Variation of parasite burden within the European badger (*Meles meles*): the effect of season, habitat, body condition, gender & age on the prevalence of *Eimeria melis* and *Capillaria*

Submitted by Elizabeth Rosemarie Anne Cottrell to the University of Exeter as a thesis for the degree of Master of Science by Research in Biosciences in October 2011

I certify that all material in this thesis which is not my own work has been identified and that no material has been previously submitted and approved for the award of a degree by this or any other University.

Signature……..

Table of Contents

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E. R. A. Cottrell [1, 2]

[1]*Food and Environment Research Agency, Woodchester Park, Tinkley Lane, Nympsfield, Gloucestershire, GL10 3UJ; and* [2]*Centre for Ecology and Conservation, University of Exeter Cornwall Campus, Tremough, Treliever Road, Penryn, Cornwall, TR10 9EZ.*

Summary

1. Parasites, although naturally occurring can have severe impacts on both an individual host and on the wider population and ecosystem.

2. This study investigates the relationship of parasite burden within the European badger (N=175) with several life history characteristics such as age, condition, gender and co-infection of bovine tuberculosis and with the environmental factors of habitat and season.

3. Using two general linear mixed models results showed significant positive relationships between *Eimeria melis* burden and age and gender, and between *Capillaria spp.*, age and month of sampling. A significant negative correlation was also observed between *Capillaria spp.* and badger body condition index. No significant results were found for habitat type or in the further GLMM's (N=124) run to investigate co-infection of bovine tuberculosis.

4. The opportunity to investigate complex parasite interactions in a wild population of known individuals is rare and a valuable opportunity. Parasite burdens in *Meles meles* were found to be extremely variable with some exhibiting very high faecal egg/oocyst output. Results suggest that such burdens of gastro-intestinal parasites do have a relationship with life history characteristics and condition and therefore should be taken into account when wildlife disease management protocols or ecological studies are carried out.

Key-words: badger, coccidia, *Capillaria*, helminth, *Eimeria melis, Meles meles*, nematode, parasite.

Introduction

THE IMPORTANCE OF PARASITES

The relationship between a parasite and its host is naturally occurring and a way of life (Perez *et al.* 2006). However, parasitism is known to play an important role in the evolution and ecology of the host organism and consequently the population and the ecosystem in which it inhabits (Combes 1996). In literature parasites are defined as a special variety of predators which do not cause immediate death to their prey (their hosts) (Perez *et al.* 2006). They instead, rely on the energy that would otherwise been allocated to normal host bodily functions and development, therefore becoming detrimental to the health & survival, longevity, and fecundity of the host (Alzaga *et al.* 2006, Coltman *et al.* 1999, Hillegrass, Waterman & Roth 2010, Hudson 1986, Stodart 1968, Tompkins & Begon 1999). The true cost of this relationship is largely unknown due to the difficulty of quantifying energy costs in natural populations (Hillegrass, Waterman & Roth 2010) and because pathogenic effects of parasites are not always obvious or easy to measure (Marcogliese & Pietrock 2011). Parasitic effects are also likely to be linked strongly to other stressors (such as variation in biotic or abiotic factors), the combination of which may be more debilitating than either stressor alone. Parasitic effects are also not likely to be independent of other species (a combination of parasites is frequently found in host organisms) leading to a more pronounced affect on the host than a monospecific infection (Marcogliese & Pietrock 2011) and adding further difficulty to interpreting parasitic relationships and understanding data and analyses.

Undoubtedly though, parasites have the potential to profoundly influence the structure and stability of natural communities (Rosalino, Torres & Santo-Reis 2006, Sheldon & Verhulst 1996) and quite possibly are a factor in regulating the population size of host species, albeit that they may mainly have an effect on individuals that are already vulnerable (Casselinello, Gomendio & Roldan 2001, Dawson & Bortolotti 2000, Tompkins & Begon 1999). Parasites may act as an important selective force, resulting in wider genetic diversity of host species and consequently their own large diversity (Alzaga *et al.* 2007, Casselinello, Gomendio & Roldan 2001, Coltman *et al.* 1999, Perez *et al.* 2006) and may be important in sexual selection and in allocation of resources for cognitive development (Sheldon & Verhulst 1996).

The vertebrate intestine can be considered one of the major ancestral sites for parasites. In evolution, access to the bodies of vertebrate hosts is likely to have occurred through accidental ingestion and survival in the intestine would have been favoured by the free availability of nutrition (Wakelin 1996), and by the immune system tolerating a low intensity of infection in order to optimise energy allocation (Sheldon & Verhulst 1996). Gastro-intestinal parasite species are overall, still the commonest, although not the most pathogenic of all parasites (Wakelin 1996) and have the research advantage of being easily detectable

using non invasive methods such as microscopic faecal analysis. This allows much larger sample sizes than would be possible for parasites found elsewhere in the body that are perhaps only detectable through post-mortem.

The distribution of parasites throughout individuals in the population is far from random, but highly complex. Evidence in the field for patterns is vast and conflicting, as well as highly species dependant for both parasite and host (Anderson 2000, Taylor, Coop & Wall 2007) but even though not fully understood evidence points to relationships existing between parasite burden and such factors as age (Douche & Moram 1993, Schalk & Forbess 1997, Stodart 1968), cycles in season and within the host (Fayer 1980, Haukisalmi, Henttoren & Tenora 1988), foraging and other behaviours (Anderson 2000, Rosalino, Torres & Santos-Reis 2006), gender (Poulin 1996) and geographical range (Torres, Miquel & Motje 2001).

THE EUROPEAN BADGER

The European badger is host to a wide range of parasites, including a variety of gastro-intestinal species (Dale 2005, Hancox 1980, Rossalino & Torres 2006, Rosalino, Torres & Santos-Reis 2006, Massey, Elsheika & Morsy 2009, Torres, Miquel & Motjé 2001). It has also been the subject of extensive study in the United Kingdom due to acting as a carrier for the disease bovine tuberculosis or *Mycobacterium bovis* (bTb), a disease that in cattle is of great economical significance (costing the government in excess of £100,000,000 a year in compensation and research (Jenkins, Woodroffe & Donnelly 2010)) and for which the badger is said to act as a wildlife reservoir (Woodroffe *et al*. 2006). This extensive research on life history and behaviour in combination with access to a population of animals with data from known individuals makes the European badger the ideal model species in which to investigate the relationship parasites hold with potentially influential abiotic and biotic variables.

The European badger (*Meles meles* L.1758) is the largest remaining carnivore that naturally occurs in the UK. It is commonly regarded as a subspecies of the Eurasian badger but currently the exact classification of the badger subfamily is under taxonomic debate (Roper 2010). Badgers belong to the mustelidae, a family defined by its specialised scent glands and although normally carnivorous with well defined canines, the badger has teeth more adapted to its wide omnivorous diet (Harris & Yalden 2008). Over their wide range, the habits of the badger vary substantially but within the UK they live in social groups, in setts underground (Rogers *et al.* 2003, Roper 2010).

The basic lifestyle of a *M. meles* is a lifestyle in which you would expect diseases to thrive. Living in a high population density (up to 30 adults/km^2)(Roper 2010), badgers forage through soil or sources of decaying matter such as leaf litter, cattle faeces, silage, compost and garbage. Conditions in the sett are highly humid and of a moderate temperature, and badgers spend a large quantity of time in close proximity with

members of the same species. However, whilst like most species (Marcogliese & Pietrock 2011) the badger is vulnerable to a number of diseases and parasites (Hancox 1980) it would seem that it is very rare that badger populations are decimated by disease on anything other than a local level indicating that they are in general a hardy species (Roper 2010), and when within the parameters of a normal parasite load the majority of these parasites will be non-fatal and have little observable effect on reproduction or population numbers (Hancox 1980). It is only in the case of higher rates of infection that more obvious severe symptoms develop.

GASTRO-INTESTINAL PARASITES IN BADGERS

Because of the difficulties in studying wildlife populations the majority of parasite literature is based on studies of domestic animals. Very little research material is available specifically on parasites in the European badger, particularly in the UK and the studies that do exist are largely opportunistic or have a small sample size (Hancox 1980, Massey, Elsheika & Morsy 2009) with only a few exceptions (Dale 2005, Newman, MacDonald & Anwar 2001). This makes speculation of the specific symptoms of specific species of parasites difficult. However this is not to say that research of domestic animals is not of use for drawing general patterns, indeed many of the symptoms caused by such parasites are similar across host species, as long as such information is treated with caution. Animals experiencing heavy gastro-intestinal parasite burdens may experience symptoms including diarrhoea, vomiting, and weight loss. In badgers this is more commonly observed in cubs who appear to have either a less developed immune system or are more vulnerable to infection due to encountering a different lifestyle to adults (Newman, MacDonald & Anwar 2001). It is has also been suggested that prolonged exposure to severe parasite burdens in badgers may potentially lead to abnormal behaviour and enhanced vulnerability to road traffic accidents, and high levels of infection may be an important factor in cub mortality (Hancox 1980). Two of the most frequently occurring gastro-intestinal taxa are the helminths and coccidia.

COCCIDIA

Coccidiosis is a disease affecting the intestinal tract of animals, it is caused by protozoan organisms known as coccidia. The presence of which is widespread in vertebrates (Cassey & Ewen 2008). The primary symptom of coccidiosis is diarrhoea, but it may also cause vomiting, appetite loss and dehydration. Specifically to badgers, cubs present with swollen abdomens and diarrhoeal enteristis (Newman, MacDonald & Anwar 2001). Most information about *Eimeria* epidemiology and life cycle is taken from research carried out on domestic animals where the impacts of coccidiosis are economically important and consequently more widely researched than in wild animals (Anwar *et al*. 2000), but the wealth of information for free-living animals is slowly increasing.

