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Roger J. Quy,[1,5] **David P. Cowan,**[1] **Peter J. Haynes,**[1] **Anthony P. Sturdee,**[2] **Rachel M. Chalmers,**[3] **Adam T. Bodley-Tickell,**[2] **and Stephanie A. Bull**[4]

[1] Central Science Laboratory (MAFF), Sand Hutton, York, YO41 1LZ, UK

[2] Cryptosporidium Research Group, School of Natural and Environmental Sciences, Conventry University, Priory Street, Coventry, CV1 5FB, UK

[3] CDSC (Wales), Abton House, Wedal Road, Cardiff, CF4 3QX, UK

[4] S. W. W. Scientific Services, Microbiology Laboratory, Bridge Road, Countess Wear, Exeter, EX2 7AA, UK

[5] Corresponding author (e-mail: r.quy@csl.gov.uk)

ABSTRACT: The potential of Norway rats (*Rattus norvegicus*) to spread the parasite *Cryptosporidium parvum* was investigated by examining parasite prevalence in relation to the structure and movements of three permanent rat populations living on farmland in Warwickshire (UK) from October 1994 to March 1997. One population lived among a group of farm buildings housing cattle, while the other two had no contact with livestock, one living around a pond and its outflowing stream and the other on a rubbish tip. Overall, parasite occurrence was 24% (n = 438), but it varied according to body weight (age) with 40% of juveniles ($\leq$100 g) infected decreasing to 12% for adults >400 g, suggesting that actively breeding populations are potentially more likely to spread the parasite than non-breeding populations. There was no difference in prevalence between the three populations. The parasite was detected in more males (29%) than females (19%). Seasonally, on the livestock farm, prevalence was significantly lower in autumn (10%), but varied little (31–36%) from winter to summer. In contrast, on the arable farm, prevalence peaked in summer (50%) with a trough in winter (6%). Infection in rats appeared to last <67 days. Rats living on the livestock farm had home ranges largely confined to the cattle sheds, thereby maintaining a potential source of infection for livestock if rodent control was not part of a decontamination program. Equally, rats living around the pond on the arable farm provided a source of oocysts to contaminate the pond water, as well as being able to carry the parasite to nearby farm buildings or even to neighboring farms.

Key words: Cryptosporidium parvum, fecal samples, mark-recapture, radio-tracking, *Rattus norvegicus.*

INTRODUCTION

Cryptosporidium parvum, a protozoan parasite, has received attention in recent years as the etiological agent responsible for the gastrointestinal disease cryptosporidiosis in both humans and farmed animals worldwide (Casemore, 1990; for recent review, Fayer, 1997). Spread via the fecal-oral route, the contamination of potable water with viable oocysts has resulted in waterborne outbreaks in the human population, such as in Sheffield (UK) in 1986 when livestock, notably cattle, were suspected as the primary source of the parasites (Smith and Rose, 1990). However, little attention has been given to wildlife hosts as possible vectors of the disease. Rodents are known to carry a variety of organisms that may cause diseases in human and domestic animal populations

(Gratz, 1994). Direct contact with excreta from infected rodents can cause disease, but the potential for widespread dissemination of *C. parvum* lies in the fecal contamination of food or water supplies used by people and other animals. One rodent species, the Norway rat (*Rattus norvegicus*), is well known for its close association with human activities and the parasite was first detected in rat feces in Japan (Iseki, 1986). Since then, a survey in the UK found six out of nine farm rat populations infected (Webster and Macdonald, 1995). Neo-natal calves appear to be particularly susceptible to cryptosporidiosis (Tzipori, 1983) and rats sometimes live close to them or their food supply. As each rat may produce about 40 droppings a day (Meehan, 1984), a possible route of transmission is available. Rats are also good swimmers and they have often been implicated

in cases of other diseases, such as leptospirosis, in humans following contact with contaminated water (Gratz, 1994). Therefore, rats may be an important wildlife reservoir of cryptosporidiosis, but while this presents yet another reason to control their populations, previous studies did not assess the likelihood of people or animals becoming infected through oocysts shed by rats. This will depend as much on rat population dynamics and behavior, as on the life cycle of the parasite.

Farms frequently offer favorable conditions for rat populations to thrive and in 1993 a random survey of English and Welsh agricultural properties found 42% of 133 sites infested with rats (Meyer et al., 1995). Potentially, such environments could support many hundreds of rats, although control measures are usually instigated before the maximum carrying capacity has been reached. Whatever the short-term effectiveness of these measures at reducing infestations, reproduction by survivors and immigration from surrounding areas may offset some, or all, of the losses. This may result in a high turnover of individuals within the population, with perhaps consequential variability in the prevalence of a parasite, particularly if the hosts remain unaffected as rats appear to be when carrying *C. parvum* (Iseki, 1986). Alternatively, if the parasite is shed from its host in an environmentally-resistant form (such as an oocyst), recruits to the population may be easily infected and the prevalence may remain relatively stable.

In this study, we regularly trapped rats over 2.5 yr from three permanent populations, one living on a livestock farm and two along a watercourse and on a rubbish tip at an arable farm. The main objectives were to quantify the prevalence of *C. parvum* in rats in the presence and absence of livestock; determine variations in prevalence in relation to habitat, season, population size and structure; and assess the likelihood of infected rats spreading the parasite by contact with livestock and watercourses.

