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Experimental transmission of *Cryptosporidium parvum* isolates from wild rodents and calves to laboratory bred common voles (*Microtus arvalis*)

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Abstract

The oocysts of *Cryptosporidium parvum* isolated from *Microtus arvalis*, *Clethrionomys glareolus* and calves were used in laboratory experiments carried out on 35 *M. arvalis*. Oocysts were detected in 43% of 14 voles infected with *C. parvum* derived from calves and in 54% of 13 voles infected with *C. parvum* isolates from *C. glareolus*. All voles inoculated with oocysts from *M. arvalis* (MA-voles) developed infection. There were observed differences in courses of infection between the three groups of voles with MA-voles demonstrating the most typical curve with an acute phase of infection in the first 4 weeks and a 2-week-long phase of self-curing. The infections with 'heterologous' *C. parvum* isolates (*C. glareolus* and calf isolates) transformed into chronic infections with lower oocyst productions. A strong host age effect was observed on prevalence of *C. parvum* among voles of different ages. Eight-month-old voles did not become infected and in older animals the infections were milder. The successful cross-transmission of two *C. parvum* isolates to *M. arvalis* suggests that this species should be considered as reservoir host for this important parasite of humans and animals.

Key words

Calves, cross-transmission, *Cryptosporidium parvum*, infectivity, isolates, *Microtus arvalis*, oocyst output

Introduction

Cryptosporidium parvum (Tyzzer, 1912) is a coccidian protozoan parasite of man and other mammals. The parasite invades epithelial cells of the intestinal tract and respiratory tree of vertebrate hosts (O'Donoghue 1995, Sturdee *et al*. 1999). Infected hosts produce huge quantities of infective stages (oocysts), which are long-living and highly resistant to standard water disinfecting methods (Fayer *et al*. 1998). Thus, in assessment of water pollution with this parasite, the crucial aim is to determine the infectivity and viability of oocysts produced by different reservoir hosts which may possess different risk for public health. Recent studies have shown that the *C. parvum* complex comprises at least two distinct species (Xiao *et al*. 2000, Morgan-Ryan *et al*. 2002). The previous *C. parvum* genotype 1 (or genotype H, arising originally from humans) is now named *C. hominis* as it is almost exclusively the parasite of humans and not infective for other mammals. The term *C. parvum* is now reserved for zoonotic genotypes of what was previously called *C. parvum* genotype 2 (or genotype C, originating originally from calves) comprising many zoonotic isolates found in livestock (cattle, horses) and in a wide range of wild animals. Recent reports on these host-specific genotype adaptations (Morgan *et al*. 2000, Caccio *et al*. 2002, Xiao *et al*. 2004) suggest the hypothesis that *C. parvum* lacks host specificity should be revised again; especially, since *C. parvum* infections in wild animals are believed to act as a source of infections for livestock (Klesius *et al*. 1986) and humans (Xiao *et al*. 2000) and many field studies revealed that wildlife rodents could be reservoir hosts for *C. parvum* (Siński *et al*. 1993; Chalmers *et al*. 1995, 1997; Webster and Macdonald 1995; Bajer *et al*. 2002).

Additionally, in our laboratory, the infectivity of rodent *C. parvum* isolates for the standard mouse model (Bednarska *et al*. 2003) showed significant differences between the courses of infection with isolates of different origin and, further-

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more, the genotype involved (Bajer *et al*. 2003) showed a high level of homology to the *C. parvum* mouse genotype (Morgan *et al*. 1999).

However, the natural routes of dissemination of rodent isolates were not studied. Voles of the two most common species, *Microtus arvalis* and *Clethrionomys glareolus*, inhabit ecologically different habitats – open grassland areas and forests, respectively, but the second species is able to explore open areas adjacent to the forest, giving the possibility for parasite exchange. On the other hand, common voles share the grassland habitats (pastures and meadows) with livestock and may serve as the source of intestinal protozoa for naive calves, lambs and other domesticated animals. In order to check this hypothesis, our preliminary cross-transmission studies revealed that *C. parvum* was infective for naive common voles (Bajer *et al*. 2000). A complex picture of parasite circulation amongst different reservoir hosts is emerging. The main goal of the present study was to evaluate the role of *M. arvalis*, the common vole, in the ecology of *C. parvum* by cross-transmission experiments to determine the infectivity of *C. parvum* isolates from woodland rodents and calves for laboratory bred *M. arvalis*.

