Papers

Isosporoid coccidiosis in translocated cirl buntings (*Emberiza cirlus*)

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Four of 17 cirl buntings (*Emberiza cirlus*) involved in a trial translocation in 2004 for conservation purposes died and were examined postmortem. Two of the cirl buntings showed intestinal and hepatic lesions, including necrotising enteritis, consistent with isosporoid coccidiosis, and a third had an intestinal infestation of isosporoid coccidia. Sporulated oocysts from faecal samples from the birds were identified as *Isospora normanlevinei*, a parasite previously detected in cirl bunting populations in continental Europe. In a subsequent translocation of 75 cirl buntings from Devon to Cornwall in 2006, each brood of birds was placed in strict quarantine at low stocking density, with improved hygienic precautions and detailed health surveillance, and each bird was treated prophylactically with toltrazuril in an attempt to control the disease but not eliminate the *I normanlevinei* parasites. Seventy-two of the 75 birds were successfully reared and released, and there were no apparent clinical or pathological signs of isosporoid coccidiosis in any bird. *I normanlevinei* was detected in the released population, an indication that it had been successfully conserved.

THE cirl bunting (*Emberiza cirlus*), a member of the family Emberizidae, is red listed (within Birds of Conservation Concern 3 [Eaton and others 2009]) because of the declines in both numbers and range that the species has undergone within the UK. The population of cirl buntings in England has decreased dramatically over the past 120 years. In the 1890s the species was found in 39 counties (Aplin 1892), but by 1989 the population was estimated to be only 118 pairs in three counties, with more than 95 per cent being found in Devon (Evans 1992). The demise of the species has been linked to changes in farmland habitat, particularly the absence of winter stubble (Jeffs and Evans 2004). Once the reasons for the dramatic decline were understood, targeted conservation action by the Royal Society for the Protection of Birds (RSPB), Natural England and farmers to deliver cirl bunting-friendly habitat was implemented. This has resulted in a population increase, and the cirl bunting population in Devon is now estimated to be more than 860 pairs. This population increase has been directly linked to the targeted provision of habitat through the UK government-funded Countryside Stewardship Scheme (Peach and others 2001). Despite the increase in the number of breeding pairs, there has been no corresponding increase in range, and the population remains largely confined to a coastal strip in Devon. A key objective for the UK Biodiversity Action Plan for this species, therefore, is to establish populations away from this core population. The only way to achieve this objective for a sedentary species such as the cirl bunting is to translocate them to a site with suitable habitat. If this can be achieved, it will be a major conservation success, and the techniques used could be adapted for other species.

As cirl buntings are unlikely to recolonise favourable habitat once they have been extirpated from it, translocation was seen as the preferred method of securing a population distant from Devon. Adult cirl buntings are prone to stress, and so moving adults was discounted, as it was considered likely to result in unacceptable mortality (Jeffs and Evans 2004). In 2004, a small-scale translocation to develop rear and release techniques for nestlings was trialled. This programme was expanded to a larger translocation of birds from Devon to Cornwall in 2006. The aim was to establish a geographically separate, self-sustaining population by 2012.

Isosporoid coccidial infections, as taxonomically defined by Schrenzel and others (2005), have been reported in a wide variety of captive passerine birds, including canaries (*Serinus canaria*) (Poelma and others 1971), house sparrows (*Passer domesticus*) (Lainson 1959), greenfinches (*Carduelis chloris*) (Cooper and others 1989), Bali mynahs (*Leucospar rothschildi*) (Partington 1989), a bullfinch (*Pyrrhula pyrrhula*) (McNamee and others 1995) and in tanager species (Adkesson and oth-

