EQUINE VETERINARY JOURNAL
Equine vet. J. (1994) **26** (1) 9-13

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.

Keywords: horse; *Cryptosporidium*; cryptosporidiosis; epidemiology; parasites

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 excreted *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.* 1988; 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):

TABLE 1: Prevalence of *Cryptosporidium* infection in horses with diarrhoea

Groups	No. of animals		Detection method	Location	References
	Examined	Positive			
Immunodeficient foals					
Arabian foals	6	5	Histology	Colorado, USA	Snyder *et al.* (1978)
Arabian foals	2	2	Histology	Australia	Gibson *et al.* (1983)
Arabian foals	1	1	Histology	England	Mair *et al.* (1990)
Thoroughbred foals	2	2	Histology	Kentucky, USA	Poonacha and Tuttle (1989)
Immunocompetent foals					
Pony foals	22	22	Sugar flotation	Louisiana, USA	Coleman *et al.* (1989)
Pony foals	4	4	Acid-fast stain	Illinois, USA	DiPietro *et al.* (1988)
Foals	4	4	Acid-fast stain	France	Lengronne *et al.* (1985)
Foals	2	2	Sugar flotation	Canada	Gajadhar *et al.* (1985)
Foals	14	0	Sugar flotation	Ohio, USA	Reinemeyer *et al.* (1984)
Foals	52	0	Unspecified	Australia	Tzipori (1988)

TABLE 2: Prevalence of *Cryptosporidium* infection in horses without diarrhoea

Groups	No. of animals		Detection method	Location	References
	Examined	Positive			
Foals	55	8	Sugar flotation	Louisiana, USA	Coleman *et al.* (1989)
Foals*	68	0	Sugar flotation	Louisiana, USA	Coleman *et al.* (1989)
Foals	23	0	Histology	Colorado, USA	Snyder *et al.* (1978)
Foals	82	13	Acid-fast stain	France	Soule *et al.* (1983)
Foals	246	6	Unspecified	England	Angus (1989)
Foals	30	18	Acid-fast stain	France	Chermette *et al.* (1987)
Foals	37	6	Immunofluorescence	Ohio, USA	Xiao and Herd (1994)
Foals	29	9	Immunofluorescence	Kentucky, USA	Xiao and Herd (1994)
Foals	45	3	Unspecified	Italy	Canestri-Trotti and Visconti (1985)
Yearlings	22	18	Acid-fast stain	France	Chermette *et al.* (1987)
Yearlings	24	0	Immunofluorescence	Ohio, USA	Xiao and Herd (1994)
Yearlings	22	0	Immunofluorescence	Kentucky, USA	Xiao and Herd (1994)
Mares	48	0	Acid-fast stain	France	Soule *et al.* (1983)
Mares	49	40	Acid-fast stain	France	Chermette *et al.* (1987)
Mares	14	0	Sugar flotation	Ohio, USA	Reinemeyer *et al.* (1984)
Mares	18	0	Immunofluorescence	Ohio, USA	Xiao and Herd (1994)
Mares	53	0	Immunofluorescence	Kentucky, USA	Xiao and Herd (1994)

*Results from a 2nd year survey.

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* infection 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.* 1993a, b) 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

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; Blewett 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 *et al.* 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

recommended (Current and Garcia 1991).

Faecal smears can also be stained by using a commercial immunofluorescence stain (Meridian Diagnostics, Inc., Cincinnati, OH, USA) (Xiao and Herd 1994): oocysts stain bright apple green 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 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 diarrhoea 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 diarrhoea, 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% oocide (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; Opedenbosch 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.

References

Anderson, B.C. (1981) Patterns of shedding of cryptosporidial oocysts in Idaho calves. *J. Am. vet. med. Ass.* **178**, 982-984.

Angus, K.W. (1989) Mammalian cryptosporidiosis: a veterinary perspective. In: *Cryptosporidiosis: Proceedings of the First International Workshop*. Eds: K. W. Angus and D. A. Blewett. Moredun Research Institute, Edinburgh. pp.43-53.

