EQUINE VETERINARY EDUCATION
Equine vet. Educ. (2012) **24** (4) 206-214
doi: 10.1111/j.2042-3292.2011.00358.x

Review Article

A review of foal diarrhoea from birth to weaning

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Keywords: horse; gastrointestinal; enterocolitis; sepsis; infectious disease

Summary

Diarrhoea is among the most common clinical complaints in foals. Aetiologies, diagnostic testing and recommended interventions for specific causes of enterocolitis are summarised. Many mild to moderately affected foals can be managed in an ambulatory setting, while others will benefit from more intensive care at a referral centre.

Noninfectious aetiologies

Foal heat diarrhoea is itself a misnomer for short periods of diarrhoea seen between one and 2 weeks of age in otherwise systemically healthy foals. Attempts to identify a causative agent in mare's milk around the time of oestrus have been unsuccessful (Johnston *et al*. 1970). Further, orphans and foals maintained on milk replacer often also experience a similar episode of diarrhoea, effectively ruling out the mare as a cause (Cymbaluk *et al*. 1993; Magdesian 2005). A more likely theory involves the normal development of gastrointestinal flora, particularly as the foal begins to ingest other feeds and inoculates its gastrointestinal tract via coprophagy (Masri *et al*. 1986). Recent work examining bacterial flora and occurrence of diarrhoea in neonatal foals further supports this theory and failed to demonstrate an association between foal heat in dams and onset of diarrhoea in the foal (Kuhl *et al*. 2011). Affected foals will continue to suckle readily, are afebrile and have no clinical abnormality other than diarrhoea. Thus, owner education and benign neglect are ideal treatments.

Dietary issues can occur in both orphan and mare-fed foals. Overfeeding associated with high milk-producing mares results in a large amount of undigested lactose reaching the large intestine, where its fermentation can lead to osmotic diarrhoea. Foals receiving inappropriate or incorrectly prepared milk replacer can develop diarrhoea, secondary to overadministration of lactose or electrolytes (Cymbaluk *et al*. 1993). Primary lactose intolerance in foals is very rare, but has been reported (Koterba 1990; Roberts *et al*. 2008; Sloet van Oldruitenborgh-Oosterbaan 2008). There is also a suggested, although still unproven, association between secondary lactose intolerance and enterocolitis of clostridial or rotaviral origin (Weese *et al*. 1999).

Some foals willingly ingest large amounts of sand or other abrasive material which can be extremely irritating and damaging to intestinal mucosa. Foals with significant colonic sand can present for diarrhoea, with or without colic. Most cases resolve with medical therapy but severe cases may require surgical intervention. Previous work in adult horses has shown that removal of the animal from a sandy environment may be equally efficacious as medical treatment but results are conflicting (Leib 1997; Hammock *et al*. 1998; Hotwagner and Iben 2007).

Perinatal asphyxia syndrome (PAS) can also cause gastrointestinal mucosal damage. Hypoxia and hypoperfusion of the gastrointestinal tract can result in signs of enterocolitis, even in the absence of evidence of hypoxia to other body systems. Similar to treatment of the neurological signs of PAS, these foals require supportive care, prevention of sepsis and cautious enteral feedings.

A syndrome of necrotising enterocolitis is recognised in human premature and term neonates and reported in foals. In foals (although not infants), the syndrome has been loosely linked to clostridial infection (Bueschel *et al*. 1998; East *et al*. 1998). Regardless of aetiology, the disease is characterised by symptoms of ileus, evidence of *pneumatosis intestinalis* and necrotic intestinal mucosa/submucosa (Cudd and Pauly 1987; Eser *et al*. 2002; Magdesian 2005). Affected foals are generally very compromised and can progress rapidly to death.

