Coccidiosis in the European badger, *Meles meles* in Wytham Woods: infection and consequences for growth and survival

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SUMMARY

In total 1502 faecal samples were collected from a population of European badgers (*Meles meles*) between 1992 and 1995 at Wytham Woods, Oxfordshire, UK. Two coccidia species, *Eimeria melis* and *Isospora melis*, were identified. Cubs showed a marked seasonal pattern of infection with *E. melis*, with infection occurring at significantly higher intensity and prevalence than in adults. There was preliminary evidence to suggest that infantile coccidiosis in badgers may be associated with impaired growth and increased mortality.

Key words: badger, coccidiosis, *Eimeria melis*, *Isospora melis*, intensity, prevalence.

INTRODUCTION

The consequence of parasitic infection in individuals can impact on the dynamics and community structure of a host population and influence the behaviour of its members (Hamilton, 1982, 1990; Dobson & Hudson, 1986; Hudson, 1986; Gregory & Keymer, 1989; Gulland, 1995). Such host–parasite interactions have been the subject of both theoretical (Anderson & May, 1978, 1982, 1991) and experimental studies (Scott & Dobson 1989; Zuk, 1990). Despite the confounding effects of innumerable uncontrolled variables, studies in the wild offer the additional opportunity of assessing the ecological and evolutionary role of parasites in nature (Holmes, 1982). Long-term studies in which temporal changes in the assemblages of parasites effecting the host population are monitored are particularly important (Scott & Dobson, 1989). Of particular interest are those studies of wild mammal populations which have examined host–parasite interactions in detail at the level of the individual (Meade, 1983; Festa-Bianchet, 1990; Gulland, 1991; Schaller, 1972; Packer, 1979; Ransom, 1981; Packer *et al*. 1988).

Much of the parasitological research on badgers has focused on helminths, for which a number of species have been identified (reviewed by Hancox, 1980). Relatively little attention has been given to protozoan parasites, especially their epidemiological impact, though records exist for badgers infected with both flagellates and sporozoans: *Giardia* sp. (Mackinnon & Dibb, 1938), *Leishmania tropica* (Maruashvili & Bardzhadze, 1977), *Trypanosoma pestanai* (Macdonald *et al*. 1999), *Babesia missirolii* (Pierce & Neal 1974; Anwar & da Silva, 1988; Macdonald *et al*. 1999). *Eimeria melis* (Kotlan & Pospech, 1933; Kamiya & Suzuki, 1975; Anwar *et al*. 2000) and *Isospora melis* (Pellerdy, 1955; Anwar *et al*. 2000). The preliminary report by Anwar *et al*. (2000) on coccidial infections in badgers indicated a potential for *E. melis* to be pathogenic in cubs, warranting further investigation.

Badger cubs raised in captivity have been documented to suffer diarrhoeal enteritis (Rewell, 1948; Ratcliffe, 1974; Neal, 1977) leading to morbidity and mortality. This pathology is consistent with coccidial infection, as is the tendency for infection primarily among immunologically immature individuals (Koudela, 1999; Daugschies, Meyer & Joachim, 1999). Coccidiosis, the disease caused in a host by a host-specific coccidian parasite, is a pandemic disease of considerable economic importance to livestock farming (Shirley & Tomley, 1998; Trees *et al*. 1999; Williams, 1999). However, there have been few studies on the incidence and impact of coccidiosis in wild animals and these are usually limited to necropsies of a few individuals (Cowan, 1985; Snyder, *et al*. 1990; Oksanen, 1994; Sainsbury & Gurnell, 1995; Geisel *et al*. 1995; Forbes, 1997; Aramini, Stephen & Dubey, 1998).

The population of badgers, *Meles meles*, in Wytham Woods, Oxfordshire, offers an unusual opportunity to investigate the detailed impact of coccidiosis in the wild and to test whether the prevalence and intensity of infection varies with sex, age or season (Festa-Bianchet, 1990). Kruuk's

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(1978*a*, *b*) pioneering study of badgers in Wytham Woods has formed a platform on which a longitudinal study of known individuals has run continuously since 1987 (Woodroffe, Macdonald & da Silva, 1993; da Silva, Macdonald & Evans, 1994; Woodroffe & Macdonald, 1995; Tuyttens *et al*. 1999). This study has revealed variations in behaviour, physical condition, reproductive success and survival between years (da Silva, Woodroffe & Macdonald, 1993; Woodroffe & Macdonald, 1995). Part of this variation may be explicable in terms of resource availability, but there is also preliminary evidence on inter-annual variation in the prevalence of two blood parasites in this population (Macdonald *et al*. 1999), one of which may be pathological. In common with blood parasitaemias, coccidia can also be studied using non-destructive sampling techniques, allowing samples to be collected from all the badgers in the population on repeated occasions.