Evidence from cattle (Fayer 1980) and from birds, such as three species of exotic passerine in New Zealand suggests that a low level of coccidia within the gut is normal and in most cases asymptomatic (Cassey & Ewen 2008), unless burdens become severe (often due to a combination of other stressors on the individual making it prerequisitably vulnerable) when the effect of coccidia burdens becomes much more apparent and may result in death (Newman, MacDonald & Anwar 2001). However studies like this often do not account for unseen effects in life history and energy allocation (Sheldon & Verhulst 1996) such as the ability to escape from predators, as was shown to be reduced in hares by Alzaga *et al.* 2008, further adding to the evidence that parasites can act as a selective force within their hosts.

The first record of coccidian infection in the European badger was by Kotlan and Perspech (1933) who reported *Eimeria melis* as a new species, they also reported a second coccidia species which later became known as *Isospora melis* (Anwar *et al.* 2000). Almost all members of the genus *Eimeria* are considered monoxenous due to the life cycle of the parasite being completed within a singular host (Anwar *et al.* 2000, Fayer 1980). They are also considered mostly stenoxenous because usually each species of *Eimeria* parasitises a single species of host, although some cross transmission has been shown in experimental situations (Anwar *et al.* 2000, Fayer 1980).

Across the *Eimeria* genus life cycles are very similar and species are most commonly differentiated through oocyst structure (the method which will be used in this study), but other factors are sometimes used such as structure of endogenous stages, host species, location of endogenous stages within the host, and cross immunity, contributing to an estimated 2,644,736 identified species of *Eimeria* (Fayer 1980). Transmission of oocysts takes place through ingestion of contaminated faecal matter, *Eimeria* then multiplies in the gut and when the cycle is complete is excreted once more into the environment (Fayer 1980).

NEMATODES

Helminth parasites are also extremely widespread in vertebrate populations, and highly diverse. Although many helminth transmission routes remain unclear, information on *Capillaria* (the second most common parasite taxa found in this experiment) transmission is available, albeit not in vast quantities. Similarly to coccidia, research has been biased towards those parasites that affect humans and domestic animals with a more limited emphasis of study on wild animals, however many species have being investigated and even though the variation is vast, findings represent a theoretical template which can be used as a base for comparative study (Anderson 2000). Also similarly to coccidia, evidence would suggest that a low level burden of helminth parasites is normal and rarely fatal. Research has showed infected badgers in most cases exhibit few or no obvious symptoms, particularly in badgers on the continent where a much higher diversity and prevalence of helminth parasites is observed (Torres, Miquel & Motjé 2001), but may have

long term less obvious, debilitating symptoms which remain unseen, their effects upon the host originating in pathological alterations of intestinal structure and function (Wakelin 1996). As with other gastro-intestinal disorders severe *Capillaria* infection can cause vomiting, diarrhoea and weight loss, and infection appears to have effects on energy allocation particularly with respect to breeding success (Hudson 1986, Vandegrift, Raffel & Hudson 2008) and immunology and life history characteristics (Sheldon & Verhulst 1996).

The nematodes (phylum nemathelminthes) or roundworms, are elongated cylindrical worms. Most are free living but a large number of species (approximately 33% of genera) parasitise animals and plants (John & Petri 2006), to date 594 species have been investigated, 16-17,000 have been described, and an estimated 40,000 species exist (Anderson 2000). Amongst these is the family Capillariinae, of the superfamily Tricuroidea. Capillariinae are extremely common in the gut, urinary tract and respiratory system of vertebrates (Anderson 2000) and the eggs can be found in faecal analysis. Worldwide, multiple species of *Capillaria* have been identified in the badger. This is not surprising seeing as it is such a versatile animal with an extremely wide variation in diet and habitat. In the UK however the list of species is somewhat smaller, even more so due to recent reclassification reducing the number of species. Of those identified (it is very difficult to identify helminths at species level using faecal analysis and frequently records of *Capillaria* are not identified down to species level (Rosalino, Torres & Santos-Reis 2006)) there are records of *Capillaria erinacei* (Jones, Neal & Harris 1980 found it in 25% of badgers sampled in Cornwall) and *Capillaria mustelorum,* however these have been reclassified as *Capillaria* (synonym *Aonchotheca*) *putorii* (Butterworth 1980, Fahmy 1964) other records of which have also been found in the UK (Millan *et al.* 2004)

AIMS/HYPOTHESES

This study will assess the link between common parasites of the badger and multiple explanatory variables within the population. The effect of season, habitat, body condition, gender & age will be tested against the prevalence of *eimeria melis* and *Capillaria* species. This study aims to offer a rare insight into the parasite ecology and its effect on a free living animal.

Materials and methods

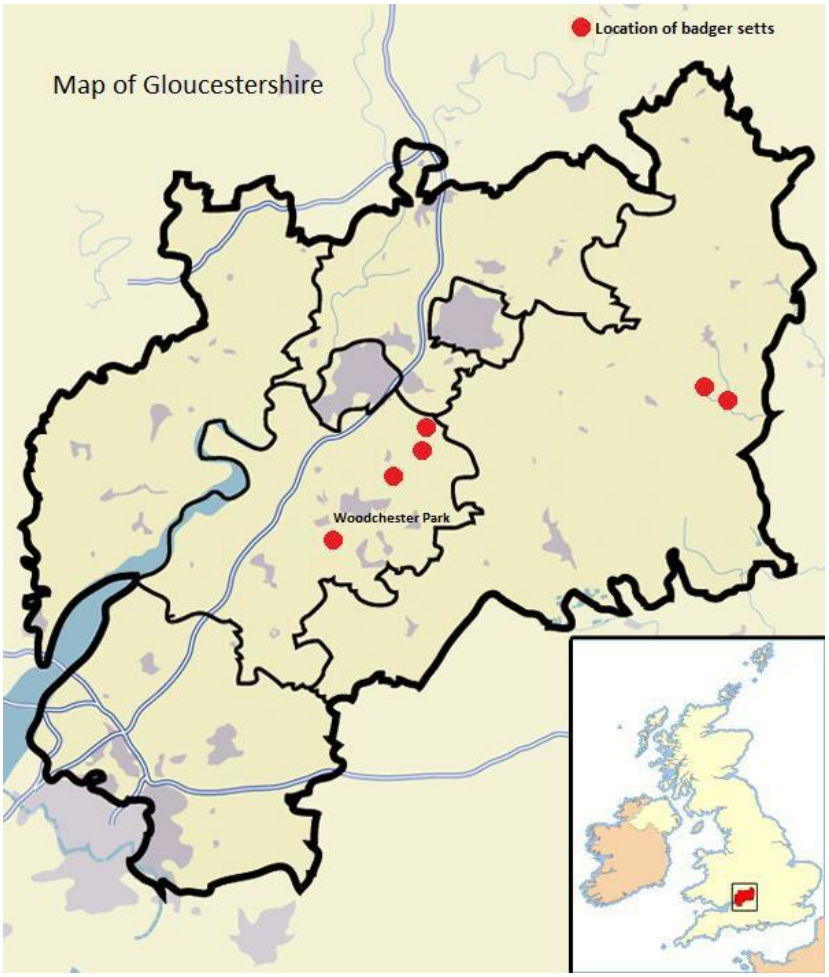
STUDY SITE

Badger samples were obtained from Woodchester Park and the surrounding Gloucestershire area during established research projects (Map 1). Samples were obtained from these sources rather than from

latrines as procedure allowed all faeces to be obtained from individually identifiable badgers, and cross referenced with morphometric data.

Woodchester Park, in Gloucestershire, England, is the site of a long-term research project investigating the role of the Badger in the transmission of *Mycobacterium bovis* to domestic cattle. The study site has been researched intensively since 1981 using a continuous regime of live trapping and sampling in combination with territorial mapping using bait marking (Delahay *et al.* 2000, Rogers *et al.* 2003). The site itself provides ideal conditions for badgers such as cover in the form of woodland combined with slopes and a soil type suitable for excavating setts (Roper 2010) and is surrounded by key feeding habitats (Rogers *et al.* 2003). The site covers 7km² of Cotswold sandstone escarpment and has a resident badger population comprising of 25 social groups (Delahay *et al.* 2000, Rogers *et al.* 2003)

The badgers from the surrounding area were obtained from 5 distinct social groups in separate locations (Bridleway (51˚50’29.84”N 1°45’26,55”W), Butlers (51˚47’26.61”N 2˚08’37.24”W), Frith Wood (51˚46’08.30”N 2˚11’10.50”W), Overtown (51˚48’35.55”N 2˚08’37.24”W) and Sandy Hill (51˚50’29.84”N 1˚45’26,55”W)) Habitat was predominantly woodland but the surroundings of the sett varied somewhat.



Map. 1. Showing the location of setts from which badgers originated.

COLLECTION OF SAMPLES AND BADGER MORPHOMETRIC DATA

A total of 176 faecal samples were collected from 131 badgers. Badgers were sampled from a total of 28 social groups spread over Gloucestershire. Within the Woodchester Park area 23 social groups and 144 samples were taken from 108 Individuals and from the surrounding Gloucestershire area 5 Social groups and 32 samples from 23 Individuals were taken. The Social groups were identified in Woodchester from bait marking analysis within the park (Delahay *et al*. 2000) and external to the park where bait marking was not possible, by a distance that was >2km between main setts. Samples were collected during May, June, August, September and October 2010. All other Gloucestershire badgers and no Woodchester badgers were sampled during August.

Badgers were caught using cage traps positioned at active setts and pre-baited with peanuts for a week prior to setting. After capture they were transported to the sampling facility in holding cages and anaesthetised via intramuscular injection (details in Appendix A). Data for body weight and length (nose to tip of tail bone) was recorded along with sex, age and body condition (a qualitative scale of 5 based on visual assessment made by the sampler taking into account fur condition, weight and overall health). BTb status in the Woodchester badgers was determined as excretor, exposed or negative based on blood samples via jugular venepuncture, alongside swabs of sputum, pus from any open wound and urine which were taken and used for *M. bovis* analysis using bacterial culture and Brock ELISA antibody test (Chambers *et al*. 2008, Clifton-Hadley, Wilesmith & Stuart 1993). Following recovery badgers were returned to setts and released at point of capture. (Details of further samples taken but not used in this study can be found in Appendix B.)

