Review Article

Review of equine Cryptosporidium infection

LIHUA XIAO and R. P. HERD

Department of Veterinary Preventive Medicine, The Ohio State University, 1900 Coffey Road, Columbus, Ohio 43210, USA.

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Introduction

Diarrhoea is one of the most important diseases of foals and 70–80% are affected within the first 6 months of life (Palmer 1985). Although the cause is multifactorial and changes in the physiology and nutrition of foals are sometimes responsible, diarrhoea is frequently the result of infection by enteropathogens such as bacteria, viruses and nematodes (Palmer 1985; Tzipori 1985). There is increasing evidence that Cryptosporidium infection can also cause diarrhoea in foals (Austin et al. 1990; Kim 1990).

Cryptosporidiosis in horses is caused by Cryptosporidium parvum, a coccidial parasite infective to mammals. C. muris has been reported in foals in Italy (Canestri-Trotti and Visconti 1985), but its identity was questionable because the reported size of oocysts was within the range of C. parvum (Kim 1990). Like other coccidia, the infective stage for Cryptosporidium is the sporulated oocyst. Unlike the others, the sporulation of Cryptosporidium occurs within the microvillous border of the small intestine. Therefore, newly excrated Cryptosporidium oocysts are infective to other hosts immediately. In addition, there are two types of oocysts in Cryptosporidium: (1) thin-walled oocysts that exist within the intestine shortly after their formation and re-initiate infection (autoinfection), and (2) thick-walled oocysts that are excreted in faeces and are responsible for the transmission of infection from one animal to another (Current and Garcia 1991).

Prevalence of Cryptosporidium infection in horses

Cryptosporidiosis in horses was first described in Arabian foals with combined immunodeficiency disease. Five of 6 immunodeficient Arabian foals in Colorado, USA, that died of adenoviral infection were infected with Cryptosporidium (Snyder et al. 1978). Clinical cryptosporidiosis was further reported in 2 Arabian foals in Australia and 1 Arabian foal in the United Kingdom, all with combined immunodeficiency disease (Gibson et al. 1983; Mair et al. 1990). Fatal cryptosporidiosis also occurred in 2 Thoroughbred foals with an immunodeficiency that resulted from the failure of passive transfer of colostral immunoglobulin from mares to foals (Poonacha and Tuttle 1989). These results suggested that an immunodeficiency was necessary for Cryptosporidium infection in horses.

Immunodeficiency, however, may not be a necessary predisposition for cryptosporidiosis. Studies in Canada, France, Spain and the United States have shown the presence of Cryptosporidium infection in immunocompetent foals with diarrhoea (Table 1). Cryptosporidium infection has also been found in normal foals without diarrhoea, with infection rates of 2.4–60.0% (Table 2). Cryptosporidium infection in horses therefore appears to be widespread and to cause diarrhoea in immunocompetent foals under certain conditions. Previously, equids had been considered to be resistant to Cryptosporidium infection (Coleman et al. 1989), and it was thought that Cryptosporidium infections in immunocompetent horses were incidental findings (Kim 1990).

Epidemiology of Cryptosporidium infection

Age

Most early studies on equine Cryptosporidium infection were conducted with neonatal foals (Snyder et al. 1978; Gibson et al. 1983; Canestri-Trotti and Visconti 1985; Gajadhar et al. 1985; Chermette et al. 1987; DiPietro et al. 1988; Fernandez et al. 1989; Coleman et al. 1989; Poonacha and Tuttle 1989; Mair et al. 1990). Cryptosporidium infection was found in these animals, suggesting that the infection was a neonatal problem, as in calves, in which oocyst excretion usually occurs in animals <4 weeks of age (Anderson 1981; Blewett 1989a; Henriksen 1989). However, Cryptosporidium infection does occur in older foals: 13 of 82 3–15-week-old foals examined in France were infected (Soule et al. 1983). Infection was also found in 3 Quarter Horses aged 100–123 days in Louisiana (Coleman et al. 1989). A sensitive and accurate detection method showed that foals in Ohio had infection between 4 and 23 weeks of age, with no significant difference in infection rates between 4 and 19 weeks (Xiao and Herd 1994).

Clinical cryptosporidiosis was also reported in older foals in France: a 10-month-old foal infected with Cryptosporidium had diarrhoea (Chermette et al. 1987), and 4 foals with Cryptosporidium infection developed diarrhoea at 6 weeks of age, and were still diarrhoeic at 9–10 months (Lengronne et al. 1985). We have observed 2 foals with chronic diarrhoea and severe Cryptosporidium infection at 6 months and 7 months of age (Xiao and Herd, 1994). Differences in management probably account for the different epidemiology of Cryptosporidium infection between calves and foals and farms.