Here we present results on the effects of coccidiosis on the badger population in Wytham Wood.

MATERIALS AND METHODS

Study site

The study was performed in Wytham Woods (OS ref: SP 462,080; for detailed description of study area see Kruuk, 1978*a*, *b* and da Silva *et al*. 1993). Population socio-spatial distribution was assessed during this study using standard bait-marking techniques in 1993, 1994 and 1995 (Kruuk, 1978*a*, *b*; Stewart, Anderson & Macdonald, 1997; Feore & Montgomery, 1999) which revealed that the population is largely geographically discrete and organized into 20 social-groups. Taking these groups as a whole the outer limit of the most peripheral latrine sites has consistently embraced an area measuring 6 km^2

Trapping protocol

Between 1993 and 1995 each sett in the study area was trapped on at least 4 occasions per year (following the general methodology of Cheeseman & Harris, 1982), an annual schedule that has been followed since 1987. Badgers were trapped in steel mesh cage traps ($850 \times 370 \times 380$ mm) baited with peanuts. Trapping sessions took place in early January, late May, August and October/November (trapping was suspended during the period February–May, so as not to disturb the final trimester of gestation or the period during which altricial young are highly dependent).

Traps were sited at all of the active setts associated with each group and each site was trapped for 3 successive days. Badgers were anaesthetized using 0·2 ml of Ketamine hydrochloride (Vetalar, Pharmacia & Upjohns, Crawley, E. Sussex, UK) per kg bodyweight (Hunt, 1976; Mackintosh *et al*. 1976) and upon initial capture all individuals were permanently marked with a tattoo on the left inguinal region (Cheeseman & Harris, 1982). The age (latterly known from year of birth, and previously inferred from toothwear, see da Silva & Macdonald, 1989) sex and social group affiliation (based on the site at which it was trapped) of each badger was recorded. Badgers were released at the site of capture after approximately 3 h, by which time they had fully recovered from anaesthesia. Each individual was dabbed with a temporary colour marker so that it could be released without handling if recaptured during the remainder of that trapping session.

Faecal sample collection

Faeces were collected by 2 different methods. (1) Fresh faecal samples were collected at the end of each month from November 1992 to October 1993 inclusively from hinterland-latrines situated within the home range of each of 10 social groups (10 samples per social group, only 5 in autumn due to reduced terrestrial defecation rates). The exclusive use of these latrines by the respective groups was established by bait marking (Delahay *et al*. 2000). (2) During each trapping session faeces were collected from individually marked, anaesthetized badgers following administration of an enema consisting of 7·5 ml of warm soapy water per kg bodyweight ('induced sample'). Additional individuals were sampled during supplementary live-trapping during May, June and July. All induced samples were collected within a 3-h badger processing period to limit circadian variations in oocyst output. Induced faecal samples were collected throughout 1993 and 1994 and analysed for the periods winter (January–March), spring (April–June), summer (July–September) and autumn (October–December), these periods respectively correspond to parturition and post-partum mating, lactation, cub independence and intensive feeding prior to winter lethargy. Further samples were collected from cubs in 1995.