Materials and methods

Animals

Thirty five *M. arvalis* of both sexes and of different ages were taken from the local breeding colony which had been established in Department of Parasitology in the year 1998. Before inoculation, animals were treated by following anthelmintics: Drontal (Bayer) at a dose of 250 mg/animal in drinking water, 0.02% Baycox solution (Bayer) in drinking water (0.2 ml/animal) and Metronidazol (Polpharma S.A.) at a dose of 20 mg/kg of body weight. Additionally, for the control of infectivity of *C. parvum* isolate from naturally infected calves, 6 males of C57BL/6 mice were used. Mice were treated with anthelmintic Pyrantelum (Terpol) at a dose of 100 mg/kg of body weight and Metronidazol at a dose of 20 mg/kg of body weight. During the experiment all animals were kept isolated, in individual cages with wire bottoms preventing autoinfection.

Isolates

Individual isolates of *C. parvum* oocysts were obtained from naturally infected voles (*C. glareolus* and *M. arvalis*) from Mazury Lake District, NE Poland (Bajer *et al*. 2002, Bednarska *et al*. 2003). The earlier sequence analysis of nested-PCR products on COWP gene of these two groups of isolates revealed the high similarity to 'mouse' genotype of *C. parvum* (Bajer *et al*. 2003). The *C. parvum* isolate from naturally infected calves was passaged through C57BL/6 mice and determined as *C. parvum* genotype C (Bajer *et al*. 2004).

Oocysts were concentrated using the modified Sheather's sugar flotation technique (Garcia and Bruckner 1988, Bajer *et al*. 2002), stained using direct immunofluorescent assay (IFA) (Mer*I*Fluor *Cryptosporidium/Giardia*, Meridian Diagnostics, Inc, Cincinnati, Ohio, USA) and counted using microscopy. Voles and mice were orally inoculated with 0.2 ml of the oocysts in suspension. Four experimental groups were established using 35 parasite free common voles:

(1) 13 common voles were each inoculated with 10^4 *C. parvum* oocysts originating from naturally infected bank voles *C. glareolus* (CG-voles);

(2) 14 common voles were each inoculated with 10^4 *C. parvum* oocysts originating from calves (C-voles);

(3) 5 common voles were each inoculated with 5×10^2 *C. parvum* oocysts originating from naturally infected common voles (MA-voles);

(4) 3 common voles were not inoculated to act as the control group.

A complete list of isolates, infective doses and their origin is provided in Table I.

Monitoring of infection

Faecal samples from experimental voles were collected weekly until 72 dpi. Samples were weighted and concentrated using Sheather's sugar flotation and stained with IFA. Oocysts were counted using Olympus BX70 microscopy with the filter system for fluorescein isothiocyanate (FITC) at ×1000 magnification. The daily output of oocysts was calculated per 1 g of faeces.

Statistical analysis

Comparison of the courses of infection arising from the 3 isolates, including days post infection, host sex and host age as the factors was performed by 3-way analysis of variance (3-way ANOVA) with normal errors after normalization of data by log ($\times + 1$) transformation (Bajer *et al*. 2002) using the software package Statgraphics Version 7.

Prevalence (percentage of animals infected) was analyzed by maximum likelihood techniques based on log linear analysis of contingency tables, implemented by Statgraphics Version 7. Days post infection (10 points), host sex (2 sexes) and age (4 groups) and parasite prevalence (0, 1) were entered as the factors.

Results

All 3 *C. parvum* isolates were infective to *M. arvalis*. Oocysts of *C. parvum* were detected in the faeces of 60% of 32 experimentally infected animals. The three voles from the control group remained uninfected. All C57BL/6 mice became infected with *C. parvum* isolate from calves.