Sources of infection

Several studies in France, using acid-fast staining of faecal smears, suggested that foals got their Cryptosporidium infection from mares (Lengronne et al. 1985; Chermette et al. 1987, 1989):
failed to support the view that mares are the major source of infection found in 2 mares in one US study using a similar detection method, but this was observed only after their foals had begun passing oocysts (DiPietro et al. 1998). Excretion of oocysts was also found in 2 mares in one US study using a similar detection method, but this was observed only after their foals had begun passing oocysts (DiPietro et al. 1998).

Examination of foals every 2 weeks from March to October showed that oocyst excretion in foals began at 4 weeks of age. Because the prepatent period for Cryptosporidium infection is widespread in other animal species (Xiao and Herd 1994), the infection rate was over 80%, higher than that of foals (Chermette et al. 1987, 1989). Yearlings had an infection rate similar to that of mares. The infection in mares was asymptomatic, but some infected yearlings excreted soft stools (Chermette et al. 1987). It was proposed, therefore, that mares were asymptomatic carriers and transmitted the infection to foals by contaminating the environment. Excretion of oocysts was also found in 2 mares in one US study using a similar detection method, but this was observed only after their foals had begun passing oocysts (DiPietro et al. 1988). In other studies, 48 mares in France (Soule et al. 1983) and 14 mares in the USA (Reinemeyer et al. 1984) were free of Cryptosporidium infection.

The use of a more sensitive and specific detection method failed to support the view that mares are the major source of infection for their foals. Immunofluorescence assay did not reveal the presence of Cryptosporidium in 18 mares examined in Ohio and 53 mares examined in Kentucky, although 17.6-31.0% of foals on these farms were infected (Xiao and Herd 1994). Acid-fast staining is known to have low specificity in detecting Cryptosporidium infection. The role of mares in the transmission of Cryptosporidium is therefore uncertain.

Because of the high infection rate and intensity of Cryptosporidium in foals, foals are most likely to get their infection from other infected foals, following an infection path similar to the calf-to-calf faecal–oral route of bovine Cryptosporidium infection (Henriksen 1989). This theory was substantiated by a recent study in Ohio, in which the chronological process of Cryptosporidium infection in foals on a farm was followed (Xiao and Herd 1994). On this farm, mares and their foals were kept in individual stalls and paddocks from birth for 9 days. They were then pastured together with older foals and their dams. A faecal examination of mares by immunofluorescence assay in early June revealed no Cryptosporidium infection, although a 27.8% infection rate of Giardia was present. Examination of foals every 2 weeks from March to October showed that oocyst excretion in foals began at 4 weeks of age. Giardia infection, which occurs later than Cryptosporidium infection in other animal species (Xiao et al. 1999), appeared much earlier in foals (2 weeks) than did Cryptosporidium infection on this farm. Cryptosporidium oocyst excretion in foals could be expected to occur earlier if foals were infected by mares, because the prepatent period for Cryptosporidium spp. (4–5 days) is shorter than that of Giardia spp. (1–2 weeks).

Because Cryptosporidium infection is widespread in other

### Table 1: Prevalence of Cryptosporidium infection in horses with diarrhoea

| Groups                  | No. of animals | Detection method | Location          | References               |
|-------------------------|----------------|------------------|-------------------|--------------------------|
| Arabian foals           |                |                  |                   |                          |
| Arabian foals           | 6              | 5                | Histology         | Colorado, USA            |
| Arabian foals           | 2              | 2                | Histology         | Australia                |
| Arabian foals           | 1              | 1                | Histology         | England                  |
| Thoroughbred foals      | 2              | 2                | Histology         | Kentucky, USA            |
| Immunodeficient foals   |                |                  |                   |                          |
| Pony foals              | 22             | 22               | Sugar flotation   | Louisiana, USA           |
| Foals                   | 4              | 4                | Acid-fast stain   | Illinois, USA            |
| Foals                   | 2              | 2                | Acid-fast stain   | France                   |
| Foals                   | 14             | 0                | Sugar flotation   | Ohio, USA                |
| Foals                   | 52             | 0                | Unspecified       | Australia                |

*Results from a 2nd year survey.