Bacterial infection

Clostridial infection is most commonly caused by *C. perfringens* or *C. difficile* in foals (Browning *et al*. 1991; Netherwood *et al*. 1996; East *et al*. 1998). Additionally, foals and adult horses can harbour and shed both *C. difficile* and *C. perfringens type A* without developing clinical disease, although adults are less likely to do so

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(Tillotson *et al*. 2002; Båverud *et al*. 2003; Medina-Torres *et al*. 2011). *Clostridium perfringens type C* appears to be associated with significant disease but additional work on both prevalence of infection and ideal methods of diagnostic testing are needed (Bueschel *et al*. 1998; East *et al*. 1998; Tillotson *et al*. 2002). The presence or absence of clostridial enterotoxin may help define the pathogenicity of these potential infectious agents (Netherwood *et al*. 1998; Weese *et al*. 2001). These infections can be primary in nature or secondary to antimicrobial administration (Båverud *et al*. 2003; Arroyo *et al*. 2004). Regardless of the specific clostridial aetiology, affected foals can present with acute colic and haemodynamic compromise, often prior to the development of clinical diarrhoea. Significant intestinal necrosis may occur and will contribute to rapid decompensation and secondary bacteraemia. Clinical pathological abnormalities will often include significant panleucopenia, hypoproteinaemia, and hyperfibrinogenaemia (Jones *et al*. 1987; East *et al*. 1998; Magdesian *et al*. 2002). In general, affected animals will have adequate transfer of passive immunity (East *et al*. 1998).

Salmonella can cause diarrhoea in horses of any age. The most typical source for infection in a foal is the dam or another mare/foal pair kept in close proximity (Traub-Dargatz and Besser 2007). As seen with Salmonellosis in older horses, severity of infection will vary based on the host-adaptation of the species involved. The most common serovars are Typhimurium, Newport, Anatum and Agona (Traub-Dargatz and Besser 2007). Infection in neonates is associated with relatively high mortality in some retrospective work, but this has not been uniformly verified (Netherwood *et al*. 1996; Frederick *et al*. 2009). Affected foals are usually febrile, have varying degrees of systemic compromise, and have profound leucopenia often with a toxic left shift (Lester 2003). Localised infections of bones, joints, central nervous system or umbilicus can develop due to bacteraemia, even after recovery from the initial episode of enterocolitis; thus, owners should be cautioned to carefully observe affected foals after the resolution of diarrhoea.

A variety of other bacterial agents have occasionally been associated with diarrhoea in the young foal, including *E. coli, Bacteroides fragilis, Enterococcus* and *Aeromonas* spp. (Myers *et al*. 1987; Holland *et al*. 1989, 1996; Browning *et al*. 1991). However, a true causative relationship has not been well proven for these agents.

Diarrhoea is one of the most common clinical signs for foals with systemic sepsis (Hollis *et al*. 2008; Sanchez *et al*. 2008). Diarrhoea occurring secondary to sepsis is most commonly due to mucosal hypoperfusion and sepsis-related inflammatory mediators. A specific gastrointestinal pathogen is rarely isolated from faeces of affected foals but presumptively bacteraemia in this population is associated with enteric bacterial translocation. Further, approximately 50% of foals with clinical diarrhoea aged less than one month are bacteraemic (Hollis *et al*. 2008; Sanchez *et al*. 2008; Frederick *et al*. 2009).

Although classically a pulmonary pathogen, *Rhodococcus equi* can have extrapulmonary effects, including enterocolitis, abdominal abscessation, lymphadenitis, peritonitis and hepatitis (Reuss *et al*. 2009). The number of infected animals is likely small but it is an important consideration in foals presenting at greater than 30 days of age, particularly those with evidence of respiratory disease or from farms with a history of *R. equi* infection. In a population of referred cases (150 foals with *R. equi*), 74% of foals had at least one extrapulmonary site of infection – these included diarrhoea and other forms of abdominal infection (Reuss *et al*. 2009).