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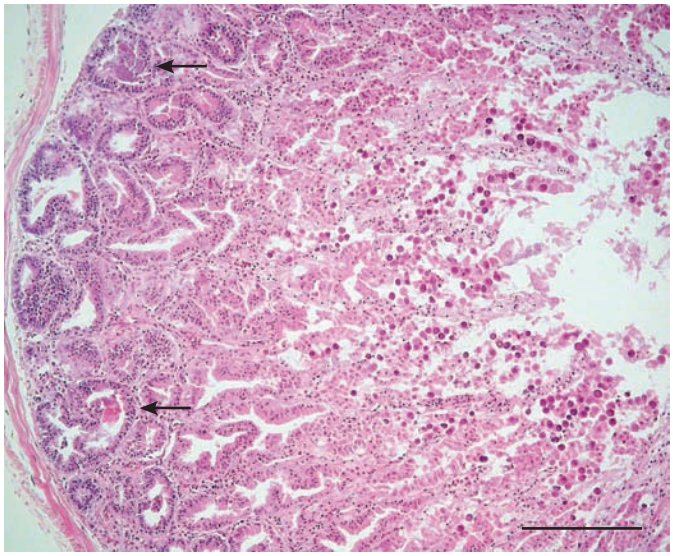

FIG 1: Numerous coccidial gamonts on the mucosal surfaces of the duodenum of a cirl bunting (CB1). Autolytic changes contribute to the disruption of mucosal morphology, and inflammation is minimal, but there are occasional crypts dilated with necrotic debris and heterophils (arrows). Haematoxylin and eosin. Bar=250 μm

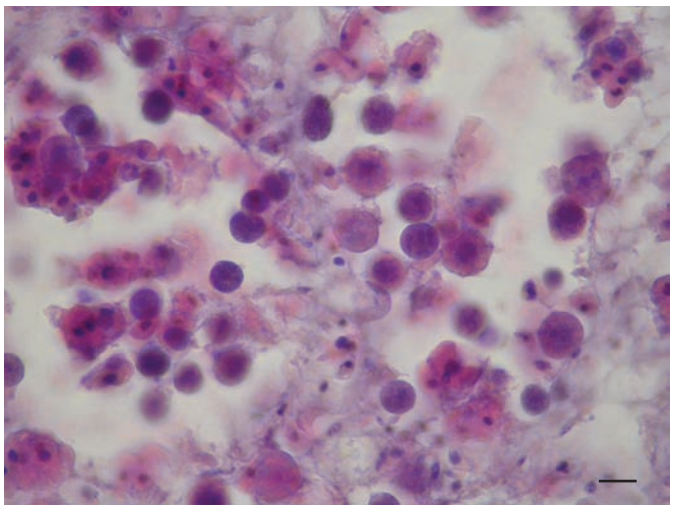

FIG 2: Numerous coccidial gamonts present among epithelial cells in the superficial mucosa of the duodenum of a cirl bunting (CB2). Morphology is suboptimal due to autolysis. Haematoxylin and eosin. Bar=10 μm

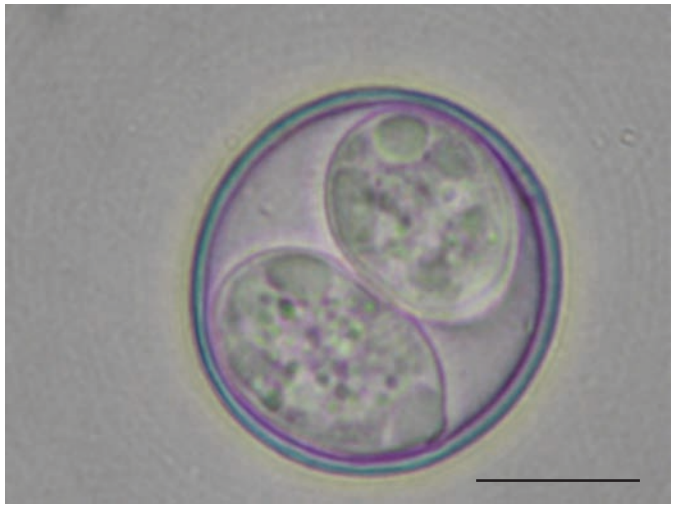

FIG 3: Sporulated oocyst of *Isospora normanlevinei* containing two sporocysts (isosporoid oocysts) identified in a pooled faecal sample from cirl buntings. Bar=10 μm