### Table 2: Prevalence of Cryptosporidium infection in horses without diarrhoea

| Groups                  | No. of animals | Detection method | Location          | References               |
|-------------------------|----------------|------------------|-------------------|--------------------------|
| Foals                   | 55             | 8                | Sugar flotation   | Louisiana, USA           |
| Foals                   | 68             | 0                | Sugar flotation   | Louisiana, USA           |
| Foals                   | 23             | 0                | Histology         | Colorado, USA            |
| Foals                   | 82             | 13               | Acid-fast stain   | France                   |
| Foals                   | 246            | 6                | Unspecified       | England                  |
| Foals                   | 30             | 18               | Acid-fast stain   | France                   |
| Foals                   | 37             | 6                | Immunofluorescence| Ohio, USA                |
| Foals                   | 29             | 9                | Immunofluorescence| Kentucky, USA            |
| Foals                   | 45             | 3                | Unspecified       | Italy                     |
| Yearlings               | 22             | 18               | Acid-fast stain   | France                   |
| Yearlings               | 24             | 0                | Immunofluorescence| Ohio, USA                |
| Yearlings               | 22             | 0                | Immunofluorescence| Kentucky, USA            |
| Mares                   | 48             | 0                | Acid-fast stain   | France                   |
| Mares                   | 49             | 40               | Acid-fast stain   | France                   |
| Mares                   | 14             | 0                | Sugar flotation   | Ohio, USA                |
| Mares                   | 18             | 0                | Immunofluorescence| Ohio, USA                |
| Mares                   | 53             | 0                | Immunofluorescence| Kentucky, USA            |

*Results from a 2nd year survey.
animal species, horses presumably can also get infection from other infected animals if they are housed or pastured together. Management of cattle and horses on the same farms and pastures has been implicated as a risk factor for equine Cryptosporidium infection in one study (Chermette et al. 1989). Alternate grazing between horses and ruminants as a control method for nematode infections may also lead to equine Cryptosporidium infection in certain circumstances.

Oocyst excretion patterns

Few studies have been done to elucidate the pattern of oocyst shedding in Cryptosporidium-infected foals. Experimental infections of colostrum-deprived foals and sucking foals, using oocysts from calves, showed that oocyst excretion by foals was very brief (Tzipori 1983). The mean period of oocyst shedding in naturally infected pony foals was 21.5 days (12–36 days) in one study (DiPietro et al. 1988) and 10 days (2–18 days) in another (Coleman et al. 1989). These results are similar to the oocyst excretion pattern of calves experimentally or naturally infected with C. parvum (Anderson 1981; Biewett 1989a; Henriksen 1989). In the study of Xiao and Herd (1994) on an Ohio Thoroughbred farm, oocyst excretion in foals began at 4 weeks of age and ended at 23 weeks, much later than that reported by most studies on equine or bovine Cryptosporidium infections. The oocyst shedding also lasted longer and was intermittent. Faecal examination every 2 weeks from March to October revealed that 7 of 25 infected foals were positive twice, 3 were positive 3 times, and 1 was positive 4 times. The longest interval between oocyst shedding was 6 weeks in 4 foals.

Clinical features of equine Cryptosporidium infection

Cryptosporidium parvum differs from other coccidia in that it invades the microvillous border and not the cytoplasm of host cells. The predilection infection site in horses is the distal small intestine, especially the ileum (Snyder et al. 1978; Gibson et al. 1983; Poonacha and Tuttle 1989; Mair et al. 1990), although in immunodeficient horses other organs such as the stomach, large intestine and pancreatic and bile ducts can also be involved (Snyder et al. 1978). Although brush border maldigestion can occur if infection extends to the proximal region of the small intestine, Cryptosporidium mainly causes malabsorption by the massive occupation by protozoa of the absorptive surface and villous atrophy (Tzipori 1988). Diarrhoea occurs as a result of overgrowth of intestinal microflora and changes in osmotic pressure. Compared to coccidiosis, diarrhoea in cryptosporidiosis is milder and non-haemorrhagic.

The role of Cryptosporidium infection in the aetiology of diarrhoea of immunocompetent foals is not certain. Although Cryptosporidium infection was implicated as the cause of diarrhoea by some researchers (Gajadhar et al. 1985; Lengronne et al. 1985; DiPietro et al. 1988), others found no Cryptosporidium infection in diarrhoeic foals (Reinemeyer et al. 1984; Soule et al. 1983; Chermette et al. 1987, 1989; Xiao and Herd, 1994). Experimental infection of colostrum-deprived and sucking foals with oocysts isolated from calves also failed to cause clinical signs (Tzipori 1983). However, when the infection rates of Cryptosporidium in diarrhoeic and normal foals were compared in the same study, higher infection rates were found in diarrhoeic animals (Angus 1989; Coleman et al. 1989).