MATERIALS AND METHODS

Three permanent rat populations were located on two farms approximately 5 km apart in the county of Warwickshire (UK). One population lived in and around the farm buildings at Warwickshire College at Moreton Morrell (52°12′N, 1°34′W) which covered an area of about 2 ha and housed dairy cattle, beef bulls, calves, and sheep. Proprietary animal feeds and, depending on season, maize and grass silage also were stored in the buildings; the latter two were stored in plastic-sheet-lined clamps. Straw bales were stacked in and around some buildings depending on the time of year and the area also included a slurry lagoon. Rats were present at all times throughout the study, but the farm staff and a pest control contractor carried out occasional poison treatments using anticoagulant rodenticides. Other colonies of rats, apparently transitory, were located and sampled whenever opportunities arose: these included animals living in a game-rearing unit (0.45 ha approximately) 100 m from the farm buildings, a stable block 600 m away and a small wood (spinney) between two maize fields 500 m away.

The other two permanent rat populations were located on the second farm (52°10′N, 1°32′W) which, apart from a few sheep, did not keep livestock. One population lived around the banks of a small pond (0.13 ha approximately including the banks) and its outflowing stream and the other on a rubbish tip about 600 m away. Throughout the study, both of these sites were surrounded by fields in which either wheat or oil seed rape was grown. According to the farmer, no manure or animal slurry was applied to these fields, only chemical fertilizers. Water levels in the pond were maintained by rainwater and drainage from the surrounding fields and a nearby vehicle testing facility. No specific measures were taken by the farmer to control the rats at the pond or tip, although the tip was occasionally set on fire to burn the rubbish. At times during the winter months, a metal drum feeder filled with wheat was left on the banks of the pond to attract game-birds, but it was also used by rats. Occasional poison treatments were carried out against rats living around the farm buildings, which were about 450 m from both the pond and the tip. Rat colonies found elsewhere on the farm were also sampled for as long as each colony persisted. One of these (referred to hereafter as the "stream" population) became established 700 m downstream from the pond and about 200 m from the buildings. In the second year of the study, this part of the stream was dug up and two ponds were formed; the

area around these ponds was quickly recolonized by rats when the excavations ceased.

Trapping was carried out at 3 to 6 wk intervals from October 1994 until March 1997. Single-capture live-traps baited with whole wheat were laid unset for 1 wk at all sites where rat signs were discovered. The traps were then set for two nights the following week, with an additional two nights trapping the week after if <10 animals had been caught. A sample of three to four fresh fecal pellets was collected from each rat and stored in a sterile screw-topped pot into which drops of distilled water were added to keep the feces moist. If rats were found dead in the traps without fresh feces, the animals were dissected and a sample of the intestinal contents collected from the rectum or cecum. All rats caught at Warwickshire College and all those caught on the arable farm after April 1995 were transferred to an inhalation chamber and anesthetized with diethyl ether (towards the end of the study this was replaced with isoflurane (Abbot Laboratories Ltd., Queenborough, Kent, UK)). When unconscious, each animal was weighed, sexed, the head + body length recorded and a passive integrated transponder (Trovan ID 100 microtag, RS Biotech, Northants, UK) implanted subcutaneously between the shoulder blades (Quy and Cowan, 1996). Each transponder transmits a unique identification number when energized by an electromagnetic field. After full recovery from the anesthetic, each rat was released at its point of capture. In subsequent trapping sessions, recaptures were identified and a new fecal sample was tested, enabling a history of the infection in those individuals to be obtained. Before April 1995, all rats (except for 13 which were radio-tagged and released) caught on the arable farm were humanely killed after a fecal sample was obtained.

To follow the movements of potentially infected rats, each healthy animal ≥300 g was also fitted with a radio-transmitter (Biotrack, Wareham, Dorset, UK) while under the anesthetic. The radio-tag contained a thermistor which monitored body temperature. A fall in temperature increased the tag's pulse rate, which indicated either that the rat was dead or that the tag had come off. Every effort was made to recover tags from rats presumed dead. Tracking was carried out 1 to 2 wk after each trapping session, using a Yagi antenna attached to a Televilt RX900 receiver (Televilt Intern. AB, Lindesberg, Sweden). Daytime refuges were located for each tagged rat, while movements were followed mostly at night. Fixes were obtained by triangulation, by first finding the strongest signal in one direction, then moving to another place and estimating where the

line of the strongest signal in the second place intercepted the first. The estimated position of the animal was recorded as a grid reference to the nearest 1 m. Fixes were obtained every 6 hr, but occasionally more frequently if an individual was particularly active, or less frequently if its position seemed not to alter. The radio-tracking data were analyzed using Ranges V software (ITE, Wareham, Dorset, UK). Home ranges for individuals were calculated by the minimum convex polygon method (Mohr, 1947) for each rat with >10 fixes. Areas occupied by populations, divided by sex, season and site, were calculated by the same method using all fixes from all tagged rats. Differences between the proportions of fixes associated with site and season were examined by chi-square analysis with Yates' correction in 2×2 tables (Campbell, 1974). The level of significance α in all statistical tests was $P \leq 0.05$.

