following is an attempt to streamline an approach to swine diarrhea diagnostics with the acknowledgment that situations or conditions can vary. Seldom are disease processes singular in any given unit. Evaluation often requires tailoring to specific situations in which case discussion with a diagnostic pathologist is encouraged to provide a source of appropriate tests for you and your client's needs.

## Tissue Submission

Samples should be selected from sacrificed untreated animals in acute stages of disease or animals dead for a short period of time (usually less than 3 hours). Submitting live animals to a diagnostic laboratory for a full evaluation may be ideal in some situations. When submitting live animals, attempt to provide a cross-section of clinical presentations. Animals in the acute stage to animals in more advanced stages that have not undergone antimicrobial therapy should be included. Additionally, providing diagnosticians an accurate verbal or written history allows for timely, efficient, and informed decisions on tests as well as approaches to providing solutions.

Having appropriate containers for 10% buffered neutral formalin and fresh tissue samples available that are permanently and properly identified before necropsy may seem elementary; however, it provides for a good deal less work in the long run.

Collection of blood samples from euthanatized animals for potential testing is always a good idea. Ideally, samples of solid organs for bacteriology should be the first obtained. *Fresh lung should be obtained first and packaged separately from other tissues. Fresh samples of liver, kidney, and spleen, should they be warranted, can be packaged together. Samples of intestine should be packaged together and never mixed with other tissue samples. Enteric contents for electron microscopic examination should be placed in a well-sealed container without addition of preservatives.* Tissues collected for histopathology should be no greater than 0.25-inch thick and placed in approximately 9 parts 10% buffered neutral formalin to 1 part tissue. Double packaging of formalin-fixed tissues is recommended to reduce risks of leakage that may subsequently render submissions worthless because of drying. Table 2 provides a brief outline of samples for swine diarrhea evaluations.

## Serology

Although serology for enteric pathogens is not commonly necessitated in swine, it is often imperative in modern management systems for definitive evaluation of exposure to TGE virus. Screening tests used by most laboratories for TGE serology do not differentiate between antibody to TGE virus and porcine respiratory coronavirus. Differential serologic tests as yet are not widely available but can provide a rapid means, in conjunction with other diagnostic tests, to ascertain exposure to TGE virus.

## Prevention

The complex nature of neonatal pig diarrhea in conjunction with many of today's management systems makes the expectation of total

**Table 2.** NEONATAL SWINE DIARRHEA DIAGNOSTIC SAMPLE SUBMISSION

<b>Histopathology/ Immunohistochemistry (Formalin-flushed Unopened 1.5- to 2.0- cm Sections)</b>	<b>Bacteriology</b>	<b>Fluorescent Antibody</b>	<b>Electron Microscopy</b>
Duodenum: 1 section Jejunum: 2-4 sections Ileum: 3-6 sections Colon: 2 sections opened Sections of other tissues as indicated by clinical signs and gross lesions.	Ileum and mid-jejunum: fresh chilled 10- to 15- cm segments Cecum: preferably the tail of the cecum tied off Colon: 2 to 3 loops with mesenteric attachment intact	Ileum and lower jejunum: 3-6 approximately 5-cm segments rotavirus and coronavirus [TGE]	At least 10 mL of fresh cecal and ileal content in a well-sealed container
Immunohistochemical tests for TGE, rotavirus as well as other enteric pathogens can be conducted on formalin-fixed samples often in lieu of or as further conformation for fluorescent antibody tests.	Mesenteric lymph node Liver (in separate container)		

prevention unrealistic. Control at an economic level should be the major objective. Obviously, the occurrence of clinical disease and mortality is dependent on a balance between environmental contamination, degree of colostral immunity, and other resistance factors present in the neonatal pig.

Specific management suggestions are difficult because of differences in unit size, facilities, and labor. Broad management principles are most applicable.

1. Increase specific resistance of the neonate by vaccination of the dam or neonate.
2. Provide maximum nonspecific resistance with adequate colostrum and optimal animal husbandry practice.
3. Reduce the degree of exposure of neonates to infectious agents and to a stressful environment.

Unfortunately there is no vaccination protocol for poor management. Extensive environmental contamination continually exposes neonates to pathogens and provides a significant degree of stress.

Chilling is directly responsible for many of the deaths during the first postparturient day and increases susceptibility to disease.<sup>2, 8</sup> Environmental temperature can significantly affect various aspects contributing to health and development of pigs such as immunophysiology and nutrition.<sup>13, 14, 15</sup> These effects are even more pronounced in neonates owing to their limited ability to cope with thermal extremes.<sup>5, 15</sup> Further stress is often induced by inconsistencies in temperature where improper

thermostatic control or ventilation (drafts) results in undulating temperatures.

Environmental temperatures can have a significant impact on the response of neonatal pigs exposed to bacterial endotoxin.<sup>15</sup> Neonatal dependence on a controlled thermal environment increases disease susceptibility and mortality in neonatal pigs housed at cool temperatures. Diarrhea reduces the neonatal pig's ability to maintain its rectal and surface temperature within a cold environment.<sup>15</sup>

Behavioral responses of diarrheic pigs tend toward moving to cooler environments for unknown reasons. This gravitation toward cooler thermal zones occurs despite obvious negative physiologic effects.<sup>2</sup> Consequently, diarrheic neonatal pigs require even more attention and care in terms of thermal environment than their healthy counterparts.<sup>2</sup> Unit personnel should be trained to understand these behavioral responses of the neonate to provide efficient and effective care in an effort to reduce mortality.

## SUMMARY

To be effective, swine practitioners should develop a unit health program.<sup>25</sup> Development should involve unit managers, owners, and employees involved in day-to-day operations. Emphasis on training personnel and management to reduce disease and collection of accurate records is necessary.

Routine diagnostics are needed to solve disease problems. Communication with laboratory personnel to ascertain what samples are needed for diagnosis of particular problems cannot be overemphasized. General diagnosis of disease problems outlined by Vinson<sup>25</sup> can be similarly followed within the specifics of diarrheal problems within units.

1. Observe symptoms exhibited by pigs, i.e., huddling, fecal material around perineum, extreme thirst, etc.
2. Evaluate the degree of morbidity and potential production losses.
3. Analyze possible specific causes of symptoms, i.e., environmental cleanliness, affected litter distribution, age of affected neonates, and other populations affected.
4. Examine live animals, i.e., obtain serum samples from a random population, take rectal temperatures of affected neonates, and evaluate fecal pH.
5. Necropsy dead or dying pigs [that] appear to represent the problem.
6. Submit live pigs or appropriate tissues from necropsied pigs to a diagnostic laboratory.
7. Re-evaluate environmental conditions that may be contributing to the problem (remember, unit employees are a part of the pigs' environment).



8. Evaluate management procedures contributing to the disease problem, i.e., lack of adherence to all-in all-out, rapid turn-around decreasing cleaning time etc.<sup>25</sup>

Following this format and communicating with diagnosticians should provide for positive results for the producers both entities serve.

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