Infectivity, prepatent and patent periods

Oocysts were detected in faeces of 6 from 14 (43%) C-voles infected with *C. parvum* isolate from calves and from 7 of 13 (54%) CG-voles infected with *C. parvum* isolates from *C. gla-*

Table I. Doses and oocyst output during infection with three *Cryptosporidium parvum* isolates in *Microtus arvalis*

Isolate origin/ no. of vole	Age (months)	Sex	Dose (no. of oocysts)	Result of transmission	Oocysts/g faeces mean	range
Calves						
C – 1	8	M	10000	–	–	–
C – 2	8	M	10000	–	–	0–2400
C – 3	5	M	10000	+	566	0–2288
C – 4	5	F	10000	+	656	0–4800
C – 5	5	F	10000	+	884	–
C – 6	8	M	10000	–	–	0–9600
C – 7	6	M	10000	+	1123	0–5280
C – 8	6	M	10000	+	956	0–8000
C – 9	6	M	10000	+	1078	–
C – 10	5	M	10000	–	–	–
C – 11	8	M	10000	–	–	–
C – 12	8	M	10000	–	–	–
C – 13	6	M	10000	–	–	–
C – 14	5	M	10000	–	–	–
Mean			10000		405	0–9600
C. glareolus						
CG – 1	4	M	10000	+	756	0–2164
CG – 2	4	M	10000	+	1117	0–3692
CG – 3	4	F	10000	+	1166	0–4800
CG – 4	4	F	10000	+	2206	0–19200
CG – 5	4	F	10000	+	1121	0–3600
CG – 6	8	F	10000	–	–	–
CG – 7	4	F	10000	–	–	–
CG – 8	4	F	10000	–	–	–
CG – 9	4	F	10000	–	–	–
CG – 10	4	M	10000	–	–	–
CG – 11	4	M	10000	+	1032	0–7200
CG – 12	4	M	10000	+	16	0–160
CG – 13	4	M	10000	–	–	–
Mean			10000		570	0–19200
M. arvalis						
MA – 1	4	M	500	+	1054	0–3908
MA – 2	5	M	500	+	232	0–560
MA – 3	5	M	500	+	589	0–2952
MA – 4	5	M	500	+	1288	0–4800
MA – 5	4	M	500	+	245	0–1964
Mean			500		681	0–4800

reolus. All 5 MA-voles infected with *C. parvum* isolates originating from *M. arvalis* shed oocysts (Table I). The differences in infectivity between three groups of isolates were not quite significant ($\chi^2 = 4.94$, $df = 2$, $P = 0.085$).

For 2 MA-voles, 3 C-voles and 3 CG-voles the prepatent period lasted less than 7 dpi. Three MA-voles, 3 C-voles and 4 CG-voles began oocyst shedding between 7 and 14 dpi. The patent period for MA-voles lasted 5 weeks, for C-voles 6 weeks and for CG-voles 7 weeks (Fig. 1).

Comparison of infection courses

The differences in the course of infection between 3 groups of voles infected with the three *C. parvum* isolates were on the border of significance (species of isolate origin × oocyst shedding: $F_{2,277} = 10.38$, $P = 0.06$) (Fig. 1). The highest overall mean oocyst output during the patent period was found in MA-voles infected with the homologous isolates (681 oocysts/g of faeces) with the maximum on 21 and 28 dpi of 1270 and 1760 oocysts/g of faeces, respectively. The highest individual oocyst output found in MA-voles was 4800 oocysts/g of faeces for vole MA-4 on 14 dpi, and 3908 oocysts/g of faeces for vole MA-1 on 21 dpi. The MA-voles finished oocyst shedding on 42 dpi (Fig. 1).

The intensity of infection measured as an overall mean oocyst output was about 20% lower in CG-voles (570 oocysts/g of faeces). Maximum daily oocyst output was observed in this group on dpi 14 and 21, and was 1580 and 1423 oocysts/g of faeces, respectively (Fig. 1). The overall highest daily individual oocyst output was found in CG-voles and was 19200 oocysts/g faeces for vole CG-4 on 14 dpi (Table I). Oocysts were not detected in faeces of CG-voles 72 dpi.