Badger Body Condition Index was calculated as an objective measure of body condition (as a contrast to the qualitative measure above which is vulnerable to sampling bias) using the relationship between weight and body length and calculated against mean values for season from past Woodchester data (1997-2009) for adult males, adult females and cubs separately (Tomlinson, Unpublished PhD thesis, Woodroffe & MacDonald 1995). Habitat type was defined using maps of the sett location and prior surveying of the area.

Faecal samples were recovered from a metal tray placed under holding cages during badger recovery, the Woodchester Park badgers received an 5 ml ‘Micralax’ enema (comprised of sodium citrate and sodium alkysukphoacetate) whilst under anaesthetic to stimulate defaecation. Other Gloucestershire area badgers did not receive enemas because it was not necessary to analyse faeces for *M.bovis*, however most animals defecated without. A 4g subsample of recovered faeces was removed and mixed with 4ml of 70% ethanol in individual plastic containers. This was stored for a minimum of 24 hours to destroy any bTb bacteria and render it safe for analysis. Samples were stored in a refrigerator at 4°C. All faecal samples were collected

early in the morning to account for differences in circadian rhythms as far as possible, however faeces may have been excreted at any point during the previous night.

FAECAL ANALYSIS METHODS

Levels of gastro-intestinal parasite burdens were determined by excreted oocyst/ova counts. This was carried out using an adapted version of the "McMaster" faecal flotation method (Tomlinson, Unpublished Phd thesis). Using weighing scales and a wooden spatula, 6g of the faeces/ethanol mixture was extracted and mixed with 39ml of tap water. Glass beads were added and the sample vigorously agitated for 2 minutes to break up the faeces. The mixture was placed through a tea strainer of a 1mm² mesh and the debris discarded as clinical waste. 15ml of the strained solution was transferred immediately to a 15ml falcon tube and spun in the centrifuge for 2 minutes at 800G. The supernatant was discarded and the remaining faecal pellet re-suspended with 15ml saturated sodium chloride/sugar solution (specific gravity 1.280) and inverted to mix. A subsample was taken from the middle of the tube using a pipette and used to fill both counting chambers of the McMaster slide. The slide was placed under a binocular microscope to stand for 2 minutes to allow for egg flotation, then using the 10x objective helminth ova and coccidial oocysts were counted and identified from both chambers. This count was multiplied by 50 to give number of oocysts/ova per gram.

A preliminary exploratory analysis by Newman, MacDonald & Anwar 2001 suggested that there were not significantly more observations of oocysts/eggs when looking at ten slides per faecal sample than for looking at two. Consequently this experiment only used a repeated measure of two (both chambers in a McMaster slide). In other studies an approximately linear relationship between number of parasites and number of oocysts is observed in multiple species, this makes faecal egg counts a valuable and reasonably accurate measure of parasite burden, and a valuable tool in monitoring wildlife populations (Newman, MacDonald & Anwar 2001).

STATISTICS METHODS

General Linear Mixed Models using the laplace approximation and the function GLMER in R were used to analyse data (R Development Core Team 2010). Parasite species were analysed individually because of large variance in life cycle that would have resulted in a combined parasite burden measure that hid potential results (Marcogliese & Pietrock 2011 caution against lumping parasites species together for this reason). Consequently a separate model was run for both *Eimeria melis* burden and *Capillaria* burden. Burden was determined as egg or oocyst count per gram and was categorised to make mulitinomial analysis possible using boundaries specified by log transforming the data ($\log_e(x)$). Categories were as follows: *Eimeria;* 1=<7, 2=<55, 3=<403, 4=<2981, 5=<22,026, 6=<162,755, 7=<1,202,604. *Capillaria;* 1=<3,

2=<7, 3=<20, 4=<54, 5=<148, 6=<403, 7=<1097, 8=<2981. *Capillaria* burden was analysed as the response variable with sex, age (adult or cub), Badger Body Condition Index (BCI), habitat and condition as explanatory variables. *Eimeria* burden was run as a similar model using sex, age, BCI and habitat as explanatory variables. Models were fitted using a poisson error structure with region of trapping, social group and individual badger as random effects.

Further to these models, in order to evaluate the relationship of parasite burden and bTb status it was necessary to run additional analyses as separate models due to the difference in sample size (only Woodchester badgers underwent testing for bTb). These GLMM's again used *Capillaria* and *E. melis* as response variables but only included those explanatory variables that remained in the minimal adequate model from the former analyses (*Capillaria* against bTb status, Month, Age and BCI. *Eimeria* against an Age-Sex interaction, bTb status and BCI), with a poisson error structure and social group and individual badger as random effects.

Results

Although a variety of parasites were observed in this study, attention was focused purely on the 2 most frequent parasite types found in faecal samples. *Eimeria melis* and *Capillaria* species (probably *Capillaria putorii*).

COCCIDIA

Eimeria melis was the most prevalent parasite recorded in faecal analysis. Only 85 faecal samples were negative for *E. melis*, the remaining 91 badgers had a burden varying from between 50 to 413,700 oocysts per gram. Burden of *Eimeria melis* (Table 1.) was highly significantly related (*P*=<0.05) to age and sex (*P*=3.996e-07, N=175). Male cubs carried the highest burden of coccidia ($\bar{}$=6.647228) followed by female cubs ($\bar{}$=5.093168), male adults ($\bar{}$=2.465513) and female adults ($\bar{}$=1.889099) (Fig. 1, Table 2.). *E. melis* was not significantly related to habitat (*P*=0.1623), condition (*P*=0.2203) month of sampling (*P*=0.469) or to BCI (*P*=0.05661).

Table 1. Showing values for the GLMM of the parasite burden of *Eimeria melis* as a response variable.

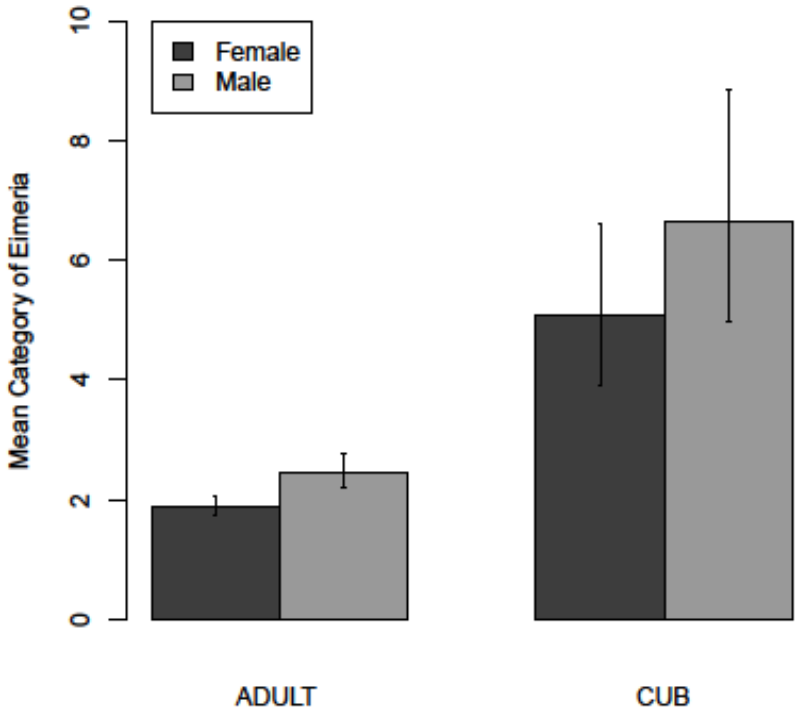
Factor	Degrees of Freedom	χ^2 Value	*P* Value
Month	4	3.8646	0.4246
Condition	3	4.1712	0.2436
Habitat	4	7.4433	0.1142
BCI	1	3.6339	0.05661 .
Age*Sex	3	32.556	3.996e-07***

Signif. codes: '***' 0.001 '**' 0.01 '*' 0.05 '.' 0.1 ' ' 1

Table 2. Showing the significant parameter estimates between *Eimeria melis* and the sex-age interaction.

	Estimate	Standard Error
Adult Female	1.889099	2.063699
Adult Male	2.465513	2.763506
Cub Female	5.093168	6.611430
Cub Male	6.647228	8.853386

Figure 1. Showing the significant relationship between mean burden of *Eimeria melis* and the age and sex categories.



CAPILLARIA

Capillaria species were the second most frequently recorded parasites in faecal samples. Based on miscroscopic analysis (of size, colour and shape) and historical records these *Capillaria* oocysts were most likely to be *Capillaria putorii*, however due to the similar nature of *Capillaria* eggs identification cannot be 100% definite. 28 samples were positive for *Capillaria* and 148 negative. The positive badgers had burdens ranging from 50 to 1800 eggs per gram. Burden was also significantly related to age (P=1.783e-06) (Fig.2) but was additionally significantly related to month (P=0.02857) (Fig. 2) and to BCI (P=0.04139) (Fig. 3). *Capillaria* burden was not significant to habitat type (P=0.1242) or to sex (P=0.6799).

Table 3. Showing values for the GLMM of the parasite burden of *Capillaria spp.* as a response variable.

Factor	Degrees of Freedom	χ^2 Value	*P* Value
Sex	1	0.1702	0.6799
Habitat	4	7.2316	0.1241
BCI	1	4.1601	0.04139 *
Month	4	10.828	0.02857 *
Age	1	22.816	1.783e-06 ***

Signif. codes: '***' 0.001 '**' 0.01 '*' 0.05 '.' 0.1 ' ' 1

Table 4. Showing the significant parameter estimates for BCI, age and month within the *Capillaria* model.

	Estimate	Standard Error
BCI	-0.14009	0.07548
Adult May	1.087357	0.8738470
Adult June	1.088913	0.8844228
Adult August	2.189208	1.8918969
Adult September	1.313152	1.0664229
Adult October	1.588960	1.2735649
Cub May	2.525982	1.7316592
Cub June	2.529597	1.7526168
Cub August	5.085636	3.7490782
Cub September	3.050514	2.1132774
Cub October	3.691231	2.5237604

Figure 2. Showing the relationship between the significant parameter estimates of *Capillaria* burden and categories of month and age of badger.

