

Coccidiosis in the European badger (*Meles meles*) from England, an epidemiological study

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SUMMARY

In total 445 faecal samples were collected from 259 European badgers (*Meles meles*) in Wytham Woods, Oxfordshire, UK (462080). Microscopical examination revealed infection with 2 species of coccidia *Eimeria melis* and *Isospora melis*. From the initial examination of each animal, point prevalence rates of 0.44 and 0.35 were calculated for *Eimeria* and *Isospora* respectively. The intensity of infection was significantly greater for *Eimeria* than *Isospora* and the distribution of intensities was highly skewed for both species, with a few individuals shedding the majority of oocysts. Incidence and recovery rates for both coccidia species were calculated from longitudinal data collected at 3-monthly intervals from a subset of the adult badger population, and the predicted prevalence rates based on these were similar to the point prevalence rates. This suggests little, if any, parasite-induced mortality in the adult population. In contrast, there was a marked and significant reduction in the point prevalence and intensity of infection with *Eimeria* from cub to adult badger suggesting a degree of acquired immunity to *Eimeria melis* on initial exposure and/or that there is significant *Eimeria*-associated mortality in the cub population. No such relationship was found for *Isospora* infection. In those adult badgers with co-infections there was a direct relationship between the intensity of *Eimeria* and *Isospora*. The taxonomic status of these parasites suggests a heteroxenous life-cycle for *I. melis*, and direct transmission of *E. melis*. However, the greater than expected prevalence of co-infection is consistent with a common source of infection, such as communal latrines.

Key words: *Eimeria melis*, *Isospora melis*, prevalence, mortality.

INTRODUCTION

The first report on coccidia in the European badger (*Meles meles*) was by Kotlan & Pospesch (1933) who reported *Eimeria melis* as a new species and a second diplosporid coccidia which resembled *Lucetina rivolta* (*Lucetina* = *Isospora*). Later, Pellérdy (1955) named this latter parasite *Isospora melis*. Kamiya & Suzuki (1975) reported coccidia oocysts, and macrogametocytes and microgametocytes found in the jejunal mucosa of a badger in Japan. They gave the size of oocysts, but they were unable to identify this parasite beyond mentioning that 'the coccidia parasite in this study probably belongs to genus *Eimeria*'.

Most of what we know of the epidemiology of coccidia is limited to studies of domesticated animals such as cattle (e.g. Taylor & Catchpole, 1994) and poultry (e.g. Henken *et al.* 1992). In general, reports of coccidia in wild animals are limited to new geographical records (e.g. McAllister, Upton & Trauth, 1994), descriptions of novel host-parasite

combinations (e.g. McAllister *et al.* 1994), crude prevalence rates (e.g. Zarzara *et al.* 1990), and isolated records of pathogenicity (Oksanen, 1994). The present paper describes for the first time coccidia infections and the results of a longitudinal study of parasites in the European badger, *Meles meles*, with details of the epidemiology of these highly prevalent and potentially pathogenic endoparasites in the UK.

MATERIALS AND METHODS

Study site and sampling

Badgers (*Meles meles*) were studied in Wytham Woods, Oxfordshire, UK (462080). The study site occupies approximately 6 km². Badger density is approximately 25 adults per km² (Woodroffe & Macdonald, 1995). Trapping and processing followed the methods described by Woodroffe & Macdonald (1995). Badgers were caught between May 1996 and August 1997 using box traps placed near the burrow entrance and baited with peanuts. Upon capture, each badger was anaesthetized by intramuscular injection of 15–20 mg/kg ketamine hydrochloride, sexed and classified as adult or cub.

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All animals were tattooed with a unique code number and released at the site of capture after full recovery from anaesthesia.

At the same time, faecal samples were collected from each animal by enema, and placed in separate vials containing 2.5% (w/v) aqueous potassium dichromate, mixed thoroughly and stored at 4 °C. Upon return to the laboratory, faecal suspensions were filtered to remove gross material, and an aliquot centrifuged at 730 *g* for 3 min. The pellet was weighed, re-suspended in a saturated solution of common salt and screened for coccidia oocysts by microscopy following the Parker & Duszynski (1986) method. The intensity of infection (recorded as oocysts per gram of pelleted faecal material) was determined directly. Where the concentration of oocysts was too great to be accurately enumerated in this way, a second faecal pellet was prepared using the McMaster technique (Davis, 1973).

Sporulation time was estimated in heavy infections. Unsporulated oocysts were allowed to sporulate at 25 °C ± 2 °C in a 15 cm Petri dish containing a layer of 2.5% K₂Cr₂O₇. To estimate the sporulation time the sample was examined twice daily.