Faecal samples were stores individually in a cold room at 4 °C, in 250 ml plastic pots using 2·5 % (w/v) aqueous potassium dichromate ($K_2Cr_2O_7$) as preservative. Levels of gastro-intestinal coccidiosis were monitored using excreted oocyst counts. Following the methods of Anwar *et al*. (2000), prior to analysis 100 ml of each faecal sample was washed through a 2 mm-guage sieve to remove large fragments of semi-digested food. The faecal suspension was then centrifuged twice at 447 ***g*** for 3 min, the supernatant discarded and the solid weighed. A suspension of these solids was then made using a saturated solution of sodium chloride (NaCl) (MAFF, 1986; Parker & Duszynski, 1986; Lloyd & Smith, 1995). An exploratory analysis of 20 faeces showed that examination of up to 10 slides per scat failed to achieve significantly greater success in

Table 1. Coccidia distribution: prevalence (1993–1995) showing the numbers of live-trapped badgers subject to induced defecation, with adults separated into females and males

Year	Class	Number in class	Parasite	Winter	Spring	Summer	Autumn	Mean (%)
1993	Female adult	40	*E. melis*	2/4	0/5	1/15	1/16	12·5
			I. melis	0/4	0/5	1/15	0/16	2·5
	Male adult	75	*E. melis*	1/8	2/10	3/31	4/26	12
			I. melis	0/8	1/10	0/31	1/26	2·7
	Cubs	75	*E. melis*		11/14	25/38	9/23	60
			I. melis		1/14	0/38	0/23	1·3
1994	Female adult	70	*E. melis*		0/4	3/49	1/17	5·7
			I. melis		1/4	0/49	0/17	1·4
	Male adult	57	*E. melis*		1/6	2/36	0/15	5·3
			I. melis		0/6	2/36	0/15	3·5
	Cubs	27	*E. melis*		2/2	15/19	2/6	70·4
			I. melis		0/2	0/19	0/6	0
1995	Cubs	57	*E. melis*		17/25	10/22	8/10	61·4

monitoring oocyst output than the use of 2 slides (ANOVA: $F_{1,18} = 1{\cdot}45$, $P = 0{\cdot}245$). Therefore, 2 slides were examined per faecal sample and the mean score calculated.

Analyses

All data were $\log_e(x+1)$ transformed and treated as a continuous variable with normal errors to account for the skewed distribution of oocysts (Anwar *et al.* 2000). As not all individuals are caught in every season the population classes analysed are comprised of different combinations of individuals on different occasions and thus only analyses at the population level are possible.

In total 1100 latrine faecal samples were examined for species of coccidia present. Oocyst counts were used as indices of the intensity of infection and seasonal patterns are given for 1992 and 1993.

Working with induced faecal samples ($n = 402$), from known individuals, oocyst counts were used to provide an index of intensity for coccidiosis, while the prevalence at which coccidia oocysts were recorded is expressed as a percentage. Cub, adult male and adult female population subclasses were examined separately, per season, from 1993 to 1995.

Rates of infectious intensity, prevalence and seasonal patterns of infection are compared between the population subclasses using ANOVA tests subject to Bonferroni correction.

Seasonal patterns in cub *E. melis* oocyst counts were examined to test the hypothesis that immunity may develop as badgers mature.

To account for inter-annual variation in infection rates, and intra-annual seasonal variation, oocyst counts of *E. melis* infection were standardized relative to the intensity of infection. For each season, each individual oocyst count was divided by the mean of the oocyst counts for that season. For annual trends, in instances where individuals were re-captured within years, seasonal mean intensity of infection provided a standardized score that was used to reflect how an individual compared overall to its contemporaries in the population. Thus, individuals with an intensity of infection greater than the population mean would have factors scoring > 1, while individuals with infection intensities lower than the mean would score < 1.

These scores were then used to test the hypothesis that *E. melis* infection may be associated with altered morbidity or mortality in the focal cub cohorts was tested. As an index of parasite induced morbidity body length was chosen as a variable that could be influenced by the stress of high parasitosis during development but could not recover from any parasite-induced retardation once skeletal development ceases. Anaesthetized badgers were laid dorsally and head-body-length was measured from the end of the snout to the base of the sacrum.

Because the head-body-length of badgers is sexually dimorphic we examined the sexes separately when testing the impact of *E. melis* infection on skeletal development using regression analyses. Length was considered after 1 and 2 years to allow for complete maturation. Annual levels of cub infection were standardized across the seasons. The survival rates of cubs with greater than mean infection, and cubs with less than mean infection were compared after 1 and 2 years using chi-squared analyses. If a cub was not re-caught by the age of 2 it was considered to be dead. While this measure also includes permanent dispersal, this factor is negligible at this site (Newman, 2000).

All results of multiple tests are expressed after Bonferroni corrections.