The lowest overall mean oocyst output was found in C-voles (405 oocysts/g of faeces). The intensity of infection measured as an overall mean oocyst output was the lowest in

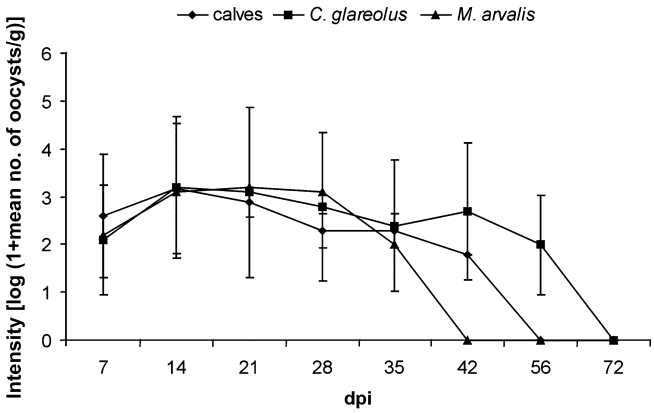

Fig. 1. Comparison of the course of *Cryptosporidium parvum* infections between isolates in experimentally infected *Microtus arvalis* (isolates × *C. parvum*; $F_{2, 277} = 10.38$; $P = 0.063$ and dpi × *C. parvum*; $F_{8, 277} = 29.90$; $P = 0.045$)

this group throughout the experiment. However, the highest daily oocyst output was 1520 oocysts/g of faeces on 14 dpi (Fig. 1) and was similar to other experimental groups. The highest daily individual oocyst outputs were 9600 oocysts/g of faeces for vole C-7 and 8000 oocysts/g of faeces for vole C-9 on 14 dpi (Table I). Oocysts were not detected in C-voles from 52 dpi. None of the voles exhibited any obvious symptoms of infection during the experiment.

Differences in prevalence and abundance of *C. parvum* between males and females were not significant but there were significant differences in prevalence or abundance of this parasite between voles of different ages ($\chi^2 = 9.47$, $df = 3$, $P = 0.024$). The age effect arose from the generally milder intensity of infection and lower prevalence among the older animals. Prevalence decreased gradually from 75% in the youngest (4 months old), through 65% in 5 months old and 50% in 6 months old, to 0% in 8 months old common voles.

Discussion

The present experimental study was the first attempt to test the infectivity of different *C. parvum* isolates for a common species of wild rodents – *M. arvalis*. With *C. parvum* isolates originating from the *M. arvalis*, *C. glareolus* and calves we reproduced possible routes of transmission in natural vole habitats used by cattle and forest rodents. The experiment suggests that host specificity is not strict for these parasite isolates and that the calf and bank vole *C. parvum* isolates are infective to more than the host species of origin. In addition, these results suggest quite complicated routes of parasite transmission in the agricultural environment and an important role for the common vole as a reservoir host of this pathogen for livestock animals and also humans.

In many transmission experiments with oocysts of livestock origin the mouse model has been used with mice of different strains and age (Bednarska *et al*. 1998, Korich *et al*. 2000, Noordeen *et al*. 2002). These revealed that *C. parvum* infections were easily transmissible from cattle to domestic mice (*Mus musculus*) but the infectivity varied with mouse strain (Sherwood *et al*. 1982, Rasmussen and Healey 1992). On the other hand, the experiment of Koudela *et al*. (1998) showed that *C. muris* oocysts isolated from cattle (probably *C. andersoni*; see Lindsay *et al*. 2000) were infective only for one species of rodents – Mongolian gerbil (*Meriones unguiculatus*) and other species, i.e., *M. arvalis*, *C. glareolus* or *Apodemus sylvaticus* remained resistant to this isolate. In our experiment in which we have used adult and not immunosuppressed animals we demonstrated that common voles were as susceptible for *C. parvum* infections as very susceptible strains of mice, e.g., C57BL/6N mice (Yang *et al*. 2000) and far more susceptible than BALB/c mice (Rasmussen and Healey 1992). Especially, young animals less than 6 months of age were susceptible hosts for this parasite and can be used for oocyst propagation in laboratory conditions.