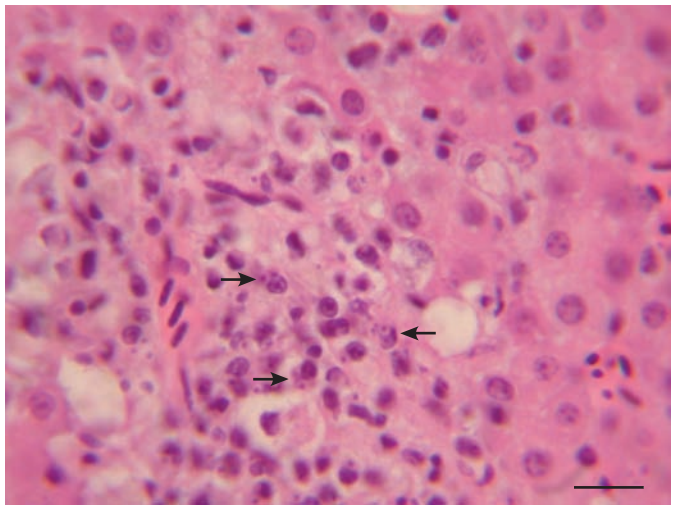

FIG 4: One of multiple foci of periportal mononuclear inflammation in the liver of a cirl bunting (CB4). Indistinct intracytoplasmic inclusions consistent with isosporoid coccidians can be seen in some of the infiltrating cells (arrows). Haematoxylin and eosin. Bar=20 μm

ers 2005). Isosporoid coccidia are a recognised cause of disease in captive passerines, secondary to high stocking densities, poor hygiene and other stressors (Greiner and Ritchie 1994, Dorrestein 2000, Sandmeier and Couteel 2006), but their pathogenic significance in free-living wild birds has not been investigated in detail as far as the authors are aware. Oocysts from two apparent species of *Isospora*, *Isospora normanlevinei* and *Isospora coluzzi,* have been identified in the faeces of healthy wild cirl buntings in Italy (Cringoli and others 1993); these parasites are probably a natural part of the cirl bunting's endemic fauna.

In the summer of 2004, 17 cirl bunting chicks were taken from the wild in Devon, reared and released in a trial translocation to establish effective techniques. This paper describes a disease outbreak among these 17 birds, associated with an isosporoid coccidial parasite infection, and the measures taken to prevent further isosporoid coccidial disease but conserve the isosporoid parasite in the reintroduced population, during a subsequent translocation of 75 birds from Devon to Cornwall in 2006.

Case histories

Trial translocation in 2004

Cirl bunting CB1 was collected from a nest in the wild in Devon on July 12, 2004, at approximately six days of age. It was colour ringed, weighed, transported to Paignton Zoo, hand-reared in a brooder at 27°C with other chicks in the same brood, and fed hourly by hand between 07.00 and 23.00. The chick was fed a diet consisting of locust, mealworm and a soft mix consisting of hard-boiled egg, Mazuri Diet A (Special Diets Service), SA37 mineral and vitamin supplement (Intervet UK) and banana. Water was given at each feed. At between 11 and 13 days of age, it was moved to a box cage for a few hours each day, where it remained from day 14; from approximately day 16 it was able to feed itself, including live mealworms from day 20. CB1 was found dead on July 27, 2004, aged approximately 21 days. A standard postmortem examination was performed (Latimer and Rakich 1994) at Paignton Zoo. The bunting was female and weighed 16 g; no significant abnormalities were evident upon gross examination. Samples of oesophagus, trachea, lung, liver, spleen, kidney, heart, proventriculus, ventriculus, duodenum, pancreas and striated muscle were fixed in neutral buffered 10 per cent formalin, and 5 µm tissue sections were cut and stained with haematoxylin and eosin. Significant histopathological abnormalities were found only in the small intestine. Moderate numbers of intraepithelial gamonts consistent with a coccidial parasite were present within the villous epithelium, associated with mild mucosal necrosis and crypt dilation by necrotic debris (Fig 1).

Cirl bunting CB2 was removed from a nest on July 21, 2004, at approximately six days of age; it was reared as for CB1. CB2 was found dead on August 6, 2004, at approximately 22 days of age.