Older foals, normally 3–12 months, can develop proliferative enteropathy caused by *Lawsonia intracellularis*. Although diarrhoea can occur, poor body condition is the most commonly noted clinical sign. Affected animals typically have small intestinal mural thickening evident ultrasonographically, which may be corrugated in appearance. Laboratory findings generally include significant hypoproteinaemia, characterised by hypoalbuminaemia or panhypoproteinaemia. Although most affected animals will survive with aggressive treatment, they will lag behind similar quality foals in financial value at yearling sales (Frazer 2008).

Increasingly recognised in older foals, Potomac Horse Fever (*Neorickettia risticii*) infection results in fever and anorexia, followed by diarrhoea within 1–2 days. This pathogen can affect foals >3 months of age (John *et al*. 1989).

Viral infection

Rotavirus is the most common viral cause of diarrhoea and most affected foals are less than 30 days of age (Conner and Darlington 1980; Netherwood *et al*. 1996; Frederick *et al*. 2009). There is a large range of disease severity, from very mild to life threatening. The virus damages small intestinal microvilli, resulting in decreased absorptive capacity and increased luminal secretion. Diarrhoea is self-limiting in many foals but some develop profuse watery diarrhoea, significant dehydration and electrolyte abnormalities. Older foals have a much greater ability to compensate for small intestinal disease with effective colonic resorption (Magdesian 2005). Both coronavirus and adenovirus have also been isolated from the faeces of diarrhoeic foals, but the true incidence and importance of these infectious agents is not fully understood (McChesney *et al*. 1973; Corrier *et al*. 1982; Studdert and Blackney 1982; Davis *et al*. 2000; Guy *et al*. 2000). Recent work examining the prevalence of various pathogens in diarrhoeic and healthy foals using PCR assays has identified coronavirus more commonly than previously recognised but further work is needed to further establish the true roles of these pathogens (Slovis *et al*. 2010).

Parasitic infection

Although most foals are completely asymptomatic in the face of *Strongyloides westeri* infection, extreme infestations can result in diarrhoea (Brown *et al*. 1997). The major source of infection for the foal is transmammary by ingestion of larvae. Ivermectin administration to the dam can prevent transmission to the foal. *Cryptosporidium parvum* has been isolated from the faeces of diarrhoeic foals, primarily those under one month of age. Originally thought to be a problem of immunocompromised foals only, *Cryptosporidium* can affect immunocompetent foals either alone or as a co-infection (Netherwood *et al*. 1996; Cole *et al*. 1998; Grinberg *et al*. 2008, 2009; Slovis *et al*. 2010). Attention should be paid to the zoonotic nature of this disease. Although other protozoal species have been isolated from healthy and sick foals; their importance as a cause of disease is still poorly understood.

Diagnostic approach

Evaluation of the diarrhoeic foal should begin with careful physical examination. Neonates are particularly likely to have concurrent sepsis, respiratory compromise or evidence of neonatal encephalopathy. Baseline laboratory evaluation should include complete blood count, biochemical profile and electrolytes, particularly for foals with evidence of systemic compromise. Venous blood gas analysis, including measurement of lactate, can be useful in any foal with significant hypovolaemia. Measurement of immunoglobulin concentration is indicated in neonates and blood culture is recommended for patients less than 30 days of age. Because approximately 50% of blood cultures from diarrhoeic foals yield growth of at least one infectious agent, blood culture can be a very useful diagnostic tool in this population (Frederick *et al*. 2009). Additionally, nasogastric intubation is indicated in any foal displaying signs of colic.

Ultrasonographic examination of the abdominal cavity should be considered in any systemically compromised diarrhoeic foal, especially if signs of colic have been observed. A linear or curvilinear probe with a frequency between 3 and 7 mHz can be used to obtain quality images, with a 5–10 mHz probe preferable for examination of the internal umbilical remnants. Ultrasonographic examination allows for identification of fluid or gas located within the visceral lumen, intestinal wall (*pneumatosis intestinalis*) or free within the peritoneum as well as examination of intestinal mural thickness, estimation of intestinal distention and evaluation for evidence of intussusception. The internal umbilical remnants can also be evaluated for evidence of infection. If indicated, ultrasonography can also be used to locate an appropriate area for abdominocentesis. Radiographs can identify sand or visceral gas distention. Diagnostic quality radiographs of the abdomen of young foals can be obtained with ambulatory equipment.