Cryptosporidium infection may therefore cause diarrhoea under some conditions.

Cryptosporidium may cause diarrhoea of horses by interacting with other enteropathogens. Although Cryptosporidium was the only pathogen found in diarrhoeic foals in some studies (Gibson et al. 1983; Gajadhar et al. 1985; Lengronne et al. 1985), other enteropathogens were also present in others (Snyder et al. 1978; Coleman et al. 1989; Poonacha and Tuttle 1989; Austin et al. 1990; Mair et al. 1990). Concurrent infections of Cryptosporidium with rotavirus (Austin et al. 1990; Poonacha and Tuttle 1989), coronavirus (Coleman et al. 1989; Mair et al. 1990), adenovirus (Snyder et al. 1978; Mair et al. 1990), Salmonella (Coleman et al. 1989) and Giardia (Xiao and Herd 1994) were present in immunodeficient or immunocompetent foals. In ruminants, Cryptosporidium infection usually causes diarrhoea of young animals in association with rotavirus, coronavirus, enterotoxigenic Escherichia coli, Salmonella and Giardia (Angus 1990; Xiao et al. 1993b).

Horses react very differently in their clinical response to Cryptosporidium infection. Infection in foals with inherited or acquired immunodeficiency usually causes acute diarrhoea and high mortality. Diarrhoea in these animals develops very early, mostly during the first month of life. It progresses very quickly and foals die 1 or 2 weeks after onset (Snyder et al. 1978; Gibson et al. 1983; Poonacha and Tuttle 1989; Mair et al. 1990).

Diarrhoea in immunocompetent neonatal foals also begins within the first month (Gajadhar et al. 1985; DiPietro et al. 1988; Fernandez et al. 1988; Coleman et al. 1989), but it is usually self-limited and lasts 1–8 days (Coleman et al. 1989). Although high mortality has been reported in this group of animals (DiPietro et al. 1988; Fernandez et al. 1988), it was probably the result of concomitant infections. Diarrhoea in older foals infected with Cryptosporidium is frequently chronic and remittent. It lasts for several months and can still be present when foals are 9–10 months old (Lengronne et al. 1985; Chermette et al. 1987; Xiao and Herd 1994). The latter is very similar to cryptosporidiosis seen in immunocompromised humans, although the diarrhoea is not as profuse (Tzipori 1988).

Diagnosis of cryptosporidiosis

Post-mortem diagnosis of cryptosporidiosis in horses can be done by searching for the developmental stages of Cryptosporidium sp. on the small intestinal mucosa (Snyder et al. 1978; Gibson et al. 1983; Gajadhar et al. 1985; Poonacha and Tuttle 1989; Mair et al. 1990). In tissue sections stained with haematoxylin and eosin, developmental stages of Cryptosporidium appear as spherical basophilic bodies (2–5 µm in diameter) over the crypts and along the sides of intestinal villi (Snyder et al. 1978; Gibson et al. 1983). Special staining procedures (Wolbach–Giemsa) were used by some researchers without achieving any improvement (Snyder et al. 1978; Gibson et al. 1983). Transmission electron microscopy can be used to confirm the diagnosis (Snyder et al. 1978; Gajadhar et al. 1985; Poonacha and Tuttle 1989). The presence of a feeder organelle is unique to Cryptosporidium.

Cryptosporidium infection in living horses is diagnosed by finding oocysts in faeces. The most widely used method is acid-fast staining of faecal smears (Lengronne et al. 1985; DiPietro et al. 1988; Fernandez et al. 1988; Chermette et al. 1987, 1989). Oocysts of C. parvum appear as bright red spherical bodies (4–6 µm in diameter) against a counter-stained background in acid-fast stained faecal smears, while yeast cells stain darkly with the background stain. Problems with acid-fast staining are the low sensitivity and specificity. Only 3–13% of the oocysts pick up the stain (Cozon 1992). Because some yeast cells also stain red, the reported specificity of acid-fast staining is only 52% (Arrowood and Sterling 1989). Experience in our laboratory has shown that non-specificity increases with old samples. Faecal samples should therefore be submitted as fresh as possible or in 10% formalin or sodium acetate-acetic acid–formalin (SAF) preservatives. Samples preserved in polyvinyl alcohol (PVA) fixative cannot be used for Cryptosporidium examinations (Current and Garcia 1991). Because oocyst excretion in faeces is intermittent (Xiao and Herd 1994), multiple samplings are necessary with all diagnostic methods. In humans, 3 samples are
The specificity of monoclonal antibodies to Cryptosporidium against the dark background, while yeast cells stain yellow or red. The specificity of monoclonal antibodies to Cryptosporidium and the sensitivity of the direct immunofluorescence assay have greatly increased the sensitivity and specificity of Cryptosporidium examinations. Immunofluorescence assay is at least 10 times more sensitive than acid-fast staining (Garcia et al. 1987; Weber et al. 1991). The disadvantages of immunofluorescence staining are the higher cost and the requirement of a fluorescence microscope.