The number of rats living around the farm buildings and in colonies elsewhere on the livestock farm was estimated immediately before each trapping session using an established tracking plate technique (Quy et al., 1993). The buildings and adjacent land were surveyed first and fresh signs of rats (feces, runs, active burrows) noted. Tracking plates were distributed on each occasion to match the distribution of these signs. Daily track scores were obtained for four consecutive days and the mean score used in the calibration curve to obtain a point estimate of population size.

Cryptosporidial oocysts were concentrated from the feces of individual rats using a modified formol-ether sedimentation technique (Casemore et al., 1985) adapted for use with a fecal parasite concentrator kit (Evergreen Scientific, Los Angeles, California, USA). Samples were stained with an immunofluorescent antibody test (IFAT), using a genus-specific monoclonal antibody according to the manufacturer's instructions (Shield Diagnostics, Dundee, UK). Samples found to be positive by IFAT were also tested using a modified Ziehl-Neelson (MZN) stain and examined by bright-field microscopy at 400× and 1000× (Casemore et al., 1985). A calibrated eye piece graticule was used to measure oocysts and confirm the species (Chalmers et al., 1994). Size and shape distinguished *C. parvum* from *C. muris*, the former 4 to 5 μm in diameter and circular in profile, the latter ovoid 5 to 6 μm by 7 to 8 μm (Bull et al., 1998). The number of oocysts in each sample was assigned to an arbitrary density assessment as high (>50 oocysts per 20 μl of fecal concentrate), medium (20–50) and low (<20). The limit of detection was about 3×10^3 oocysts per g feces, determined by calibration experi-
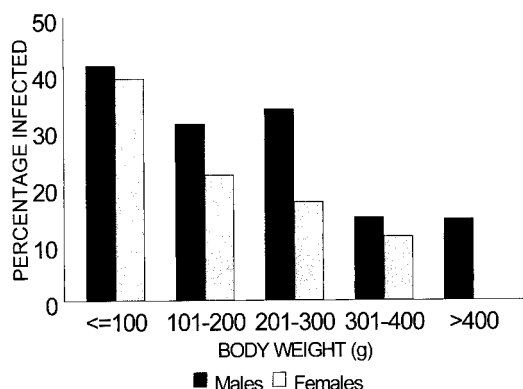
FIGURE 1. Prevalence of *Cryptosporidium parvum* in 438 Norway rats trapped on two farms in Warwickshire (UK) between October 1994 and March 1997 in relation to body weight group (g) and sex.

ments using fecal material spiked with known numbers of oocysts (Chalmers, 1996).

To facilitate data analysis, the rats were assigned to 100g body weight classes. These classes, although arbitrary, corresponded approximately to juveniles (≤100 g), young adults becoming sexually mature (101–200 g, 201–300 g) and mature adults (301–400 g, >400 g) (Bishop and Hartley, 1976). Since parasite prevalence in the population samples was related to the body weight distribution, comparisons between samples in relation to different locations, populations, seasons, and years had to take account of possible differing body weight distributions. Thus, in contingency tables, the expected number infected in each sample was obtained by multiplying the number of rats examined in each weight group by the proportion infected in the corresponding weight group of the entire data set and summing the results (Leslie et al., 1952). Although some rats were trapped in two or more sessions, the rats trapped in each session were assumed to be an independent sample of the population existing at that time. It is likely, however, that the trapping produced an unavoidably biased sample with respect to age/weight, because some small rats were probably not heavy enough to spring the traps.

RESULTS

Four hundred and thirty-eight samples were tested from 354 individual rats (189 males, 165 females), 155 samples from the livestock farm and 283 from the arable farm. The prevalence of *C. parvum* was related to body weight for each sex (χ^2 = 9.5, 3 df, P = 0.02 males; χ^2 = 9.0, 3 df, P = 0.03 females; two heaviest weight groups combined), with 40% of juveniles (≤100 g) infected decreasing to 12% for adults >400 g (Fig. 1). Overall occurrence was 24% (105/438) (Table 1), with no difference between the three permanent populations (χ^2 < 1, 2 df, P > 0.05) after adjusting for body weight. The density of oocysts was classified as low in 91% (95/105) of the samples. Overall, pooling data for all populations, more males than females were infected (χ^2 = 5.01, 1 df, P = 0.025); the body weight distributions of males and females did not differ. Resampling of recaptured rats did not appear to contradict the prevalence figures: sample occurrence, 29% males, 19% females; 29%

TABLE 1. Number of Norway rats infected with *Cryptosporidium parvum* on two farms in Warwickshire (UK) between October 1994 and March 1997.