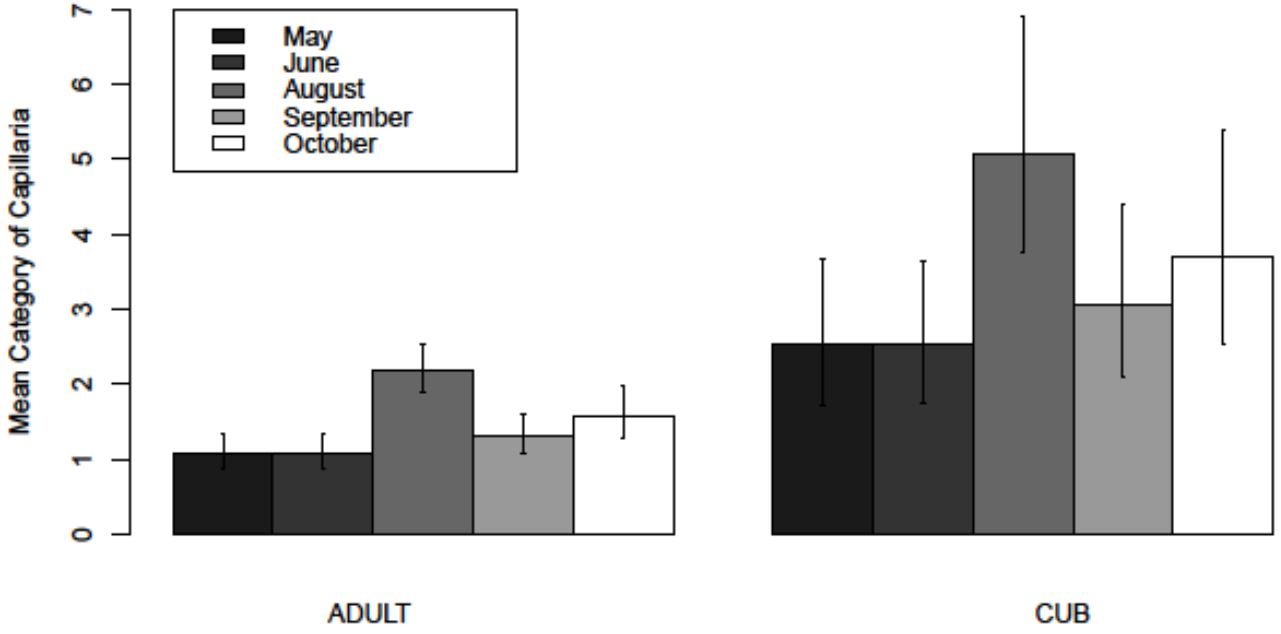
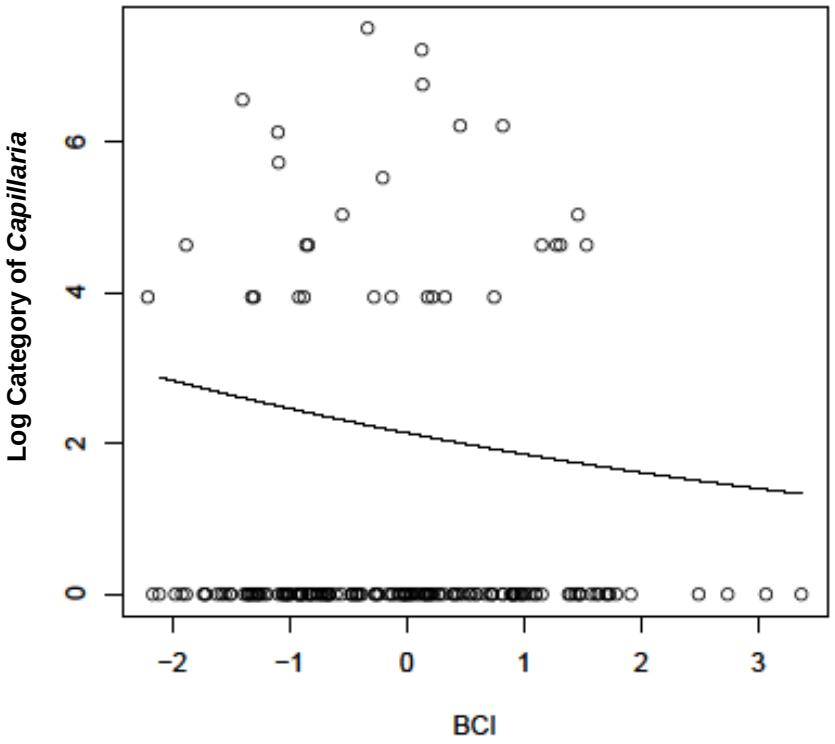


Figure 3. Showing the relationship between logged *Capillaria* count and badger body condition index.



BOVINE TUBERCULOSIS

There was no significant relationship between bTb status (N=124) and either burden of *Capillaria spp.* (*P*=0.8612) or burden of *E. melis* (*P*=0.3107).

Table 5. Showing the non significant values from the 2 models run with parasite burden of *Capillaria spp*. and *E. melis* as the response variables and individual badger status of bovine tuberculosis.

	Degrees of Freedom	Chi² Value	*P* Value
Capillaria spp.	2	0.2988	0.8612
Eimeria melis	2	2.3378	0.3107

Signif. codes: '***' 0.001 '**' 0.01 '*' 0.05 '.' 0.1 ' ' 1

Discussion

Investigating gastro-intestinal parasites showed there to be a significant correlation between parasite burden and a number of life history characteristics. *Capillaria* burden was significantly correlated with age of host, month of sampling and individual badger body condition index. *Eimeria melis* burden was significantly correlated to age of host and to gender. Unfortunately this correlation cannot infer cause and effect but is still of value to illustrate relationships and has therefore fulfilled the aim of investigating these characteristics.

AGE

This study found a significant relationship between age of host and both *E. Melis* (Table 1) and *Capillaria* (Table 3). Cubs had a higher prevalence of both parasites than adult badgers (Fig. 1 & 2). Evidence from this study therefore supports the hypothesis that cubs carry higher burdens of gastro intestinal parasites that has been found within multiple studies in the literature. Specifically; coccidial infections in rabbits showed a statistically significantly decrease of oocyst output correlating with an increase in age (Stodart 1968), and nematode infection studies in Romney sheep show that lambs exhibit lower faecal egg counts than sheep of 28 months due to the apparent acquired immune response and the ability of older sheep to suppress fecundity of the nematodes (Douche & Morum 1993)

As badger cubs are entirely fossorial until they are 8 weeks old and remain mostly underground until 10 weeks, the use of underground latrines until this time may act as a disease reservoir and aid easy transmission between cubs (Newman, MacDonald & Anwar 2001). Indeed, even the social dwelling of adult badgers underground is likely to contribute to transmittance of parasites with direct life cycles and infected adults may be the original source of infection to cubs (Newman, MacDonald & Anwar 2001). The relationship could also be explained by an acquired immunity to parasites in adults or even a high mortality rate of cubs with infections (Newman, MacDonald & Anwar 2001).

SEX

The data in this study showed that male badgers carry a significantly higher burden of *E. melis* infection (Fig. 1, Table 2) but not of *Capillaria* (Table 3). The relationship for coccidia coincides with the well documented effect of sex hormones on immunity. Whilst female hormones (in particular oestrogen) increase the resistance to parasites through stimulating humoral and cell mediated immunity, male sex hormones are said to be beneficial to parasites by suppressing immunity (Haukisalmi, Henttonen & Tenora 1988, Poulin 1996, Schalk & Forbes 1997).

Differences in behaviour, home range and diet between sexes may also lead to differences in exposure to parasites partially accounting for this relationship and differences in mating behaviour may alter stress levels possibly contributing to male vulnerability (Schalk & Forbes 1997).

The observed values for *Capillaria* infection are slightly more difficult to interpret due to mixed results in the literature. In this study the relationship was not significant, although there was an observed positive correlation between *Capillaria* infection and male badgers. Literature in the field reports mixed results for the relationship between helminth parasites and parasite burdens, which appears to be highly dependent on both the species of parasite and the species of host. Schalk and Forbes 1997 report from a review of

145 mammal host studies from both laboratory and field studies, that the majority showed a male bias in burden of parasites but not within the helminth taxa, which would correspond with the results found in this study of badgers and *Capillaria*. However, these same results do not correspond with a similar study which had a larger sample size and did show male bias to have an effect on helminth burden of mammals (Poulin 1996).

A possible explanation for this, again found in the study by Poulin 1996 found that body size has an influence on fecundity of helminths, possibly due to a larger body size and consequently a larger nutritional intake which could be advantageous to the parasite species. This may contribute to a male biased relationship due to the typical larger body size observed within mammals, and may explain why the European badger, with its gender morphometric similarity does not fit into this pattern.

SEASONAL VARIATION

This study showed a significant variance in monthly cycles of *Capillaria* burden (Fig. 2, Table 4). The faecal egg output was lowest in May and June before peaking in August and dropping again for September. Contrary to expectations the parasite burden appears to increase again in October (Fig. 2). Seasonal variation could be due to a multitude of factors. For example; there is a possibility that parasite burden variance could be directly explained by changes in food availability and types in which eggs might be found. This explanation is particularly relevant where parasites have indirect lifestyles and intermediate hosts are consumed as prey (e.g. earthworms which are implicated in the transmission of *Capillaria aerophila* (Anderson 2000)), however as the parasites investigated under this experiment are most likely be *C. putorii* (commonly found in mustelids and hedgehogs (Hancox 1980, Taylor, Coop & Wall 2007)) is considered to have a direct life cycle this is less likely, with transmission more likely accounted for in badgers by the social dwelling underground (Rogers, 1996, Roper 2010).

