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# Foal Diarrhea

## Established and Postulated Causes, Prevention, Diagnostics, and Treatments

Olimpo Oliver-Espinosa, DVM, MSc, DVSc

**KEYWORDS**

• Foal • Infections • Enterocolitis • Diagnosis • Therapy

**KEY POINTS**

- Diarrhea is one of the most important diseases in young foals and may occur in more than half of foals until weaning age.
- Several infectious and noninfectious underlying causes have been implicated but scientific evidence of pathogenesis is constantly evolving.
- Practically it is important to investigate all known different potential causes and to identify infectious agents to avoid outbreaks, as well as to evaluate in individuals the level of systemic compromise and establish an adequate therapy.
- In addition it is crucial to differentiate foals that can be managed in field conditions from those who should be sent to a referral center.
- This article reviews these aspects and recent developments in the diagnostic and therapeutic approaches.

### INTRODUCTION

Diarrhea is defined as increased frequency of defecation with increased water content in feces.[1] Foals that develop diarrhea usually have enteritis, which is associated with systemic inflammatory response syndrome (SIRS).[2] More than 50% of foals experience 1 or more bouts of diarrhea in the first 6 months of life.[3–5] There are established and putative infectious and noninfectious causes of foal diarrhea. Treatment is mainly symptomatic but some specific treatments of various causes are available.

### PATIENT HISTORY

Vaccination and worming status, information on number and ages of the affected foals, and hygienic conditions on the farm are important.[6,7] Failure of passive transfer of immunity might be reported.[8] Diarrhea commonly occurs within the first 6 months of

Department of Animal Health, National University of Colombia, Carrera 45 # 26-85, Edif. Uriel Gutiérrez, Bogotá D.C., Colombia
*E-mail address:* ojolivere@unal.edu.co

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life. Depending on the etiology, outbreaks can occur, or single foals might be affected.[9,10] Risk factors include suboptimal deworming of mares and extensive use of antibiotics.[4] Additional observations can be depression, decreased suckling reflex, weakness, colic, weight loss, and occasionally sudden death.[8]

## CLINICAL PRESENTATION

Clinical signs vary.[6,7] Initial signs may include colic, hypermotility or amotility, and abdominal distension. Fecal consistency can range from watery to pasty, with different colors, and may contain blood or casts.[7] Foals tend to dehydrate quickly and can reach severe dehydration evidenced by sunken eyeballs and a prolonged skin tent. Depression, weakness, anorexia, salivation, and bruxism are frequently observed. Body temperature varies from fever to hypothermia.[2] Cases of enteritis commonly have signs of SIRS, including congested mucous membranes, tachycardia, weak pulses, tachypnea, and cool extremities.[2,7] When signs progress, recumbence, coma, and death may occur.[7] Up to 50% of diarrheic foals less than 30 days of age with diarrhea are bacteremic.[11,12]

## CLINICAL PATHOLOGY

If dehydration is present, hematocrit is high.[13] Signs of inflammation are present in the leukogram, including leukopenia or leukocytosis, commonly due to abnormal neutrophil counts. A left shift might also be present, particularly in cases with SIRS.[11] Plasma total proteins can be increased or decreased, as in cases with sepsis, protein-losing inflammation (salmonellosis), or *Lawsonia intracellularis* infections.[14,15] Acute phase proteins, such as fibrinogen and serum amyloid A, are usually elevated.[13]

Electrolyte derangements are common and should be evaluated. Up to 66% of the foals have been reported as hyponatremic and 33% hypokalemic. Hyperchloremia or hypochloremia also can occur.[13] Foals with diarrhea usually have a strong ion metabolic acidosis with low blood pH, low plasma bicarbonate levels, and a decreased strong ion difference (strong ion difference = Na + K-Cl lactate = 36–44) due to electrolyte derangements.[11,13] Increased lactate concentrations are also common and contribute to the low SID.