RESULTS

Two coccidia species were recorded from both latrine- and induced faeces: *E. melis*, and *I. melis* (Kotlan & Pospech, 1933; Anwar *et al.* 2000)

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Table 2. Coccidia distribution: intensity (log_e *oocysts* + *1 transformed*) (1993–1995)

Year	Class	Parasite	Winter		Spring		Summer		Autumn		Annual mean	Total s.e.
			Mean	s.e.	Mean	s.e.	Mean	s.e.	Mean	s.e.		
1993	Female adult	*E. melis*	0·258	0·157	0	0	0·206	0·206	0·258	0·182	0·206	0·106
		I. melis	0	0	0	0	0·026	0·026	0	0	0·0098	0·0098
	Male adult	*E. melis*	0·025	0·025	0·285	0·207	0·2048	0·0848	0·298	0·166	0·2285	0·0723
		I. melis	0	0	0·156	0·156	0	0	0·0381	0·0381	0·034	0·0245
	Cubs	*E. melis*	N.A.	N.A.	2·186	0·456	1·311	0·211	0·933	0·294	1·358	0·169
		I. melis	N.A.	N.A.	0·07	0·07	0	0	0	0	0·0131	0·0131
1994	Female adult	*E. melis*	N.A.	N.A.	0	0	0·0258	0·0187	0·0234	0·0234	0·0238	0·0142
		I. melis	N.A.	N.A.	0·104	0·104	0	0	0	0	0·0059	0·0059
	Male adult	*E. melis*	N.A.	N.A.	0·146	0·146	0·1089	0·0762	0	0	0·0841	0·0504
		I. melis	N.A.	N.A.	0	0	0·0318	0·0281	0	0	0·0201	0·0178
	Cubs	*E. melis*	N.A.	N.A.	4·0265	0·0825	2·429	0·324	0·839	0·586	2·194	0·304
		I. melis	N.A.	N.A.	0	0	0	0	0	0	0	0
1995	Cubs	*E. melis*	N.A.	N.A.	3·575	0·152	2·426	0·247	1·317	0·242	2·494	0·161

identified by unique structural characteristics and confirmed by sporulation (Anwar *et al*. 2000).

E. melis was the most prevalent intestinal parasite in our study population. For adults no significant difference in intensity of infection was observed overall between the adult sexes and mean annual prevalence was only 8·5 %. However, mean annual prevalence of *E. melis* amongst cubs was much higher at 66·4 % across all seasons and all years (Table 1). The intensity of infection in cubs was much greater than for the adults at 1·36 $\log_e(x+1)$ oocysts per gram faeces in cubs versus 0·22 in adults in 1993 and 2·19 $\log_e(x+1)$ oocysts per gram faeces in cubs versus 0·06 in adults in 1994 (Table 2). Thus, cubs stand out as the population class most severely afflicted by *E. melis*. Data for 1993–1995 (Tables 1 and 2) show the high levels of coccidial infection and the progressive decline in intensity of infection by late autumn.

Parasitosis with *E. melis* peaked in the youngest cubs caught in 1993 with 10·5 $\log_e(x+1)$ oocysts per gram faeces recorded for the most severely infected individual. No comparisons could be made between infected and uninfected cubs, *per se*, as prevalence of infection was initially 100 % for all cubs captured. In 1993 and 1994 the intensity and prevalence of infection with *E. melis* was generally greater in cubs than in adults (Table 3).

Coccidia infection varied with season. In latrine samples, the intensity of *E. melis* infection showed a primary peak in May–August and a secondary peak in January (Fig. 1) whereas *I. melis* peaked in December and January (Fig. 2).

These results were only partly reflected by the analyses of induced samples. In 1993 there was no significant seasonal variation in adults of either sex. For cubs *E. melis* showed a general seasonal trend in intensity, with a spring peak and a decline through the summer to an autumn low, but this pattern was not significant. There was also a similar trend in the prevalence of *I. melis* with *Spring* > *Autumn* $F_{1\cdot35} = 6\cdot05$ $P = 0\cdot019$ which bordered the significant Bonferroni correction value of 0·017.

In 1994 females showed significant seasonal variation in the intensity of *I. melis* infection, peaking in spring and declining over the summer. *Spring* > *Summer* = $F_{1,51} = 15\cdot72$ $P = 0\cdot001$. In cubs, intensity and prevalence of *E. melis* infection declined through the year. However, this trend was not significant after Bonferroni correction.