A more feasible explanation for seasonal variance is that the difference in food availability and nutrition has an effect on the condition and immune response of the badgers. As August was particularly dry in Gloucestershire in 2010, weather would have influenced food availability (particularly access to earthworms) and consequently contributed to the vulnerability of badgers to parasites through overall poor condition. In particular levels of nitrogen and phosphorus can affect the abundance of pathogens (Johnson *et al.* 2010) and Vitamins A and K are said to provide protection from parasites for the host organism (Fayer 1980). Changes such as these in the host's immunological mechanisms are a likely explanation for variation in helminth burden, although whether they are due to the aforementioned or to cyclic factors in the development of the host remain to be confirmed (Haukisalmi, Henttonen & Tenora 1988).

Research also shows that host organisms may be more vulnerable to parasites and the detrimental effects of heavy parasite burdens more pronounced in extreme conditions such as drought (Cassinello, Gomendio & Rolden 2001) or severe winters (Coltman *et al*. 1999). This is particularly the case when in combination with other stressors (Cassinello, Gomendio & Rolden 2001), where extremes of temperature and a heavy parasite burden have been shown to lead to increased mortality in a variety of research (Marcogliese & Pietrock 2011).

If this seasonal variation of *Capillaria* burden is a result of poor condition in some periods of the year then it may have important consequences for disease management, particularly if the pattern is relevant to other diseases such as bTb. Investigation into links between parasites and weather data would be useful further study, as would further investigation into seasonal variation of immune response.

Another explanation of seasonal distribution is a variation in transmission. It is argued that in most nematode species the rapid build up of infection that can occur at any time of the year is evidence against this. However a study in voles showed that the rarer helminth species have different life cycle and transmission patterns which may well be influenced by temperature and moisture and would explain the monthly variation seen in *Capillaria* abundance (Haukisalmi, Henttonen & Tenora 1988). These same factors combined with aerobia are also known to affect the sporulation of coccidia oocysts. As oocysts are not infectious until they sporulate, these factors must have a significant influence on transmission, although the extent of which will vary between species (Fayer 1980) and any evidence for seasonal patterns of coccidia in this study were not significant.

CONDITION

Badger body condition index was found to be significantly negatively related to *Capillaria* burden (Fig.5). A slight positive correlation with *E. melis* was observed but this was not significant (Table 1). The *Capillaria* relationship supports the energy allocation principal found in multiple studies, where animals that are fighting parasite infections are using valuable energy which would otherwise be invested in growth. Several other studies have also found a relationship between condition and parasite burden. For example Vandegrift, Raffel and Hudson 2008 found the use of anthelmintics increased body condition in white-footed mice, and Dawson and Bortolotti 2000 found male American kestrels exhibited a negative association between body condition and hematozoan parasites.

The positive trend between coccidia and BCI, although not significant was still observable and is surprising because as BCI takes into account both weight and body length the results conflict with a major study into coccidia in the European badger. Newman, MacDonald and Anwar 2001 found that higher burdens of *E.*

melis in cubs resulted in stunted head-body length. It is possible that within the context of this study there were not enough badgers with burdens of coccidia high enough above the critical threshold to have a symptomatic effect on the badgers, particularly as trapping was not possible in July, (which lies in the peak summer months for *E. melis* burden according to Newman, MacDonald & Anwar 2001) and that the positive relationship might be due to individuals as hosts providing a better environment for the parasites whilst in a good condition.

Although it is presumed in most studies that increased parasite burden is a result of poor immune system it is equally plausible that poor physical condition simply contributes to an individual's vulnerability to infection in the first place (Dawson & Bortolotti 2000). Regardless of cause and effect, the significant relationship observed in *Capillaria* burden is evidence for the principal that parasites impose competition for nutrients and energy resources.

HABITAT

The findings of this study found the relationship between both parasite types and habitat to be non significant (Tables 1 & 3). However this may be because of limitations in the definitions of habitats for badgers, as other research has shown a substantial link between environments and food resources to parasite burden (Stodart 1968). Habitat types for this experiment were based on rudimentary descriptions of sett location and did not take into account food availability, climatic condition or quality of habitat. In addition to this variation of habitat type was small, as the preferred location for most badger setts in the UK is within areas of woodland (Roper 2010) and within the setts sampled for the study there was little variation. Most social groups would have had plentiful access to food resources. Further research might include a wider study area with variation between habitat types and topography and a measure of food availability. Results would then be a better comparison to other literature and are likely to be linked to results for seasonal variation.

IMMUNE AND BTB

The impact of parasites on the overall immune system may also make the individual more vulnerable to other infectious diseases (Sheldon & Verhulst 1996). For example there is speculation that vulnerability to bovine tuberculosis, an extremely important disease economically for which badgers are known carriers could be at least partially linked to parasite burden as an individual has only limited resources for which to fight infection, and because the presence of co-infection may alter the immune response (Massey, Elsheikha & Morsy 2009). Should this be the case it would have important implications in the realms of wildlife disease management, affecting both individual and group dynamics. However results from cultures

for M. bovis found no correlation with either burden of *Capillaria spp.* or burden of *E. melis* (Table 5). Results from a similar study by Massey, Elsheikha & Morsy 2009 also found no significant relationships between parasite burden and *M. bovis*. However, although there is a wealth of research on badgers and bTb, little is actually known about their immune functions with relation to other diseases and this subject would warrant use of more sensitive bTb testing and a more longitudinal study.

CONCLUSION

Research on the effect of parasitism in laboratory animals is well documented, however care must be taken when trying to extrapolate these results and apply them to wild free-living animals as complex interactions are often unaccounted for (Marcogliese & Pietrock 2011). This study used free living animals to try and interpret the relationships of parasite burden in *M. meles* with multiple abiotic and biotic factors. Although using wild animals has obvious drawbacks within a scientific experiment (extraneous variables that cannot be controlled for as they would be in a laboratory). It is the opinion of the author that by using individually identifiable badgers most factors could be monitored and that the data obtained provides a valuable insight into the parasite ecology of the badger.

The study found similar relationships to previous literature with respect to the significance between age sex and seasonal variation, however as far as the author is aware the significance of body condition index within *M. meles* in relation to helminths is a new finding, although a previous study has found a link between coccidia and body length. Badgers were found to have extremely wide variations in parasite burdens, some of which were very high. I conclude that the prevalence of gastro-intestinal parasites would undoubtedly have an effect on the energy resource allocation of badgers and probably on the immune system capabilities. Parasite prevalence therefore should not be overlooked when wildlife disease management protocols or ecological studies of badgers are enforced. Further research into the overall immunology of badgers and a more longitudinal study of parasites would be beneficial to the field.

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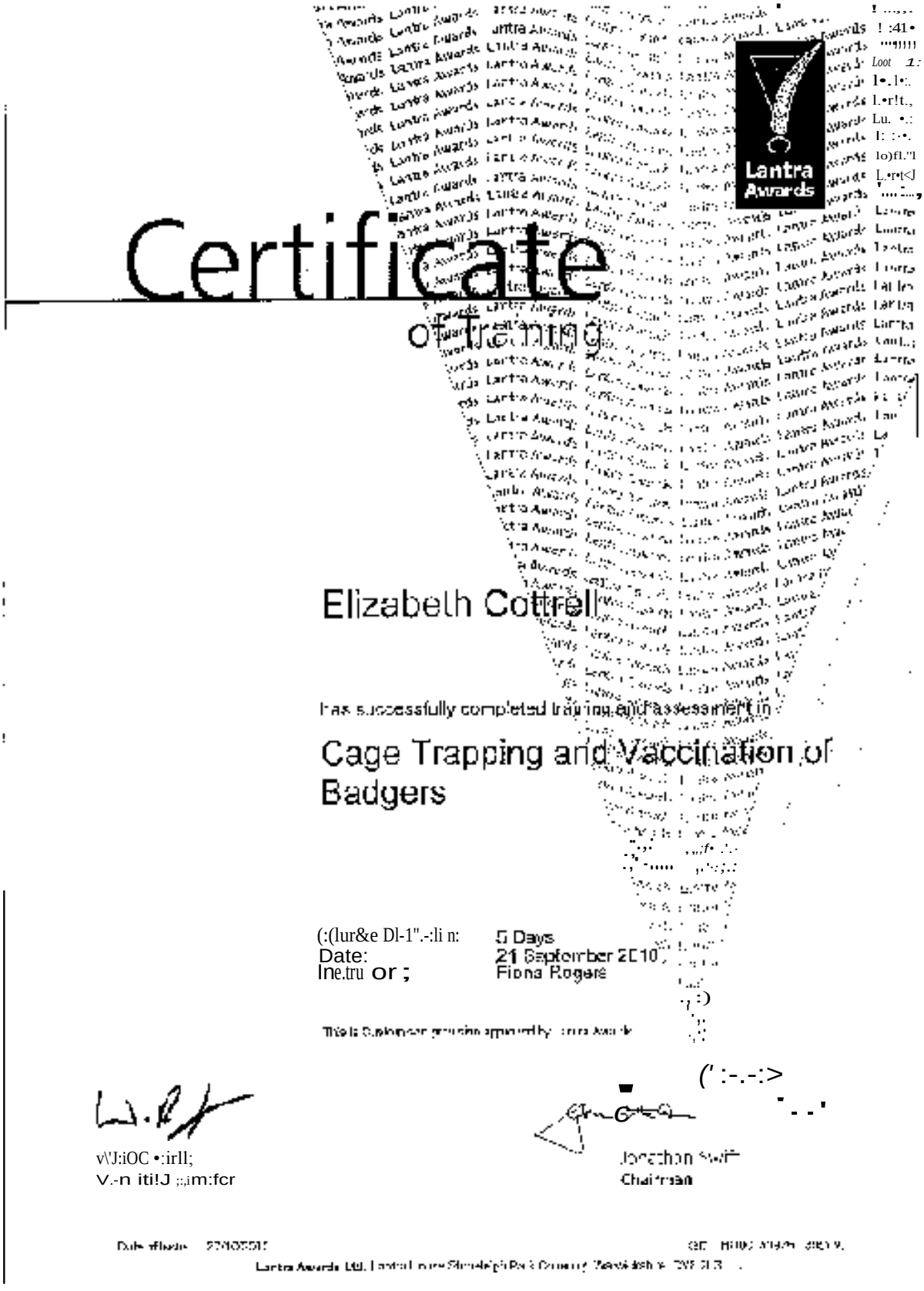
APPENDIX A - DETAILS OF ANAESTHETICS

Badgers were anesthetised via an intra muscular injection of a combination of 0.04mg/kg medetomidine hydrochloride (1mg/ml Domitor, Pfizer), 8mg/kg ketamine hydrochloride (100mg/ml Vetalar V. Pharmacia & Upjohn) and 0.8mg/kg butorphanol tartate (10mg/ml Torbugesic, Fort Dodge, Animal Health)(de Leeuw *et al.* 2004).