Urea and creatinine levels are often increased, indicating a prerenal and occasionally also a renal azotemia.[11]

## ETIOLOGY

Noninfectious causes include foal heat diarrhea, perinatal asphyxia syndrome, necrotizing enterocolitis (NEC), dietary imbalance, equine gastric ulcer syndrome, luminal irritant diarrhea, and secondary lactose intolerance.[16,17]

The most common infectious agents include rotavirus (RV), *Clostridium perfringens* types A and C, *Salmonella* spp, *C difficile*, *Cryptosporidia*, and *L intracellularis*.[2,3,6,11,15,17] Less common causes are *Coronavirus*, *Rhodococcus equi*, and *Strongyloides westeri*.[18–20] Single case reports have also been published on *Aeromona hydrophyla*,[21] *Neorickettsia risticii*,[22] candida[23] and *Listeria monocytogenes*.[24] Coinfections can occur,[22] as with *C perfringens* and *C difficile*.[25]

### *Foal Heat Diarrhea*

Foal heat diarrhea is usually a self-limiting condition in foals aged 5 days to 15 days. It occurs in 75% to 80% of neonatal foals and usually lasts 3 days to 4 days.[16] These foals have diarrhea, show no signs of systemic disease, and continue to suckle

well.[2] Individual foals may be affected by more severe and prolonged diarrheic episodes.[16] The etiology remains speculative and include changes in mare's milk composition during foal heat; however, orphan foals on milk replacer developed diarrhea at similar ages, making this theory less likely.[26] More recently, maturational changes in bacterial intestinal flora during that early life period have been elucidated and this adaptation of the microbiota may lead to foal heat diarrhea.[27]

### *Rotavirus*

RVs are currently the most frequently detected infectious agents in foals with diarrhea.[11,22] In a study using real-time polymerase chain reaction (PCR), RV was responsible for 35% to 90% of cases up to 3 months of age.[11] RV belongs to the Reoviridae, subfamily Sedoreovirinae, genus *Rotavirus*. Only group A has been associated with horse infection.[28,29] RVs tend to cause outbreaks, have an ubiquitous world distribution in the horse population, are highly contagious, replicate rapidly, and are shed in high concentrations in feces of affected animals.[2,30] Transmission is via the feco-oral route of contaminated feces or fomites. The incubation period is 1 day to 4 days.[28] Virus shedding can start before the onset of diarrhea, persists during the clinical phase, and may persist for up to12 days post resolution of diarrhea.[31] Foals with RV diarrhea presented to a referral hospital had a survival rate of 94%.[11]

RV diarrhea is primarily malabsorptive but also includes a toxin-mediated secretory component.[32] It preferentially infects the mature absorptive villous enterocytes of duodenum, jejunum, and ileum-sparing crypts, causing villous damage resulting in malabsorptive diarrhea.[32] Foals often become lactose intolerant because lactase is mainly produced by the brush border epithelium of the villous enterocyte. The RV nonstructural glycoprotein 4 (NSP4) is a viral enterotoxin, responsible for the hypersecretory component of RV diarrhea.[33]

### *Clostridium perfringens*

*C perfringens* is a gram-positive anaerobic spore-forming bacterium, associated with enterocolitis in foals and horses.[30,34] *C perfringens* was long considered part of normal microbiota of the large colon, but recent studies found low prevalence (0%–8%) in feces of horses.[35,36] In neonatal foals *C perfringens* was isolated from the feces of 90% of 3-day-old foals but isolation rates decreased over the first weeks of life.[37] The most common genotype identified (85%) is type A; *C perfringens* type C was isolated in less than 1% to 3% of neonatal foals.[37,38] In a retrospective study, *C perfringens* was the second most significant organism isolated from foals with diarrhea.[11] In some instances, *C perfringens* may cause sudden death without prior clinical manifestations. Foal diarrhea associated with *C perfringens* occurs commonly early in life; 96% of affected foals had adequate transfer of passive immunity.[34] Diarrheic foals less than 1 month of age were significantly more likely to be positive for *C perfringens* (odds ratio [OR] 15; 95% CI, 3.5–66) compared with older foals.[11] Reported mortality rates vary from 12% to 83%.[11,22] The highest mortality has been reported for infection with *C perfringens* type C.[34]