A morphological study was conducted on sporulated oocysts at ×1000 magnification using oil immersion with a calibrated ocular micrometer. All measurements are in micrometers (µm). Means ± S.E. (*n* = 50) and showing size range in parentheses (Duszynski, Eastham & Yates, 1982). The taxonomic characters of oocysts were studied as recommended by Duszynski & Wilber (1997).

Epidemiological analysis

Infection status (presence/absence) and changes in status (changed/unchanged) were recorded as binary outcomes (1/0) and analysed in GLIM with binomial errors and the logit link function with a denominator of 1's (Crawley, 1993). Intensity of infection was log-transformed and treated as a continuous variable with normal errors. Model fitting began with a maximal model and parameters were deleted by a process of backward elimination to arrive at the minimum adequate model (Crawley, 1993).

RESULTS

Morphometric analysis

Isospora. Oocysts ovoidal, measuring 32.8 ± 0.34 × 26.9 ± 0.19 µm (25.6–37.8 µm × 24–29.6 µm), Length/width (L/W) Ratio 1.22 ± 0.124 (1.10–1.57). Oocyst wall thickness: 1.8 µm; composed of 2 layers; outer layer smooth, no micropyle. No oocystic residuum or polar granule develops. Sporulation takes 72 h. Sporocysts ellipsoidal and with no Stieda

body. Sporocysts measured 21.5 ± 0.166 × 14 ± 0.12 µm (19–24 µm × 12–16.6 µm), L/W Ratio 1.55 ± 0.017 (1.33–1.85). The sporozoites: 14.2 ± 1.16 × 4.0 ± 0.17 µm (10–20 µm × 3–5 µm), round at one end and tapered at the other end.

Eimeria. Oocysts ellipsoidal, measuring 20 ± 0.18 × 15.7 ± 0.02 µm (16–22.6 µm × 13–18.8 µm), L/W Ratio 1.28 ± 0.017 (1.12–1.5). Oocyst wall about 1.2 µm thick; composed of 2 layers; outer layer smooth, no micropyle. Oocyst residuum (4.2 × 2.0 µm) and small refractile polar body (1.4 × 0.4 µm) present. Sporulation time 72 h. Sporocysts ovoidal, 11.9 ± 0.018 × 6.5 ± 0.08 µm (10–14.2 µm × 5–8.2 µm). L/W Ratio 1.83 ± 0.026 (1.55–2.4). Stieda body present. Sporozoite: 9.0 ± 0.05 × 3.24 ± 0.025 µm (8.0–9.2 µm × 2.8–3.6 µm). Refractile body, round at thick end, measuring 3.6 µm × 2.0 µm.

Epidemiological analysis

In total 445 faecal samples were collected from 259 badgers during 1996 and 1997, in 4 three-monthly surveys.

Cross-sectional estimates of prevalence

In total 259 badgers were sampled at least once. Treating infection status at first sampling as a cross-sectional survey of infection, an overall point prevalence rate of 0.44 (115/259) and 0.35 (91/259) can be calculated for *Eimeria* and *Isospora* respectively. Stratifying these results by age and sex, the point prevalence rate for *Eimeria* was found to be significantly higher in cubs than adults ($\chi^2_1 = 43.85$; $P < 0.001$), but the point prevalence rate of *Isospora* was constant with age ($\chi^2_1 = 1.93$; $P > 0.05$) (Table 1). There was no significant interaction between age and sex, for either *Isospora* ($\chi^2_1 = 0.87$; $P > 0.05$) or *Eimeria* infection ($\chi^2_1 = 0.8$; $P > 0.05$): in other words, there is no detectable difference in the prevalence rate between males and females as adults and cubs.

Mixed infections of *Isospora* and *Eimeria* were common (Table 1), and overall greater than expected by chance ($\chi^2_1 = 4.66$; $P < 0.05$). When separated into component demographic groups, only male badgers (adult + cub) had mixed infections at greater than expected frequency ($\chi^2_1 = 4.41$; $P < 0.05$), but the trend was in the same direction for all badgers and statistically indistinguishable between groups.