Oocyst examination can also be done by Sheather’s sugar flotation method (Reinemeyer et al. 1984; Gajadhar et al. 1985; Coleman et al. 1989). Oocysts appear as pink-tinged spherical bodies (4.5-5.5 μm in diameter) in sugar solution by bright-field microscopy, with 1-4 dark granules. Yeast cells do not have granules and are not in pink in colour. Phase-contrast or differential interference-contrast microscopy, however, is frequently needed to distinguish Cryptosporidium oocysts from yeast. Prompt processing and examinations of samples are also necessary, because oocysts begin to collapse and lose their spherical shape when left in sugar solution for more than 15 min (Current and Blagburn 1990).

Treatment and control

Because diarrhea caused by Cryptosporidium in foals is often mild and self-limited, treatment of affected animals may not always be necessary. Effective treatment would be useful for the occasional outbreaks with high morbidity and mortality. Unfortunately, there are no drugs available for such treatment. For foals with severe cryptosporidial diarrhea, supportive therapy with oral or parenteral rehydration and control of complicating infections are the only options to reduce mortality.

Control of Cryptosporidium infection is frustrating. The oocyst is the main target for control measures designed to reduce infection rate and intensity. This is difficult to achieve because Cryptosporidium oocysts are very resistant to adverse conditions. They are fully sporulated in the host and do not require the critical combination of environmental temperature and moisture for the development of infectivity required by other coccidia. If they are not exposed to extremes of temperature or desiccation, they can survive for several months. Most disinfectants at the concentrations used by farmers cannot kill Cryptosporidium oocysts. Oocysts can be killed only by steam heat, 10% formalin, 5% ammonia, 10 vol hydrogen peroxide, undiluted commercial bleach, or 5% octode (Antec International Ltd, Sudbury, Suffolk, UK) (Blewett 1989b; Angus 1990). Sanitation reduces the infection intensity to a certain degree, but not necessarily the infection rate. Some of the best managed horse farms in the world still have high Cryptosporidium infection rates (Xiao and Herd 1994). As there is an autoinfection stage in the Cryptosporidium life cycle, reduction in environmental contamination itself is not enough to prevent the occurrence of cryptosporidiosis.

Because most horses will eventually become infected (Tzipori and Campbell 1981; Opedembosch and Wellemans 1985; Xiao and Herd 1994), control strategies should emphasise the prevention of clinical cryptosporidiosis. It has long been known that the immune status of the host is a major factor determining the severity and duration of Cryptosporidium infection in other animal species (Current and Blagburn 1990). The high morbidity and mortality seen in immunodeficient foals infected with Cryptosporidium suggest that immunity also plays an important role in the occurrence of equine cryptosporidiosis.

Any factors that reduce immunity of horses are likely to increase the occurrence of cryptosporidiosis. Stresses such as overcrowding and early weaning, known to reduce immunity, should be avoided. Malnutrition may also play a role in equine cryptosporidiosis. Clinical cryptosporidiosis usually occurs in animals infected with other enteropathogens such as rotavirus, adenovirus, coronavirus, Salmonella and Giardia. Prevention and control of these infections may help to reduce the morbidity and mortality of Cryptosporidium infection in foals. The role of colostrum in equine cryptosporidiosis is not clear. One study implicated the lack of immunity transfer through colostrum as the cause of fatal cryptosporidiosis in 2 foals (Poonacha and Tuttle 1989). Studies in calves and humans, however, have shown no protective role of lactogenic immunity in Cryptosporidium infection (Current and Blagburn 1990). Nevertheless, lactogenic immunity is likely to reduce the occurrence of concomitant infections, thereby reducing the morbidity of Cryptosporidium infection. Foals should therefore be allowed to have adequate colostrum at birth.

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