*C perfringens* type A produces $\alpha$ toxin; type B produces $\alpha$, $\beta$, and $\varepsilon$ toxins; type C produces $\alpha$ and $\beta$ toxins; type D produces $\alpha$ and $\varepsilon$ toxins; and type E produces $\alpha$ and $\iota$ toxins.[35] Additional virulence factors include $\beta$2 toxin,[39] an enterotoxin produced by all *C perfringens* but most commonly by type A.[2,35] Recently, an additional toxin named *netF* has been identified in foals with NEC and in vitro cell cytotoxicity has been proved.[40] The role of these additional virulence factors in pathogenesis of diarrhea is still debated.

### *Salmonellosis*

*Salmonella* spp are rod-shaped, gram-negative Enterobacteriaceae.[41] There are currently more than 2200 serovars in 8 subspecies, with subspecies I responsible for 99% of clinical mammalian diseases.[41] The most frequent *Salmonella* serovars isolated from horses in the United States include Typhimurium, Newport, Anatum, Javiana, and Agona.[41] Transmission mainly occurs via the feco-oral route.[42] Diarrheic foals older than 1 month of age were significantly more likely to have fecal *Salmonella* isolated than younger foals (OR 2.6; 95% CI, 1.2–6.0).[11]

*Salmonella* colonizes the intestine using the invasion-associated type III secretion system causing inflammation and massive mucosal damage of the ileum and colon.[42]

### *Clostridium difficile*

*C difficile* is a gram-positive, anaerobic, spore-forming bacterium commonly associated with diarrhea and colitis in humans and other mammals.[35] It is considered among the most important agents of enteric disease of foals and adult horses.[11,17] *C difficile* enterocolitis in foals has been reproduced experimentally.[43] *C difficile* has been isolated from healthy and diarrheic foals and adult horses.[44] Carrier rates in healthy foals are higher at 2 weeks of age compared with older foals.[38] It has been isolated in 11% of diarrheic foals.[38] There have been limited studies on risk factors, but antibiotic administration and hospitalization seem the most important ones.[44] Transmission occurs via fecal oral route.

The 2 major toxins, toxin A (TcdA) an enterotoxin and toxin B (TcdB) a cytotoxin,[45] are responsible for causing increased paracellular permeability of mucosal surfaces, cell rounding, and eventually cell death or apoptosis.[45] The toxins also cause inflammation resulting in increased fluid exudation and mucosal damage, leading to diarrhea or pseudomembranous colitis.[45]

## *Less Common Infectious and Parasitic Agents*

### *Coronavirus*

Equine coronavirus is considered an enteropathogen in foals.[11,18] The prevalence rates of equine coronavirus in healthy and diarrheic foals in central Kentucky were 27% and 29%, respectively.[22] The importance of this agent is not known.

### *Cryptosporidium*

*Cryptosporidium* spp can infect adult horses and foals, *Crystosporidium parvum* is the most common genotype identified.[46] Infection occurs with ingested oocysts that reach the ileum. It affects mainly foals with severe combined immunodeficiency (SCID), but outbreaks in immune-competent foals have also been reported.[47] Self-limiting diarrhea can occur for up to 8 days and in older foals; chronic and intermittent diarrhea may also occur.[46]

### *Lawsonia intracellularis*

*L intracellularis* is a gram-negative obligate intracellular bacterium affecting mainly foals aged 4 months to 7 months (range 2–13 months) causing proliferative enteropathy.[48] In 2 recent studies using fecal PCR in healthy and diarrheic foals, it was not found in foals less than 4 month of age.[11,22] It is transmitted via the feco-oral route.[48]