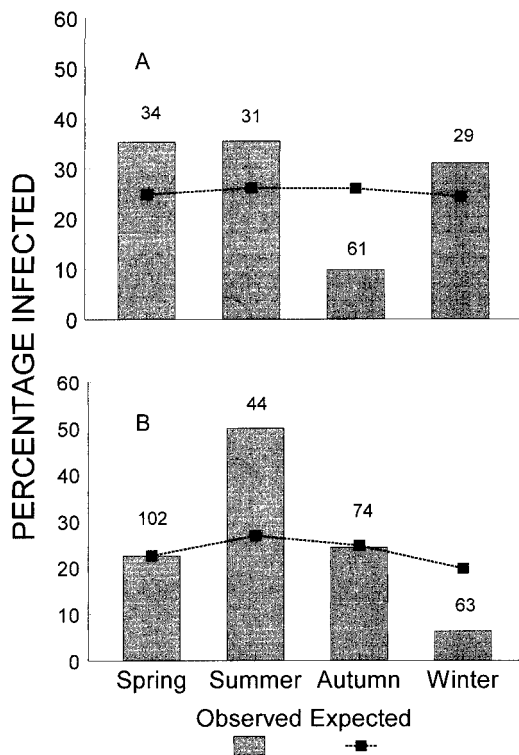| Site	Number of rats trapped	Number infected (%)
Livestock Farm		
Permanent population 1: Farm buildings	137	35 (26)
Additional sites: Stables, game unit, spinney	18	3 (17)
Subtotal	155	38 (25)
Arable farm		
Permanent population 2: Pond	169	41 (24)
Permanent population 3: Rubbish tip	86	21 (24)
Additional site: Stream	28	5 (18)
Subtotal	283	67 (24)
Grand total	438	105 (24)

FIGURE 2.   Seasonal prevalence of *Cryptosporidium parvum* in rats on (A) the livestock farm and (B) the arable farm. The expected values were calculated to allow for variations in body weight distributions between samples. The numbers above each bar are the number of animals tested in each season. Spring (March–May), summer (June–August), autumn (September–November), winter (December–February).

of males and 20% of females were found to be infected at their first capture. Seasonal differences in parasite prevalence were evident in the rats from the livestock farm ($\chi^2 = 19.1$, 3 df, $P < 0.001$) and those from the arable farm ($\chi^2 = 12.4$, 3 df, $P < 0.01$), but the trends were different on the two sites. After allowing for body weight differences, on the livestock farm there were significantly fewer infected rats (10%) than expected in the autumn and no obvious peak in prevalence at other times (winter 31%, spring 35%, summer 36%), whereas on the other farm most rats (50%) were infected in summer and least (6%) in winter (Fig. 2).

Prevalence of *C. parvum* was compared between two complete years, 1995 and 1996, for each of the three permanent rat populations. It appeared to increase in samples collected from the pond and tip rats from 23% ($n = 35$) to 28% ($n = 104$) and 18% ($n = 50$) to 39% ($n = 26$) respectively and decrease in samples from the livestock farm from 31% ($n = 52$) to 20% ($n = 70$). However, the differences were not significant (pond and farm, $\chi^2 < 1$ 1 df, $P > 0.05$; tip, $\chi^2 = 2.57$, 1 df, $P > 0.05$) and probably reflected changes in the distributions of body weights of the trapped rats (Fig. 3).

Of 279 rats tagged and released, 60 were recaptured in one or more later trapping sessions (Table 2); three were positive on each of two consecutive recaptures after 28, 44, and 49 days respectively; one rat was positive on each of three consecutive recaptures with 154 and 28 days respectively between the captures.

Estimates of the size of the rat population living in and around the buildings on the livestock farm ranged from 10 to 51 animals. The mean of six estimates for 1995 was 44.5 (34 to 53) and the mean of seven estimates for 1996 was 33.4 (19 to 48), with a gradual decline over the duration of the study. When body weight differences were taken into account, there was no difference between the prevalence of *C. parvum* in the population when it was below the overall mean of 36 rats (22%) and when it was above the mean (28%; $\chi^2 < 1$, 1 df, $P > 0.05$). Colonies elsewhere on the farm consisted of 2 to 18 animals and apparently disappeared in 2 to 4 mo.

Twenty-eight rats (11 males, 17 females) caught on the livestock farm and 56 (31 males, 25 females) on the arable farm were fitted with radio-transmitters. Home ranges were estimated for 34 rats each with 12 to 144 fixes obtained over 3 to 144 days (mean 53 days) (Table 3). On the livestock farm the calculated ranges for individuals and those grouped by sex and season were contained wholly within the area (2.45 ha approximately) covered by the farm buildings and the adjacent game-