In 1995 only cubs were monitored. The intensity of *E. melis* infection declined significantly from spring to autumn: ANOVA; *Spring* > *Summer* $F_{1,56} = 20\cdot09$ $P = 0\cdot001$; *Spring* > *Autumn* $F_{1,45} = 105\cdot54$ $P = 0\cdot001$ (*Summer* > *Autumn* $F_{1,53} = 9\cdot43$ $P = 0\cdot003$), as did *E. melis* prevalence: *Spring* > *Autumn* $F_{1,45} = 9\cdot58$ $P = 0\cdot003$.

Inter-annual comparisons by season, for both prevalence and intensity of cub infection with *E.*

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Table 3. ANOVA tests per season for 1993 and 1994 showing cubs to have significantly higher rates of infection than adults

(Intensity was measured as log_e *oocysts+1* and prevalence expressed as a percentage. N.S., Not significant. Only cubs were examined in 1995 so no inter-population class analyses were possible.)

E. melis	Year	Spring	Summer	Autumn
Intensity	1993	C > A $F_{1,27} = 18{\cdot}57$ $P = 0{\cdot}001$	C > A $F_{1,82} = 26{\cdot}62$ $P = 0{\cdot}001$	N.S.
Intensity	1994	C > A $F_{1,10} = 369{\cdot}44$ $P = 0{\cdot}001$	C > A $F_{1,102} = 200{\cdot}8$ $P = 0{\cdot}001$	C > A $F_{1,36} = 11{\cdot}89$ $P = 0{\cdot}001$
Prevalence	1993	C > A $F_{1,27} = 20{\cdot}34$ $P = 0{\cdot}001$	C > A $F_{1,82} = 26{\cdot}36$ $P = 0{\cdot}001$	N.S.
Prevalence	1994	N.S.	C > A $F_{1,102} = 107{\cdot}53$ $P = 0{\cdot}001$	N.S.

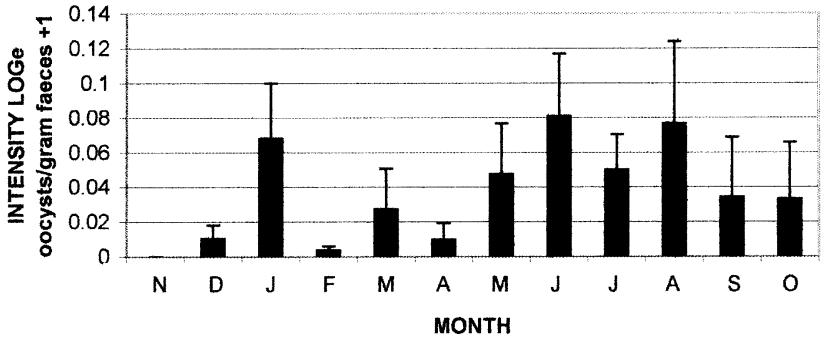

Fig. 1. Intensity of infection with *Eimeria melis* oocysts seen in faeces collected from latrines (1992–1993) showing mean with standard error bars.

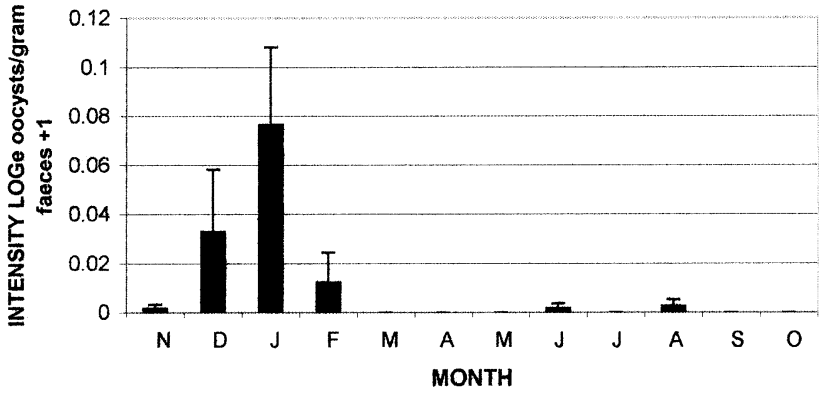

Fig. 2. Intensity of infection with *Isospora melis* oocysts seen in faeces collected from latrines (1992–1993) showing mean with standard error bars.

melis revealed no significant differences between 1993 and 1994 or between 1994 and 1995. However, 1993 did differ significantly from 1995 in *Spring* (93 > 95): ANOVA; Intensity; $F_{1,37} = 19{\cdot}64$, $P = 0{\cdot}001$ and in *Summer* (93 > 95) $F_{1,69} = 11{\cdot}94$, $P = 0{\cdot}001$.