However, far fewer laboratory studies have been done on infectivity of rodent isolates for livestock. Some of them demonstrated that *C. parvum* of domestic mouse origin was readily transmissible to livestock animals, i.e., calves and goats and also for rabbits (Klesius *et al*. 1986, O'Donoghue 1995). Successful cross-transmission of *C. parvum* has also been reported from naturally infected Siberian chipmunk (*Tamias sibiricus*) to laboratory mice but not to laboratory rats, guinea pigs and rabbits (Matsui *et al*. 2000).

In earlier experiments conducted in our laboratory we demonstrated a successful transmission of *C. parvum* isolates from three naturally infected rodent species (*C. glareolus*, *A. flavicollis* and *M. arvalis*) to C57BL/6 mice (Bednarska *et al*. 2003). However, the courses of infection differed depending on host species of origin as in the present study.

In the present study, we found some differences in the prevalence of infection and the course of infection among the 3 experimental groups, despite differences between individual isolates and the different infective doses used. The highest infectivity (100%) and the highest mean overall oocyst output were found in MA-voles infected with homologous isolates originating from common voles, despite the lowest infective doses used (Table I). The course of infection in MA-voles was fairly typical with a characteristic acute phase during the first four weeks, and a quick progressive self-cure phase in the following 2 weeks during which oocyst output fell to zero. This differs clearly from the course of infection with *C. parvum* isolates originating from common voles in C57BL/6 mice which produced chronic infections without an acute phase and self-cure process and with a lower number of excreted oocysts (Bednarska *et al*. 2003). In CG- and C-voles the acute phase appeared earlier (14 vs. 21 dpi) and was shorter, but the chronic phase characterised by continuing excretion of oocysts at a low level lasted longer and ended on 72 and 52 dpi, respectively (Fig. 1). Although generally the course of infection was very similar in these two groups of voles, the lowest infectivity and the lowest overall mean oocysts output

were found in C-voles during infection with non-rodent *C. parvum* isolate derived from calves.

Comparing our results with earlier results of Bednarska *et al*. (2003), it seems that the more 'homologous' are the *C. parvum* isolates used for cross-transmission studies, the more typical the picture of infection that appears. The course of infection with 'heterologous' isolates (i.e., derived from livestock or rodents inhabiting different habitats) is characterised by a low intensity oocyst output reflecting a chronic infection without a clear, or with a shorter, acute phase. Moreover, infections with heterologous isolates seem to avoid immunological control by the host and the self-cure process needs usually more time to complete as in the case of infections in CG-voles in our experiment (Fig. 1) or in the three groups of mice in the experiment of Bednarska *et al*. (2003).

We demonstrated that both infectivity and intensity of oocyst production decreased gradually with the age of experimental voles. The 8 months old voles did not become infected, and in 5–6 months old animals the infections were milder compared to the 4 months old ones. Similarly, during long term field studies in Mazury Lake District we observed lower prevalence and abundance of *C. parvum* infections in the oldest voles in *C. glareolus* and *M. arvalis* populations (Bajer *et al*. 2002). The probable reason for this is the maturity of the immunological system in adult individuals compared to the young ones. The same conclusion emerged from the studies carried on wild rats (Quy *et al*. 1999).

Small rodents are found commonly in urban areas, thus providing a link between rural and urban foci of different zoonotic infections. Due to the demonstrated infectivity of rodent parasite isolates for non-specific hosts (Bednarska *et al*. 2003) they could contribute to many of the sporadic human cases of cryptosporidiosis in towns and cities. Although the daily quantity of faeces excreted by rodents is small, the prevalence of *C. parvum* is often high in wildlife rodent populations (Chalmers *et al*. 1997; Siński *et al*. 1993; Bajer *et al*. 2000, 2002). Rodents spread many small droppings wherever they forage, contaminating human and animals food stuff and nearby surroundings. The increasing role of rodents as reservoir hosts for human and animal pathogens parallels the significant changes in agricultural practices in the Mazury Lake District in the last two decades. More extensive pasture practices contribute to intensified contact between animals from different habitats. The calf isolate used in this experiment was characterised previously as *C. parvum* genotype 2, which is the cause of almost 50% of human infections all over the world (Xiao *et al*. 2000, 2004; Mallon *et al*. 2003). The rodent isolates of *C. parvum* used in these experiments showed high similarity of the COWP sequence to that of the 'mouse' genotype (Morgan *et al*. 1999). And the close relationship between 'mouse' and zoonotic genotypes of *C. parvum* inform us that this isolate should be treated as potentially hazardous for human and animal health (Xiao *et al*. 2000). The successful cross-transmission of these two *C. parvum* isolates to *M. arvalis* suggests that these rodent species should be treated as an important reservoir host for this pathogen and may act as a vector in parasite transmission between forest wildlife rodents and livestock animals sharing grassland habitats with voles.