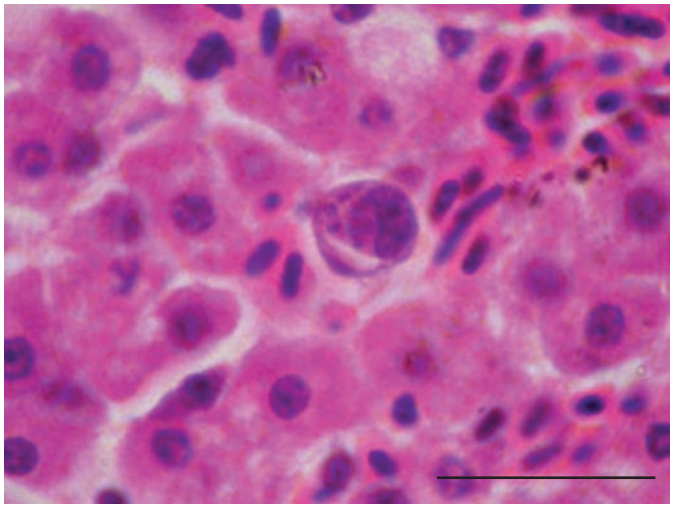

FIG 5: Schizont of *Isospora normanlevinei* in the liver of a cirl bunting (CB5). Haematoxylin and eosin. Bar=20 μm

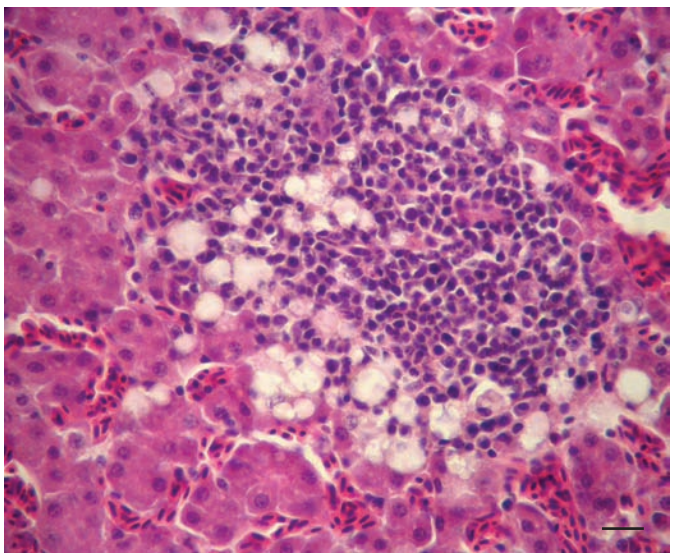

FIG 6: Liver of a cirl bunting (CB5) showing a typical focus of mononuclear inflammatory cells in a periportal location. Macrophages, some with vacuolated foamy cytoplasm, lymphocytes and plasma cells predominate. Organisms cannot be readily identified. Haematoxylin and eosin. Bar=25 μm

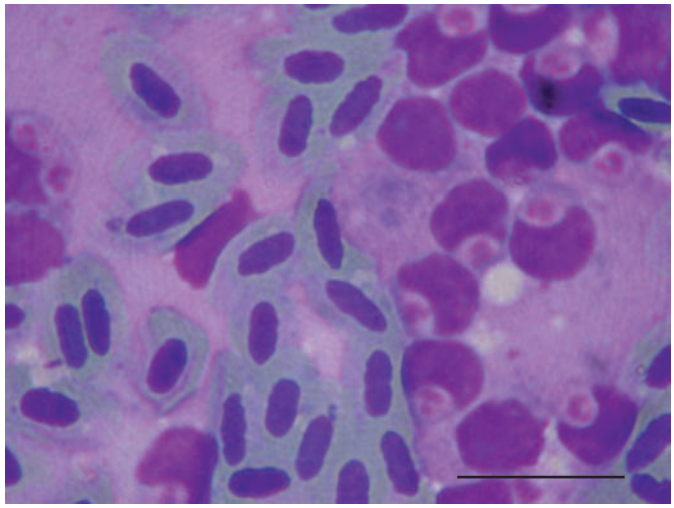

FIG 7: Merozoites of *Isospora normanlevinei* in leucocytes in a spleen impression smear from a cirl bunting (CB5). Giemsa. Bar=15 μm