Regressions associating intensity with prevalence are significant in all years (1993: $F_{1,3} = 11{\cdot}44$, $P = 0{\cdot}043$, $r^2 = 0{\cdot}79$; 1994: $F_{1,2} = 17{\cdot}77$, $P = 0{\cdot}052$, $r^2 = 0{\cdot}90$; 1995: $F_{1,3} = 41{\cdot}4$, $P = 0{\cdot}032$, $r^2 = 0{\cdot}83$) and over the complete set of data ($F_{1,12} = 26{\cdot}88$, $P = 0{\cdot}001$, $r^2 = 0{\cdot}69$). This indicates that prevalence is, at least in part, threshold detectable dependent on intensity rates.

High relative infantile infection with *E. melis* in male cubs born in 1995 was associated with significantly retarded adult head-body-length after 1 (1996) and 2 (1997) years (Regression: After 1 year:

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$F_{1,11} = 5{\cdot}18$, $r^2 = 0{\cdot}32$, $P = 0{\cdot}044$; After 2 years: $F_{1,12} = 14{\cdot}05$, $r^2 = 0{\cdot}54$, $P = 0{\cdot}003$) but the results were not significant in 1993 or 1994. However, this trend did approach significance when all years were combined for male cubs (Regression: After 1 year: $F_{1,69} = 3{\cdot}49$, $r^2 = 0{\cdot}05$, $P = 0{\cdot}066$; After 2 years: $F_{1,60} = 3{\cdot}82$, $r^2 = 0{\cdot}06$, $P = 0{\cdot}055$). If data for male cubs on infection intensity in spring are used to predict mature body-length, May (93–95, for a viable sample size) infection intensity is correlated with reduced body-length after 1 year but is not significant after 2 (Regression: May; After 1 year: $F_{1,5} = 8{\cdot}41$, $r^2 = 0{\cdot}63$, $P = 0{\cdot}034$; After 2 years $F_{1,4} = 5{\cdot}47$, $r^2 = 0{\cdot}58$, $P = 0{\cdot}079$). The same analysis for the June data does not produce a significant correlation (Regression: June; After 1 year: $F_{1,6} = 0{\cdot}42$, $r^2 = 0{\cdot}07$, $P = 0{\cdot}54$ N.S.; After 2 years $F_{1,7} = 0{\cdot}16$, $r^2 = 0{\cdot}02$, $P = 0{\cdot}669$ N.S.).

No significant effect of the intensity of infection with *E. melis* on female body-length was detected, though an association approaching significance for impaired growth after 2 years was seen from the 1995 cohort (Regression: YR2: $F_{1,8} = 4{\cdot}15$, $r^2 = 0{\cdot}34$, $P = 0{\cdot}076$).

Comparing cubs with greater than, or less than standardized infection with *E. melis* revealed that heavy infection was significantly associated with reduced survival. In 1995 the association with survivorship approached significance after 2 years (χ^2 D.F. = 2, $P = 0{\cdot}082$). Over all years there is a significant association between high infection and poor survivorship after 1 year (χ^2 D.F. = 2, $P = 0{\cdot}01$) which still approaches significance after 2 years (χ^2 D.F. = 2, $P = 0{\cdot}062$).