1995

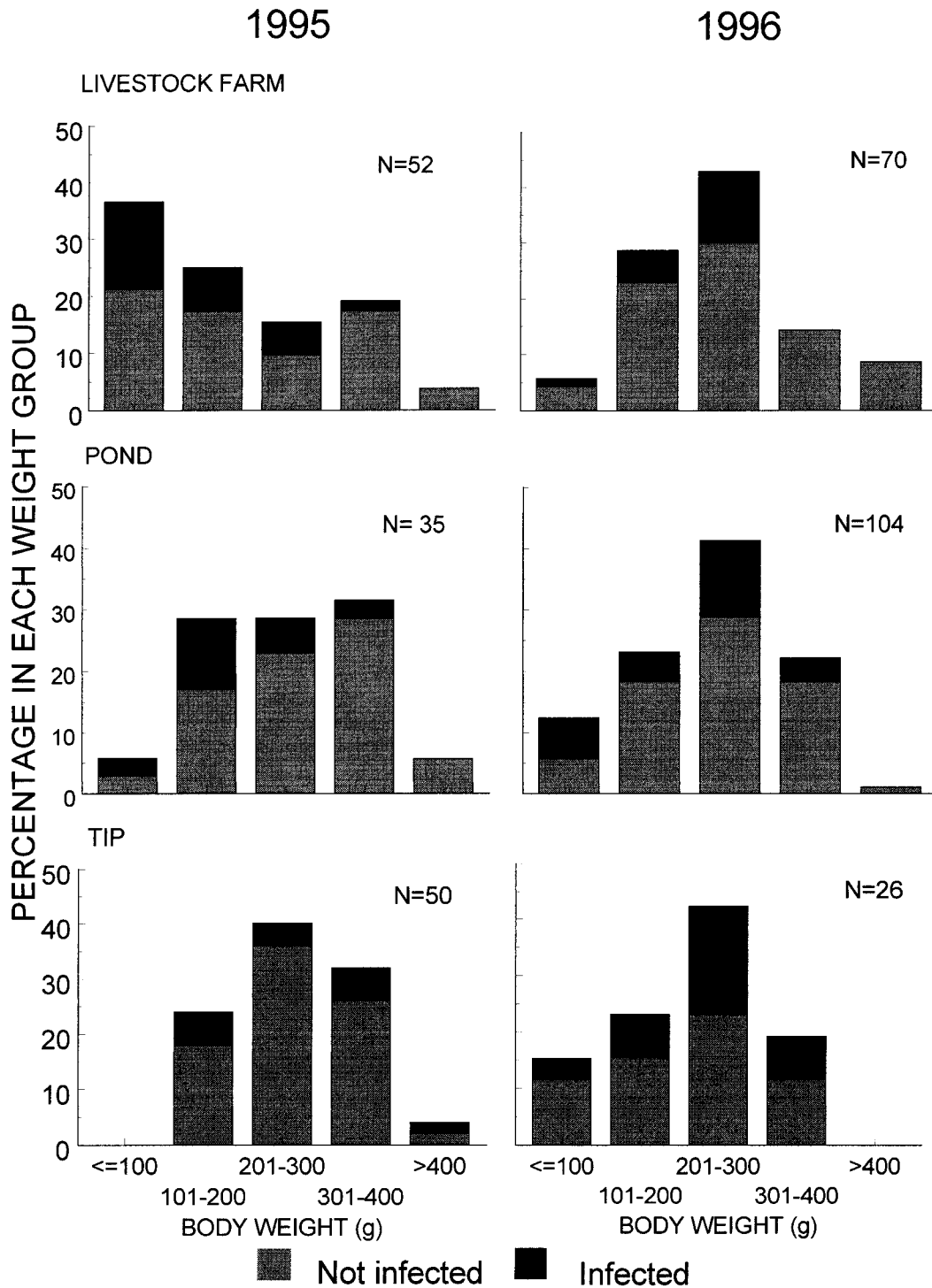## 1996

LIVESTOCK FARM

POND

TIP

FIGURE 3.   Annual comparison of *Cryptosporidium parvum* prevalence in three permanent (farm buildings, pond and rubbish tip) rat populations. N is the number of samples.

TABLE 2.   Number of rats, marked and released during the study, testing positive for *Cryptosporidium parvum* at their first and subsequent recaptures. The mean time between captures for 60 rats caught two or more times was $\bar{x} \pm SE = 66.8 \pm 4.6$ days (range 20–204).

Number of times caught	Not infected	Number of times positive			Total infected	Total
		1	2	3		
1	158	61	—	—	61	219
2	24	13	2	—	15	39
3	6	11	1	1	13	19
4	2	—	—	—	—	2
Totals	190	85	3	1	89	279

rearing unit: 77% ($n = 589$) of the fixes were located within cattle sheds and cattle feed stores. On the arable farm, depending on sex and season, the calculated range of rats tagged at the pond was 1 to 142.3 times the area of the pond (0.13 ha approximately): 88% ($n = 370$) and 70% ($n = 157$) of the fixes from the pond and stream rats respectively were located either in or by the water. There was a greater proportion of fixes from females associated with cattle than those from males ($\chi^2 = 37.4$, 1 df, $P < 0.001$; Table 3); similarly, more fixes from females were located by water (pond and stream fixes combined) than from males ($\chi^2 = 24.7$, 1 df, $P < 0.001$). The proportion of fixes associated with cattle did not vary with the season ($\chi^2 = 4.9$, 3 df, $P > 0.05$); the proportion associated with water on the arable farm varied significantly from 33% in the summer to 94% in winter ($\chi^2 = 117.2$, 3 df, $P < 0.001$). There were insufficient data to calculate ranges for the tip rats due to the apparently high disappearance rate of animals living there. Movements between distinct habitats were recorded: two male rats from the stream population that were tracked over 39 and 43 days respectively, moved to the farm buildings, a straight-line distance of about 200 m; a female released at the pond and tracked over 32 days moved to a field boundary approximately 650 m away and then back to the pond; a female first caught in November 1995 at the pond was located on a neighboring farm 600 m away in April 1996 when it was killed by the farmer; a male released in the spinney moved at least 500 m within 13 days to the game unit on the livestock farm; a juvenile male, marked with a microtag and released at the tip was recaptured three months later 650 m away at the pond.

TABLE 3.   Home ranges (ha) of rats based on the minimum convex polygon method for individuals with >10 fixes and, by pooling all fixes for all rats, for each sex and season. Below each range grouped by sex and season, the percentage of fixes found in cattle sheds on the livestock farm and by water on the arable farm are shown.