### *Rhodococcus equi*

*R equi*, a gram-positive facultative intracellular pathogen, is a common etiologic agent in foal pneumonia.[49] It has been reported that up to 50% of foals with *R equi* bronchopneumonia also had intestinal lesions on necropsy, and 4% of foals had intestinal lesions without pneumonia.[49]

### *Sepsis in foals*

Diarrhea is common in septic foals, due to hemodynamic alterations leading to gastrointestinal (GI) mucosal hypoperfusion, inflammatory mediators associated with SIRS, and dysmotility.[2]

### *Other Infectious Agents*

The role of enterotoxigenic *Escherichia coli* in foal diarrhea has been suggested, but there is only 1 report of a single case where it was proposed as causative agent.[50] *Aeromona aerophila* has been isolated from 9% of diarrheic foals[51] but its role in foal diarrhea is unknown. *S westeri* infects foals early in its life, but its role in diarrhea is questionable; however, in 1 study it was associated with diarrhea when fecal egg count was greater than 2000 eggs/g of feces.[20] Enterotoxigenic *Bacteroides fragilis* was isolated from the feces of 10 of 40 Thoroughbred foals with naturally acquired diarrhea.[52] In another study, using PCR, *N risticii* was found in 4% of foals with diarrhea.[22] The importance of the latter 2 agents is currently unknown.

## NONINFECTIOUS DIARRHEA

### *Necrotizing Enterocolitis*

NEC is a disease primarily of premature infants and consists of necrotic injury to the mucosal and submucosal layers of the GI tract. The distal small intestine and proximal colon are most commonly affected.[2] NEC has been reported in foals. These foals showed severe weakness, inability to stand, colic, ileus, gastric reflux, intolerance to enteral feeding, shock signs, and evidence of pneumatosis intestinalis in the large colon.[53]

### *Hypoxic Gastrointestinal Damage*

Peripartum asphyxia syndrome in foals can cause ischemic damage to different organs, such as GI tract, kidneys, heart, and brain.[54]

### *Equine Gastric Ulcer Syndrome and Diarrhea*

Equine gastric ulcer syndrome occurs in foals from 2 days to 9 months of age[17] and has been associated with diarrhea; however, a recent consensus statement questions this association.[55]

### *Luminal Irritant Diarrhea*

Foals can develop pica and may ingest large amounts of sand or other abrasive materials that can be extremely irritating and damaging to intestinal mucosa that may result in diarrhea with colic.[17]

### *Diarrhea Due To Lactose Intolerance*

Primary lactose intolerance in foals is rare.[56] Secondary lactose intolerance produced by small intestinal enteritis associated with RV and *C difficile* has been also reported.[57,58]

### *Dietary Diarrhea*

Dietary intolerance in foals can cause diarrhea. It is more commonly seen in foals receiving enteral nutrition with milk replacers.[2,17]

## DIAGNOSIS

Establishing a diagnosis of diarrhea is easily achieved whereas establishing the cause of the diarrhea is often difficult. The minimum database of a foal with diarrhea should

include a thorough history, physical examination, complete blood cell count, biochemistry profile, and determination of serum immunoglobulin concentrations. If available or in severe cases, blood gas analysis can also be useful. Abdominal ultrasound can be useful to determine signs of NEC and to assess intestinal wall thickness. Abdominal radiography can be used to determine presence of sand and gas accumulations. A major differential in diarrheic foals is sepsis and if available a sepsis score and blood culture should be performed.

Additionally, fecal testing is recommended and tests should be based on the most likely causative agent of a given case based on age and clinical signs (**Table 1**). Testing options for various causes are presented in **Table 2**. Fecal panels are offered by various laboratories, but results have to be interpreted with caution, because they have not been critically validated. A positive diagnostic test does not always confirm that this agent is the etiologic cause of the diarrhea. Several pathogens can be found in feces of healthy as well as diarrheic animals.