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References

Bajer A., Bednarska M., Pawełczyk A., Behnke J.M., Gilbert F.S., Siński E. 2002. Prevalence and abundance of *Cryptosporidium parvum* and *Giardia* spp. in wild rural rodents from Mazury Lake District region of Poland. *Parasitology*, 125, 21–34.

Bajer A., Bednarska M., Siński E. 2004. Molekularne badania inwazji *Cryptosporidium* spp. *Medycyna Weterynaryjna,* in press.

Bajer A., Behnke J.M., Bednarska M., Kanicka M., Siński E. 2000. The common vole (*Microtus arvalis*) as a competent host for *Cryptosporidium parvum*. *Acta Parasitologica*, 45, 178.

Bajer A., Caccio S., Bednarska M., Behnke J.M., Pieniążek N.J, Siński E. 2003. Preliminary molecular characterization of *Cryptosporidium parvum* isolates of wildlife rodents from Poland. *Journal of Parasitology*, 89, 1053–1055.

Bednarska M., Bajer A., Kuliś K., Siński E. 2003. Biological characterization of *Cryptosporidium parvum* isolates of wildlife rodents in Poland. *Annals of Agricultural and Environmental Medicine*, 10, 163–169.

Bednarska M., Bajer A., Siński E. 1998. Calves as a potential reservoir of *Cryptosporidium parvum* and *Giardia* sp. *Annals of Agricultural and Environmental Medicine*, 5, 135–138.

Caccio S., Pinter E., Fantini R., Mezzaroma I., Pozio E. 2002. Human infection with *Cryptosporidium felis*: case report and literature review. *Emerging Infectious Diseases*, 8, 85–86.

Chalmers R.M., Sturdee A.P., Bull S.A., Miller A. 1995. Rodent reservoirs of *Cryptosporidium*. *Royal Society of Chemistry Special Publication*, 168, 51–66.

Chalmers R.M., Sturdee A.P., Bull S.A., Miller A., Wright S.E. 1997. The prevalence of *Cryptosporidium parvum* and *C. muris* in *Mus domesticus*, *Apodemus sylvaticus* and *Clethrionomys glareolus* in agricultural system. *Parasitology Research*, 83, 478–482.

Fayer R., Trout J.M., Jenkins M.C. 1998. Infectivity of *Cryptosporidium parvum* oocysts stored in water at environmental temperatures. *Journal of Parasitology*, 84, 1165–1169.

Garcia L.S., Bruckener D.A. 1988. Macroscopic and microscopic examination of faecal specimens. In: *Diagnostic medical parasitology* (Eds. L.S. Garcia and D.A. Bruckener). Elsevier Science Publishing, New York, 377–391.

Klesius P.H., Haynes T.B., Malo L.K. 1986. Infectivity of *Cryptosporidium* sp. isolated from wild mice for calves and mice. *Journal of the American Veterinary Medical Association*, 189, 192–193.

Korich D.G., Marshall M.M., Smith H.V., O'Grady J., Bukhari Z., Fricker C.R., Rosen J.P., Clancy J.L. 2000. Inter-laboratory comparison of the CD-1 neonatal mouse logistic dose-response model for *Cryptosporidium parvum* oocysts. *Journal of Eukaryotic Microbiology*, 47, 294–298.

Koudela B., Modry D., Vitovec J. 1998. Infectivity of *Cryptosporidium muris* isolated from cattle. *Veterinary Parasitology*, 76, 181–188.

Lindsay D.S., Upton S.J., Owens D.S., Morgan U.M., Mead J.R., Blagburn B.L. 2000. *Cryptosporidium andersoni* n. sp. (Api-

complexa: Cryptosporiidae) from cattle, *Bos taurus*. *Journal of Eukaryotic Microbiology*, 47, 91–95.