At gross postmortem examination, CB2 was found to be male and weighed 13 g; no significant abnormalities were detected apart from the small intestinal contents, which were black/brown in colour. Microscopic examination of the large intestinal contents in a direct wet mount at magnifications of x10 and x100 demonstrated 28,000 coccidial opg. Large intestinal contents and a swab from the liver were plated on to Columbia blood agar including 5 per cent horse blood (QCM Laboratories), incubated aerobically at 25°C, and observed at one, two and five days, when bacterial isolates were tentatively identified using API biochemical tests (bioMérieux) as *Staphylococcus aureus* from the liver; no significant bacteria were cultured from the intestine. A swab from the liver was placed on Sabouraud's agar including chloramphenicol (QCM Laboratories), incubated aerobically at 25°C and observed for fungal elements at one, two, five, seven and 14 days, but none was seen. Samples of liver, lung, kidney, heart, proventriculus, ventriculus, brain and striated muscle, prepared as described above and examined histologically, were unremarkable. In the small intestine, there were numerous developmental stages (gamonts and meronts) of a coccidial parasite (Fig 2).

Cirl bunting CB3 was removed from a nest on July 12, 2004 and reared as described above until fledging, when wild seeds, mixed millet and the commercial feed Sluis (Haiths) were added to the diet, and the bird was transferred to a pre-release uncovered outdoor aviary, measuring approximately 3 x 2 x 2 m high, constructed of timber and wire mesh and with solid wood floors. The aviary contained a raised box cage, an array of branches, feeding utensils and a natural earthen floor. CB3 was found dead on August 30, 2004; autolysis of the carcase prevented interpretation of the cause of death.

Pooled faecal samples collected from the aviary floor and fittings on September 13, 2004 were positive for coccidial oocysts, which were sporulated to aid identification. Oocysts were extracted from the faeces by flotation in saturated sodium chloride solution. The faecal samples were soaked in water, mashed and washed into a centrifuge tube. This was spun at 550 *g* for three minutes and the supernatant was discarded. The pellet was disrupted and resuspended in saturated sodium chloride solution, and the tube was topped up to give a positive meniscus onto which a coverslip was placed. The samples were then centrifuged at 138 *g* for three minutes and then the coverslip was lifted off and the liquid that adhered to it rinsed with water to wash off the oocysts. The oocysts were suspended in 2 per cent potassium dichromate solution and then examined under a compound microscope (x 400 magnification). The oocysts were spherical, measuring 24 μm in diameter; each contained two sporocysts of approximately 15.2 x 11.36 μm (Fig 3). These measurements and morphology suggested that only one species of isosporoid coccidian was present, identified as *I normanlevinei* (Cringoli and others 1993).

Cirl bunting CB4 was removed from a nest on July 23, 2004, at approximately six days of age and reared as for CB3. It was found dull, 'fluffed-up' and inactive on October 1. It was treated with 20 mg/kg bodyweight enrofloxacin (Baytril; Bayer) but was found dead on October 2, at approximately 2.5 months of age. At postmortem examination, this cirl bunting, of undetermined sex, was emaciated and weighed 16 g. No parasites were detected in impression smears stained with Giemsa taken from the liver or spleen. A modified Ziehl-Neelsen stain of large intestinal contents was negative for parasites. Microscopic examination of large intestinal contents revealed multiple round oocysts consistent with unsporulated coccidial oocysts. Histopathological examination indicated that a moderate multifocal mononuclear inflammatory infiltrate was present in the liver (Fig 4), with a predominantly periportal distribution. Indented parasites within mononuclear cells were identified as merozoites of an isosporoid coccidian. Samples of heart, proventriculus, ventriculus, intestines, pancreas, brain, sciatic nerve, striated muscle, oviduct and skin were also examined by histopathology, but no abnormalities were detected. A blood smear collected from a cage mate of CB4 revealed *Haemoproteus coatneyi*. This is the first record of this parasite occurring in the UK, but in general it is not known to cause any morbidity in buntings (Bennett and others 1993).

The remaining 13 cirl buntings in this trial translocation were reared successfully and soft-released from the aviaries, with food being provisioned throughout the autumn and winter.