Angus, K.W. (1990) Cryptosporidiosis in ruminants. In: *Cryptosporidiosis of Man and Animals*. Eds: J. P. Dubey, C. A. Speer and R. Fayer. CRC Press, boca Raton. pp.83-103.

Arrowood, M.J. and Sterling, C.R. (1989) Comparison of conventional staining methods and monoclonal antibody-based method for *Cryptosporidium* oocyst detection. *J. clin. Microbiol.* **27**, 1490-1495.

Austin, S.M., DiPietro, J.A. and Foreman, J.H. (1990) Cryptosporidium sp.: A cause of diarrhoea in immunocompetent foals. *Eq. Pract.* **12**, 10-14.

Blewett, D.A. (1989a) Quantitative techniques in *Cryptosporidium* research. In: *Cryptosporidiosis: Proceedings of the First International Workshop*. Eds: K. W. Angus and D. A. Blewett. Moredun Research Institute, Edinburgh. pp.85-95.

Blewett, D.A. (1989b) Disinfection and oocysts. In: *Cryptosporidiosis: Proceedings of the First International Workshop*. Eds: K. W. Angus and D. A. Blewett. Moredun Research Institute, Edinburgh. pp.107-115.

Canestri-Trotti, G. and Visconti, S. (1985) Inagine parasitologica su protozoi intestinali in equini dell'esercito italiano. *Atti Soc. Ital. Sci. Vet.* **39**, 758.

Chermette, R., Boufassa, S., Soule, C., Tarnau, C., Couderc, O. and Lengronne, D. (1987) La cryptosporidiose equine: une parasitose meconnue. *Bulletin CEREOPA*, 13e Journee d'Etudes, Paris, 1987, pp.81-94.

Chermette, R., Tarnau, C., Boufassa-Ouzrout, S. and Couderc, O. (1989) Survey on equine cryptosporidiosis in Normandy. In: *Coccidia and Intestinal Coccidiomorphs: Proceedings of the Vth International Coccidiosis Conference*. Ed: P. Yvore. INRA Service des Publications, Versailles. pp.493-498.

Coleman, S.U., Klei, T.R., French, D.D., Chapman, M.R. and Corstvet, R.E. (1989) Prevalence of *Cryptosporidium* sp in equids in Louisiana. *Am. J. vet. Res.* **50**, 575-577.

Cozon, G., Cannella, D., Biron, F., Piens, M.-A., Jeannin, M. and Revillard, J.-P. (1992) *Cryptosporidium parvum* sporozoite staining by propidium iodide. *Int. J. Parasitol.* **22**, 385-389.

Current, W.L. and Blagburn, B.L. (1990) *Cryptosporidium:* Infection in man and domesticated animals. In: *Coccidiosis of Man and Domestic Animals*. Ed: P. L. Long. CRC Press, Boca Raton. pp.155-185.

Current, W.L. and Garcia, L.S. (1991) Cryptosporidiosis. *Clin. Microbiol. Rev.* **4**, 325-358.

DiPietro, J.A., Kirkpatrick, C.E., Baszler, T.V., Todd, K.S., Jr and Austin, S.M. (1988) Cryptosporidium spp infection of pony foals. *Proc. 69th Ann. Mtg. Conf. Res. Work. Anim. Dis.* p.38 (abstr.)

Fernandez, A., Gomez-Villamando, J.C., Carrasco, L. Perea, A., Quezada, M. and Gomez, M.A. (1988) Brote diarreico en potros asociado a criptosporidios. *Med. Vet.* **5**, 311-313.

Gajadhar, A.A., Caron, J.P. and Allen, J.R. (1985) Cryptosporidiosis in two foals. *Can. vet. J.* **26**, 132-134.

Garcia, L.S., Brewer, T.C. and Bruckner, D.A. (1987) Fluorescence detection of *Cryptosporidium* oocysts in human faecal specimens by using monoclonal antibodies. *J. clin. Microbiol.* **25**, 119-121.