## INITIAL RESUSCITATION

### *Rehydration*

To correct hypovolemia, crystalloids, colloids, or combinations of these 2 can be used. Replacement crystalloid fluids for foals include lactated Ringer solution, Normosol R, and Plasma-Lyte 148.[59] Hypertonic solutions are not commonly used in foals, because foals may not be tolerant of large sodium loads.[60] Synthetic colloids can be used as 3-mL/kg to 5-mL/kg boluses if needed.[60] Synthetic colloids have been associated with renal failure and a worse outcome in human medicine and their use is currently questioned in horses as well. An attempt should be made to correct hypovolemia without synthetic colloids if deemed feasible. Plasma is in foals a better choice of colloids than synthetic colloids. In case colloids are used, administration as constant rate infusion (CRI) of 1 mL/kg/h to 2 mL/kg/h to has been suggested to avoid or minimize the risks for anaphylactoid reactions.[60]

**Table 1**
**Common infectious agents of foal diarrhea sorted by age range of foals**

| Age Range | Common Causes |
|---|---|
| < Two weeks | Foal heat diarrhea<br>*C perfringens*<br>*Rotavirus*<br>*C difficile*<br>Septicemia<br>*Cryptosporidium*<br>Neonatal asphyxia<br>NCE |
| Two weeks–two months | *Rotavirus*<br>*Cryptosporidium*<br>*Salmonella* spp<br>*S westeri* |
| > Two mo | *L intracellularis*<br>*S westeri*<br>*N risticii* |
| All ages | *Salmonella* spp<br>Lactose intolerance<br>Luminal irritants |

**Table 2**
**Diagnostic tests for associated causes of foal diarrhea**

| Etiologic Agent | Test | Characteristics |
|---|---|---|
| Foal heat diarrhea | Clinical examination | Based on clinical presentation |
| RV | Electron microscopy | Limited use in the field |
| | Latex particle agglutination | Sensitivity not as high as ELISA, rapid, simple, and useful in the field |
| | ELISA | Most show high sensitivity |
| | RT-PCR | Commercially available, rapid and sensitive |
| *C perfringens* | Fecal culture | Considered normal GI inhabitant in horses, but recent studies have not confirmed it. |
| | PCR | Detect toxin genes, best suited as adjunctive test because toxigenic strains are often found in healthy horses. |
| | ELISA | To detect specific toxins, it is deemed more accurate than gene detection PCR |
| | ELISA + toxin gene PCR | The strongest evidence of *C perfringens*–associated disease |
| *Salmonella* | Fecal culture | Isolation is criterium of diagnosis; 5 daily consecutive samplings in adult horses have 97% sensitivity. Number of samples not determined in foals. |
| | PCR | Sensitivity from 80% to 100% and specificity that ranged from 85% to 98%. Potential for misclassification |
| *C difficile* | Fecal culture | Isolation is not confirmatory. Normal horses may harbor it, and there are nontoxigenic strains |
| | CTA | Best available test, but technically demanding, time consuming, and not readily available |
| | PCR for TcdA and TcdB | High sensitivity, short turnaround time. False positives in carriers |
| | Toxin A/B ELISA, | Some tests validated in horses. Reported sensitivity and specificity of 84% and 96%, respectively, compared with CTA |
| Coronavirus | RT-PCR | High sensitivity. There are other tests such virus isolation, immunofluorescent antibody test, virus neutralization. Significance of detection not determined yet. |
| *Cryptosporidium* | Fecal modified AF | Low cost and simple methodology but low sensitivity |
| | PCR | High sensitivity and specificity |
| | ELISA | Similar sensitivity to AF |
| *L intracellularis* | PCR | Indicates bacteria shedding and active infection |
| | Warthin-Steiner silver stain | Postmortem diagnosis. Strongly suggestive. The curved bacilli presence |
| *R equi* | PCR | Has been used in foals with diarrhea |
| *S westeri* | Fecal flotation | Commonly used |
| Lactose intolerance | Intestinal lactose absorption test | Commonly used to determine it |

*Abbreviations:* AF, acid-fast stain; CTA, cell cytotoxicity assay; RT-PCR, reverse transcription–PCR.