Isosporoid coccidial infection in a cirl bunting at Paignton Zoo

At the same time, the authors were investigating an outbreak of *Isospora* species disease in cirl buntings at Paignton Zoo; one case is described here because the preservation of the parasites and pathological lesions was significantly better than in the above four cases. CB5 was an 18-month-old cirl bunting kept under a similar regimen to the fledglings in the pre-release aviaries, which was part of a proposed captive breeding project at Paignton Zoo; it died on November 24, 2004. At postmortem examination, CB5 weighed 17 g and was in poor body condition. Heart blood smears and impression smears from the liver and spleen were made. Intraleucocytic merozoites of an isosporoid coccidian were seen in two of three heart blood smears. Impression smears from the liver contained a schizont (Fig 5) and showed periportal inflammation (Fig 6), and, in the spleen, merozoites of an isosporoid coccidian were observed (Fig 7). Coccidiosis was the likely cause of death of this bird.

Disease prevention during translocation of cirl buntings in 2006

In 2006, 75 cirl buntings were translocated from Devon to Cornwall. The birds were taken from their nests at approximately six days of age, transported to an isolated site in Cornwall, reared in brooders and later box cages, and soft-released from aviaries. In response to the cases of isosporoid coccidiosis described above, several changes in the birds' management were made for the larger translocation. Each brood of chicks was placed in quarantine throughout the rearing stages, in the brooder, box cage and aviary. Aviculture carers, who were dedicated to working with the translocated cirl buntings alone, changed their clothing and cleaned their hands using a skin decontaminant (F10 Hand Gel; F10 Biocare) between servicing each brood. Separate tools were used for each brood, and stocking densities were reduced, with no more than one brood of up to four chicks in each brooder, box cage or aviary. Footbaths containing disinfectant (Virkon; Russell Mainstream Supply) were placed between the brooder, box cage and aviary areas for disinfection of the carers' footwear. The brooders and box cages were cleaned after every feed with disinfectant wipes (F10 Wipes; F10 Biocare) and the aviaries were cleaned daily to reduce the parasite load in the environment through dilution. Toltrazuril (Baycox; Bayer) was administered orally, diluted in water, at a dose of 12.5 mg/kg bodyweight on two days per week when the birds were being hand-fed, and subsequently in drinking water on two days per week at a dilution of 45 mg toltrazuril per litre. The recommendations of Norton and others (2004) were considered in devising this dose rate. Faecal samples collected from the nest and transportation box at three, 10, 17, 24, 31 days after the birds were captured (collected in the morning) were examined microscopically at x 100 magnification to monitor for coccidial parasites. Coccidial oocysts were detected in one faecal sample from a nest and in one sample from a brooder. All subsequent samples were negative for coccidial oocysts, and as a result, the frequency of toltrazuril medication was reduced to once weekly. Faecal samples continued to test negative for coccidial oocysts. On the understanding that eradication of the isosporoid coccidial infection might be contraindicated in maintaining the health of the cirl buntings after their release, three faecal samples from three free-living cirl buntings, released in summer 2006, were collected in January 2007 and examined by the salt flotation method. One sample contained *Isospora* species oocysts that were identified as *I normanlevinei*. No isosporoid coccidial disease was detected through clinical or pathological investigations in any cirl bunting in 2006, and 72 of the 75 wild-caught birds were successfully released in 2006.

Discussion

During captive rearing for translocation in 2004, four of 17 cirl bunting chicks died over a three-month period. Pathological changes associated with an isosporoid coccidial infection were detected in two of these birds, and coccidial oocysts were detected in the faeces and intestine of another. No other significant pathological findings were detected; thus, the authors surmise that isosporoid coccidiosis probably was the cause of this disease outbreak. Sporulated oocysts found in faecal samples from the same group of birds were identified as *I normanlevinei*; therefore, this was the probable agent of the disease. Infection with more than one isosporoid coccidian parasite cannot be ruled out, because extraintestinal and intestinal coccidial forms were detected in CB4, which might indicate concurrent infection with two isosporoid coccidial species. As *I normanlevinei* has been detected in free-living cirl buntings within their geographical range in Europe, this parasite is assumed to be an endemic parasite that has coevolved with cirl buntings, and to also be native to the UK. The work of Schrenzel and others (2005) on passerine bird species suggested that the majority of isosporoid coccidia cospeciate with their passerine host and are thus host-specific. However, more than one species of isosporoid coccidial parasite was present in five of the 23 passerine bird species studied by Schrenzel and others (2005), and individual birds from four of these species harboured more than one coccidial species. Sequencing of coccidial parasite DNA from the intestinal and extraintestinal tissues could be used to establish the number of parasite species present in the cirl buntings described here.