In severe cases of enterocolitis, particularly those with significant or recurrent pain, it can be challenging to completely rule out a surgical lesion. A positive response to initial therapy, in conjunction with imaging findings, can support the diagnosis of enterocolitis. An exploratory laparotomy will be indicated for foals that fail to respond to initial therapy or remain painful despite the provision of analgesic agents.

Identification of an aetiological agent is not always possible, but the desire to pursue a diagnosis often depends upon the number and/or severity of animals affected. In a retrospective evaluation of diarrhoeic foals with complete diagnostic testing, 55% of foals (122/223 enrolled) tested were positive for one or more pathogen (Frederick *et al*. 2009). Many recommended tests do not provide immediate results; hence nonspecific therapy is typically instituted prior to the return of diagnostic tests. While the results of these diagnostic tests may not dictate treatment for individual animals, the information is useful for farm level prevention and biosecurity measures. Commonly performed diagnostic procedures are summarised in **Table 1**. Case details and clinical signs can lead to directed sample submission. One should note that testing time does not include time associated with sample transport to an appropriate laboratory.

The most meaningful method of identifying clostridial infection is not entirely clear. The combination of bacteriological culture and enterotoxin assays may assist in differentiation of infection from colonisation with nonpathological species but additional research is needed to better validate testing mechanisms for clostridial infection (Weese *et al*. 2000, 2001; Arroyo *et al*. 2004). *Rhodococcus equi* is easily isolated from the faeces of asymptomatic and uninfected foals, therefore diagnosis depends upon intra-abdominal sampling (abdominocentesis or aspirate of peripheral abscess) or an index of suspicion (supported by pulmonary disease). Further confirmation of systemic *R. equi* can be made with vapA PCR testing (blood, transtracheal wash, abdominal fluid sample or faeces), previously demonstrated to be extremely sensitive and specific for identification of pulmonary disease (Sellon *et al*. 2001; Halbert *et al*. 2005; Pusterla *et al*. 2007).

Treatment

Treatment strategies are directed at both provision of supportive care (fluid therapy, nutrition) and treatment for specific infections, as indicated by clinical assessment and diagnostic testing.

Analgesics are indicated in any foal displaying signs of colic secondary to enteritis. If pain is persistent or severe, imaging is often useful to rule out a surgical lesion. Most dosages and regimes suggested for use in foals are extrapolated from those used in adult horses. Pharmacokinetic and behavioural effects of butorphanol

TABLE 1: Diagnostic procedures for common infectious agents

Agent	Sample	Test	Testing time	Comments
Clostridium difficile	Faeces	Toxin A/B ELISA	Minutes to hours	
Clostridium perfringens	Faeces	Enterotoxin ELISA	Minutes to hours	High sensitivity; samples should be kept cool (not frozen) if possible
Clostridium spp.	Faeces	Culture	Days to weeks	Low specificity; samples should be transported anaerobically
Salmonella spp.	Faeces	Culture	Days to weeks	5 samples needed to conclude negative
	Faeces	PCR	Hours to days	High sensitivity, specificity variable
Neorickettsia risticii	Faeces, plasma	PCR	Days to week	Collect samples prior to treatment
	Blood	IFA	Days to week	Poor sensitivity
Rotavirus	Faeces	ELISA	Minutes to hours	High sensitivity, equal or better than EM
Coronavirus	Faeces	PCR	Hours to days	
Lawsonia intracellularis	Faeces Tissue	PCR	Days	Poor sensitivity, best to submit both tests
	Serum	IFA	Days to week	Best to submit both tests
Cryptosporium parvum	Faeces	Acid fast stain IFA	Minutes to hours Days to week	
Strongyloides westeri	Faeces	Faecal float	Minutes to hours	Prepatent period of 5–7 days
All viruses and protozoa	Faeces	Electron microscopy	Days to weeks	Poor sensitivity depending upon sample, very specific