Longitudinal estimates of incidence, recovery and prevalence

Only 5 cubs (2 female, 3 male) were sampled for coccidia more than once. However, the faeces of 110

Table 1. Point prevalence of *Eimeria melis* and *Isospora melis* and mixed infections of the two from first survey ($n = 259$), stratified by age and sex

	Male cub (%)	Female cub (%)	Male adult (%)	Female adult (%)
<i>Eimeria melis</i>	22 (95.6)	23 (88.5)	36 (32.7)	34 (34)
<i>Isospora melis</i>	9 (39)	13 (50)	39 (35.5)	30 (30)
Mixed	9 (39)	12 (46)	16 (14.5)	14 (14)
Uninfected	1 (4.3)	2 (7.7)	51 (46.4)	50 (50)
Total	23 (100)	26 (100)	110 (100)	100 (100)

Table 2. Intensity of infection with *Eimeria melis* and *Isospora melis* (as oocysts per gram of faeces) from first survey ($n = 259$), stratified by age and sex

	<i>Isospora melis</i>		<i>Eimeria melis</i>	
	Geom. mean	Range	Geom. mean	Range
Overall	10.4	1–6804	59	1–16157
Cub	12.6	1–535	369.8	1–16157
Adult	9.8	1–6804	18.1	1–570
Male	8.6	1–549	67.6	1–10305
Female	12.3	1–6804	51.5	1–16157

adult badgers (57 female, 53 male) was examined at roughly 3-monthly intervals up to a maximum of 4 times (0, 3, 6 and 9 months). From these data it is possible to make a crude estimate of the rate at which adult badgers acquire and recover from these infections.

Three-monthly incidence rate

The incidence rate of *Eimeria* and *Isospora* was assessed at a 3-monthly time-scale. Above this, insufficient badgers were sampled to give meaningful results. All sample series for which an individual badger was negative for one survey and then sampled in the following 3-monthly survey were included.

For *Eimeria*, a total of 100 series were taken from both males and females. Of these, 24% (95% CI 15.5–32.5) became positive on the second survey. The incidence rate by sex was 0.18 (0.052–0.308) for females and 0.29 (0.115–0.463) for males, and there was therefore no significant difference between the sexes ($\chi^2_1 = 1.524$, $P > 0.05$).

Of 104 series for *Isospora*, 17% (16.9–21.7%) became positive on the second survey. By sex, the incidence rate was 0.23 (0.02–0.49) for females and 0.11 (0.01–0.29) for males, again not significantly different ($F_{1,103} = 0.1688$, $P > 0.05$). There was no significant difference in incidence between *Isospora* and *Eimeria* ($\chi^2_1 = 2.281$, $P > 0.05$).

One of the assumptions of this analysis of incidence is that adult badgers do not acquire resistance with each bout of infection. One way of testing this is to compare the incidence rate in badgers which have recovered from infection with

the overall incidence. Too few instances of this were available for statistical analysis, but the data are suggestive: 33% (3/9) of badgers became *Eimeria* positive within 3 months of recovering from an *Eimeria* infection; 25% (2/8) of badgers became *Isospora* positive within 3 months of recovering from an *Isospora* infection. These values are very close to the overall incidence rates of 0.24 and 0.17 for *Eimeria* and *Isospora* respectively, and the implication is that acquired immunity does not increase significantly in adult life.

Three-monthly recovery rate

The analysis of recovery rates followed the same protocol as incidence, but taking the series starting point to be a parasite-positive sample. For *Eimeria*, of 43 positive samples, 63% (49–77%) became negative on the second survey (i.e. 3 months later). The recovery rates for females (0.6; 0.35–0.85) and males (0.64; 0.32–0.96) were not significantly different ($\chi^2_1 = 0.1$, $P > 0.05$).

For *Isospora*, of 39 positive samples, 74% (60–88%) became negative on the second survey. Again, the recovery rates of females (0.76; 0.55–0.97) and males (0.73; 0.46–1.0) were not significantly different ($\chi^2_1 = 0.07$, $P > 0.05$). There was no demonstrable difference in recovery rate between *Isospora* and *Eimeria* ($\chi^2_1 = 1.1$, $P > 0.05$).

Prevalence rate from incidence and duration of infection

We have already estimated the point prevalence rates of *Eimeria* and *Isospora* from the initial survey

(Table 1). We can also calculate the expected prevalence from the incidence and recovery rates through the relationship:

$$\text{prevalence} = \text{incidence} \times \text{duration of infection} \quad (1)$$

where the duration of infection is the reciprocal of the recovery rate. Obviously, this assumes that there is no mortality associated with infection; if there were, then those animals would be lost to the survey and therefore would not appear in the analysis, reducing the estimate of incidence and elevating the estimate of recovery, and compounding to create an underestimate of prevalence.

Calculating overall prevalence by Equation (1), our prevalence estimates for *Eimeria* ($0.24 \times 1/0.63 = 0.38$) and *Isospora* ($0.17 \times 1/0.74 = 0.23$) are similar to the directly observed point prevalence rates in adult badgers of 0.33 for both *Eimeria* and *Isospora* in adult badgers from the initial survey (Table 1). This is suggestive of little, if any, parasite-induced mortality in adults.