APPENDIX B - FURTHER SAMPLES

Animals used in this study were trapped as part of a long term research project investigating the social structure of badgers and TB prevalence (Woodchester Park badgers) or as part of a research programme to develop the oral vaccination for TB (other Gloucestershire badgers). Consequently other samples were taken that were not used within this investigation. At Woodchester; further tests were carried out on the samples taken for *M.bovis* culture using STAT-PAK® (Chambers *et al*. 2008) multi-antigen test and IFN-λ ELISA tests. Breeding status (determined by lactation or descending of testicles), toothwear (on a four point scale) and temperature (taken from rectum) were also recorded. In the Gloucestershire badgers blood samples were taken to determine uptake of bait. Hair and whisker samples were also taken from both areas for stable isotope analysis.

Training Certificate in Badger Trapping and Vaccination



Certificate of training

Elizabeth Cottrell

has successfully completed training and assessment in

Cage Trapping and Vaccination of Badgers

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Managing Wildlife Disease – A Literature Review

E. R. A. Cottrell [1, 2]

[1]Food and Environment Research Agency, Woodchester Park, Tinkley Lane, Nympsfield, Gloucestershire, GL10 3UJ; and [2]Centre for Ecology and Conservation, University of Exeter Cornwall Campus, Tremough, Treliever Road, Penryn, Cornwall, TR10 9EZ.

Introduction – What Is Wildlife Disease?

Disease is present in all ecosystems: it is an integral, natural component of ecological communities (Delahay, Smith & Hutchings 2009). An important factor in population biology, it affects survival, reproduction, dispersal, community structure and genetic diversity (Berger, Speare & Hyatt 1999). Under normal circumstances a balance is maintained between host species and pathogens or parasites (Delahay, Smith & Hutchings 2009). It is only in some cases, when additional pressure is put on populations, perhaps through environmental degradation or the facilitated travel of novel pathogens (Berger, Speare & Hyatt 1999) that this balance is altered and intervention is perhaps necessary to manage disease in wildlife populations (Wobeser 2002). This review will outline such situations, including the causes and the incidences in which it is necessary for management to occur. It will also evaluate the management strategies used and the potential and known impacts on wildlife, ecosystems and the economy.

Disease, as a wide definition, can be referred to as any impairment of normal functions (Delahay, Smith & Hutchings 2009). However this definition does not exclude injury or poisoning and for application to wildlife disease management is insufficient. More important for the purposes of management and to this review is infectious disease. This can be defined as disease caused by pathogens (infectious agents), either transmitted directly between organisms or with indirect contact through the environment or through vectors. Pathogens may be microorganisms such as bacteria, fungi, protozoa and viruses, or they may be multicellular organisms (parasites) such as helminths or arthropods (Delahay, Smith & Hitchings 2009). Pathological change may be caused by infection, stress or any other factor which disrupts the natural balance of an ecological relationship, producing characteristic symptoms and illness, and is the primary concern of contagious wildlife situations that require management.

Why Manage Disease?

Management of disease in wildlife populations is a fairly recent concept when compared with the history of disease management in humans and domesticated animals. The belief that an infection can be eliminated or its circulation in wild animals restricted, is due in part to recent developments in the field of ecological epidemiology (Artois *et al*. 2001). In the past, infectious disease in free–living animals only received attention during major events of consequence to the health of humans (Wobeser 2002). Even now, where disease management is undertaken in the majority of cases it is because of potential threat to some aspect of human life, such as human health, health of domestic animals or animals highly valued by humans (Wobeser 2002). Management therefore, is unlikely to occur unless the disease present in wildlife is damaging to humans, either physically or economically, or perhaps if we feel a moral responsibility. Even so, the implementation of control measures to wildlife disease should not be undertaken lightly as the presumption that intervention will benefit wildlife is over simplistic (Artois *et al*. 2001) and it may do more harm than good if poorly planned or under funded (Woodroffe 1999).

The reasoning behind intervention is extendable to multiple situations; one of which is when a wild species acts as a reservoir for infectious pathogens (Artois *et al*. 2001), which can subsequently prevent the elimination of the disease from domestic animals (Wobeser 2002). For this to become a problem in terms of veterinary public health, a pathogen needs to be sustained and transmitted in the wild-host population and passed regularly to domestic stock or humans (Artois *et al*. 2001). For example, such is the case of the pathogen *Mycobacterium bovis*, the cause of bovine tuberculosis, in the United Kingdom. A combination of factors has led to the recurrence of tuberculosis in livestock, which is increasingly economically damaging, costing the UK government over £100,000,000 a year, and in 2008 alone was responsible for the breakdown of 2378 herds of cattle, profoundly damaging the livelihoods of farmers (Jenkins, Woodroffe & Donnelly 2010). One of the causes of its persistence is likely to be the wildlife reservoir in badgers (*Meles meles*) and deer living in close vicinity to livestock (Delahay *et al*. 2007, McDonald *et al*. 2008).

It should be stressed however that evidence of disease in wild animals is not necessarily evidence of their existence as a reservoir (Artois *et al*. 2001). Evidence of *M. Bovis* has been found throughout Mammalia in the United Kingdom, yet it is highly unlikely that certain species come into close enough contact to transmit infection to livestock, for example the common shrew (*Sorex araneus*), grey squirrel (*Sciurus carolinensis*) and wood mouse (*Apodemus sylvaticus*) (Delahay *et al*. 2007). It is also likely that infected wildlife populations can coexist with livestock and not risk cross-infection, such is the case in wild boars (*Sus scrofa*) and the transmission of the classical swine fever virus, CSF (Artois *et al*. 2002). Although wild

species may act as a vector for disease, the primary reservoir may well be within the livestock undetected (ISGC 2007, Artois *et al*. 2001, Woodroffe *et al*. 2006).

Another situation where disease management is readily engaged is in cases where disease threatens a species, either of great importance (ecological, economical or political) or which is endangered. Such is the case in species such as the Tasmanian devil (*Sarcophilus harrisii)*, the largest surviving marsupial carnivore, which suffers from Tasmanian devil facial tumour disease (DFTD) (Lachish, Jones & McCallum 2007) or the Ethiopian wolf (*Canis simensis*) the world's rarest canid, threatened by rabies (Haydon *et al*. 2006), where special care must be taken prevent the eradication of species, either directly through disease or through any implemented disease management (Delahay, Smith & Hutchings 2009).

A final rationale for disease management is the feeling of a moral responsibility to act. For instance with diseases either directly or indirectly caused or worsened by humans. An example of such a situation is where habitat destruction adds additional pressure to an already struggling population and increases the susceptibility of individuals to a certain disease. This is the case in the Australian koala (*Phascolarctos cinereus*) which becomes more vulnerable to diseases such as *Chlamydia* when extra stress is brought about by habitat fragmentation (Augustine 1998, Phillips 2000). Habitat destruction also affects the distribution and density of populations perhaps forcing animals to live closer to one another than they would naturally, therefore facilitating disease transmission and may limit access to normal food sources. It may also alter natural social structure causing further dispersal or alterations to behaviour and may isolate populations to an extent that through inbreeding depression, immune systems become compromised. In the case of the Tasmanian devil this lack of genetic diversity has caused extreme vulnerability to the infectious cancer DFTD which now threatens extinction (Lachish, Jones & McCallum 2007, McCallum 2008). Another example of human impact aggravating a disease and a potential moral obligation is through facilitating the transport of the pathogen. The rapid global transport of people, goods and animals will assist in the dissemination of infectious agents both between and within continents (Berger, Speare & Hyatt 1999) and this is likely to unbalance potentially stable natural disease systems. It is likely that the spread of chytridiomycosis in amphibians, and consequently the threat of extinction to the majority of amphibian species worldwide has been worsened by such human activity spreading this fungus to further vulnerable populations (Skerratt *et al*. 2007).

Methods of Disease Management

Once the decision to manage disease has been made, implementing disease control is not as simple as deciding upon a method and carrying out a plan, as multiple factors always need to be taken into consideration. Selection of the appropriate management technique to control infection requires in depth

understanding of the nature and ecology of the disease, which must include the cause, pathogen-host interaction, the diseases course and its effects on both the individual and the population (Wobeser 2002). Selection of the management protocol will also be greatly dependent on the reasons for managing the disease itself. For instance a strategy to eliminate a disease in endangered species will have greatly different priorities to that of a disease carried in what is considered to be a pest species or a non-endangered disease reservoir.

In principle, the control of infectious disease can be realised by multiple strategies, which can be classified into four categories: prevention, control, eradication and doing nothing. When using reactive methods such as control or eradication the disease agents can be targeted within the host, free in the external environment or in some vector or alternative host (Wobeser 2002). In practice this control can be divided into two principals; limiting the number of susceptible individuals and reducing the transmission between or to those individuals (Matthews *et al*. 2003).

The methods in which these strategies can be applied are summarised into three sections below; reducing population density, medication and isolation with captive husbandry.

REDUCING POPULATION DENSITY

The first method of disease control is to reduce the population density of the host. Logically, it would follow that by reducing population size the number of infected and susceptible individuals is reduced, resulting in a lower incidence of the disease. Once the population density is reduced below the critical threshold at which transmission can occur the disease should then disappear (Artois *et al*. 2001), or reach a more tolerable level within the population. It is important however to continue to monitor the population following the implementation of disease control in order to prevent a subsequent recovery of population numbers that may in turn make animals more susceptible to the original disease. This effect was observed in previous rabies control programmes (Smith & Cheeseman 2002).