Home range	Livestock farm		Arable farm	
	Farm buildings	Game unit	Pond	Stream
Male	1.6		1.51	2.99
% fixes by cattle/water (n)	53.8 (106)		83.2 (197)	64.1 (128)
Female	1.50		18.2	0.50
% fixes by cattle/water (n)	82.0 (483)		92.5 (173)	96.6 (29)
Spring (Mar–May)	1.69		18.5	1.85
% fixes by cattle/water (n)	77.0 (261)		92.8 (207)	58.5 (65)
Summer (Jun–Aug)	0.82		1.28	0.39
% fixes by cattle/water (n)	73.1 (108)		12.1 (33)	60.0 (25)
Autumn (Sep–Nov)	0.99		0.13	2.35
% fixes by cattle/water (n)	83.0 (135)		98.4 (61)	77.8 (18)
Winter (Dec–Feb)	1.39		0.20	1.53
% fixes by cattle/water (n)	71.8 (85)		98.6 (69)	87.5 (49)
Mean individual ± SE	0.07 ± 0.02	0.16	0.39 ± 0.17	0.28 ± 0.11
(n)	(12)	(1)	(16)	(5)

DISCUSSION

The discovery in the UK of *C. parvum* in at least four species of rodents including the house mouse (*Mus domesticus*), wood mouse (*Apodemus sylvaticus*), bank vole (*Clethrionomys glareolus*) and Norway rat suggests that a significant wildlife reservoir exists (Chalmers et al., 1997; Webster and Macdonald, 1995). Rats and house mice are of particular concern, because of their close association with human activities and therefore the potential to transmit the parasite to humans and livestock. Although rat numbers have probably declined in the UK over the last 100 yr following habitat changes and improvements in control techniques (Harris et al., 1995), this species is highly adaptable and many farms still retain the potential to support very large populations (Quy et al., 1995). The livestock farm, which was part of an agricultural college, was perhaps atypical of most farms in that relatively undisturbed places with suitable harborage for rats were limited. Nevertheless, the rat population persisted throughout the study period, although demographic changes in the latter half suggested that the population might have been declining (Fig. 3). The decrease in the number of juvenile rats caught reflected either higher mortality or reduced birth rates and could have been caused, in part, by the rodenticide treatments. That the population censuses also indicated a fall in numbers suggested that changes in trapability were not responsible. The apparent fall in the prevalence of *C. parvum* on the livestock farm between 1995 and 1996 can therefore be accounted for by changes in the age (weight) structure. Thus, populations of similar size, but different age structures, may have different infection rates with, consequently, different risks regarding the likelihood of transmitting the parasite, solely from the number of infected rats at large. The relationship between body weight (age) and the prevalence of *C. parvum* has been demonstrated in *Rattus rattus* (Miyaji et

al., 1989), but Webster and Macdonald (1995) did not find it in a sample of 73 *R. norvegicus.*

The rats living on the arable farm were not subjected to the same degree of continual disturbance from farming activities as those on the livestock farm. Nest sites for rats living around the pond were largely undisturbed, but harvesting of crops in the surrounding fields followed by ploughing meant disruption to the food supply. The tip rats were even less fortunate, as those living near the tip face were exposed to frequent fires and infilling thereby destroying harborage. Both of these populations might have been more susceptible to the effects of severe weather and predators, compared with those living in the relatively sheltered environment provided by farm buildings. Norway rats are capable of breeding all year round if there is a stable supply of food and cover (Leslie et al., 1952) and the appearance of a large proportion of juveniles (i.e., rats ≤100 g body weight) in the catch would suggest recently successful breeding. Furthermore, with an age related effect, an apparent increase in the prevalence of the parasite would be expected, but more rats were found to be infected in the summer than at other times, after allowing for the presence of juveniles. Older (heavier) rats were becoming infected more often than in the other seasons. Whether this was caused by the presence of juveniles or not is unknown, but a temporary increase in population density and ranging behavior might have led to more frequent animal to animal contact and hence offered more scope for transmitting the parasite, particularly if there was more contact between infected and susceptible individuals. Perhaps the absence of a substantial increase in either population density and/or ranging behavior prevented a significant increase in the number of infected rats on the livestock farm during the summer; this may have been caused by a combination of habitat factors and rodent control measures in and around the farm buildings. Different sea-

sonal trends in rats have been reported, with spring and winter peaks and a summer trough (Webster and Macdonald, 1995) and, for other rodents, no consistency in seasonal trends between years (Sinski et al., 1993). Chalmers et al., (1997) reported autumnal peaks in the prevalence of *C. parvum* in house mice, wood mice and bank voles. House mice were present in the farm buildings on the livestock farm during the course of this present study and might have visited the same places as some of the rats. Therefore, it is surprising that a possible peak occurrence of the parasite in mice coincided with the lowest incidence in rats. Perhaps rats have to eat mice or mouse droppings in order to become infected, which they may not do when other food is plentiful, as is often the case on livestock farms throughout the year. Alternatively, prevalence among rats might coincide with shedding of the parasite by infected livestock. Calves were present between the months of September and January each year and it was known that 39% were infected during the period of this study. However, prevalence among adult cattle was <10% and rat activity was more closely associated with them than with the calves.