The necrotic enteritis seen in CB1 was mild and was not nearly as severe as that typically reported in transmural lymphocytic enteritis associated with coccidiosis in, for example, captive Nashville warblers (*Vermivora ruficapilla*) (Swayne and others 1991). Isosporoid infections in the intestines of birds are of variable pathogenicity (Schmidt and others 2003). In many cases, infections in the natural host may be grossly inapparent and characterised solely by the presence of organisms in enterocytes and ova in faeces, without pathological changes. In heavy infections, gross lesions can range from increased fluid content in the intestinal lumen to fibrinonecrotic enteritis. Histologically, these latter cases have a predominantly mononuclear mucosal inflammatory reaction, associated with acute mucosal necrosis. In the cirl buntings described in this paper, autolytic changes in the intestinal sections hindered interpretation, but mild to moderate necrotising enteritis accompanied by the presence of intraepithelial organisms was recorded. Secondary bacterial infection was suspected to have contributed to the enteric lesions in CB1. Hepatic lesions in isosporoid coccidial infections of birds are typical of those caused by apicomplexan parasites, being characterised by multifocal non-suppurative inflammation with a portal or sinusoidal distribution. The hepatic necrosis that can be a feature of severe infections was not present in the cases reported here.

The quarantine, hygiene and other management changes, and prophylactic toltrazuril treatment instituted in 2006 were associated with an absence of isosporoid coccidial disease. It has already been established in the literature that measures to reduce stress may reduce the excretion of isosporoid oocysts, and improved hygiene and prevention of overcrowding can reduce the level of orofaecal transmission of isosporoid coccidia, and all these measures are important in controlling this disease in passerine birds (Dorrestein 2000, Zinke and others 2004, Sandmeier and Couteel 2006). Considering the success in rearing cirl buntings in 2006, the authors believe that sanitation and stocking density had an influence on the disease outbreak in 2004. The authors believe that an isosporoid coccidian is part of the natural parasitic fauna of cirl buntings in the UK because in 2006, isosporoid coccidia were detected in a nest sample from free-living native cirl buntings in Devon, and isosporoid coccidia completed their life cycle in the cirl buntings described here in 2004; these parasites tend to be host-specific, and there were no exotic passerines of the Emberizinae at Paignton Zoo in 2004. However, the authors were concerned that the control methods implemented in 2006 might have eradicated the isosporoid coccidia, which might have disadvantaged the birds once they were reintroduced to the wild if they were subsequently re-exposed to the parasite. The detection of *Isospora* species in one faecal sample from a reintroduced cirl bunting assuaged these concerns. Only a few coccidian parasites are required to stimulate immunity, and they are in general very immunogenic (Shirley and others 2005).

De Leo and Dobson (2002) considered it unwise to eradicate parasites from translocated hosts, particularly in situations where the hosts might be exposed to the parasite again in the destination environment and the parasite had coadapted with the host over a prolonged period, as is likely to be the case with the cirl bunting and *I normanlevinei*. Studies on the effect of eradicating the introduced pathogen rinderpest virus from the Serengeti ecosystem have shown that pathogens/parasites have a significant role in the ecological community (De Leo and Dobson 2002). Therefore, there are good reasons for attempting to conserve 'native' parasites in translocated and reintroduced populations, and the present paper presents preliminary evidence that it is possible to do this even when the parasites are pathogenic.

These findings suggest that isosporoid coccidiosis can be a hazard to the translocation of cirl buntings and possibly other passerine birds, and this disease should be considered in future conservation projects involving the translocation of rare passerine species. Hygiene and prophylactic measures are well established and were effective in controlling disease in the cirl buntings reared for reintroduction in this programme, without the need to eradicate the isosporoid parasite.

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Notes