Intensity of infection

The frequency distribution of intensity of infection, measured as oocysts per gram of faeces, was extremely overdispersed for both *Eimeria* and *Isospora* (Table 2). Of those animals infected, the geometric mean intensity of infection was significantly greater for *Eimeria* than *Isospora* ($F_{1,205} = 28.66$, $P < 0.01$).

Of those infected, the intensity of *Eimeria* infection decreased dramatically and significantly from cub to adult ($F_{1,112} = 40.6$, $P < 0.01$), but there was no significant difference in intensity of infection between males and females ($F_{1,112} = 1.136$, $P > 0.05$), nor was there any interaction between the two factors ($F_{1,112} = 0.03$, $P > 0.05$) (Table 2). The intensity of *Isospora* infection did not vary by age ($F_{1,88} = 0.71$, $P > 0.05$), sex ($F_{1,88} = 1.24$) or the interaction of the two ($F_{1,88} = 1.53$, $P > 0.05$) (Table 2).

For those animals with concurrent *Isospora* and *Eimeria* infections, intensity significantly and positively co-varies: in other words, when the intensity of one parasite is high, the other is also high ($F_{1,48} = 4.4$, $P < 0.05$). Stratifying for age, this trend holds true for adult badgers ($F_{1,28} = 4.6$, $P < 0.05$) but disappears in cubs ($F_{1,19} = 0.143$, $P > 0.05$).

However, the intensity of infection with one parasite does not appear to be affected by the presence or absence of the other species. Thus, there is no significant change in *Eimeria* intensity when co-infected with *Isospora* ($F_{1,113} = 0.75$, $P > 0.05$) or vice versa ($F_{1,89} = 0.01$, $P > 0.05$).

Although only 5 badgers were sampled more than once for coccidia, there are recapture records for 19 animals first sampled for coccidia as cubs (including captures during trapping in 1998). The initial

intensity of *Eimeria* infection was considerably lower in those cubs which were recaptured 6 months later (169 oocysts/g), compared with those which were not (518 oocysts/g); in fact, none of the latter group was seen greater than 1 month after initial capture. However, the difference is not significant ($\chi^2_1 = 2.3$, $P > 0.05$). There was only a negligible difference in mean intensity of *Isospora* infection between recaptured and unrecaptured individuals (10.7 versus 10.2 oocysts/g) ($\chi^2_1 = 0.04$, $P \geq 0.05$).

DISCUSSION

Taxonomy and morphometric analysis

There have been only 3 previous reports of *Isospora melis* and *Eimeria melis* in badgers.

The first report on coccidia of the European badger (*Meles meles*) was by Kotlan & Pospesch (1933) who reported 2 species: *Eimeria melis* as a new species with oocysts measuring $17\text{--}24\ \mu\text{m} \times 14\text{--}15\ \mu\text{m}$; and a diplosporid coccidia which resembled the cat coccidia *Lucetina rivolta* (*Lucetina* is a synonym of *Isospora*), with oocysts measuring $26\text{--}34 \times 20\text{--}27\ \mu\text{m}$. However, Kotlan & Pospesch were not able to infect badgers with *L. rivolta* collected from cats and also could not infect cats with the putative *Lucetina* oocysts collected from badgers. Later, Pellérdy (1955) recovered the same *Lucetina*-like oocysts ($27\text{--}31\ \mu\text{m} \times 18\text{--}24\ \mu\text{m}$) from badgers and named the parasite as a new species, *Isospora melis*. Finally, Kamiya & Suzuki (1975) reported coccidia oocysts measuring $20\text{--}21\ \mu\text{m} \times 14\text{--}17\ \mu\text{m}$, also macrogametocytes and microgametocytes found in the jejunal mucosa of a badger in Japan. They mentioned that 'the coccidia parasite in this study probably belongs to genus *Eimeria*', and this was therefore probably *Eimeria melis*.

We conclude that the species described in the present paper are those reported before as *Eimeria melis* and *Isospora melis*, based on the similarity of taxonomic characters with those previously reported. There is some variation in size between our measurements and those reported earlier. Size variation has been reported in other species of coccidia; the size of oocysts change not only in different individuals but also at different stages of infection (Joyner, 1982).