Of those rats found to be infected, most were excreting numbers of oocysts equivalent to or just above the level of detection ($\geq 3 \times 10^3$/g feces). The length of time over which oocysts of *C. parvum* could be shed was estimated from four rats found to be infected on consecutive captures. This varied from 28 to 182 days, but assumes that those rats were infected continuously between captures and had not lost the parasite and then acquired new infections. Most of the infected rats that were recaptured were positive only once (24/28; Table 2), suggesting that the parasite would have been detectable for less than the average time between captures of 67 days. A roof rat (*R. rattus*) that shed *C. parvum* oocysts for over six weeks has been reported (Miyaji et al., 1989). Although young rats were more likely to be infected than adults, 11 rats apparently became infected when adult after testing negative at their first capture, when they were either juvenile or already adult.

In order for infected rats to pass on the parasite to other animals, their feces must presumably contaminate food, water or surfaces used by any susceptible individuals. On the livestock farm most tagged rats were located in and around a single large building divided into dairy cow cubicles, calving pens, a lambing shed and silage store with an adjacent stack of straw bales. Direct physical contact between tagged rats and livestock or their feed was not observed, because there was so much cover that it was difficult to spot rats even when their location had been determined. All individual and group home ranges were within the area covered by the farm buildings and the adjacent game-rearing unit with most fixes recorded from the buildings used for housing cattle. It is possible to speculate on how many oocysts infected rats might shed into the environment. Assuming each animal voids 40 droppings each weighing 0.5 g and that there are a minimum of 3,000 oocysts per g, one individual could release 60,000/day. If a farm was infested with an average population of 100 rats of which at least 24 were infected at any one time, nearly 526 million oocysts could contaminate the area occupied by those individuals each year. The mean home range of rats occupying the buildings on the livestock farm was 700 m^2, thus if all infected rats occupied the same area, the rate of contamination would be about 751,000 oocysts/m^2. If cattle were infected also and rats rarely moved outside the building containing them, it could be argued that the rats acquired the parasite from infected livestock. Equally, eliminating the infection in livestock without also eliminating the rats might be a wasted effort, particularly as the infective dose appears to be low (Blewett et al., 1993; Dupont et al., 1995). Similarly, spot treatments to clean up watercourses might also fail. Rats living

around the pond on the arable farm did not always limit their activities to the pond boundary. Food sources were often intermittent and for limited periods abundant, particularly in summer when rats moved into the fields to feed as the crops matured. However, most fixes were recorded from the pond surrounds and rats were occasionally seen swimming in the water and fresh droppings were found close to the water's edge. A similarly speculative assessment of the potential contamination of the pond habitat, including the water, results in 135,000 oocysts/m^2, lower than the farm buildings because the rats had larger home ranges. The relatively large home ranges calculated for females at the pond and in the spring (Table 3) were caused by an outlying fix for one rat that moved 650 m away and then back again. Taylor (1978) recorded movements of tagged rats living away from farm buildings and calculated mean ranges of 340 m for females and 660 m for males: some of those rats included farm buildings in their home range but they nested 500 m away. Such movements would enable *C. parvum* to be transferred to farm buildings that had been decontaminated. No pond rats moved to the buildings on the arable farm, but two rats did so from the stream population. These movements appeared to be permanent changes in homesite. Eliminating potential sources of immigrants simultaneously with eradication of an infected rat population, although ideal, is rarely practicable.

The results showed that rats living in different environments had strikingly similar infection rates. This suggests that factors other than transfer from other species may be important in the maintenance of the parasite in rat populations. The livestock farm used in this study had no history of cryptosporidiosis outbreaks and it is likely that the observed incidence of the parasite represented background levels. Rats that did not live near livestock or watercourses were just as likely to be infected and therefore this species should be seen as a significant vector of *C. parvum*. The

infection appears to be relatively short-lived in the majority of animals with juveniles being particularly susceptible. How they become infected is not certain, whether from other rats or other rodents, but actively breeding populations with good survival of young should be seen as potentially more of a hazard than non-breeding populations. Factors not studied here, but which should also be considered, are the viability of oocysts shed by rats and any similarity between strains of the parasite found in rats and those in livestock and humans. Recent studies have suggested that some isolates of the parasite from humans are genetically distinct from those found in calves (Awad-El-Kariem et al., 1998) and that person-to-person transmission may be more common than animal-to-human transmission. However, it is not clear that investigation of outbreaks of cryptosporidiosis among humans not involving livestock, directly or indirectly, has always eliminated commensal rodents as a source, given that they are widespread in both urban and agricultural areas.

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LITERATURE CITED

AWAD-EL-KARIEM, F. M., H. A. ROBINSON, F. PETRY, V. MCDONALD, D. EVANS, AND D. CASEMORE. 1998. Differentiation between human and animal isolates of *Cryptosporidium parvum* using molecular and biological markers. Parasitology Research 84: 297–301.

BISHOP, J. A., AND D. J. HARTLEY. 1976. The size and age structure of rural populations of *Rattus norvegicus* containing individuals resistant to the anticoagulant poison warfarin. Journal of Animal Ecology 45: 623–646.