have been evaluated in foals, although data is not yet available regarding analgesia. Butorphanol (0.05–0.1 mg/kg bwt i.v.) has a duration of approximately 2 h in neonatal foals and multiple administrations may be indicated after ruling out a surgical lesion (Arguedas *et al*. 2008). Opioids and α_2 agonists (generally xylazine at 0.5–1.0 mg/kg bwt i.v.) can be used together to manage pain and allow more thorough work-up of the patient, although the use of α_2 agonists is contraindicated in cases of severely compromised foals due to their cardiovascular effects. Additionally, flunixin meglumine (1 mg/kg bwt i.v. q. 12 h) can be used judiciously to manage pain and for anti-inflammatory/anti-endotoxic effects. Evaluation of flunixin in foals suggests that elimination of the drug may differ from adult horses, but physiological activity of the medication is similar and dosing schemes likely apply (Semrad *et al*. 1993; Crisman *et al*. 1996). Meloxicam, a COX-2 selective nonsteroidal drug, has been used for abdominal pain in adult horses. Dosing and pharmacokinetic data are established for meloxicam in adults, although efficacy in mitigating endotoxaemia in the horse and dosing regimens for foals have not been evaluated (Toutain *et al*. 2004). It is also important to avoid nonsteroidal anti-inflammatory drugs in cases with azotaemia or significant dehydration prior to fluid therapy.

Fluid therapy is necessary for many diarrhoeic foals and can be administered via either the oral or i.v. route. Intravenous fluid therapy is typically indicated for any hypovolaemic foal unable or unwilling to suckle routinely. The need for continuous vs. bolus therapy depends upon the availability of resources in conjunction with the severity of illness. If a foal is ambulatory, suckling well, only slightly dehydrated and has no evidence of ileus or reflux, oral fluid and electrolyte supplementation may be adequate, especially in older foals. The enteral route is rarely used as a sole source of fluid therapy in neonates with diarrhoea.

Initial volume replacement depends upon the degree of hypovolaemia. With hypovolaemic shock, a combination of crystalloid and colloid replacement is often recommended, especially in neonates, as many foals with enteritis or enterocolitis have some degree of protein loss within the gastrointestinal tract. Initial crystalloid volume replacement is best provided with a balanced electrolyte solution. Initial boluses of 10–20 ml/kg bwt i.v. can safely be given over 30 min and repeated as indicated by response to therapy. Other options include physiological saline solution or isotonic bicarbonate, but they are typically used to address specific strong ion imbalances, hyperkalaemia or severe metabolic academia of inorganic origin. Hyponatraemia should be addressed cautiously, increasing systemic sodium concentration at a rate no greater than 0.5 mmol/l/h (Magdesian 2005). After initial resuscitative therapy, maintenance fluid administration (100 ml/kg bwt/day for neonates, 50 ml/kg bwt/day for foals over 30 days of age) can be initiated, normally with isotonic balanced electrolyte solutions or isotonic bicarbonate. Rarely, relatively hypotonic maintenance