DISCUSSION

Assessing gastro-intestinal parasites from faecal oocyst/egg counts (MAFF, 1986; Parker & Duszynski, 1986; Lloyd & Smith, 1995) is an indirect method that provides an analogue of infection. An approximately linear relationship between number of parasites per gram faeces and total parasite burden has been observed for many parasite species (Sachs & Sachs, 1968; Keymer & Hiorns, 1986; Pritchard *et al*. 1990). However, once the gut is saturated with parasites the rate of excretion in the faeces may no longer rise in direct proportion to intestinal infection, though differences between infected cubs and immune adults in this study are very apparent. The estimation of parasite burden by coprological analysis remains a useful technique, however, for examining infections in live hosts as well as the prevalence and intensity of infection rates in extant host populations (Hall, 1982, Michael & Bundy, 1989). It is appropriate to consider an infection with reference to background levels within years, as it may be largely related to the general conditions affecting the health of that particular cohort at that time. An individual with a less than average intensity of infection in one year might be more physiologically stressed than an individual in a different year with a much lower absolute intensity of infection.

A number of studies have used faecal oocyst counts to quantify infections with coccidia (Aramini *et al*. 1988; Davis & Dubey, 1995; Lloyd & Smith, 1995; Chappell *et al*. 1996; Hobbs *et al*. 1999). Since the transmission of infection is dependent upon the rate at which coccidia oocysts are shed, faecal oocyst counts are critical to the effective monitoring of population health.

Badger cubs are entirely fossorial until they are 8 weeks old and do not emerge above ground regularly until they are 10 weeks old, usually in late April (Neal, 1990). The increase in *E. melis* oocysts in latrine faeces in May–August suggests that the faecal contribution of cubs, once above ground, might be determining the pattern of oocyst deposition at latrines. This inference accords with observations of cubs defecating at terrestrial latrines close to the sett (personal observation) from May onwards. Cubs already have high rates of infection when trapped and sampled in May. Before this and for their first 10 weeks, they must defecate beneath ground and subterranean latrine sites have been documented in sett chambers (Roper, 1992). The faeces they deposit within the sett will have a high oocyst content and may act as a reservoir that could potentially transmit infection between annual cub cohorts.

Alternatively, initial infection could come from contact with infected adults. However, survival of infection with coccidia is known to result in strong acquired immunity and only 1 in 10 adult badgers was recorded with any *E. melis* infection. Combining this with the low oocyst intensity in faeces deposited exclusively by adult badgers at terrestrial latrine sites before cub emergence, limits, but does not exclude, the likelihood of the general adult population being the source of early cub infection. Perhaps significantly, the minor peak in *E. melis* and main peak in *I. melis* in January correspond with the latter stages of badger gestation (Woodroffe & Macdonald, 1995). This is a time when raised levels of oestrogens, progesterones and corticosterones can lead to immunosuppression that can increase susceptibility to parasitoses (Lloyd, 1983; Alexander & Stimson 1988; Brabin & Brabin, 1992). Hence, it is possible that cubs could become infected via maternal transmission at parturition. Recrudescence of coccidia infections attributable to a compromised immune system during pregnancy have been recorded for infections with *Eimeria* in rabbits (Hobbs *et al*. 1999). Further study will be needed to elucidate sources of infection.

Host–parasite interactions can exhibit sex-specific differences and many studies have shown females to mount a more effective immune response (Wein-

stein, San & Segal, 1984; Grossman, 1989; Schuurs & Verheul, 1990). In this present study there was no evidence to suggest that prevalence, and therefore immune-susceptibility of exposure risk to either coccidial species varied with gender at any stage of maturity (also seen by Anwar *et al*. 2000). A similar homology between the sexes has also been noted for 8 *Eimeria* species infecting rabbits (Hobbs *et al*. 1999).

I. melis occurred at a much lower prevalence and intensity than the eimerian coccidia. This may be because *Eimeria* species are known to be monoxenous (i.e. with direct life-cycles), transmitted by the faecal–oral route. Lindsay & Blagburn (1994) reported that *Isospora* species with a stieda body in their sporocysts are also generally monoxenous. However, badger *I. melis* lack a stieda body (Anwar *et al*. 2000), and thus, according to Lindsay & Blagburn (1994), may be facultatively heteroxenous, utilizing a paratenic host. The predation of badgers on small mammals in Wytham is rare (personal observation), preferring to eat earthworms (*Lumbricus terrestris*) where possible (Kruuk, 1978*a*, *b*) and this may limit the opportunity for infection to occur with a heteroxenous parasite. The absence of *I. melis* infection in some seasons suggests that exposure to the source of infection may be sporadic, though the January latrine peak in infection could reflect the contribution of immunocompromised pregnant/lactating females. However, from induced samples, no significant differences were found between the adult sexes or between cubs and adults. Further investigation of potential paratenic hosts is ongoing.