BLEWITT, D. A., S. E. WRIGHT, D. P. CASEMORE, N. E. BOOTH, AND C. E. JONES. 1993. Infective dose size studies on *Cryptosporidium parvum* using gnotobiotic lambs. Water Science and Technology 27: 61–64.

BULL, S., R. CHALMERS, A. P. STURDEE, A. CURRY, AND J. KENNAUGH. 1998. Cross-reaction of an anti-*Cryptosporidium* monoclonal antibody with sporocysts of *Monocystis* species. Veterinary Parasitology 77: 195–197.

CAMPBELL, R. C. 1974. Statistics for biologists. Cambridge University Press, London, UK, 385 pp.

CASEMORE, D. P. 1990. Epidemiological aspects of human cryptosporidiosis. Epidemiology and Infection 104: 1–28.

————, M. ARMSTRONG, AND R. L. SANDS. 1985. Laboratory diagnosis of cryptosporidiosis. Journal of Clinical Pathology 38: 1337–1341.

CHALMERS, R. M. 1996. The distribution of *Cryptosporidium* in livestock and wild animal populations on a Warwickshire farm. Ph.D. Thesis, Coventry University, Coventry, UK, 243 pp.

————, A. P. STURDEE, D. P. CASEMORE, A. CURRY, A. MILLER, N. D. PARKER, AND T. M. RICHMOND. 1994. *Cryptosporidium muris* in wild house mice (*Mus musculus*): First report in the UK. European Journal of Protistology 30: 151–155.

————, A. P. STURDEE, S. A. BULL, A. MILLER, AND S. E. WRIGHT. 1997. The prevalence of *Cryptosporidium parvum* and *C. muris* in *Mus domesticus*, *Apodemus sylvaticus* and *Clethrionomys glareolus* in an agricultural system. Parasitology Research 83: 478–482.

DUPONT, H. L., C. L. CHAPPELL, C. R. STERLING, P. C. OKHUYSEN, J. B. ROSE, AND W. JAKUBOWSKI. 1995. The infectivity of *Cryptosporidium parvum* in healthy volunteers. New England Journal of Medicine 332: 855–859.

FAYER, R. (ED.) 1997. *Cryptosporidium* and cryptosporidiosis. CRC Press Inc., Boca Raton, Florida, 251 pp.

GRATZ, N. G. 1994. Rodents as carriers of disease. *In* Rodent pests and their control, A. P. Buckle and R. H. Smith (eds.). CAB International, Wallingford, Oxon, UK, pp. 85–108.

HARRIS, S., P. MORRIS, S. WRAY, AND D. YALDEN. 1995. A review of British mammals: Population estimates and conservation status of British mammals other than cetaceans. Joint Nature Conservation Committee, Peterborough, UK, 168 pp.

ISEKI, M. 1986. Two species of *Cryptosporidium* naturally infecting house rats, *Rattus norvegicus*. Japanese Journal of Parasitology 35: 521–526.

LESLIE, P. H., U. M. VENABLES, AND L. S. V. VEN-ABLES. 1952. The fertility and population structure of the brown rat (*Rattus norvegicus*) in cornricks and some other habitats. Proceedings of the Zoological Society (London) 122: 187–238.

MEEHAN, A. P. 1984. Rats and mice: their biology and control. Rentokil Ltd., East Grinstead, UK, 383 pp.

MEYER, A. N., A. SHANKSTER, S. D. LANGTON, AND G. JUKES. 1995. National commensal rodent survey 1993. Environmental Health 103: 127–135.

MIYAJI, S., T. TANIKAWA, AND J. SHIKATA. 1989. Prevalence of *Cryptosporidium* in *Rattus rattus* and *R. norvegicus* in Japan. Japanese Journal of Parasitology 38: 368–372.

MOHR, C. O. 1947. Table of equivalent populations of North American small mammals. American Midland Naturalist 37: 223–249.

QUY, R. J., AND D. P. COWAN. 1996. Monitoring the activities of free-living wild rats implanted with transponders. *In* Proceedings of the Brighton crop protection conference—pests and diseases, F. M. McKim (ed.). The British Crop Protection Council, Farnham, UK, pp. 847–852.

————, ————, C. V. PRESCOTT, J. E. GILL, G. M. KERINS, G. DUNSFORD, A. JONES, AND A. D. MACNICOLL. 1995. Control of a population of Norway rats resistant to anticoagulant rodenticides. Pesticide Science 45: 247–256.

————, ————, AND T. SWINNEY. 1993. Tracking as an activity index to measure gross changes in Norway rat populations. Wildlife Society Bulletin 21: 122–127.

SINSKI, E., E. HLEBOWICZ, AND M. BEDNARSKA. 1993. Occurrence of *Cryptosporidium parvum* infection in wild small mammals in District of Mazury Lake (Poland). Acta Parasitologica 38: 59–61.

SMITH, H. V., AND J. B. ROSE. 1990. Waterborne cryptosporidiosis. Parasitology Today 6: 8–12.

TAYLOR, K. D. 1978. Range of movement and activity of common rats (*Rattus norvegicus*) on agricultural land. Journal of Applied Ecology 15: 663–677.

TZIPORI, S. 1983. Cryptosporidiosis in animals and humans. Microbiological Review 47: 84–96.

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