fluids may be appropriate for neonatal foals with low volume diarrhoea. Often, these fluids contain 5% dextrose, thus are not practical if bolus fluid supplementation is chosen. For those foals able to maintain caloric requirements through suckling from the mare, bolus administration of replacement crystalloid fluids (i.e. 1–2 l i.v. q. 4–6 h) can be used to replace ongoing losses. The ability to intermittently recheck electrolyte and acid-base status with stall-side analysers can help direct therapy. Therapeutic goals involve maintaining blood pH above 7.2 and sodium above 130 mmol/l and overcorrection should be avoided (Magdesian 2005). Volume resuscitation alone usually resolves metabolic acidaemia associated with hypoperfusion. However, if acidaemia remains (pH <7.2–7.25) despite adequate perfusion, bicarbonate supplementation is indicated. Bicarbonate deficit is calculated as base deficit (mmol/l) × bwt (kg) × 0.4. Conveniently, 8.4% sodium bicarbonate has a bicarbonate concentration of 1 mmol/ml. Typically, half the calculated deficit is given as a bolus and the remainder as a constant rate infusion over 12 h. A potential complication associated with rapid administration of sodium bicarbonate is overwhelming the foal's ability to remove carbon dioxide, if there are concurrent respiratory disorders.

Oral supplementation can also be administered if electrolyte or bicarbonate deficits exceed volume deficits. Baking soda (1 g = 12 mmol HCO_3) can be administered orally (separately from opportunities to nurse for suckling foals). Oral administration of light salt (consists of 1 : 1 KCl : NaCl) can provide adequate replacement for smaller deficits (0.1 g/kg bwt *per os* q. 8–12 h). Water and electrolytes can be administered via nasogastric tube, considering the gastric capacity, rate of gastric emptying and potential for ileus when applicable.

Colloidal therapy is useful in adult patients with hypovolaemic shock, hypoproteinaemia (total protein <40.0 g/l) or clinical signs of oedema, as they typically remain within the intravascular space (Magdesian 2003; Seahorn and Seahorn 2003). Hyperimmune plasma (10 ml/kg bwt/h, normally 1–2 l) not only provides plasma proteins to improve vascular oncotic pressure but also immunoglobulins of use to the neonatal or critical foal (Porter and Green 2003; Sykes and Furr 2005). Compromised neonatal foals with failure of transfer of passive immunity (IgG less than 8.0 g/l) should receive at least 1 l of plasma for provision of immune factors. For foals with ongoing gastrointestinal protein loss, administration of plasma can be repeated either as bolus therapy or a CRI at 1–2 ml/kg bwt/h. Serial testing of IgG concentrations in sick neonates can guide the need for additional plasma due to loss or consumption from the primary disease process. Hetastarch (3–5 ml/kg bwt i.v. boluses up to 10 ml/kg bwt/day maximum) can be used for initial resuscitation in hypovolaemic shock in conjunction with plasma or as a less expensive replacement colloid (Jones *et al*. 1997; McFarlane 1999). Coagulopathies secondary to Hetastarch administration have been a concern in both human and equine patients, although recent work in an experimental model of endotoxaemia in adult horses failed to identify any additional effect beyond the endotoxaemia-induced coagulopathy (Pantaleon *et al*. 2007).