Mallon M., Macleod A., Wastling J., Smith H., Reilly B., Tait A. 2003. Population structures and the role of genetic exchange in the zoonotic pathogen *Cryptosporidium parvum*. *Journal of Molecular Evolution*, 56, 407–417.

Matsui T., Fujino T., Kajiama J., Tsuji M. 2000. Infectivity to experimental rodents of *Cryptosporidium parvum* oocysts from Siberian chipmunks (*Tamias sibiricus*) originated in the People's Republic of China. *Journal of Veterinary Medical Science*, 62, 487–489.

Morgan U.M., Sturdee A.P., Singleton G., Gomez M.S., Gracenea M., Torres J., Hamilton S. G., Woodside D.P., Thompson R.C. 1999. The *Cryptosporidium* "mouse" genotype is conserved across geographic areas. *Journal of Clinical Microbiology*, 37, 1302–1305.

Morgan U.M., Weber R., Xiao L., Sulaiman I., Thompson R.C., Ndiritu W., Lal A., Moore A., Deplazes P. 2000. Molecular characterisation of *Cryptosporidium* isolates obtained from human immunodeficiency virus-infected individuals living in Switzerland, Kenya, and the United States. *Journal of Clinical Microbiology*, 38, 1180–1183.

Morgan-Ryan U.M., Fall A., Ward L.A., Hijjawi N., Sulaiman I., Fayer R., Thompson R.C., Olson M., Lal A., Xiao L. 2002. *Cryptosporidium hominis* n. sp. (Apicomplexa: Cryptosporidiidae) from *Homo sapiens*. *Journal of Eukaryotic Microbiology*, 49, 433–440.

Noordeen F., Horadagoda N.U., Faizal A.C., Rajpakse R.P., Razak M.A., Arulkanthan A. 2002. Infectivity of *Cryptosporidium parvum* isolated from asymptomatic adult goats to mice and goat kids. *Veterinary Parasitology*, 103, 217–225.

O'Donoghue P.J. 1995. *Cryptosporidium* and cryptosporidiosis in man and animals. *International Journal for Parasitology*, 25, 139–195.

Quy R.J., Cowan D.P., Haynes P.J., Sturdee A.P., Chalmers R.M., Bodley-Tickell A.T., Bull S.A. 1999. The Norway rat as a reservoir host of *Cryptosporidium parvum*. *Journal of Wildlife Diseases*, 35, 660–670.

Rasmussen K.R., Healey M.C. 1992. Experimental *Cryptosporidium parvum* infections in immunosuppressed adult mice. *Infection and Immunity*, 60, 1648–1652.

Sherwood D., Angus K.W., Snodgras D.R., Tzipori S. 1982. Experimental cryptosporidiosis in laboratory mice. *American Society for Microbiology*, 38, 471–475.

Siński E., Hlebowicz E., Bednarska M. 1993. Occurrence of *Cryptosporidium parvum* infection in wild small mammals in District of Mazury Lakes (Poland). *Acta Parasitologica*, 38, 59–61.

Sturdee A.P., Chalmers R.M., Bull S.A. 1999. Detection of *Cryptosporidium* oocysts in wild mammals of mainland Britain. *Veterinary Parasitology*, 80, 273–280.

Webster J.P., Macdonald D.W. 1995. Cryptosporidiosis reservoir in wild brown rats (*Rattus norvegicus*) in the UK. *Epidemiology and Infection*, 115, 207–209.

Xiao L., Morgan U.M., Fayer R., Thompson R.C., Lal A.A. 2000. *Cryptosporidium* systematics and implications for public health. *Parasitology Today*, 16, 287–292.

Xiao L., Fayer R., Ryan U., Upton S.J. 2004. *Cryptosporidium* taxonomy: recent advances and implications for public health. *Clinical Microbiology Reviews*, 17, 72–97.

Yang S., Benson S.K., Du C., Healey M.C. 2000. Infection of immunosuppressed C57BL/6N adult mice with a single oocyst of *Cryptosporidium parvum*. *Journal of Parasitology*, 86, 884–887.

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