Gibson, J.A., Hill, M.W.M. and Huber, M.J. (1983) Cryptosporidiosis in Arabian foals with severe combined immunodeficiency. *Aust. vet. J.* **60**, 378-379.

Henriksen, Sv.Aa. (1989) Epidemiology of cryptosporidiosis in calves. In: *Cryptosporidiosis: Proceedings of the First International Workshop*. Eds: K. W. Angus and D. A. Blewett. Moredun Research Institute, Edinburgh. pp.79-83.

Kim, C.W. (1990) Cryptosporidiosis in pigs and horses. In: *Cryptosporidiosis of Man and Animals*. Eds: J. P. Dubey, C. A. Speer and R. Fayer. CRC Press, Boca

Raton. pp.105-111.

Lengronne, D., Regnier, G., Veau, P., chermette, R., Boufassa, S. and Soule, C. (1985) Cryptosporidiose chez des poulains diarrheiques. *Point. Vet.* **17**, 528-529.

Mair, T.S., Taylor, F.G.R., Harbour, D.A. and Pearson, G.R. (1990) Concurrent cryptosporidium and coronavirus infections in an Arabian foal with combined immunodeficiency syndrome. *Vet. Rec.* **126**, 127-130.

Opedenbosch, E.V. and Wellemans, G. (1985) Detection of antibodies to *Cryptosporidium* by indirect · immunofluorescence (drop method). *Vlaams Diergeneesk. Tijdschr.* **54**, 49-54.

Palmer, J.E. (1985) Gastrointestinal diseases of foals. *Vet. Clin. North. Am.: Equine Pract.* **1**, 151-168.

Poonacha, K.B. and Tuttle, P.A. (1989) Intestinal cryptosporidiosis in two Thoroughbred foals. *Equine Pract.* **11**, 6-8.

Reinemeyer, C.R., Kline, R.C. and Stauffer, G.D. (1984) Absence of *Cryptosporidium* oocysts in faeces of neonatal foals. *Equine vet. J.* **16**, 217-218.

Snyder, S.P., England, J.J. and McChesney, A.E. (1978) Cryptosporidiosis in immunodeficient Arabian foals. *Vet. Pathol.* **15**, 12-17.

Soule, C., Plateau, E., Perret, C., Chermette, R. and Feton, M.M. (1983) Observation de cryptosporidies chez le poulain. Note preliminaire. *rec. Med. Vet.* **159**, 719-720.

Tzipori, S. (1983) Cryptosporidiosis in animals and humans. *Microbiol. Rev.* **47**, 84-96.

Tzipori, S. (1985) The relative importance of enteric pathogens affecting neonates of domestic animals. *Ad. vet. Sci. comp. Med.* **29**, 103-206.

Tzipori, S. (1988) Cryptosporidiosis in perspective. *Adv. Parasitol.* **27**, 63-129.

Tzipori, S. and Campbell, I. (1981) Prevalence of *Cryptosporidium* antibodies in 10 animal species. *J. clin. Microbiol.* **14**, 455-456.

Weber, R., Bryan, R.T., Bishop, H.S., Wahlquist, S.P., Sullivan, J.J. and Juranek, D.D. (1991) Threshold of detection of *Cryptosporidium* oocysts in human stool specimens: evidence for low sensitivity of current diagnostic methods. *J. clin. Microbiol.* **29**, 1323-1327.

Xiao, L. and Herd, R.P. (1994) Epidemiology of equine *Cryptosporidium* and *Giardia* infections. Equine *vet. J.* **26**, 14-17.

Xiao, L., Herd, R.P. and Rings, D.M. (1993a) Diagnosis of *Cryptosporidium* infection by immunofluorescence assays on a sheep farm with neonatal diarrhea. *Vet. Parasitol.* **47**, 17-23

Xiao, L., Herd, R.P. and Rings, D.M. (1993b) Concurrent infections of *Giardia* and *Cryptosporidium* on two Ohio farms with calf diarrhea. *Vet. Parasitol.* in press.

Received for publication: 9.12.92

Accepted: 23.2.93