CULLING

Culling, or lethal control, is perhaps the most frequent method historically used to reduce population size of host species. Although theoretically it can be a cheaper and potentially effective way of reducing disease transmission, in most cases where monitored it appears to have had mixed effects and not to be the most cost effective solution. Culling can be achieved through a multitude of measures including gassing, shooting, poisoning and trapping (Artois *et al*. 2001). During the decision process when considering lethal control techniques, it is of great importance to consider both the welfare to the animals involved directly and also any indirect effects on non target species or the environment, such as changes in predator-prey

systems (Ritchie & Johnson 2009) or unintentional uptake of poison. It is also important to consider the long term repercussions of the cull such as a reduced gene pool, or as in some situations, that attempts to reduce population size might be offset by compensatory reproduction and immigration (Artois *et al*. 2001, Baker & Harris 2005). It is also worth mentioning that in implementing culling, an artificial situation is created in which the population is unstable, growing and with a young biased population which could potentially lead to a population becoming unusually mobile, in turn accelerating the spread of disease (Holmala & Kauhala 2006). It may be that in order to be effective culling needs to be partnered with other methods to reduce recruitment rate such as fertility control (Artois *et al*. 2001). If this method of disease control is to be implemented then it may be important that it occurs early on in the epidemic, before the geographical range of the infection has spread. In Tasmanian devils with DFTD, the infective nature of the disease was realised too late and to carry out a lethal control programme would now be ineffective and almost impossible without wiping out the whole species (McCullum 2008). Lethal control can be divided into two categories;

Proactive Culling

The aim of proactive culling is to reduce the population size indiscriminately across a wide affected area. In most cases it is desirable to remove as large a proportion of the host species as possible; in others it may be desirable to just reduce the population to the threshold density of disease transmission (ISGC 2007). This method was used to reduce red fox (*Vulpes vulpes*) densities in Europe (Smith & Cheeseman 2002) and was trialled in the UK to reduce the population of badgers (ISGC 2007).

Reactive Culling

Reactive culling is undertaken as a response to a confirmed disease outbreak, perhaps in a local livestock herd. The aim would be to remove all animals with access to the infected area (ISGC 2007).

This type of culling is used commonly within livestock for severely contagious diseases (Matthews *et al*. 2003). In recent history it was used with success to control the 2001 foot and mouth epidemic (a rapidly spreading disease caused by a highly contagious aphthovirus) alongside strict movement controls (Ferguson, Donnelly & Anderson 2001, Hutber, Kitching & Conway 1999). Also with the 2003 avian influenza epidemic, where infected poultry was culled, alongside pre-emptive culling of flocks which had been in contact with infected birds (Stegeman *et al*. 2004). However both these diseases had limited spread to wildlife populations (perhaps due to the swift measures taken to eliminate infection from domestic animals) and therefore there was no need to control free living animals.

Culling in relation to badgers and bovine tuberculosis

Bovine tuberculosis is a serious disease of cattle (McDonald *et al*. 2008, ISGC 2007) with a wide range of mammalian hosts (Delahay *et al*. 2007, McDonald *et al*. 2008). Test and slaughter programmes have been used with success in many countries but a persistent problem still lingers in areas of New Zealand, Ireland and the United Kingdom (McDonald *et al*. 2008). It is particularly a problem for British farmers (ISGC 2007), where in 2006 alone disease control restrictions were in place on 4% of Great Britain's 90,000 herds, subjecting 22,000 cattle to compulsory slaughter, costing tax payers in excess of £80,000,000 on testing, researching and compensating farmers (McDonald *et al*. 2008), this cost has increased in subsequent years and in 2008 cost in excess of £100,000,000 (Jenkins, Woodroffe & Donnelly 2010).

The reoccurrence in disease has largely been implicated to the European badger *Meles meles* (Woodroffe *et al*. 2006, ISGC 2007) a widespread but protected mammal (Protection of Badgers Act 1992, Woodroffe *et al*. 2006). This implication led to the Randomised Badger Culling Trial (RBCT) in 1998. The RBCT was conducted in 30 *M. bovis* high risk areas over England. Annual culling of >20,000 badgers was carried out in the two categories, proactive and reactive culling alongside a third control group (Woodroffe *et al*. 2006, ISGC 2007). Controversially this trial led the Independent group on cattle TB to believe that although it appears badgers transmit *M. bovis* to cattle and vice versa, culling in some cases could make the disease problem worse due to weakening in the social structure of badgers leading to increased perturbation of potentially infected badgers (ISGC 2007, McDonald *et al*. 2008, Tuyttens & MacDonald 1998). It was concluded that culling actually exacerbated the spread of tuberculosis in the surrounding areas of the cull (ISGC 2007) and within culled areas the initial reduction of *M. bovis* was not sustained. Furthermore, the economic benefits did not offset the costs of culling (Jenkins, Woodroffe & Donnelly 2010). This finding reinforces the need for intensive research on wild animal movements before implementing controls.

Culling in relation to foxes and rabies

Rabies is a highly fatal disease which can infect all species of mammals, including humans and domestic animals, although it appears some species are more susceptible than others. In Europe rabies is historically common in red foxes and more recently has been noted to be on the increase in raccoon dogs (*Nyctereutes procyonoides*) particularly in Estonia (Holmala & Kauhala 2006). Rabies occurs in all continents except Antarctica, although some countries and islands have remained rabies free through strict import regimes and eradication programmes (Holmala & Kauhala 2006).

The spread of rabies is partially determined by population density, quality of habitat and social organisation. As the fox lives in pairs or family groups with overlapping territories it is vulnerable to

infection. Where foxes share territories with racoon dogs with rabies, risks become increased and combined populations exceed the threshold density for disease (Holmala & Kauhala 2006).

Traditionally, attempts to eradicate rabies were undertaken with the mindset that if enough foxes were destroyed then eradication of the virus would follow. Hunting, poisoning and gassing of dens was carried out in Europe for decades (Smith & Cheeseman 2002, Holmana & Kauhala 2006). Unfortunately this failed to reduce the density of susceptible individuals below the critical threshold, and did not prevent the spread of rabies in continental Europe (with the exception of Denmark which is of a peninsular nature) (Holmana & Kauhala 2006).

It may be that the failure of culling in most wildlife diseases, despite models predicting success (Barlow 1996) is due to increased migration or perturbation of animals or compensatory reproduction (Smith & Cheeseman 2002, Tuyttens & MacDonald 1998). Social instability and the reestablishment of territories may also increase the frequency of bites between individuals and therefore increase the chance of disease transmission (Holmana & Kauhala 2006). It is clear that many factors need to be understood fully before carrying out a lethal control method of disease management.

FERTILITY CONTROL

Recently fertility control has arisen as a method of controlling disease; previously its use was limited to a method of growth control for populations which became larger than the desired levels, either in wildlife parks or situations creating some form of conflict (with humans, other wildlife or environmental). In terms of reducing disease it would work in principally the same manner as culling, by reducing the population density and consequently the levels of infection within a population. Furthermore depending on how disease dynamics are influenced by reproductive investment and the age structure of the host, fertility control could offer epidemiological advantages (Tuyttens & MacDonald 1998). For the purposes of this review fertility control excludes surgical intervention, which is considered largely impractical to wild ranging animal populations and refers only to the management of birth rates through manipulation of hormones and their derivatives, or through immuno-contraceptives (van Aarde & Jackson 2006).

Fertility control has been used with some success to reduce the size of several wild populations, for example; of elk (*Cervus elaphus)* with use of gonadotropin-releasing hormone GnRH with no observable difference in reproductive behaviours or lasting body condition (Conner *et al*. 2006), of wild horses and deer with no immediately observable social organisation or behavioural change (Kirkpatrick *et al*. 1997) and of levonorgestrel implants in experiments with grey kangaroos (*Macropus giganteus*) with no observed change in females in time spent in allocated to most normal activities. (Poiani *et al*. 2002). It is also used

widely as a method of reducing African elephant numbers (van Aarde & Jackson 2007). Use of fertility control is also being researched for use with the European badger, which in urban environments creates conflict (Davison *et al*. 2008) but as a protected species cannot be managed with lethal control.

There are now also several cases where this fertility control induced reduction in population size and decreased intra-species contact could lead to a lowered disease incidence. For example in the brushtail possum (*Trichosurus vulpecula*), a solitary species where contact between individuals is rarely made other than for mating encounters. Evidence based on surgical sterilisation (as a model for fertility control that blocks endocrine control) showed a reduced transmission of the common pathogen *Leptospira interrogans* serovar *balcanica* by 88% in males and 67% in females (Ramsey 2007).

An advantage of this approach is that in most situations fertility control is considered to be more humane and ethical than the alternative lethal control (Hardy & Braid 2007, Kirkpatrick & Turner 1985, Poiani *et al*. 2002, Tuyttens & Macdonald 1998), and although a lower reproductive output may have effects on the social structure of a population it is likely that these effects are less severe than the compensatory immigration which can occur as a result of culling (Smith & Cheeseman 2002). Additionally fertility control when used within an intelligent management scheme has the benefit of being targeted and reversible whereas lethal methods of management permanently remove genes from the gene pool, altering the natural selection process (Kirkpatrick & Turner 1985).

However the delivery of contraception can be labour intensive and costly (although much reduced if orally delivered as proposed in Smith & Cheeseman 2002), in many cases it also requires frequent boosters, which in turn incur the issues of recapture and identification (van Aarde & Jackson 2006).

Fertility control may also have undesirable side effects and energy costs on health and natural behaviour of the target organism, for example hormonally treated animals may remain in sexual heat and be subjected to harassment by males (van Aarde & Jackson 2006), animals may experience loss of libido or produce sterile young (Kirkpatrick & Turner 1985), animals may lose their territories (Ramsey 2007) or reducing reproductive rates may have consequences on the stability of the age and social structure of the group, potentially influencing the well being of individual (van Aarde & Jackson 2006).