Broad-spectrum parenteral antimicrobial therapy should be administered to any diarrhoeic foal less than one month of age due to the high incidence of bacteraemia (Hollis *et al*. 2008; Corley and Hollis 2009; Frederick *et al*. 2009). A typical initial broad-spectrum regimen includes amikacin (22–25 mg/kg bwt i.v. q. 24 h) or gentamicin (6.6 mg/kg bwt i.v. q. 24 h in foals >2 weeks, 12 mg/kg bwt i.v. q. 24 h <2 weeks) combined with potassium penicillin (22,000–44,000 u/kg bwt i.v. q. 6 h) or ampicillin (25 mg/kg bwt i.v. q. 6 h) (Durr 1976; Brown *et al*. 1984; Ensink *et al*. 1996; Magdesian *et al*. 1998; Mckenzie and Furr 2003; Bucki *et al*. 2004; Corley and Hollis 2009). Therapeutic drug monitoring can be used with aminoglycoside therapy to ensure adequate dosing while reducing the possibility of toxicity. Ceftiofur (5 mg/kg bwt i.v. q. 12 h) or cefquinome (1 mg/kg bwt i.v. or i.m q. 24 h) can be used as a monotherapeutic approach for broad-spectrum prophylaxis (Allan 2003; Thomas *et al*. 2006; Meyer *et al*. 2009; Rodich *et al*. 2009; Widmer *et al*. 2009). Broad-spectrum antimicrobial treatment is indicated for treatment of foals with known Salmonella infection (particularly if less than 90 days of age) and this treatment should continue past clinical recovery to prevent potential secondary sites of infection (Lester 2003). For suspected or proven clostridial infections, metronidazole (15 mg/kg bwt *per os* q. 6–8 h or 25 mg/kg bwt *per os* q. 12 h, 15 mg/kg bwt i.v. q. 6 h) is recommended (Sweeney *et al*. 1986; Giguère 2008), based upon regional patterns of resistance (Magdesian *et al*. 2002). For *Lawsonia intracellularis*, chloramphenicol (50 mg/kg bwt *per os* q. 6–8 h), oxytetracycline (10 mg/kg bwt i.v. q. 24 h) followed by doxycycline (10 mg/kg bwt *per os* q. 12 h), erythromycin (25 mg/kg bwt *per os* q. 12 h) or clarithromycin (7.5 mg/kg bwt *per os* q. 12 h) are most commonly recommended (Atherington and McKenzie 2006; Frazer 2008; Pusterla and Gebhart 2009; Sampieri *et al*. 2010). Length of treatment is dictated by clinical status and improvement of clinicopathological findings but a minimum of 3 weeks of treatment is likely indicated (Pusterla and Gebhart 2009). In cases of rhodococcal enterocolitis, treatment is similar to pulmonary *R. equi*, generally using a combination of macrolide (azithromycin at 10 mg/kg bwt *per os* q. 12 h or clarithromycin at 7.5 mg/kg bwt *per os* q. 12 h) and rifampin (5 mg/kg bwt *per os* q. 12 h).

There are numerous antidiarrhoeal agents available, with varying amounts of evidence supporting their use. Bismuth subsalicylate and kaolin/pectin (each 2–5 ml/kg bwt *per os* divided into 2–4 oral doses) are thought to coat the enteric mucosa and subsalicylate may have some anti-inflammatory effects. Activated charcoal has been used primarily as an adsorbent. Di-tri-octahedral smectite[1] (30 ml *per os* q. 12 h) has been shown to adsorb

clostridial toxins (Weese *et al*. 2003; Lawler *et al*. 2008). It is important to administer oral medications separately, to avoid nonspecific binding and reduced absorption of the other medications. Probiotic preparations, although commonly used, may provide unintended microbial species and have minimal evidence to support efficacy (Weese 2002). Current work supports the use of products containing *Saccharomyces* species in equine patients (Desrochers *et al*. 2005; Weese and Rousseau 2005); however, there is increasing evidence of fungaemia in immunosuppressed human patients treated with *Saccharomyces* (Venugopalan *et al*. 2010). Bulk laxatives (0.5–1.0 g/kg bwt psyllium *per os*) may stimulate clearance of luminal sand (Hotwagner and Iben 2007; Landes *et al*. 2008). Ease of psyllium administration can be improved through admixture with mineral oil prior to administration via nasogastric tube.

Gastric ulcer prophylaxis is also used variably in cases of enterocolitis. Omeprazole (1 mg/kg bwt *per os* q. 24 h), ranitidine (1.5 mg/kg bwt i.v. q. 8 h or 6.6 mg/kg bwt *per os* q. 8 h) or sucralphate (10–20 mg/kg bwt *per os* q. 6–8 h) may be used for prevention of ulcers in foals. However, the benefit of prophylaxis is unproven and the incidence of gastric ulceration in neonates at one institution appeared unrelated to administration of ulcer prophylaxis (Barr *et al*. 2000). There is additional concern that human ICU patients treated with ulcer prophylaxis may be more likely to develop respiratory or gastrointestinal infections (Kappstein *et al*. 1991; Dinsmore *et al*. 1997; Ortiz *et al*. 1998; Crill and Hak 1999).