The speed and volume of infusions depend on the degree of hypovolemic, cardiovascular status and plasma protein levels; however, there are no studies in foals to suggest accurate fluid rates. The most recent recommendation based on pediatric patients suggests an initial fluid bolus of 20 mL/kg crystalloid solution over 10 minutes to 20 minutes.[59] Perfusion should be reassessed after the bolus and, if still inadequate, this bolus can be repeated up to 3 times in total. Clinical signs of adequate perfusion include normalization of tachycardia, improvement in mentation, return of suckle reflex, and urine voidance. If possible, laboratory parameters should be re-evaluated. After 3 boluses, continuous rate infusion of fluids is recommended if possible. If response to fluid therapy is inadequate (perfusion does not normalize), inotrope therapy should be added. Dobutamine diluted in isotonic saline, 5% dextrose, or lactated Ringer solution is used and the dose should be titrated from 1 μg/kg/min to 3 μg/kg/min up to 5 μg/kg/min CRI. The remainder of the dehydration should be corrected in within 12 hours to 24 hours and ongoing losses added to the fluid regimen.

### *Maintenance*

Maintenance amounts of fluid need to be added if the foal is not allowed to nurse. The maintenance fluid needs in foals should be calculated using the Holliday-Segar formula for body weight (BW)[59]:

1 kg to 10 kg = 100 mL/kg/d

11 kg to 20 kg = 1000 mL + 50 mL for each kg >10 kg/d

Greater than 20 kg of weight = 1500 mL+25 mL for each kg/d >20 kg

Correction of electrolyte imbalances during maintenance fluid therapy is crucial for recovery. This also aids in balancing the acid base status of foals. Hyponatremia should be corrected at a rate not exceeding 0.5 mEq/h to avoid central pontine myelinolysis. Hyponatremia can be partially corrected by increasing the plasma sodium concentration by 2 mEq/L to 4 mEq/L to abolish seizure activity.[60] Hypokalemia is treated with empiric supplementation of fluids with 10 mEq/L to 40 mEq/L of potassium chloride and it depends on the existing plasma potassium concentration. Hyperkalemia can be empirically treated with administration of 20 mL/kg of 0.9% sodium chloride containing calcium (eg, 0.4 mEq/L to 1 mL/kg of 23% calcium borogluconate) and 4 mg/kg/min to 8 mg/kg/min of dextrose.[60] Isotonic sodium bicarbonate (1.3%) can be used with added dextrose as an alternative treatment. In refractory and severe cases of hyperkalemia, insulin treatment can be tried.

Sodium bicarbonate therapy should be reserved for foals with severe refractory acidosis, often due to hyperchloremia. In these cases, isotonic sodium bicarbonate (1.3%) intravenously (IV) can help ameliorate signs of acidosis by supplying sodium while decreasing the chloride load. Alternatively oral sodium bicarbonate can be administered.

All foals with compromised systemic status usually benefit from glucose administration. A starting dose is 4 mg/kg/min. The rate can be increased to 6 mg/kg/min to 8 mg/kg/min if needed.[60,61] Glucose should be administered separately from fluid therapy if possible, using a fluid pump. This allows adjusting fluid and glucose therapy separately.

## ANTI-INFLAMMATORY AND ANTIENDOTOXIC TREATMENTS

Nonsteroidal anti-inflammatory drugs (NSAIDs) should be used cautiously in foals less than 1 month old due to renal and gastric side effects.[2] Polymyxin B (6000 U/kg IV) can be used to treat endotoxemia.[62] Plasma transfusion should be considered for foals with failure of passive transfer of immunity. Because foals with diarrhea may use their globulins despite initially adequate levels, assessment of immunoglobulin concentration should be repeated periodically to assess the need for further plasma transfusion.