Models show that alone, fertility control is a slower, less effective method of controlling disease than culling (Barlow 1996, Holmala & Kauhala 2006) as it relies on natural mortality to reduce population density (van Aarde & Jackson 2006), but when combined with culling or vaccination it is more effective than the alternatives (Smith & Wilkinson 2003) and it is of use where culling is perceived as too controversial or is illegal (Kirkpatrick *et al*. 1997).

MEDICATION

Cases of direct medical treatment of wild populations are infrequent because of the substantial practical difficulties of handling wild animals and because of the high cost of veterinary drugs. Nevertheless in some cases where individual animals are valuable or endangered medical treatment has been used with success (Delahay, Smith & Hutchings 2009). Examples of such occasions include the treatment of illnesses of the mountain gorilla (*Gorilla gorilla beringei*) where habituation for research purposes has increased vulnerability to human infections, of mange in cheetahs (*Acinonyx jubatus*) (Delahay, Smith & Hutchings 2009), and of lungworm in big horn sheep (Schmidt *et al*. 1979). Treatment options for *Chlamydia* in the Australian koala are also being investigated (Griffith 2010).

A more commonly used approach than direct treatment to an individual is to vaccinate the population.

VACCINATION

A well documented method of disease control is immunisation (Artois *et al*. 2001, Matthews *et al*. 2003). It is argued that as mass immunisation has had a dramatic effect on the spread of disease in humans and domesticated animals, and that providing the correct vaccines are available and delivery is possible it should therefore function well on free living animals (Wobeser 2002). The purpose of vaccination is to render the individual resistant to an infectious agent (Wobeser 2002), either with the intention of benefitting the individual, for example the successful vaccination of roan antelope (*Hippotragus equinus*) against environmentally borne anthrax (de Vos, van Rooyen & Kloppers 1973). Or to reduce transmission within the population and consequently the spread of disease, such as with rabies in the red fox (Holmala & Kauhala 2006, Smith & Wilkinson 2003) and bovine tuberculosis in the European badger (Corner *et al*. 2009), where vaccination programmes have been initiated in order to prevent the spread of infection to other organisms.

Vaccination has several advantages; whilst (unlike culling) animals are not removed from the social group and so the natural network of territories is preserved, it also has a lesser disturbance of the natural population dynamics, although there may be unknown effects from trapping distress and from a reduction of disease induced mortality. It is also vastly more publicly acceptable from an animal welfare point of view (Peterson, Mertig & Liu 2006), and may be an extremely valuable tool for use in disease management in endangered species, or where individual animals are valuable. Similar to all methods of disease control, the necessary number of target animals should be planned beforehand. Failure to immunise enough animals could result in a failure of eradication or of a resistance to the disease developing, allowing endemicity to persist and a return of disease to unmanageable levels (Artois *et al*. 2001). Consequently it is also

important when vaccinating that the population continue to be monitored after disease control is implemented (Smith & Cheeseman 2002).

The effectiveness and success of vaccination, as with any type of disease management, may depend on multiple factors, each varying greatly depending on the nature of the disease and of the host, for instance whether the disease is acute (short term but severe) or chronic (long term or reoccurring), and characteristics of the host species such as replacement rate and lifespan (Smith & Cheeseman 2002). Populations with a higher density or species with a higher recruitment rate will require a larger proportion of animals to be vaccinated and a more prolonged programme (Wobeser 2002). In situations where a disease is acting as an important population control, vaccination may work best when complemented by another type of control such as fertility control (Smith and Wilkinson 2002).

Unfortunately the development of a vaccine itself is not always possible, in the case of DFTD it is unlikely that a vaccine is feasible because vaccinations against cancer have rarely been achieved (McCullum 2008). Even where immunisation is possible, delivery to a wild population is challenging and expensive (McCullum 2008), although sometimes cheaper than the alternatives. For example vaccination against *M. bovis* in badgers is likely to be more cost effective than culling (ISGC 2007, Jenkins, Woodroffe & Donnelly 2010), and in the case of rabies in foxes in France it became more cost effective than culling after four years (Holmala & Kauhala 2006). Consequently it should not be dismissed as a viable method of disease management.

Methods of Delivering Vaccine

In most situations practicality prevents the individual capture and handling of wildlife for immunisation, although this may be possible in situations where the number of animals is small, they are highly valued (Wobeser 2002), or animals/setts are easily located in the wild (Delahay *et al*. 2003), and such situations have led to past successes (Smith & Wilkinson 2003). Vaccine can be delivered via injection or recently and more efficiently by oral bait (Artois *et al*. 2001, Holmala & Kauhala 2006, Smith & Wilkinson 2003, Wobeser 2002). Oral vaccination was first used in Switzerland in 1978 as a response to the rabies virus outbreak. Seven European countries have since become rabies free as a consequence (Holmala & Kauhala 2006), and analyses of the vaccine in foxes in France suggest a successful eradication of the virus where previous lethal control produced only a "transient lull" (Aubert 1999).

Using baited vaccine delivery instead of injected reduces the labour cost and intensity of vaccination programmes and eliminates the possible trap stress, which could potentially lower the immune system of the target organism (Delahay *et al*. 2003). However, the effect of any non target species inadvertently

ingesting the bait must be considered, and it must be ensured that excess bait or deployment does not damage to the environment (Wobeser 2002). Consequently bait development is normally a lengthy, expensive process, likely to be restricted to use in a small number of serious diseases (Cagnacci *et al*. 2007, Wobeser 2002). For instance diseases that threaten a species considered of value to humans such as the parapox virus in the red squirrel (Sciurus vulgaris) where oral vaccination may be a viable solution (Tompkins *et al*. 2001).

ISOLATION, CAPTIVE BREEDING AND INSURANCE POPULATIONS

When a whole population is threatened with the risk of extinction, a first step to ensure survival of the species is to isolate uninfected individuals in captivity and through captive breeding maintain an insurance population from which healthy individuals could be released into the wild (McCullum 2008), either after resistance to the disease has been bred or once the threat has passed. This method has been used to some success in several species such as the Arabian oryx, Californian condor and the black-footed ferret (Frankham 2008) but runs the risk of altering behaviour through a change in natural selection criteria whilst living in captivity, and also of inducing a genetic bottleneck (Frankham 2008, McCullum 2008). Insurance populations do however function well as a short term solution where options are limited. Such is the case in Tasmanian devils (McCullum 2008) and with Chydriomycosis in amphibians (Berger, Speare & Hyatt 1999) both of which are threatened with extinction through disease. Another option outlined by McCullum 2008 is the possible introduction of the threatened species (in this case the Tasmanian devil) to islands where they could continue to function within their wild niche, thus avoiding the issues of captive breeding. Although creating new issues in relation to the ethics of introducing a novel species to a naïve island population, even if they have an empty niche for a similar species type. Protecting populations with physical barriers in situ is also an attractive option (McCullum 2008) and has been used successfully in cases of chronic wasting disease in the United States of America (Vercauteren, Lavelle & Hygnstrom 2006) and to prevent the spread of CSF into wild boar (Artois *et al*. 2002), but is an expensive option and the displacement of natural migration may have long term undesirable effects.

CAPTIVE BREEDING AND CHYTRIDIOMYCOSIS IN AMPHIBIANS WORLDWIDE

Amphibians have recently undergone what is described as “the most spectacular loss of biodiversity due to disease in recorded history” on a global scale, and evidence points to the probable extinction of some 122 species and the rapid decline of another 435 since 1980 (Skerrat *et al*. 2007). Declines have primarily been attributed to habitat disturbance (approximately half of the declining species) (Skerratt *et al*. 2007), caused by acts such as logging, wetland degradation, cattle damage, fish introduction and pollution (Berger, Speare & Hyatt 1999). Yet the remaining 202 species have likely experienced decline due to the spread of

chytridiomycosis, caused by *Batrachochytrium dendrobatidis,* a fungal infection affecting in particular, stream dwelling isolated populations (Berger, Speare & Hyatt 1999). This waterborne fungal infection is highly transmissible and fatal to adult or newly emergent frogs (Berger, Speare & Hyatt 1999), the spread of which may have been exacerbated by movement of the pathogen aided by human transport (Skerratt *et al*. 2007), either through intentional trade of frogs or unintentional movement in fruit or cargo (Berger, Speare & Hyatt 1999).

Australia, (the likely source of the original *Batrachochytrium* fungus (Berger, Speare & Hyatt 1999)), in an attempt to prevent the extinction of the amphibian population, has implemented a disease management strategy which includes quarantine, recovery projects and extended research on determining distribution, preventing the spread and understanding the epidemiology and pathogenesis (Skerratt *et al*. 2007). Such research is essential if any attempt to prevent further declines is to be realised and is equally important in the planning of captive husbandry which may lead to breeding disease resistant individuals and to reintroduction (Skerratt *et al*. 2007).

However it would appear that globally Australia is well ahead in the recognition of the threat of chytridiomycosis and if the further spread of disease and amphibian decline is to be prevented there needs to be a cooperative global strategy of adequate quarantine, surveillance and control reached quickly and efficiently (Skerratt *et al*. 2007).

Conclusion

Wild animals by definition are free living and consequently controlling or treating them is more difficult than with captive animals such as livestock. Access is more challenging to these animals and assessing the state of disease in individuals and the population is considerably more difficult (Artois *et al*. 2001).

Management is however not always necessary and should not be undertaken lightly as unseen effects may make the spread of disease worse. Where management is necessary there is no perfect means of completing the task and thorough research is necessary into the potential effects on the targeted population and side effects on other species and the environment before carrying out a plan. Surveillance such as this is incredibly difficult to obtain, particularly where funding is not present or in countries where data is not published (Artois *et al*. 2001).

A variety of strong evidence suggests that to manage disease effectively in wild populations an approach involving a combination of control methods will be the most effective. As in most cases decisions about which type of management protocol to employ are ultimately guided by funding allocation rather than the ideals of wildlife professionals (Artois *et al*. 2001), cost effectivity can become a higher priority than other

factors. However the development of oral bait has the potential to reduce labour costs in disease management substantially whilst also being a very effective means of delivering control. It should not therefore be overlooked as a method of delivering vaccination and fertility control on the future, two methods which I regard to be at the forefront of managing wildlife disease.

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