Infection was most highly prevalent in recently emerged cubs (*ca* 12 weeks old) that had just become available for trapping. By November, however, oocyst intensity had dropped, on average, to a few hundred oocysts per gram. For such a ubiquitous parasite as *E. melis* a decline in infection rates between cubs and adults suggests either a behavioural change resulting in reduced exposure to sources of infection or a change in the immune profile of the population with age. This may be caused through acquired immunity or the death of innately susceptible individuals. Comparable high levels of infection are known to cause morbidity in dogs (*C. domesticus*) infected with *Cryptosporidium parvum* (Lloyd & Smith, 1995) and Wapiti (*Cervus elaphus*) infected with *E. wapiti* and *E. zurenii* (Foreyt & Largerquist, 1994).

Impaired badger development, assessed as head-body length, was associated with high levels of coccidiosis. This trend was most evident from the 1995 data. Anderson (1993) discussed the potential for seasonal or annual fluctuations in parasite development as a product of climate variability. In 1995 there was a marked summer drought. Earthworms (badgers principle food, Kruuk & Parish, 1982; Neal, 1990; da Silva *et al*. 1993) are less abundant at the surface of dry soil. Hence, reduced food availability may have added to the stresses cubs experienced that year and potentiated the morbidity incurred by the *E. melis* coccidiosis in 1995. An alternative hypothesis that malnutrition reduced the cubs' resistance and resilience to infection in 1995 must also be considered. Initial levels of coccidiosis in 1995 were no higher than in 1993 or 1994 but intensity of infection remained high throughout the summer of 1995, showing a slower rate of developing immunity than in the other years. This may have been caused by the cub cohort's nutritionally-impaired ability to mount an effective immune response against the parasitosis. Either mechanism remains consistent with the hypothesis that *E. melis* infection can impact upon cub development.

The symptoms of badger cub infection with high levels of *E. melis* were apparent when these individuals were handled. Cubs had diarrhoeal enteritis with a swollen abdomen and produced soft dark faeces. Coccidial infections are characterized by chronic wasting e.g., Chae *et al*. (1998); Daugschies *et al*. (1999); Lindsay, Dubey & Blagburn (1997). Cubs suffering from *E. melis* were thus likely to experience poor health. Interactions between nutrition and parasite action are well documented (Bundy & Golden, 1987, Crompton, 1987, 1993). Host nutritional status can affect the parasite abundance and conversely parasite infection can influence the host's feeding behaviour and metabolism (Müller-Graf, 1994). Three mechanisms, not mutually exclusive, could thus militate for impaired cub growth observed: malnutrition through malabsorption and metabolic incompetence (Symons, 1976; Crompton & Stephenson, 1990; Solomons, 1993), inappetance and anorexia (Daugschies *et al*. 1999) and increased susceptibility to secondary infections due to damaged organ development (Slater & Keymer, 1986). The extent of parasitosis may also be self-reinforcing; as the cub is weakened by the infection so it becomes more susceptible to further infection. Malnutrition, by interaction with the immune system may decrease cellular and humoral defences (Behnke, 1990; Castro, 1990) and/or induce defects in cellular immunity (Michael & Bundy, 1992).

The more general significance of impaired head-body-length development in males but not females requires explanation. Adult male badgers were 3% longer than females in this study and grew faster, attaining this extra 3% length advantage by the August of their first year (Newman, 2000). Differential growth rates resulted in male cubs achieving their length advantage over females by the time they were 6 months old, thus male cubs had a higher growth rate earlier in the spring than females, during which time levels of coccidial infection were seen to be at their highest (Newman, 2000). Hence, male

cubs experienced the physical stresses of a more rapid growth rate and high parasitosis simultaneously; thus the high physiological demand of infection may have caused growth to suffer.

Two mechanisms could explain the interaction between infection and mortality. With severe infection cubs may simply die of the illness directly or through concurrent infections. Alternatively, their lethargic behaviour could leave them open to other causes of death through either direct threats or competition. Additionally, the subnormal stature of cubs surviving high initial infection may place them at a competitive disadvantage as they mature.

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