### *Absorptive Agents*

Adsorptive agents, such as kaolin/pectin and bismuth subsalicylate, have been used but efficacy studies are lacking. These agents are used at 0.5 mL/kg to 4 mL/kg, 1 to 4 times daily.[63]

Di-tri-octahedral smectite can has been shown to neutralize *C difficile* and *C perfringens* toxins in vitro. It is possible that in vivo neutralization of bacterial toxins in the gut could also occur.[64]

### *Modification of the Gastrointestinal Microbiota*

The use of probiotics in neonatal foals cannot be recommended based on currently available data. The use of fecal microbiota transplant has been proposed based on its remarkable success in treating recurrent *C difficile* infection in humans[65] (see Marcio Carvalho Costa and Jeffery Scott Weese's article, "Understanding the Intestinal Microbiome in Health and Disease," in this issue).

### *Adjunct Therapies*

The prophylactic use of antiulcer medications is controversial in neonatal foals but indicated in older foals. It has been shown that neonatal foals on antiulcer medication had a higher risk of developing diarrhea.[66] Omeprazole is the only approved drug for the treatment of EGUS, and 4 mg/kg orally, every 24 hours, is the recommended dose.[49]

Gastroprotectants should be considered if NSAIDs are given for prolonged periods of time.

In foals with signs of SIRS or sepsis, the use of broad-spectrum antibiotics is warranted. The antibiotics commonly used are a combination of aminoglycosides (mainly amikacin 20–25 mg/kg IV every 24 h) and penicillin (22.000–44.000 IU/kg IV every 6 h).[67]

### *Specific Therapies*

Metronidazole (15–25 mg/kg body weight every 8 h) is widely used for clostridial diarrhea, but there are no current objective data supporting its use.[67] Vancomycin has been used in cases of resistance and in humans is used in severe cases of *C difficile*–associated diarrhea.[68–74] Bacitracin was used in the past but is no longer available.[68]

Antibiotic treatment in foals with known *Salmonella* infection is indicated due to the risk for bacteremia and sepsis. The treatment should be continued beyond clinical recovery to prevent secondary seeding.[6] Antimicrobials effective against *Salmonella* spp include extended-spectrum cephalosporin or ampicillin-sulbactam alone or in combination with an aminoglycoside (gentamicin or amikacin) or fluorquinolones.[67]

Antimicrobial choices recommended for treating *L intracellularis* include oxytetracyclines, macrolides, or chloramphenicol.[48] Treatment of rhodococcal enterocolitis is similar to pulmonary *R equi* infection using macrolide antimicrobials combined with

rifampin.[49] Therapy for cryptosporidiosis is largely supportive in foals, but therapy with the aminoglycoside paromomycin could be attempted.[6] In cases of *S westeri*–associated diarrhea, anthelmintics (oxibendazole [15 mg/kg] or ivermectin [0.2 mg/kg]) should be used.[75]

## NUTRITIONAL SUPPORT

Foals with mild to moderate diarrhea without evidence of colic should be allowed to continue nursing. In contrast, any foal with abdominal discomfort will likely benefit from a period of brief GI rest (12–24 h).[2] Fluid therapy has to be administered if a foal less than 4 weeks old is not nursing for more than 6 hours. In cases of suspected lactose intolerance, exogenous lactase (Lactaid, 3000–6000 FCC U orally every 6 h) can be used.[2] Reintroduction to feeding should be slow and gradual. Feeding of 10% of body weight per day divided into hourly or 2-hour feeding intervals through a nasogastric tube or feeding tube is an adequate starting level.[76]

## PREVENTION AND MANAGEMENT

Foal infectious diarrhea prevention should focus on 3 important aspects of disease prophylaxis: minimize exposure to pathogens (disinfection and isolation), increasing immunity (vaccination), and optimization of management practices.[77]

Detection of a sick foal demands that in-contact animals should not be moved to other locations, because they may be a source of infection. People traffic should be curtailed by ensuring that healthy animals are attended to first, followed by in-contact foals, and finally clinical cases.[78]

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