Nutritional management

Foals with mild to moderate diarrhoea and no evidence of colic should generally be allowed to continue suckling. The benefits of continued oral intake often outweigh the risk of exacerbation of enteritis by additional suckling. Oral intake of as little as 60 ml of milk every few hours can provide nutrition for enterocytes (Ousey *et al*. 1997). Any foal with colic, abdominal distention or suspected rotavirus or clostridial infection will likely benefit from a period of brief gastrointestinal rest (12–24 h) (Magdesian 2005). Villous damage can result in transient maldigestion and lactase deficiency, thus exogenous lactase (Lactaid, 3000–6000 FCC units *per os* q. 6 h) during re-feeding is a useful adjunct in some foals (McAuliffe and Slovis 2008; Sloet van Oldruitenborgh-Oosterbaan 2008). For foals kept from suckling for longer than 12–24 h in older foals or 4–6 h in neonates, caloric requirements should be provided parenterally (Buechner-Maxwell 1998; Magdesian 2005). The addition of dextrose (typically 5% of a maintenance fluid rate or a rate of 4–8 mg/kg bwt/min i.v.) to fluid therapy is adequate to meet caloric requirements for up to 24–48 h. For more intensive or prolonged cases that cannot tolerate oral nutrition, partial or total parenteral nutrition (PPN or TPN) can be provided with the addition of amino acids with or without lipids to dextrose solutions. There is significant information available elsewhere regarding the preparation and administration of these TPN solutions (Buechner-Maxwell 2005; McKenzie and Geor 2009). Although it can be costly to administer, provision of parenteral nutrition can prevent a catabolic state associated with prolonged malnutrition. Intermittent blood glucose concentrations should be monitored with patients requiring parenteral feeding or young foals on limited suckling to ensure euglycaemia. One should note that neither euglycaemia nor hyperglycaemia rule out the presence of a negative energy balance.

Nursing care includes helping weak foals to stand every few hours. Thick bedding will aid in the prevention of decubitis ulcers and allow for absorption of diarrhoea. Cleaning the perineum and application of petroleum jelly or a similar lubricant will also help prevent skin irritation. Additionally, the dam should be milked frequently when not nursing the foal.

Preventative measures for spread of infection

Biosecurity is particularly important for prevention of the spread of infectious agents to other mares and/or foals. Strict isolation procedures should be used for affected foals, considering traffic flow, cleaning and grooming tools and other potential fomites. Ideally, potentially infected faeces should be contained and disposed of instead of composted on the property. Separate clothing and footwear should be used for affected animals, be they permanent and disinfected between animals or disposable. Footbaths and careful handwashing procedures are very important for minimising contamination, along with disinfection of all in-contact items. Quaternary ammonium disinfectants are effective against most bacterial pathogens and phenolic disinfectants (One-Stroke[2] and Tek-trol[3]) are effective against rotavirus, even when contaminated with organic matter. Clostridial spores are particularly resistant to disinfectants; the most effective agent for clearing environmental spores is bleach. Farm personnel are key to a good biosecurity plan, thus educating them is a valuable investment of time and effort (Dwyer 2001).

Thus far, available maternal vaccination against specific enteric pathogens is somewhat limited. Prepartum vaccination of broodmares against rotavirus appears safe, but reported efficacy appears variable (Powell *et al*. 1997; Barrandeguy *et al*. 1998). Data regarding prevention of other enteric pathogens in foals through vaccination is restricted to anecdotal reports.

Authors' declaration of interests

No conflicts of interest have been declared.

Manufacturers' addresses

[1]Biosponge, Platinum Performance, Buellton, California, USA.
[2]One Stroke Environ, Pro-Ag Products, Winnipeg, Manitoba, Canada.
[3]Tek-trol, Agri-Labs, St. Joseph, Missouri, USA.

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