Epidemiology

It is sometimes assumed that an absence of coccidia oocysts in a faecal sample represents an absence of infection. Of course, it is possible that oocysts are present but at undetectable levels, as has been recorded in sheep and goats which discharge oocysts continuously at low numbers (Pellérdy, 1974). Indeed, the total absence of parasites is thought unlikely under conditions of continuous exposure to infection (Wakelin & Apanius, 1997). Therefore

what we term incidence may, at least in part, be the rate of recrudescence of an existing infection rather than a newly acquired infection. Similarly, what we call recovery (or immunity) may not be the elimination of parasites, but rather a degree of remission sufficient to reduce oocyst levels below a detectable threshold.

Exposure to infection

There is no evidence that the prevalence, and therefore the risk of infection with either *Eimeria* or *Isospora*, is different in males and females, either as cubs or adults. The inference is that both exposure and susceptibility to infection are constant between the sexes. Prevalence of *Eimeria* does decrease with age. However, the intensity of *Eimeria* infection also decreases, which, for such a notoriously ubiquitous microparasite as *Eimeria*, suggests a change in immune profile of the population from cub to adult (either acquired or through the death of innately susceptible individuals – see below), rather than change in exposure to parasites.

Eimeria species are known to be monoxenous (i.e. they have direct life-cycles), transmitted by the faecal–oral route. In contrast, some genera of *Isospora* complex are heteroxenous. Lindsay & Blagburn (1994) reviewed mammalian *Isospora* and indicated that species which lack a Stieda body and are not sporulated when shed – which from our observations would therefore include *Isospora melis* – may be facultatively heteroxenous.

Although the badger is classified in the Carnivora (family Mustelidae), observations on their dietary habits reveal them to be generalist or opportunist feeders (Roper, 1994). According to Kruuk (1978), in Wytham and through much of the UK they feed predominantly on the earthworm *Lumbricus terrestris*, supplemented by other invertebrates, with rodents only rarely eaten. As 35% of badgers harboured *Isospora* infection, it is unlikely that the source of infection is vertebrate prey. Either badger isosporan infection is monoxenous – in this case they are not in the category suggested by Lindsey & Blagburn (1994) – or it is likely that earthworms are transmitting the parasite. Against this hypothesis, non-vertebrate hosts of coccidia appear rare (Snyder *et al.* 1990).

However, the greater than expected co-incidence of *Eimeria* and *Isospora* infections suggests either that the risk factors for infection with one species overlap with those for the other, or that the presence of one directly increases the risk of acquiring the other (e.g. by immuno-compromising the host). The latter explanation is contradicted by the observation that a co-infection with one parasite does not correlate with an increase in intensity of the other. Yet the former explanation seems unlikely if the mode of transmission of *Eimeria* is monoxenous and

of *Isospora* heteroxenous, and might indicate that *Isospora* is at least partly directly transmitted, like *Eimeria*, explaining the co-incidence by co-exposure, say in communal latrines.

Mortality and immunity

Wytham badgers are either acquiring and resolving *Eimeria* and *Isospora* infections, or relapsing and recovering from a single chronic infection, with high frequency. The result is a high point prevalence of both infections in the Wytham badger population. Indeed, in the cub population virtually all individuals were infected at first sampling, and it is reasonable to assume that all would be exposed to infection at some point.

The considerable reduction in both prevalence and intensity of *Eimeria* infection from cub to adult can be interpreted either as infection-associated mortality in those cubs with the highest intensities of infection, or the acquisition of immunity to infection leading to a reduction in the mean intensity of infection and the consequent loss of patent infection in some animals, or a combination of the two. Conversely, the prevalence and intensity of *Isospora* infection does not appear to change with age, and the mean intensity of *Isospora* infections was also considerably lower than that of *Eimeria*.

If re-appearance in traps could be treated as a measure of cub survival, then the much lower initial intensity of infection with *Eimeria* – but not *Isospora* – in those which survived to be trapped at least 6 months later suggests that *Eimeria* – and not *Isospora* – can cause death in the most susceptible of cubs. However, the sample size was small, and the observation is not significant.

In contrast, there is indirect evidence, from the comparison of the point prevalence rate with the prevalence rate estimated from incidence and recovery, to suggest that adult badgers do not suffer any significant mortality from *Eimeria* or *Isospora* infections. Neither do they appear to acquire significant additional immunity to infection at this age. This would be expected if either those badgers not competent to fight infection died before reaching maturity, and/or the level of acquired immunity asymptotes in adulthood to a maximum upon repeated or prolonged exposure to the parasite as is seen for other chronic endoparasitic infections (Cox, 1998). It is worth noting in this context that first exposure to *Eimeria* occurs very early in Wytham badgers: almost all cubs were found to have contracted infections, and any immunity may therefore develop very early in life.

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